SurVive

First Aid

Student Resource

Table of Contents

Environmental

Poisoning

SURVIVE STUDENT RESOURCE

Version 1.5 (May 2021)

Welcome to the Survive First-Aid Student Resource.

Getting Started

To navigate through this resource, simply click on the stacked icon in the top left of the page. You will then be able to see a menu of all topic areas on the left. You can also search from this menu.

Content

Content in this this booklet is sourced from public downloads and open-source information provided by the Australian Resuscitation Council (ARC). The content has not been altered in any way, only presented as a single document.

All content can be found at:

Australian Resuscitation Council

Additional content is sourced from public downloads and open-source information provided by:

- Wilderness Medical Society
- Asthma Australia
- Allergy and Anaphylaxis Australia
- Stroke Foundation Australia
- The Heart Foundation
- The Centre for Clinical Interventions (CCI)
- The Black Dog Institute
- Wilderness Medicine Society
- The Australian Venom Research Unit
- Australasian Society of Clinical Immunology and Allergy Ltd (ASCIA)
- The Asthma Foundation of Victoria

- RAW Medicine
- BICORescue

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PDF

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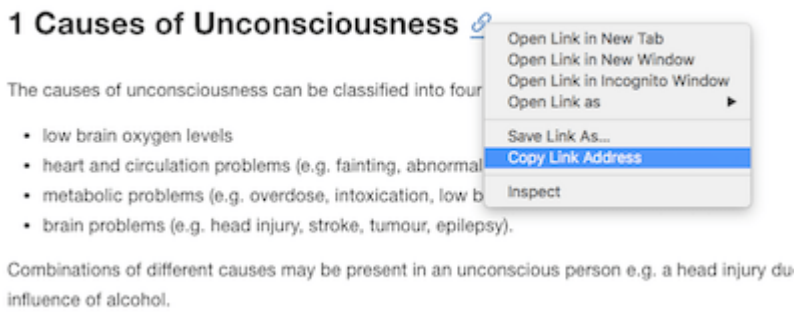
Links

Every heading in this book is linked. If you are communicating with your instructor about a particular topic, you can link the nearest heading by right-clicking (or long press on mobile/tablet) to the right of the heading, and copy the link as shown in the image below. The link can then be pasted in an email e.g. https://survive-student-resource.austererisk.com/general/anzcor_3_unconscious_victim.html#1-causes-of-unconsciousness



Support

- Clinical: adam@survivefirstaid.com.au

- Technical: Austere Risk Knowledge Base

AUSTERE RISK
MEDICAL | SAFETY | RESCUE

Change Log

v1.5 (May 2021)

Added the following resources:

- ANZCOR Guideline 9.2.12 - Recognition and First Aid Management of the Seriously Ill Person Including Sepsis
- ANZCOR Guideline 9.2.11 - First Aid Management of the Agitated Person
- Survive First-Aid Pocket Handbook:
  - Evidence-based Guide to Heat Stress
  - Assess the Cold Patient
  - Care for the Cold Patient

v1.4 (May 2021)

Updated the following guidelines to reflect ANZCOR changes:

- ANZCOR BLS Flowchart
- ANZCOR Guideline 2 - Managing an Emergency
- ANZCOR Guideline 3 - Recognition and First Aid Management of the Unconscious Person
- ANZCOR Guideline 4 - Airway
- ANZCOR Guideline 5 - Breathing
- ANZCOR Guideline 6 - Compressions
- ANZCOR Guideline 7 - Automated External Defibrillation in Basic Life Support
- ANZCOR Guideline 8 - Cardiopulmonary Resuscitation
- ANZCOR Guideline 9.1.1 - First Aid for Management of Bleeding
- ANZCOR Guideline 9.1.5 - Harness Suspension Trauma - First Aid Management
- ANZCOR Guideline 9.2.1 - Recognition and First Aid Management of Suspected Heart Attack
- ANZCOR Guideline 9.2.2 - Stroke
- ANZCOR Guideline 9.2.9 - First-aid Management of a Diabetic Emergency
- ANZCOR Guideline 9.2.10 - The Use of Oxygen in Emergencies
- ANZCOR Guideline 9.3.5 - Resuscitation and First Aid for Divers who have Breathed Compressed Gas
- ANZCOR Guideline 9.4.1- Australian Snake Bite
- ANZCOR Guideline 9.4.2 - Australian Spider Bite

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v1.3 (March 2020)

Added the following guidelines:

- Wilderness Medical Society Clinical Practice Guidelines for the Treatment and Prevention of Drowning: 2019 Update
- Wilderness Medical Society Clinical Practice Guidelines for Diabetes Management
- Wilderness Medical Society Clinical Practice Guidelines for the Out-of-Hospital Evaluation and Treatment of Accidental Hypothermia: 2019 Update
- Wilderness Medical Society Clinical Practice Guidelines for Water Disinfection for Wilderness, International Travel, and Austere Situations

Updated the following guidelines:

- Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Heat Illness: 2019 Update
- Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Treatment of Frostbite: 2019 Update
- Wilderness Medical Society Clinical Practice Guidelines for Spinal Cord Protection
- Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Treatment of Acute Altitude Illness: 2019 Update

EVACUATION GUIDELINES

- Please be aware that these are recommendations only.
- They are based on best-practice and the most current available information.
- Every first-aid scenario is dependent on responder training, patient presentation, environmental considerations and available resources- all of which should be taken into account and form part of your treatment and evacuation plan.

RAPID EVACUATION FOR ANY UNRESPONSIVE PATIENT

PRESENTATION	CONSIDER EVACUATION	CONSIDER RAPID EVACUATION
Unresponsive-CPR	RAPID	Rapid evacuation for all unresponsive patients (including deteriorating LOC) and unstable vital signs
Unresponsive-Breathing	RAPID	Rapid evacuation for all unresponsive patients (including deteriorating LOC) and unstable vital signs
Spinal Injury	All patients with relevant MOI	All patients with relevant MOI and any signs and symptoms of spinal injury
Shock	All patients with injuries significant enough to cause uncontrolled shock	Deteriorating patients, uncontrollable bleeding, airway problems and unstable vital signs
Abdominal	Sudden unexplained onset of severe pain or constant unexplained pain lasting more than 24 hrs	Blood in urine, faeces or vomit; any acute abdo pain associated with women of childbearing age; any complications of pregnancy
Anaphylaxis	Any abnormal reaction or first time exposure with systemic involvement	Any patient who requires EpiPen administration
Asthma	Any persistent asthma event or where the trigger cannot be removed	Any asthma event unresponsive to salbutamol and/or any patient with underlying comorbidities (eg: chest infection)
Chest Pain	Any patient complaining of cardiac chest pain	If patient has no medication, has never experienced chest pain before or symptoms last more than 10 mins
Choking	Any patient requiring back blows/chest thrusts that doesn’t rapidly improve with clearing of the obstruction	Any patient who becomes unresponsive

PRESENTATION	CONSIDER EVACUATION	CONSIDER RAPID EVACUATION
Diabetes- HYPO	For known diabetics, monitor resolved hypoglycaemia for 24hrs (provided patient does not have compounding issues- eg: trauma). Consider evac for relapse or first-time presentations	If patient shows no signs of improvement, relapses, has altered level of consciousness (including persistent behavioural changes) or becomes unresponsive
Diabetes- HYPER	All T2/NIDDM patients experiencing hyperglycemia in a wilderness setting; monitor T1/IDDM regularly and consult/manage according to diabetes action plan	Altered level of consciousness, unresponsive
Seizures	For known epileptics, monitor resolved seizures for 24hrs (provided patient does not have compounding issues- eg: trauma). Consider evacuation for relapses	Seizures related to head injury, trauma, unknown causes or lasting longer than 5 mins. Evacuation for ALL first-time seizures
Stroke	RAPID	Any patient who ‘fails’ the F.A.S.T test
Bleeding- External	Controlled bleeding with associated unstable vital signs	Any patient requiring a tourniquet or haemostatic dressing
Bleeding- Internal	All patients with suspected internal bleeding	Immediate evacuation for internal bleeding with compromised vital signs
Burns	Partial thickness burn greater than 10% of TBSA; Any full thickness burn	Major burns of the hand, face, feet or genitals; burns with inhalation injury; electrical burns; circumferential burns; burns to a medically ill patient
Blisters/ Minor Lacerations + Wounds	Related to patient comfort and logistical considerations- should be considered for any patient showing signs of localised infection	Any patient with signs of systemic infection

PRESENTATION	CONSIDER EVACUATION	CONSIDER RAPID EVACUATION
Head Injury	All patients who have a relevant MOI, changes in LOC caused by a head injury (including concussion) should be assessed by a health professional; Acute onset of unexplained headache	Any persistent decreased LOC or deterioration
Limb + Joint Injuries	Directly related to patient comfort and logistical considerations- should be considered for any patient with uncontrollable pain levels or persistent dislocation	Any patient with signs of distal hypoperfusion
Penetrating Injury	Any patient with a penetrating injury	Any patient with signs of shock or breathing problems as a result of injury
Bites and Stings	Directly related to patient comfort and logistical considerations- should be considered for any patient with uncontrollable pain	Any patient with systemic effects, breathing or circulatory problems. Any patient who requires a Pressure Immobilisation Bandage
Hypothermia	Any patient that doesn't respond to rewarming interventions	Persistent moderate/ severe patients
Hyperthermia	Any patients that don't respond to cooling interventions	Altered LOC or seizing patients
Hyponatraemia	Any patients that don't respond to cooling interventions	Altered LOC or seizing patients
Lightning	RAPID	Anyone suspected of a lightning strike injury
Drowning	RAPID	Anyone suspected of a drowning episode
Altitude Emergencies	Descent for all patients showing persistent signs of AMS lasting longer than 24 hours	Rapid descent for all patients showing signs of advanced AMS (HAPE, HACE), breathing problems or decreased LOC
Diving Emergencies	RAPID	Pain or breathing issues after diving, any altered LOC, any complications of rapid ascent

PRESENTATION	CONSIDER EVACUATION	CONSIDER RAPID EVACUATION
Avalanche Emergencies	RAPID	Any patient that has required rescue (self or bystander) following an avalanche emergency (possible unknown internal injuries)
Mental Health	Patients who show a deteriorating level of mental health where responder intervention does not improve situation	Any patient at risk of harm to themselves or others; suicidal ideations; psychotic episodes

Survive First-Aid Pocket Handbook

This book has been developed as a pocket reference for those that have completed a course with Survive First Aid Pty Ltd.

First Aid Courses should be refreshed every 3 years, with CPR, Asthma and Anaphylaxis training refreshed annually.

Content/Guidelines have been sourced from the list of providers on the Home page of this app.

Contents

Abbreviations

Abbreviation	Definition
A	Absence of
AMS	Acute Mountain Sickness
Angio	Vessel
Anterior	From
Anti	Against
Arterio	Artery
Bi	Two
BLS	Basic Life Support
B.O.M	Bites, Odors, Medi-Alerts
BP	Blood Pressure
Brady	Slow
BSI	Body Substance Isolation
cap	Capsule of Medicine
Cardio	Heart
CC	Chief Compliant
cm	Centimeters
CO	Carbon Monoxide
CPR	Cardio Pulmonary Resuscitation
Crani	Skull
CSMS	Circulation, Sensation, Motion & Strength
Derm	Skin
Distal	Away from the body
Dys	Difficult
Ectomy	Removal
Encephalo	Brain
Entero	Intestine
g	Grams
Gastro	Stomach
HACE	High Altitude Cerebral Edema
Haemo	Blood
HAPE	High Altitude Pulmonary Edema
Hemi	Half
Hepato	Liver

Abbreviation	Definition
HPI	History of Present Illness
HR	Heart Rate
Hyper	High
Hypo	Low
ICP	Intra-cranial Pressure
IM	Intramuscular
Inferior	Lower
Itis	Inflammation
IV	Intravenous
L/min	Litres per minute
LLF	Look Listen Feel
Lateral	Side
Lingual	Tongue
LLS	Lake Lousie Score
LOC	Level of Consciousness
m	Meters
Medial	Middle
mg	Milligrams
MI	Myocardial Infarction
min	Minutes
MOI	Mechanism of Injury
Myo	Muscle
Neuro	Nerve
NSAID	Non Steroidal Anti- inflammatory Drug
Ologist	Specialist
ORS	Oral Rehydration Solution
Ostomy	Opening
OTC	Over the counter
Oxia	Oxygen
PEARL	Pupils, Equal & Reactive to Light
Pedi	Children
Pnea	B - Normal Breathing?
Pneumo	Air

Abbreviation	Definition
Poly	Many
Post	After
Posterior	Back
Pre	Before
Prone	Lying on front
Proximal	Closer to the body
Psych	Mind
Retro	Behind
RICE	Rest Ice Compression Elevation
ROM	Range of Motion
RR	Respiratort Rate
S/S	Signs and Symptoms
SC	Sub Cutaneous (Under the Skin)
Scleros	Hardening
Scopy	look at or into
SOB	Shortness of Breath
Superior	Upper
Supine	Lying on back
tab	Tablet
Tachy	Fast
tbs	Teaspoon
tbsp	Tablespoon
Thorax	Chest
TM	Trademark
TX	Treatment
Uni	One
Uria	Urine
Vasc	Vessel
Viscera	Internal organs

First-Aid Aims and Principles

First Aid is the care given to an injured or Ill person before medical assistance arrives or is sourced. Whilst administering any First Aid it is important the following aims are adhered too:

- Maintain Life
- Protect the Unconscious
- Prevent the Condition Getting Worse
- Promote Recovery
- Seek Medical Assistance
- And Above all **DO NO HARM!**

Whilst administering First Aid it can be very difficult to recognize the variety of illness or injuries and therefore decide what appropriate treatment to administer.

It is important First Aiders adhere to a system and structure provided through training and then provide care to a patient based on a Patients, Signs (what you see or hear), Symptoms (what a patient feels or tells you) and Medical History (what happened and what pertinent history there is). Throughout First Aid it is imperative that you treat a patient with care and reassurance as this will undoubtedly help the situation and improve the chance that the patient will respond to treatment.

Primary Survey

When first initiating First Aid it is important to undertake the Primary Survey in the appropriate order of **DRSABCD** and to follow the Emergency Response Plan.

Basic Life Support

EMERGENCY ACTION PLAN

Unconscious Patient

D - Dangers?
R - Responsive?
S - Send for Help
A - Open Airway
B - Normal Breathing?
C - Start CPR
D - Defibrillator & Deadly Bleeds

Check for dangers to:

- Self
- Bystanders
- Patient

Questions:

- What are some specialised dangers we need to consider when working in a remote or wilderness environment?
- What should we do if the dangers to ourselves is too much?

D - Dangers?
R - Responsive?
S - Send for Help
A - Open Airway
B - Normal Breathing?
C - Start CPR
D - Defibrillator & Deadly Bleeds

The AVPU Scale

- **ALERT:** person, place, time, event
- **VERBAL:** R - Responsive? to a verbal command? (eg: squeeze my hand)
- **PAIN:** R - Responsive? to a painful stimuli? (eg: trapezius squeeze)
- **UNRESPONSIVE:** no R - Responsive? to any of the above

D - Dangers?
R - Responsive?
S - Send for Help
A - Open Airway
B - Normal Breathing?
C - Start CPR
D - Defibrillator & Deadly Bleeds

- Call 000

H.E.L.P

Make sure that you have the following information ready before you call:

- Hazards
- Emergency type
- Location
- Patient details

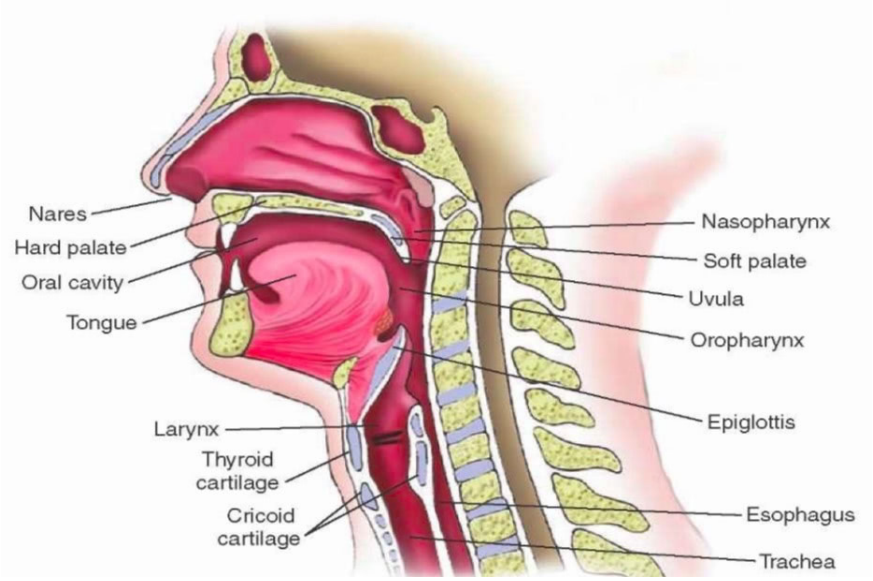
D - Dangers?
R - Responsive?
S - Send for Help
A - Open Airway
B - Normal Breathing?
C - Start CPR
D - Defibrillator & Deadly Bleeds

Make sure that the airway is:

- Open
- Clear

Use the Head Tilt and Chin Lift technique

Remember: **Airway always trumps spinal!**



D - Dangers?
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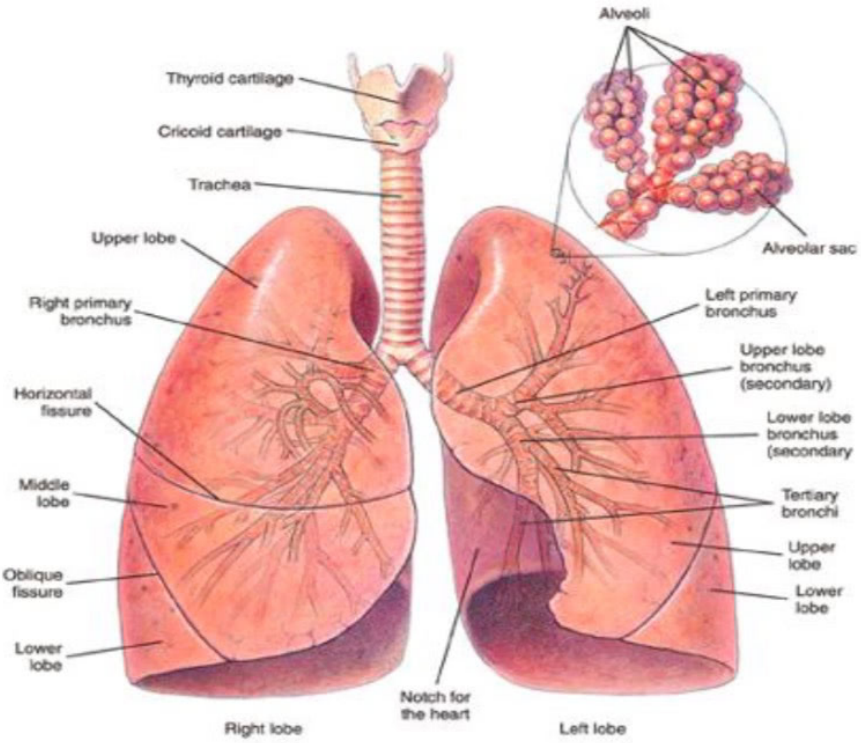
Make sure that the breathing is:

- Normal
- Equal

Look, Listen and Feel for 10 seconds



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Ensure that you consider the following:

- Is the surface suitable for CPR?
- Is my hand and body position correct?
- Am I compressing 1⁄3 of chest depth?
- Is my timing correct?
- Are my rescue breaths effective?

30 COMPRESSIONS : 2 BREATHS 5 CYCLES : 2 MINUTES 100 BEATS PER MINUTE

HANDS-ONLY CPR IS OK!!

D - Dangers?
R - Responsive?
S - Send for Help
A - Open Airway
B - Normal Breathing?
C - Start CPR
D - Defibrillator & Deadly Bleeds

ATTACH DEFIB ASAP

Make sure you consider:

- Do I need to dry or shave the patient?
- Am I putting the pads in the right place?
- Is the environment suitable?

STOP DEADLY BLEEDS ASAP

Make sure you remember:

- Direct pressure on the wound
- Positional pressure on the limb
- Do I need to use a torniquet?

Basic Life Support

EMERGENCY ACTION PLAN

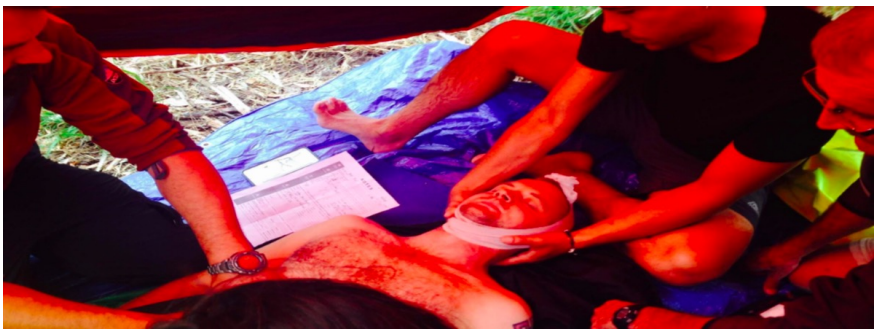
Conscious Patient

D - Dangers?
R - Responsive?
S - Send for Help
A - Open Airway
B - Normal Breathing?
C - Start CPR
D - Defibrillator & Deadly Bleeds
E - Evaluate Injury & Environment
F - Find out the Facts
G - Get Ready to Go

Evaluate Injury & Environment

- **Treat** life threatening injuries and illness
  - Asthma, anaphylaxis, hypoglycaemia etc.
- Treat **distracting** injuries
  - Long-bone fractures, burns,

- Treat for **environment**
  - Remove wet clothing, hypo-wrap etc.



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Find out the Facts

- Follow the N.O.T.E.S sheet format:
  - Need to know?
  - Observations
  - Total Body Exam
  - Enquire
  - Summary + Spinal Assess

Remember:

- Record vital signs at regular intervals
- Get “down to skin” in total body exam
- Ask plenty of questions!

D - Dangers?
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G - Get Ready to Go

Get Ready to Go & Final Treatments

- **Complete** any treatments or packaging
- Prepare an **IMIST AMBO** handover
- **Re-assess** evacuation plans
- Make sure that all **rescue team** members are on the same page
- **Smooth and steady** movements- especially if lifting or carrying

Observations & Vital Signs Ranges

VITAL SIGNS	NORMAL	MODERATE	SEVERE
LEVEL OF CONSCIOUSNESS	A+O x 4	ALERT ONLY	V, P or U
HEART RATE	60 - 100	100 - 120	120+ (without cause)
RESPIRATORY RATE	8 - 16	16 - 22	22+ (with cause)
SKIN	Pink, warm, dry	Pale, cool, clammy	Pale, cold blue
PUPILS	PEARL	Dilated/sluggish/pinpoint	Dilated/fix
BLOOD SUGAR	4 - 6 mmol	2 - 4 mmol	Low

I.M.I.S.T A.M.B.O

- **I**dentify the patient
- **M**echanism of injury/ nature of illness
- **I**njuries or info related to complaint

- **S**igns
- **T**reatment
- **A**llergies
- **M**edications
- **B**ackground
- **O**ther pertinent info

Level of Consciousness (LOC) AVPU

Key to the AVPU Scale

- **A&O x 4** - Patient knows Person, Place, Time and Event
- **A&O x 3** - Patient knows Person, Place and Time
- **A&O x 2** - Patient knows Person and Place
- **A&O x 1** - Patient knows Person
- **A** - Patient responds but doesn't know who they are
- **V** - Patient responds to a verbal stimulus
- **P** - Patient only responds to a Pain Stimulus
- **U** - Patient is Unresponsive

Heart Rate (HR)/Pulse

The rate of a normal pulse can vary greatly and be between 50 – 100 beats per minute for an adult and as much as 160 in an infant. Details of the HR can however tell you if a patient is stable over time and give you an indication of the extent of the injury/illness.

Note: It is best to take the Radial Pulse of a patient, as this is both less invasive for the patient and also important when assessing Blood Pressure.

Respiratory Rate (RR)

In general it is effortless to breathe however if this becomes labored it is an indication that your patient has something wrong. The respiratory rate is best done by sight or feel of the chest rising and falling. As with the HR detail the Rate (Breaths per minute), Rhythm (Regular or Irregular) and Quality (Deep or Shallow).

Skin Color: Temperature: Moisture, (SCTM)

Every person irrespective of their ethnic/cultural background and genetics will have pink skin on the inside of their mouth, soles of feet and palms of hands.

Examples of SCTM:

- PWD - Pink, Warm & Dry - Normal
- PCC - Pale, Cool & Clammy - Shock
- RHD - Red, Hot & Dry - Hyperthermia

Pupils

Normal Pupils are equal in size, round in shape and have a normal reaction to light. They become smaller with increased light and larger with less light. This is described as PEARL, (Pupils, Equal and Reactive to Light). A patients Pupils can be tested by shining a torch briefly in each eye or use sun light by covering each eye and then exposing to the light again.

Anything that is different to PEARL should be noted, as it may be a significant discovery with regards to an injury or illness, i.e. Fixed, Dilated, Unequal.

Total Body Exam (Head to Toe)

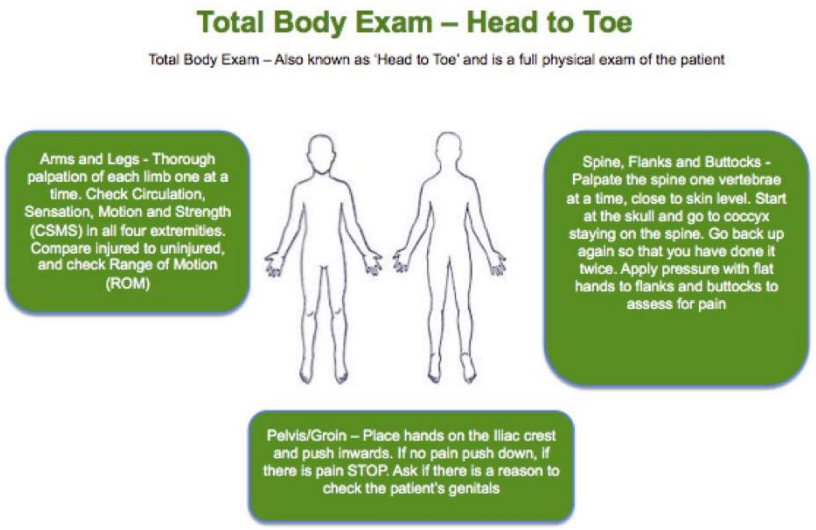
This is a systematic, physical check of the whole body for any injuries sustained. Whilst conducting this physical check, it is worth remembering to conduct a B.O.M check (Bites, Odors, Medi-alerts) and ensure you Look Ask and Feel (LAF) as you systematically move down the body. Look for bruising, bleeding, swelling, or anything out of the ordinary. Ask about pain or tenderness. Feel for deformities, unusual hardness or softness.

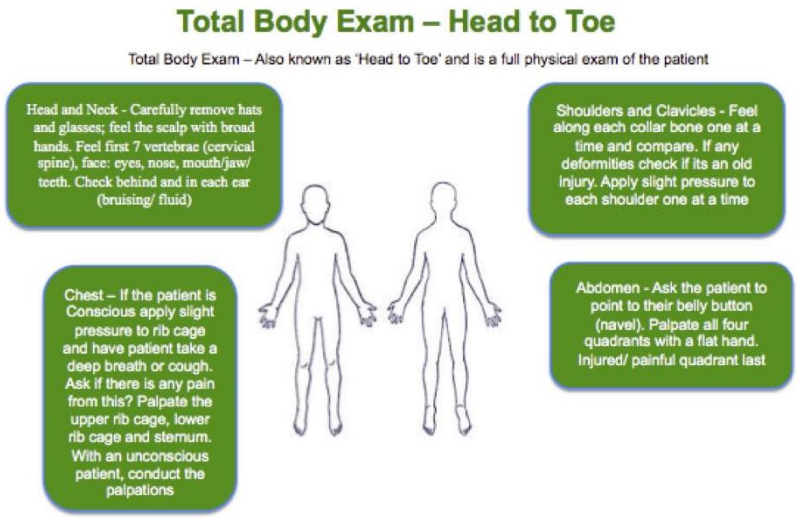
If you find a painful area ensure to expose it to skin level and examine before moving on. The objective of this ‘Exam” is to ensure you get a full picture of all the injuries that have been sustained.

Start at the head and work towards the feet using broad hands and explaining to the patient what you are doing at all times (even if the patient is unconscious)

If there is any pain or an injury with the neck, back or pelvis always suspect a spinal injury and treat accordingly. Ensure you check Circulation, Sensation, Motion and Strength (CSMS) in all four extremities. Please note that the Chief Complaint (CC) should already have been exposed and possibly treated in an earlier stage of your Emergency Action Plan (Evaluating Injury and Shock) prior to commencing this component of your secondary survey.

It is important during the Full Body Exam that you respect the individual’s cultural background and ensure that the exam is always undertaken with their permission. Continually talk to the patient during the exam explaining whet you are about to do and ensure they are comfortable and at ease.





Enquire (SAMPLE history)

A Sample medical history is an important part of the Secondary Survey and allows you to really investigate how the patient is feeling. Be compassionate and confident (good bedside manner), ask non-leading through questions, and record the information you receive. Again it is important to respect the cultural/ethnic background of your patient and ensure you speak to them in a respectful and appropriate manner.

- **SYMPTOMS** - How do you feel? Open ended Questions?
- **ALLERGIES** - What To? Severity of Reaction? Medication?
- **MEDICATIONS** - Prescription, Homeopathic, Illegal?
- **PERTINENT MEDICAL HISTORY** - What, When, Is it Related?
- **LAST INS & OUTS** - Urine, Faeces, Vomit, Diarrhea, Menstrual?
- **EVENTS** - What events occurred to make you feel like this?

Pain

- **ONSET** – Sudden or Gradual?
- **PROVOKES** – Movement, Touch?
- **QUALITY** – Dull or Sharp?
- **RADIATES** – Does the pain move?
- **SEVERITY** – Out of 10?
- **TIME** – How Long?

Calling Emergency Services

The person who calls emergency services should provide clear concise information to the operator. Other appropriate information to give to the operator would be the callers name, phone number or details and how to get back in contact.

When speaking to an operator you may need to spell words to ensure the information given is accurate. In such a case the use of the International Phonetic Alphabet will help

H.E.L.P

- **Hazards**– Detail any hazards emergency services attending need to be aware off.
- **Emergency Type** – Detail what has happened and what help you require
- **Location** - Detail exact location of where you are (nearest road head is often required)
- **Patient Details** – Describe what is going on with your Patient(s)

International Phonetic Alphabet

LETTER	WORD	PRONUNCIATION
A	Alpha	Al fah
B	Bravo	Brah Voh
C	Charlie	Char Lee
D	Delta	Dell Tah
E	Echo	Eck Oh
F	Foxtrot	Foks Trot
G	Golf	Golf
H	Hotel	Hoh Tell
I	India	In Dee Ah
J	Juliet	Jew Lee Ett
K	Kilo	Key Loh
L	Lima	Lee Mah
M	Mike	Mike
N	November	No Vem Ber
O	Oscar	Oss Car
P	Papa	Pah Pah
Q	Quebec	Keh Beck
R	Romeo	Row Me Oh
S	Sierra	See Air Rah
T	Tango	Tang Go
U	Uniform	You Nee Form
V	Victor	Vick Tah
W	Whisky	Wiss Key
X	X-Ray	Ecks Ray
Y	Yankee	Yang Key
Z	Zulu	Zoo Loo

Two-way Radio Guide

When using the Two-way Radio Network it is vital that you are clearly understood. To do this it is important to understand the differences between a telephone and a two-way radio. Two-way radios are not like a telephone. Telephones and mobile phones are "full-duplex" which means you can talk and listen at the same time. Two way radios are "simplex" which means you cannot hear anyone while you have the talk button pressed.

- **It is extremely important to take turns talking.** To help with this, standard radio procedures have been created.
- **The single most important mistake people make is failing to identify themselves.** There may be several people using the same channel as you, so it's important to know who you are directing your transmission to. To call someone, say the name of the person you want to call, followed by the words "THIS IS," then say your name and "OVER."

  Example: "Susan, THIS IS John, OVER."
- It's important to say the name of the person you want to contact before saying your name, as this will get their attention, and they will listen to the rest of your message. The word "OVER" leaves no doubt about whose turn it is to talk and avoids any confusion. It's important to say "OVER", so they know when you are done speaking.
- **It's basic "radio etiquette" to establish contact** and make sure that you have the other person's attention before you start your message. If you hear someone calling you, acknowledge his or her call by saying, "GO AHEAD" or "STAND BY" This lets the caller know that you heard them. Also remember, it may take someone a while to get to their radio and respond. Be patient in waiting for a reply.
- Because only one person can talk at a time, it is more important to LISTEN on a 2-way radio than to talk!
- When your business is finished, the person who started the conversation should end it by saying their name and the word "OUT," which leaves no mistake that contact has ended.
- Always release the push-to-talk (PTT) button whenever you stop talking. If you forget and keep it pushed down while you are trying to think of something to say, the radio continues to transmit, making your battery run down faster and making "dead air" so that nobody else can speak or be heard. In an emergency, it could prevent someone with vital information from getting through. If you need to collect your thoughts, release the PTT so that somebody else can break in case they have an emergency or additional information
- It is always best to speak in short simple phrases on the radio and toss the conversion back and forth with the word "OVER."
- Don't speak immediately when you press the PTT (push to talk), especially with digital radios which among all their benefits have slightly longer delay. Ideally, wait 2-3 seconds. If you speak as soon you press the PTT button, it can chop off your the first syllable or word, making you hard to understand.

Radio User's Language

Expression	Meaning
Go Ahead	Resume transmission
Say Again	Re-transmit your message
Stand-by	Transmission has been acknowledged, but I am unable to respond now
Roger	Message received and understood
Affirmative	Yes (avoid yup, nope, etc.)
Negative	No
Over	Transmission finished
Out	Communication is over and the channel is available for others.

Sample Dialog

Below is a sample dialog that puts these standards to use.

Adam: *Mike, this is Adam. Over.*

Mike: *Adam, this is Mike, Stand By. Over.*

Mike: *Adam, this is Mike, Go Ahead. Over.*

Adam: *Mike, there is a fire at 123 Main St. Over.*

Mike: *Adam, this is Mike, confirming a fire at 123 main St. The fire department will be notified. Over.*

Adam: *Mike, this is Adam, address is confirmed, thanks for the help. Over and Out.*

Radio Usage Tips

- Be brief and to the point.
- Stay off the radio unless absolutely necessary.
- Engage your brain before your mouth.
- Think about how best to make yourself understood.
- Listen before you begin your transmission.
- Make sure the channel is clear. Wait a full second AFTER you push-to-talk and BEFORE you begin to speak. This will insure the beginning of your message is heard.
- Speak ACROSS the microphone rather than into it to improve intelligibility. Use a natural speaking voice. The only way to overcome loud ambient noise is to shield the microphone from the wind, point it away from the source of noise or wait until the noise passes.
- You should be aware that any portable radio is much less effective when worn on your belt, because your body absorbs the radio signal. This is very noticeable with low powered radios.
- **DO NOT** shout into the radio. It only distorts your transmission.
- **DO NOT** turn the volume all the way up. This drains the battery and causes distortion. It also has no effect on outgoing transmission quality.

Triage

Priority One – Red Tag (require immediate surgery or other life-saving intervention). Immediate resuscitation and stabilization required

- Control of severe bleeding required
- Open and stabilize airway in an unconscious casualty

Priority Two – Orange Tag (condition stable, but requires close monitoring and will need hospital care)

- Treatment but no active resuscitation required
- Burns, Crush injury, Spinal, Head, Chest, Abdominal

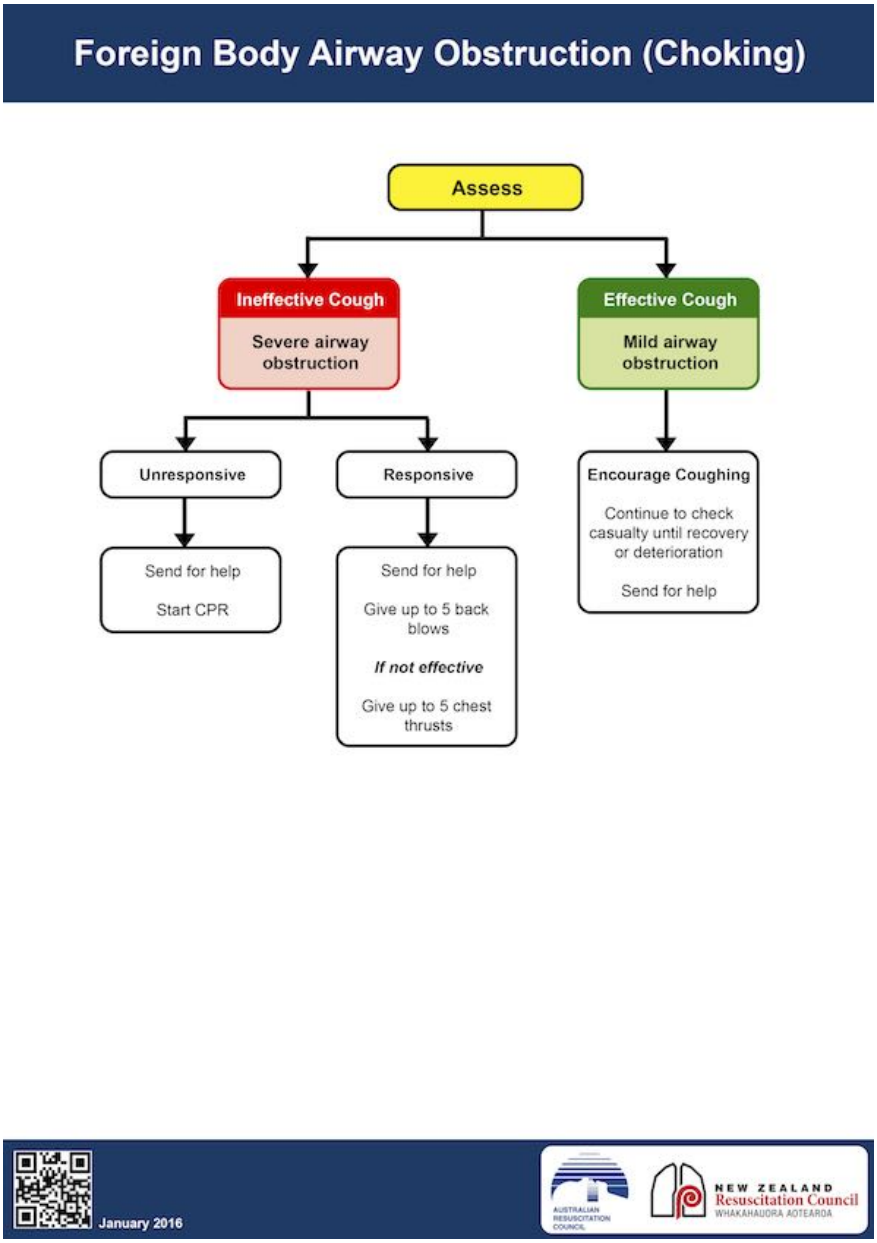
Priority Three – Green Tag (will require care but not immediately)

- Walking wounded
- Cuts, Grazes, Sprains, Closed Fractures

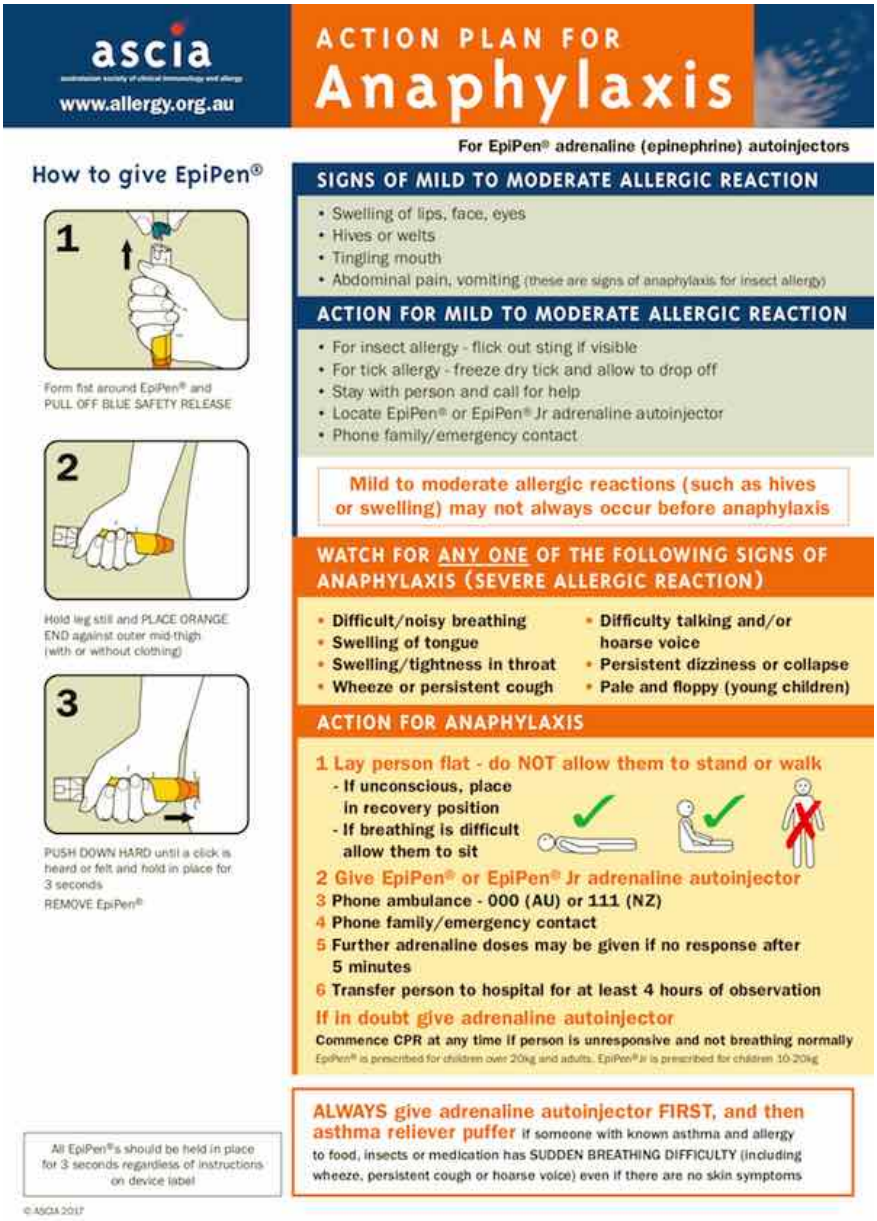
Priority Zero - Black Tag (deceased or in such life- threatening medical crisis that they are unlikely to survive given the care available)

- Decapitation
- Massive head or torso injuries

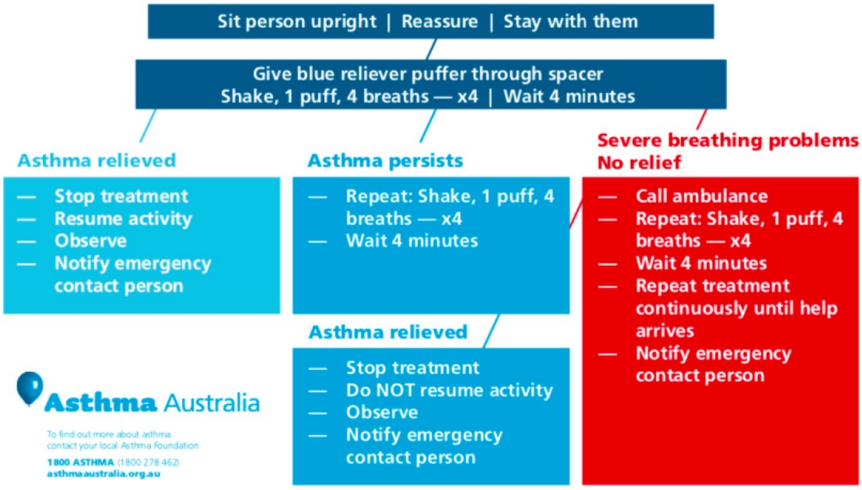
Choking



Anaphylaxis


ascia
www.allergy.org.au
ACTION PLAN FOR
Anaphylaxis
For EpiPen® adrenaline (epinephrine) autoinjectors
How to give EpiPen®
1
Form fist around EpiPen® and PULL OFF BLUE SAFETY RELEASE
2
Hold leg still and PLACE ORANGE END against outer mid-thigh (with or without clothing)
3
PUSH DOWN HARD until a click is heard or felt and hold in place for 3 seconds
REMOVE EpiPen®
All EpiPen®s should be held in place for 3 seconds regardless of instructions on device label
© ASCIA 2017
SIGNS OF MILD TO MODERATE ALLERGIC REACTION
• Swelling of lips, face, eyes
• Hives or welts
• Tingling mouth
• Abdominal pain, vomiting (these are signs of anaphylaxis for insect allergy)
ACTION FOR MILD TO MODERATE ALLERGIC REACTION
• For insect allergy - flick out sting if visible
• For tick allergy - freeze dry tick and allow to drop off
• Stay with person and call for help
• Locate EpiPen® or EpiPen® Jr adrenaline autoinjector
• Phone family/emergency contact
Mild to moderate allergic reactions (such as hives or swelling) may not always occur before anaphylaxis
WATCH FOR ANY ONE OF THE FOLLOWING SIGNS OF ANAPHYLAXIS (SEVERE ALLERGIC REACTION)
• Difficult/noisy breathing
• Swelling of tongue
• Swelling/tightness in throat
• Wheeze or persistent cough
• Difficulty talking and/or hoarse voice
• Persistent dizziness or collapse
• Pale and floppy (young children)
ACTION FOR ANAPHYLAXIS
1 Lay person flat - do NOT allow them to stand or walk
- If unconscious, place in recovery position
- If breathing is difficult allow them to sit
2 Give EpiPen® or EpiPen® Jr adrenaline autoinjector
3 Phone ambulance - 000 (AU) or 111 (NZ)
4 Phone family/emergency contact
5 Further adrenaline doses may be given if no response after 5 minutes
6 Transfer person to hospital for at least 4 hours of observation
If in doubt give adrenaline autoinjector
Commence CPR at any time if person is unresponsive and not breathing normally
EpiPen® is prescribed for children over 20kg and adults. EpiPen® Jr is prescribed for children 10-20kg
ALWAYS give adrenaline autoinjector FIRST, and then asthma reliever puffer if someone with known asthma and allergy to food, insects or medication has SUDDEN BREATHING DIFFICULTY (including wheeze, persistent cough or hoarse voice) even if there are no skin symptoms

Asthma



Pressure Immobilisation

When treating a patient that requires a Pressure Immobilisation Bandage, there are two components that must be satisfied:

1. pressure over the bitten limb, and
2. general immobilisation.

This involves the application of:

- A broad (minimum 7.5 cm wide) elastic bandage to the entire bitten limb at a very firm pressure of at least 40 mmHg for an arm and 55 mmHg for a leg. The SMART Snake Bandage is perfect for this.
- Splints to effectively immobilize the entire limb, in combination with laying the patient down and completely still to minimize any movement.

TREATMENT:

- Keep the victim at rest, reassured and under constant observation
- Commence resuscitation if necessary, following ANZCOR Guideline 8: Cardiopulmonary Resuscitation
- Apply a Pressure Immobilisation Bandage to the entire limb:
- Apply a broad pressure bandage over the bite site as soon as possible
  - The bandage should be firm and tight, you should be unable to easily slide a finger between the bandage and the skin
  - To further restrict lymphatic flow and to assist in immobilisation of the limb, apply a further pressure bandage, commencing at the fingers or toes of the bitten limb and extending upward covering as much of the limb as possible.
  - Splint the limb including joints on either side of the bite, to restrict limb movement.
  - Rapid evacuation for any patient who requires a Pressure Immobilisation Bandage

DO NOT...

DO NOT cut or excise the bitten area, or attempt to suck venom from the bite site

DO NOT wash the bitten area

DO NOT apply an arterial tourniquet

DO NOT try to catch, chase or kill the snake, as this may lead to another bite

DO NOT give alcohol, tea, stimulants, food or medications without medical advice

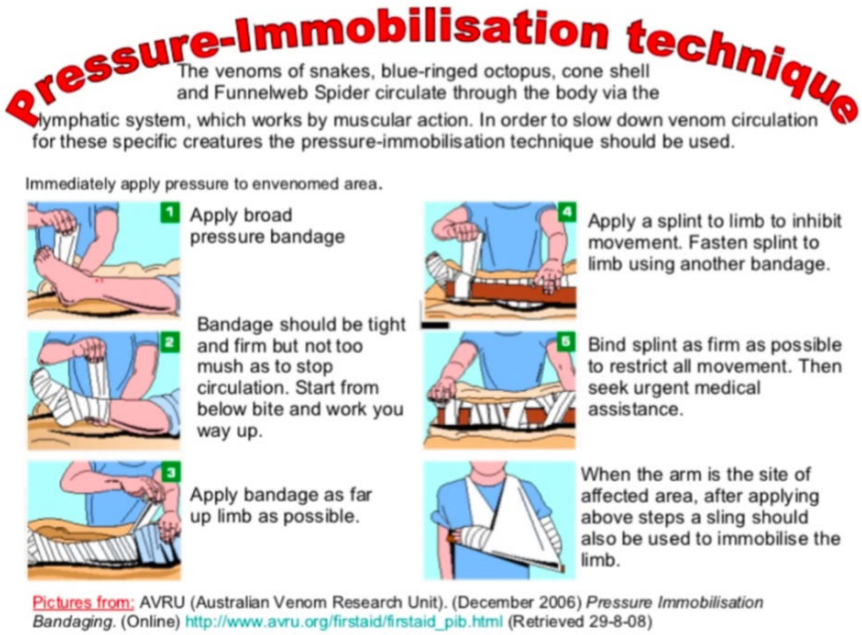
DO NOT wash the wound, apply hot or cold packs, apply electric shocks, and

DO NOT suck the wound or use suction from any device

DO NOT allow the patient to walk or run after a snake bite

DO NOT remove or loosen the pressure immobilisation bandages unless advised to do so by medical personnel

DO NOT ignore the urgency of obtaining medical assistance in favour of reliance on traditional medicines or home remedies



Pictures from: AVRU (Australian Venom Research Unit). (December 2006) *Pressure Immobilisation Bandaging*. (Online) http://www.avru.org/firstaid/firstaid_pib.html (Retrieved 29-8-08)

Heart Attack


Will you recognise your heart attack?
Heart Foundation
Do you feel any
pain
pressure
heaviness
tightness
In one or more of your
chest
neck
jaw
arm/s
back
shoulder/s
You may also feel
nauseous
a cold sweat
dizzy
short of breath
Yes
1 STOP and rest now
2 TALK tell someone how you feel
If you take angina medicine
• Take a dose of your medicine.
• Wait 5 minutes. Still have symptoms? Take another dose of your medicine.
• Wait 5 minutes. Symptoms won't go away?
Are your symptoms severe or getting worse?
or
Have your symptoms lasted 10 minutes?
Yes
3 CALL 000* and chew 300mg aspirin, unless you have an allergy to aspirin or your doctor has told you not to take it
*Triple Zero
• Ask for an ambulance.
• Don't hang up.
• Wait for the operator's instructions.
*If calling Triple Zero (000) does not work on your mobile phone, try 112.
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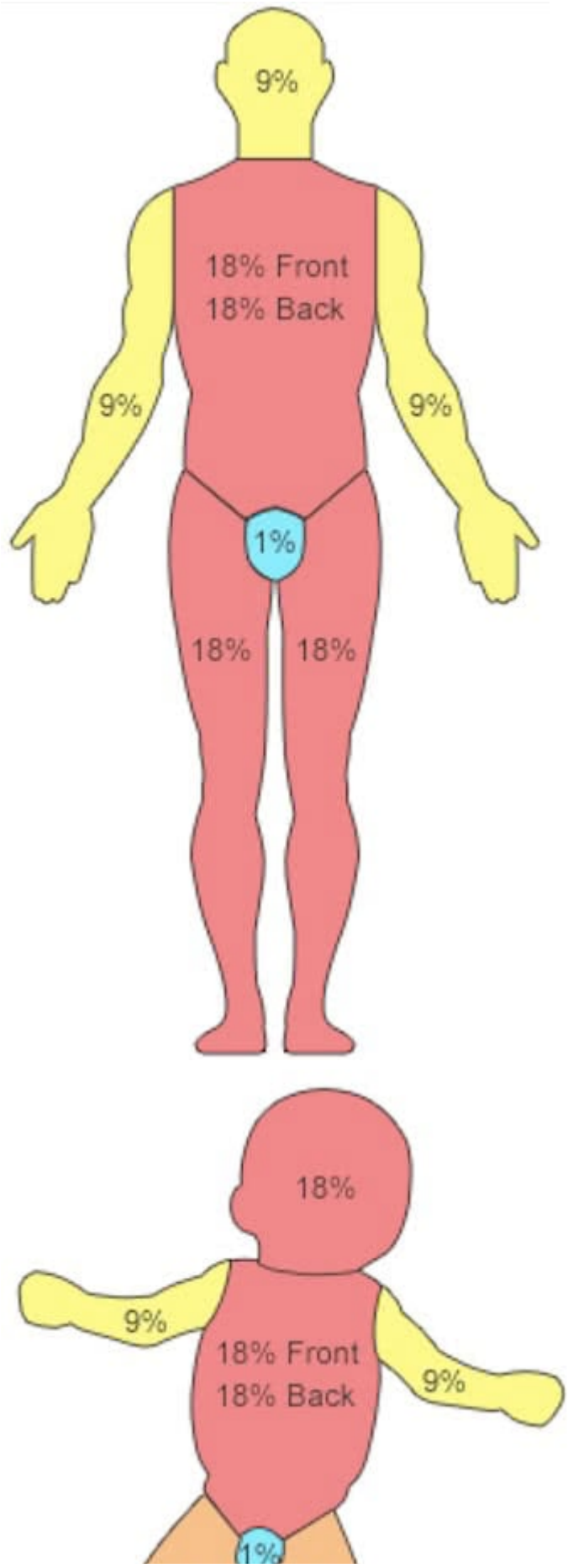
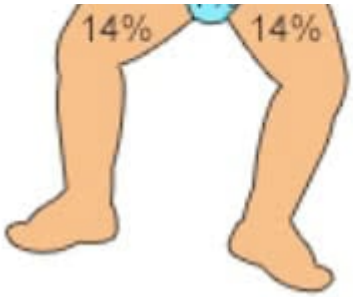
HFN0389-JUN17

Stroke


Recognise
STROKE
Think F.A.S.T.
F
Has their
FACE
drooped?
A
Can they lift both
ARMS?
S
Is their
SPEECH
slurred and do they
understand you?
T
Call 000,
TIME
is critical
If you see
any of these
symptoms
Act FAST call 000
Stroke
FOUNDATION

Burns


9%
18% Front
18% Back
9%
9%
1%
18%
18%
18%
9%
18% Front
18% Back
9%
1%



Heat Stress / Hyperthermia

Hypothermia

ASSESS COLD PATIENT

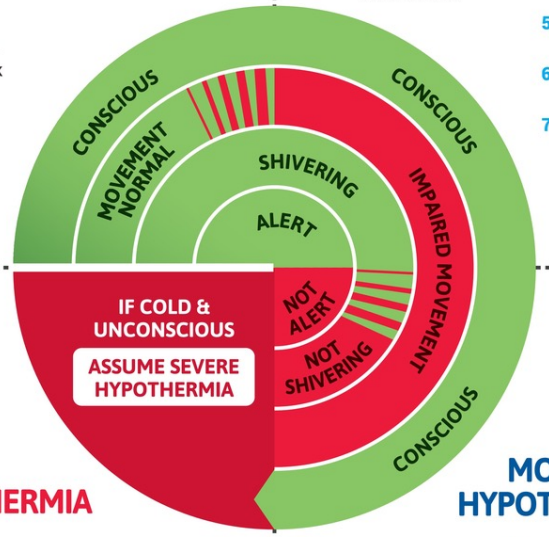
1. From outside ring to centre: assess Consciousness, Movement, Shivering, Alertness
2. Assess whether normal, impaired or no function
3. The colder the patient is, the slower you can go, once patient is secured
4. Treat all traumatized cold patients with active warming to upper trunk
5. Avoid burns: following product guidelines for heat sources; check for excessive skin redness

COLD STRESSED, NOT HYPOTHERMIC

1. Reduce heat loss *(e.g., add dry clothing)*
2. Provide high-calorie food or drink
3. Move around/ exercise to warm up

MILD HYPOTHERMIA

1. Handle gently
2. Have patient sit or lie down for at least 30 min.
3. Insulate/ vapour barrier
4. Give heat to upper trunk
5. Give high-calorie food/drink
6. Monitor for at least 30 min.
7. Evacuate if no improvement



SEVERE HYPOTHERMIA

1. Treat as Moderate Hypothermia, and
   - ***a) IF*** no obvious vital signs, ***THEN*** 60-second breathing / pulse check, or assess cardiac function with cardiac monitor
   - ***b) IF*** no breathing / pulse, ***THEN*** Start CPR
2. Evacuate carefully ASAP

MODERATE HYPOTHERMIA

1. Handle gently
2. Keep horizontal
3. No standing/walking
4. No drink or food
5. Insulate/ vapour barrier
6. Give heat to upper trunk
7. Volume replacement with warm intravenous fluid (40-42°C)
8. Evacuate carefully

Funded by the Government of Canada | Canada

BICOrescue.com

Baby It's COLD OUTSIDE

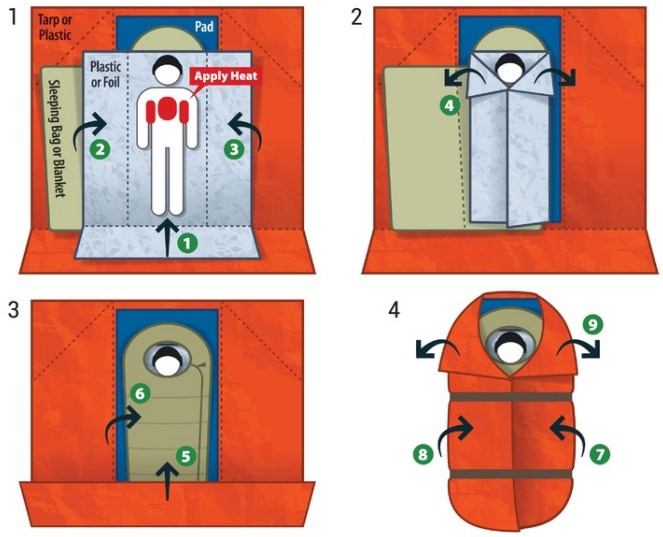
CARE FOR COLD PATIENT

SUGGESTED SUPPLIES FOR SEARCH/RESPONSE TEAMS IN COLD ENVIRONMENTS:

- **1** - Tarp or plastic sheet for vapour barrier outside sleeping bag
- **1** - Insulated ground pad
- **1** - Hooded sleeping bag (or equivalent)
- **1** - Plastic or foil sheet (2 x 3 m) for vapour barrier placed inside sleeping bag
- **1** - Source of heat ***for each team member*** *(e.g., chemical heating pads, or warm water in a bottle or hydration bladder),* or ***each team*** *(e.g., charcoal heater, chemical / electrical heating blanket, or military style Hypothermia Prevention and Management Kit [HPMK])*

INSTRUCTIONS FOR HYPOTHERMIA WRAP "The Burrito"

1. *Dry or damp clothing:* *Leave clothing on*
2. *Very wet clothing:*
   - ***IF*** Shelter / Transport is ***less than*** 30 minutes away, ***THEN*** *Wrap immediately*
   - ***IF*** Shelter / Transport is ***more than*** 30 minutes away, ***THEN*** *Protect patient from environment, remove wet clothing and wrap*
3. *Avoid burns: follow product instructions; place thin material between heat and skin; check hourly for excess redness*



Sources: BICOrescue.com; Zafren, Giesbrecht, Danzl et al. Wilderness Environ Med. 2014, 25:S66-85.

Helicopter Evacuation Guidelines

General Guidelines

- Approach and depart helicopter from the front or forward-sides- ideally in the space defined by '10 + 2' on a clock.
- Always establish visual contact with the pilot before approaching. If unsure, do not approach.
- Where possible, maintain radio communications with the pilot at all times.
- When approaching at night, await a predetermined signal such as a the flashing of a head-torch or vehicle headlights.
- Never approach or depart toward the tail of the helicopter; always be aware of the tail rotor.
- Always approach and depart from the downhill slope (if present)
- Carry all equipment and patients below waist level.

- Secure all loose equipment on the ground (including wind-socks, lights and flares).
- Protect patients by using natural features or building a small fortress (using packs, bags, etc), between them and the aircraft.
- At night, designate the landing zone by crossing vehicle headlights or using head-torches in a similar manner.
- Small fires may be lit to designate landing zone corners, however must be well-controlled and attended at all times.
- Never shine lights or laser beams directly at the pilot.
- All non-essential lights should be extinguished.
- Always be aware that helicopters will approach and land from a downwind direction

Landing Zone Specifications

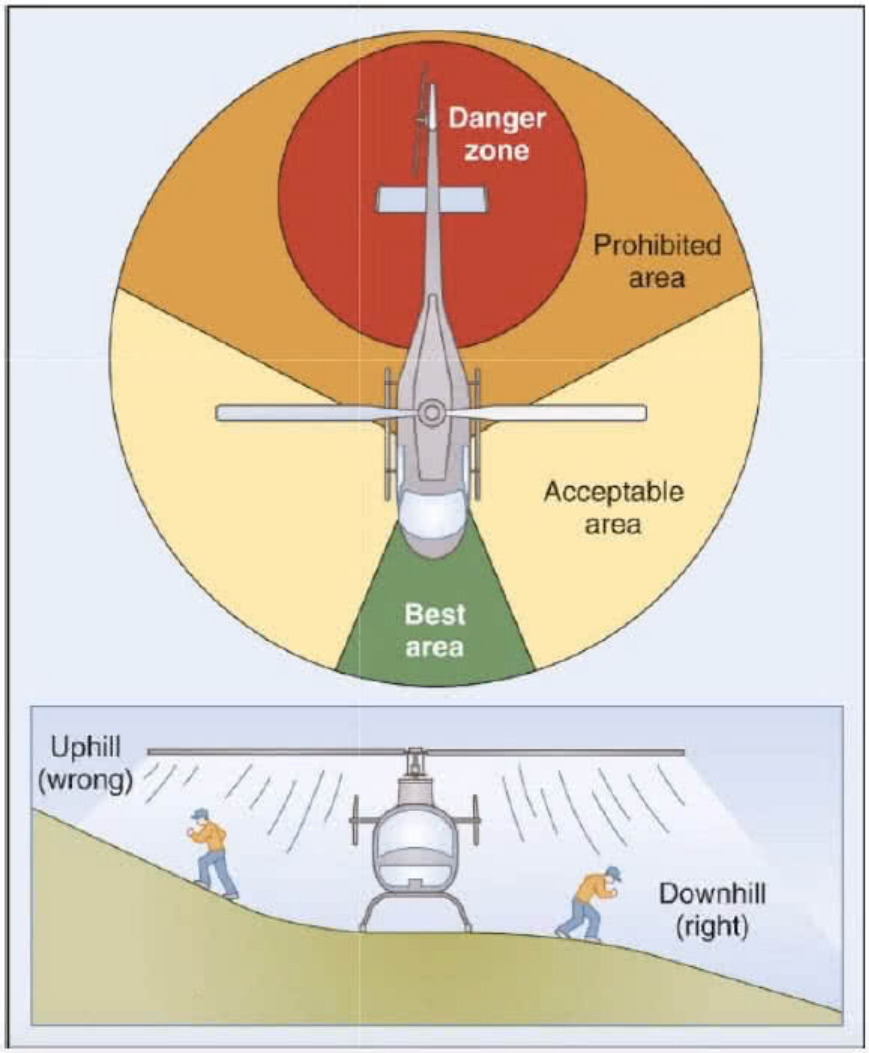
- Landing zones should, ideally, measure a 30m x 30m square, with minimal ground debris or overhead structures in the vicinity.
- The landing zone should be as flat as possible. In the case where flat land is not available, the slope should not exceed 5 degrees.
- A wind-sock should be placed on the upwind edge of the square, securely fastened. A small fire may serve this purpose.
- On a beach or similar surface, a large ‘H’ can be used to designate the middle of the landing zone.
- The landing zone must be kept free of personnel and debris at all times.

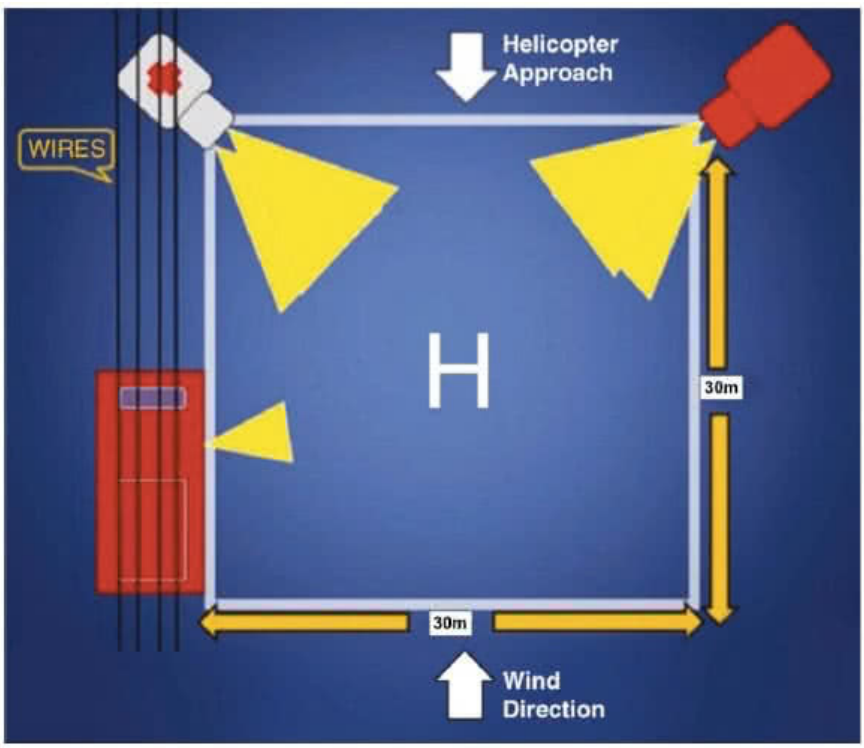
Land here
(day)

LZ unsafe
(day)

Land here
(night)

LZ unsafe
(night)


Danger zone
Prohibited area
Acceptable area
Best area
Uphill (wrong)
Downhill (right)


Helicopter Approach
WIRES
H
30m
30m
Wind Direction

Evacuation Guidelines

- Please be aware that these are recommendations only.
- They are based on best-practice and the most current available information.
- Every first-aid scenario is dependent on responder training, patient presentation, environmental considerations and available resources- all of which should be taken into account and form part of your treatment and evacuation plan.

RAPID EVACUATION FOR ANY UNRESPONSIVE PATIENT

PRESENTATION	CONSIDER EVACUATION	CONSIDER RAPID EVACUATION
Unresponsive-CPR	RAPID	Rapid evacuation for all unresponsive patients (including deteriorating LOC) and unstable vital signs
Unresponsive-Breathing	RAPID	Rapid evacuation for all unresponsive patients (including deteriorating LOC) and unstable vital signs
Spinal Injury	All patients with relevant MOI	All patients with relevant MOI and any signs and symptoms of spinal injury
Shock	All patients with injuries significant enough to cause uncontrolled shock	Deteriorating patients, uncontrollable bleeding, airway problems and unstable vital signs
Abdominal	Sudden unexplained onset of severe pain or constant unexplained pain lasting more than 24 hrs	Blood in urine, faeces or vomit; any acute abdo pain associated with women of childbearing age; any complications of pregnancy
Anaphylaxis	Any abnormal reaction or first time exposure with systemic involvement	Any patient who requires EpiPen administration
Asthma	Any persistent asthma event or where the trigger cannot be removed	Any asthma event unresponsive to salbutamol and/or any patient with underlying comorbidities (eg: chest infection)
Chest Pain	Any patient complaining of cardiac chest pain	If patient has no medication, has never experienced chest pain before or symptoms last more than 10 mins
Choking	Any patient requiring back blows/chest thrusts that doesn’t rapidly improve with clearing of the obstruction	Any patient who becomes unresponsive

PRESENTATION	CONSIDER EVACUATION	CONSIDER RAPID EVACUATION
Diabetes- HYPO	For known diabetics, monitor resolved hypoglycaemia for 24hrs (provided patient does not have compounding issues- eg: trauma). Consider evac for relapse or first-time presentations	If patient shows no signs of improvement, relapses, has altered level of consciousness (including persistent behavioural changes) or becomes unresponsive
Diabetes- HYPER	All T2/NIDDM patients experiencing hyperglycemia in a wilderness setting; monitor T1/IDDM regularly and consult/manage according to diabetes action plan	Altered level of consciousness, unresponsive
Seizures	For known epileptics, monitor resolved seizures for 24hrs (provided patient does not have compounding issues- eg: trauma). Consider evacuation for relapses	Seizures related to head injury, trauma, unknown causes or lasting longer than 5 mins. Evacuation for ALL first-time seizures
Stroke	RAPID	Any patient who ‘fails’ the F.A.S.T test
Bleeding- External	Controlled bleeding with associated unstable vital signs	Any patient requiring a tourniquet or haemostatic dressing
Bleeding- Internal	All patients with suspected internal bleeding	Immediate evacuation for internal bleeding with compromised vital signs
Burns	Partial thickness burn greater than 10% of TBSA; Any full thickness burn	Major burns of the hand, face, feet or genitals; burns with inhalation injury; electrical burns; circumferential burns; burns to a medically ill patient
Blisters/ Minor Lacerations + Wounds	Related to patient comfort and logistical considerations- should be considered for any patient showing signs of localised infection	Any patient with signs of systemic infection

PRESENTATION	CONSIDER EVACUATION	CONSIDER RAPID EVACUATION
Head Injury	All patients who have a relevant MOI, changes in LOC caused by a head injury (including concussion) should be assessed by a health professional; Acute onset of unexplained headache	Any persistent decreased LOC or deterioration
Limb + Joint Injuries	Directly related to patient comfort and logistical considerations- should be considered for any patient with uncontrollable pain levels or persistent dislocation	Any patient with signs of distal hypoperfusion
Penetrating Injury	Any patient with a penetrating injury	Any patient with signs of shock or breathing problems as a result of injury
Bites and Stings	Directly related to patient comfort and logistical considerations- should be considered for any patient with uncontrollable pain	Any patient with systemic effects, breathing or circulatory problems. Any patient who requires a Pressure Immobilisation Bandage
Hypothermia	Any patient that doesn't respond to rewarming interventions	Persistent moderate/ severe patients
Hyperthermia	Any patients that don't respond to cooling interventions	Altered LOC or seizing patients
Hyponatraemia	Any patients that don't respond to cooling interventions	Altered LOC or seizing patients
Lightning	RAPID	Anyone suspected of a lightning strike injury
Drowning	RAPID	Anyone suspected of a drowning episode
Altitude Emergencies	Descent for all patients showing persistent signs of AMS lasting longer than 24 hours	Rapid descent for all patients showing signs of advanced AMS (HAPE, HACE), breathing problems or decreased LOC
Diving Emergencies	RAPID	Pain or breathing issues after diving, any altered LOC, any complications of rapid ascent

PRESENTATION	CONSIDER EVACUATION	CONSIDER RAPID EVACUATION
Avalanche Emergencies	RAPID	Any patient that has required rescue (self or bystander) following an avalanche emergency (possible unknown internal injuries)
Mental Health	Patients who show a deteriorating level of mental health where responder intervention does not improve situation	Any patient at risk of harm to themselves or others; suicidal ideations; psychotic episodes

N.O.T.E.S Form

N.O.T.E.S

SurVive First Aid

N — Need to know?

Who? Name: _____ Age: _____ D.O.B_____

What? (MOI or NOI): _____

Chief Complaint? _____

Contact Person (Name & Phone)? _____

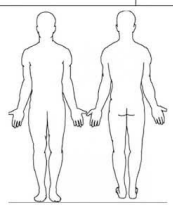
O — Observations

TIME												
LOC - AVPU												
HR	Rate	Rhythm	Quality	Rate	Rhythm	Quality	Rate	Rhythm	Quality	Rate	Rhythm	Quality
RR	Rate	Rhythm	Quality	Rate	Rhythm	Quality	Rate	Rhythm	Quality	Rate	Rhythm	Quality
SKIN Color/Temp/Moist												
PUPILS – PEARL												

T — Total Body Exam

Abrasion (A)
Haemorrhage (H)
Burn (B)
Contusion (C)
Deformity (D)
Fracture (F)
Laceration (L)
Object (O)
Pain (P)
Rigidity (R)
Swelling (S)
Tenderness (T)

Blood Loss
☐ <500ml
☐ >500ml



Onset: _____

Provokes: _____

Quality: _____

Radiates: _____

Severity: _____

Time: _____

SPINAL CONSIDERATIONS – FIRSTLY CONSIDER MOI – FALLS >1M SPEED

NO PAIN ON SPINE ☐Normal LOC ☐Normal CSMS ☐Stable Vitals ☐No Distractions☐ Sober ☐< 55 ☐No Bone Disease☐

E — Enquire

Symptoms: _____

Allergies: _____

Medications: _____

Past / Pertinent History: _____

Last Ins & Outs: _____

Events: _____

Medication Given	
Name:	
Dosage:	
Frequency:	
Time Given:	
Name:	
Dosage:	
Frequency:	
Time Given:	

S — Summary

Incident Description: _____

Treatment: _____

EMS: Time of Call: _____Time of Arrival: _____Time of Departure: _____

Hazards: _____

Emergency Type: _____

Location: _____

Patient Details: _____

HANDOVER

Identify Patient: _____

Mechanism of Injury or Medical Condition: _____

Injuries or Info related to complaint: _____

Signs: LOC_____ HR _____ RR _____ SCTM _____ PUPILS_____

Treatment: _____

Allergies: _____

Medications: _____

Background: _____

Other pertinent info: _____

WILDERNESS PSYCHOLOGICAL FIRST AID

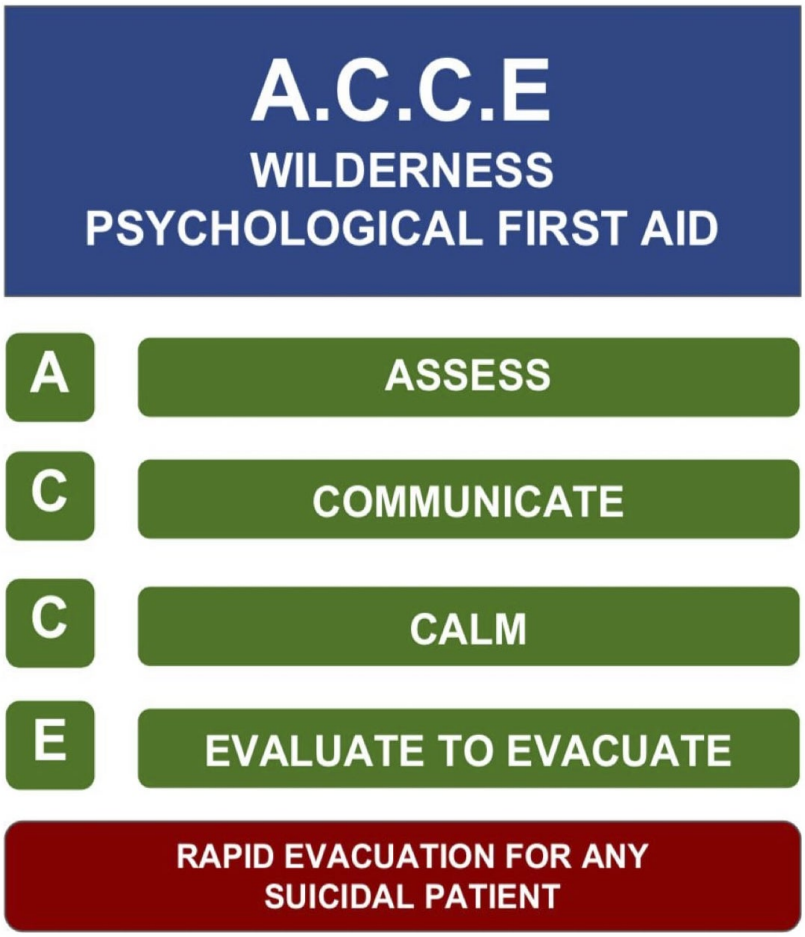
Mental Health First Aid Services

PROVIDER	PHONE	WEB
Lifeline Australia	131114	https://www.lifeline.org.au
Lifeline New Zealand	0800543354	https://www.lifeline.org.nz
Kids Helpline	1800551800	https://kidshelpline.com.au
MensLine Australia	1300789978	https://www.mensline.org.au/
Suicide Call Back Service	1300659467	https://www.suicidecallbackservice.org.au/
Beyond Blue	1300224636	https://www.beyondblue.org.au/
Open Arms - Veterans & Family Counselling	1800011046	https://www.openarms.gov.au/

State Crisis Numbers

STATE	PHONE	PROVIDER
NSW	1800011511	Mental Health Line
VIC	1300651251	Suicide Help Line
QLD	13432584	13 HEALTH
TAS	1800332388	Mental Health Services Helpline
SA	131465	Mental Health Assessment and Crisis Intervention Service
WA	1300555788	Mental Health Emergency Response Line
NT	1800682288	Mental Health Line
ACT	1800629354	Mental Health Triage Service

The A.C.C.E Model



The Mental Health Continuum

When using the "A.C.C.E' Model, keep it simple

What we're really looking for when using this assessment tool is whether the patient/incident is closer to **GREEN**, or closer to **RED**.

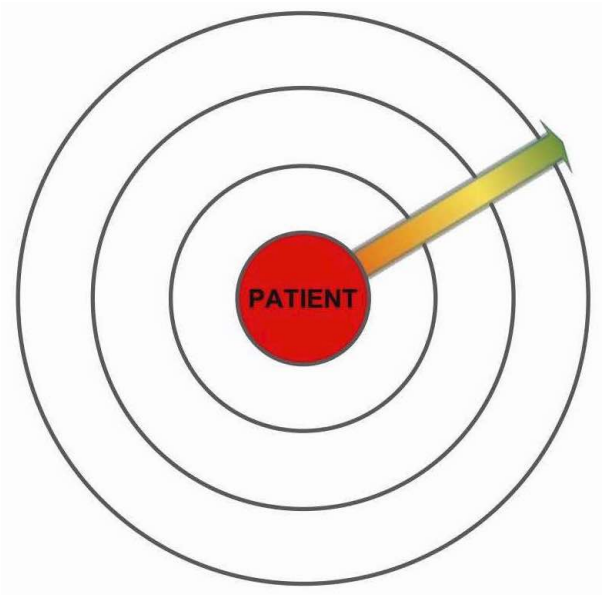
Assess: Individual

WHAT HAPPENED? Is the patient's behaviour consistent with the events?	Aware, coherent, insightful about self and situation	Confused, no insight about risks, can't self-regulate emotions, poor memory of events	Confused, incoherent, worsening memory of events	Poor situational awareness, no memory of events
HOW DOES THE *PATIENT* APPEAR TO BE FEELING?	Self-aware, calm, measured, composed	Irritable, impatient, anxious, upset	Angry, agitated, disturbed	Out-of-control, 'Frozen', raging
HOW DO *YOU* RATE THE PATIENT'S LEVEL OF DISTRESS?	Appropriate for situation, stress easily self-regulated	Elevated but can still function and perform tasks readily	Elevated, with difficulty functioning +/or performing normal tasks	High, unable to function , cannot perform normal tasks

Assess: Environment

HOW FAR ARE YOU FROM HELP?		Help is readily available; Reliable comms; Clear road access	Help available, but delayed; Intermittent comms; Difficult road access	Self-help only available; Restricted comms; Limited road access	Help is not available; No comms; No road access
WHAT RESOURCES DO YOU HAVE?		Hard-top shelter; Access to multiple comms devices; Access to multiple reliable vehicles; Access to defined Emerg Action Plan	Tent/Tarp shelter; Access to two comms devices only; Access to one reliable vehicle only; Emerg Action Plan exists, not on hand	Natural shelter only; Access to one comms device only- limited battery life; Uncertain access to vehicles; Emerg Action Plan insufficient for situation	No shelter available; No comms; No access to vehicles; No Emerg Action Plan in place
WHAT ARE THE CONDITIONS LIKE?	TERRAIN	Safe/easy	Rough/unknown	Difficult	Dangerous
	WEATHER	Calm/temperate	Changeable	Inclement	Extreme
	TIME OF DAY	Morning	Midday	Afternoon	Night
	ANIMALS	Not present	Present, known, contained	Present, unknown, uncontained	Present, dangerous

Assess: Group



Think of the **GROUP** (i.e. those under your duty of care) as a bullseye with the **PATIENT/INCIDENT** at the center and group members at various distances according to their exposure.

Questions:

1. Where to various group members get placed on the bullseye?
2. What requirements do they have (e.g. medical, food, shelter)
3. Do the total group requirements outweigh your resources

Communicate

Reassurance

Reassure the patient that they are safe and that their emotions are normal for the situation

Communication + Interaction Style

- Introduce yourself with your name and your role
- Keep your words clear, concise, simple and calm
- Maintain an even tone and pace of speech; don't raise your voice
- Give regular updates to the patient about what is happening in the situation
- Be non-judgemental, and demonstrate active listening
- Use age-appropriate language
- Maintain good eye contact
- Be patient

Physical Space

- Move the patient away from distressing sights
- Keep children with parents/caregivers where possible
- Your physical proximity to the patient largely depends on the situation and person – however, if you are unsure or wary (or if the patient is displaying high levels of agitation/aggression) don’t get too close and don’t restrict their movement

Calm

Breathing

- The aim here is to slow their breathing.
- You can try the following technique: breathe in through the nose for 4 seconds, breathe out through the nose for 4 seconds. Continue until the body settles.
- If the patient is having a panic attack or hyperventilating, try the same technique. It may also help to breathe and count with them to demonstrate the pace and depth of the breath

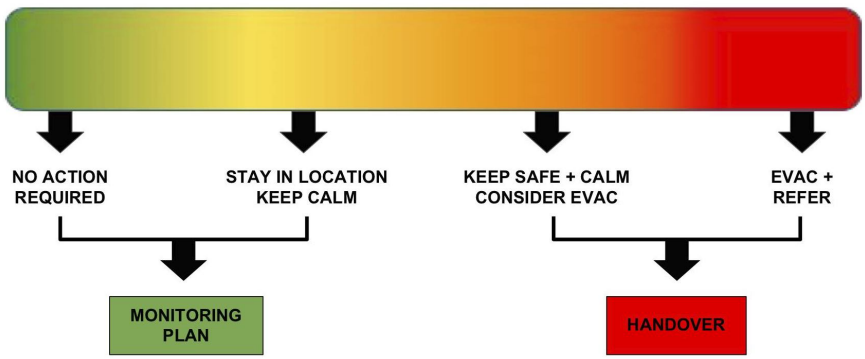
Grounding

- The aim here is to focus the patient on tangible things. You can try the following technique:
    1. Identify 4 things you can see, 4 things you can hear, and 4 things you can feel
    2. Identify 3 things you can see, 3 things you can hear, and 3 things you can feel
    3. Identify 2 things you can see, 2 things you can hear, and 2 things you can feel
    4. Identify 1 thing you can see, 1 thing you can hear, and 1 thing you can feel

Distraction

- The aim here is to get the patient thinking about something other than the incident/their condition
- Talk to the patient and ask them questions about their interests/hobbies/experiences/familiy/pets, etc
- Give the patient a simple physical/behavioural tasks (eg: hold onto a rope, roll up bandages, calm someone else, help setup/take down a tent, etc.)

Eval to Evac



Monitor +/ or Handover

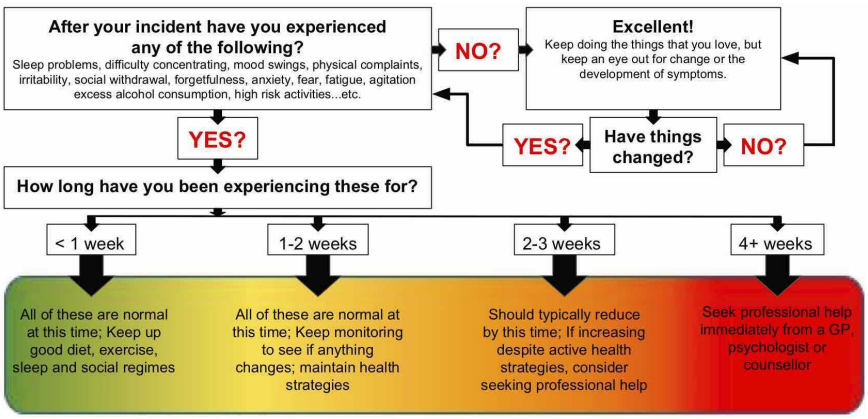
Monitor

1. Trigger
    - What caused the incident?
2. Treatment
    - What did you do that worked?
3. Tomorrow
    - How are you going to implement the effective treatment going forward?

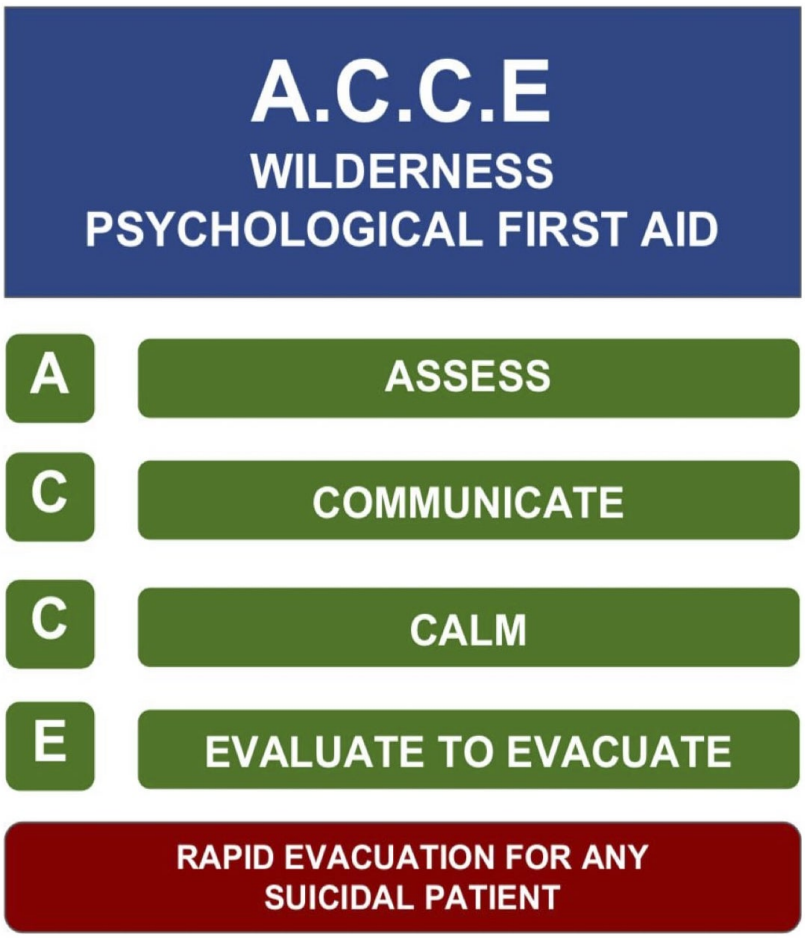
Handover

1. Incident
    - Brief description of what happened.
2. Signs & Symptoms
    - How the patient presented to you; what you noticed.
3. Treatment
    - What did you do for them? How did they respond?

Self Care



Notes



Notes about the A.C.C.E Model

- This model is **adaptable** to almost any **remote** or **wilderness** situation, regardless of the length of time or distance from resources.
- Focus of the model is **situational management** rather than individual support
- Designed from **evidence-based research** into critical incidents and management of patients with psychological presentations
- **Remember:** the aim of PFA is to keep the patient calm and safe until further help arrives.

Resources

The following resources are from:
https://www.cci.health.wa.gov.au/Resources/Looking-After-Others

Centre for Clinical Interventions

What is anxiety?

Many people experiencing the symptoms of anxiety can begin to wonder if there is something really wrong with them. One typical fear is that they might be going crazy. Unfortunately, the reactions and comments from other people such as, ‘just get yourself together’ are not very helpful.

Although you might feel alone in your struggle against anxious moods, the reality is that many people experience these moods either from time to time, or on a more regular basis. In fact, it is estimated that 1 in every 5 experience significantly anxious mood at some time in their life.

Anxiety can effect any kind of person at any stage of their life, whether they are an introvert or an extrovert, socially active or shy, youthful or elderly, male or female, wealthy or poor. Whatever your distinction, you can become anxious. That means that any person you know is also fair game. So remember, you are not alone.

Understanding Anxiety

Feeling afraid is very much a part of the experience of being human. It occurs in response to realistically anticipated danger and therefore is a survival instinct. For example, if a ferocious animal confronted us it is likely that we would respond with fear. This response is important because it initiates a whole series of physical and behavioural changes that ultimately serve to protect us. In this example, when confronted by an animal, the feeling of fear would probably lead us to either run for our lives or become sufficiently ‘pumped up’ to physically defend ourselves. As you can see from this example, the experience of fear is part of a process of survival.

The experience of anxiety is very similar to the experience of fear - the main difference is that anxiety occurs in the absence of real danger. That is, the individual may think that they are in danger but the reality is that they are not. To illustrate this, think of the anxiety one may feel when walking down a poorly lit alley. The individual may feel anxious because they perceive some potential danger. This may not mean that there is any real danger in walking down this particular alley, but what causes the experience of anxiety is that the person believes that they are in danger. Therefore, the experience of anxiety and fear are basically the same except that in the case of anxiety, there may not be any actual danger - the person just thinks there is.

Fight/Flight Response

It is important to fully understand the way our bodies react to threat or danger, whether real or imagined. When a person is in danger, or believes that they are in danger a number of changes occur. This response has been named the fight/flight response. As previously explained, when confronted with danger we will typically flee from the situation, or stand and fight. The main purpose of the fight/flight response is to protect the individual. It is therefore important to remember that the

experience of anxiety is not in itself, harmful. When a person's fight/flight response is activated, three major systems are affected. These are the physical, cognitive and behavioural systems.

Physical System

When we believe that we are in danger, our whole physical system undergoes some major, temporary changes designed to enhance our ability to either run away, or stand and be ready to fight. Physically, as soon as danger is perceived, the brain sends a message to our autonomic nervous system. Our autonomic nervous system has two sections: the sympathetic branch and the parasympathetic branch. These two sections control the physical changes that occur in the fight/flight response. The sympathetic branch is the part that activates the various areas of the body to be ready for action. When the sympathetic branch is activated, it includes all areas of the body, and therefore, the person experiences physical changes from head to toe. To get things moving, the sympathetic nervous system releases two chemicals from the adrenal glands on the kidneys. These chemicals are called adrenalin and noradrenalin and are basically messengers that serve to maintain the physical changes for a sufficient amount of time. So what are these physical changes that the sympathetic mechanism produces when you are anxious?

1. An increase in heart rate and strength of beat. One physical change that is quite noticeable to the person experiencing the fight/flight response, is an increase in heart rate and the strength of heartbeat. An increase in heart rate enables blood to be pumped around the body faster, so that oxygen gets delivered more promptly to the various tissues of the body and waste products can be efficiently eliminated.
2. A redistribution of blood from areas that aren't as vital to those that are. There is also a change in blood flow - away from places where it is not needed (such as skin, fingers and toes) towards the places it is likely to be needed (large organs and muscles). This is very useful because if we were attacked and cut in some way we would be less likely to bleed to death, as the blood will be with the vital organs. This physical change results in the skin looking pale and feeling cold, and also in the experience of cold, numb and tingling fingers and toes.
3. An increase in the rate and depth of breathing. As well as changes to heart rate, there are also changes to the speed and depth of breathing. This is very important, as it provides the tissues with the extra amount of oxygen required to prepare for action. The feelings produced by this increase in breathing can include breathlessness, choking or smothering feelings, tightness and pain in the chest, and sighing and yawning. One of the main side effects of this increase in breathing is that the blood supply to the head is actually decreased. This is not dangerous but can produce a collection of unpleasant symptoms, including: dizziness, light-headedness, blurred vision, confusion, feelings of unreality and hot flushes.
4. An increase in sweating. Another physical change in the fight/flight response is an increase in sweating. This causes the body to become more slippery,

making it harder for a predator to grab, and also cooling the body and thus preventing it from overheating.

5. Widening of the pupils of the eyes. One effect of the fight/flight response that people are often unaware of, is that the pupils widen to let in more light, which may result in the experience of blurred vision, spots before the eyes, or just a sense that the light is too bright. This change enables the person to more effectively use their sight to identify any hidden dangers such as something lurking in the shadows.
6. Decreased activity of the digestive system. The decreased activity of the digestive system allows more energy to be diverted to systems more immediately related to fight or flight. The range of effects you might notice as a result of this body change are a decrease in salivation, resulting in a dry mouth and decreased activity in the digestive system, often producing feelings of nausea, a heavy stomach or even constipation.
7. Muscle tension. Finally, many of the muscle groups tense up in preparation for fight/flight and this results in subjective feelings of tension, sometimes resulting in aches and pains and trembling and shaking. The whole physical process is a comprehensive one that often leaves the individual feeling quite exhausted.

Behavioural System

As already mentioned, the two main behaviours associated with fear and anxiety are to either fight or flee. Therefore, the overwhelming urges associated with this response are those of aggression and a desire to escape, wherever you are. Often this is not possible (due to social constraints) and so people often express the urges through behaviours such as foot tapping, pacing or snapping at people.

Cognitive System

As the main objective of the fight/flight response is to alert the person to the possible existence of danger, one major cognitive change is that the individual begins to shift their attention to the surroundings to search for potential threat. This accounts for the difficulty in concentrating that people who are anxious experience. This is a normal and important part of the fight/flight response as its purpose is to stop you from attending to your ongoing chores and to permit you to scan your surroundings for possible danger. Sometimes an obvious threat cannot be found. Unfortunately, most of us cannot accept not having an explanation for something and end up searching within themselves for an explanation. This often results in people thinking that there is something wrong with them - they must be going crazy or dying.

Restoration of the Systems

Once the immediate danger has abated, the body begins a process of restoration back to a more relaxed state. This is once again controlled by the autonomic nervous system. This time it instructs the parasympathetic branch to begin the process of counteracting the sympathetic branch. As a result, the heart rate begins to slow, breathing rate slows, the body’s temperature begins to lower and

the muscles begin to relax. Part of the process of restoration is that the systems do not return to normal straight away. Some arousal continues and this is for a very good reason. In primitive times, if a wild animal confronted us it would be foolish to relax and be off guard as soon as the animal began to back off. The chances of danger continuing in such a case causes the body to remain prepared for the need to once again face danger. Therefore, some residual effects of the fight/flight response remain for some time and only gradually taper off. This can leave the individual feeling ‘keyed up’ for some time afterwards. This helps to understand why it is that people can feel anxious for ongoing periods of time when no obvious stressor is present.

What Causes Anxiety?

The combination of factors which result in an individual developing an anxiety disorder differ from person to person. However, there are some major factors that have been identified, which may be common to sufferers. These factors can be effectively divided into biological and psychological causes.

Biological Factors

A genetic factor has been linked to the development of anxiety disorders. For example, in obsessive-compulsive disorder, about 20% of first-degree relatives have also suffered from the condition. Overall, based on family studies, it has been suggested that individuals may inherit a vulnerability to developing an anxiety disorder.

Psychological Factors

Having this genetic vulnerability does not imply that those individuals will develop an anxiety disorder. A great deal depends on the lifestyle of that person, the types of life stressors they have encountered and their early learning. For example, if we were taught to fear certain neutral situations as a child it can become difficult to extinguish these learned patterns of behaviour. Therefore, we may have developed certain patterns of thinking and behaving which contribute to the development of an anxiety disorder.

Summary

As you can see from this description of the fight/flight response, anxiety is an important emotion that serves to protect us from harm. For some people the fight/flight response becomes activated in situations where no real danger is present. The types of situations vary greatly from person to person. For example, simply anticipating poor performance on an examination can be enough to activate the fight/flight response. An anxiety disorder is usually diagnosed when a person cannot manage to function adequately in their daily life due to the frequency and severity of the symptoms of anxiety. It is important to keep in mind however, that some anxiety is functional, enabling us to get to work on time, meet demands, cross busy streets and remain aware of our surroundings.

Coping with Stress

Stress and Stressors

Stress is something that is part of normal life, in that it is experienced by everyone from time-to-time. However, some people suffer from stress which is so frequent or so severe that it can seriously impact on their quality of life. Stress can come from a huge range of sources (stressors), such as:

- Relationships with others
- Work-related issues
- Study demands
- Coping with illness
- Life changes, such as marriage, retirement, divorce
- Day-to-day activities and tasks
- Positive events, such as organising holidays or parties
- Juggling many roles or tasks at the same time Some people are aware of what tends to trigger their stress, and this increases their ability to either prevent stress or to handle it more effectively. Many others are less able to deal with stress, and identifying stressors is a key step in this. If you often experience stress, take some time to consider what tends to set it off for you.

Symptoms of Stress

Some people do not even notice that they are stressed until symptoms begin to occur, including:

- Irritability or moodiness
- Interrupted sleep
- Worrying or feeling of anxiety
- Back and neck pain
- Frequent headaches, minor to migraine
- Upset stomach
- Increased blood pressure
- Changes in appetite
- Rashes or skin breakouts
- Chest pains
- Making existing physical problems worse
- More susceptible to cold/flu and slower recovery These symptoms reduce quality of life, and people suffering from stress may notice that work performance or relationships suffer more as a result. You may be able to use some the strategies listed here, or you may find it useful to consult a professional for more help.

Stress Management Tips

1. Identify your stressors, and see if there are some things within your control to manage better. Some things will be beyond your control, for example if you work a job that is based on working towards deadlines then you can’t change

this without changing jobs. But perhaps you can control some aspects, such as scheduling to have at least a short lunch break each day, or to go to bed earlier so that you have more energy to cope with the daytime.

2. Build regular exercise into your life - as well as being part of a healthy , balanced lifestyle and giving you more energy, many people find that working out at the gym or playing sport helps them to unwind.
3. Make sure that you eat and sleep well.
4. Take time out for family, friends and recreational activities. Most of us know that this is important but we do not all do it. If you find it hard to make time for this, perhaps you need to take deliberate steps to have time out, such as set aside one evening a week where you meet up with friends or enjoy a hobby, or set aside one day of the weekend for relaxing at home.
5. Problem-solving techniques can be a useful way of clarifying the problem, brainstorming possible solutions, and then choosing one to put into action after listing the pros and cons of each option. See the handout Problem Solving for more details about this.
6. Learn calming techniques such as controlled breathing and progressive muscle relaxation, to train your mind and body to become more relaxed. These techniques require practice but can be helpful with regular use. See handouts Calming Technique and Progressive Muscle Relaxation.
7. You may wish to speak to a professional about assertiveness training and communication skills which can help you to deal with challenging situations more effectively, thereby reducing stress. See the handout Assertive Communication.
8. Last but definitely not least, consider whether there is negative thinking which is contributing to your stress. Negative thinking can make us worry more than is necessary, increasing stress, and generally does not motivate us to take positive actions. See the handouts Thinking & Feeling, Analysing Your Thinking and Changing Your Thinking.

What is Panic?

To understand panic, we need to understand fear. You can think of fear as an automatic alarm response that switches on the moment there is danger. Think about what would happen to you if a dangerous animal approached you. For most people it would be panic stations! You, and almost everyone, would go through a whole series of bodily changes, like your heart pumping, breathing faster, sweating, all in order to respond to the danger in front of you. This alarm response would probably lead us to either run for our lives or become sufficiently ‘pumped up’ to physically defend ourselves. This alarm response is an important survival mechanism called the fight or flight response.

Sometimes, however, it is possible to have this intense fear response when there is no danger – it is a false alarm that seems to happen when you least expect it. It is like someone ringing the fire alarm when there is no fire! Essentially, a panic attack is a false alarm.

Many people experience some mild sensations when they feel anxious about something, but a panic attack is much more intense than usual. A panic attack is usually described as a sudden escalating surge of extreme fear. Some people portray the experience of panic as ‘sheer terror’. Let’s have a look at some of the symptoms of a panic attack:

Panic Attack Symptoms

- Skipping, racing or pounding heart
- Sweating
- Trembling or shaking
- Shortness of breath or difficulty breathing
- Choking sensations
- Chest pain, pressure or discomfort
- Nausea, stomach problems or sudden diarrhoea
- Dizziness, lightheadedness, feeling faint
- Tingling or numbness in parts of your body
- Hot flushes or chills
- Feeling things around you are strange, unreal, detached, unfamiliar, or feeling detached from body
- Thoughts of losing control or going crazy
- Fear of dying

As you can see from the list, many of the symptoms are similar to what you might experience if you were in a truly dangerous situation. A panic attack can be very frightening and you may feel a strong desire to escape the situation. Many of the symptoms may appear to indicate some medical condition and some people seek emergency assistance.

Characteristics of a Panic Attack

- It peaks quickly - between 1 to 10 minutes
- The apex of the panic attack lasts for approximately 5 to 10 minutes (unless constantly rekindled)
- The initial attack is usually described as “coming out of the blue” and not consistently associated with a specific situation, although with time panics can become associated with specific situations
- The attack is not linked to marked physical exertion
- The attacks are recurrent over time
- During an attack the person experiences a strong urge to escape to safety

Many people believe that they may faint whilst having a panic attack. This is highly unlikely because the physiological system producing a panic attack is the opposite of the one that produces fainting. Sometimes people have panic attacks that occur during the night when they are sleeping. They wake from sleep in a state of panic. These can be very frightening because they occur without an obvious trigger. Panic attacks in, and of themselves, are not a psychiatric condition. However, panic attacks constitute the key ingredient of Panic Disorder if

the person experiences at least 4 symptoms of the list previously described, the attacks peak within about 10 minutes and the person has a persistent fear of having another attack.

Panic Disorder and Agoraphobia

Someone with panic disorder has a persistent fear of having another attack or worries about the consequences of the attack. Many people change their behaviour to try to prevent panic attacks. Some people are affected so much that they try to avoid any place where it might be difficult to get help or to escape from. When this avoidance is severe it is called Agoraphobia.

Panic Disorder is more common than you think. A recent study reported that 22.7% of people have reported experience with panic attacks in their lifetime. 3.7% have experienced Panic Disorder and 1.1% have experienced Panic Disorder plus Agoraphobia. * These numbers equate to millions of people world wide. If left untreated, Panic Disorder may become accompanied by depression, other anxiety disorders, dependence on alcohol or drugs and may also lead to significant social and occupational impairment.

Biology and Psychology of Panic

Panic attacks (the key feature of Panic Disorder) can be seen as a blend of biological, emotional & psychological reactions. The emotional response is purely fear. The biological & psychological reactions are described in more detail below.

Biological Reactions 1: Fight or Flight

When there is real danger, or when we believe there is danger, our bodies go through a series of changes called the fight/flight response. Basically, our bodies are designed to physically respond when we believe a threat exists, in case we need to either run away, or stand and fight. Some of these changes are:

- an increase in heart rate and strength of heart beat. This enables blood and oxygen to be pumped around the body faster.
- an increase in the rate and depth of breathing. This maintains oxygen and carbon dioxide levels.
- an increase in sweating. This causes the body to become more slippery, making it harder for a predator to grab, and also cooling the body.
- muscle tension in preparation for fight/flight. This results in subjective feelings of tension, sometimes resulting in aches and pains and trembling and shaking. When we become anxious and afraid in situations where there is no real danger, our body sets off an automatic biological “alarm”. However, in this case it has set off a “false alarm”, because there is no danger to ‘fight’ or run from.

Biological Reactions 2: Hyperventilation & Anxious Breathing

When we breathe in we obtain oxygen that can then be used by the body, and we breathe out to expel the product of our metabolic processes - carbon dioxide. The body naturally maintains optimal levels of both oxygen and carbon dioxide. When we are anxious, the optimal level of carbon dioxide is disrupted because we begin to hyperventilate, or breathe too much. If the body cannot return carbon dioxide levels to the optimal range, we experience further symptoms such as dizziness, light-headedness, headache, weakness and tingling in the extremities, and muscle stiffness. For people with panic, these physiological sensations can be quite distressing, as they may be perceived as being a sign of an oncoming attack, or something dangerous such as a heart attack. However these are largely related to the fight or flight response and overbreathing, not physical problems.

Psychological Reactions 1: Thinking Associated with Panic

We've described the physical symptoms of panic. People who panic are very good at noticing these symptoms. They constantly scan their bodies for these symptoms. This scanning for internal sensations becomes an automatic habit. Once they have noticed the symptoms they are often interpreted as signs of danger. This can result in people thinking that there is something wrong with them, that they must be going crazy or losing control or that they are going to die. There are a number of types of thinking that often occur during panic, including:

- Catastrophic thoughts about normal or anxious physical sensations (eg "My heart skipped a beat - I must be having a heart attack!")
- Over-estimating the chance that they will have a panic attack (eg "I'll definitely have a panic attack if I catch the bus to work")
- Over-estimating the cost of having a panic attack: thinking that the consequences of having a panic attack will be very serious or very negative.

Psychological Reactions 2: Behaviours that Keep Panic Going

When we feel anxious or expect to feel anxious, we often act in some way to control our anxiety. One way you may do this is by keeping away from situations where you might panic. This is called avoidance, and can include:

- Situations where you've had panic attacks in the past
- Situations from which it is difficult to escape, or where it might be difficult to get help, such as public transport, shopping centres, driving in peak hour traffic
- Situations or activities which might result in similar sensations, such as physical activity, drinking coffee, having sex, emotional activities such getting angry

A second response may be to behave differently, or to use "safety behaviours". The following are examples of these; you might make sure you are near an escape route, carry medication with you, or ensure that you are next to a wall to lean on. Or you may use other more subtle methods like trying to distract yourself from your anxiety by seeking reassurance, reading something, or bringing music

to listen to. Although this may not seem harmful to begin with, if you become dependent on these behaviours you can become even more distressed if one day it's not possible to use them.

What is Bipolar Disorder?

Bipolar Disorder or Manic Depression is a mood disorder, and is the name given to the experience of abnormal moods or exaggerated mood swings. This illness is characterised by the experience of extremely "high" moods where one becomes extremely euphoric or elated, and the experience of extremely "low" moods where one becomes extremely sad and finds it difficult to experience pleasure. The high moods are called manic episodes and the low moods are called depressive episodes. These episodes can range from mild to severe and affect how a person thinks, feels, and acts. However, it is important to remember that some people may experience different patterns associated with their disorder. For example, some people may experience only one episode of mania but more frequent episodes of depression.

Bipolar Disorder occurs in approximately 1% of the population, that is, about 1 in every 100 will experience an episode that will probably require hospital care. This illness affects men and women equally, and typically begins in their early to late 20s.

Features of Bipolar Disorder

Manic Episodes

Mania is an extreme mood state of this disorder. It describes an abnormally elevated, euphoric, driven and/or irritable mood state. Hypomania is the term given to the more moderate form of elevated mood. It can be managed often without the need for hospitalisation as the person remains in contact with reality. However, it is very easy to move rapidly from hypomania into a manic episode. Symptoms of mania include:

- Irritability: Irritability as described in the Oxford dictionary means "quick to anger, touchy." Many people, when in an elevated mood state, experience a rapid flow of ideas and thoughts. Because of this rapid thought process, they become easily angered when people don't seem to comprehend their ideas or enthusiasm for some new scheme.
- Decreased need for sleep: One of the most common symptoms of mania and often an early warning sign is the increased experience of energy and lack of need for sleep.
- Rapid flow of ideas: People who are becoming manic experience an increase in the speed at which they think. They move more quickly from one subject to another. Sometimes thoughts can become so rapid that they begin to make no sense, developing into a jumbled, incoherent message that the listener can no longer understand. Grandiose ideas: It is common for people who are manic to think that they are more talented than others, or have unique gifts.

As the person's mood becomes more elevated, these beliefs can become delusional in nature, with people.

- Uncharacteristically poor judgement: A person's ability to make rational decisions can become impaired and they may make inappropriate decisions or decisions that are out of character.
- Increased sexual drive: People when they become manic often experience increased libido, and may make less well-judged decisions about the sexual partners.

Depressive Episodes

Depression is a mood state that is characterised by a significantly lowered mood. Its severity, persistence, duration, and the presence of characteristic symptoms can distinguish a major depressive episode, the other extreme mood state of bipolar disorder, from a milder episode of depression. The most common symptoms of depression include:

- Persistent sad, anxious, or empty mood: People often describe depression as an overwhelming feeling of sadness and hopelessness. They may experience a loss of enjoyment in the activities of everyday life that they used to take a lot of pleasure in.
- Poor or disrupted sleep: A person when they are depressed often experience sleep disturbances, and this can be due to increased anxiety. They then find it difficult to fall asleep, or wake up frequently during the night worrying about day-to-day events or wake early in the morning and are unable to get back to sleep.
- Feelings of worthlessness or hopelessness: Sometimes people become overwhelmed with a sense of their own inability to be of use to anyone, and can become convinced that they are useless and worthless. Thoughts may revolve around the hopelessness of the situation and the future.
- Decreased interest in sex: As the person becomes more depressed, they gradually become less interested in social activities and sex.
- Poor concentration: Thinking can become slowed and the person can have difficulty in making decisions. They find it difficult to concentrate on reading a book or on the day to day tasks such as shopping. This can often create anxiety or agitation in a person.
- Thoughts of suicide, or suicide attempts: When a person becomes overwhelmed by their feelings of hopelessness and despair, they may have thoughts of ending their lives or make plans to commit suicide.

Mixed Episodes

A mixed episode is characterised by the experience of both depressive and manic symptoms nearly everyday for a period of time. The person experiences rapidly alternating moods, eg, irritability, euphoria, sadness, and there may be insomnia, agitation, hallucinations and delusions, suicidal thoughts, etc.

Diagnosis of Bipolar Disorder

Correctly identifying an illness can help you begin to explore the various treatment options available to you so that you can better manage your illness. As such, having an accurate diagnosis is the beginning of becoming well. The following diagnoses are based on the definitions and criteria used in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) by the American Psychiatric Association, 1994.

Bipolar I Disorder is the most common and prevalent of the different bipolar mood disorders. It is characterised by the experience of full-blown manic episodes and severe depressive episodes. The patterns of abnormal mood states are very varied and different individuals may experience a different course of the illness. Many physicians refer to bipolar I disorder as a relapsing and remitting illness, where symptoms come and go. It is therefore, important to ensure that treatment is continued even if the symptoms are no longer present, to prevent an episode relapse.

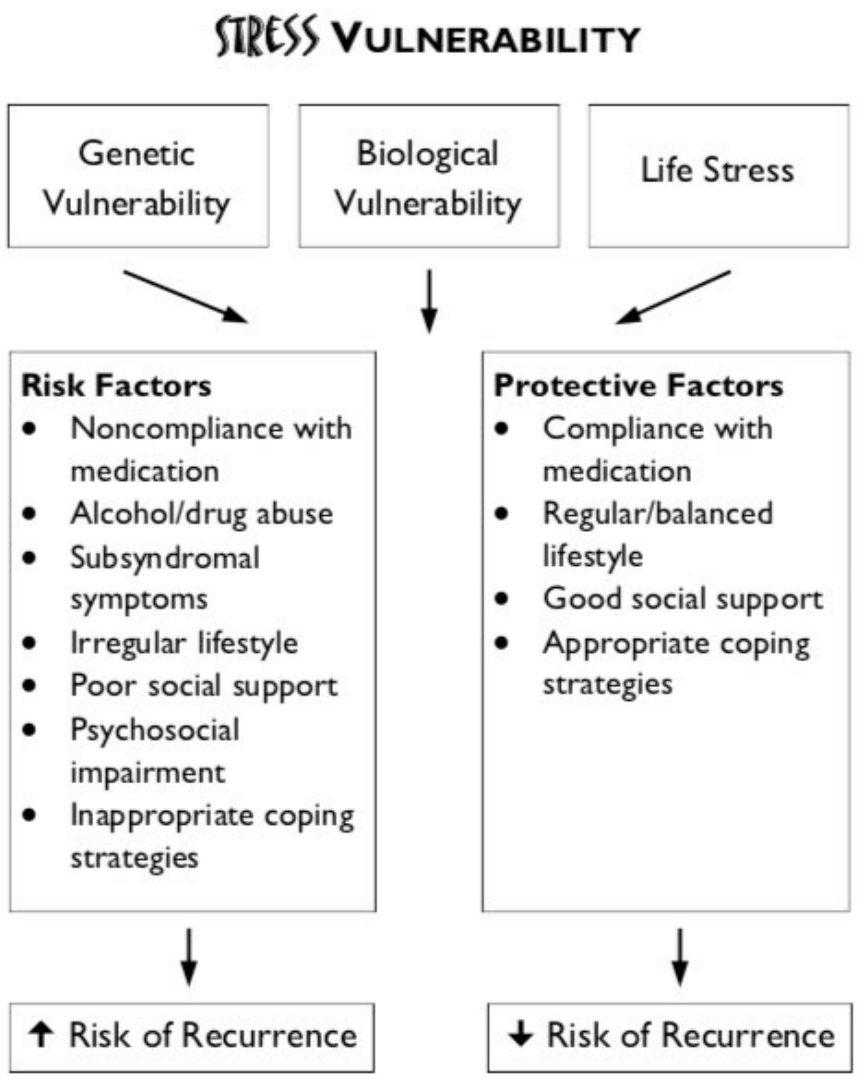
Bipolar II Disorder is characterised by the experience of full-blown episodes of depression and episodes of hypomania (i.e., with mild manic symptoms) that almost never developed into full-fledged mania.

Cyclothymic Disorder is characterised by frequent short periods of mild depressive symptoms and hypomania, mixed in with short periods of normal mood. Though a patient with cyclothymic disorder does not experience major depression or mania, they may go on to develop bipolar I or II disorder. Patients with bipolar I or bipolar II may experience frequent mood cycling. Patients who experience more than four episodes of hypomania, mania, and/or depression in a year are said to experience Rapid Cycling. These patients tend to alternate between extreme mood states separated by short periods of being well, if at all.

Note: A proper diagnosis should only be made by a medical doctor or psychiatrist, or a trained mental health practitioner. The information provided in this handout is not enough for an accurate diagnosis to be made by anyone who is not a trained mental health professional or physician. Please speak to an appropriate professional if you have any concerns or questions regarding the information provided here.

What Causes Bipolar Disorder?

No one factor has been identified to cause bipolar disorder, that is, it is not caused by a person, event, or experience. There are a number of factors that interact with each other that may contribute to the development of this disorder in some people. In this handout, we present to you a way of understanding how all these factors come together to trigger the onset of this illness.



First, we begin by looking at three key factors in this model, namely: genetic vulnerability, biological vulnerability and life stress.

Genetic Vulnerability

Bipolar disorder tends to run in families. First degree relatives of people with bipolar disorder have an increased risk of developing bipolar disorder. Children of bipolar patients face an 8% risk of getting the illness versus 1% in the population. Children of bipolar patients also face an increased risk (12%) of getting unipolar depression (i.e., depression only, without mania). Identical twins are also more likely to both develop this disorder than fraternal twins. While these results indicate to some extent that this disorder is genetically inherited, they also suggest that there are other factors that may contribute to its development.

Biological Vulnerability

This refers to possible biochemical imbalances in the brain that makes a person vulnerable to experiencing mood episodes. An imbalance of brain chemicals or an inability for them to function properly may lead to episodes of “high” or “low”

moods.

Life Stress

Stressful events or circumstances in a person's life, such as, family conflicts, employment difficulties, bereavement, or even positive events, such as getting married, having children, moving house, etc, can place extra demands on the person, leading to them feeling stressed, frustrated, anxious, sad, etc. The occurrence of bipolar disorder can thus be explained as an interaction of the 3 above factors. A person who is genetically and/or biologically vulnerable may not necessarily develop bipolar disorder. These vulnerabilities are affected by how they cope with stressors in their life. For example, a person who has a family history of diabetes may not develop diabetes if they are careful with they eat and have enough exercise. This brings us to a discussion on protective and risk factors.

Protective & Risk Factors

A risk factor is something that will increase the chances of a person who is already vulnerable becoming ill. Examples of risk factors are: poor or maladaptive coping strategies, alcohol or drug use, irregular daily routines, interpersonal conflicts, stressful events, etc. Protective factors, on the other hand, are those that can help to prevent a vulnerable person from becoming ill. Protective factors include:

- good coping strategies,
- good social support networks,
- effective communication and,
- problem solving skills, etc.

It is when the risk factors outweigh the protective factors, that the chances of developing the disorder are high. This principle applies when considering the risk of recurrence as well.

Course of Illness

While some patients may experience long periods of normal moods, most individuals with bipolar disorder will experience repeated manic and/or depressive episodes throughout their lifetime. The ratio of manic episodes to depressive episodes will vary from one individual to the next, as will the frequency of episodes. Some individuals may experience only two or three episodes in their lifetime while others may experience a rapid cycling pattern of four or more episodes of illness per year. Whatever the pattern, it is important that bipolar patients learn effective ways of managing their illness and preventing the recurrence of further episodes.

Medications for Bipolar Disorder

The recognised standard treatment for bipolar disorder is medication, which focuses on controlling or eliminating the symptoms and then maintaining the symptom-free state by preventing relapse. The effective use of medication requires that you work closely with your medical practitioner. Some patients may respond well and experience few side effects with one type of medication, while others may do better with another. Thus, when taking medication, it is important that you monitor its effects and consult with your doctor.

Principles of Medication Management

1. For medication to be of benefit, you should carefully follow the prescribed treatment and take note of your symptoms and side effects.
2. If side effects develop, these should be reported to your doctor as soon as possible to avoid prolonged discomfort. It is strongly advised that you do not stop medication abruptly before first consulting with your doctor. This could bring on a return of a manic or depressive episode.
3. Alcohol, illicit medications, and other prescribed medicines may cause your medication for bipolar disorder to be ineffective and may increase side effects. You should report all other medications and substances you are taking to your doctor to ensure that none adversely interact with the medication prescribed for bipolar disorder.
4. Effective medical management of bipolar disorder requires you to monitor your symptoms and side effects, and work with your doctor to adjust dosages or types of medications.

Phases of Treatment

There are usually three phases to medical treatment for bipolar disorder. The most important aim, if you are experiencing an episode of mania, hypomania, or major depression, is to control or eliminate the symptoms so that they can return to a normal level of day-to-day functioning. The duration of this acute phase of treatment may last from 6 weeks to 6 months. Sometimes, longer periods are necessary in order to find the most effective medications with minimal side effects.

In continuation treatment, the main aim is to maintain the symptom-free state by preventing relapse, which is the return of the most recent mood episode.

The third phase, the maintenance phase, is critical and essential for all patients with bipolar disorder. The goal for maintenance treatment is to prevent recurrence, that is, to prevent new episodes of mania, hypomania, or depression from occurring. For bipolar patients, as with other medical conditions such as diabetes or hypertension, maintenance treatment may last 5 years, 10 years, or a lifetime. But remember, prolonged symptom control will help you to function better in your daily lives.

For all phases of treatment and all medications, patients must take the prescribed medication/s on a daily basis. Unlike medications like paracetamol or antibiotics that are taken only when a person actually experiences a headache or has the

‘flu, medications for bipolar disorder must be taken regularly – on both good days and bad days – at the same dosage.

Types of Medications for Bipolar Disorder

Mood Stabilisers

A mood stabiliser is a medication that is used to decrease the chance of having further episodes of mania or depression. They are the first line agents for bipolar disorder. Depending on the associated symptoms with this disorder, anti-depressants or antipsychotics may also be used. A mood stabiliser is given to a person as a maintenance medication because it regulates mood swings but doesn’t take away the cause. Feeling well does not mean you can stop taking mood stabilisers, it means the medication is keeping you stable. The most common mood stabilisers are Lithium Carbonate, Carbamazepine, and Sodium Valproate. Sometimes these medications are used on their own or in combination with other medications.

Antidepressants

Antidepressants can also be used with mood stabilisers in the acute, continuation, and/or maintenance phases of medical treatment. Thereisnooneparticularantidepressantthatis more effective than the others in bipolar disorder. In fact, there is a significant risk for antidepressants to induce or cause a “switch” to manic or hypomanic episodes, especially if a patient on antidepressants is not taking a mood stabiliser. Common antidepressants include:

- Selective serotonin reuptake inhibitors (SSRIs) - fluoxetine, paroxetine, sertraline
- Tricyclics - imipramine, amitriptyline, desipramine, dolthiepin
- Monoamine oxidase inhibitors (MAOIs) - phenelzine and tranylcypromine

Antipsychotic Medication

Antipsychotics may also be used both in the acute phase of the disorder and sometimes as a longer term treatment. Common antipsychotic agents include haloperidol, chlorpromazine, thioridazine, risperidone, and olanzapine. These medications are often combined with mood stabilisers to assist in controlling hallucinations, or delusions, to induce sleep, to reduce inappropriate grandiosity, or decrease irritability or impulsive behaviours. These medications are usually not used for treating hypomania. Although antipsychotics are most often used in treatment of the acute phase of mania, some patients may continue on smaller dosages to ensure that they do not experience a relapse of psychotic or manic symptoms.

Another often used medication is clonazepam, which is classed under the benzodiazepines. This is used as an adjunct with other medications (mood stabilisers and antipsychotics) to aid in inducing sleep, reducing psychomotor agitation, and slowing racing thoughts and pressured speech.

Remember that it is very important that you talk openly with your prescribing doctor or psychiatrist and not to stop your medication without first discussing it with them.

Psychotherapy for Bipolar Disorder

Although effective medications have been found for bipolar disorder, many patients still experience episode recurrences and relapse. Some experience between- episode symptoms that may not be serious enough to be considered a full-blown episode, but could still cause some discomfort and interference with day-to-day activities. A high rate of relapse and episode recurrences could be because of medication non- compliance, alcohol and drug use, high stress levels, many between-episode symptoms, and poor daily functioning. These issues have alerted mental health professionals to try psychotherapy and psychosocial interventions, in addition to medication, to improve illness outcome and quality of life for bipolar patients.

Cognitive Behavioural Therapy

A treatment approach that has been well researched for a wide range of adult psychiatric disorders is cognitive behavioural therapy (CBT), which has recently been adapted to bipolar disorder. Although CBT for bipolar disorder is relatively new, it has been used in the treatment of a range of psychiatric disorders including unipolar depression, generalised anxiety disorder, panic disorder, social phobia, and eating disorders. It has also been applied as an adjunctive treatment for disorders such as obsessive-compulsive disorder, personality disorders, and schizophrenia. This information package is based on this approach. CBT is a structured and time-limited intervention. It is a comprehensive psychological therapy in which there is an emphasis on collaboration between therapist and patient, and on active participation by the patient in achieving therapeutic goals.

CBT is also focused on problem solving. The central aim of CBT is to teach patients how their thoughts and beliefs play an important role in the way they respond to situations and people. The CBT approach also teaches patients the tools that could them to make their response more helpful. CBT can play a role in teaching bipolar patients about their disorder and helping them deal with adjustment difficulties. CBT can also help patients cope with everyday stressors through active problem solving, and teach patients to monitor and regulate their own thoughts, moods, and activities, and thus be prepared to manage between-episode symptoms.

Research

CBT for bipolar disorder has been evaluated in a controlled trial here at the Centre for Clinical Interventions. The results of our study showed that CBT for bipolar disorder was effective in helping patients feel less depressed and more

confident about managing their illness. While this type of psychosocial treatment is still being evaluated worldwide, preliminary results from a number of studies have been positive.

Summary

Because bipolar patients experience episode recurrences and some difficulty in everyday living, some form of psychosocial treatment is recommended as an addition to medication. Recent research has found that cognitive behavioural therapy for bipolar disorder appears to be beneficial for patients. However, bipolar patients are reminded that this is an adjunctive treatment and must not be considered as a substitute for medication.

What is Depression?

Many people experiencing the symptoms of depression might begin to wonder if there is something really wrong with them. One typical fear is that they might be going crazy. Unfortunately, the reactions and comments from other people such as, “Just get yourself together!” are not very helpful.

Although you might feel alone in your struggle against depressive moods, the reality is that many people experience these moods from time to time, or even regularly. In fact, it is estimated that 1 in every 4 people experience significantly depressed mood at some time in their life.

Depression can affect any kind of person at any stage of their life. You may be an introvert or an extrovert, socially active or shy, youthful or elderly, male or female, wealthy or poor. Whatever your distinction, you can become depressed. That means that any person you know is fair game. So remember, you are not alone.

Depression is a word used in everyday language to describe a number of feelings, including sadness, frustration, disappointment and sometimes lethargy. However, in clinical practice, the term "Depression" or "Major

Depression" differs from these everyday 'down' periods in three main ways:

- Major Depression is more intense
- Major Depression lasts longer (two weeks or more)
- Major Depression significantly interferes with effective day-to-day functioning

In this handout, the word depression is referring to Major Depression or a clinical depression.

Depression as a Syndrome

A syndrome is a collection of events, behaviours, or feelings that often go together. The depression syndrome is a collection of feelings and behaviours that have been found to characterise depressed people as a group. You may find that you experience all or some of these feelings and behaviours. There are many individual differences to the number of symptoms and the extent to which different symptoms are experienced. These symptoms are described in this next section.

Mood

Depression is considered to be a disorder of mood. Individuals who are depressed, describe low mood that has persisted for longer than two weeks. In mild forms of depression, individuals may not feel bad all day but still describe a dismal outlook and a sense of gloom. Their mood may lift with a positive experience, but fall again with even a minor disappointment. In severe depression, a low mood could persist throughout the day, failing to lift even when pleasant things occur. The low mood may fluctuate during the day – it may be worse in the morning and relatively better in the afternoon. This is called ‘diurnal variation,’ which often accompanies a more severe type of depression.

In addition to sadness, another mood common to depression is anxiety.

Thinking

Individuals who are depressed think in certain ways, and this thinking is an essential feature of depression. It is as much a key symptom of depression as mood or physical symptoms. Those who are depressed tend to see themselves in a negative light. They dwell on how bad they feel, how the world is full of difficulties, how hopeless the future seems and how things might never get better. People who are depressed often have a sense of guilt, blaming themselves for everything, including the fact they think negatively. Often their self-esteem and self- confidence become very low.

Physical

Some people experience physical symptoms of depression.

- Sleep patterns could change. Some people have difficulty falling asleep, or have interrupted sleep, others sleep more and have difficulty staying awake
- Appetite may decline and weight loss occurs, while others eat more than usual and thus gain weight
- Sexual interest may decline
- Energy levels may fall, as does motivation to carry out everyday activities. Depressed individuals may stop doing the things they used to enjoy because they feel unmotivated or lethargic

Interacting with Other People

Many depressed people express concern about their personal relationships. They may become unhappy and dissatisfied with their family, and other close, relationships. They may feel shy and anxious when they are with other people, especially in a group. They may feel lonely and isolated, yet at the same time, are unwilling or unable to reach out to others, even when they have the opportunities for doing so.

It is important to understand that depression is not caused by one thing, but probably by a combination of factors interacting with one another. These factors can be grouped into two broad categories – biology and psychology. Many

biological and psychological factors interact in depression, although precisely which specific factors interact may differ from person to person.

Biological Factors

The biological factors that might have some effect on depression include: genes, hormones, and brain chemicals.

Genetic Factors

Depression often runs in families, which suggests that individuals may inherit genes that make them vulnerable to developing depression. However, one may inherit an increased vulnerability to the illness, but not necessarily the illness itself. Although many people may inherit the vulnerability, a great many of them may never suffer a depressive illness.

Hormones

Research has found that there are some hormonal changes that occur in depression. The brain goes through some changes before and during a depressive episode, and certain parts of the brain are affected. This might result in an over- or under-production of some hormones, which may account for some of the symptoms of depression. Medication treatment can be effective in treating these conditions.

Brain Chemicals (Neurotransmitters)

Nerve cells in the brain communicate to each other by specific chemical substances called neurotransmitters. It is believed that during depression, there is reduced activity of one or more of these neurotransmitter systems, and this disturbs certain areas of the brain that regulate functions such as sleep, appetite, sexual drive, and perhaps mood. The reduced level of neurotransmitters results in reduced communication between the nerve cells and accounts for the typical symptoms of depression. Many antidepressant drugs increase the neurotransmitters in the brain.

Psychological Factors

Thinking

Many thinking patterns are associated with depression. These thinking patterns include:

- overstressing the negative
- taking the responsibility for bad events but not for good events
- having inflexible rules about how one should behave
- thinking that you know what others are thinking and that they are thinking badly of you

Loss

Sometimes people experience events where loss occurs, and this can bring on depression. The experience of loss may include the loss of a loved one through bereavement or separation, loss of a job,loss of a friendship, loss of a promotion, loss of face, loss of support, etc.

Sense of Failure

Some people may stake their happiness on achieving particular goals, such as getting ‘As’ on their exams, getting a particular job,earning a certain amount of profit from a business venture, or finding a life partner. If for some reason they are not able to achieve those goals,they might believe that they have failed somehow, and it is this sense of failure that can sometimes bring on, or increase, depression.

Stress

An accumulation of stressful life events may also bring on depression. Stressful events include situations such as unemployment, financial worries, serious difficulties with spouses, parents or children, physical illness, and major changes in life circumstances.

Conclusion

While we cannot do much about the genes we have inherited, there are a number of things we can do to overcome depression, or to prevent us from becoming depressed. Your doctor may have suggested medication, especially in a severe depression. While taking medication can be of assistance in overcoming depression, psychological treatments are also available. Ask your doctor or mental health practitioner for more details.

Psychotherapy for Depression

Depression can be treated with medical treatments such as antidepressant medication or electroconvulsive therapy, and psychotherapy. Please see your medical doctor or psychiatrist for more information about medical treatments as this will not be discussed in this handout.

We’re now going to talk briefly about two psychological therapies that have been proven to be effective most of the time. You might have come across words such as “best practice” “evidence-based practice,” “evidence-based treatment” or “evidence-supported therapy.” These words refer to a particular type of treatment or therapy that has been evaluated and has proven to be effective. For the treatment of depression,the evidence-supported therapies include cognitive therapy and behaviour therapy.

Cognitive Therapy

The aim of cognitive therapy is to help individuals realise that they can influence their mood by identifying and changing their thoughts and beliefs. When people are depressed, they often think very negative thoughts about themselves, their lives, and their future. This further worsens their mood. Cognitive therapy focuses on discovering and challenging unhelpful assumptions and beliefs, and developing helpful and balanced thoughts. Cognitive therapy is also structured, time-limited, and focused on the ‘here-and-now.‘ This form of treatment for depression has been proven to be effective when individuals are able to acquire the skills that are being taught in therapy.

Behaviour Therapy

Depressed people tend to feel lethargic and unmotivated. They often stay at home and avoid going out and interacting with people. As such, they may miss out on opportunities that help lift their mood. Behaviour therapy aims to identify and change aspects of behaviour that may perpetuate or worsen the depression. Some behavioural strategies include: goal setting, activity scheduling, social skills training, and structured problem solving.

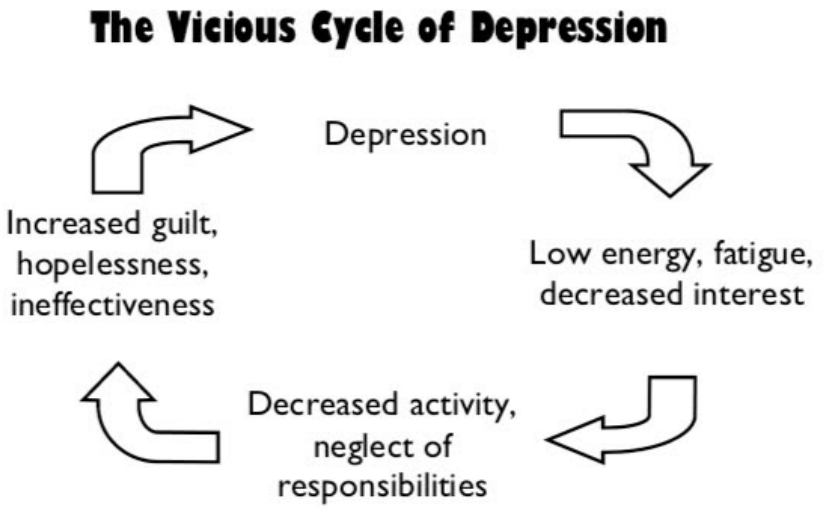
In Summary

These two therapies have been shown to be effective most of the time. Often, a combination of these therapies are offered for people who experience depression. This information package focuses on providing information on the cognitive and behavioural aspects of depression, which includes suggested strategies for how you could better manage your mood.

The Vicious Cycle of Depression

The symptoms of depression can bring about some drastic changes in a depressed person’s life, daily routines, and their behaviour. Often it is these changes that makes the depression worse and prevents the depressed person from getting better. For example, a lack of motivation or a lack of energy can result in a depressed person cutting back on their activities, neglecting their daily tasks and responsibilities, and leaving decision-making to others. Have you noticed these changes in yourself when you are depressed? You may find that you have become less and less active, don’t go out much anymore, avoid hanging out with friends, and stopped engaging in your favourite activity. When this happens, you have become locked in the vicious cycle of depression, which might look like this:

The Vicious Cycle of Depression



When your activity level decreases, you may become even less motivated and more lethargic. When you stop doing the things you used to love, you miss out on experiencing pleasant feelings and positive experiences. Your depression could get worse.

Similarly, when one begins neglecting a few tasks and responsibilities at work or at home, the list may begin to pile up. As such, when a depressed person thinks about the things they have to do, they may feel overwhelmed by the pile of things they have put off doing. This may result in them feeling guilty or thinking that they are ineffective or even, a failure. This will also worsen the depression.

Reversing the Vicious Cycle of Depression

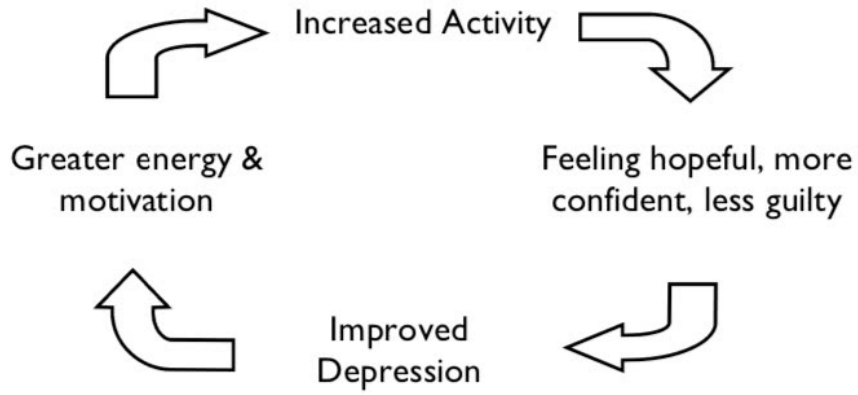
One of the ways of breaking the vicious cycle of is through the use of medication. Medication such as antidepressants can help change your energy level and improve sleep. Another way is to simply increase your activity level, especially in pleasurable activities and tackling your list of tasks and responsibilities, but doing it in a realistic and achievable way, so that you set yourself up to succeed.

Becoming more active has a number of advantages:

- Activity helps you to feel better
- Activity helps you to feel less tired
- Activity can help you think more clearly

When the depression cycle is broken, it will look like this:

Reversing The Vicious Cycle of Depression



Here's a list of possible fun things to do. You can add your own to this list.

1. Soaking in the bathtub
2. Collecting things (coins, shells, etc.)
3. Going for a day trip
4. Going to see a comedy at the movies
5. Going to the beach
6. Playing squash/tennis/badminton
7. Having a barbecue at the park
8. Going for a walk, jog, or hike
9. Listening to uplifting music
10. Gardening

Try some of them out and evaluate how you feel before and after the activity. Chances are, you'll find that you'll feel a little better. The important thing is to persist – keeping your activity levels up is the first step to breaking out of that vicious cycle! The second step is to look at how thinking patterns contribute to the vicious cycle of depression. The "Improving how you feel" information sheet starts to look more closely at this.

Grief and Bereavement

Uncomplicated Grief

Grief and loss are part of life and is experienced by most of us at some point in life. People deal with grief in many different ways, and not necessarily going through a predictable group of 'stages,' although some do. How people grieve can depend on the circumstances of the loss (e.g., sudden death, long illness, death of a young person) as well as past experiences of loss. There is no time limit on grief - some people get back to their usual routine fairly quickly, others take longer. Some people prefer time alone to grieve, others crave the support and company of others. Below are just some of the range of experiences which can be part of uncomplicated grief:

- Symptoms of depression or anxiety, such as poor sleep, lowered appetite, low mood, feeling of anxiety - for some people the anxiety will be more obvious, for others the depression.

- A sense of the loss not quite being ‘real’ at first, or refusal to believe it has occurred
- Feeling disconnected from others, sense of numbness
- Guilt about not initially feeling pain about the loss
- Worries about not grieving ‘normally’ or ‘correctly’
- Mood swings and tearfulness
- Guilt about interactions with the person who has died (e.g. I should have spent more time with her or I wish we didn’t have that argument)
- Waves of sadness or anger which can be overwhelming and sometimes suddenly triggered by reminders
- Seeking reminders of the person who has died, e.g. being in their home or with their belongings, or perhaps at times even feeling you see or hear the deceased person
- Guilt about gradually getting back to ‘normal’ life and at times not ‘remembering’ to feel sad

Coping with Uncomplicated Grief

Most people going through the pain described above will eventually adjust to the loss and return to normal life, although of course carrying some sadness about the loss. Most people do not require medication or counselling to manage uncomplicated grief, and should simply be supported to go through their individual grief process. It is important to maintain a healthy diet and some physical activity during this time. Some people may find it helpful to engage in counselling or to attend groups with others who have suffered a recent loss.

Complicated Grief

Complicated grief is a general term for describing when people adjust poorly to a loss. This is very difficult to define, as there is no standard which limits what is normal or healthy grief.

Below are some warning signs which may suggest that a person is not coping well with grief and may be at a greater risk of the grieving process taking longer to resolve or being more difficult:

- Pushing away painful feelings or avoiding the grieving process entirely
- Excessive avoidance of talking about or reminders of the person who has died
- Refusal to attend the funeral
- Using distracting tasks to avoid experiencing grief, including tasks associated with planning the funeral
- Abuse of alcohol or other drugs (including prescription)
- Increased physical complaints or illness
- Intense mood swings or isolation which do not resolve within 1-2 months of the loss
- Ongoing neglect of self-care and responsibilities

Again, it is important to emphasise that there are no ‘rules for grieving’ and that many of the items above may occur as part of uncomplicated grief. However, people who are coping very poorly one month after a loss may continue to cope poorly 1-2 years later, so if these warning signs are present then it is often worthwhile seeking some help early on, to increase the chances of adjusting in the long term.

Coping with Complicated Grief

Psychological therapy can support people to safely explore feelings of grief and connect with painful feelings and memories, paving the way for resolution. Therapy may also support people to use strategies such as relaxation, engaging in positive activities, and challenging negative thoughts, in order to combat the associated symptoms of anxiety and depression. Antidepressant medication may also be used to alleviate depression associated with grief, and this can be useful in conjunction with psychological strategies. Tranquilizing medications can interfere with the natural grieving process. Although early help is recommended, health professionals are able to support people to work through complicated grief even years after the loss.

What is Anger?

Normal Anger

Anger is a normal human emotion. Everyone feels annoyed, frustrated, irritated, or even very angry from time to time. Anger can be expressed by shouting, yelling, or swearing, but in extreme cases it can escalate into physical aggression towards objects (eg. smashing things) or people (self or others). In some cases, anger might look much more subtle, more of a brooding, silent anger, or withdrawal. In a controlled manner, some anger can be helpful, motivating us to make positive changes or take constructive action about something we feel is important. But when anger is very intense, or very frequent, then it can be harmful in many ways.

What Causes Anger?

Anger is often connected to some type of frustration— either things didn’t turn out the way you planned, you didn’t get something you wanted, or other people don’t act the way you would like. Often poor communication and misunderstandings can trigger angry situations. Anger usually goes hand-in-hand with other feelings too, such as sadness, shame, hurt, guilt, or fear. Many times people find it hard to express these feelings, so just the anger comes out. Perhaps the anger is triggered by a particular situation, such as being caught in a traffic jam, or being treated rudely by someone else, or banging your thumb with a hammer while trying to hang a picture-hook. Other times there is no obvious trigger—some people are more prone to anger than others. Sometimes men and women handle anger differently, but not always.

Problems Associated With Anger

Uncontrolled anger can cause problems in a wide range of areas of your life. It may cause conflicts with family, friends, or colleagues, and in extreme situations it can lead to problems with the law. But some of the other problem effects of anger may be harder to spot. Often people who have a problem with anger feel guilty or disappointed with their behaviour, or suffer from low self-esteem, anxiety, or depression. There are also physical side-effects of extreme or frequent anger, such as high blood pressure, and heart disease. Some studies suggest that angry people tend to drink more alcohol, which is associated with a wide range of health problems.

Do I Have a Problem with Anger?

Perhaps you have already identified that anger is a problem for you, or someone else has mentioned it to you. But if you are not sure whether anger is a problem for you, consider the following questions:

- Do you feel angry, irritated, or tense a lot of the time?
- Do you seem to get angry more easily or more often than others around you?
- Do you use alcohol or drugs to manage your anger?
- Do you sometimes become so angry that you break things, damage property, or become violent?
- Does it sometimes feel like your anger gets out of proportion to the situation that set you off?
- Is your anger leading to problems with relationships, such as with family, friends, or at work?
- Have you noticed that others close to you sometimes feel intimidated or frightened of you?
- Have others (family, friends, colleagues, health professionals) mentioned that anger might be a problem for you?
- Do you find that it takes you a long time to ‘cool off’ after you have become angry or irritated?
- Have you ended up in trouble with the law as a result of your anger, for example getting into fights?
- Do you find yourself worrying a lot about your anger, perhaps feeling anxious or depressed about it at times?
- Do you tend to take your frustration out on loved ones or people less powerful than you, rather than dealing with the situation that triggered your anger?

If you answered ‘yes’ to any of these questions, it may be that anger is a problem for you. It may be that addressing your anger can allow you to live a much more positive and rewarding life.

How Can I Manage Anger Better?

You may have heard about ‘anger management’ and wondered what it involves. Anger management can be addressed in groups or through individual therapy, and there are also a lot of self-help resources available. Anger management is not

just about counting to ten before you respond (although that is often a good idea). It is about helping you to better understand why you get angry, what sets it off and what are the early warning signs, and about learning a variety of strategies for managing those feelings more constructively. You may wish to read through our ‘Anger Coping Strategies’ handout for more information about this.

Anger Coping Strategies

Anger and Problem Anger

Anger is a normal human emotion, and can range from mild irritation to an intense rage or fury. Our handout ’What Is Anger?’ provides more detail about the difference between normal anger and problem anger, and some questions to help you identify whether anger may be a problem for you. This handout includes a number of tips which you may use to help you to cope better with your anger. You may wish to practice some of these on your own, or you may wish to combine them with individual or group therapy for extra support.

Triggers and Early Warning Signs

One of the first steps in managing your anger is to identify what types of situations usually trigger your anger. Make a list of the things which usually set you off, for example:

- being cut off in traffic
- running late for an appointment
- other people running late
- your son/daughter leaving their schoolbag in the hall
- your partner not putting away the dishes
- a colleague falling behind on a project

Some of these situations you may be able to avoid, such as planning ahead to avoid running late. Other situations are less in your control, such as being cut off in traffic, but what you can control is your response. Once you have finished listing your common trigger situations, make a separate list of the warning signs for your anger. What is it that usually happens in your body when you get angry? Becoming aware of your body’s alarm bells helps you to spot anger early on, which gives you a better chance of putting other coping strategies into practice. Some common warnings are:

- tightness in chest
- feeling hot or flushed, sweating
- grinding teeth
- tense muscles or clenched fists
- pounding or racing heart
- biting your nails

Why Am I Angry?

When you notice these warning signs, stop and ask yourself what it is that is making you angry. Often there will be something going on that is quite reasonable to feel angry about, so allow yourself to acknowledge this. But it is also important to be clear about the cause of our anger so that we don't respond in a way that is out of proportion (eg. staying angry all day about someone else using up the last of the milk) or take out the anger on the wrong person (eg. getting angry at family members when it is your boss you are angry with).

Taking Out The Heat

When you notice yourself becoming angry, there are a number of techniques which you can use to 'take the heat out' of your anger. These include:

Time Out: This simply means removing yourself from the situation for a period of time, to give yourself a chance to 'cool down' and think things through before you act. For example, when you notice yourself becoming angry during an argument with your partner, say "I need to take time out, let's talk about this calmly when I get back" and then go for a walk.

Distraction: If you cannot change the situation, it can help to distract yourself from whatever is making you angry by counting to ten, listening to music, calling a friend to chat about something else, or doing housework. For example, if you are stuck in traffic and getting angry, put on the radio and try to find a song you like, or count the number of times the chorus is sung.

Silly Humour: While it is not always possible to just 'laugh your problems away,' you can often use humour to help you to take a step back from your anger. For example, if you are angry with a colleague and refer to them as 'a stupid clown,' think about what this means literally. Imagine or draw them dressed in a clown suit, with big shoes and a red nose. If you picture this image every time they do something which bothers you, it will be much easier to keep things in perspective.

Relaxation: Just as our bodies are strongly affected by our emotions, we can also influence our emotional state with our physical state. Relaxation techniques, such as taking slow deep breaths or progressively tensing and relaxing each of your muscle groups, can help to reduce anger.

Self-Talk and Helpful Thinking

How you are thinking affects how you are feeling, so focussing on negative thoughts such as "this is so unfair" will maintain the angry feeling. Make a list of more balanced statements you can say to yourself before, during and after difficult situations. For example: Before: I know I can handle this, I have strategies to keep my anger under control and can take time out if I need to. During: Remember to keep breathing and stay relaxed. There is no need to take this personally. I can manage this. After: I handled that well. Even though I felt angry I didn't raise my voice too much and I think I got a better result

Assertiveness and Practice

Another key strategy in managing anger is to learn to be assertive. Assertiveness means expressing your point of view in a clear way, without becoming aggressive. You may wish to read other handouts about this topic. Finally, because anger is often an automatic response, all of these techniques require a lot of practice.

Facts About Sleep

The Nature of Sleep

Sleep is such an important part of our lives, yet many of us don't pay much attention to it. It is usually not until we have problems with sleep that we notice it and start to try to understand the nature of sleep. As well as humans, other mammals, reptiles and birds all sleep, while fish, amphibians and insects do not (although they may rest). Some animals sleep in many short bursts, while others, like humans, prefer to sleep in one long block.

We all know what sleep looks like - we recognise a sleeping person because they have their eyes closed, will usually be lying down, breathing in a slow rhythm, with relaxed muscles and generally keeping still, although they may rearrange their bodies every so often. Being asleep is being unconscious to most things happening around you, but is different from a coma or passing out because sleeping people can be woken up, by loud noises or bright lights or touch.

Stages of Sleep

Research tells us that there are two types of sleep:

- **REM rapid-eye-movement sleep:** this type of sleep occurs for about 25% of the night, and is characterised by electrical activation of the brain, very relaxed muscles and body becoming immobile, and rapid eye movements as the eyes dart back and forth under closed eyelids. REM sleep provides energy to the brain and body and supports daytime performance. Dreams often occur during REM sleep, although they can occur at any stage.
- **NREM non-rapid-eye-movement sleep:** this type of sleep occurs during the other 75% of the time, and can be further broken down into 4 stages:
    - Stage 1: this stage is light sleep, between being awake and falling asleep
    - Stage 2: this stage is the onset of sleep, when the person begins to become disengaged from their surroundings. Body temperature drops and breathing and heart rate become regular.
    - Stages 3 & 4: These stages are the deepest and most restorative sleep, known as 'delta sleep' - Stage 3 is a transition into Stage 4, or 'true delta.' During these stages, blood pressure drops, breathing becomes slower, muscles are relaxed and receiving more blood supply, tissue growth and repair occurs, and hormones are released (including growth hormone, which is why growing teenagers need to sleep more).

Role and Function of Sleep

Sleep is essential to humans, just like air, water and food. When necessary, people can cope without sleep for periods of time, but the longer we are awake the stronger the urge to sleep becomes. The exact role and function of sleep has been a topic of debate for researchers, but most agree that sleep serves a restorative purpose, both psychologically and physiologically. It is thought that delta sleep (stages 3 & 4) is most involved with restoring the body and physical energy, while REM sleep is most important for restoring mental function such as memory and concentration. Sleep is important for general physical health, restoring energy, repairing injuries or illness, growth, psychological well-being and mood, concentration, memory, work performance, and getting along with others.

Effects of Lack of Sleep

People vary in terms of how much sleep they need - while the average sleep duration for adults is 7-8.5 hours per night, some people function well with 4-5 hours and others require 9-10 hours. Whatever your individual needs, lack of sleep or poor sleep quality can have effects including:

- Poor attention, concentration and memory
- Irritability and other mood disturbances
- Impaired judgement and reaction time
- Poor physical coordination (dangerous for driving)

The seriousness of these effects depends on how bad the sleep deprivation is (e.g. less sleep vs. no sleep; one night's poor sleep vs. chronic problems) and the tasks and responsibilities of the day. If you have ongoing problems with sleep, it is important to seek help.

How Well do Good Sleepers Sleep?

Good sleepers usually take less than 30 minutes to fall asleep at the beginning of the night and will wake up once or twice during the night. In other words, it is unrealistic to expect to fall asleep immediately on getting into bed or to never wake up at all during the night. Even the best sleepers in the world don't achieve this! Also, everybody, even the best sleepers, will have a night now and then when it takes them a long time to get to sleep. This is often triggered by a stressful event and will usually pass after a night or two. Similarly, everybody will have a night now and then when they find it difficult to get back to sleep after waking in the middle of the night.

What is Insomnia?

Primary insomnia is more than just transient sleep difficulties, it is persistent problems with sleep, lasting for more than one month, and may include:

- Difficulty falling asleep - also known as onset insomnia
- Waking up on and off during the night - also known as middle insomnia
- Waking up very early and not returning to sleep
- Unsatisfactory sleep quality

These are different to other sleep problems, such as excessive daytime sleepiness, effects of shift work and jet-lag, or nightmares and sleepwalking. Surprisingly, insomnia is the most common psychological health problem - it has been estimated that 15-30% of the adult population suffers from insomnia, with twice as many women as men suffering. Insomnia becomes more common as we get older, but it affects a range of ages. Most of us experience problems with sleep at some point in our lives, generally when under stress, but you should consider seeking help for what we call chronic insomnia. This is when your problems with sleep have lasted for more than one month or if you cannot get a good night's sleep without sleeping pills.

Insomnia is often associated with other psychological disorders such as depression, generalised anxiety disorder, and post-traumatic stress disorder. People may underplay the importance of insomnia by regarding it as just a symptoms of another issue, when in fact it may require treatment in its own right.

Impact of Insomnia

Although insomnia is common, it is certainly not a minor issue. Ongoing sleep problems can impact on your functioning during the daytime as well as night. People who do not sleep well may experience:

- Low mood or easily irritable
- Poor memory & concentration
- Trouble staying alert
- Worry about not sleeping
- Poor work performance
- Conflict in relationships
- Less quality of life

If you are experiencing some of these consequences of poor sleep, then you may need to seek help.

Causes of Insomnia

There is a wide range of factors which may contribute to insomnia. Just some of these factors are:

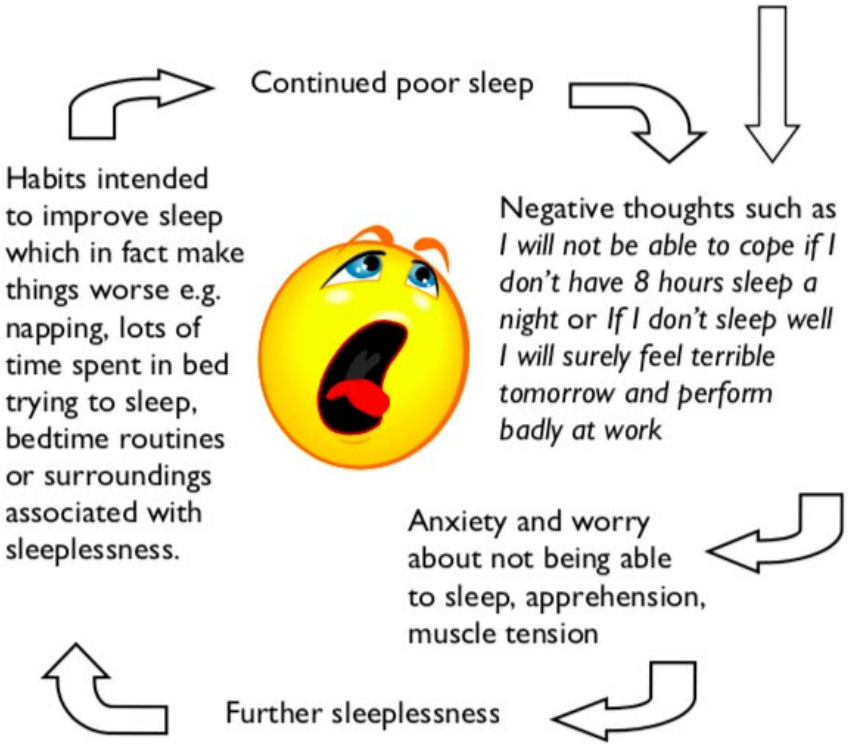
- Respiratory problems such as sleep apnea
- Restless legs or twitching legs during sleep
- Pain
- Side effects of medications
- Alcohol - leads to more fitful, less refreshing sleep
- Caffeine and nicotine
- Severe anxiety or depression
- Stressful life events
- Habits such as daytime napping
- Dependence on sleeping medication

There may be one set of factors associated with the initial causes of insomnia, and quite another that keeps insomnia going and makes into a chronic problem.

The Vicious Cycle of Insomnia

People can become stuck in a vicious cycle of insomnia, developing habits or beliefs which help keep the sleep problems going beyond the original cause. For example:

Initial poor sleep due to stress, pain, or other reason



Management of Insomnia

There are many medications which are used to treat insomnia, yet these are usually only effective in the short- term. For long-term management of sleep problems, you may need to consider strategies such as sleep hygiene, cognitive therapy, and reducing your stress levels.

Sleep Hygiene

What is Sleep Hygiene?

'Sleep hygiene' is the term used to describe good sleep habits. Considerable research has gone into developing a set of guidelines and tips which are designed to enhance good sleeping, and there is much evidence to suggest that these strategies can provide long-term solutions to sleep difficulties.

There are many medications which are used to treat insomnia, but these tend to be only effective in the short-term. Ongoing use of sleeping pills may lead to dependence and interfere with developing good sleep habits independent of medication, thereby prolonging sleep difficulties. Talk to your health professional

about what is right for you, but we recommend good sleep hygiene as an important part of treating insomnia, either with other strategies such as medication or cognitive therapy or alone.

Sleep Hygiene Tips

1. Get regular. One of the best ways to train your body to sleep well is to go to bed and get up at more or less the same time every day, even on weekends and days off! This regular rhythm will make you feel better and will give your body something to work from.
2. Sleep when sleepy. Only try to sleep when you actually feel tired or sleepy, rather than spending too much time awake in bed.
3. Get up & try again. If you haven't been able to get to sleep after about 20 minutes or more, get up and do something calming or boring until you feel sleepy, then return to bed and try again. Sit quietly on the couch with the lights off (bright light will tell your brain that it is time to wake up., or read something boring like the phone book. Avoid doing anything that is too stimulating or interesting, as this will wake you up even more.
4. Avoid caffeine & nicotine. It is best to avoid consuming any caffeine (in coffee, tea, cola drinks, chocolate, and some medications. or nicotine (cigarettes. for at least 4-6 hours before going to bed. These substances act as stimulants and interfere with the ability to fall asleep
5. Avoid alcohol. It is also best to avoid alcohol for at least 4-6 hours before going to bed. Many people believe that alcohol is relaxing and helps them to get to sleep at first, but it actually interrupts the quality of sleep.
6. Bed is for sleeping. Try not to use your bed for anything other than sleeping and sex, so that your body comes to associate bed with sleep. If you use bed as a place to watch TV, eat, read, work on your laptop, pay bills, and other things, your body will not learn this connection.
7. No naps. It is best to avoid taking naps during the day, to make sure that you are tired at bedtime. If you can't make it through the day without a nap, make sure it is for less than an hour and before 3pm.
8. Sleep rituals. You can develop your own rituals of things to remind your body that it is time to sleep - some people find it useful to do relaxing stretches or breathing exercises for 15 minutes before bed each night, or sit calmly with a cup of caffeine-free tea.
9. Bath time. Having a hot bath 1-2 hours before bedtime can be useful, as it will raise your body temperature, causing you to feel sleepy as your body temperature drops again. Research shows that sleepiness is associated with a drop in body temperature.
10. No clock-watching. Many people who struggle with sleep tend to watch the clock too much. Frequently checking the clock during the night can wake you up (especially if you turn on the light to read the time. and reinforces negative thoughts such as "Oh no, look how late it is, I'll never get to sleep" or "it's so early, I have only slept for 5 hours, this is terrible. "
11. Use a sleep diary. This worksheet can be a useful way of making sure you have the right facts about your sleep, rather than making assumptions. Because a diary involves watching the clock (see point 10. it is a good idea to

only use it for two weeks to get an idea of what is going and then perhaps two months down the track to see how you are progressing.

12. Exercise. Regular exercise is a good idea to help with good sleep, but try not to do strenuous exercise in the 4 hours before bedtime. Morning walks are a great way to start the day feeling refreshed!
13. Eat right. A healthy, balanced diet will help you to sleep well, but timing is important. Some people find that a very empty stomach at bedtime is distracting, so it can be useful to have a light snack, but a heavy meal soon before bed can also interrupt sleep. Some people recommend a warm glass of milk, which contains tryptophan, which acts as a natural sleep inducer.
14. The right space. It is very important that your bed and bedroom are quiet and comfortable for sleeping. A cooler room with enough blankets to stay warm is best, and make sure you have curtains or an eye mask to block out early morning light and earplugs if there is noise outside your room.
15. Keep daytime routine the same. Even if you have a bad night sleep and are tired it is important that you try to keep your daytime activities the same as you had planned. That is, don't avoid activities because you feel tired. This can reinforce the insomnia.

The following resources are from: https://www.blackdoginstitute.org.au

Post-Traumatic Stress Disorder

What is post-traumatic stress disorder?

Post-traumatic stress disorder (PTSD) is a group of stress reactions that can develop after we witness a traumatic event, such as death, serious injury or sexual violence to ourselves or to others. PTSD can happen after we've been through one traumatic event, or after repeated exposure to trauma. Sometimes, PTSD can develop after hearing details about devastating and traumatic events many times, like the experience of some emergency workers. It's important to seek help to manage PTSD. There are effective treatments for PTSD, and you can feel better.

- We have strong reactions to traumatic events
- At times in our lives we may encounter traumatic experiences that threaten the life or safety of ourselves or others. Most of us will have a very strong reaction to these extreme and distressing events.
- Feelings of fear, sadness, anger and grief are common after a traumatic event. This is part of our natural human response to danger.
- Often, these feelings settle in time
- Over time, with support from family and friends, we start to make sense of what's happened. These feelings usually fade and we recover. However, sometimes witnessing a distressing event can lead to severe feelings of fear

and anguish that stay with us for a long time. These feelings start to interfere with our lives and stop us doing what we used to do. When this happens, we need help to get through it.

Causes of post-traumatic stress disorder

Traumatic events threaten the life and safety of ourselves or others. They can involve actual or threatened death, serious injury, or sexual abuse. Experiencing or hearing about a traumatic event or multiple traumatic events can lead to post-traumatic stress disorder (PTSD). Experts are still learning more about why some of us develop post-traumatic stress disorder (PTSD).

Why do some people get PTSD?

- Anyone can develop PTSD after a traumatic event. Factors that may contribute to developing PTSD, include:
  - past life experiences such as childhood trauma or sexual abuse
  - experiencing trauma over a long period of time
  - having a job that exposes you to repeated trauma such as police, ambulance officer, firefighter or military
  - genetic factors (family history of mental health issues)
  - previous mental health issues like anxiety and depression
  - brain chemistry
  - your body’s stress response
  - your support network (colleagues, family and friends and access to professional help).

Treatments for post-traumatic stress disorder

It's important to get treatment for PTSD. Treatments include psychological therapies, physical treatments (medication), and exercise, mindfulness and self-help strategies. Talk to your GP or mental health professional about the best treatment for you. Often a combination of treatments works best.

Important things to know about getting treatments for PTSD:

- There are good resources and professionals to help you with PTSD.
- You need a thorough check from a health professional before treatment is prescribed.
- Psychological therapies and medication are the most established ways to treat PTSD.
- New evidence shows that exercise and mindfulness are very useful for PTSD. They can be used together with physical and psychological treatments.
- Exercise helps other conditions that can occur with PTSD, like depression, anxiety, sleep problems, cardiovascular disease and obesity.

You can get better!!

Many people who have had PTSD have been able to seek help, return to work, and live active, fulfilling lives.

There are three broad categories of treatment for PTSD:

- psychological treatments (talking therapies)
- physical treatments (medications)
- exercise, mindfulness and self-help.

Often, a combination of treatments works best. Find the best treatment for you.

Everybody has a different experience. Your symptoms, any co-conditions (like anxiety or depression), and your personal preferences will influence which treatments are best for you.Talk to your GP or mental health professional about the best treatment for you. Sometimes, a team will be involved in your care. It’s still important that one professional coordinates and has overall responsibility for your treatment.

Suicide & Self-Harm

Warning signs for suicide and self-harm

By learning the warning signs, anyone can help to prevent suicide.

When people are thinking about ending their life, there are sometimes signs you may notice. They may be feeling distraught and can’t see a way out of their problems. People show signs in different ways, so it is best to be aware of both verbal and non-verbal cues. If you're worried that someone is thinking about taking their own life, it's important to talk to them. People usually don't want to end their life. They want the pain to stop. There is a sense of isolation and hopelessness if they don't think they have the support to get through it. Talking to people can reduce stigma and encourage them to seek help, factors proven to lower risks of suicidal behaviours.

Don't ignore threats of suicide

Many people who take their own life give some kind of warning beforehand. When people express suicidal thoughts, these need to be taken seriously. Even if you're not sure, it's better to help straight away than to be unsure and not act at all. Talk to the person and get professional advice from others.

Be aware of subtle changes in behaviour

If someone is acting out of the ordinary, this is no cause for alarm but a sign to pay attention to their behaviours.

These signs might include:

- sleep changes (too much sleep or too little)
- withdrawing from family and friends
- loss of interest in things
- changes in eating
- irritability, being moody or easily upset
- self-harming (e.g. cutting)
- putting affairs in order, giving things away, saying goodbyes, writing suicide notes or goodbye letters

- risky behaviour (e.g. consuming excessive alcohol or other drug use)
- decreased academic or work performance
- mentioning or joking about suicide, death or dying

Take note of feelings and how they are expressed

Some people choose to talk about how they are feeling, however this does not apply to everyone. They might be feeling hopeless, depressed, angry and irritable, distressed, worthless, exhausted, like there's no way out of their problems or no reason for living at all.

They might say things which suggest that:

- They see themselves as a burden e.g. "You'd be better off without me."
- They can't see a way out of their situation e.g. "I've had enough." or "I'm over it."
- They're feeling a sense of hopelessness e.g. "There's nothing to live for." or "There's no point."

Get support now

There is help for you and you can stay safe. Make the call for help.

Are you or someone near you in immediate danger?

- Call Emergency Services on 000; or
- Go to a hospital emergency department

Are you having suicidal thoughts and need someone to talk to?

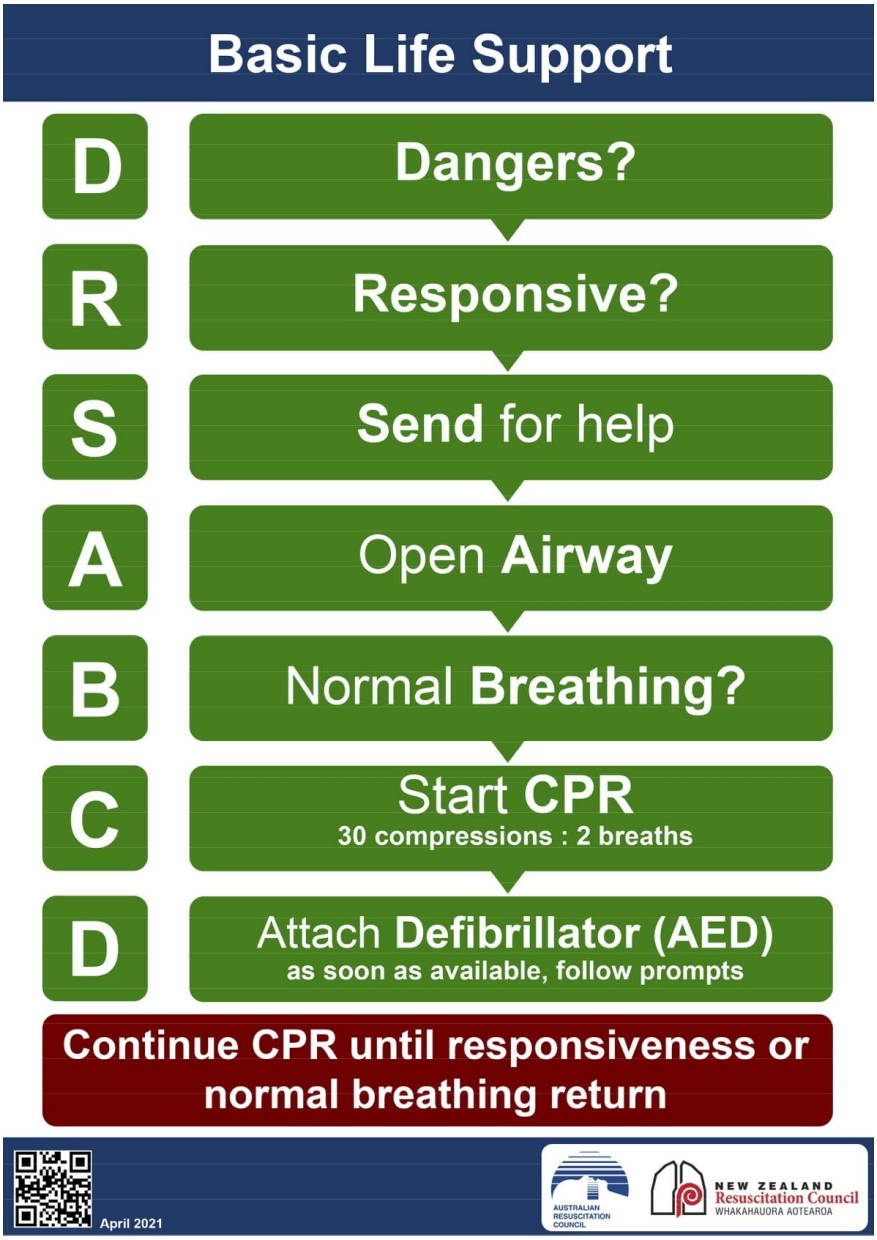
- Call Lifeline on 13 11 14
- Talk to someone like:
  - a GP, counsellor, psychologist or psychiatrist family or friends
  - a school, university or TAFE counsellor
  - a teacher or coach
  - a work colleague
  - a church minister or religious leader

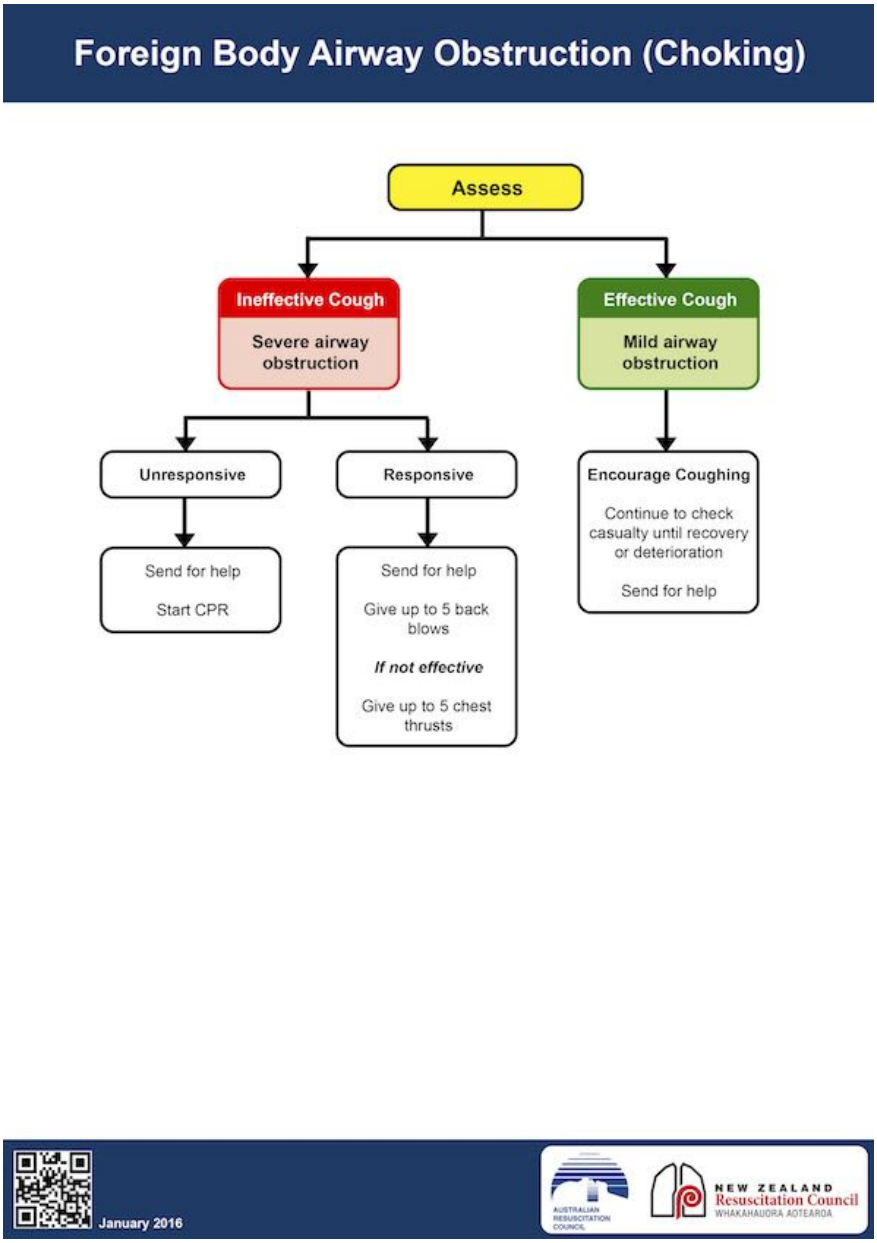
Safety Planning

If you or someone you know is having suicidal thoughts, safety planning can assist during tough times. BeyondNow is a free safety planning app (created by beyondblue). It can help you if you're having suicidal thoughts and distress. Please note: you should work with a health professional or support person to create your plan. BeyondNow is not the only form of support you should receive.

Resources and links

Resources and links have been compiled with others at the top of this handbook


Basic Life Support
D
Dangers?
R
Responsive?
S
Send for help
A
Open Airway
B
Normal Breathing?
C
Start CPR
30 compressions : 2 breaths
D
Attach Defibrillator (AED)
as soon as available, follow prompts
Continue CPR until responsiveness or normal breathing return
April 2021
AUSTRALIAN RESUSCITATION COUNCIL
NEW ZEALAND Resuscitation Council WHAKAHAUORA AOTEAROA


Foreign Body Airway Obstruction (Choking)
Assess
Ineffective Cough
Severe airway obstruction
Effective Cough
Mild airway obstruction
Unresponsive
Responsive
Encourage Coughing
Continue to check casualty until recovery or deterioration
Send for help
Send for help
Start CPR
Send for help
Give up to 5 back blows
If not effective
Give up to 5 chest thrusts
January 2016
AUSTRALIAN RESUSCITATION COUNCIL
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WHAKAHAUORA AOTEAROA

ANZCOR Guideline 2 - Managing an Emergency

Summary

Who does this guideline apply to?

This guideline applies to all who are in need of immediate care ('person' or 'person in need').

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders or first aid providers, first responders and health professionals.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) recommends that in all emergencies, the rescuer should:

1. Quickly assess the situation.
2. Ensure safety for the rescuer, person in need and bystanders (this may mean moving the person in need).
3. Send for help (call an ambulance).

Individuals who are unresponsive and breathing normally should be positioned into a lateral, side-lying recovery (lateral recumbent) position.

If the person in need is unresponsive and not breathing normally, follow the ANZCOR Basic Life Support Flowchart (Guideline 8).

Where more than one person requires attention, the care of an unconscious person has priority.

Guideline

1 Priorities in an Emergency

Early recognition is a key step in initiating early management of an emergency situation.1-

In all emergencies, the rescuer should:

- quickly assess the situation
- check for danger (assess and manage risks to the rescuer and others. This may mean moving the person in need)
- send for help (call an ambulance).

If the person is unresponsive and not breathing normally, follow the ANZCOR Basic Life Support Flowchart (Guideline 8).

Where more than one person requires attention, **THE CARE OF AN UNCONSCIOUS PERSON HAS PRIORITY.**

2 General principles of management

After ensuring safety for the person in need, rescuer and bystanders and sending for help, the management of the collapsed or injured person involves:

- prevention of further harm or injury
- checking response to verbal and tactile stimuli ('talk and touch')
- care of airway, and breathing
- control of bleeding (Guideline 9.1.1)
- checking for physical (eg. alert jewellery) or electronic alert devices (eg. smartphone application) that may be relevant to assessment or management
- protection from the weather
- other first aid measures depending on the circumstances
- gentle handling
- reassurance
- continued observation.

2.1 Moving a person in need

The condition of a collapsed or injured person may be made worse by movement: increasing pain, injury, blood loss and shock. However, a person lying in a hazardous area, for example on a road or railway, may need to be moved to ensure safety.

A rescuer should move a person when needed to:

- ensure the safety of both rescuer and the person in need
- protect from extreme weather conditions
- enable evacuation from difficult terrain
- enable the care of airway and breathing (e.g. turning the unconscious breathing person onto the side or turning a collapsed person onto their back to perform cardiopulmonary resuscitation)
- enable the control of severe bleeding.

ANZCOR suggests that an unresponsive person who is breathing normally is positioned into a lateral, side-lying recovery (lateral recumbent) position as opposed to leaving them supine.[1,4-5] [2015/2020 CoSTRs, weak recommendation, very-low-certainty evidence]

It is reasonable to roll a face-down unresponsive person onto their back to assess airway and breathing and initiate resuscitation. [Good Practice Statement] Concern for protecting the neck should not hinder the evaluation process or life saving procedures.[6]

Ideally, the most experienced rescuer should take charge and stay with the person in need while another rescuer is sent to seek help. If movement is necessary and help is available, the rescuer in charge should explain clearly and simply the method of movement to the assistants, and to the person in need if they are conscious.

When ready to move the person in need:

- avoid bending or twisting the person's neck and back: a spinal injury (Guideline 9.1.6) can be aggravated by rough handling
- try to have three or more people to assist in the support of the head and neck, the chest, the pelvis and limbs while moving the person. A spine board may be used if available
- a single rescuer may need to drag the person. Either an ankle drag or arm-shoulder drag is acceptable
- make prompt arrangements for transport by ambulance to hospital.

2.2 Specific management of a person in need at a Road Accident

- Approach with caution and make the accident scene as safe as possible.
- Do not touch a vehicle, or attempt to rescue a person from within ten metres[7] of a fallen power line unless an appropriate electrical authority has declared the area safe.
- Use hazard lights, road triangles, or torches to warn oncoming traffic of the accident scene. Bystanders may also be used where it is safe to do so.
- Turn off the ignition of a crashed vehicle and activate the park brake. If unable to activate the park brake, place a chock under a wheel. Be cautious that airbags that have not deployed may activate following a crash.
- Remove a motorbike helmet from a person if it is necessary to manage the airway, assist breathing or control bleeding.
- If an unconscious breathing person can be managed within the vehicle, do not remove them from the vehicle unless there is a threat to life. Clear the airway of foreign material; maintain head tilt and jaw support and continuously reassess the airway and breathing.
- If the person in the vehicle is unconscious and not breathing normally despite opening the airway, remove the person from the vehicle if possible and commence CPR immediately following the ANZCOR Basic Life Support Flowchart (Guideline 8).

2.3 Specific Management of Electric Shock

- When power lines are in contact with a vehicle or a person, do not approach until the situation is declared safe by authorities. The rescuer should ensure that all bystanders remain at least ten metres clear of any electrified material;

examples being a car body, cable, pool of water.[7] Metal and water conduct electricity and may be extremely hazardous.

- In a **domestic** or similar situation it is essential to promptly separate the person in need from the electricity supply. Turn off the supply of electricity and, if possible, unplug the appliance from the power outlet. Until the power is off, avoid direct skin contact with the person or any conducting material.
- If the person is unresponsive and not breathing normally, follow the ANZCOR Basic Life Support Flowchart (Guideline 8).
- Other injuries may require treatment. Burns are common and should be managed following ANZCOR Guideline 9.1.3.
- Promptly refer all who have suffered an electric shock for medical assessment.
- Assess the person who has been struck by lightning: if unresponsive and not breathing normally, follow the ANZCOR Basic Life Support Flowchart (Guideline 8).

References

1. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science With Treatment Recommendations. Resuscitation 2020;156:A240-A82.
2. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Circulation 2020;142:S41-S91.
3. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2020;156:A35-A79.
4. Zideman DA, Singletary EM, De Buck EDJ, et al. Part 9: First aid 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e225-e61.
5. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science With Treatment Recommendations. Circulation 2020;142:S284-S334.
6. Hood N, Considine J. Spinal immobilisaton in pre-hospital and emergency care: a systematic review of the literature. Australasian Emergency Nursing Journal 2015;18:118-37.
7. Electrical Safety: Look up and Live. Energy Safe Victoria. Accessed 12 November 2020 from https://esv.vic.gov.au/campaigns/look-up-and-live/ 2020.

Further Reading

- ANZCOR Guideline 3 Recognition and First Aid Management of the Unconscious Person

- ANZCOR Guideline 4 Airway
- ANZCOR Guideline 5 Breathing
- ANZCOR Guideline 8 Cardiopulmonary Resuscitation
- ANZCOR Guideline 9.1.3 Burns
- ANZCOR Guideline 9.1.6 Management of suspected spinal injury
- ANZCOR Guideline 9.2.3 Shock

About this Guideline

- Search date/s: ILCOR literature search details and dates are available on the CoSTR page of the ILCOR website (https://costr.ilcor.org) and the relevant CoSTR documents:
  - CPR Prior to Call for Help (BLS): Systematic Review https://costr.ilcor.org/document/cpr-prior-to-call-for-help-task- force-systematic-review-costr
- Questions/PICOs: Are described in the CoSTR documents (https://costr.ilcor.org)
- Method: Mixed methods including ARC NHMRC methodology before 2017 and ILCOR GRADE methodology described in ILCOR publications since 2017.
- Principal reviewers: Julie Considine, Hugh Grantham
- Approved: April 2021
- Guideline superseded: August 2016

ANZCOR Guideline 3 - Recognition and First

Aid Management of the Unconscious Person

Summary

Who does this guideline apply to?

This guideline applies to all persons who are unconscious. **Unconsciousness** is a state of unrousable, unresponsiveness, where the person is unaware of their surroundings and no purposeful response can be obtained.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders or first aid providers, first responders and health professionals.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) recommends that in all emergencies, the rescuer should manage the unconscious person who is breathing normally as follows:

1. Check for danger (assess and manage risks to the rescuer and others).
2. Assist the unconscious person to the ground and position on their side. Ensure the airway is open (Guideline 4). Do not leave the person sitting in a chair nor put their head between their knees.
3. **Call an ambulance.**
4. Promptly stop any bleeding (Guideline 9.1.1).
5. Constantly re-check the person's condition for any change.

If the person is unresponsive and not breathing normally, follow ANZCOR Basic Life Support Flowchart (Guideline 8).

Guideline

1 Causes of Unconsciousness

The causes of unconsciousness can be classified into four broad groups:

- low brain oxygen levels
- heart and circulation problems (e.g. fainting, abnormal heart rhythms)
- metabolic problems (e.g. overdose, intoxication, low blood sugar)
- brain problems (e.g. head injury, stroke, tumour, epilepsy).

Combinations of different causes may be present in an unconscious person e.g. a head injury due to the influence of alcohol.

2 Recognition

Before loss of consciousness, the person may experience yawning, dizziness, sweating, change from normal skin colour, blurred or changed vision, or nausea.

Assess the collapsed person's response to verbal and tactile stimuli ('talk and touch'), ensuring that this does not cause or aggravate any injury. This may include giving a simple command such as, "open your eyes; squeeze my hand; let it go". Then grasp and squeeze the shoulders firmly to elicit a response.

A person who fails to respond or shows only a minor response, such as groaning without eye opening, should be managed as if unconscious. [Good practice statement]

3 Management

If the person is unresponsive and not breathing normally, follow ANZCOR Basic Life Support Flowchart (Guideline 8).

With an unconscious breathing person, care of the airway takes precedence over any injury, including the possibility of a spinal injury (Guideline 9.1.6). An unconscious person must be handled gently and every effort made to avoid any twisting or forward movement of the head and spine.[1]

ANZCOR suggests that an unresponsive person who is breathing normally be positioned into a lateral, side-lying recovery (lateral recumbent) position as opposed to leaving them supine.[2-4] [2015/2020 CoSTR, weak recommendation, very-low-certainty evidence]

1. Ensure the safety of both the person and rescuer.
2. Assist the unconscious person to the ground and position them on the side. Ensure their airway is open (Guideline 4). Do not leave the person sitting in a chair nor put their head between their knees.
3. **Call an ambulance.**
4. Promptly stop any bleeding (Guideline 9.1.1).
5. Constantly re-check the person's condition for any change.
6. Ideally, the most experienced rescuer should stay with the person.

References

1. Hood N, Considine J. Spinal immobilisaton in pre-hospital and emergency care: a systematic review of the literature. Australasian Emergency Nursing Journal 2015;18:118-37.
2. Zideman DA, Singletary EM, De Buck EDJ, et al. Part 9: First aid 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e225-e61.
3. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science With Treatment Recommendations. Circulation 2020;142:S284-S334.
4. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science With Treatment Recommendations. Resuscitation 2020;156:A240-A82.

Further Reading

- ANZCOR Guideline 4 Airway
- ANZCOR Guideline 8 Cardiopulmonary Resuscitation
- ANZCOR Guideline 9.1.1 Principles of Control of Bleeding for First Aiders
- ANZCOR Guideline 9.1.6 Management of Suspected Spinal Injury

About this Guideline

- Search date/s: ILCOR literature search details and dates are available on the CoSTR page of the ILCOR website (https://costr.ilcor.org) and the relevant CoSTR documents:
  - CPR Prior to Call for Help (BLS): Systematic Review https://costr.ilcor.org/document/cpr-prior-to-call-for-help-task- force-systematic-review-costr
- Questions/PICOs: Are described in the CoSTR documents (https://costr.ilcor.org)
- Method: Mixed methods including ARC NHMRC methodology before 2017 and ILCOR GRADE methodology described in ILCOR publications since 2017.
- Principal reviewers: Julie Considine, Hugh Grantham
- Approved: April 2021
- Guideline superseded: January 2016

ANZCOR Guideline 4 - Airway

Summary

Who does this guideline apply to?

This guideline applies to all persons who need airway management. Airway management is required to provide an open airway when a person is unconscious, has an obstructed airway, or needs rescue breathing.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders or first aid providers, first responders and health professionals.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. Check for danger (assess and manage risks to the rescuer and others)
2. In an unconscious person, care of the airway takes precedence over any injury, including the possibility of spinal injury.
3. To assess breathing and airway, leave the person in the position in which they have been found, unless fluid or matter obstructs the airway.
4. To clear the airway the mouth should be opened and the head turned slightly downwards to allow any obvious foreign material (e.g. food, vomit, blood and secretions) to drain.
5. If the airway becomes compromised during resuscitation, promptly roll the person onto their side to clear the airway. Once the airway is clear, reassess for responsiveness and normal breathing. If the person is unresponsive and not breathing normally, follow ANZCOR Basic Life Support Flowchart (Guideline 8).
6. For an unresponsive adult or child, open the airway using the head tilt-chin lift. For an infant, open the airway by placing the head in the neutral position and support the jaw from falling back.
7. Manage Foreign Body Airway Obstructions using the Choking Algorithm (Figure 4).

Guideline

1 General Principles

When someone is unconscious, all muscles are relaxed. If they are left lying on their back, the tongue, which is attached to the back of the jaw, falls against the back wall of the throat and blocks air from entering the lungs. Other soft tissues of the airway may worsen this obstruction. The mouth falls open but this tends to block, rather than open, the airway.

The unconscious person is further at risk because of being unable to swallow or cough out foreign material in the airway. This may cause airway obstruction, or laryngeal irritation and foreign material may enter the lungs. For this reason the rescuer should not give an unconscious person anything by mouth, and should not attempt to induce vomiting.

If foreign material irritates the vocal cords, a protective reflex muscular spasm (laryngeal spasm) prevents the entry of material into the lungs. This may result in partial or complete airway blockage of the entrance to the trachea (windpipe) with the person often making an abnormal noise (stridor) during attempts to breathe. Airway closure due to laryngeal spasm can be complete; in this case there is no noise (stridor) because there is no airflow. That can persist until the person becomes blue or unconscious from lack of oxygen. When consciousness is lost, the spasm usually relaxes.

In an unconscious person, care of the airway takes precedence over any injury , including the possibility of spinal injury (Refer to Guideline 9.1.6). All who are unconscious should be handled gently with no twisting or bending of the spinal column and especially the neck.[1] If it is necessary, move the head gently to obtain a clear airway. Where possible, an assistant should support the head when an injured person is being moved, but no time should be wasted in detailed positioning.

The person should not be routinely rolled onto the side to assess airway and breathing - leave them in the position in which they have been found. This has the advantages of simplified teaching, taking less time to perform and avoids movement. The exceptions to this would be where the airway is obstructed with fluid (water or blood) or matter (sand, debris, vomit). Here, the person should be promptly rolled onto their side to clear the airway.

The mouth should be opened and the head turned slightly downwards to allow any obvious foreign material (e.g. food, vomit, blood and secretions) to drain. Loose dentures should be removed, but well-fitting ones can be left in place. Visible material can be removed by using the rescuer's fingers. Case series reported the finger sweep as effective for relieving foreign body airway obstruction (FBAO) in unconscious adults and children aged >1yr. However, five case reports documented harm to the person's mouth or biting of the rescuer's finger[1].

If the airway becomes compromised during resuscitation, promptly roll the person onto their side to clear the airway. Once the airway is clear, reassess for responsiveness and normal breathing, then begin resuscitation as appropriate following the ANZCOR Basic Life Support Flowchart (Guideline 8).

Regurgitation is the passive flow of stomach contents into the mouth and nose. Although this can occur in any person, regurgitation and inhalation of stomach contents is a major threat to an unconscious person. It is often unrecognised because it is silent and there is no obvious muscle activity. **Vomiting** is an active process during which muscular action causes the stomach to eject its contents.

In resuscitation, regurgitation and vomiting are managed in the same way: by prompt positioning the person on their side and manual clearance of the airway prior to continuing rescue breathing.

If the person begins to breathe normally, they can be left on their side with appropriate head tilt. If not breathing normally, the person must be rolled on their back and resuscitation commenced.

2 Airway Management

Airway management is required to provide an open airway when the person:

- is unconscious
- has an obstructed airway
- needs rescue breathing.

For unresponsive adults and children, it is reasonable to open the airway using the head tilt- chin lift manoeuvre.[1] [Good practice statement] For lay rescuers performing compression-only CPR, there is insufficient evidence to recommend the use of any specific passive airway manoeuvre.[1] However, the value of maintaining an unobstructed airway is recognised. [Good practice statement]

2.1 Head Tilt/Chin Lift

One hand is placed on the forehead or the top of the head. The other hand is used to provide Chin Lift. The head (NOT the neck) is tilted backwards (see Figure 1). It is important to avoid excessive force, especially where neck injury is suspected. When the person is on their side, the head will usually remain in this position when the rescuer's hands are withdrawn.

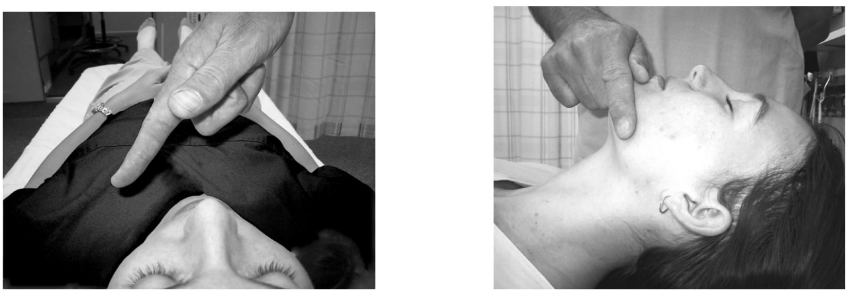


Figure 1: Head tilt/chin lift manoeuvre

Chin lift is commonly used in conjunction with Backward Head Tilt. The chin is held up by the rescuer's thumb and fingers in order to open the mouth and pull the tongue and soft tissues away from the back of the throat.

A suggested technique is to place the thumb over the chin below the lip and supporting the tip of the jaw with the middle finger and the index finger lying along the jaw line. Be careful that the ring finger does not squash the soft tissues of the neck. The jaw is held open slightly and pulled away from the chest.

2.2 Children and Infants

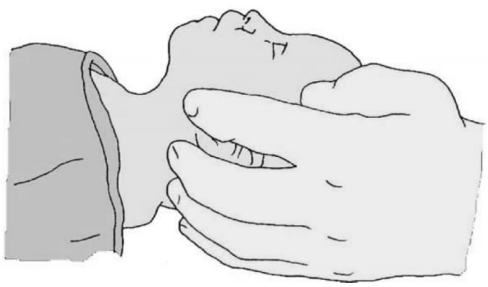
An **infant** is defined as younger than one year, a **child** as one to eighteen years of age (or up to onset of puberty if the age is unknown). In both cases the principle is to maintain an open airway.

Children

Children should be managed as for adults.

Infants

In an infant, the upper airway is easily obstructed because of the narrow nasal passages, the entrance to the windpipe (vocal cords) and the trachea (windpipe). The trachea is soft and pliable and may be distorted by excessive backward head tilt or jaw thrust. Therefore, in an infant the head should be kept neutral and maximum head tilt should not be used (Figure 2). The lower jaw should be supported at the point of the chin while keeping the mouth open. There must be no pressure on the soft tissues of the neck. If these manoeuvres do not provide a clear airway, the head may be tilted backwards very slightly with a gentle movement. [Good practice statement]



Infant in Neutral Position

(Reproduced Courtesy of European Resuscitation Council)

Figure 2: Infant in neutral position

3 Recognition of Upper Airway Obstruction

Airway obstruction may be partial or complete, and present in the conscious or the unconscious person. Typical causes of airway obstruction may include, but are not limited to:

- relaxation of the airway muscles due to unconsciousness
- inhaled foreign body
- trauma to the airway
- anaphylactic reaction.

The symptoms and signs of obstruction will depend on the cause and severity of the condition. Airway obstruction may occur gradually or suddenly, and may lead to complete obstruction within a few seconds. As such the person should be observed continually.

In the conscious person who has inhaled a foreign body, there may be extreme anxiety, agitation, gasping sounds, coughing or loss of voice. This may progress to the universal choking sign, namely clutching the neck with the thumb and fingers (as shown in Figure 3).

Figure 3: Universal choking sign

Airway obstruction will cause the diaphragm muscle to work harder to achieve adequate ventilations. The abdomen will continue to move out but there will be loss of the natural rise of the chest (paradoxical movement), and in-drawing of the spaces between the ribs and above the collar bones during inspiration.

Partial obstruction can be recognised where:

- breathing is labored
- breathing may be noisy
- some escape of air can be felt from the mouth.

Complete obstruction can be recognised where:

- there may be efforts at breathing
- there is no sound of breathing
- there is no escape of air from nose and/or mouth.

Airway obstruction may not be apparent in the non-breathing unconscious person until rescue breathing is attempted.

4 Management of Foreign Body Airway Obstruction

(Choking)

A Foreign Body Airway Obstruction (FBAO) is a life-threatening emergency. Chest thrusts or back blows are effective for relieving FBAO in conscious adults and children with low risk of harm (only 4 observational studies report harm from back blows and 5 observational studies report harm from chest thrusts).[3-5] Life-threatening complications associated with use of abdominal thrusts (including the Heimlich Manoeuvre) have been reported in 52 observational studies.[3-5] Therefore, the use of abdominal thrusts in the management of FBAO is not recommended and, instead back blows and chest thrusts should be used. [Good practice statement] These techniques should be applied in rapid sequence until the obstruction is relieved. More than one technique may be needed: there is insufficient evidence to determine which should be used first.

4.1 Assess Severity

The simplest way to assess severity of a FBAO is to assess for effective cough.

4.2 Effective Cough (Mild Airway Obstruction)

The person with an effective cough should be given reassurance and encouragement to keep coughing to expel the foreign material. If the obstruction is not relieved the rescuer should call an ambulance.

4.3 Ineffective Cough (Severe Airway Obstruction)

Conscious person

If the person is conscious send for an ambulance and perform up to five sharp, back blows with the hand in the middle of the back between the shoulder blades. Check to see if each back blow has relieved the airway obstruction. The aim is to

relieve the obstruction with each blow rather than to give all five blows. An infant may be placed in a head downwards position prior to delivering back blows, i.e. across the rescuer's lap.[1,2] [Good practice statement]

If back blows are unsuccessful the rescuer should perform up to five chest thrusts. To perform chest thrusts, identify the same compression point as for CPR and give up to five chest thrusts. These are similar to chest compressions but sharper and delivered at a slower rate. The infant should be placed in a head downwards and on their back across the rescuer's thigh, while children and adults may be treated in the sitting or standing position.[1,2] [Good practice statement]

With each chest thrust, check to see whether the airway obstruction has been relieved. The aim is to relieve the obstruction rather than deliver all five chest thrusts. If the obstruction is still not relieved and the person remains responsive, continue alternating five back blows with five chest thrusts.

Unconscious person

ANZCOR suggests against the use of blind finger sweeps.[3,4] [2020 CoSTR, weak recommendation, very-low-certainty evidence] ANZCOR suggests that rescuers consider the manual extraction of visible items in the mouth.[3,4] [2020 CoSTR, weak recommendation, very- low-certainty evidence] If the person is unresponsive and not breathing normally, follow ANZCOR Basic Life Support Flowchart (Guideline 8).

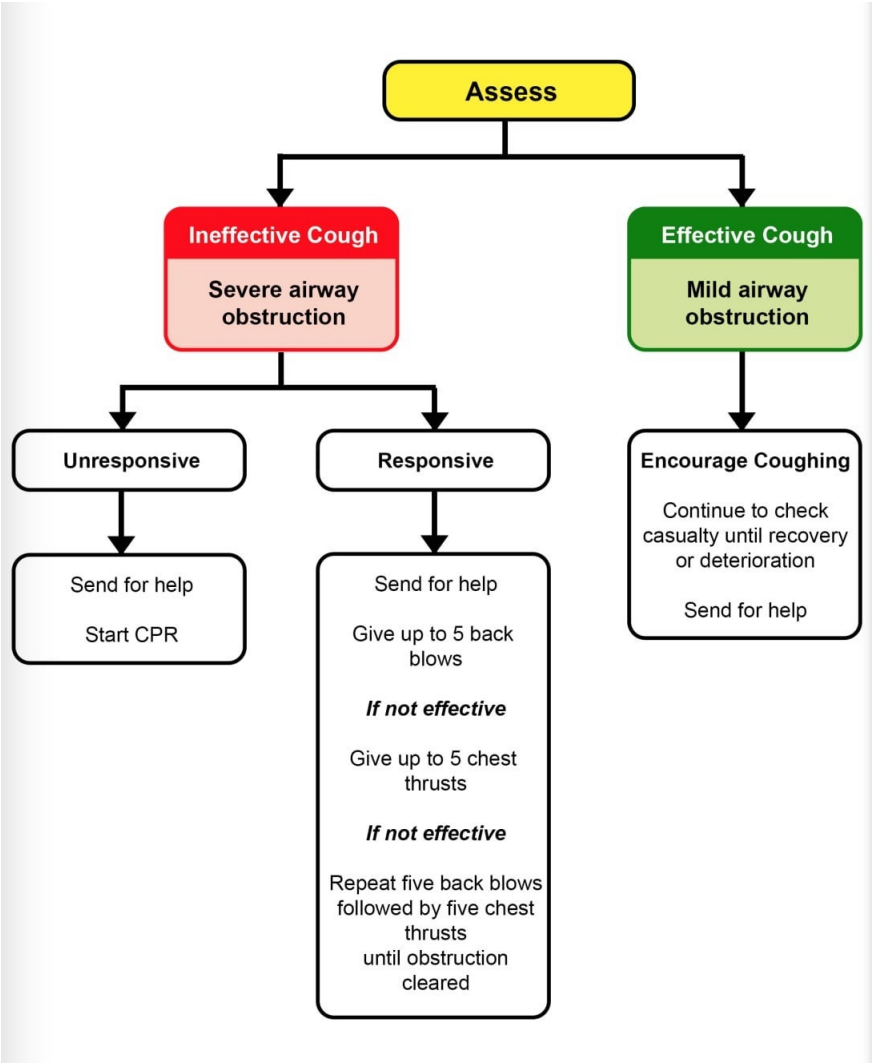


Figure 4: Management of Foreign Body Airway Obstruction (Choking) Algorithm

References

1. Hood N, Considine J. Spinal immobilisaton in pre-hospital and emergency care: a systematic review of the literature. Australasian Emergency Nursing Journal 2015;18:118-37.
2. Koster RW, Sayre MR, Botha M, et al. Part 5: Adult basic life support: 2010 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation 2010;81:e48-e70.
3. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Circulation 2020;142:S41-S91.
4. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2020;156:A35-A79.

5. Couper K, Abu Hassan A, Ohri V, et al. Removal of foreign body airway obstruction: A systematic review of interventions. Resuscitation 2020;156:174-81.

Further Reading

ANZCOR Guideline 9.1.6 Management of Suspected Spinal Injury

About this Guideline

- Search date/s: ILCOR literature search details and dates are available on the CoSTR page of the ILCOR website (https://costr.ilcor.org) and the relevant CoSTR documents:
  - Airway Management in Drowning (BLS 856): Scoping Review https://costr.ilcor.org/document/bls-856-airway-management- in-drowning-tf-scoping-review
  - Removal of Foreign Body Airway Obstruction (BLS 368): Systematic Review https://costr.ilcor.org/document/removal-of-foreign-body- airway-obstruction-tfsr-costr
- Questions/PICOs: Are described in the CoSTR documents (https://costr.ilcor.org)
- Method: Mixed methods including ARC NHMRC methodology before 2017 and ILCOR GRADE methodology described in ILCOR publications since 2017.
- Principal reviewers: Julie Considine, Hugh Grantham, Alan Morrison, Helen Liley, Jason Acworth
- Acknowledgment Thanks to Charlotte Foley for illustrations
- Approved: April 2021
- Guideline superseded: January 2016

ANZCOR Guideline 5 - Breathing

Summary

Who does this guideline apply to?

This guideline applies to all persons in need of resuscitation.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders or first aid providers, first responders and health professionals.

Recommendations.

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. Persons who are gasping or breathing abnormally and are unresponsive require resuscitation.
2. When assessing breathing, rescuers should look, listen and feel: **LOOK** for movement of the upper abdomen or lower chest; **LISTEN** for the escape of air from nose and mouth; and **FEEL** for movement of air at the mouth and nose.
3. The ratio of compressions to rescue breaths is 30:2.
4. Mouth to mouth, mouth to nose, mouth to mask and bag valve mask are all viable methods of rescue breathing. Mouth to stoma should be used where a person has had a laryngectomy.
5. Risk of disease transmission is very low and rescuers need not be deterred from providing rescue breaths without a barrier device. However, rescuers should consider using a barrier device if this is available.
6. Those who are trained and willing to give rescue breaths do so for all persons who are unresponsive and not breathing normally.

Guideline

Normal breathing is essential to maintaining life. A person who is gasping or breathing abnormally and is unresponsive requires resuscitation.[1,2]

1 Causes of Ineffective Breathing of Acute Onset

Breathing may be absent or ineffective as a result of:

- direct depression of, or damage to, the breathing control centre of the brain
- upper airway obstruction
- paralysis or impairment of the nerves and/or muscles of breathing
- problems affecting the lungs
- drowning
- suffocation.

2 Assessment of Breathing

There is a high incidence of abnormal gasping (agonal gasps) after cardiac arrest.[1,4] All rescuers should use a combination of unresponsiveness and absent or abnormal breathing to identify the need for resuscitation.[1,2] [Good practice statement]

The rescuer should maintain an airway and assess for normal breathing:

- **LOOK** for movement of the upper abdomen or lower chest
- **LISTEN** for the escape of air from nose and mouth
- **FEEL** for movement of air at the mouth and nose.

Movement of the lower chest and upper abdomen does not necessarily mean the person has a clear airway. Impairment or complete absence of breathing may develop before the person loses consciousness.

3 Rescue Breathing

If the unconscious person is unresponsive and not breathing normally after the airway has been opened and cleared, the rescuer must immediately begin chest compressions and then rescue breathing. Give 30 compressions and then two breaths, allowing about one second for each ventilation following the ANZCOR Basic Life Support Flowchart (Guideline 8).[1,2] [2015/ CoSTR, weak recommendation, very-low-certainty evidence]

ANZCOR suggests that those who are trained and willing to give breaths do so for all persons who are unresponsive and not breathing normally.[1-4] [2015/2020 CoSTR weak recommendation, very-low-certainty evidence]

3.1 Mouth to mouth

Kneel beside the victim's head. Maintain an open airway (refer to ANZCOR Guideline 4).

Take a breath, open your mouth as widely as possible and place it over the person's slightly open mouth. While maintaining an open airway, pinch the nostrils (or seal nostrils with rescuer's cheek) and blow to inflate the person's lungs. Because the hand supporting the head comes forward some head tilt may be lost and the airway may be obstructed. Pulling upwards with the hand on the chin helps to reduce this problem.

For mouth to mouth ventilation, it is reasonable to give each breath in a short time (one second) with a volume to achieve chest rise regardless of the cause of cardiac arrest.[5] [Good practice statement] Care should be taken not to over-inflate the chest. [Good practice statement]

Look for rise of the chest during each inflation. If the chest does not rise, possible causes are:

- obstruction in the airway (tongue or foreign material, or inadequate head tilt, chin lift)
- insufficient air being blown into the lungs
- inadequate air seal around mouth and or nose.

If the chest does not rise, ensure correct head tilt, adequate air seal and ventilation. After inflating the lungs, lift your mouth from the person's mouth, turn your head towards their chest and listen and feel for air being exhaled from the mouth and nose.

3.2 Mouth to nose

The mouth to nose method may be used:

- where the rescuer chooses to do so
- where the person's jaws are tightly clenched
- when resuscitating infants and small children.

The technique for mouth to nose is the same as for mouth to mouth except for sealing the airway. Close the mouth with the hand supporting the jaw and push the lips together with the thumb. Take a breath and place your widely opened mouth over the person's nose (or mouth and nose in infants) and blow to inflate the lungs. Lift your mouth from the person's nose. Look for the fall of the chest, and listen and feel for the escape of air from the nose and mouth.

If the chest does not move, there is an obstruction, an ineffective seal, or insufficient air being blown into the lungs. In mouth-to-nose resuscitation a leak may occur if the rescuer's mouth is not open sufficiently, or if the person's mouth is not sealed adequately. If this problem persists, use mouth-to-mouth resuscitation. If blockage of the nose prevents adequate inflation, the rescuer should use mouth-to-mouth resuscitation.5,6. [Good practice statement]

3.3 Mouth to mask

Mouth to mask resuscitation is a method of rescue breathing which avoids mouth-to-mouth contact by using a resuscitation mask. Rescuers should take appropriate safety precautions when feasible and when resources are available to

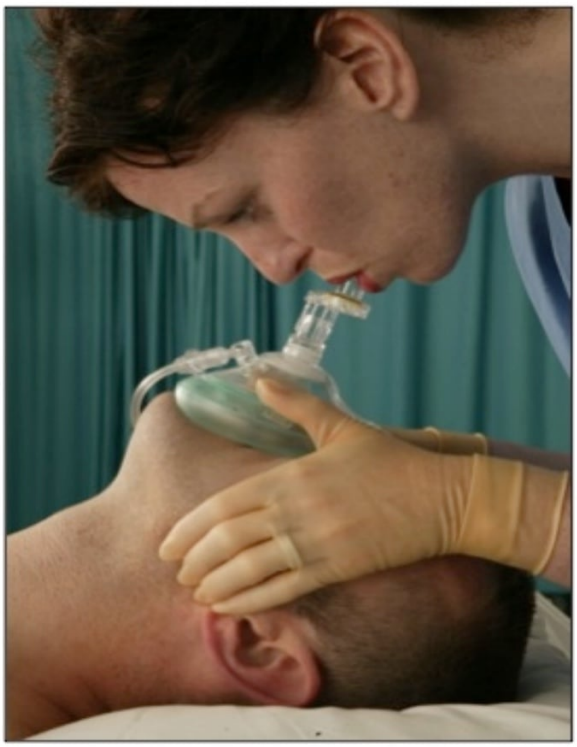
do so, especially if a person is known to have a serious infection (e.g. HIV, tuberculosis, Hepatitis B virus or SARS).[7] [Good practice statement]

Position yourself at the person's head and use both hands to maintain an open airway and to hold the mask in place to maximise the seal. Maintain head tilt and chin lift. Place the narrow end of the mask on the bridge of the nose and apply the mask firmly to the face. (Figure 1)

Inflate the lungs by blowing through the mouthpiece of the mask with sufficient volume and force to achieve chest movement. Remove your mouth from the mask to allow exhalation.

Turn your head to listen and feel for the escape of air. If the chest does not rise, recheck head tilt, chin lift and mask seal.

Failure to maintain head tilt and chin lift is the most common cause of obstruction during resuscitation.



Mouth to mask method
(Reproduced Courtesy of European Resuscitation Council)

Figure 1: Mouth to Mask method

3.4 Bag valve mask

For rescuers trained in its use, bag valve mask ventilation is an alternative method option of providing rescue breathing A bag valve mask device is a self-inflating bag attached to a non- rebreathing valve and face mask. There is an option to connect the bag to a reservoir and oxygen supply. There are different size devices for infants, children and adults: it is important to select the size

appropriate to the person. Successful bag valve mask ventilation requires a patent airway (Guideline 4), adequate mask seal, and adequate ventilation technique. Prolonged bag valve mask ventilation or poor technique may introduce air into the stomach increasing the risk of regurgitation of gastric contents.

It is recommended that when bag valve mask ventilation is used, two trained rescuers provide ventilation for a non-breathing person: one to manage the airway, mask and seal, and the second to operate the bag.[8] ANZCOR considers bag valve mask ventilation an extension to Basic Life Support thus it is not mandated in Basic Life Support training programs. [Good practice statement]

3.5 Mouth to neck stoma

A person with a laryngectomy has had the larynx (voice box) removed and breathes through a hole in the front of their neck (stoma). A stoma will be more obvious when the person is on their back for Rescue Breathing and the head is put into backward tilt. If a tube is seen in the stoma, always leave it in place to keep the hole open for breathing and resuscitation.

The rescuer should place their mouth over the stoma and perform rescue breathing as described above. If the chest fails to rise, this may be due to a poor seal over the stoma, or the person having a tracheostomy rather than laryngectomy thus allowing air to escape from the mouth and nose or a blocked stoma or tube. If stoma or tube is blocked use back blows and chest thrusts in an attempt to dislodge the obstruction (Refer to Guideline 4). [Good practice statement]

4 Risks

No human studies have addressed the safety, effectiveness, or feasibility of using barrier devices to prevent person-to-rescuer contact during rescuer breathing.[7] Nine clinical reports advocate the use of barrier devices to protect the rescuer from transmitted disease: three studies showed that barrier devices can decrease transmission of bacteria in controlled laboratory settings.[7]

The risk of disease transmission is very low and need not deter rescue breathing without a barrier device. If available, rescuers should consider using a barrier device.[7] [Good practice statement]

References

1. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2020; **142** (16_suppl_1): S41-S91.
2. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2020; **156** : A35-A79.

3. Perkins GD, Travers AH, Berg RA, et al. Part 3: Adult basic life support and automated external defibrillation. 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2015; **95** : e43-e69.
4. Travers AH, Perkins GD, Berg RA, et al. Part 3: Adult Basic Life Support and Automated External Defibrillation 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2015; **132** (16 suppl 1): S51-S83.
5. Koster RW, Sayre MR, Botha M, et al. Part 5: Adult basic life support: 2010 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Resuscitation* 2010; **81** (1): e48-e70.
6. de Caen AR, Kleinman ME, Chameides L, et al. Part 10: Paediatric basic and advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2010; **81** (1): e213- e59.
7. Soar J, Mancini ME, Bhanji F, et al. Part 12: Education, implementation, and teams: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2010; **81** (1): e288-e330.
8. Jesudian MC, Harrison RR, Keenan RL, Maull KI. Bag-valve-mask ventilation; two rescuers are better than one: preliminary report. *Crit Care Med* 1985; **13** (2): 122-3.

Further Reading

- ANZCOR Guideline 4 Airway
- ANZCOR Guideline 9.3.2 Resuscitation of the Drowning Victim
- ANZCOR Guideline 10.1 Basic Life Support Training

About this Guideline

- Search date/s: ILCOR literature search details and dates are available on the CoSTR page of the ILCOR website (https://costr.ilcor.org) and the relevant CoSTR documents:
  - Harm to Rescuers from CPR (BLS): Scoping Review https://costr.ilcor.org/document/harm-to-rescuers-from-cpr- scoping-review
  - Starting CPR (ABC vs CAB) (BLS): Scoping Review https://costr.ilcor.org/document/starting-cpr-abc-vs-cab-tfsr- costr
- Questions/PICOs: Are described in the CoSTR documents (https://costr.ilcor.org)
- Method: Mixed methods including ARC NHMRC methodology before 2017 and ILCOR GRADE methodology described in ILCOR publications since 2017.

- Principal reviewers: Julie Considine, Hugh Grantham
- Approved: April 2021
- Guideline superseded: January 2016

ANZCOR Guideline 6 - Compressions

Summary

Who does this guideline apply to?

This guideline applies to all persons who are unresponsive and not breathing normally.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders or first aid providers, first responders and health professionals.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. All rescuers should perform chest compressions for all persons who are unresponsive and not breathing normally.
2. Interruptions to chest compressions should be minimised.
3. Those who are trained and willing to give rescue breaths do so for all persons who are unresponsive and not breathing normally.

Guideline

All rescuers should perform chest compressions for all persons who are unresponsive and not breathing normally.[1,2] [Strong recommendation, moderate certainty of evidence] ANZCOR suggests that those who are trained and willing to give rescue breaths do so for all who are in cardiac arrest.[1], [Weak recommendation, very-low-certainty evidence]

1 Recognition of the need for Chest Compressions

All rescuers, including health care professionals, should use unresponsiveness and absence of normal breathing to identify the need for resuscitation.[1-4] [Good practice statement] Palpation of a pulse is unreliable and should not be performed to confirm the need for resuscitation.[5-8] [Good practice statement]

2 Locating the site for Chest Compressions

ANZCOR suggests performing chest compressions on the lower half of the sternum.[1,2,4,9] [CoSTR 2015/2020, weak recommendation, very-low-certainty evidence] In making this recommendation, we place a high value on consistency with current treatment recommendations in the absence of compelling data suggesting the need to change the recommended approach.[1,2,4,9] Place the heel of their hand in the centre of the chest with the other hand on top (Figure 1). [Good practice statement]

Avoid compression beyond the lower limit of the sternum. Compression applied too high is ineffective and if applied too low may cause regurgitation and/or damage to internal organs. [Good practice statement]

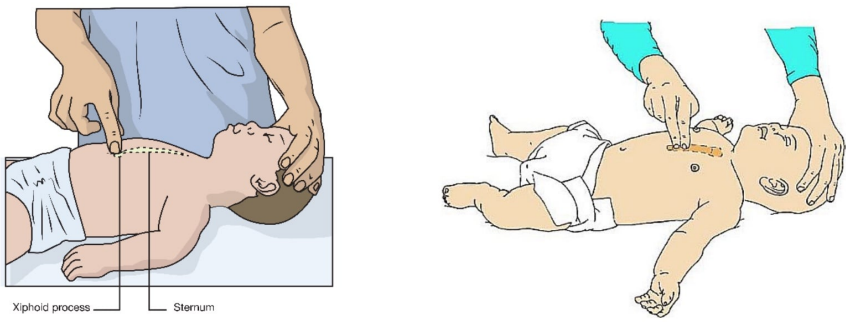
 Locating the site of chest compressions in adult CPR

Figure 1: Location of chest compressions

3 Method of Compression

3.1 Infants

In infants, ANZCOR suggests the two finger technique should be used by lay rescuers in order to minimise transfer time from compression to ventilation. Having obtained the compression point the rescuer places two fingers on this point and compresses the chest (Figure 2). [Good practice statement]



(adapted courtesy of European Resuscitation Council)

Figure 2: Method of compression for infants

3.2 Children and Adults

Either a one or two hand technique can be used for performing chest compressions in children.[5-8] (Figure 3) [Good practice statement]

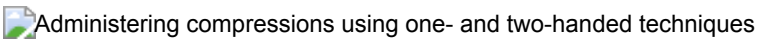

Administering compressions using one- and two-handed techniques

Figure 3: Administering compressions using one- and two-handed techniques

Interruptions to chest compressions must be minimized.[3,4] [2015 CoSTR, weak recommendation, low- quality evidence] A person requiring chest compressions should be placed on their back on a firm surface before commencing chest compressions to optimise the effectiveness of compressions.[1,2,10] [CoSTR, weak recommendation, very-low-certainty evidence] Compressions should be rhythmic with equal time for compression and relaxation. The rescuer must avoid either rocking backwards and forwards, or using thumps or quick jabs. Rescuers should allow complete recoil of the chest after each compression.[1,2,11] [2020 CoSTR, weak recommendation, very-low-certainty evidence]

3.3 Pregnant women

There are no published studies of optimum positioning in pregnant women undergoing cardiopulmonary resuscitation (CPR) so recommendations to date are extrapolated from manikin studies or studies of pregnant women who are not in cardiac arrest. Good quality, uninterrupted chest compressions as described above should be the immediate priority in all pregnant women who are unresponsive and not breathing normally.[1,2,4,9] [Good practice statement]

In noticeably pregnant women, standard CPR should be commenced immediately. Once CPR is in progress, if there are sufficient resources available, rescuers should place padding such as a towel, cushion or similar object under the right hip to tilt the woman's hips (approximately 15-30 degrees) to the left but leave her shoulders flat to enable good quality chest compressions. The reason for this position in pregnant women is to move the weight of the pregnant uterus off of her major blood vessels in the abdomen. If a tilted position is not possible or tilting the hips compromises the quality of chest compressions, then chest compressions should be performed as described as above with the woman on her back.

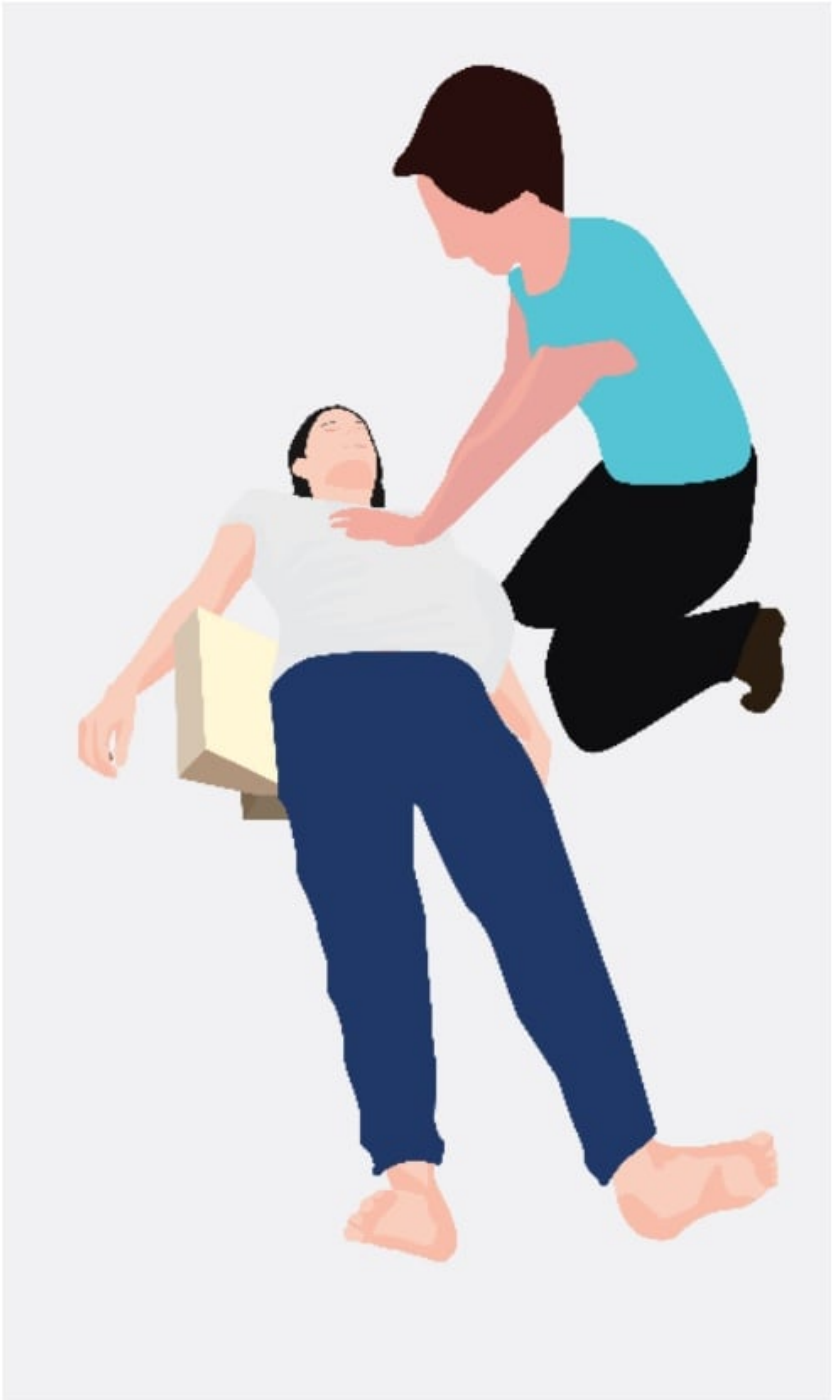


Figure 4: Padding the noticeably pregnant woman

4 Depth of Compressions

The lower half of the sternum should be depressed approximately one third of the depth of the chest with each compression. This equates to more than 5cm in adults,[1,2,4,9,11] approximately 5cm in children[5,6,12,13] and 4 cm in infants.[5,6,12,13] [2015/2020 CoSTR, weak recommendation, very-low-certainty evidence] ANZCOR places greater importance on adequate compression depth.

Although there is some evidence suggesting detriment with compression depths greater than 6cm, the clinical reality of being able to tell the difference between 5 or 6 cm and adjust compressions accordingly is questionable. Inadequate compression depth is definitely associated with poor outcomes. ANZCOR has elected not to put an upper limit on compression depth as the risk of too shallow compressions outweighs the risk of compressions that are too deep.[3,4] [Good practice statement]

5 Rate of Compressions

Rescuers should perform chest compressions for all ages at a rate of 100 to 120 compressions per minute (almost 2 compressions/second).[1,2,4,9,11] [CoSTR 2015/2020, strong recommendation, very-low-certainty evidence] This does not imply that 100-120 compressions will be delivered each minute since the number will be reduced by interruptions for breaths given by rescue breathing. ANZCOR acknowledges that compression rates will vary between and within providers and survival rates are optimised at compressions rates of 100-120 compressions per minute. There is some evidence that compressions rates less than 100 or greater than 140 compressions per minute are associated with lower rates of survival.[1,2,4,9,11]

6 CPR Quality

The compression rate and depth is variable among rescuers and compressions may be worse in the first 5 minutes of the arrest.[14] One manikin study of rescuer CPR showed that compressions became shallow within one minute, but providers became aware of fatigue only after 5 min.[7] When performing compressions, if feasible, change rescuers at least every two minutes to prevent rescuer fatigue and deterioration in chest compression quality, particularly depth.[1,2,15-17] [Good practice statement] Changing rescuers performing chest compressions should be done with a minimum of interruptions to compressions.[14,17]

7 Feedback

There is no high level evidence that the use of CPR feedback devices during real time CPR improves survival or return of spontaneous circulation.[1-4] CPR prompt / feedback devices may be considered for clinical use to provide data as part of an overall strategy to improve quality of CPR at a systems level.[1-4] [CoSTR 2015/2020, weak recommendation, very-low-certainty evidence]. ANZCOR places a higher value on resource allocation and cost effectiveness than widespread implementation of a technology with uncertain effectiveness during real time CPR. We acknowledge that data provided by CPR feedback devices may benefit other victims as part of a broader quality improvement system.[1-4]

8 Risks

Rib fractures and other injuries are common but acceptable consequences of CPR given the alternative of death.[15] [Good practice statement] CPR should be initiated for presumed cardiac arrest without concerns of harm to patients not in cardiac arrest.[3,4] [CoSTR 2015, strong recommendation, very-low- certainty evidence] In making this recommendation, ANZCOR places a higher value on the survival benefit of CPR initiated by laypersons for patients in cardiac arrest against the low risk of injury in patients not in cardiac arrest.[3,4]

References

1. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2020; **142** (16_suppl_1): S41-S91.
2. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2020; **156** : A35-A79.
3. Perkins GD, Travers AH, Berg RA, et al. Part 3: Adult basic life support and automated external defibrillation. 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2015; **95** : e43-e69.
4. Travers AH, Perkins GD, Berg RA, et al. Part 3: Adult Basic Life Support and Automated External Defibrillation 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2015; **132** (suppl 1): S51-S83.
5. Maconochie IK, Aickin R, Hazinski MF, et al. Pediatric Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2020; **142** (16_suppl_1): S140-S84.
6. Maconochie IK, Aickin R, Hazinski MF, et al. Pediatric Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2020; **156** : A120-A55.
7. de Caen AR, Kleinman ME, Chameides L, et al. Part 10: Paediatric basic and advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2010; **81** (1): e213-e59.
8. Kleinman ME, De Caen AR, Chameides L, et al. Part 10: Pediatric Basic and Advanced Life Support 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2010; **122** (16 suppl 2): S466- S515.
9. Perkins GD, Handley AJ, Koster RW, et al. European Resuscitation Council Guidelines for Resuscitation 2015: Section 2. Adult basic life support and automated external defibrillation. *Resuscitation* 2015; **95** : 81-99.

10. Holt J, Ward A, Mohamed T-Y, et al. The optimal surface for delivery of CPR: A systematic review and meta-analysis. *Resuscitation* 2020; **155** : 159-64.
11. Considine J, Gazmuri RJ, Perkins GD, et al. Chest compression components (rate, depth, chest wall recoil and leaning): A scoping review. *Resuscitation* 2020; **146** (Jan): 188-202.
12. Maconochie IK, de Caen AR, Aickin R, et al. Part 6: Pediatric basic life support and pediatric advanced life support 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2015; **95** : e147-e68.
13. De Caen AR, Maconochie IK, Aickin R, et al. Part 6: Pediatric Basic Life Support and Pediatric Advanced Life Support. *Circulation* 2015; **132** (16 suppl 1): S177-S203.
14. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *Jama* 2005; **293** (3): 299-304.
15. Koster RW, Sayre MR, Botha M, et al. Part 5: Adult basic life support: 2010 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Resuscitation* 2010; **81** (1): e48-e70.
16. Sayre MR, Koster RW, Botha M, et al. Part 5: adult basic life support: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Circulation* 2010; **122** (16_suppl_2): S298-S324.
17. International Liaison Committee on Resuscitation. Part 2. Adult basic life support. 2005 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Resuscitation* 2005; **67** : 187-200.

About this Guideline

- Search date/s: ILCOR literature search details and dates are available on the CoSTR page of the ILCOR website (https://costr.ilcor.org) and the relevant CoSTR documents:
  - Alternative Compression Techniques (BLS 374): Systematic Review https://costr.ilcor.org/document/alternative-compression- techniques-bls-374-tfsr
  - Chest Compression Rate (BLS): Scoping Review https://costr.ilcor.org/document/chest-compression-rate- scoping-review
  - Feedback for CPR Quality (BLS 361): Systematic Review https://costr.ilcor.org/document/feedback-for-cpr-quality-bls- 361-tf-sr
  - Firm Surface for CPR (BLS): Systematic Review https://costr.ilcor.org/document/firm-surface-for-cpr-tfsr-costr
  - Hand Position During Compressions (BLS 357): Systematic Review https://costr.ilcor.org/document/hand-position-during- compressions-bls-357-tf-systematic-review

    - Harm for CPR to Victims not in Cardiac arrest (BLS 353): Systematic Review https://costr.ilcor.org/document/harm-from-cpr-to-victims-not- in-cardiac-arrest-tfsr-costr-
    - Rescuer Fatigue in CC Only CPR (BLS 349): Scoping Review https://costr.ilcor.org/document/rescuer-fatigue-in-cc-only-cpr- bls-349-scoping-review
    - Starting CPR (ABC vs CAB) (BLS): Systematic Review https://costr.ilcor.org/document/rhythm-check-timing-tfsr- costr
- Questions/PICOs: Are described in the CoSTR documents (https://costr.ilcor.org)
- Method: Mixed methods including ARC NHMRC methodology before 2017 and ILCOR GRADE methodology described in ILCOR publications since 2017.
- Principal reviewers: Julie Considine, Hugh Grantham
- Approved: April 2021
- Guideline superseded: January 2016

ANZCOR Guideline 7 - Automated External Defibrillation in Basic Life Support

Summary

The importance of defibrillation has been well established as part of overall resuscitation, along with effective cardiopulmonary resuscitation (CPR). An Automated External Defibrillator (AED) must only be used for persons who are unresponsive and not breathing normally.

With cardiac arrest, time to defibrillation is a key factor that influences a person's chance of survival. A defibrillator should be applied to the person who is unresponsive and not breathing normally as soon as it becomes available so that a shock can be delivered if necessary.

Who does this guideline apply to?

This guideline applies to all persons who are unresponsive and not breathing normally.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders or first aid providers, first responders and health professionals.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. For all who are unresponsive and not breathing normally, chest compressions should be commenced immediately and continued until an AED is applied. The AED should be applied as soon as it becomes available so that a shock can be delivered if necessary.
2. The use of publicly accessible AEDs is recommended to increase survival rates in those who have cardiac arrest.
3. Pads are to be placed to ensure that a shock is delivered on an axis through the heart. Typical pad placement in adults and children is the anterior-lateral position.

4. Although AEDs are extremely safe, rescuers should take care not to touch a person during shock delivery.

Guideline

1 Background

The importance of defibrillation has been well established as part of overall resuscitation, along with effective cardiopulmonary resuscitation (CPR). An Automated External Defibrillator (AED) must only be used for persons who are unresponsive and not breathing normally. CPR must be continued until the AED is turned on and pads attached. The rescuer should then follow the AED prompts.

The time to defibrillation is a key factor that influences survival. For every minute defibrillation is delayed, there is approximately 10% reduction in survival if the victim is in cardiac arrest due to Ventricular Fibrillation (VF).[1] CPR alone will not save a person in VF. Hence a defibrillator should be applied to the person in need as soon as it becomes available so that a shock can be delivered if necessary.

The development of AEDs has made defibrillation part of basic life support. AEDs can accurately identify the cardiac rhythm as 'shockable' or 'non shockable'.

2 Which rescuers should use an AED?

AED use should not be restricted to trained personnel. Allowing the use of AEDs by individuals without prior formal training can be beneficial and may be life saving. Since even brief training improves performance (e.g. speed of use, correct pad placement), it is recommended that training in the use of AEDs (as a part of BLS) be provided.[2,3] [Good practice statement]

The use of AEDs by trained lay and professional responders is recommended to increase survival rates in those who have cardiac arrest.[2]

3 Public Access to AEDs

ANZCOR recommends the implementation of public access AED programs for out-of-hospital cardiac arrest.[4,5] [2020 CoSTR, strong recommendation, low-certainty evidence] Issues such as early detection of cardiac arrest, optimising AED availability, AED signage, novel delivery methods, public awareness, device registration, mobile apps for AED retrieval and personal access defibrillation should be considered as part of all PAD programs.[4,5] [Good practice statement] ANZCOR suggests that AED signage is consistent with international recommendations. [Good practice statement]

Deployment of home AEDs for high-risk individuals who do not have an implantable cardioverter defibrillator (ICD) is safe and feasible, and may be considered on an individual basis, but has not been shown to change overall survival rates.[2] [Good practice statement] Use of AEDs in public settings

(airports, casinos, sports facilities, etc.) where witnessed cardiac arrest is likely to occur can be useful if an effective response plan is in place.[2] An AED can and should be used on pregnant women who are in cardiac arrest.

Use of AEDs is reasonable to facilitate early defibrillation in hospitals.[2] Studies to date have shown AEDs are effective in decreasing the time to first defibrillation during in-hospital cardiac arrest.[2]

4 Pad Placement

4.1 Pad placement - Adults

Effective pad placement ensures that a shock is delivered on an axis through the heart. Place pads on the exposed chest in an anterior-lateral position: one pad slightly below the collar bone on the person's right chest and one pad on the person's left side below the arm pit (Figure 1). Acceptable alternatives are the anterior-posterior position, where one pad is placed on the upper back between the shoulder blades and the other on the front of the chest (slightly to the left, if possible); and apex-posterior.[1,4]

In large-breasted individuals it is reasonable to place the left electrode pad lateral to the left breast to avoid breast tissue.[1,4,5] All pads have a diagram on the outer covering demonstrating the area suitable for pad placement.[1] [Good practice statement] Pad to skin contact is important for successful defibrillation. Rescuers may need to remove moisture or excessive chest hair prior to the application of pads but emphasis must be on minimizing delays in shock delivery.[1] [Good practice statement]

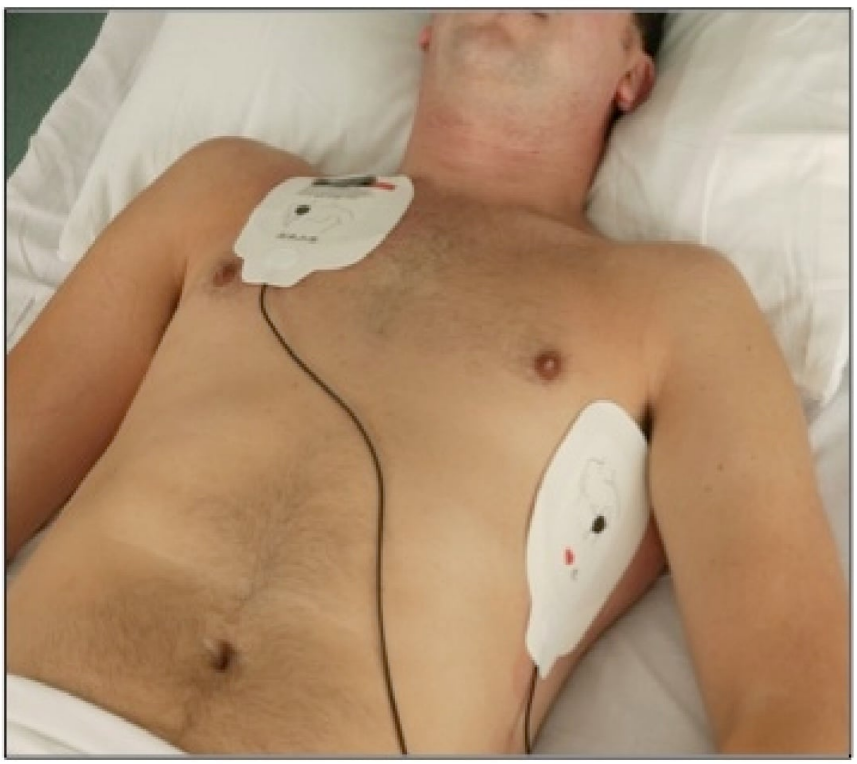


Figure 1: Anterior-lateral pad placement

Avoid placing pads over implantable devices. If there is an implantable medical device the defibrillator pad should be placed at least 8cm from the device.[1] Do not place AED electrode pads directly on top of a medication patch because the patch may block delivery of energy from the electrode pad to the heart and may cause small burns to the skin. Remove medication patches and wipe the area before attaching the electrode pad.[1]

4.2 Pad placement - Children and Infants

Standard adult AEDs and pads are suitable for use in children older than 8 years. Ideally, for those under 8 years (including infants < 1 year) paediatric pads and an AED with a paediatric capability should be used (see guideline 12.1). These pads also are placed as per the adult and the pads and come with a diagram of where on the chest they should be placed.[6]

If the AED does not have a paediatric mode or paediatric pads then it is reasonable to proceed with standard adult AED pads.[2] Ensure the pads do not touch each other on the child's chest.[6] Apply the pad firmly to the bare chest in the anterior-lateral position as shown for adults in Figure 1. If the pads are too large (a particular risk in younger children and infants) and there is a danger of pad-to-pad arcing, use the front-back position (antero-posterior): one pad placed on the upper back (between the shoulder blades) and the other pad on the front of the chest, if possible slightly to the left.[7]

5 Defibrillation Safety

Rescuers should follow the prompts: care should be taken not to touch the person during shock delivery. There are no reports of harm to rescuers from attempting defibrillation in wet environments.[2] [Good practice statement] In the presence of oxygen, there are no case reports of fires caused by sparking when shocks were delivered using adhesive pads.[2] [Good practice statement]

References

1. Sunde K, Jacobs I, Deakin CD, et al. Part 6: Defibrillation: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 201 0; **81** (1): e71-e85.
2. Soar J, Mancini ME, Bhanji F, et al. Part 12: Education, implementation, and teams: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2010; **81** (1): e288-e330.
3. Finn JC, Bhanji F, Lockey A, et al. Part 8: Education, implementation, and teams 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2015; **95** : e203-e24.
4. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and

Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation* 2020; **142** (16_suppl_1): S41-S91.
5. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2020; **156** : A35- A79.
6. de Caen AR, Kleinman ME, Chameides L, et al. Part 10: Paediatric basic and advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. *Resuscitation* 2010; **81** (1): e213- e59.
7. Biarent D, Bingham R, Eich C, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 6. Paediatric life support. *Resuscitation* 2010; **81** (10): 1364-88.

Further Reading

- ANZCOR Guideline 8 Cardiopulmonary Resuscitation
- ANZCOR Guideline 11.4 Electrical Therapy for Adult Advanced Life Support
- ANZCOR Guideline 12.6 Techniques in Paediatric Advanced Life Support

- Search date/s: ILCOR literature search details and dates are available on the CoSTR page of the ILCOR website (https://costr.ilcor.org) and the relevant CoSTR documents:
  - Automated External Defibrillator use in Drowning (BLS 856):? Scoping Review https://costr.ilcor.org/document/bls-856-automated-external-defibrillator-use-in-drowning-aed-use-tf-scoping-review
  - CPR Prior to Defibrillation (BLS): Systematic Review https://costr.ilcor.org/document/cpr-prior-to-defibrillation-tfsr-costr
  - Pad Size, Orientation and Placement (BLS): Scoping Review https://costr.ilcor.org/document/pad-size-orientation-and- placement - scoping-review
- Questions/PICOs: Are described in the CoSTR documents (https://costr.ilcor.org)
- Method: Mixed methods including ARC NHMRC methodology before 2017 and ILCOR GRADE methodology described in ILCOR publications since 2017.
- Principal reviewers: Julie Considine, Hugh Grantham
- Approved: April 2021
- Guideline superseded: January 2016

ANZCOR Guideline 8 - Cardiopulmonary Resuscitation (CPR)

Summary

Who does this guideline apply to?

This guideline applies to all persons who are unresponsive and not breathing normally.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders or first aid providers, first responders and health professionals, excluding those specifically trained in paediatric basic or advanced life support. Those trained in paediatric BLS and ALS should refer to guidelines 12.1 and 12.2, respectively.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. Rescuers must start CPR if the person is unresponsive and not breathing normally.
2. Bystander CPR should be actively encouraged.
3. Compression-to-ventilation ratio be 30:2 for all ages.
4. All rescuers perform chest compressions for all who are not breathing normally. Rescuers who are trained and willing to give rescue breaths are encouraged to do so.
5. Chest compressions should be provided at a rate of approximately 100 - 120 /min.
6. Rescuers should aim to minimise interruptions to chest compressions.

Guideline

1 What is Cardiopulmonary Resuscitation?

Cardiopulmonary resuscitation (CPR) is the technique of chest compressions combined with rescue breathing. The purpose of CPR is to temporarily maintain a circulation sufficient to preserve brain function until specialised treatment is available. Rescuers must start CPR if the person is unresponsive and not breathing normally.[1,2] Even if the person takes occasional gasps, rescuers should start CPR.[1,2] [Good practice statement] CPR should commence with chest compressions.[1-4] [2015/2020 CoSTR, weak recommendation, very-low-certainty evidence] and interruptions to chest compressions must be minimised.[3,4] [2015 CoSTR, weak recommendation, low-certainty evidence]

1.1 Bystander CPR

Early high-quality CPR saves lives.[1-4] ANZCOR recommends that CPR is started for presumed cardiac arrest without concerns of harm to persons not in cardiac arrest.[1-4] [CoSTR 2015/2020, strong recommendation, very-low-certainty evidence]

2 Compression-to -Ventilation Ratio

ANZCOR suggests a compression-ventilation ratio of 30:2 compared with any other compression-ventilation ratio in people in cardiac arrest.[1] [CoSTR 2015, weak recommendation, low-certainty evidence] Compressions must be paused to allow for ventilations.

3 Steps of Resuscitation

Initial steps of resuscitation are:

DRSABCD

1. DANGERS Check for danger (assess and manage risks to the rescuer and others)
2. RESPONSIVENESS Check for response (if unresponsive)
3. SEND Send for help
4. AIRWAY Open the airway
5. BREATHING Check breathing (if not breathing / abnormal breathing)
6. CPR Start CPR (give 30 chest compressions followed by two breaths)
7. DEFIBRILLATION Attach an Automated External Defibrillator (AED) as soon as available and follow the prompts.

3.1 Chest Compressions

All rescuers should perform chest compressions for all those who are unresponsive and not breathing normally. [CoSTR 2015, strong recommendation, very-low-certainty evidence] ANZCOR suggests that those who are trained and

willing to give breaths do so for all persons in cardiac arrest. [CoSTR 2015, weak recommendation, very-low-certainty evidence]

When rescuers perform chest compressions they should be at a rate of approximately 100 - 120 /min.[3] [CoSTR 2015/2020, strong recommendation, very-low-certainty evidence] (See ANZCOR Guideline 6 Compressions)

3.2 Minimise Interruptions to Chest Compressions

CPR should not be interrupted to check for response or breathing. ANZCOR places a high priority on minimising interruptions to chest compressions. We seek to achieve this overall objective by balancing it with the practicalities of delivering 2 effective breaths between cycles of chest compressions to the patient without an advanced airway.[1] [Good practice statement]

3.3 Multiple Rescuers

When more than one rescuer is available ensure:

- that an ambulance has been called
- all available equipment has been obtained (e.g. AED).

3.4 Duration of CPR

The rescuer should continue cardiopulmonary resuscitation until any of the following conditions have been met:[1]

- the person responds or begins breathing normally
- it is impossible to continue (e.g. exhaustion)
- a health care professional arrives and takes over CPR
- a health care professional directs that CPR be ceased.

[Good practice statement]

4 Risks

CPR should be initiated for presumed cardiac arrest without concerns of harm to persons not in cardiac arrest.[1] [CoSTR 2015, strong recommendation, very-low-certainty evidence] In making this recommendation, ANZCOR places a higher value on the survival benefit of CPR initiated by rescuers for persons in cardiac arrest against the low risk of injury in persons not in cardiac arrest.[1] [CoSTR 2015, values and preferences statement]

The risk of disease transmission during training and actual CPR performance in normal non pandemic circumstances is very low.[3] A systematic review found no reports of transmission of hepatitis B, hepatitis C, human immunodeficiency virus (HIV) or cytomegalovirus during either training or actual CPR when high-risk activities, such as intravenous cannulation were not performed.[3] If available, the use of a barrier device during rescue breathing is reasonable.[3] [Good practice statement] After resuscitating a person, the rescuer should reassess and re-evaluate for resuscitation-related injuries.[3] [Good practice statement]

References

1. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: 2020 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Circulation 2020;142:S41-S91.
2. Olasveengen TM, Mancini ME, Perkins GD, et al. Adult Basic Life Support: International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2020;156:A35-A79.
3. Perkins GD, Travers AH, Berg RA, et al. Part 3: Adult basic life support and automated external defibrillation. 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Resuscitation 2015;95:e43-e69.
4. Travers AH, Perkins GD, Berg RA, et al. Part 3: Adult Basic Life Support and Automated External Defibrillation 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Circulation 2015;132:S51-S83.

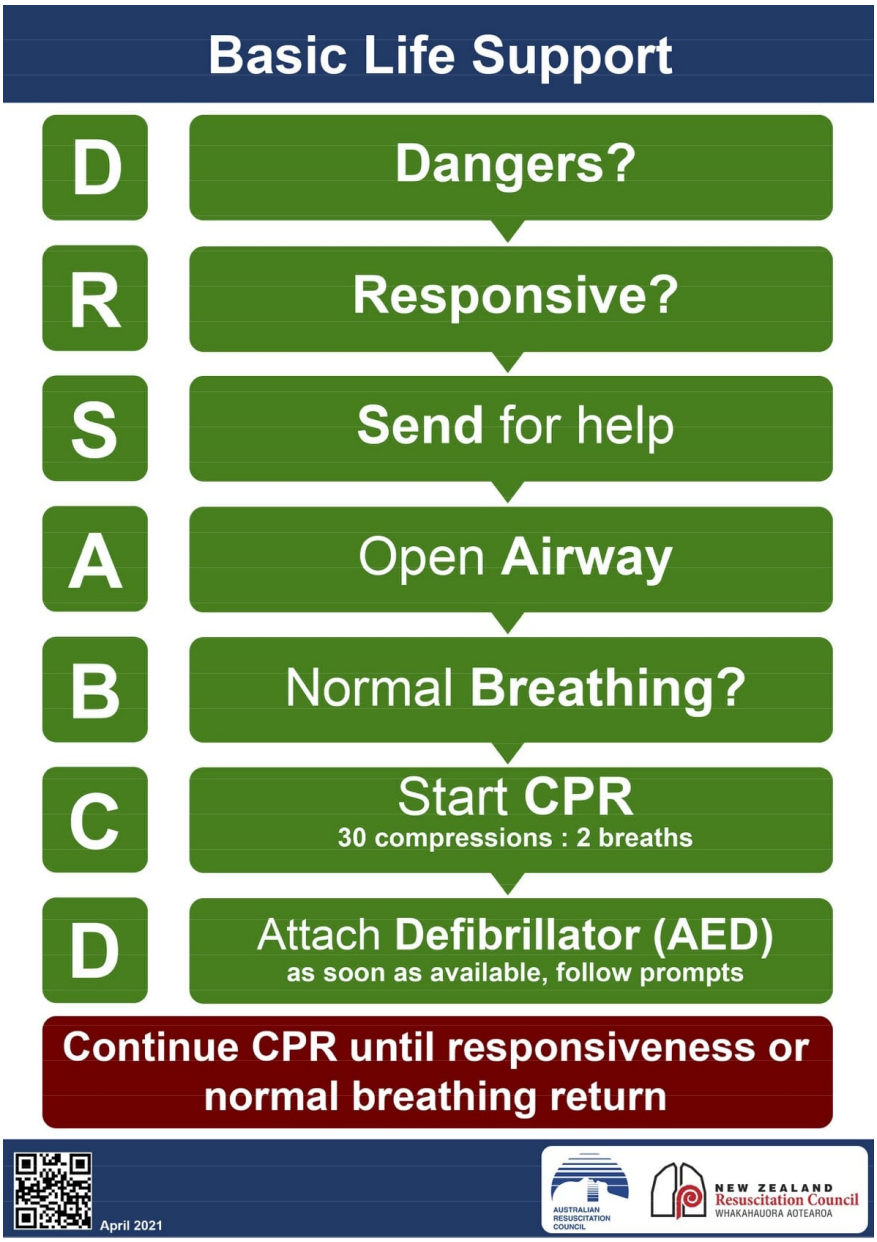
Further Reading

ANZCOR Guideline 2 Priorities in an Emergency ANZCOR Guideline 3 Unconsciousness ANZCOR Guideline 4 Airway ANZCOR Guideline 5 Breathing ANZCOR Guideline 6 Compressions ANZCOR Guideline 7 External Automated Defibrillation (AED) in Basic Life Support (BLS) ANZCOR Guideline 9.3.2 Resuscitation of the Drowning Victim

About this Guideline

- Search date/s: ILCOR literature search details and dates are available on the CoSTR page of the ILCOR website (https://costr.ilcor.org) and the relevant CoSTR documents:
  - Chest Compression rate (BLS): Scoping Review https://costr.ilcor.org/document/chest-compression-rate- scoping-review
  - CPR: Chest Compression to Ventilation Ration - Adult (BLS): Systematic Review https://costr.ilcor.org/document/cpr-chest-compression-to-ventilation-ratio-adult
  - CPR Prior to Defibrillation (BLS): Systematic Review https://costr.ilcor.org/document/cpr-prior-to-defibrillation-tfsr- costr
  - CPR: Compression to Ventilation Ratio - Bystander - Adult (BLS): Systematic Review https://costr.ilcor.org/document/cpr-compression-to-ventilation-ratio-bystander-adult
  - CPR: Compression to Ventilation Ratio - Dispatch Assisted- Adult (BLS): Systematic Review https://costr.ilcor.org/document/cpr-compression-to-ventilation-ratio-dispatch-assisted-adult

    - Feedback for CPR Quality (BLS): Systematic Review https://costr.ilcor.org/document/feedback-for-cpr-quality-bls- 361-tf-sr
    - Starting CPR (ABC vs CAB) (BLS): Systematic Review https://costr.ilcor.org/document/starting-cpr-abc-vs-cab-tfsr- costr
- Questions/PICOs: Are described in the CoSTR documents (https://costr.ilcor.org)
- Method: Mixed methods including ARC NHMRC methodology before 2017 and ILCOR GRADE methodology described in ILCOR publications since 2017.
- Principal reviewers: Julie Considine, Hugh Grantham
- Approved: April 2021
- Guideline superseded: January 2016



ANZCOR Guideline 9.1.1 - First Aid for Management of Bleeding

Summary

Who does this guideline apply to?

This guideline applies to adults, children and infants.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. Firm pressure on or around the wound is the most effective way to stop most bleeding. [Good Practice Statement]
2. In life-threatening bleeding, control of bleeding takes priority over airway and breathing interventions. [Good Practice Statement]
3. We suggest to use an arterial tourniquet for life-threatening limb bleeding that is not controlled by direct wound pressure.[1] [CoSTR 2020, weak recommendation, very low certainty of evidence]

Abbreviations

ANZCOR: Australian and New Zealand Committee on Resuscitation CoSTR: Consensus on Science with Treatment Recommendations (from International Liaison Committee on Resuscitation - ILCOR) CPR: Cardiopulmonary Resuscitation

Guideline

1 External Bleeding

The use of pressure on or around the wound is usually the fastest, easiest and most effective way to stop external bleeding.[1,2,3,4] [Good Practice Statement] Other methods should be used if direct pressure alone does not control severe bleeding. The aim is to stop further bleeding whilst waiting for help to arrive. There is no evidence that elevating a bleeding part will help control bleeding[1,5] and there is the potential to cause more pain or injury.

Bleeding should be managed as severe, life-threatening bleeding in the following situations:

- amputated or partially amputated limb above wrist or ankle
- shark attack, propeller cuts or similar major trauma to any part of the body
- bleeding not controlled by local pressure
- bleeding with signs of shock, i.e. pale and sweaty plus pulse rate >100, or capillary refill > 2 sec and/or decreased level of consciousness

1.1 Management

- Use standard precautions (e.g. gloves, protective glasses) if readily available.
- Management of all bleeding begins with application of pressure on or around the wound.
- If bleeding is severe or life-threatening, controlling the bleeding takes priority over airway and breathing interventions. Lie the person down, apply pressure and call for an ambulance.
- If there is severe, life threatening bleeding from a limb, not controlled by pressure, we suggest to apply an arterial tourniquet above the bleeding point, if trained in its use and one is available.[1] [CoSTR 2020: weak recommendation, very low certainty of evidence]
- If there is severe, life-threatening bleeding from a wound site not suitable for tourniquet, or from a limb when a tourniquet is not available or has failed to stop the bleeding, we suggest to apply a haemostatic dressing, if trained in its use and one is available.[1,5] [CoSTR 2020: weak recommendation, low quality evidence]
- For the majority of non-life-threatening cases, first aiders should follow the sequence of DRSABCD, where control of bleeding follows establishing airway and commencing CPR if required.
- If the person is unresponsive and not breathing normally, follow the Basic Life Support Flowchart. [Refer to ANZCOR Guideline 8]

1.2 Direct Pressure Method

Where the bleeding point is identified, the rescuer, a bystander or the injured person should control bleeding by:

- Applying firm, direct pressure sufficient to stop the bleeding. Pressure can be applied using hands or a pad over the bleeding point.
- If bleeding continues, apply a second pad and a tighter bandage over the wound. If bleeding still continues, check that the pad and bandage are correctly applied, directly over the bleeding. If not, it may be necessary to remove the pad(s) to ensure that a specific bleeding point has not been

missed. Applying firmer pressure, only using 1 to 2 pads over a small area, will achieve greater pressure over the bleeding point than continuing to layer up further pads.

To assist in controlling bleeding, where possible:

- Advise the person to lie down and remain still
- Restrict movement by immobilizing a bleeding limb

1.3 Embedded Objects

If there is an obvious embedded object causing bleeding, use pressure around the object. [Good Practice Statement]

- Do not remove the embedded object because it may be plugging the wound and restricting bleeding.
- Apply padding around or on each side of the protruding object, with pressure over the padding.

Pressure application methods may be insufficient to control bleeding. It may still be necessary to use other measures including an arterial tourniquet or haemostatic dressings.

1.4 Arterial tourniquet

- Arterial tourniquets should only be used for life-threatening bleeding from a limb, where the bleeding cannot be controlled by direct pressure. Ideally, a tourniquet **should not** be applied over a joint or wound, and **must not** be covered up by any bandage or clothing.
- Commercially manufactured windlass tourniquets such as those based on military designs are more effective than improvised tourniquets[1]. An example of a military tourniquet is shown in Fig 1 below. Effective use of commercial tourniquets is optimal when first aid providers are trained in proper application techniques.
- All arterial tourniquets should be applied in accordance with the manufacturer's instructions (or 5 cm above the bleeding point if no instructions) and tightened until the bleeding stops.
- If a tourniquet does not stop the bleeding its position and application must be checked. Ideally the tourniquet is not applied over clothing nor wetsuits and is applied tightly, even if this causes local discomfort.
- If bleeding continues, a second tourniquet (if available) should be applied to the limb, preferably above the first.
- If a correctly applied tourniquet(s) has failed to control the bleeding consider using a haemostatic dressing in conjunction with the tourniquet.[1,5] [Good Practice Statement]
- An elastic **venous** tourniquet (designed to assist drawing blood samples or inserting intravenous cannulae) is **not** suitable for use as an arterial tourniquet.
- Improvised tourniquets are unlikely to stop all circulation to the injured limb without risk of tissue damage. Improvised tourniquets which do not stop all circulation can increase bleeding. Nonetheless, in the context of life-

threatening bleeding, an improvised tourniquet is likely to be better than no tourniquet. Tourniquets, ideally of a similar broad width to commercial types, can be improvised using materials from a first aid kit (e.g. triangular bandage, elastic bandage) from clothing, a surfboard leg rope or other available similar items. Improvised tourniquets should be tightened by twisting a rod or stick under the improvised tourniquet band, similar to the windlass in commercial tourniquets.

- The time of tourniquet application must be noted and communicated to emergency/paramedic personnel. Once applied, the person requires urgent transfer to hospital and the tourniquet should not be removed until the person receives specialist care.

Figure 1: Combat Application Tourniquet

1.5 Haemostatic dressings

- Haemostatic dressings are impregnated with agents that help stop bleeding. The haemostatic dressings included in the CoSTR 2015 recommendations contained the products kaolin and chitosan.[6] They are commonly used to control bleeding in the surgical and military settings but their use in the civilian, non-surgical setting is becoming more common. An example is shown in Fig 2 below.
- When available and the first aid provider is trained in their use, we suggest that haemostatic dressings are of most value in the following situations [CoSTR 2015: weak recommendation, very low quality evidence]:[5]
  - Severe, life-threatening bleeding not controlled by wound pressure, from a site not suitable for tourniquet use.
  - Severe, life-threatening bleeding from a limb, not controlled by wound pressure, when the use of a tourniquet(s) alone has not stopped the bleeding, or a tourniquet is not available.
- Haemostatic dressings must be applied as close as possible to the bleeding point, held against the wound using local pressure (manually initially) then

held in place with the application of a bandage (if available). Haemostatic dressings should be left on the bleeding point until definitive care is available.

The need to control the bleeding is paramount. The risks associated with the first aid use of tourniquets and haemostatic dressings are less than the risk of uncontrolled severe, life-threatening bleeding. These adjuncts provide temporary bleeding control and rapid transfer to hospital remains critically important.

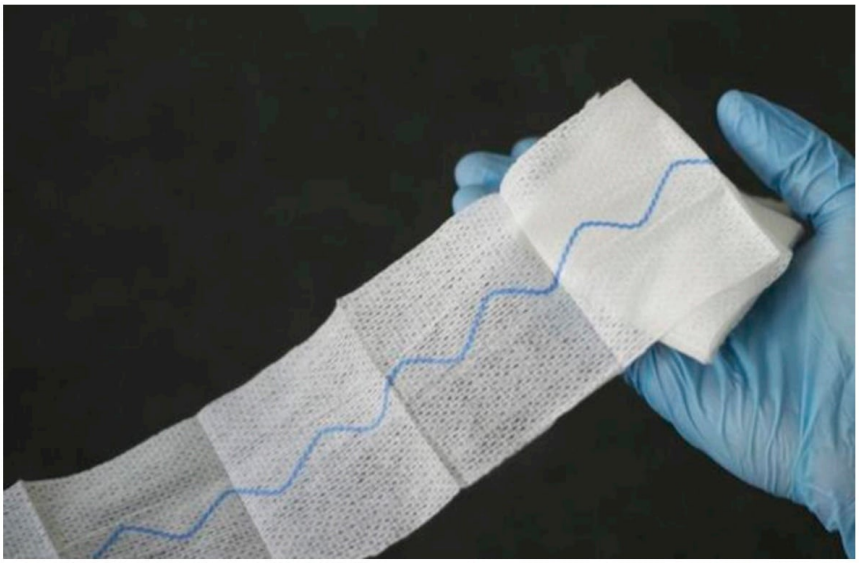
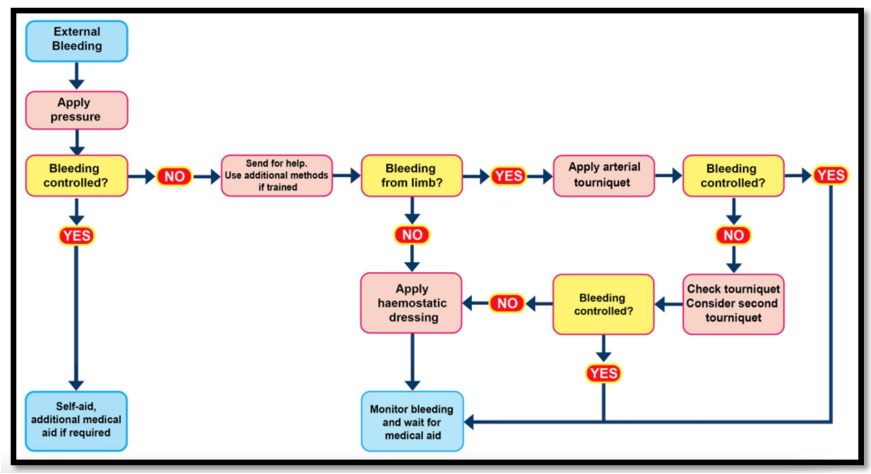


Figure 2: Kaolin impregnated gauze (an example of a haemostatic dressing)

1.6 Flow Chart for First Aid Control of External Bleeding



2 Internal Bleeding

2.1 Recognition

Internal bleeding may be difficult to recognise, but should always be suspected where there are symptoms and signs of shock. [Refer to ANZCOR Guideline 9.2.3]

Internal bleeding includes bruising, locally contained bleeding (e.g. an 'egg on the head') and the bleeding associated with injury or disease of organs in the abdomen or chest, as well as fractures. Severe bleeding may also occur from complications of pregnancy.

Symptoms and signs may include:

- pain, tenderness or swelling over or around the affected area
- the appearance of blood from a body opening, eg:
  - bright red and/or frothy blood coughed up from the lungs
  - vomited blood which may appear bright red or as dark brown 'coffee grounds'
  - blood-stained urine
  - vaginal bleeding or bleeding from the penis
  - rectal bleeding which may be bright red or black and 'tarry'
- shock in the case of severe bleeding

2.2 Management

Severe internal bleeding is life-threatening and requires urgent treatment in hospital.

- Send for an ambulance.
- Lie the person down
- Treat shock [Refer to ANZCOR Guideline 9.2.3]

2.3 Closed Bleeding in a Limb

- If there is bruising to a limb and no external bleeding, we suggest to use pressure and a cold pack if available.[5] [CoSTR 2015: weak recommendation, low quality evidence]
- If closed bleeding in a limb is causing severe swelling or pain, or the person is showing signs of shock [Refer to ANZCOR Guideline 9.2.3], send for an ambulance [Good Practice Statement]

3 Nose Bleed (Epistaxis)

For a nose bleed:

- Pressure must be applied equally to both sides of the nose, over the soft part below the bony bridge (usually between the thumb and index finger).
- The person should lean with the head forward to avoid blood flowing down the throat.
- Encourage the person to spit out blood rather than swallow it as swallowed blood irritates the stomach, and causes vomiting which can worsen the bleeding.
- The person should remain seated at total rest for at least 10 minutes. On a hot day or after exercise, it might be necessary to maintain pressure for at least 20 minutes.
- If bleeding continues for more than 20 minutes seek medical assistance.

4 Management of All Severe Bleeding

- Call for an ambulance
- Reassure the person
- Assist the person into a position of comfort, preferably lying down
- Keep the person warm
- Monitor the vital signs at frequent intervals
- Administer oxygen if available and trained to do so [Refer to ANZCOR Guideline 9.2.10]
- **Do not** give any food or drink orally, including medications
- Treat shock [Refer to ANZCOR Guideline 9.2.3]
- If the person is unresponsive and not breathing normally, follow the Basic Life Support Flowchart [Refer to ANZCOR Guideline 8]

References

1. Nathan P. Charlton, Janel M. Swain, Jan L. Brozek, Maja Ludwikowska, Eunice Singletary, David Zideman, Jonathan Epstein, Andrea Darzi, Anna Bak, Samer Karam, Zbigniew Les, Jestin N. Carlson, Eddy Lang & Robby Nieuwlaat (2020): Control of Severe, Life-Threatening External Bleeding in the Out-of-Hospital Setting: A Systematic Review, Prehospital Emergency Care, 2020, DOI: 10.1080/10903127.2020.
2. Walker S.B., Cleary S., Higgins M. Comparison of the FemoStop device and manual pressure in reducing groin puncture site complications following coronary angioplasty and coronary stent placement. International Journal of Nursing Practice. Dec. 2001. **7** (6):366-75.
3. Simon A., Bumgarner B., Clark K., Israel S. Manual versus mechanical compression for femoral artery hemostasis after cardiac catheterization. American Journal of Critical Care. Jul 1998. **7** (4):308-13.
4. Naimer S.A., Chemla F. Elastic adhesive dressing treatment of bleeding wounds in trauma victims. American Journal of Emergency Medicine. 2000. **18** :816-819.
5. Zideman, D. A., Singletary, E. M., De Buck, E.,et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. Resuscitation , 95, e225. http://www.cprguidelines.eu/assets/downloads/costr/S0300-9572(15)00368-8_main.pdf00368-8_main.pdf) Accessed 21/11/
6. Devlin JJ, Kircher S, Kozen BG et al. Comparison of Chitoflex®, CELOX™ , and QuickClot™ in Control of Hemorrhage. 2011. Journal of Emergency Medicine. 2011;41(3), 237- 245

Further Reading

- ANZCOR Guideline 9.2.3 Shock
- ANZCOR Guideline 9.1.4 Head Injury
- ANZCOR Guideline 9.2.10 The Use of Oxygen in Emergencies

About this Guideline

- Search date/s: ANZCOR search: October 2020
- Question/PICO: Population: Adults and children with severe, life- threatening external bleeding in an out-of-hospital setting. Bleeding from both compressible and non- compressible external sites were included. Nasal and oral bleeding were excluded.
- Intervention: All bleeding control methods applicable for use by trained or untrained first aid providers including manufactured or improvised tourniquets, hemostatic dressings or agents, cryotherapy, direct manual pressure, pressure points, pressure dressings or bandages, or elevation of the injured area.
- Comparisons: Direct manual pressure alone or any other management technique listed in Intervention, if a comparison was available. Outcomes: Outcomes were prioritized for importance a priori, as per the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) approach (Table 1).
- Study Designs: Randomized controlled trials (RCTs) and non- randomized comparative studies were included. If there were no or limited comparative studies, then case series of 4 or more subjects and simulation studies were also included. Conference abstracts and trial protocols were excluded. All languages were included where an English abstract was available.
- Method: Systematic Review (ILCOR First Aid Task Force, CoSTR) published April 20
- Primary reviewers: Finlay Macneil
- Other consultation: Nil
- Worksheet: See https://www.ilcor.org/
- Approved: April 2021
- Guidelines superseded: ANZCOR Guideline 9.1.1 - July 2017

ANZCOR GUIDELINE 9.1.3 – BURNS

Guideline

Who does this guideline apply to?

This guideline applies to adult, child and infant victims.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid providers.

1 Definition

A burn is an injury caused by heat, cold, electricity, chemicals, gases, friction and radiation (including sunlight). A significant burn for the purpose of this document includes:

- burns greater than 10% of total body surface area (TBSA)
- burns of special areas—face, hands, feet, genitalia, perineum, and major joints
- full-thickness burns greater than 5% of TBSA
- electrical burns
- chemical burns
- burns with an associated inhalation injury
- circumferential burns of the limbs or chest
- burns in the very young or very old
- burns in people with pre-existing medical disorders that could complicate management, prolong recovery, or increase mortality
- burns with associated trauma.

All infants and children with burns should be medically assessed.

2 Initial Approach

- Ensure safety for rescuers, bystanders and the victim.
- Do not enter a burning or toxic atmosphere without appropriate protection.
- Stop the burning process:
  - Stop, Drop, Cover and Roll

    - Smother any flames with a blanket.
- Move away from the burn source to a safe environment as soon as possible.
- Assess the adequacy of airway and breathing.
- Check for other injuries.
- If safe, and if trained to do so, give oxygen to all victims with smoke inhalation or facial injury, following The Use of Oxygen in Emergencies (ANZCOR Guideline10.4).
- Call for an ambulance.

The aims of first aid treatment of burns should be to stop the burning process, cool the burn and cover the burn. This will provide pain relief and minimize tissue loss.

3 Heat/Thermal/Contact Burns

- These include flame, scald, blast (hot gas), inhalation injury and direct heat contact.
- IMMEDIATELY cool burns with cool running water (CoSTR 2015: strong recommendation/low quality evidence)^1. ANZCOR recommends cooling for 20 minutes^2.
- If possible, remove all rings, watches, jewellery or other constricting items from the affected area without causing further tissue damage.
- Remove wet, non-adherent clothing as clothing soaked with hot liquids retains heat.
- Cover the burnt area with a loose and light non-stick dressing, preferably clean, dry, lint free (non-fluffy) material e.g. plastic cling film.
- Cover unburnt areas and keep the rest of the victim warm to reduce the risk of hypothermia.
- Where feasible elevate burnt limbs to minimise swelling.

DO NOT peel off adherent clothing or burning substances.

DO NOT use ice or ice water to cool the burn as further tissue damage may result.

DO NOT break blisters.

DO NOT apply lotions, ointments, creams or powders other than hydrogel.

3.1 Inhalation Burn

Always assume inhalation injury if there are burns to the face, nasal hairs, eyebrows or eyelashes, or if there is evidence of carbon deposits in the nose or mouth. Coughing of black particles in sputum, hoarse voice and/or breathing difficulties may indicate damage to the airway.

An inhalation burn should be suspected when an individual is trapped in an enclosed space for some time with hot or toxic gas, steam or fumes produced by a fire, chemicals etc. An inhalation injury may result from irritant gases such as ammonia, formaldehyde, chloramines, chlorine, nitrogen dioxide and phosgene. These agents produce a chemical burn and an inflammatory response.

Do not assume the burn victim is stable following an inhalation injury simply because the victim is breathing, talking and able to get up. Some agents produce delayed pulmonary inflammation which may develop up to 24 hours later.

- Remove to fresh air.
- Assess and manage the airway (ANZCOR Guideline 4).
- Give oxygen if available and trained to do so, following The Use of Oxygen in Emergencies (ANZCOR Guideline 10.4).
- Call for an ambulance.

3.2 Electrical Burns

Electrical burns, including lighting strike, are often associated with other injuries including involvement of the cardiac and respiratory systems, loss of consciousness and trauma.

The priorities in the management of the electric shock victim are to:

- Isolate/turn off the power supply without touching the victim
- Commence cardiopulmonary resuscitation if required following the Basic Life Support Flow Chart (ANZCOR Guideline 8)
- Cool burns if safe to do so, with cool running water for 20 minutes
- Give oxygen if available and trained to do so, following The Use of Oxygen in Emergencies (ANZCOR Guideline 10.4)
- Call an ambulance.

Lightning may cause cardiac arrest.

- Commence cardiopulmonary resuscitation if required following the Basic Life Support Flow Chart. (ANZCOR Guideline 8).

3.3 Radiation Burns

Radiation burns may be caused by solar ultraviolet radiation (sunburn), welder’s arc, lasers, industrial microwave equipment and nuclear radiation.

- Cover radiation burns with a clean, dry dressing to prevent infection.

3.4 Chemical Burns

Government regulations on hazardous substances and work, health and safety require the manufacturer or importer of a hazardous chemical to prepare a safety data sheet (SDS) for the chemical3,4. A supplier must provide a SDS to a workplace at the time of first supply or upon request. These SDS’s provide first aid information specific to each chemical and include information relevant to eye contact, skin contact, inhalation and ingestion.

The aim of first aid for chemical burns is not to cool the burn but to dilute the chemical.

- Avoid contact with any chemical or contaminated material, using appropriate personal protection equipment.

- Remove the victim to a safe area.
- Remove the chemical and any contaminated clothing and jewellery as soon as practical.
- Brush powdered chemicals from the skin.
- Without spreading the chemical to unaffected areas, IMMEDIATELY run cool running water directly onto the area for one hour or until the stinging stops.
- Apply a non-adherent dressing even if no burn mark is obvious.
- If chemical enters the eye, open and flush the effected eye(s) thoroughly with water (CoSTR 2015: weak recommendation/very low quality evidence)^1 for as long as tolerated and refer the victim for urgent medical attention. If only one eye is affected then flush with the head positioned so as the affected eye is down to avoid spread of the chemical to the unaffected eye. The flushing of the eye is more important than immediate transfer for medical care.
- Refer to instructions on the container for further specific treatment.
- If available, in hard copy or on the internet, refer to Safety Data Sheets (SDS) for specific treatment.
- Call the Poisons Information Centre5,6 for further advice.

DO NOT attempt to neutralise either acid or alkali burns, because this will increase heat generation which may cause more damage.

DO NOT apply cling wrap or hydrogel dressings to chemical burns.

Acknowledgements

Australian and New Zealand Burns Association (ANZBA) http://anzba.org.au/ Accessed 20/11/

References

1. Zideman, D. A., Singletary, E. M., De Buck, E.,et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. *Resuscitation, 95* , e225. http://www.cprguidelines.eu/assets/downloads/costr/S0300-9572(15)00368-8_main.pdf00368-8_main.pdf) Accessed 19/11/
2. http://anzba.org.au/care/first-aid/ Accessed 19/11/
3. http://www.safeworkaustralia.gov.au/sites/swa/whs-information/hazardous-chemicals/sds/pages/sds Accessed 19/11/
4. http://www.epa.govt.nz/hazardous-substances/using-storing/pages/safety-data-sheets.aspx Accessed 19/11/
5. New Zealand Poison Information phone: 0800 POISON (0800 764 766)
6. Australian Poisons Information Centres phone: 13 11 26

Further Reading

ANZCOR Guideline 2 Managing an Emergency

ANZCOR Guideline 8 Cardiopulmonary Resuscitation

ANZCOR Guideline 9.2.3 Shock

ANZCOR Guideline 9.3.3 Hypothermia: First Aid and Management

ANZCOR Guideline 9.5.1 Emergency Management of a Victim who has Been Poisoned

ANZCOR Guideline 10.4 TheUse of Oxygen in Emergencies

ANZCOR GUIDELINE 9.1.4 – HEAD INJURY

Guideline

Who does this guideline apply to?

This guideline applies to adult, child and infant victims.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid providers.

1 Introduction

Head injury may be caused by a number of mechanisms including, falls, assaults, motor vehicle crashes, sporting injuries and, less commonly, penetrating injuries. A victim may sustain a significant head injury without loss of consciousness or loss of memory (amnesia). Therefore, loss of consciousness or memory loss should not be used to define the severity of a head injury or to guide management.^1

The initial first aid for a victim with head injury includes assessing and managing the airway and breathing, whilst caring for the neck until expert help arrives.

There is insufficient evidence to support or refute the use by first aiders of simplified concussion scoring systems such as the Sport Concussion Assessment Tool (SCAT), the Glasgow Coma Scale (GCS) or Alert, Voice, Pain, Unresponsive (AVPU) versus standard first aid without a scoring system. (CoSTR 2015)^2 The serious consequences of not recognising concussion in the first aid environment warrants advising all victims who have sustained a head injury, regardless of severity, to seek assessment by an health care professional or at a hospital.

2 Recognition

A brain injury should be suspected if the victim has a reported or witnessed injury, has signs of injury to the head or face such as bruises or bleeding, or is found in a confused or unconscious state. A victim may have a brain injury without external

signs of injury to the head or face. Serious problems may not be obvious for several hours after the initial injury.

3 Management

- **Call an ambulance** if there has been a loss of consciousness or altered consciousness at any time, no matter how brief.
- A victim who has sustained a head injury, whether or not there has been loss of consciousness or altered consciousness, should be assessed by a health care professional.

- Check for response: an unconscious victim should be managed according to ANZCOR Guideline 3.
- Ensure that the airway is clear (ANZCOR Guideline 4).
- Protect the neck whilst maintaining a clear airway (ANZCOR Guideline 9.1.6).
- Identify and control any significant bleeding with direct pressure if possible (ANZCOR Guideline 9.1.1).

All victims who appear to have suffered a head injury (including a minor head injury) should be assessed by a health care professional before continuing with sport or other activity.

If the victim is unresponsive and not breathing normally, commence resuscitation following the Basic Life Support Flowchart (ANZCOR Guideline 8).

References

1. Reed D. Adult Trauma Clinical Practice Guidelines: Initial Management of Closed Head Injury in Adults, 2nd Ed. 2011. NSW Institute of Trauma & Injury Management: North Sydney. http://www.itim.nsw.gov.au/images/3/3d/Closed_Head_Injury_CPG_2nd_Ed_Full_documen t.pdf Accessed 19/11/
2. Zideman, D. A., Singletary, E. M., De Buck, E.,et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. Resuscitation, 95 , e225. http://www.cprguidelines.eu/assets/downloads/costr/S0300-9572(15)00368-8_main.pdf00368-8_main.pdf) Accessed 19/11/

Further Reading

ANZCOR Guideline 2 Managing an Emergency

ANZCOR Guideline 3 Recognition and First Aid Management of the Unconscious Victim

ANZCOR Guideline 4 Airway

ANZCOR Guideline 5 Breathing

ANZCOR Guideline 8 Cardiopulmonary Resuscitation

ANZCOR Guideline 9.1.6 Management of Suspected Spinal Injury

ANZCOR Guideline 9.2.3 Shock

ANZCOR Guideline 9.1.5 - First Aid Management of Harness Suspension Trauma

Summary

Who does this guideline apply to?

This guideline applies to adults, adolescents and children

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. Send for an ambulance [Good Practice Statement]
2. Rescue the person and place in a lying position as soon as it is safe to do so [Good Practice Statement]

Abbreviations

ANZCOR: Australian and New Zealand Committee on Resuscitation

Guideline

1 Introduction

Suspension trauma, or orthostatic shock, has been reported to affect people who are suspended within a body harness for a prolonged period of time (more than 10 min).[1,2,3] It may result in loss of consciousness or death. This is thought to occur as a result of low blood pressure due to blood pooling in the legs combined with an increase in the activity of a part of the nervous system that slows the heart (vagal tone).[3]

2 Recognition

The signs and symptoms of suspension trauma are the same as shock. Harness suspension trauma should be considered where the person has been suspended by a harness, and are exhibiting any of the following:[4]

- feeling faint or dizzy
- breathlessness
- sweating
- looking pale
- nausea
- low blood pressure
- loss of responsiveness

3 Management

- Call for an ambulance.
- If safe to do so, free the person from the harness.
- If not responding, manage as per ANZCOR Basic Life Support flow chart [Refer to ANZCOR Guideline 8]
- Rest the responding person in a position of comfort, ideally lying down, and provide reassurance.
- Loosen or remove harness.
- Administer oxygen if available.
- Look for and manage any associated injuries, particularly for those who may have fallen or been electrocuted.
- Monitor level of response and breathing at frequent intervals.

> Some agencies have previously recommended that those with suspension trauma are maintained in a sitting position and avoid lying flat for 30 minutes[5]. This review has found no evidence to support this practice and it may be harmful.

References

1. Turner N.L., Wassell J.T., Whisler R., Zwiener J. Suspension tolerance in a full-body safety harness, and a prototype harness accessory. [Evaluation Studies. Journal Article] Journal of Occupational & Environmental Hygiene. **5** (4):227-31, 2008.
2. Lee C., Porter K.M. Suspension trauma. [Journal Article. Review. Emergency Medicine Journal. **24** (4):237-8, 2007.
3. Rauch S, Schenk K, Strapazzon G, Dal Cappello T, Gatterer H, Palma M, Erckert M, Oberhuber L, Bliemsrieder B, Brugger H, Paal P. Suspension syndrome: a potentially fatal vagally mediated circulatory collapse - an experimental randomized crossover trial. European journal of applied physiology. 2019 Jun;119(6):1353-65.
4. Adish A., Robinson L., Codling A., Harris-Roberts J., Lee C., Porter K. Evidence based review of the current guidance on first aid measures for

suspension trauma. Health and Safety Executive. Research Report RR708. May 2009. http://www.hse.gov.uk/research/rrpdf/rr708.pdf
5. Raynovich B, Rwaili FT and Bishop P. (2009). Dangerous suspension. Understanding suspension syndrome & prehospital treatment for those at risk. JEMS : a journal of emergency medical services. 34(8):44-51, 53;

Further Reading

- ANZCOR Guideline 3 Recognition and First Aid Management of the Unconscious Person
- ANZCOR Guideline 4 Airway
- ANZCOR Guideline 8 Cardiopulmonary Resuscitation
- ANZCOR Guideline 9.1.6 Management of Suspected Spinal Injury
- ANZCOR Guideline 9.2.3 Shock

About this Guideline

- Search date/s: January 2020
- Question/PICO : For people suspended in a harness for a prolonged period awaiting rescue, what interventions are effective in the prevention of poor clinical outcomes
- Method: Scoping literature review
- Primary reviewers: Finlay Macneil
- Other consultation: Kevin Nation
- Worksheet: https://resus.org.au/download/worksheets/worksheets_to_support_guidelines/gl-9-1-5-harness-suspension-trauma-worksheet-final.pdf
- Approved: April 2021
- Guidelines superseded: ARC Guideline 9.1.5 - July 2009

ANZCOR GUIDELINE 9.1.6 – MANAGEMENT OF SUSPECTED SPINAL INJURY

Guideline

Who does this guideline apply to?

This guideline applies to adult, child and infant victims.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid providers. This guideline is equally applicable to healthcare professionals working in the pre-hospital setting.

1 Introduction

The spine is made up of 33 separate bones, known as vertebrae, extending from the base of the skull to the coccyx (tailbone). Each vertebra surrounds and protects the spinal cord (nerve tissue). Fractures or dislocations to the vertebral bones may result in injury to the spinal cord. The direct mechanical injury from the traumatic impact can compress or sever the nerve tissue. This is followed by secondary injury caused by ongoing bleeding into the spinal cord as well as continued swelling at the injured site and surrounding area.

The possibility of spinal injury must be considered in the overall management of all trauma victims. The risk of worsening the spinal injury in the prehospital period is probably less than previously thought, yet to minimise the extent of the secondary injury, caution must be taken when moving a victim with a suspected spinal injury.

Spinal injuries can occur in the following regions of the spine:

- the neck (cervical spine)
- the back of the chest (thoracic spine)
- the lower back (lumbar spine).

The cervical spine is most vulnerable to injury, which must be suspected in any victim with injuries above the shoulders. More than half of spinal injuries occur in the cervical region. Suspected spinal injuries of the neck, particularly if the victim is unconscious, pose a dilemma for the rescuer because correct principles of airway management often cause some movement of the cervical spine.

2 Recognition

The most common causes of spinal cord injury are:

- a motor vehicle, motor cycle or bicycle incident as an occupant, rider, or pedestrian
- an industrial accident (i.e. workplace)
- a dive or jump into shallow water or water with obstacles or being "dumped" in the surf
- a sporting accident (e.g. rugby, falling from a horse)
- a fall from greater than a standing height (e.g. ladder, roof)
- falls in the elderly population
- a significant blow to the head
- a severe penetrating wound (e.g. gunshot).

The symptoms and signs of a spinal injury depend on two factors: firstly the location of the injury and secondly, the extent of the injury – whether there is just bone injury or associated spinal cord injury, and whether the spinal cord injury is partial or complete. It will be difficult to elicit symptoms and signs in victims with an altered conscious state.

2.1 Symptoms

Symptoms of spinal injury include:

- pain in the injured region
- tingling, numbness in the limbs and area below the injury
- weakness or inability to move the limbs (paralysis)
- nausea
- headache or dizziness
- altered or absent skin sensation.

2.2 Signs

Signs of spinal injury include:

- head or neck in an abnormal position
- signs of an associated head injury
- altered conscious state
- breathing difficulties
- shock
- change in muscle tone, either flaccid or stiff
- loss of function in limbs
- loss of bladder or bowel control

- priapism (erection in males).

3 Management

The priorities of management of a suspected spinal injury are:

1. calling for an ambulance
2. management of airway, breathing and circulation
3. spinal care.

An awareness of potential spinal injury and careful victim handling, with attention to spinal alignment, is the key to harm minimisation.

3.1 The Conscious Victim

Tell the victim to remain still but do not physically restrain if unco-operative. Those with significant spinal pain will likely have muscle spasm which acts to splint their injury. Keep victim comfortable until help arrives. If it is necessary to move the victim from danger (e.g. out of the water, off a road), care must be taken to support the injured area and minimise movement of the spine in any direction. Ideally, only first aid providers or health care professionals trained in the management of spinal injuries, aided by specific equipment, should move the victim.

3.2 The Unconscious Victim

Airway management takes precedence over any suspected spinal injury. It is acceptable to gently move the head into a neutral position to obtain a clear airway. If the victim is breathing but remains unconscious, it is preferable that they be placed in the recovery position. The victim should be handled gently with no twisting. Aim to maintain spinal alignment of the head and neck with the torso, both during the turn and afterwards. In victims needing airway opening, use manoeuvres which are least likely to result in movement of the cervical spine. Jaw thrust and chin lift should be tried before head tilt.

4 Spinal Immobilisation Techniques and Devices

The clinical importance of prehospital immobilisation in spinal trauma remains unproven. There have been no randomised controlled trials to study immobilisation techniques or devices on trauma victims with suspected spinal cord injury. All existing studies have been retrospective or on healthy volunteers, manikins or cadavers[1]. Prehospital spinal immobilisation has never been shown to affect outcome and the estimates in the literature regarding the incidence of neurological deterioration due to inadequate immobilisation may be exaggerated. Spinal immobilisation can expose victims to the risks associated with specific devices and the time taken in application leads to delays in transport time.[2,3]

4.1 Cervical Collars

The use of semi rigid (SR) cervical collars by first aid providers is not recommended (CoSTR 2015, weak recommendation, low quality evidence).[5]

ANZCOR recommends all rescuers in the pre-hospital environment review their approach to the management of suspected spinal injury with regards to SR cervical collars. Consistent with the first aid principle of preventing further harm, the potential benefits of applying a cervical collar do not outweigh harms such as increased intracranial pressure, pressure injuries or pain and unnecessary movement that can occur with the fitting and application of a collar. In suspected cervical spine injury, ANZCOR recommends that the initial management should be manual support of the head in a natural, neutral position, limiting angular movement (expert consensus opinion). In healthy adults, padding under the head (approximately 2cm) may optimise the neutral position.[6]

The potential adverse effects of SR cervical collars increase with duration of use and include:

- unnecessary movement of the head and neck with the sizing and fitting of the collar
- discomfort and pain
- restricted mouth opening and difficulty swallowing[8]
- airway compromise should the victim vomit[8]
- pressure on neck veins raising intra-cranial pressure[9] (harmful to head injured victims)
- hiding potential life-threatening conditions[10].

4.2 Spinal Boards

Rigid backboards placed under the victim can be used by first aiders should it be necessary to move the victim. The benefits of stabilizing the head will be limited unless the motion of the trunk is also controlled effectively during transport.11,12 Victims should not be left on rigid spinal boards. Healthy subjects left on spine boards develop pain in the neck, back of the head, shoulder blades and lower back. The same areas are at risk of pressure necrosis.13,14,1^5 Conscious victims may attempt to move around in an effort to improve comfort, potentially worsening their injury.

Paralysed or unconscious victims are at higher risks of development of pressure necrosis due to their lack of pain sensation. Strapping has been shown to restrict breathing and should be loosened if compromising the victim.16,1^7

Victims may be more comfortable on a padded spine board, air mattress or bead filled vacuum mattress; devices used by some ambulance services.18,

4.3 Log Roll

The log roll is a manoeuvre performed by a trained team, to roll a victim from a supine position onto their side, and then flat again, so as to examine the back and/or to place or remove a spine board.[20]

4.4 Children

After road traffic accidents, conscious infants should be left in their rigid seat or capsule until assessed by ambulance personnel. If possible, remove the infant seat or capsule from the car with the infant/child in it. Children under eight years of age may require padding under their shoulders (approximately 2.5cm) for neutral spinal alignment.[21]

References

1. Hood N, Considine J: Spinal Immobilisation in Pre-hospital and Emergency Care: A Systematic Review of the Literature. Australasian Emergency Nursing Journal 2015; 18(3):118-
2. Hauswald M, Hsu M, Stockoff C: Maximizing Comfort and Minimizing Ischemia: A Comparison of Four methods of Spinal Immobilization. Prehospital Emergency Care. 2000; 4: 250-
3. Hauswald M, Ong G, Tandberg D, Omar Z: Out-of-hospital Spinal immobilization: Its Effect on Neurologic Injury. Academic Emergency Medicine. 1998; vol5, 3: 214-
4. Arishita G, Vayer J, Bellamy R: Cervical Spine Immobilization of Penetrating neck Wounds in a Hostile environment. Journal of Trauma. 1989; vol 29, 3: 332-
5. Zideman, D. A., Singletary, E. M., De Buck, E.,et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. Resuscitation, 95_ , e225. http://www.cprguidelines.eu/assets/downloads/costr/S0300-9572(15)00368-8_main.pdf00368-8_main.pdf) Accessed 19/11/
6. De LorenzoR, Olsen J, Boska M, Johnston R, Hamilton G, Augustine J, Barton R: Optimal Positioning for Cervical Immobilization. Annals of Emergency Medicine. 1996; vol 28, 3: 301- 308
7. Schriger D, Larmon B, LeGassick T, Blinman T: Spinal Immobilization on a Flat backboard: does it Result in neutral position of the Cervical Spine. Annals of Emergency Medicine. 1991; vol 20,8: 878-
8. Houghton D, Curley J: Dysphagia caused by a hard cervical collar. British Journal of Neurosurgery. 1996: vol 10, 5: 501-
9. Davies G, Deakin C, Wilson A: The effect of a rigid collar on intracranial pressure. Injury. 1996; vol 27, 9: 647- 649
10. Ramasamy A, Midwinter M, Mahoney P, Clasper J: Learning the lessons from conflict: Pre- hospital cervical spine stabilisation following ballistic neck trauma. Injury, Int. J. Care Injured. 2009; 40: 1342- 1345
11. Mazolewski P, Manix T: The Effectiveness of Strapping techniques in spinal Immobilization. Annals of Emergency Medicine. 1994; vol 23, 6: 1290- 1295
12. Perry S, McLellan B, McIlroy W, Maki B, Schwartz M, Fernie G: The Efficacy of Head Immobilization Techniques During Simulated Vehicle Motion. Spine. 1999; vol 24, 17: 1839- 1844
13. Main P, Lovell M: A review of seven support surfaces with emphasis on their protection of the spinally injured. Journal Accid Emerg Med. 1996; 13: 34- 37

14. Sheerin F, de Frein R: The Occipital and Sacral Pressures Experienced by Healthy Volunteers Under spinal immobilization: A Trial of Three Surfaces. Journal of Emergency Nursing. 2007; vol 33, 5: 447- 450
15. Chan D, Goldberg R, Mason J, Chan L: Backboard versus Mattress Splint Immobilization: A Comparison of Symptoms Generated. Journal of Emergency Medicine. 1995; vol 14,3: 293-
16. Shafermeyer R, Ribbeck B, Gaskins J, Thomason S, Harlan M, Attkisson A: Respiratory Effects of Spinal immobilization in Children. Annals of Emergency Medicine. 1991; vol 20, 9: 1017-
17. Totten V, Sugarman D: Respiratory Effects of Spinal Immobilization. Prehospital Emergency Care. 1999; vol 3: 347-
18. Walton R, DeSalvo J, Ernst A, Shahane A: Padded vs unpadded Spine Board for Cervical Spine Immobilization. Acad Emerg Med. 1995; vol 2: 725-
19. Cordell W, Hollingsworth J, Olinger M, Stroman S, Nelson D: Pain and Tissue-Interface Pressures During Spine-Board Immobilization. Annals of Emergency Medicine. 1995; vol 26,1: 31-
20. Boissy P, Shrier I, Brière, Mellete J, Fecteau L, Matheson G, Garza D, Meeuwisse W, Segal E Boulay J, Steele R: Effectiveness of Cervical Spine Stabilization Techniques. Clin J Sports Med. 2001; vol 21, 2: 80-
21. Nypaver M and Treloar D. Neutral cervical spine positioning in children. Annals of Emergency Medicine 1994; vol 23, 2: 208- 11

Further Reading

- ANZCOR Guideline 2 Priorities in an emergency
- ANZCOR Guideline 3 Unconsciousness
- ANZCOR Guideline 4 Airway
- ANZCOR Guideline 8 Cardiopulmonary Resuscitation

ANZCOR GUIDELINE 9.1.7 - EMERGENCY MANAGEMENT OF A CRUSHED VICTIM

INTRODUCTION

Crush injuries may result from a variety of situations, including vehicle entrapment, falling debris, industrial accident or by prolonged pressure to a part of the body due to their own body weight in an immobile victim.(1-5) Crush syndrome refers to the multiple problems that may subsequently develop, most commonly as a result of crush injuries to the limbs, particularly the legs.(1) Crush syndrome results from disruption of the body's chemistry and can result in kidney, heart and other problems. The likelihood of developing acute crush syndrome is directly related to the compression time, therefore victims should be released as quickly as possible, irrespective of how long they have been trapped.(1)

MANAGEMENT

- Ensure the scene is safe, and that there is no risk of injury to the rescuer or bystanders.
- Call an ambulance,
- If it is safe and physically possible, all crushing forces should be removed from the victim as soon as possible.^1
- A victim with a crush injury may not complain of pain, and there may be no external signs of injury. All victims who have been subjected to crush injury, including their own body weight, should be taken to hospital for immediate investigation 2-
- Keep the victim warm, treat any bleeding.5,6 (ARC Guideline 9.1.1)
- Continue to monitor the victim's condition. If the victim becomes unresponsive and is not breathing normally, follow Australian Resuscitation Council and New Zealand Resuscitation Council Basic Life Support Flowchart (Guideline 8) if possible.
- DO NOT leave the victim except if necessary to call an ambulance
- DO NOT use a tourniquet for the first aid management of a crush injury.

NOTE

Crushing force applied to the head, neck, chest or abdomen can cause death from breathing failure or heart failure so must be removed promptly. Although the victim may appear to be alert and not distressed, there is a risk of deterioration so ongoing reassessment of the victim's condition is essential.

REFERENCES

1. Greaves I, Porter K and Smith JE, Consensus statement on the early management of crush injury and prevention of crush syndrome. J R Army Med Corps. 2003;149(4):255-9
2. Aoki N, Demsar J, Zupan B, Mozina M. Predictive modelling for estimating risk of crush syndrome: a data mining approach. J Trauma. 2007;62(4:940-5)
3. Brown AA and Nicholls RJ, Crush syndrome: A report of 2 cases and a review of the literature. Brit J Surg. 1977;64(6):397- 402
4. Burns K, Cone DC, Portereiko JV. Complex extrication and crush injury. Prehosp Emerg Care. 2010;14(2):240- 4
5. Jagodzinski NA, Weerasinghe C and Porter K, Crush injuries and crush syndrome - A review. Part 1: The systemic injury. Trauma. 2010;12(2):69-88
6. Hatamizadeh P, Najafi I, Vanholder R, Rashid-Farokhi F, et al., Epidemiologic Aspects of the Bam Earthquake in Iran: The Nephrologic Perspective. Am J Kid Dis. 2006;47(3):428-38

LEVEL OF EVIDENCE Level III- Level IV

CLASS OF RECOMMENDATION Class B - Acceptable

FURTHER READING

ARC Guideline 8 Cardiopulmonary Resuscitation

ARC Guideline 9.1.1 Principles for the control of bleeding for first aiders

Wilderness Medical Society Practice Guidelines for Treatment of Eye Injuries and Illnesses in the Wilderness: 2014 Update

Ryan Paterson, MD; Brandy Drake, MD; Geoffrey Tabin, MD; Frank K. Butler Jr, MD; Tracy Cushing, MD, MPH

From the Kaiser Permanente Medical Group—Colorado (Dr Paterson); the Valley View Hospital Glenwood Springs, CO (Dr Drake); the Denver Health Medical Center/University of Colorado School of Medicine, Denver, CO (Drs Paterson and Cushing); the University of Utah School of Medicine, Salt Lake City, UT (Dr Tabin); and the US Army Institute of Surgical Research, Committee on Tactical Combat Casualty Care, Defense Board, Fort Sam Houston, TX (Dr Butler).

A panel convened to develop an evidence-based set of guidelines for the recognition and treatment of eye injuries and illnesses that may occur in the wilderness. These guidelines are meant to serve as a tool to help wilderness providers accurately identify and subsequently treat or evacuate for a variety of ophthalmologic complaints. Recommendations are graded on the basis of the quality of their supporting evidence and the balance between risks and benefits according to criteria developed by the American College of Chest Physicians. This is an updated version of the original guidelines published in Wilderness & Environmental Medicine 2012;23(4):325–336.

Introduction

Although eye complaints constitute approximately 3% of visits to emergency departments each year, their incidence in the wilderness is unknown.[1] Eye problems in the wilderness represent a challenging group of complaints for several reasons: access to proper equipment is limited, access to proper medications may be limited, and most practitioners in the wilderness are not specially trained.[2] The Wilderness Medical Society (WMS) published a set of guidelines on eye injuries in the wilderness in 2001. A panel was convened at the WMS Mountain Medicine meeting in Park City, UT, in February 2011 to update those guidelines with the most relevant evidence-based information.

Methods

The panel was convened at the 2011 Wilderness and Mountain Medicine WMS conference in Park City, UT. The WMS members were selected on the basis of clinical interest, research experience, or ophthalmologic expertise. Relevant

articles were identified through the PubMed and Cochrane Collaboration databases using keyword searches with the appropriate terms corresponding to each topic. Studies were reviewed, and the level of evidence was assessed. Data regarding specific wilderness treatment of eye injuries are sparse; therefore, we evaluated the data regarding eye injuries and illness in general and adapted these to the wilderness setting. The panel used a consensus approach to develop recommendations regarding diagnosis, treatment, and prevention of ocular injuries in the wilderness. These recommendations have been graded on the basis of clinical strength as outlined by the American College of Chest Physicians (ACCP) (see online Supplementary Table 1) and in conjunction with prior WMS practice guidelines.[3]

EXAMINATION AND EQUIPMENT

Examination of the eye in the wilderness may be difficult owing to lack of specialized equipment, but many basic elements of the eye examination can be completed in austere settings. The Table provides a list of equipment and medications that should be included in both a basic and an advanced eye kit. A basic kit is appropriate for short excursions, whereas the basic plus the advanced kit could be used for prolonged expeditions, especially to remote locations.

PRE-TRIP PLANNING AND PREVENTION

All people who have a history of any ophthalmologic problems who are participating in wilderness activities should have a complete eye examination within 3 months of any extended trip. Participants should be encouraged to bring all of their own equipment and medications, including glasses, goggles, sunglasses, contacts, lens solution, and any specific medications that they are currently taking or anticipate requiring. Additionally, participants should bring a copy of their lens prescription to aid in the acquisition of new corrective wear if needed or simply bring extra glasses or contacts if they will be in austere settings. Prevention of eye illness and injuries is paramount. Many eye illnesses are a result of accident, and thus may not be preventable, yet adequate sun and protective eyewear, good hygiene, and proper hand washing can prevent a large number of the conditions addressed in these guidelines.

Medical Eye Complaints

ACUTE VISION LOSS IN THE WHITE EYE

Vision loss can occur in both the red and white eye. As these are quite rare, data on these illnesses in the wilderness are very limited; therefore, wilderness management is adapted from clinic or hospital treatments to the wilderness setting as appropriate. There are numerous causes of acute vision loss; regardless of the cause, any vision loss should be considered an emergency, and all patients with vision loss should be considered for emergent evacuation (see the Table).

CENTRAL RETINAL ARTERY OCCLUSION

Central retinal artery occlusion (CRAO) is an ischemic stroke of the retina, usually as a result of an embolic event, that causes abrupt vision loss.[4] Treatment options in the wilderness are limited, but there are case reports to suggest improvement in visual acuity after providing highflow oxygen.[5] There are also several case studies that report improvement with hyperbaric oxygen therapy. Central retinal artery occlusion is currently an approved indication for hyperbaric oxygen therapy by the Hyperbaric Oxygen Committee of the Undersea and Hyperbaric Medicine Society.[5] If oxygen or hyperbaric therapies are available, they should be strongly considered for patients with suspected CRAO. Although a 2009 Cochrane Review found 2 randomized controlled trials evaluating 2 potential treatments for CRAO, pentoxifylline tablets and enhanced external counterpulsation, both trials showed improved retinal artery flow with their treatments, but neither showed improvement in vision.[6] These treatments are not available in the wilderness. Evacuation of these patients should be emergent.

- Oxygen:1C
- Hyperbaric oxygen therapy:1C
- External counterpulsation:2B
- Pentoxifylline:2B
- Emergent evacuation: 1C

Table - basic and advanced equipment and medications

Basic equipment	Basic medications
Light source: ideally would be penlight with blue filter, but bright headlamp is reasonable option	Artificial tears, individual bullet packs to avoid contamination
Cotton-tipped applicators	Fluorescein strips
Paperclip for lid retraction	Erythromycin ophthalmic 0.5% ointment
	Proparacaine 0.5% drops
	Oral pain medicine

Advanced equipment	Advanced medications
Metal eye shield: can be improvised from anything that will protect eye from further damage	Fluoroquinolone ophthalmic eye drops, such as moxifloxacin 0.5%
Magnifying glass	Prednisolone 1% drops
Fine forceps	Moxifloxacin 400-mg tablets
Small needle, such as 23G or tuberculin syringe	Prednisone 20-mg tablets
Direct ophthalmoscope	Atropine ophthalmic 1% ointment
Wire speculum for lid retraction	Pilocarpine 2% drops
	Diamox oral 250-mg tablets
	Topical NSAID, such as ketorolac, diclofenac, or bromfenac eye drops

- Equipment: 1C; medications: 1C.

NSAID, nonsteroidal anti-inflammatory drug.

CENTRAL RETINAL VEIN OCCLUSION

Central retinal vein occlusion (CRVO) is a blockage of the venous outflow from the eye leading to profound, painless vision loss. It can be a result of a thrombus, external compression, or vasculopathy, and usually occurs in people older than 50 years who have hypertension.[7] Patients with CRVO will usually have an afferent pupillary defect[4] : when a light is shone in the normal eye, both pupils will constrict, but when the light is then quickly shone in the abnormal eye, both pupils will dilate. It should be noted that many diseases that cause a unilateral optic neuropathy or retinal problem (such as ischemic optic neuropathy, retinal detachment, and CRVO) can lead to an afferent pupillary defect. It is not necessarily important to differentiate these conditions, but to recognize this abnormal finding. Although the treatments of CRVO in the hospital are complicated and involve laser treatments and intravitreal steroid administration, it is nevertheless reasonable to start treatment in the wilderness with topical steroids, if available (prednisolone 1%, 2 drops in the affected eye 4 times daily). Evacuation should be emergent, as these patients need specialist care and treatments.

- Topical steroid:1C
- Emergent evacuation:1C

RETINAL DETACHMENT

A retinal detachment is characterized by vision loss or floaters and flashes of bright light in a patient's visual field. The vision loss is often described as a fixed cloudy or curtains like defect in the visual field. This condition is usually painless.

Visual loss is variable, depending on where on the retina the tear has occurred.[4] These patients should be emergently evacuated, as surgical management may be necessary, and there is no effective field treatment.

- Emergent evacuation:1C

PERIOCULAR INFLAMMATION

Inflammation of the periocular region includes preseptal cellulitis, orbital cellulitis, and dacrocystitis. Preseptal cellulitis is an infection limited to the space anterior to the orbital septum, which includes the superficial tissue surrounding the eye and the eyelids. Orbital cellulitis is more extensive and involves the soft tissues in the bony orbit (deep to the orbital septum). Clinically, preseptal cellulitis presents with swelling and inflammation of the eyelid without involvement of the eye itself. Orbital cellulitis presents with inflammation within the orbit, including bulging of the eye (proptosis), swelling of the conjunctiva (chemosis), painful extraocular movements, and possible visual disturbance.[8] Additionally patients with orbital cellulitis may be systemically ill and have a fever. Periorbital cellulitis, while isolated to the preseptal space, is often associated with marked swelling, which makes the differentiation of preseptal from orbital cellulitis difficult. Preseptal cellulitis can be caused by spread from sinusitis, conjunctivitis, or blepharitis. Causes that may be more commonly encountered in the wilderness include trauma and insect bites.[8] Orbital cellulitis is usually caused by a sinus infection.[9] Additionally, preseptal cellulitis can spread and cause orbital cellulitis. There are limited data regarding management of these entities in the wilderness. Rapid identification of orbital cellulitis is paramount, as orbital cellulitis is a true medical emergency. Failure to treat this infection quickly can lead to blindness, intracranial infection, and possibly death. Initial management of both conditions requires antibiotics that cover Staphylococcus aureus, Streptococcus pneumoniae,and Haemophilus influenzae.[8]

Consideration should also be given toward coverage of anaerobes and methicillin-resistant S aureus(MRSA).[9] Treatment of both preseptal and orbital cellulitis should begin with amoxicillin/clavulanate 875 mg orally, 2 times daily for 10 days, but if that is not available, then oral fluoroquinolones (such as moxifloxacin) may be substituted.[10] If the practitioner is certain that an infection is isolated to the preseptal space, evacuation can be nonemergent. However, if there is any question that the infection may involve the orbit, or a presumed preseptal cellulitis is not responding to antibiotic treatment, evacuation should be emergent.[11] Dacryocystitis is an infection of the lacrimal sac that typically arises from obstruction of the lacrimal duct, which causes pooling of tears in the lacrimal sac and subsequent infection.[4] It presents as pain, swelling, and redness over the lacrimal duct, which is located at the nasal corner of the eye. The most common pathogens involved areS aureus, Streptococcus species, and H influenzae.[12] Dacrocystitis can be treated initially with oral antibiotics as well as warm compresses, if available. The preferred antibiotic is amoxicillin/clavulanate 875 mg orally, 2 times daily for 10 days, but if that is not included in your kit, oral fluoroquinolones (such as moxifloxacin) will likely suffice.[10] If this condition worsens, the patient should be evacuated, nonemergently.

- Antibiotics:1C
- Emergent evacuation for orbital cellulitis:1C
- Nonemergent evacuation for periorbital cellulitis:1C

ACUTE RED EYE

There are numerous conditions that can cause redness of the eye, and the severity of these conditions can range from mild to severe. There are 3 main tests that can be quickly and easily accomplished to help make the diagnosis:fluorescein staining, relief of pain with anesthetic drops, and pupillary status.[11] These tests can be easily performed in the wilderness if the supplies are available. The medical causes of the red eye can be broadly divided into conditions that are fluorescein positive (corneal ulcer or erosion, herpes keratitis), and those that are negative. Those that are negative can be further divided into conditions that resolve with an initial dose of topical anesthetic (conjunctivitis, blepharitis, ultra- violet [UV] keratitis) and those that do not (acute angle- closure glaucoma [AACG], iritis, scleritis).

ACUTE ANGLE-CLOSURE GLAUCOMA

Acute angle-closure glaucoma is caused by a blockage of aqueous outflow from the eye resulting in increased intraocular pressure (IOP). Symptoms include deep and severe pain, blurry vision, halos around lights, headache, and often nausea and vomiting.[13] Signs of this diagnosis include a nonreactive pupil that is mid dilated (4 mm to 6 mm), decreased visual acuity, “steamy” or “hazy” cornea, and a red eye.[14] Measurement of IOP in the field is often impossible owing to the absence of measurement devices, but gentle palpation of the globe through a closed lid may reveal a taut globe indicating elevated IOP.[13,15,16] The palpation should be done very gently and also in comparison with the normal eye. AACG is a medical emergency and requires emergent evacuation, as the definitive treatment is surgical. Standard treatment of AACG includes timolol 0.5%, 1 drop in the affected eye 2 times daily, topical steroids such as prednisolone 1%, 1 drop 4 times daily, and oral acetazolamide (Diamox), 500 mg 2 times daily.[4,17] Pilocarpine 1% to 2%, 1 drop every 15 minutes for 2 doses should be given 1 hour after other treatments have been given. Additionally, patients should be advised to lie flat for at least 1 hour.[17]

- Medical management:1B
- Surgical management:1A
- Emergent evacuation:1C

IRITIS

Nontraumatic iritis (also called anterior uveitis) is inflammation of the iris that is inflammatory or infectious. Many symptoms are similar to previously noted conditions including pain, redness of the conjunctiva, blurred vision, and photophobia. The patient may also have a history of iritis, which is a good clue to the diagnosis. One important examination finding is photophobia with shining of a penlight in the unaffected eye (consensual photophobia).[14] Standard therapy for

iritis includes corticosteroids, such as prednisolone 1% eye drops, 1 drop into the affected eye every hour if severe, every 4 hours if mild.[18] If drops are unavailable, or if the condition is worsening, oral steroids can be given, prednisone 40 mg to 60 mg oral daily. If there is concern for herpetic lesions or other serious infection, steroids should be avoided. In this case, a topical nonsteroidal anti-inflammatory drug (NSAID) may be beneficial. Cycloplegic (mydriatic) drops, such as atropine, are a key component to pain control as they relieve ciliary spasm.[18] Place 1 drop in the affected eye 2 times daily. Evacuation should be emergent, as iritis can be complicated by scarring of the pupil and elevated IOP, leading to decreased vision or blindness.[11]

- Topical steroids:1C
- Systemic steroids:2C
- Mydriatic drops:1C
- Topical NSAID: 2C
- Emergent evacuation:1C

HERPES KERATITIS

Herpes keratitis is diagnosed by a dendritic staining pattern on fluorescein examination. Often, patients have had previous episodes of ocular herpes infections.[13] Treatment includes oral or ophthalmic preparations of trifluridine or acyclovir, although these are often not included in expedition kits.[19] Participants should be encouraged to bring their own medicine if they have a history of this condition. Steroids should be avoided for patients with suspected herpes keratitis.[20] Although some texts advocate debridement of herpetic lesions with a cotton-tipped swab, that has not been proven to be efficacious and should only be done by experienced providers if deemed necessary.[19]

- Oral or ophthalmic antivirals:1A

CONJUNCTIVITIS

Among fluorescein-negative medical eye conditions, conjunctivitis, blepharitis, and UV keratitis can usually be treated in the field, and are typically not serious. Conjunctivitis is the most common cause of a red eye.[13] Common etiologies include allergies, viruses, and bacteria. Conjunctivitis always involves the palpebral conjunctiva, and the examiner must look under the lid to evaluate for this inflammation. The hallmark of allergic conjunctivitis is itching. Bacterial conjunctivitis gives a purulent discharge, whereas viral conjunctivitis tends to give a watery discharge. Treatment of allergic and viral conjunctivitis is primarily supportive, and bacterial conjunctivitis is treated with antibiotics. Although bacterial conjunctivitis is usually a self-resolving condition, studies have demonstrated that use of antibiotics results in faster clinical and microbiological remission.[21] There are no studies showing superiority of any one antibiotic over another; therefore, choice should be based on availability.[22] Erythromycin ointment, a 1-cm ribbon placed in the lower conjunctival sac 3 or 4 times daily for 7 days, is a reasonable option. It is important to note that viral conjunctivitis is extremely contagious, and proper hand washing is essential in wilderness settings to avoid spread throughout a group or to the contralateral eye.[23] Topical

anesthetic eye drops are diagnostically beneficial and provide acute relief for corneal ulcers, herpes keratitis, and conjunctivitis; however, it is important to note that they should not be used chronically as they are toxic to the corneal epithelium.[23]

- Topical or systemic antibiotics:1A
- Hand washing:1C

Altitude and the Eye

The environmental conditions at altitude, including hypoxia and low atmospheric pressure, pose several problems for the eye and may result in problems both for the previously healthy eye and for the patient with a history of ocular disease. Because the cornea receives most of its oxygen from the ambient air, the cornea becomes hypoxic at altitude and may not function normally.[24] In addition, patients with a history of intraocular gas bubbles should not go to high altitude owing to expansion of the gas bubble at decreasing atmospheric pressure. Patients should consult with their ophthalmologist regarding the safety of traveling to altitude.

ALTITUDE AFTER RADIAL KERATOTOMY, LASER-ASSISTED STROMAL IN SITU KERATOMILEUSIS, OR PHOTOREFRACTIVE KERATOTOMY

Hypoxia at altitude causes edema and thickening of the cornea.[25,26] Although this thickening has been shown not to cause visual disturbances in healthy eyes, it can be problematic for patients who have undergone radial keratotomy (RK), laser-assisted stromal in situ keratomileusis (LASIK), or photorefractive keratotomy (PRK). Radial keratotomy involves making incisions into the cornea, weakening its stability. The edema caused by the hypoxia of high altitude results in a flattening of these corneas, causing increased far-sightedness[24] and a significant decrease in visual acuity. The amount of vision change partially depends on the amount of residual myopia that a patient has at sea level, but is difficult to predict before altitude exposure. Although most of these changes will resolve with return to normal altitude, it is recommended that patients who have had RK take various levels of corrective lenses during their excursion. The cheapest option may be to wear glasses under goggles and to bring several levels of corrective glasses. The best option, however, would be to bring several levels of prescription goggles in the event of decreased visual acuity at altitude. Patients should consult with their ophthalmologist to obtain various strengths of eyewear. Eyes that have received LASIK and PRK undergo fewer refractive changes at altitude. There have been 2 studies demonstrating blurring of vision at altitude, and 2 studies demonstrating no change. A recent study of 12 climbers with LASIK-treated eyes noted no visual changes with changing altitude, despite dry eye associated with high altitude mountaineering being a known complication of LASIK.[27] Similarly, post-PRK patients seem to have excellent vision at altitude. There has only been 1 study of this PRK population, and it demonstrated no significant change in vision at altitude.[28]

- Caution at high altitude if history of RK:1C
- Safety of LASIK/PRK at altitude:1C

- Extra goggles/glasses if history of RK:1C

HIGH-ALTITUDE RETINAL HEMORRHAGE

At altitude an increase in retinal blood flow and subsequent retinal vein dilation[11] can lead to retinal hemorrhages, which have been well described since the 1970s.[29] The percentage of climbers experiencing retinal hemorrhages ranges from 4% to 82%, depending on the study.[29] Most cases of retinal hemorrhage do not cause visual disturbances, unless the hemorrhage involves the macula. Patients with retinal hemorrhages can continue ascent if no visual disturbances are present, but should be instructed to descend in the setting of altered vision.[11] Additionally, the presence of high altitude retinal hemorrhage has been associated with altitude illness, and its presence should alert providers to the possible development of altitude illness.

- Descent if visual changes:1C

Diving and the Eye

The hyperbaric environment can cause a number of effects on the eye. The primary concerns regarding dive medicine and the eye include ensuring that patients have normal visual acuity while diving, and making sure that patients who have a surgical intraocular gas bubble do not dive. Divers who normally require corrective lenses should dive with prescription goggles or soft contact lenses. Diving after refractive surgery such as LASIK and PRK is safe.[30] Diving after RK carries a hypothetical risk of corneal incision rupture, although in practice this is not seen, and diving is therefore considered safe. Additionally, divers who have had RK cannot dive for 6 months postoperatively to ensure adequate wound healing of the corneal incisions. Divers with a history of any type of eye surgery should always consult with their ophthalmologist before diving.[31]

- Safety of diving after refractive surgery:1C

OCULAR AND PERIOCULAR BAROTRAUMA

A complete description of the mechanism and pathophysiology of barotrauma are beyond the scope of this review; however, in brief, when air in a closed space is exposed to higher atmospheres of pressure, it is compressed. If this occurs while diving with a facemask, the lids, skin, and eyes can be drawn out into the mask. This force can be very strong and cause significant injury, resulting in periorbital ecchymosis, edema, subconjunctival hemorrhage, and potentially hyphema. The prevention of ocular and periocular barotrauma can be achieved by frequently filling one's mask with exhaled air from the nose to increase the volume of air in the mask.[31–33]

- Mask pressure equalization:1C

Traumatic Eye Injuries

The treatment of traumatic ocular emergencies in the wilderness hinges on rapid assessment, stabilization, and evacuation. Often eye injuries do not occur in isolation and may be associated with other potentially life- threatening injuries. Periocular and ocular traumas include many disorders, but these guidelines are limited to the most commonly encountered conditions in the wilderness. Ocular trauma is responsible for approximately 3% of emergency department visits in North America annually[34] and is a leading cause of preventable blindness worldwide. Its true incidence in the wilderness is unknown as epidemiologic data are absent from the literature.

PERIOCULAR TRAUMA

Eyelid lacerations

Falls, branches, rocks, or animal bites can cause eyelid lacerations. Eyelid lacerations can be classified as simple or complex. Simple lid lacerations are often horizontal and partial thickness, and do not involve the lid margin. This type of laceration can be managed with irrigation and typical wound care and has an excellent prognosis.[35] Eyelid lacerations that are complex, namely, full thickness, and involve the lid margin or the medial or lateral end of the palpebral fissures require evacuation and emergent ophthalmologic evaluation and repair, ideally within 36 hours.[35,36] Immediate treatment consists of prompt irrigation and the application of antibiotic ointment with shielding to protect the eye as evacuation commences. If an associated ruptured globe is suspected, there should be no further examination, manipulation, or irrigation of the eye. Treatment should be as outlined in the section on ruptured globe.

- Wound care:1B
- Antibiotic ointment:1B
- Eye shield:1C
- Emergent evacuation if complex eyelid laceration:1B

Orbital fractures

A direct blow to the face or orbit may cause fractures of any of the 7 facial bones that form the orbit. Orbital fractures can lead to significant ocular injury and subsequent visual impairment in approximately 17% of cases.[37] Thus, it is important for the wilderness provider to be able to perform a basic ophthalmologic examination to screen for severe injury, including entrapment of ocular muscles and pupillary or vision derangements. If signs of entrapment or visual derangements are present, emergent evacuation is required. Clinically significant retro-orbital hemorrhage can cause a compressive optic neuropathy and is a true ophthalmologic emergency. Signs include markedly elevated IOP, vision loss, and an afferent pupillary defect. If it is within the wilderness practitioner’s scope of training and practice, an emergent lateral cantholysis and canthotomy may restore vision in patients with compressive optic neuropathy attributable to retro-orbital hemorrhage. Therefore, emergent, expert management both in the field (with a cantholysis/canthotomy procedure, if indicated) and during emergent

evacuation to the closest emergency center are essential. As a result of consensus review in both maxillofacial and ophthalmologic practice patterns, patients with suspected orbital fractures should avoid blowing their nose until cleared to do so by a specialist. Patients may find relief from congestion through the use of nasal decongestant sprays, and if systemic steroids are available, they may be used to decrease orbital edema.[38,39] Beyond this basic medical management, emergent evacuation is imperative if signs of ocular muscle entrapment, traumatic vision loss, or suspected globe rupture are present.

- Orbital fractures
- Nose blowing:1C
- Decongestant sprays:1C
- Systemic steroids:2C
- With entrapment or visual derangements, emergent evacuation:1B
- Retro-orbital hemorrhage
- Lateral cantholysis:1B
- Emergent evacuation:1B

OCULAR TRAUMA

Globe rupture

Globe rupture or an open globe results from trauma and is an ophthalmologic emergency that requires emergent evacuation from the wilderness. A soft eye often characterizes an open globe, although palpation of the eye must be avoided in globe rupture as it may cause progression of the injury. If the anterior segment is involved in the rupture, the iris may prolapse into the wound, causing an irregular or a keyhole-shaped pupil. Such a change in pupil shape is extremely suggestive of a serious anterior segment injury that is in need of emergent evacuation for an ophthalmology consultation. The complications of globe rupture are endophthalmitis, defined as infection and inflammation of the contents of the globe, or potential vision loss, or both. In one review, lacerations of the globe that failed to self-seal and did not have intraocular tissue prolapse were at higher risk for development of endophthalmitis.[40] It has also been shown that primary repair more than 24 hours from injury is an independent risk factor in developing endophthalmitis.[40] Treatment of endophthalmitis generally requires antibiotics and corticosteroids that can be administered by a variety of routes.[40] Because of the likelihood of contamination after globe rupture in the wilderness setting, a strong pathophysiologic rationale exists for the early administration of broad-spectrum antibiotics, if available, that will cover Clostridium species,S aureus, Streptococcus species, and Pseudomonas species. The selected antibiotic must also have good eye penetration, which makes moxifloxacin, 400 mg orally once daily, a good antibiotic choice. Topical antibiotics should be avoided. In the wilderness, the patient’s eye should be shielded to avoid further injury. A pressure patch should not be used as it can result in the expulsion of the intraocular contents. All foreign bodies, if large enough to be visualized, should be left in place and splinted with a shield until evaluated by a specialist.[41]

- Shielding:1C

- Early antibiotics:1B
- Steroids:1B
- Emergent evacuation:1A

Hyphema

A hyphema is a collection of blood in the anterior chamber between the iris and the cornea, generally after trauma. The collection of blood is usually an isolated finding, but can accompany a dislocated lens, ruptured globe, or retinal injury. The majority of hyphemas resolve without treatment, but patients need to be monitored for secondary complications including corneal staining, rebleeding, and acute elevation in IOP. Therefore, this condition in the wilderness requires emergent evacuation.[42] There is no difference between ambulation and complete rest on the risk of secondary hemorrhage or time to rebleeding.[42] Despite this report, in the wilderness, based on the current pathophysiologic understanding of hemostasis, activity should be restricted to walking only. Available evidence regarding the use of cycloplegics or corticosteroids in traumatic hyphema is lacking.[42] Historically, cycloplegics (atropine 1%, every 8 hours) and topical corticosteroids (prednisolone acetate 1%, 4 times a day) have been used to decrease inflammation and improve a patient's comfort, and should be considered for this purpose.[42] If clinical signs of increased IOP are present, such as headache, nausea, or vomiting, or if the affected globe is taut under palpation in comparison with the unaffected eye, then addition of topical or systemic IOP-lowering medications are indicated.[15] In the wilderness setting, acetazolamide may be available and can be used for this purpose at a dose of 500 mg orally twice daily.[43] Analgesics and antiemetics should be used as clinically indicated to keep the patient comfortable and to minimize emesis, which can elevate IOP and worsen bleeding.[15,42,44] Patient positioning should keep the head elevated above 30 degrees at all times. There is no difference between a single patch versus a binocular patch on the risk of secondary hemorrhage or time to rebleeding.[42] Given the risk of rebleeding, NSAIDs should be avoided.[44] Although no direct evidence is available, near work or tasks (eg, reading) are also not recommended as the accommodation reflex moves the iris and can worsen rebleeding. A rigid shield is not effective in preventing rebleeding, but may be used to prevent further trauma.[42] If a hyphema is found, emergent evacuation for expert consultation is recommended.

- Activity restriction:1C
- Corticosteroids or cycloplegics:1C
- IOP-lowering medications:1A
- Acetazolamide:1B
- Analgesics and antiemetics:1C
- Use of a rigid shield:1B
- Avoidance of NSAIDs:1B
- Emergent evacuation:1B

Corneal abrasion

Corneal abrasions are one of the most frequently encountered ocular conditions and are most commonly caused by a foreign body, a direct blow to the eye, or the use of contact lenses. If a corneal foreign body is evident, then prompt removal is recommended. It is important to consider open globe injury with any corneal trauma. If open globe is suspected or deep corneal epithelial defects are apparent, then evacuation is emergent. Corneal abrasions should be treated with topical antibiotics (such as erythromycin ophthalmic 0.5% ointment, 1 cm every 8 hours),[45,46] cycloplegics (such as atropine 1%, 1 drop every 8 hours),[46] NSAIDs (such as ketorolac 0.5%, 1 drop every 8 hours),[47] and frequent use of artificial tears.[46] Sunglasses may help reduce photophobia, but there is no evidence to support eye patching for corneal abrasions. Bandage soft contact lenses can provide significant relief, restore depth perception, and improve function, but should be used only if 1) there is no retained corneal foreign body; 2) they are used in conjunction with a prophylactic topical antibiotic drop; and 3) they are removed within 24 to 48 hours. Topical anesthetic eye drops are diagnostically beneficial and provide acute relief for corneal abrasions and ulcers—but it is important to note that they should not be used chronically as they are potentially toxic to the corneal epithelium.[18] Despite this axiom, a recent prospective, double-blind, randomized trial of tetracaine versus saline for the treatment of uncomplicated corneal abrasions concluded that there was no significant difference in corneal healing and an overall patient perception of improved pain control.[48]

- Topical antibiotics:1A
- Cycloplegics:1A
- NSAIDs:1A
- Artificial tears:1C
- Sunglasses:1C
- Avoidance of patching:1A
- Emergent evacuation for open globe or deep epithelial defects:1C

Corneal ulcers

Corneal ulceration differs from abrasion in that the cause of acute ulceration is often infectious. Additionally, whereas a corneal abrasion is damage to the surface epithelial tissue of the cornea, ulceration involves deeper layers of the cornea. Signs include significant pain in the eye, white or gray infiltrate on the cornea on penlight evaluation, and an epithelial defect on fluorescein examination.[11] Risk factors for ulceration include previous corneal abrasion as well as contact lens wear, particularly with improper care of contact lenses or continued use for prolonged periods. Emergent treatment is important, as ulcers can enlarge, causing decreased visual acuity and corneal scarring or perforation. If a corneal ulcer is present, contact lenses must be discontinued.

If the patient is within 4 hours of an ophthalmologist's evaluation, it is preferable that the ophthalmologist obtains a culture before initiating treatment. If the patient is more than 4 hours from an expert evaluation, then empiric treatment should begin. Empiric treatment includes fourth-generation fluoroquinolone eye drops, such as moxifloxacin 0.5%. The patient should be loaded with the drops by

instilling 1 drop every 5 minutes for the first 30 minutes, then 1 drop every 30 minutes for 6 hours, followed by 1 drop every hour until the patient is able to see an ophthalmologist.[49,50] Oral Fluoroquinolones are warranted for wilderness cases if nothing else is available (such as moxifloxacin, 400 mg daily for 7 days). Cycloplegic medication (such as atropine 1%, 1 drop every 8 hours) can also be given to prevent synechiae formation and decrease pain in severe cases. Evacuation should be emergent.[11]

- Antibiotics, topical:1B
- Antibiotics, systemic:1C
- Cycloplegic:1B
- Emergent evacuation:1C

Chemical eye injuries

Chemical eye injuries in the wilderness are limited to case reports and case series, the most robust of which is related to the spitting cobra (genus Naja), primarily found in Africa. Other chemical injuries have been incurred through skunk musking, jellyfish stings, and exploding or spraying of cooking gases. The spitting cobra can eject its venom several meters into the eyes of its victim.[51] The venom can cause severe chemosis, blepharitis, and corneal irritation. Opacification with corneal and subconjunctival neovascularization (termed the“corneal opacification syndrome”)often leads to blindness and is most often associated with the black cobra (Naja nigricollis).[52,53] There is some evidence from rabbit studies that tetracycline drops and systemic heparin have a protective effect in guarding against corneal opacification syndrome.[52] Currently, there is no indication for topical or intravenous antivenom administration.[54] Prevention of chemical injuries is the most important tenet in this category, as treatment in the wilderness is largely supportive for such injuries. In general, rapid, large- volume irrigation and analgesia are the mainstays of treatment.[54] Ultimately, after chemical eye injury, emergent evacuation and expert consultation are encouraged.[54]

- Large-volume irrigation:1C
- Topical antibiotics:1C
- Evacuation:1C
- Animal-related bites and envenomations—systemic and topical antibiotics:1C
- Emergent evacuation:1C

Ultraviolet keratitis

Ultraviolet keratitis is a self-limited, inflammatory disorder of the cornea caused by UV rays. Symptoms of severe pain, burning, and tearing with a red eye, 6 to 12 hours after exposure to UV rays, should raise the suspicion of this condition. Although no prospective studies have looked at treatment of UV keratitis specifically, treatment is similar to that for corneal abrasions. That includes topical antibiotic ointments, anti-inflammatory drugs, and cycloplegics. (See previous section regarding dosing.) Artificial tears and patching are not recommended. Systemic analgesia is also recommended if severe pain is present. Evacuation should commence in a nonemergent manner if treatment is not available in the

wilderness setting. Prevention is the mainstay of treatment, with adequate sunglasses with side-shields being the most important mechanism for prevention.[55]

- Sunglasses:1C
- Topical antibiotics:1C
- Cycloplegics:1C
- NSAIDs:1C
- Artificial tears:1C
- Nonemergent evacuation:1C

Corneal frostbite

Corneal frostbite has been described only in case reports and book passages. Its treatment would follow similar guidelines as for other corneal epithelial disorders, including those listed under treatment for UV keratitis.[56–58]

- Topical antibiotics:1C
- Cycloplegics:1C
- NSAIDs:1C
- Artificial tears:1C

Traumatic iritis

Traumatic iritis is acute inflammation of the anterior uveal tract that can occur after trauma. In the wilderness, the diagnosis is made from a history of pain and photophobia that is often consensual. Inflammation can occur in the absence of visible ocular trauma.[14] This condition is often self-limited, and topical steroids (such as prednisolone acetate 1%, 1 drop every 2 hours when awake for the first week and then tapered slowly) and NSAIDs (such as ketorolac 0.5%, 1 drop every 8 hours)[59] can reduce inflammation, whereas cycloplegics (such as atropine 1%, 1 drop every 8 hours) can make the patient more comfortable until the inflammation is resolved.[60] Evacuation should commence in a nonemergent manner if treatment is not available in the wilderness setting.

- NSAIDs:1B
- Oral steroids:1B
- Topical steroids:1B
- Cycloplegics:1B
- Nonemergent evacuation:1C

Subconjunctival hemorrhage

Bleeding between the conjunctiva and sclera causes a collection of blood that appears bright red. Although impressive looking, this condition will resolve in days to weeks without treatment.[60] Unless there are signs of a ruptured globe or basilar skull fracture, no backcountry treatment or evacuation is required.

Conclusions

Wilderness eye injuries encompass a diverse group of illnesses that often require specialized equipment, medications, and expertise. This review provides an evidence-based overview of the most commonly encountered eye injuries; however, evidence regarding wilder- ness treatment is limited to case reports and the extrapolation of clinical and hospital care. Treatment in the wilderness is often based on available supplies and treatments rather than on the most evidence-based interventions. However, with the proper tools and physical examination skills, most providers can determine the need for further intervention or evacuation in cases of ocular pathology in the wilderness.

Supplementary tables

Supplementary ACCP Table 1 and Evidence Table 2 are available online at http://dx.doi.org/10.1016/j.wem.2014.08.008.

References

1. Cohn MJ, Kurtz D. Frequency of certain urgent eye problems in an emergency room in Massachusetts.JAm Optom Assoc. 1992;63:628– 633.
2. Forgey W.Wilderness Medical Society: Practice Guidelines for Wilderness Emergency Care.Guilford, CT: Globe Pequot Press; 2001.
3. Guyatt G, Gutterman D, Baumann MH, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians Task Force.Chest. 2006;129:174–181.
4. Marx J, Hockberger R, Walls R. Rosen's Emergency Medicine. 7th ed. Philadelphia, PA: Mosby; 2010.
5. Butler FK Jr, Hagan C, Murphy-Lavoie H. Hyperbaric oxygen therapy and the eye. Undersea Hyperb Med. 2008;35:333– 387.
6. Fraser SG, Adams W. Interventions for acute non-arteritic central retinal artery occlusion.Cochrane Database Syst Rev. 2009(1):CD001989.
7. Kiire CA, Chong NV. Managing retinal vein occlusion. BMJ. 2012;344:e499.
8. Howe L, Jones NS. Guidelines for the management of periorbital cellulitis/abscess.Clin Otolaryngol Allied Sci. 2004;29:725– 728.
9. Baring DE, Hilmi OJ. An evidence based review of periorbital cellulitis.Clin Otolaryngol. 2011;36:57– 64.
10. Pinar-Sueiro S, Sota M, Lerchundi TX, et al. Dacryocystitis: systematic approach to diagnosis and therapy.Curr Infect Dis Rep. 2012;14:137– 146.
11. Butler F. The eye in the wilderness. In: Auerbach PS, ed. Wilderness Medicine.6th ed. St. Louis, MO: Mosby; 2011.
12. Mandell GL, Bennett JE, Dolin R.Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 7th ed. Philadelphia, PA: Churchill Livingstone/Elsevier; 2010.
13. Mahmood AR, Narang AT. Diagnosis and management of the acute red eye.Emerg Med Clin North Am. 2008;26: 35 – 55, vi.
14. Dargin JM, Lowenstein RA. The painful eye.Emerg Med Clin North Am. 2008;26:199–216, viii.
15. Brandt MT, Haug RH. Traumatic hyphema: a comprehensive review.J Oral Maxillofac Surg. 2001;59:1462– 1470.

16. Baum J, Chaturvedi N, Netland PA, Dreyer EB. Assessment of intraocular pressure by palpation.Am J Ophthalmol. 1995;119:650– 651.
17. Choong YF, Irfan S, Menage MJ. Acute angle closure glaucoma: an evaluation of a protocol for acute treatment. Eye (Lond). 1999;13:613– 616.
18. Wilson SA, Last A. Management of corneal abrasions.Am Fam Physician. 2004;70:123– 128.
19. Wilhelmus KR. Antiviral treatment and other therapeutic interventions for herpes simplex virus epithelial keratitis. Cochrane Database Syst Rev. 2010(12):CD002898.
20. Morrow GL, Abbott RL. Conjunctivitis.Am Fam Physician. 1998;57:735– 746.
21. Sheikh A, Hurwitz B. Antibiotics versus placebo for acute bacterial conjunctivitis. Cochrane Database Syst Rev. 2006;(2):CD001211. Updated 2012.
22. Høvding G. Acute bacterial conjunctivitis.Acta Ophthalmol. 2008;86:5– 17.
23. Rapuano CJ.American Academy of Ophthalmology Cornea/External Disease Panel. Preferred Practice Pattern Guidelines. Conjunctivitis.San Francisco, CA: American Academy of Ophthalmology; 2008.
24. Mader TH, Tabin G. Going to high altitude with preexisting ocular conditions.High Alt Med Biol. 2003;4:419– 430.
25. Bosch MM, Barthelmes D, Merz TM, et al. New insights into changes in corneal thickness in healthy mountaineers during a very-high-altitude climb to Mount Muztagh Ata. Arch Ophthalmol. 2010;128:184– 189.
26. Morris DS, Somner JE, Scott KM, McCormick IJ, Aspinall P, Dhillon B. Corneal thickness at high altitude.Cornea. 2007;26:308– 311.
27. Dimmig JW, Tabin G. The ascent of Mount Everest following laser in situ keratomileusis. J Refract Surg. 2003;19:48– 51.
28. Mader TH, Blanton CL, Gilbert BN, et al. Refractive changes during 72-hour exposure to high altitude after refractive surgery.Ophthalmology. 1996;103:1188– 1195.
29. Hackett PH, Rennie D. Rales, peripheral edema, retinal hemorrhage and acute mountain sickness. Am J Med. 1979;67:214– 218.
30. Huang ET, Twa MD, Schanzlin DJ, Van Hoesen KB, Hill M, Langdorf MI. Refractive change in response to acute hyperbaric stress in refractive surgery patients.J Cataract Refract Surg. 2002;28:1575– 1580.
31. Butler FK Jr. Diving and hyperbaric ophthalmology.Surv Ophthalmol. 1995;39:347– 366.
32. Butler FK, Gurney N. Orbital hemorrhage following face- mask barotrauma.Undersea Hyperb Med. 2001;28:31– 34.
33. Butler FK, Bove AA. Infraorbital hypesthesia after maxillary sinus barotrauma.Undersea Hyperb Med. 1999;26: 257 – 259.
34. Bord SP, Linden J. Trauma to the globe and orbit.Emerg Med Clin North Am. 2008;26:97–123, vi-vii.
35. Chang EL, Rubin PA. Management of complex eyelid lacerations.Int Ophthalmol Clin. 2002;42:187– 201.
36. Murchison AP, Bilyk JR. Management of eyelid injuries. Facial Plast Surg. 2010;26:464– 481.
37. Brown MS, Ky W, Lisman RD. Concomitant ocular injuries with orbital fractures.J Craniomaxillofac Trauma. 1999;5:41– 48.

38. Lelli GJ Jr, Milite J, Maher E. Orbital Floor fractures: evaluation, indications, approach, and pearls from an ophthalmologist's perspective.Facial Plast Surg. 2007; 23:190– 199.
39. Joseph JM, Glavas IP. Orbital fractures: a review.Clin Ophthalmol. 2011;5:95– 100.
40. Zhang Y, Zhang MN, Jiang CH, Yao Y, Zhang K. Endophthalmitis following open globe injury.Br J Ophthalmol. 2010;94:111– 114.
41. Pokhrel PK, Loftus SA. Ocular emergencies.Am Fam Physician. 2007;76:829– 836. 42.Gharaibeh A, Savage HI, Scherer RW, Goldberg MF, Lindsley K. Medical interventions for traumatic hyphema. Cochrane Database Syst Rev. 2011;1:CD005431. Updated 2013.
42. FDA. Diamox. 2012. Available at: http://www.drugs.com/pro/diamox.html. Accessed March 5, 2012.
43. Walton W, Von Hagen S, Grigorian R, Zarbin M.Management of traumatic hyphema. Surv Ophthalmol.2002;47:297– 334.
44. Upadhyay MP, Karmacharya PC, Koirala S, et al. The Bhaktapur eye study: ocular trauma and antibiotic prophylaxis for the prevention of corneal ulceration in Nepal.Br J Ophthalmol. 2001;85:388– 392.
45. Fraser S. Corneal abrasion.Clin Ophthalmol. 2010;4:387–390.
46. Weaver CS, Terrell KM. Evidence-based emergency medicine. Update: do ophthalmic nonsteroidal anti-inflammatory drugs reduce the pain associated with simple corneal abrasion without delaying healing? Ann Emerg Med. 2003;41:134– 140.
47. Waldman N, Densie IK, Herbison P. Topical tetracaine used for 24 hours is safe and rated highly effective by patients for the treatment of pain caused by corneal abrasions: a double-blind, randomized clinical trial.Acad Emerg Med. 2014;21:3784– 3782.
48. American Academy of Ophthalmology Cornea/External Disease Panel, Preferred Practice Pattern Guidelines, Bacterial Keratitis-Limited Revision.San Francisco, CA: American Academy of Ophthalmology; 2011.
49. Shah VM, Tandon R, Satpathy G, et al. Randomized clinical study for comparative evaluation of fourth- generation fluoroquinolones with the combination of fortified antibiotics in the treatment of bacterial corneal ulcers.Cornea. 2010;29:751– 757.
50. Grüntzig J. Spitting cobra ophthalmia (Naja nigricollis) [in German].Klin Monatsbl Augenheilkd. 1984;185:527– 530.
51. Ismail M, al-Bekairi AM, el-Bedaiwy AM, Abd-el Salam MA. The ocular effects of spitting cobras: I. The ringhals cobra (Hemachatus haemachatus) venom-induced corneal opacification syndrome.J Toxicol Clin Toxicol. 1993;31:31–41.
52. Ismail M, al-Bekairi AM, el-Bedaiwy AM, Abd-el Salam MA. The ocular effects of spitting cobras: II. Evidence that cardiotoxins are responsible for the corneal opacification syndrome.J Toxicol Clin Toxicol. 1993;31:45– 62.
53. Chu ER, Weinstein SA, White J, Warrell DA. Venom ophthalmia caused by venoms of spitting elapid and other snakes: report of ten cases with review of epidemiology, clinical features, pathophysiology and management.Toxicon. 2010;56:259– 272.

54. McIntosh SE, Guercio B, Tabin GC, Leemon D, Schimelpfenig T. Ultraviolet keratitis among mountaineers and outdoor recreationalists.Wilderness Environ Med. 2011; 22:144– 147.
55. Lantukh VV. Effect of northern climate on the eye [in Russian].Vestn Oftalmol. 1983;1:58– 61. 57.Korablev AF. Case of transitory keratopathy in a skier caused by low air temperature and maximal physical exertions [in Russian].Vestn Oftalmol. 1981;2:72.
56. Forsius H. Climatic changes in the eyes of Eskimos, Lapps and Cheremisses.Acta Ophthalmol (Copenh). 1972;50: 532 – 538.
57. Kempen JH, Altaweel MM, Holbrook JT, et al. Multicenter Uveitis Steroid Treatment (MUST) Trial Research Group. Randomized comparison of systemic antiinflammatory therapy versus fluocinolone acetonide implant for intermediate, posterior, and panuveitis: The Multicenter Uveitis Steroid Treatment Trial.Ophthalmology. 2011;118:1916– 1926.
58. Cronau H, Kankanala RR, Mauger T. Diagnosis and management of red eye in primary care. Am Fam Physician. 2010;81:137– 144.

Wilderness Medical Society Clinical Practice Guidelines for Spinal Cord Protection

Author links open overlay panel Seth C.Hawkins MD[1] Jason Williams BS, NRP, DiMM[2] Brad L.Bennett PhD [3]ArthurIslas MD[4] Dietrich Whitfield Kayser MD[5] Robert Quinn MD[5]

The Wilderness Medical Society reconvened an expert panel to update best practice guidelines for spinal cord protection during trauma management. This panel, with membership updated in 2018, was charged with the development of evidence-based guidelines for management of the injured or potentially injured spine in wilderness environments. Recommendations are made regarding several parameters related to spinal cord protection. These recommendations are graded based on the quality of supporting evidence and balance the benefits and risks/burdens for each parameter according to the methodology stipulated by the American College of Chest Physicians. Key recommendations include the concept that interventions should be goal oriented (spinal cord/column protection in the context of overall patient and provider safety) rather than technique oriented (immobilization). This evidence-based, goal-oriented approach does not support the immobilization of suspected spinal injuries via rigid collars or backboards.

Keywords

spinal injury
spinal immobilization
cervical spine injury
cervical spine immobilization
spinal motion restriction

Introduction

Techniques for immobilization and extrication of the patient with a real or potential spine injury have been implemented for decades, albeit without high-quality evidence supporting their use. Such techniques addressed well-intentioned concerns about inflicting further serious injury. However, there is little evidence to support the effectiveness or necessity of these techniques, and increasing evidence suggests that such interventions may be harmful. Historic principles of

out-of-hospital spinal injury care have been more influenced by medicolegal implications and untested theory than by clinical or scientific evidence. The high cost (in terms of dollars, resources, and risk of patient injury) of defensive medicine in this regard is unlikely to be justified in the nonwilderness environment. In wilderness environments, any decision to immobilize a spine is even more significant and can be directly associated with the potential for further injury to the patient and to rescuers. When a person is injured in the wilderness, rescuers may be risking their own lives to provide a safe extrication. Under these circumstances, the need for sound evidence in clinical decision making is even more paramount. This is especially true for interventions that may introduce vastly more complex operations, such as converting a walkout of a nonimmobilized patient into a carryout of an immobilized patient.

In an effort to develop proper guidelines for spinal cord protection (SCP) in the wilderness environment based on best existing evidence, an expert panel was convened in 2011 to develop evidence-based guidelines.[1] The guidelines were revised in 2014.[2] This current publication marks the 2019 update to the original guidelines. A key philosophical difference between this and prior guidelines is the move away from technique-based principles, determining when and how to immobilize, to goal-based principles for determining how to best protect the spine from initial or secondary injury and provide safety.

Methods

The original practice guidelines were created by experts in the field who convened at the Wilderness Medical Society annual meeting in 2011 and published their findings in 2013. Members of the current revision team have a variety of professional backgrounds: 2 orthopedic surgeons, 2 experienced academic paramedics (1 military and 1 civilian), 1 emergency and emergency medical services (EMS) physician, and 1 family practitioner with sports medicine fellowship training. Relevant articles were identified through the PubMed and Cochrane Collaboration databases using keyword searches with the appropriate terms corresponding to each topic. Peer-reviewed studies related to spine immobilization and SCP including randomized controlled trials, observational studies, and case series were reviewed, and the level of evidence supporting the conclusions was assessed. Abstract-only reports were not included. Conclusions from review articles that did not perform systematic meta-analysis were not considered in the formulation of recommendations but may be cited in an effort to provide context. When no relevant studies were identified, the expert panel recommendation was based on risk versus benefit perceptions derived from patient care experience, case studies, and topical review publications. The panel used a consensus approach to develop recommendations regarding management of potential or actual spinal injuries in the wilderness. These recommendations have been graded based on clinical strength as outlined by the American College of Chest Physicians (online Supplementary Table).[3]

Scope of the Problem

Historically, the incidence of spinal cord injury (SCI) in the United States has been estimated at 54 cases per million people per year, representing 3% of hospital trauma admissions.[4,5] The National SCI Statistical Center found that 38% of these injuries were due to vehicle accidents, 32% from falls, and 14% from violence. A Norwegian epidemiologic study[6] revealed an incidence of cervical spine fractures of 12 out of 100,000 per year. Of these injuries, 60% were secondary to falls, and 21% were secondary to motor vehicle collisions. The incidence of open surgery for these injuries was 3 out of 100,000 per year.

Previous studies have shown that 2 to 10% of patients with SCI will demonstrate neurologic deterioration (ascending SCI) after initial neurologic testing. Factors attributed to neurologic deterioration include initiation of traction/immobilization and intubation (early [<24 h]), sustained hypotension (delayed [2–7 d]), and vertebral artery injury (late [>7 d]). Effectiveness of prehospital care and method of immobilization/transport have not been linked to neurologic deterioration.[7, 8, 9]

Authors have noted an improvement in the neurological status of patients with spinal cord injury arriving in emergency departments over the past 30 y. During the 1970s, 55% of patients referred to SCI centers arrived with complete neurologic lesions, whereas in the 1980s that number decreased to 39%.[10] This improvement in neurological status has been attributed to EMS initiated in the early 1970s. However, there is no specific evidence to support the belief that this improvement has anything to do with EMS protocols. It is likely that improvements in automobile safety and design, along with compulsory seat belt use laws, are at least partially responsible for these observations. Review of data from the national automotive sampling system data files between 1995 and 2001 revealed 8412 cases of cervical spine injury.[11] Approximately half (45%) were in unrestrained occupants, and the remainder consisted of belted only (38%), airbag only (9%), and both (8%) restraint systems.

It is important to interject some *a priori* clarity to the publication of these guidelines. Many articles have been repeatedly quoted in the literature as offering case evidence of neurologic deterioration in the presence of SCI secondary to inadequate out-of-hospital immobilization.[12, 13, 14, 15, 16, 17, 18] Careful review of these cases, however, reveals that virtually all represent missed or late diagnoses *after* hospital admission or deterioration that occurred while *under treatment* for a known diagnosis.

The focus of these guidelines is to present an evidence-based approach to out-of-hospital care in wilderness environments that minimizes the possibility of neurologic deterioration or injury exacerbation in the presence of an existing or potential SCI from the time of extrication to arrival at a medical facility.

Spinal immobilization itself is not a benign procedure. In addition to the risk of further injury to the patient as a consequence of increasing the danger of rescue, spinal immobilization itself is associated with documented risks and rather extreme discomfort. Although the expert panel was unable to identify a single well-documented case in the literature of out-of-hospital neurologic deterioration as a direct consequence of improper or inadequate immobilization, many cases have documented severe morbidity, and even mortality, secondary to immobilization itself.[5,19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31]

For the purpose of developing proper guidelines for SCP, it is important to recognize and/or attempt to differentiate 5 types of spinal injury scenarios: 1) an uninjured spine, 2) a stable spine injury without existing or potential neurological compromise, 3) an unstable, or potentially unstable, spine injury without apparent neurologic compromise, 4) an unstable spine injury with neurologic compromise, and 5) an injured patient with unknown spinal injury status. Historically, if immobilization were to be used, it was thought to be indicated for numbers 3, 4, and 5. However, a close re-evaluation of the evidence calls into question whether spinal immobilization is actually helpful in the wilderness out-of-hospital environment for any of these types of patients.

The phrase “clearing the spine” has many definitions depending on circumstances and the training level of the provider. It is generally regarded as more vernacular than academic. For instance, depending on the environment and caregiver, a “cleared” patient may have no evidence or suspicion of spine injury whatsoever, a low enough probability of injury to not need to have vertebral or SCI considered and not need radiographic imaging based on decision rule criteria (eg, national emergency x-radiography utilization study [NEXUS]),[32] or radiographic imaging with no demonstrable injury. Furthermore, some wilderness medicine educational organizations teach that “clearing” the spine is performed only for determining evacuation modality and not for actually determining the presence or absence of spinal injury. Such definitive determination is only made at a receiving facility.

In the modern era of out-of-hospital trauma care, any discussion regarding “clearing the spine” relative to immobilization debates a moot point. If there is no evidence that spinal immobilization helps patients, but increasing evidence that it hurts patients, it should not be considered as an intervention in the first place. Identifying potential vertebral or spinal cord injuries then assumes its rightful place as one among many considerations in managing a traumatized patient, with no special algorithm required. All that is required is the intuitive consideration of reducing motion if injury is suspected.

Most importantly, the philosophy and biomechanical physics behind the concept that spinal immobilization is a desired goal has been questioned.[33,34] A more recent theoretical argument maintains that spinal motion restriction (SMR) should be the desired goal and not strict immobilization.[34,35] Although these sound similar, motion restriction is very different from immobilization, both theoretically and in terms of technique. This concept of SMR has gained popularity in out-of-hospital care. The American College of Surgeons Committee on Trauma, American College of Emergency Physicians, and the National Association of EMS Physicians, as well as other endorsing agencies, have published a joint position statement advocating for SMR rather than immobilization in the trauma patient.[36] A main point of consensus is that current practices do not provide true immobilization of the spine, but with the goal of SMR a potentially injured spine may be protected by minimizing unwanted movement.[35]

However, although it may have fewer risks and may vastly simplify the logistics of wilderness rescue operations, there is no evidence that SMR is any more protective of the spinal cord than spinal immobilization. Cadaver studies and Newtonian physics suggests that physiological movement is unlikely to result in

further SCI in a patient with possible or actual vertebral or SCI.[33] Therefore, critical to our analysis of the literature is the understanding that the greater the degree to which an intervention produces absolute immobilization, the less desirable it may be. This runs counter to out-of-hospital standard of care for the past half century, but it appears to be a more evidence-based perspective.[31]

SMR is the most recently proposed mechanism for protecting injured spines from exacerbation of injury, but as noted, there is scant eviddence that physiological motion in fact causes further injury. Because of this, another theoretical dialogue has evolved suggesting that our goal in managing the injured spine should not be mechanistic (eg, “spinal motion restriction”) but instead should be goal oriented. Most authors following this principle have adopted the goal-oriented terminology of “spinal cord protection.” The basic gist of this argument is that we know our goal is to protect the spinal cord, but we do not have good evidence to support how to do that. Most authors using the goal-oriented term SCP believe the evidence suggests SMR is the best current mechanism to accomplish that goal and that the mechanism of spinal immobilization specifically does not meet the goal of SCP.[37, 38, 39]

The wilderness environment is a multiuser domain. From loggers to biologists to ultraendurance runners, there are more individuals in these environments now than ever before.[40] Consequently, there is overlap into many other medical disciplines.[31] In the sports medicine discipline, the National Athletic Training Association has been evaluating this issue and is currently considering SMR not only for sports in wilderness environments, but also on the sideline and courtside environments as well. Its recommendations stated that “sports medical teams must now recognize the concepts of SMR as compared to spinal immobilization” and that “techniques employed to move the spine injured athlete-patient from the field to the transportation vehicle should minimize spinal motion.”[41]

Although the focus of our discussion is the wilderness environment, in writing these guidelines, we have not found that our conclusions are confined to them.[42] Indeed, there appear to be consistent themes in treatment of possible SCI that transcend operational environment and are universal to out-of-hospital care. We encourage readers to see where our discussion of wilderness-specific SCP overlaps into their operational environment, and we would suggest that most of our discussion is in fact applicable to the entirety of out-of-hospital medical operations.

Results

Guidelines related to spinal immobilization and SCP, the evidence supporting them, and their recommendation grades are described.

Preferred Position for the Injured Spine

Although no studies have specifically evaluated an optimal generic position for the injured spine, clinical evidence (derived from imaging and patient care experience with traction, manipulation, and operative reduction) would strongly suggest that

neutral alignment is preferred.

Recommendation

Neutral alignment should be restored and maintained using nonrigid tools during extrication, unless such a maneuver is met with resistance, increased pain, or new or worsening neurologic deficit (**Evidence grade:** 1C).

Methods of Extrication With Possible Cervical Spine Injury

Analysis of neck motion during extrication from an automobile using an infrared 6-camera motion-capture system revealed that strategies permitting individuals to exit the vehicle under their own volition with cervical collar in place resulted in less motion of the cervical spine than extrication by experienced paramedics.[43] A similar biomechanical study recently corroborated these findings.[44] Dixon et al also reinforced self-extrication as the method of choice; self-extrication from a motor vehicle resulted in less spinal motion compared with different extraction methods. Their study included self-extraction with no cervical collar and only verbal instructions, extraction with cervical collar and physical assistance, extraction with spine board through rear and passenger side of vehicle, and short ejection jacket through driver door. Six trained emergency professionals assisted with each method, reflective markers were placed on the victim's bony prominences, and motion was tracked using circumferential cameras. They concluded that self-extraction with verbal instructions and no assistive devices was the most stable extraction method. Of note, use of backboards resulted in more motion, which was increasingly the case as victim body weight increased.[45]

A radiographic comparison showed superior immobilization of the normal cervical spine during extrication from an automobile with a Kendrick extrication device (KED) plus Philadelphia collar compared with short board, tape, and collar.[46] Similar benefit has been demonstrated in other studies with the KED, as well as similar devices.[47], [48], [49]

However, all this presupposes that immobilization is a desired outcome. Should the desire simply be motion restriction, it is likely that many options are equally viable. The most important principle would be to not cause further harm to the patient. Currently our author group cannot find case studies in which harm was caused by failure to place a cervical collar or a backboard, but we found increasing evidence, both actual and theoretical, that placement of these tools can cause harm. Furthermore, with clear instructions, patients appear capable of maintaining a stable neck for extrication without cervical collar.[45]

Recommendation

Patients requiring extrication should be encouraged to reduce movement of the neck, especially painful movement, and allowed to exit the situation under their own volition if alert and reliable. If injuries or other circumstances such as unconsciousness prevent controlled self-extrication, patients' cervical spines should be packaged to reduce passive motion and the airway adequately

managed without a goal of absolute immobilization. There is no requisite role for commercially made or improvised rigid cervical collars in an out-of-hospital environment (**Evidence grade:** 1C).

Moving the Patient With Real or Potential Spine Injury

Manual cervical traction is the standard technique for moving patients with known spine trauma in the hospital setting. This is done in an effort to keep the spine in the anatomic position and to prevent distortion of the spine, which might occur otherwise. Traction is often used for stabilization and reduction of unstable spine injuries. In the monitored hospital setting, up to 68 kg of cervical traction has been used safely in the reduction of unstable spine injuries.[50] Excessive traction can be dangerous in a grossly unstable spine injury and therefore should be avoided in the unmonitored setting.

Lift and slide transfer to a backboard results in superior stabilization of the entire spine compared with log-roll. One study also compared 2 methods of providing additional manual cervical spine stabilization relative to maintaining simultaneous stabilization of the thoracolumbar spine: the head squeeze and the trap squeeze. With the head squeeze maneuver, the lead rescuer lets the patient's head rest in his or her palms, with hands on both sides of the head with fingers placed so that the ulnar fingers can grab the mastoid process below and the second and third fingers can apply a jaw thrust if necessary. With the trap squeeze, the rescuer grabs the patient's trapezius muscles on either side of the head with his or her hands (thumbs anterior to the trapezius muscle) and firmly squeezes the head between the forearms with the forearms placed approximately at the level of the ears (Figure 1). The trap squeeze was superior to head squeeze in this study, particularly with simulation of an agitated patient.[51]

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Figure 1. Demonstration of trap squeeze technique for manual cervical spine stabilization. (Quinn et al.[2] Reprinted with permission from the Wilderness Medical Society. ©2014 Wilderness Medical Society.)

The superiority of the lift and slide transfer over the log-roll in providing stabilization of the entire spine has also been demonstrated in other studies.[52,53]

We are unaware of any evidence that would preclude transportation in the lateral decubitus position. Patients with spine injury are frequently placed in the lateral decubitus position without ill effect when hospitalized.

Lateral positioning is of interest because airway protection is paramount and traumatic brain injuries may occur concurrently with potential or actual cervical spine injuries. In a cadaver study, unstable C5-6 motion was monitored with electromagnetic sensors as 4 participants performed log rolled transfer and 2 participants used lateral position. The study concluded that in 5 of 6 planes there was no significant difference in range of motion. However, in the medial to lateral plane, 1.4 mm of motion was recorded and was found to be statistically significant. These results suggest that lateral positioning is appropriate in certain situations.[54]

Recommendation

The lift and slide transfer with trap squeeze is preferred to the log-roll when transferring patients when motion restriction is desired. In the case of facial fractures, an unconscious patient, or other scenarios concerning for airway compromise, the lateral position may be considered. Light to moderate traction should be used when returning a cervical spine to the anatomic position and transferring a patient (**Evidence grade:** 1C).

Effectiveness of Spinal Immobilization in Reducing the Incidence of Neurologic Sequelae

A Cochrane review found no randomized controlled trials of spinal immobilization. The authors of that review concluded that the effect of spinal immobilization on mortality, neurological injury, spinal stability, and adverse effects in trauma patients remains uncertain.[5] Because airway obstruction is a major cause of preventable death in trauma patients and spinal immobilization can contribute to airway compromise, the authors concluded that the possibility that immobilization may increase morbidity and mortality cannot be excluded.

Another study retrospectively reviewed all patients reporting to 2 university hospitals with acute blunt traumatic spinal or spinal cord injuries transported directly from the injury site to the hospital. One hospital was located in New Mexico (US) and the other was located in Malaysia. None of the 120 patients treated at the Malaysian university hospital had spinal immobilization during transport, whereas all 334 patients treated at the US university did. There was less neurologic disability in the patients who were not immobilized (odds ratio 2; P= 0.04).[55]

Recommendation

SCP should be considered an appropriate goal in patients with actual or suspected spinal injury; current evidence suggests SMR and not immobilization is the safest and most effective means of SCP (**Evidence grade:** 2C).

Effectiveness of the Cervical Collar in Immobilization of the Cervical Spine

Although use of the cervical collar is considered the gold standard in immobilization of the cervical spine, little evidence exists to indicate its effectiveness in immobilizing the cervical spine or that immobilization of the cervical spine is helpful in either patient field management or patient outcome.

An assumption exists that the neutral anatomic position is desired with an injured spine and that the cervical collar accomplishes this goal. However, 1 study demonstrated that more than 80% of adults require 1 to 5 cm of occipital padding in addition to a cervical collar to maintain the cervical spine in the neutral position relative to the torso, dependent upon physical characteristics and muscle development.[56]

A separate assumption exists that the cervical collar restricts motion of the cervical spine. When studied, use of a cervical collar was better than no immobilization, but it did not effectively reduce motion in an unstable spine model.[57] Another study analyzed cervical motion with no collar and with 3 different cervical collar types.[53] Although there was a decrease in the amount of motion generated in every plane of motion as a result of wearing each of the 3 collars, none of the changes proved to be significantly different. In another study, a rigid cervical collar combined with a backboard reduced cervical motion to 34% of normal.[58] Use of head blocks and a backboard reduced motion to 12% of normal. Addition of a rigid cervical collar to the use of head blocks provided no added immobilization benefit, but it did limit mouth opening.

These results have been somewhat contradicted by Podolsky et al,[59] who demonstrated in a similar study that neither collars alone nor sandbags and tape provided satisfactory restriction of cervical spine motion. In their study, addition of a rigid cervical collar to the sandbags and tape resulted in a statistically significant reduction in neck extension. Lador et al[60] demonstrated cervical distraction at the site of injury with the use of a rigid collar, as well as creation of a pivot point in the cervical spine where the collar meets the skull and shoulders. Others have also demonstrated abnormal separation between vertebrae with the use of cervical collars in the presence of a dissociative injury.[61]

Should ligamentous and bony structure integrity be compromised, traction that would normally pull the spine into neutral alignment may simply place tension on the spinal cord. Ivancic[62] performed a biomechanical investigation of 2 types of cervical collars and 2 types of cervicothoracic orthoses. Even though this study demonstrated increasing effectiveness of immobilization with the more constrained devices, particularly with middle and lower cervical spine flexion and extension, the most restrictive device still allowed 58% of axial rotation and 54% of lateral bending. Another study showed that ski patroller use of cervical collars and the removal of ski helmets led to significant cervical spine movement. The

authors recommended against helmet removal and cervical collar use.[63] There is a tremendous variety of helmet designs, and each may have its own benefit or risk in regard to a cervical spine injury. Each also may have its own method of fastening and removal. Therefore, in keeping with goal-oriented SCP, removal of a helmet may not be in the patient's best interest.

Rigid cervical collars are also difficult to apply correctly and are often incorrectly applied even by those who believe they are competent in this skill. When studied, 89% of providers made at least 1 error in placement, and competence was not related to confidence.[64]

Independent of whether cervical collars are effective, their use may be associated with complications related to the collar itself. Cervical orthoses can increase the risk of aspiration and impede the ability to establish an adequate airway. These devices have also been shown to directly compromise respiration. Ay et al[25] demonstrated decreases in forced expiratory volume in 1 second (FEV_1) and forced vital capacity with both the KED and long spinal backboard. Another study showed a 15% decrease in FEV_1 with a cervical collar and backboard and noted that respiratory restriction was more pronounced with age.[17] Others have demonstrated similar findings.[22,23,25] Cervical collars have also been associated with elevated intracranial pressure,[30,65, 66, 67, 68] pressure ulcerations,[69, 70, 71, 72] increased venous congestion complicating global brain injury,[73] unintentional strangulation by a cervical collar after attempted suicide by hanging,[73] and concealments of important physical findings such as soft tissue injuries, tracheal deviation, or subcutaneous air.[30,69, 70, 71, 72,74] These could all complicate evaluation and management of patients in wilderness medical care.

Although the expert panel remains unaware of any specific cases of documented neurologic deterioration occurring secondary to absent or inadequate out-of-hospital immobilization, many cases of documented neurologic deterioration, and even death, have now been reported with the use of a cervical collar in patients with ankylosing spondylitis.[26,27,75] In these patients with bony vertebral bridging, the rigid collar places focused stress on unstable portions of spine, thus increasing risk of neurologic injury; use should be considered contraindicated. Overall, rigid cervical collars have numerous identified risks and no demonstrated benefit.

Recommendation

Commercial or improvised soft cervical collars should be considered one of several tools available to aid in reducing cervical spine motion, if that is a desired goal. It should not be used if the presence of the collar in itself compromises emergent patient care. There is no requisite role for rigid cervical collars in wilderness out-of-hospital trauma care (**Evidence grade:** 2B).

Recommendation

If the medical history is known, use of any rigid cervical collar is contraindicated in ankylosing spondylitis. Patients with suspected injury should have their neck supported in a position of comfort (**Evidence grade:** 1B).

Use of Backboard

Several studies have demonstrated that a vacuum mattress provides significantly superior spine stability/motion restriction, increased speed of application, and markedly improved patient comfort when compared to a backboard[76], [77], [78], [79], [80], [81] and a cervical collar alone[82] (Figure 2). Vacuum mattress immobilization of the potentially injured spine is the current recommendation of the International Commission for Mountain Emergency Medicine.[83]

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Figure 2. Demonstration of patient with spinal cord protection implemented via spinal motion restriction of the neck and back using a vacuum splint rather than rigid cervical collar and long board.

Recommendation

Vacuum mattress (Figure 2) provides superior motion restriction and improved patient comfort (with corresponding decreased risk of pressure sores) and is preferred over a backboard for motion restriction of either the entire spine or specific segments of concern. Backboards and other rigid carrying devices may be used for temporary patient movement if needed but should not be applied as a medical tool with an immobilization goal (**Evidence grade:** 1C).

Immobilizing the Cervical Spine

Anderson et al[84] performed a meta-analysis of data regarding immobilization of the asymptomatic cervical spine in blunt trauma patients. Their analysis revealed that an alert, asymptomatic patient without a distracting injury or neurologic deficit who is able to complete a functional range-of-motion examination may safely avoid cervical spine immobilization without radiographic evaluation (sensitivity 98%; specificity 35%; negative predictive value 100%; positive predictive value 4%). Although the sensitivity and negative predictive values quoted provide reassurance that clinically relevant injuries are not being missed, the low specificity and positive predictive value would indicate that a large number of patients (96%) are being immobilized unnecessarily.

NEXUS prospectively evaluated 5 parameters in selected emergency department patients with blunt trauma: no midline cervical tenderness, no focal neurologic deficits, normal alertness, no intoxication, and no painful/distracting injury.[32] Approximately 34,000 patients were evaluated. Cervical spine injuries were identified in 818, of which 578 were clinically significant. All but 8 of the 818 patients were identified using the criteria (sensitivity 99%; specificity 13%; negative predictive value 100%; positive predictive value 3%). Only 2 of the 8 had a clinically significant injury, 1 of which required surgery. As with the immobilization data, the positive predictive value would indicate that 97% of patients are still subjected to unnecessary immobilization and imaging.

EMS data were prospectively collected on 8975 patients with regard to 5 out-of-hospital clinical criteria—altered mental status, neurologic deficit, spine pain or tenderness, evidence of intoxication, or suspected extremity fracture—the absence of which identify out-of-hospital trauma patients without a significant spine injury. The authors identified 295 patients with spine injuries (3%). Spine injury was identified by the out-of-hospital criteria in 280 of 295 (94%). The criteria missed 15 patients. Thirteen of 15 had stable injuries (stable compression or vertebral process injuries). The remaining 2 would have been captured by more accurate out-of-hospital evaluation.[85] A similar prospective study with the same criteria collected data on 13,483 patients.[86] Sensitivity of the EMS protocol was 92% resulting in nonimmobilization of 8% of the patients with spine injuries, none of whom developed neurologic compromise.

Maine has used a prehospital selective spine assessment protocol since 2002. Patients with qualified mechanism of injury (axial load, blunt trauma, motor vehicle collision, adult fall from standing height) are not immobilized if they are reliable (no intoxication or altered mental status), have no distracting injury, have a normal neurological examination, and have no spine pain tenderness. During one 12 month study period only one patient with an unstable spine fracture and 19 stable fractures were found to have been not immobilized by the protocol in approximately 32,000 trauma encounters.[87] The protocol had a sensitivity of 94%, negative predictive value of 100%, specificity of 59%, and positive predictive value of 0%. The single unstable spine injury occurred in an 86-y-old female who injured her back while moving furniture 1 week prior to calling EMS; she had a T6-7 subluxation requiring fixation and was without neurologic injury. Elderly patients (>65 y of age) represented the largest number of stable spine fractures without

neurologic compromise but also demonstrated a higher risk of complications (pain, pressure sores, respiratory compromise) from spinal immobilization. Further data from the same study population published separately revealed that 1301 of 2220 patients were immobilized on the basis of the protocol: 416 (32%) were unreliable, 358 (28%) were considered to have distracting injuries, 80 (6%) had an abnormal neurologic examination, and 709 (54%) had spine pain or tenderness.[87] Of the 2220 patients, only 7 acute spine fractures were identified, of which all were appropriately immobilized under then-current guidelines.

Studies have also validated the prehospital use of the Canadian C-spine protocol.[88, 89, 90, 91, 92, 93, 94, 95, 96, 97] This protocol investigates 3 questions relevant to whether a patient requires cervical spine radiographs: 1) Is a high-risk factor present (age >65 y, dangerous mechanism, paresthesia)? 2) Is a low-risk factor present that allows safe assessment of range of motion (simple rear-end motor vehicle accident, ambulatory at any time since injury, sitting position in the emergency department, delayed onset of neck pain, absence of midline cervical spine tenderness)? 3) Is the patient able to actively rotate the neck 45° to the left and right?

In 1 study, the NEXUS criteria were compared to the Canadian C-spine criteria by 394 physicians evaluating 8283 patients, with an overall incidence of 169 (2%) of clinically important spine injuries.[95] The Canadian C-spine rule was more sensitive (99% vs 91%; $P<0.001$) and more specific (45 vs 37%; $P<0.001$) at detecting spine injuries.

A study of 6500 patients evaluated the relationship between mechanism of injury and spinal injury.[98] The authors concluded that the mechanism of injury does not affect the ability of clinical criteria to predict spinal injury. It should come as no surprise that this is the case and that no specific mechanism of injury will prove predictive in a meaningful capacity. There are certainly many cases in which minimal trauma can result in profound cervical spine injury with neurologic deficit (eg, an elderly patient following a minor fall). On the other hand, individuals often escape serious injury even after high-energy trauma.

Konstantinidis et al[99] reported on 101 evaluable patients with cervical spine injury. Distracting injuries were present in 88 patients (87%). Only 4 patients (4%) had no pain or tenderness on the initial examination of the cervical spine. All 4 patients had bruising and tenderness to the upper anterior chest. None of these 4 developed neurologic sequelae or required surgical stabilization or immobilization.

Work by Rahmatalla et al suggests that, if motion restriction is desired, a vacuum splint (Figure 2) is more effective than a cervical collar at limiting cervical spine motion.[82]

Recommendation

If SCP is desired, appropriately trained personnel, usig either the NEXUS criteria or the Canadian C-spine rule, can safely and effectively make decisions in the prehospital setting regarding whether cervical spine motion should be reduced (**Evidence grade:** 1A). If SCP is desired, a vacuum splint (Figure 2) is preferable to a rigid collar (**Evidence grade:** 1B).

Penetrating Trauma

Clinically significant spinal injury is rare in the setting of a stab wound but not uncommon following a gunshot wound (GSW).[100] Neurological deficit from penetrating assault is generally established and final at presentation.[21,101,102] In the civilian setting, where GSWs are predominately low velocity, spinal instability rarely occurs. DuBose et al reviewed 4204 patients sustaining GSWs to the head, neck, and torso in a civilian setting.[102] None of the 4204 patients demonstrated spinal instability, and only 2 of 327 (1%) required any form of operative intervention for decompression. They concluded that routine spinal imaging and immobilization is unwarranted in examinable patients without symptoms consistent with spinal injury.

High-velocity penetrating injury of the cervical spine is associated with a high incidence of major vascular injury and airway injury requiring advanced airway protection. Cervical spine immobilization has been associated with a higher incidence of morbidity, and even mortality, when used in the presence of penetrating cervical trauma.[19,21,24,28,103] Haut et al evaluated 45,284 patients with penetrating trauma and showed overall mortality to be twice as high in spine-immobilized patients (15 vs 7%; $P<0.001$).[21] A common observation in these studies is that cervical spine immobilization could mask important clinical signs, such as tracheal deviation, expanding hematoma, and diminished or absent carotid pulse, and may impair successful endotracheal intubation.[19,28,103]

The Committee on Tactical Combat Casualty Care recommended a balanced approach to cervical spine precautions when a significant mechanism of injury exists.[104,105] The Prehospital Trauma Life Support Executive Committee concluded that there are no data to support routine spine immobilization in patients with penetrating trauma to the cranium, neck, or torso.[102] More recently, the American College of Surgeons Committee on Trauma, American College of Emergency Physicians, and National Association of EMS Physicians,[36] as well as the Eastern Association for the Surgery of Trauma,[106] published joint position statements recommending that spine immobilization not be used routinely for adult patients with penetrating trauma. This is also consistent with recommendations from leading multiauthor wilderness EMS textbooks and wilderness medicine textbooks.[37,107]

Recommendation

Spinal immobilization should not be performed for isolated penetrating trauma (**Evidence grade:** 1B).

Discussion

The practice of spinal immobilization has been predicated entirely on philosophical, theoretical, and medicolegal grounds, and the justification for its use remains unchanged despite nearly a half century of widespread use. Despite a lack of evidence clearly supporting spinal immobilization, an absence of documented cases of neurologic deterioration as a result of inadequate

immobilization, and in the face of accumulating data challenging both the philosophical and theoretical grounds of immobilization, no randomized controlled trials have yet been performed in an attempt to validate its ongoing use or stratify any risk/benefit ratio. The financial harm to the system is likely enormous, measured in both direct and indirect costs. Conversely, the routine use of spinal immobilization in the wilderness environment not only increases the financial cost of rescue operations, it also greatly increases the time, logistics, danger, and complexity of the operation, thereby also exacting a cost in terms of increased morbidity and mortality to not only the patient but to rescue personnel as well.

In the wilderness environment, the goal of spinal assessment and care should not be to definitively rule out or recognize all forms of spine injury. Rather, the goal should be to minimize the risk of missing and/or exacerbating a potentially unstable spine injury. The risk of missing such an injury should be appropriately calibrated against the risk of exposing rescuers to the potential for serious injury or causing further injury to the patient. It appears the NEXUS criteria and components of the Canadian C-spine rule are overly restrictive, particularly in regard to the mechanism of injury, when used in the wilderness environment to evaluate cervical spine injury. Although similar algorithms have not been developed for the thoracolumbar spine, one could argue that similar rules and conditions would be appropriately applicable.

It is fortuitous and insightful that the vacuum splint has become popular in the rescue environment. Not only is this device portable and rapidly deployable, but it appears quite likely to provide superior spine motion restriction (should that be desired) in addition to its other packaging and evacuation benefits, not the least of which is enhanced patient comfort and a decrease in the likelihood of complications associated with a cervical collar and backboard (Figure 2).

After careful and meticulous review of the literature, and in combination with the collective expertise of the authors, we recommend that there is no medical role for rigid backboards or rigid cervical collars in a wilderness environment.

Definitive spinal evaluation can and should be performed upon arrival at an appropriate medical center but is not a feasible goal for wilderness medical care.

When patients have sustained blunt trauma, with or without concomitant penetrating trauma, the mechanism of injury must be evaluated as it relates to the overall context of the patient and scene. Judgment regarding the likelihood of associated spinal injury should be individualized, as no reasonable guidelines are practical given the wide and disparate combinations of trauma and injury. As previously discussed, in appropriate circumstances, severe spine trauma can result from minimal trauma (particularly in the elderly), yet patients can often escape serious injury following the most dramatic trauma and do not appear to require any more aggressive intervention than passive motion restriction with soft interventions like padding or encouragement of conscious patients not to move in any way that is painful, all of which should be intuitive interventions anyway.

If the patient is suspected of having a serious spinal injury, it is likely even more important that the spine not be immobilized. This principle may appear counterintuitive, but the chance of immobilization causing harm increases the less

alert a patient is (with regard to airway or delay in care attempting to immobilization) and the more injured the spine is (an actual vertebral or SCI is more likely to have significant deleterious effects from spasming and inflammation than a strain, sprain, or contusion). All patients with evidence of neurologic deficit should have SCP principles implemented, avoiding total immobilization.

Previous practice guidelines, including our own, have presented algorithms suggesting range of motion testing as a tool for evaluating need for attention to possible SCI. The premise for range of motion testing is based on the well-validated use of flexion/extension cervical spine radiographs to clear a cervical spine. For years (prior to magnetic resonance imaging), this procedure served as the “gold standard” used to definitively clear the cervical spine, based on the knowledge that a standard lateral c-spine x-ray may appear normal in the presence of significant soft tissue injury with underlying spine instability. Flexion/extension cervical spine radiographs have been routinely performed under the direct volition of the patient under the premise that alert patients will not cause themselves neurologic harm in the presence of an injury with the capacity to do so. To our knowledge, no adverse patient reaction has been reported after many years of use, and this further argues against the necessity for immobilization. The ability to perform the maneuver, and the extent to which range of motion should occur, should be left entirely to the alert patient; pain alone should not be used as a disqualifier to interrupt the maneuver. This technique may remain useful as another tool in determining whether SMR is even desirable in the first place.

Deciding whether to explore SCP measures can be safely accomplished by practitioners with at least a basic working knowledge of the fundamental elements. That is, the practitioner should be able to recognize degrees of major trauma, identify mechanisms of injury with the potential to cause spinal injury, perform a basic physical examination of the spine and neurologic system, recognize distracting injuries, and consequently recommend passive SMR or soft padding or vacuum splinting.

Conclusions

The scant and low quality of scientific evidence available does not support the current rationale for immobilizing a potential spine injury in the wilderness environment. The authors believe that a goal-oriented approach offers the best compromise between unnecessary immobilization and the risk of causing further damage in the presence of spinal injury. The goal-oriented approach would set SCP as the ultimate treatment goal. Although the best techniques to achieve this goal are not yet clear and require further research, current evidence suggests that SMR may be the most appropriate mechanism currently available. Current evidence also suggests that rigid immobilization via collar or backboard is not an effective or safe means to accomplish this goal and can result in a worse patient outcome in both blunt and penetrating trauma. Although these guidelines cover many of the relevant issues related to spine injury, questions remain that should serve as focus for future research. We would suggest this research should be

equally goal oriented and focus on the best techniques to prevent occurrence or exacerbation of spinal column or SCI and not spring from an a priori assumption that immobilization is necessary.

Author Contributions

All authors participated in review of the literature and analysis of studies. SCH and RQ drafted the original manuscript. All authors participated in critical revision of the manuscript and approved the final manuscript.

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References

1

R. Quinn, J. Williams, B. Bennett, G. Stiller, A. Islas, S. McCord**Wilderness Medical Society practice guidelines for spine immobilization in the austere environment**

Wilderness Environ Med, 24 (3) (2013), pp. 241-252

Article Download PDFView Record in ScopusGoogle Scholar

2

R.H. Quinn, J. Williams, B.L. Bennett, G. Stiller, A.A. Islas, S. McCord**Wilderness Medical Society practice guidelines for spine immobilization in the austere environment: 2014 Update**

Wilderness Environ Med, 25 (4 Suppl) (2014), pp. S105-S117

Article Download PDFView Record in ScopusGoogle Scholar

3

G. Guyatt, D. Gutterman, M.H. Baumann, D. Addrizzo-Harris, E.M. Hylek, B. Phillips, *et al.***Grading strength of recommendations and quality of evidence in clinical guidelines**

Chest, 129 (1) (2006), pp. 174-181

Article Download PDFCrossRefView Record in ScopusGoogle Scholar

4

Spinal cord injury (SCI) 2016 facts and figures at a glance

J Spinal Cord Med, 39 (4) (2016), pp. 493-494

Google Scholar

5

I. Kwan, F. Bunn, I. Roberts**Spinal immobilisation for trauma patients**

Cochrane Database Syst Rev, 2 (2001), p. CD002803
View Record in ScopusGoogle Scholar

6

H.L. Fredo, S.A. Rizvi, B. Lied, P. Ronning, E. Helseth**The epidemiology of traumatic cervical spine fractures: a prospective population study from Norway**
Scand J Trauma Resusc Emerg Med, 20 (2012 Dec 21), p. 85
CrossRefGoogle Scholar

7

J.S. Harrop, A.D. Sharan, A.R. Vaccaro, G.J. Przybylski**The cause of neurologic deterioration after acute cervical spinal cord injury**
Spine (Phila PA 1976), 26 (4) (2001), pp. 340-346
View Record in ScopusGoogle Scholar

8

J. Farmer, A. Vaccaro, T.J. Albert, S. Malone, R.A. Balderston, J.M. Cotler**Neurologic deterioration after cervical spinal cord injury**
J Spinal Disord, 11 (3) (1998), pp. 192-196
CrossRefView Record in ScopusGoogle Scholar

9

L.F. Marshall, S. Knowlton, S.R. Garfin, M.R. Klauber, H.M. Eisenberg, D. Kopaniky, *et al.***Deterioration following spinal cord injury. A multicenter study**
J Neurosurg, 66 (3) (1987), pp. 400-404
CrossRefView Record in ScopusGoogle Scholar

10

M.N. Hadley, B.C. Walters, P.A. Grabb, N.M. Oyesiku, G.J. Przybylski, D.K. Resnick, *et al.***Cervical spine immobilization before admission to the hospital**
Neurosurgery, 50 (3 Suppl) (2002), pp. S7-S17
View Record in ScopusGoogle Scholar

11

B. Claytor, P.A. MacLennan, G. McGwin Jr., L.W. Rue 3rd, J.S. Kirkpatrick**Cervical spine injury and restraint system use in motor vehicle collisions**
Spine (Phila PA 1976), 29 (4) (2004), pp. 386-389
View Record in ScopusGoogle Scholar

12

J.W. Davis, D.L. Phreaner, D.B. Hoyt, R.C. Mackersie**The etiology of missed cervical spine injuries**
J Trauma, 34 (3) (1993), pp. 342-346
CrossRefView Record in ScopusGoogle Scholar

13

H.H. Bohlman**Acute fractures and dislocations of the cervical spine: an analysis of three hundred hospitalized patients and review of the**

literature
J Bone Joint Surg Am, 61 (8) (1979), pp. 1119-1142
CrossRefView Record in ScopusGoogle Scholar

14
B. Jeanneret, F. Magerl, J.C. Ward**Over distraction: a hazard of skull traction in the management of acute injuries of the cervical spine**
Arch Orthop Trauma Surg, 110 (5) (1991), pp. 242-245
View Record in ScopusGoogle Scholar

15
V.S. Prasad, A. Schwartz, R. Bhutani, P.W. Sharkey, M.L. Schwartz**Characteristics of injuries to the cervical spine and spinal cord in polytrauma patient population: experience from a regional trauma unit**
Spinal Cord, 37 (8) (1999), pp. 560-568
CrossRefView Record in ScopusGoogle Scholar

16
D.C. Reid, R. Henderson, L. Saboe, J.D. Miller**Etiology and clinical course of missed spine fractures**
J Trauma, 27 (9) (1987), pp. 980-986
CrossRefView Record in ScopusGoogle Scholar

17
V.Y. Totten, D.B. Sugarman**Respiratory effects of spinal immobilization**
Prehosp Emerg Care, 3 (4) (1999), pp. 347-352
CrossRefView Record in ScopusGoogle Scholar

18
J. Toscano**Prevention of neurological deterioration before admission to a spinal cord injury**
Paraplegia, 26 (3) (1988), pp. 143-150
CrossRefView Record in ScopusGoogle Scholar

19
W.B. Vanderlan, B.E. Tew, N.E. McSwain Jr.**Increased risk of death with cervical spine immobilisation in penetrating cervical trauma**
Injury, 40 (8) (2009), pp. 880-883
Article Download PDFView Record in ScopusGoogle Scholar

20
L.E. Stuke, P.T. Pons, J.S. Guy, W.P. Chapleau, F.K. Butler, N.E. McSwain**Prehospital spine immobilization for penetrating trauma-review and recommendations from the prehospital trauma life support executive committee**
J Trauma, 71 (3) (2011), pp. 763-770
CrossRefGoogle Scholar

21
E.R. Haut, B.T. Kalish, D.T. Efron, A.H. Haider, K.A. Stevens, A.N. Kieninger, *et al*.**Spine immobilization in penetrating trauma: more harm than good?**
J Trauma, 68 (1) (2010), pp. 115-120

View Record in ScopusGoogle Scholar

22

D. Bauer, R. Kowalski**Effect of spinal immobilization devices on pulmonary function in the healthy, nonsmoking man**
Ann Emerg Med, 17 (9) (1988), pp. 915-918
Article Download PDFView Record in ScopusGoogle Scholar

23

R.W. Schafermeyer, B.M. Ribbeck, J. Gaskins, S. Thomason, M. Harlan, A. Attkisson**Respiratory effects of spinal immobilization in children**
Ann Emerg Med, 20 (9) (1991), pp. 1017-1019
Article Download PDFView Record in ScopusGoogle Scholar

24

A. Ramasamy, M. Midwinter, P. Mahoney, J. Clasper**Learning the lessons from conflict: pre-hospital cervical spine stabilization following ballistic neck trauma**
Injury, 40 (12) (2009), pp. 1342-1345
Article Download PDFView Record in ScopusGoogle Scholar

25

D. Ay, C. Aktas, S. Yesilyurt, S. Sankaya, A. Cetin, E.S. Ozdogan**Effects of spinal immobilization devices on pulmonary function in healthy volunteer individuals**
Ulus Travma Acil Cerrahi Derg, 17 (2) (2011), pp. 103-107
CrossRefView Record in ScopusGoogle Scholar

26

P. Thumbikat, R.P. Hariharan, G. Ravichandran, M.R. McClelland, K.M. Mathew**Spinal cord injury in patients with ankylosing spondylitis: a 10-year review**
Spine (Phila PA 1976), 32 (26) (2007), pp. 2989-2995
View Record in ScopusGoogle Scholar

27

A. Clarke, S. James, S. Ahuja**Ankylosing spondylitis: inadvertent application of a rigid collar after cervical fracture, leading to neurological complications and death**
Acta Orthop Belg, 76 (3) (2010), pp. 413-415
View Record in ScopusGoogle Scholar

28

W.B. Vanderlan, B.E. Tew, C.Y. Seguin, M.M. Mata, J.J. Yang, H.M. Horst, *et al.***Neurologic sequelae of penetrating cervical trauma**
Spine (Phila PA 1976), 34 (24) (2009), pp. 2646-2653
View Record in ScopusGoogle Scholar

29

J.B. Brown, P.E. Bankey, A.T. Sangosanya, J.D. Cheng, N.A. Stassen, M.L. Gestring**Prehospital spinal immobilization does not appear to be beneficial and may complicate care following gunshot injury to the torso**

J Trauma, 67 (4) (2009), pp. 774-778
View Record in ScopusGoogle Scholar

30

W. Ham, L. Schoonhoven, M.J. Schuurmans, L.P. Leenen**Pressure ulcers from spinal immobilization in trauma patients: a systematic review**
J Trauma Acute Care Surg, 76 (4) (2014), pp. 1131-1141
CrossRefView Record in ScopusGoogle Scholar

31

J.G. Ten Brinke, S.R. Groen, M. Dehnad, T.P. Saltzherr, M. Hogervorst, J.C. Goslings**Prehospital care of spinal injuries: a historical quest for reasoning and evidence**
Eur Spine J, 27 (12) (2018), pp. 2999-3006
CrossRefView Record in ScopusGoogle Scholar

32

J.R. Hoffman, W.R. Mower, A.B. Wolfson, K.H. Todd, M.I. Zucker**Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. National emergency X-radiography utilization study group**
N Engl J Med, 343 (2) (2000), pp. 94-99
View Record in ScopusGoogle Scholar

33

M. Hauswald**A re-conceptualisation of acute spinal care**
Emerg Med J, 30 (9) (2013), pp. 720-723
CrossRefView Record in ScopusGoogle Scholar

34

M. Hauswald, D. Braude**Spinal immobilization in trauma patients: is it really necessary?**
Curr Opin Crit Care, 8 (6) (2002), pp. 566-570
View Record in ScopusGoogle Scholar

35

S.C. Hawkins, R.B. Simon, J.P. Beissinger, D. Simon**Vertical Aid: Essential Wilderness Medicine for Climbers, Trekkers, and Mountaineers**
The Countryman Press, New York, NY (2017)
Google Scholar

36

P.E. Fischer, D.G. Perina, T.R. Delbridge, M.E. Fallat, J.P. Salomone, J. Dodd, *et al.***Spinal motion restriction in the trauma patient–a joint position statement**
Prehosp Emerg Care, 22 (6) (2018), pp. 659-661
CrossRefView Record in ScopusGoogle Scholar

37

S.C. Hawkins, M.G. Millin, W. Smith**Wilderness emergency medical services and response systems**

P.S. Auerbach (Ed.), Wilderness Medicine (7th ed.), Elsevier, Philadelphia, PA (2017), pp. 1200-1213
View Record in ScopusGoogle Scholar

38
W. Smith**Technical rescue interface introduction: principles of basic technical rescue, packaging, and patient care integration**
S.C. Hawkins (Ed.), Wilderness EMS, Wolters Kluwer, Philadelphia, PA (2018), pp. 433-458
View Record in ScopusGoogle Scholar

39
S.C. Hawkins**Introduction to wilderness EMS: words matter in wilderness EMS**
S.C. Hawkins (Ed.), Wilderness EMS, Wolters Kluwer, Philadelphia, PA (2018), pp. 4-13
View Record in ScopusGoogle Scholar

40
C.A. Asplund, C. Madden, A. Campbell, A.A. Islas**Preparticipation evaluation of the wilderness athlete and adventurer**
Clin J Sport Med, 25 (5) (2015), pp. 381-382
CrossRefView Record in ScopusGoogle Scholar

41
National Athletic Trainers Association**Appropriate care of the spine injured athlete: updated from the 1998 document**
Available at: https://www.nata.org/sites/default/files/Executive-Summary-Spine-Injury.pdf, Accessed 23rd Jan 2019
Google Scholar

42
J. Gallagher, J. Lyng, S.C. Hawkins, R. MacDonald, M. Millin**Physician oversight of specialized emergency medical services**
Prehosp Emerg Care, 4 (2018), p. 102
View Record in ScopusGoogle Scholar

43
J.S. Shafer, R.S. Naunheim**Cervical spine motion during extrication: a pilot study**
West J Emerg Med, 10 (2) (2009), pp. 74-78
View Record in ScopusGoogle Scholar

44
M. Dixon, J. O'Halloran, N.M. Cummins**Biomechanical analysis of spinal immobilization during prehospital extrication: a proof of concept study**
Emerg Med J, 31 (9) (2014), pp. 745-749
CrossRefView Record in ScopusGoogle Scholar

45

M. Dixon, J. O'Halloran, A. Hannigan, S. Keenan, N.M. Cummins, *et al.***Confirmation of suboptimal protocols in spinal immobilization?**
Emerg Med J, 32 (2015), pp. 939-945
CrossRefView Record in ScopusGoogle Scholar

46

J.M. Howell, R. Burrow, C. Dumontier, A. Hillyard**A practical radiographic comparison of short board technique and kendrick extrication device**
Ann Emerg Med, 18 (9) (1989), pp. 943-946
Article Download PDFView Record in ScopusGoogle Scholar

47

A.F. Graziano, E.A. Scheidel, J.R. Cline, L.J. Baer**A radiographic comparison of prehospital cervical immobilization methods**
Ann Emerg Med, 16 (10) (1987), pp. 1127-1131
Article Download PDFView Record in ScopusGoogle Scholar

48

D.R. Chandler, C. Nemejc, R.H. Adkins, R.L. Waters**Emergency cervical-spine immobilization**
Ann Emerg Med, 21 (10) (1992), pp. 1185-1188
Article Download PDFView Record in ScopusGoogle Scholar

49

A. Cohen, R. Bosshard, J.D. Yeo**A new device for the care of acute spinal injuries: the Russell extrication device (RED)**
Paraplegia, 28 (1990), pp. 151-157
CrossRefView Record in ScopusGoogle Scholar

50

A.S. Lee, J.C. MacLean, D.A. Newton**Rapid traction for reduction of cervical spine dislocations**
J Bone Joint Surg Br, 76 (3) (1994), pp. 352-356
View Record in ScopusGoogle Scholar

51

P. Boissy, I. Shrier, S. Briere, J. Mellete, L. Fecteau, G.O. Matheson, *et al.***Effectiveness of cervical spine stabilization techniques**
Clin J Sport Med, 21 (2) (2011), pp. 80-88
CrossRefView Record in ScopusGoogle Scholar

52

M. Horodyski, B.P. Conrad, G. Del Rossi, C.P. DiPaola, G.R. Rechtine 2nd**Removing a patient from the spine board: Is the lift and slide safer than the log roll?**
J Trauma, 70 (5) (2011), pp. 1282-1285
CrossRefView Record in ScopusGoogle Scholar

53

G. Del Rossi, T.P. Heffernan, M. Horodyski, G.R. Rechtine**The effectiveness of extrication collars tested during the execution of spine-board transfer techniques**

Spine J, 4 (6) (2004), pp. 619-623
View Record in ScopusGoogle Scholar

54

P.K. Hyldmo, M.B. Horodyski, B.P. Conrad, S. Aslaksen, J. Røislien, M. Prasarn, *et al.***Does the novel lateral trauma position cause more motion in an unstable cervical spine injury than a logroll maneuver?**
Am J Emerg Med, 35 (11) (2017), pp. 1630-1635
Article Download PDFView Record in ScopusGoogle Scholar

55

M. Hauswald, G. Ong, D. Tandberg, Z. Omar**Out-of-hospital spinal immobilization: its effect on neurologic injury**
Acad Emerg Med, 5 (3) (1998), pp. 214-219
CrossRefGoogle Scholar

56

N.E. McSwain Jr.**Spine management skills**
Prehospital Trauma Life Support (2nd ed.), Educational Direction, Akron, OH (1990), pp. 225-256
Google Scholar

57

M. Horodyski, C.P. DiPaola, B.P. Conrad, G.R. Rechtine 2nd**Cervical collars are insufficient for immobilizing an unstable cervical spine injury**
J Emerg Med, 41 (5) (2011), pp. 513-519
Article Download PDFView Record in ScopusGoogle Scholar

58

M. Holla**Value of a rigid collar in addition to head blocks: a proof of principle study**
Emerg Med J, 29 (2) (2012), pp. 104-107
CrossRefView Record in ScopusGoogle Scholar

59

S. Podolsky, L.J. Baraff, R.R. Simon, J.R. Hoffman, B. Larmon, W. Ablon**Efficacy of cervical spine immobilization methods**
J Trauma, 23 (6) (1983), pp. 461-465
CrossRefView Record in ScopusGoogle Scholar

60

R. Lador, P. Ben-Galim, J.A. Hipp**Motion within the unstable cervical spine during patient maneuvering: the neck pivot-shift phenomenon**
J Trauma, 70 (1) (2011), pp. 247-250
View Record in ScopusGoogle Scholar

61

P. Ben-Galim, N. Dreiangel, K.L. Mattox, C.A. Reitman, S.B. Kalantar, J.A. Hipp**Extrication collars can result in abnormal separation between vertebrae in the presence of a dissociative injury**
J Trauma, 69 (2) (2010), pp. 447-450
View Record in ScopusGoogle Scholar

62

P.C. Ivancic**Do cervical collars and cervicothoracic orthoses effectively stabilize the injured cervical spine? A biomechanical investigation**
Spine (Phila PA 1976), 38 (13) (2013), pp. E767-E774
View Record in ScopusGoogle Scholar

63

J. Murray, D.A. Rust**Cervical spine alignment in helmeted skiers and snowboarders with suspected head and neck injuries: comparison of lateral C-spine radiographs before and after helmet removal and implications for ski patrol transport**
Wilderness Environ Med, 28 (3) (2017), pp. 168-175
Article Download PDFView Record in ScopusGoogle Scholar

64

M. Kreinest, S. Goller, G. Rauch, C. Frank, B. Gliwitzky, C.G. Wölfl, *et al.***Application of cervical collars—an analysis of practical skills of professional emergency medical care providers**
PLoS One, 10 (11) (2015), p. e0143409
CrossRefGoogle Scholar

65

G.R. Craig, M.S. Nielsen**Rigid cervical collars and intracranial pressure**
Intensive Care Med, 17 (8) (1991), pp. 504-505
View Record in ScopusGoogle Scholar

66

G. Davies, C. Deakin, A. Wilson**The effect of a rigid collar on intracranial pressure**
Injury, 27 (9) (1996), pp. 647-649
Article Download PDFView Record in ScopusGoogle Scholar

67

J.H. Raphael, R. Chotai**Effects of the cervical collar on cerebrospinal fluid pressure**
Anaesthesia, 49 (5) (1994), pp. 437-439
View Record in ScopusGoogle Scholar

68

J.C. Kolb, R.L. Summers, R.L. Galli**Cervical collar-induced changes in intracranial pressure**
Am J Emerg Med, 17 (2) (1999), pp. 135-137
Article Download PDFView Record in ScopusGoogle Scholar

69

A.R. Mawson, J.J. Biundo Jr., P. Neville, H.A. Linares, Y. Winchester, A. Lopez**Risk factors for early occurring pressure ulcers following spinal cord injury**
Am J Phys Med Rehabil, 67 (3) (1988), pp. 123-127
CrossRefView Record in ScopusGoogle Scholar

70

B. Blaylock**Solving the problem of pressure ulcers resulting from cervical collars**
Ostomy Wound Manage, 42 (4) (1996), pp. 26-28
View Record in ScopusGoogle Scholar

71
S.C. Liew, D.A. Hill**Complication of hard cervical collars in multi-trauma patients**
Aust N Z J Surg, 64 (2) (1994), pp. 139-140
CrossRefView Record in ScopusGoogle Scholar

72
H.A. Linares, A.R. Mawson, E. Suarez, J.J. Biundo**Association between pressure sores and immobilization in the immediate post-injury period**
Orthopedics, 10 (4) (1987), pp. 571-573
View Record in ScopusGoogle Scholar

73
M. Lemyze, A. Palud, R. Favory, D. Mathieu**Unintentional strangulation by a cervical collar after attempted suicide by hanging**
Emerg Med J, 28 (6) (2011), p. 532
CrossRefView Record in ScopusGoogle Scholar

74
A.O. Oteir, P.A. Jennings, K. Smith, J. Stoelwinder**Should suspected cervical spinal cord injuries be immobilised? A systematic review protocol**
Inj Prev, 20 (3) (2014), p. e5
CrossRefGoogle Scholar

75
A. Maarouf, C.M. McQuown, J.A. Frey, R.A. Ahmed, L. Derrick**Iatrogenic spinal cord injury in a trauma patient with ankylosing spondylitis**
Prehosp Emerg Care, 21 (3) (2017), pp. 390-394
CrossRefView Record in ScopusGoogle Scholar

76
D. Chan, R.M. Goldberg, J. Mason, L. Chan**Backboard versus mattress splint immobilization: a comparison of symptoms generated**
J Emerg Med, 14 (3) (1996), pp. 293-298
Article Download PDFView Record in ScopusGoogle Scholar

77
R.S. Hamilton, P.T. Pons**The efficacy and comfort of full-body vacuum splints for cervical-spine immobilization**
J Emerg Med, 14 (5) (1996), pp. 553-559
Article Download PDFView Record in ScopusGoogle Scholar

78
D.R. Johnson, M. Hauswald, C. Stockhoff**Comparison of a vacuum splint device to a rigid backboard for spinal immobilization**
Am J Emerg Med, 14 (4) (1996), pp. 369-372

Article Download PDFView Record in ScopusGoogle Scholar

79

P.W. Main, M.E. Lovell**A review of seven support surfaces with emphasis on their protection of the spinally injured**
J Accid Emerg Med, 13 (1) (1996), pp. 34-37
CrossRefView Record in ScopusGoogle Scholar

80

M.E. Lovell, J.H. Evans**A comparison of the spinal board and the vacuum stretcher, spinal stability and interface pressure**
Injury, 25 (3) (1994), pp. 179-180
Article Download PDFView Record in ScopusGoogle Scholar

81

M.D. Luscombe, J.L. Williams**Comparison of a long spinal board and vacuum mattress for spinal immobilisation**
Emerg Med J, 20 (5) (2003), pp. 476-478
View Record in ScopusGoogle Scholar

82

S. Rahmatalla, J. DeShaw, J. Stilley, G. Denning, C. Jennissen**Comparing the efficacy of methods for immobilizing the cervical spine**
Spine (Phila PA 1976), 44 (1) (2019), pp. 32-40
CrossRefView Record in ScopusGoogle Scholar

83

J. Ellerton, I. Tomazin, H. Brugger, P. Paal**Immobilization and splinting in mountain rescue**
High Alt Med Biol, 10 (4) (2009), pp. 337-342
CrossRefView Record in ScopusGoogle Scholar

84

P.A. Anderson, R.D. Muchow, A. Munoz, W.L. Tontz, D.K. Resnick**Clearance of the asymptomatic cervical spine: a meta-analysis**
J Orthop Trauma, 24 (2) (2010), pp. 100-106
View Record in ScopusGoogle Scholar

85

R.M. Domeier, R.W. Evans, R.A. Swor, E.J. Rivera-Rivera, S.M. Frederiksen**Prospective validation of out-of-hospital spinal clearance criteria: a preliminary report**
Acad Emerg Med, 4 (6) (1997), pp. 643-646
CrossRefView Record in ScopusGoogle Scholar

86

R.M. Domeier, S.M. Frederiksen, K. Welch**Prospective performance assessment of an out-of-hospital protocol for selective spine immobilization using clinical spine clearance criteria**
Ann Emerg Med, 46 (2) (2005), pp. 123-131
Article Download PDFView Record in ScopusGoogle Scholar

87

J.H. Burton, M.G. Dunn, N.R. Harmon, T.A. Hermanson, J.R. Bradshaw**A statewide, prehospital emergency medical service selective patient spine immobilization protocol**
J Trauma, 61 (1) (2006), pp. 161-167
CrossRefView Record in ScopusGoogle Scholar

88

J.H. Burton, N.R. Harmon, M.G. Dunn, J.R. Bradshaw**EMS provider findings and interventions with a statewide EMS spine-assessment protocol**
Prehosp Emerg Care, 9 (3) (2005), pp. 303-309
CrossRefView Record in ScopusGoogle Scholar

89

G. Bandiera, I.G. Stiell, G.A. Wells, C. Clement, V. De Maio, K.L. Vandemheen, *et al.***The Canadian C-spine rule performs better than unstructured physician judgment**
Ann Emerg Med, 42 (3) (2003), pp. 395-402
Article Download PDFView Record in ScopusGoogle Scholar

90

G. Dickinson, I.G. Stiell, M. Schull, R. Brison, C.M. Clement, K.L. Vandemheen, *et al.***Retrospective application of the NEXUS low-risk criteria for cervical spine radiography in Canadian emergency departments**
Ann Emerg Med, 43 (4) (2004), pp. 507-514
Article Download PDFView Record in ScopusGoogle Scholar

91

D. Kerr, L. Bradshaw, A.M. Kelly**Implementation of the Canadian C-spine rule reduces cervical spine x-ray rate for alert patients with potential neck injury**
J Emerg Med, 28 (2) (2005), pp. 127-131
Article Download PDFView Record in ScopusGoogle Scholar

92

I.G. Stiell, C.M. Clement, A. O'Connor, B. Davies, C. Leclair, P. Sheehan, *et al.***Multicentre prospective validation of use of the Canadian C-spine rule by triage nurses in the emergency department**
CMAJ, 182 (11) (2010), pp. 1173-1179
CrossRefView Record in ScopusGoogle Scholar

93

I.G. Stiell, J. Grimshaw, G.A. Wells, D. Coyle, H.J. Lesiuk, B.H. Rowe, *et al.***A matched-pair cluster design study protocol to evaluate implementation of the Canadian C-spine rule in hospital emergency departments: Phase III**
Implement Sci, 2 (2007), p. 4
Google Scholar

94

I.G. Stiell, G.A. Wells, K.L. Vandemheen, C.M. Clement, H. Lesiuk, V.J. De Maio, *et al.***The Canadian C-spine rule for radiography in alert and stable**

trauma patients
JAMA, 286 (15) (2001), pp. 1841-1848
View Record in ScopusGoogle Scholar

95
I.G. Stiell, C.M. Clement, R.D. McKnight, R. Brison, M.J. Schull, B.H. Rowe, *et al.***The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma**
N Engl J Med, 349 (26) (2003), pp. 2510-2518
View Record in ScopusGoogle Scholar

96
C. Vaillancourt, M. Charette, A. Kasaboski, J. Maloney, G.A. Wells, I.G. Stiell**Evaluation of the safety of C-spine clearance by paramedics: design and methodology**
BMC Emerg Med, 11 (2011), pp. 1-11
Google Scholar

97
C. Vaillancourt, I.G. Stiell, T. Beaudoin, J. Maloney, A.R. Anton, P. Bradford, *et al.***The out-of-hospital validation of the Canadian C-spine rule by paramedics**
Ann Emerg Med, 54 (5) (2009), pp. 663-671
View Record in ScopusGoogle Scholar

98
R.M. Domeier, R.W. Evans, R.A. Swor, J.B. Hancock, W. Fales, J. Krohmer, *et al.***The reliability of prehospital clinical evaluation for potential spinal injury is not affected by the mechanism of injury**
Prehosp Emerg Care, 3 (4) (1999), pp. 332-337
CrossRefView Record in ScopusGoogle Scholar

99
A. Konstantinidis, D. Plurad, G. Barmparas, K. Inaba, L. Lam, M. Bukur, *et al.***The presence of nonthoracic distracting injuries does not affect the initial clinical examination of the cervical spine in evaluable blunt trauma patients: a prospective observational study**
J Trauma, 71 (3) (2011), pp. 528-532
View Record in ScopusGoogle Scholar

100
K. Inaba, G. Barmparas, D. Ibrahim, B.C. Branco, P. Gruen, S. Reddy, *et al.***Clinical examination is highly sensitive for detecting clinically significant spinal injuries after gunshot wounds**
J Trauma, 71 (3) (2011), pp. 523-527
CrossRefView Record in ScopusGoogle Scholar

101
P. Rhee, E.J. Kuncir, L. Johnson, C. Brown, G. Velmahos, M. Martin, *et al.***Cervical spine injury is highly dependent on the mechanism of injury following blunt and penetrating assault**
J Trauma, 61 (2006), pp. 1166-1170

CrossRefView Record in ScopusGoogle Scholar

102

J. DuBose, P.G.R. Teixeira, P. Hadjizacharia, M. Hannon, K. Inaba, D.G. Green, *et al.***The role of routine spinal imaging and immobilisation in asymptomatic patients after gunshot wounds**
Injury, 40 (8) (2009), pp. 860-863
Article Download PDFView Record in ScopusGoogle Scholar

103

Y. Barkana, M. Stein, A. Scope, R. Maor, Y. Abramovich, Z. Friedman, *et al.***Prehospital stabilization of the cervical spine for penetrating injuries of the neck—is it necessary?**
Injury, 31 (5) (2000), pp. 305-309
Article Download PDFView Record in ScopusGoogle Scholar

104

F.K. Butler Jr., J. Hagmann, E.G. Butler**Tactical combat casualty care in special operations**
Mil Med, 161 (suppl) (1996), pp. 3-16
View Record in ScopusGoogle Scholar

105

National Association of Emergency Medical Technicians, F.K. Butler, S.D. Giebner, N. McSwain, *et al.* (Eds.), Prehospital trauma life support manual. 8th ed. (Military), Jones and Bartlett Learning, Burlington, MA (2014)
Google Scholar

106

C.G. Velopulos, H.M. Shihab, L. Lottenberg, M. Feinman, A. Raja, J. Salomone, *et al.***Prehospital spine immobilization/spinal motion restriction in penetrating trauma: a practice management guideline from the Eastern Association for the Surgery of Trauma (EAST)**
J Trauma Acute Care Surg, 84 (5) (2018), pp. 736-744
View Record in ScopusGoogle Scholar

107

S.C. Hawkins (Ed.), Wilderness EMS, Wolters Kluwer, Philadelphia, PA (2018)
Google Scholar

Wilderness Medical Society Practice Guidelines for Basic Wound Management in the Austere Environment: 2014 Update

Robert H. Quinn, MD; Ian Wedmore, MD; Eric L. Johnson, MD; Arthur A. Islas, MD; Anne Anglim, MD, MS; Ken Zafren, MD; Cindy Bitter, MD, MA; Vicki Mazzorana, MD

From the Department of Orthopaedic Surgery, University of Texas Health Science Center, San Antonio, TX (Dr Quinn); the Madigan Army Medical Center, Tacoma, WA (Dr Wedmore); the Wound Healing & Hyperbaric Medicine Clinic, Saint Alphonsus Hospital, Boise, ID (Dr Johnson); the Department of Family and Community Medicine, Texas Tech Health Science Center, El Paso, TX (Dr Islas); the Department of Infectious Diseases/Hospital Medicine, Saint Peter's Hospital, Helena, MT (Dr Anglim); the Division of Emergency Medicine, Stanford University Medical Center, Stanford, CA (Dr Zafren); the Department of Emergency Medicine, Missouri Baptist Medical Center, St. Louis, MO (Dr Bitter); and the Division of Emergency Medicine, Veterans Administration Medical Center, Las Vegas, NV (Dr Mazzorana).

In an effort to produce best-practice guidelines for wound management in the austere environment, the Wilderness Medical Society convened an expert panel charged with the development of evidence-based guidelines for the management of wounds sustained in an austere (dangerous or compromised) environment. Recommendations are made about several parameters related to wound management. These recommendations are graded based on the quality of supporting evidence and the balance between the benefits and risks or burdens for each parameter according to the methodology stipulated by the American College of Chest Physicians. This is an updated version of the original guidelines published inWilderness & Environmental Medicine2014;25(3):295-310.

Key words: wound care, wound management, wound closure, wound infection, burn care, blister care, evacuation, austere environment

Introduction

The skin is the largest organ system in the human body. In remote and wilderness environments, caring for injuries to the skin is a fundamental necessity. The reported incidence of injury varies considerably. A review of the National Outdoor Leadership School (NOLS) incident database from 1998 to 2002 included 1940 incidents of injuries, illness, and near-miss accidents over 630,937 program-days.(1) Nonathletic soft tissue injuries accounted for 31% of the incidents. In

emergency departments, 12 million visits for traumatic wounds are reported yearly.(2) Flores et al (3) reported 14.8% of 100,000 outdoor recreational injuries were lacerations. In a study of medical incidents and evacuations in the wilderness setting, McIntosh et al (4) noted that 4% constituted injuries to skin or wound infections, 3.7% were burns, and 2.7% were blisters. Burns, even if minor, can result in significant morbid- ity and the need for evacuation. In the above NOLS study, 5% of total injuries were burns. Of the 488 patients evacuated in that study, 7 (23% of burn victims) were because of burns. Many series of outdoor injuries show burns as 2% to 8% of wilderness injuries, but they account for a relatively high percentage of evacuations, morbidity, and mortality. (5–10) Although the incidence of wounds sustained in the wilderness varies, the numbers are significant. Even “minor”wounds such as blisters, abrasions, and small burns can present significant management challenges in a backcountry environment. In an effort to develop proper guidelines for basic wound management in the austere environment, based on the best existing evidence, an expert panel was convened to develop evidence-based guidelines.

Methods

A panel of experts in the field was convened at the Wilderness Medical Society Annual Meeting in Snowmass, CO, in July 2010. Members were selected on the basis of clinical interest or research experience. Relevant articles were identified through the PubMed and Cochrane Collaboration databases using key word searches with the appropriate terms corresponding to each topic. Peer-reviewed studies including randomized controlled trials, observational studies, and case series were reviewed, and the level of evidence supporting the conclusions was assessed. Abstract-only studies were not included. Conclusions from review articles were not considered in the formulation of recommendations but are cited in an effort to provide context. When no relevant studies were identified, the expert panel recommendation was based on perceptions of risk vs benefit derived from patient care experience. The panel used a consensus approach to develop recommendations regard- ing basic wound management in the wilderness. All final recommendations were unanimously approved. These recommendations have been graded on the basis of clinical strength as outlined by the American College of Chest Physicians (ACCP) (see onlineSupplementary ACCP Table 1). (11)

Results

Guidelines related to basic wound care in the austere environment, the evidence supporting them, and their recommendation grades are described subsequently. The purpose of this manuscript is to provide guidelines rather than to serve as an exhaustive literature review; therefore, only the most pertinent evidence relevant to the recommendations is succinctly discussed.

GOALS OF WOUND MANAGEMENT

The general goals of wound management in the wilderness environment should include the following: 1) achieve hemorrhage control; 2) minimize risk of infection; 3) promote optimal healing; 4) reduce discomfort and minimize disability associated with management; 5) minimize loss of function; 6) optimize cosmetic outcome; and 7) implement definitive care when possible and practical.

DEFINITIONS

For the purpose of discussion in this manuscript, the following terms are defined:

Wound type (by exposure)

- Clean: a simple wound (eg, cut produced by a blade) in an area of the body with low bacterial count, treated shortly after the wound occurred.
- Dirty: a wound in an area with a high bacterial count (eg, axilla, groin) or presenting late (46 hours after wounding) in which case bacterial counts are expected to be at levels that could increase risk of infection.
- Contaminated: a wound impregnated with organic soil (swamps, jungle), claylike soil, fecal mate- rial, or a wound already infected.

WOUND EVALUATION

A thorough history should be obtained, including an accurate history of the injury and the environment in which it occurred. Patients who are immunocompromised owing to an underlying medical condition or medication pose increased challenges to the remote provider. For example, diabetes, certain rheumatologic conditions, clotting disorders, and cancer, as well as a number of medications (eg, corticosteroids), can affect wound care and outcome. Immunization status (especially tetanus and rabies) is important to obtain. Although it is unlikely that immunizations (such as tetanus) will be available to the provider, status may be an important determinant of whether or not evacuation is necessary. Knowledge of the mechanism of injury and of the environment in which it occurred plays an important role in wound management. Animal and human bites may introduce complex bacteria, as may environments such as marine and those that may contain a significant concentration of fecal material or other contaminants. Patients with burns may have significant associated inhalation injury. The anatomic location and extent of a wound may have significant management implications. Important anatomic considerations include significant cosmetic area (especially the face), communication with a joint, association with neurovascular or tendon injury, location that may be associated with significant acute disability (impacting mobilization), and relationship to an associated foreign body. Thorough evaluation of the wound is important. This requires a blood less field and proper lighting, and may require anesthesia, all of which may pose challenges in the wilderness environment. Evaluation should include an assessment of neurovascular status and tendon function.

ANESTHESIA

Evaluation and management of a traumatic wound is facilitated by the appropriate use of anesthesia. Intra- dermal or subcutaneous injection is the most common method of anesthesia delivery. There are many commercial anesthetic preparations available for intradermal or subcutaneous delivery but the most common fall into 2 WMS Practice Guidelines for Basic Wound Management S classes: the amide class (eg, lidocaine and bupivacaine) and the ester class (eg, chloroprocaine). Pain from the injection itself can be lessened with the use of smaller gauge needles, warming the anesthetic solution to room temperature, and buffering lidocaine solutions (1 mL of sodium bicarbonate to 9 mL of lidocaine).(12,13) This buffering technique has been studied and is recommended with lidocaine only. The addition of epinephrine to these solutions causes vasoconstriction at the site and prolongs the effect of the anesthetic. It has been taught that epinephrine should be avoided when wounds involve anatomic areas with end arterioles, such as the digits, nose, penis, and earlobes, but there exists literature that supports safe utilization of this agent in digital blocks.(14,15) In addition to intradermal and subcutaneous injections, topical anesthetics have demonstrated efficacy, although a longer time (20–30 minutes) is required to achieve the desired effect.(16-20) The American Acad- emy of Pediatrics recommends the use of topical anesthetics, such as LET (4% lidocaine, 0.1% epinephrine, 0.5% tetracaine) for closure of simple lacerations.(18,19) Allergic reactions to anesthetic agents are very rare. If a history of allergic reaction is reported, utilization of an anesthetic from a different class is recommended. Use of diphenhydramine subcutaneously or intradermally has been noted in the literature for use in an anesthetic compound allergy.(17)

Recommendation

Intradermal or subcutaneous anesthesia may be used to facilitate wound evaluation and management. Recommendation grade: 1A.

Recommendation

Topical anesthesia can be used as an alternative to intradermal or subcutaneously injected anesthesia. Recommendation grade: 1B.

IMMUNIZATION

Tetanus

Tetanus is the only vaccine-preventable disease that is infectious but not contagious. The need for active immunization, with or without passive immunization, depends on the condition of the wound and the patient’s immunization history. Tetanus immunization status should be evaluated for all patients with a traumatic wound and treated appropriately based on the patient’s history and risk of infection.(21–23) Table 1 shows current recommendations for tetanus. Development of clinical tetanus can probably be delayed with oral antibiotics (penicillin and likely others) and should be used if evacuation of an

unimmunized patient with a tetanus- prone wound will be delayed or is logistically complicated. This technique is often used in patients claiming an allergy to tetanus toxoid.

Recommendation

Tetanus immunization, if indicated based on a patient's history and exposure, should be given to all patients with a traumatic wound. Recommendation grade: 1C.

Table 1.Tetanus recommendations (3)

History of immunization (previous toxoid doses received)	Clean and minor wounds	All other wounds[a]
Unknown/less than a series of 3 doses	Toxoid[b]	Toxoid and tetanus immune globulin
3 or more—last within 5 years	No prophylaxis	No prophylaxis
3 or more—last within 10 years	No prophylaxis	Toxoid
3 or more—last > 10 years	Toxoid	Toxoid and tetanus immune globulin

All immunocompromised patients should receive tetanus toxoid and immune globulin.

[a] Such as contaminated with soil, feces, or saliva. Includes puncture wounds, avulsions, missile injuries, crush wounds, burns, and frostbite. [b] Tetanus toxoid formulations are administered based on age. No toxoid administration should be given to an infants to 6 weeks of age.

Rabies

Although clinical rabies is an infrequently encountered disease in the developed world, substantial reservoirs of the disease are present in the United States, notably raccoons, skunks, and foxes. Details regarding rabies prophylaxis are beyond the scope of this paper. After rabies virus exposure in previously un-vaccinated persons, prompt postexposure prophylaxis (PEP) is nearly universally effective in preventing human rabies.(24) PEP combines local wound care, infiltration with human rabies immunoglobulin (HRIG) into and around the wound, and multiple doses of rabies cell-culture vaccine.

Recommendation

Rabies PEP is effective in preventing human rabies. Recommendation grade: 1B. In the wilderness setting, the one critical intervention that can be performed in the field is appropriate wound cleansing. This should be done with soap and water or, if available, irrigation with a virucidal agent (ie, povidone-iodine; or the more

commonly available chlorine dioxide). Local wound care may significantly decrease rabies risk. Wounds that might require suturing should undergo delayed primary closure at 4 days.

Recommendation

Wounds with a risk of rabies exposure should be irrigated, preferably with a virucidal agent, and closure should be delayed for 4 days. Recommendation grade: 1C. When an exposure has occurred, the likelihood of rabies infection varies with the nature and extent of the exposure. All bites, regardless of body site or evidence of gross trauma, represent a potential risk. Although risk for transmission might increase with wound severity, transmission also occurs from bites by some animals (eg, bats) that inflict rather subtle injury compared with larger-bodied carnivores. Prophylaxis for suspected rabies exposure is a medical urgency, but not an emergency, and requires definitive S120 Quinn et al medical care (the initiation of PEP), preferably within 24 to 48 hours. This will generally require evacuation, then prompt medical attention (in consultation with public health authorities). It should be noted that it is never too late to give PEP if clinical rabies has not yet developed.

WOUND CARE HEMOSTASIS

Hemostasis is an important part of wound care, both to prevent further blood loss and to allow examination and treatment of injuries.

Direct pressure

Direct pressure has been considered to be the gold standard for controlling hemorrhage since at leastAD30 during the Roman reign of Tiberius.(25) Despite this long history, it is only recently that research to evaluate direct pressure has been performed. Recent studies show that manual direct pressure generates an average of 180 mm Hg, which is sufficient to control most bleeding.(26) Multiple studies have also shown manual pressure to be effective for controlling bleeding after arterial puncture for catheterization.(27,28)

Recommendation

Direct pressure is the method of choice for acutely controlling hemorrhage in the vast majority of cases. Recommendation grade: 1B.

Pressure dressings

Pressure dressings have also been shown to provide effective hemostasis, providing about half the compression force of direct pressure (90 mm Hg).(29)

Recommendation

Once acute hemorrhage is controlled, pressure dressings represent the treatment of choice for maintenance of hemorrhage control in most wounds. Recommendation grade: 1B.

Extremity elevation

Elevation of a bleeding extremity to control hemorrhage is not supported or disputed by any controlled studies. There is however little downside to extremity elevation.(30

Recommendation

Extremity elevation may provide some value in hemorrhage control, with little significant risk, if doing so does not otherwise alter the immobilization or evacuation plan. Recommendation grade: 2C.

Pressure points

Pressure points had previously been recommended when direct pressure was ineffective in controlling bleeding. The only controlled study on pressure points, however, showed them to be of no utility in controlling bleeding.(31)

Recommendation

Pressure points have no role in hemorrhage control and attempts at implementation may delay the use of more effective methods. Recommendation grade: 1C.

Tourniquets

Tourniquets were once controversial. The successful use of tourniquets in saving lives by the military in the last 15 years has markedly changed the role of tourniquets for immediate hemostasis. Much of the prior controversy involved improper application of tourniquets by un- trained individuals or use of improper devices. Improper use or use of improper devices can worsen bleeding and morbidity. There is now little controversy concerning tourniquet use by individuals trained to use them properly.(32) The following recommendations are based on use of appropriate tourniquets by appropriately trained individuals.

Tourniquets are clearly indicated as the first intervention to control life-threatening arterial bleeds in extremities.(33,34) There is an abundance of literature to support this primary use of tourniquets.(35) Lower pressure bleed- ing can typically be controlled with direct pressure and pressure dressings as previously described, but if these methods are unsuccessful then tourniquets are indicated. Furthermore, situations exist in which a“tourniquet first” approach may be warranted as a stopgap. Any wound situation in the wilderness makes control of blood loss to be of very high priority. In these austere situations, any blood loss is detrimental and consideration should be given for a tourniquet to be applied to all extremity bleeding to provide immediate hemostasis. This allows one either to effect rescue without further blood loss or to control blood loss immediately until

an effective pressure dressing can be applied. Immediate tourniquet use is also recommended in mass casualty situations for immediate control of bleeding.(36,37) Other techniques can be used later to allow tourniquet removal. In these situations the goal is to apply the tourniquet quickly to control blood loss immediately. As soon as another method of bleeding control has been applied, the tourniquet can be released.

Recommendation

Tourniquet application is an effective means to control arterial bleeding and should be considered the primary intervention to control life-threatening arterial bleeds in extremities. Recommendation grade: 1A.

Recommendation

Tourniquet application is an effective means to control bleeding that has failed to substantially decrease with other less aggressive techniques. Recommendation grade: 1B.

Tourniquet basics

A tourniquet can be placed for up to 2 hours with mini- mal risk of complication.(38–40) A tourniquet must have a width sufficient to obstruct blood flow. The minimum acceptable width is 4 cm (1.5 inches). If placement of a tourniquet fails to control bleeding, a second tourniquet should be placed immediately adjacent and proximal to the first, thereby increasing the tourniquet width and its effectiveness. The tourniquet must have a windlass device of some sort; otherwise sufficient force to over- come arterial pressure cannot be developed. A standard belt cannot be used as a tourniquet because it cannot be pulled tight enough to occlude arterial flow. A tourniquet must always be applied with sufficient force to occlude arterial pressure, which can be confirmed by the absence of distal pulses after proper application.^41 Failure to do so, occluding only venous pressure, can increase bleeding.(42) When used as a stopgap technique, the tourniquet is applied immediately to control bleeding. The injured individual can then be moved or further treated until a proper dressing can be applied. Once this dressing is in place, the tourniquet can be released. If there is no further bleeding, the tourniquet is no longer required. If bleeding recurs, the tourniquet can be tensioned again to regain control of bleeding. A tourniquet that has been left on for longer than 2 hours should remain in place until definitive medical evaluation occurs.(43) There is no utility in the old recommendation of releasing a tourniquet every so often to allow a“little perfusion” to occur. Bleeding is either controlled in the first 2 hours or not. There are many cases of limb salvage with prolonged tourniquet times, or secondary to other reasons for lack of limb perfusion. Although individual variation exists, most limbs can tolerate up to 6 hours of ischemia time. After 2 hours of tourniquet time there is no evidence that the rate of limb salvage is improved with intermittent perfusion.(42,43)

Recommendation

A tourniquet should not be released for the sole purpose of providing intermittent perfusion. Recommendation grade: 1B.

Recommendation

Application of a tourniquet as a short-term stopgap means of hemorrhage control is appropriate when immediate control is necessary because of logistical considerations such as mass casualties, or if there is a need to move the patient immediately with removal of tourniquet as soon as another method of bleeding control has been applied. Recommendation grade: 1B.

Hemostatic agents

Hemostatic dressings are often used for significant bleed- ing when a tourniquet cannot be applied to a wound for hemostasis or when regular pressure dressings are in- effective. Wounds in the neck or other noncompressible areas, for example, are not amenable to direct pressure or tourniquet use. Numerous animal and military studies document the utility of hemostatic dressings in these cases.(44–48) Although some early hemostatic dressing had deleterious effects on wounds, this is not the case with the latest generation of dressings.(49) The only downside of the latest agents is their high cost in comparison to standard pressure dressings. Hemostatic agents are avail- able as both powder and impregnated gauze. Impregnated gauze is the most widely recommended form owing to concerns of powder washing out of the wound with arterial bleeds and concerns of embolization risk that have been seen with some powders. For proper use, the hemostatic agent must be placed directly into the wound and pressure applied for a minimum of 5 minutes. Hemostatic agents should be placed first into the wound, not on top of other standard dressings. There are many commercial hemostatic dressings on the market, with others continuously under development. The Department of Defense (DOD) through its Tactical Combat Casualty Care (TCCC) committee is an unbiased resource that constantly evaluates both these agents and tourniquets and provides recommendations based on the best available science. The TCCC recommendations, which represent a living document with continuous updates and training videos on tourniquet and hemostatic agent use, can be found at http://www.naemt.org/education/TCCC/guidelines_curriculum.

Recommendation

Hemostatic agents may be effective in hemorrhage control in situations where more traditional methods are not effective. Recommendation grade: 1B.

WOUND PREPARATION AND CLEANING

Characteristics of wound debris, rather than the debris itself,may be important determinants of wound infection. Wound debris that consists of large particles with- out electrical charge (eg, glass, gravel) is largely inert and is unlikely to contribute to wound infection or to impair healing. Organic soils (swamp, bog,

jungle) or soils with high clay content that hold ionic charge interfere markedly with leukocyte function and may decrease the amount of bacteria required for wound infection by a factor of 1000.(50) Soil contaminants in dirt, as well as the silica contained in dirt itself, cause an inflammatory reaction. Removal may facilitate healing. Contaminants with high bacterial content (eg, fecal material) should be diluted or removed when possible. For wounds sustained in the marine environment, irrigation with potable water should be considered because of the bacterial complexity and load of seawater. Recommendation An attempt at wound cleansing is recommended in the presence of high bacterial contaminants and dirt. Recommendation grade: 1C.

Recommendation

A foreign body composed of reactive or contaminated material should be removed in the field if removal can be performed easily and with a low risk of complication. Recommendation grade: 1C.

Retained foreign bodies larger than“debris”may pose a further challenge. Removal of foreign bodies that are composed of inert material and that are not readily visible on wound exploration may cause more harm than benefit. On the other hand, bodies composed of organic material can be very reactive and may cause significant inflammation. The following foreign bodies should be removed: reactive materials (eg, wood and vegetative material); contaminated clothing material; and any materials located in the foot, causing impingement of neurovascular structures or impairment of function. Large foreign bodies adjacent to, or penetrating into, vital structures or cavities, including the eye, should be left in place.

Aseptic technique

There is no evidence that sterile, rather than clean, technique decreases the incidence of wound infection after management of lacerations. Studies comparing the use of sterile vs non-sterile gloves for the management of lacerations (51,52) or for minor surgery (53) have shown no difference in infection rates.

Recommendation

Wounds should be treated using a clean field, including gloves and instruments; sterility is not necessary. Recommendation grade: 1A.

Irrigation

For the purpose of this discussion, the following definitions are recognized:

Volume

- Low: < 1L of fluid
- High: > 1L of fluid

Pressure

- Low: <6 psi (the pressure generated by a bulb syringe or pinhole in a fluid bag or container).
- High: 6–15 psi (the pressure generated by an intravenous catheter on a syringe).
- Very high: >15 psi (the pressure generated by a powered pulse irrigator on “high” setting).

The effectiveness of irrigation in decreasing the acute bacterial load in a contaminated wound and in removing foreign debris is well documented.(54,55) However, there exists less evidence supporting the role of irrigation in reducing the incidence of wound infection. High- pressure irrigation (6–15 psi) is more effective at removing bacteria and foreign debris than low-pressure irrigation. In the field, 8 psi can be exerted by fluid delivered by a 35-mL syringe through a 19-gauge needle. Very high pressure irrigation (> 15 psi) is more likely to cause direct tissue damage,(56–58) and to prop- agate bacteria and debris deeper into the wound.(56) Although very high pressure irrigation decreases bacterial counts acutely, it may result in a significantly greater rebound bacterial count at 48 hours (59) when compared with low- or high-pressure irrigation. Recent studies have shown that irrigation can also remove beneficial growth factors and chemokines in the wound exudate. One prospective randomized trial has shown a lower incidence of both wound infection and inflammation with high-pressure irrigation of wounds encountered in an emergency department.(58) Another prospective randomized study on open fractures showed a lower incidence of wound inflammation and infection with high pressure as opposed to very high pressure irrigation.(59) The optimal volume of irrigation that should be used is unknown. One study has demonstrated that increasing volume of irrigant from 0.1 L to 1 L increased the effectiveness of bacterial removal, but further increases up to 10 L provided no additional benefit.(60) High-pressure, low-volume irrigation results in less microscopic and macroscopic tissue damage and can be as effective as high-pressure, high-volume lavage at removing bacteria if performed within3 hours of contamination.(56–58) Wounds that are small (o1 cm), superficial, and not grossly contaminated likely would not benefit from irrigation.

Recommendation

The use of high-pressure irrigation (6–12 psi) is recommended to lower wound infection rates, especially in the case of open fractures. Recommendation grade: 1A.

Recommendation

Irrigation should be performed as quickly as practical as there is a direct correlation between timing and effective- ness of irrigation. Recommendation grade: 1B.

Recommendation

Irrigation should include at least 1 L of irrigant. Recommendation grade: 1C.

Irrigation: solution

Wounds irrigated with tap water have demonstrated an equivalent, or lower, incidence of infection compared with wounds irrigated using sterile saline solution.(45,61–65) A Cochrane review including 11 randomized controlled trials showed no statistically significant differences in infection rates between wounds (including acute and chronic wounds in adults and children) that were cleansed with tap water or normal saline solution.(66)

Recommendation

In a wilderness setting, potable water is the preferred solution for wound irrigation. Recommendation grade: 1A.

Irrigation: additives

Additives to irrigation solution have included antibiotics, antiseptics (povidone-iodine, benzalkonium chloride, chlorhexidine gluconate), and surfactants (such as castile soap). Although some of these agents have demonstrated efficacy in decreasing acute bacterial counts in contaminated wounds,(55,56,67) all are toxic to tissues, can increase problems associated with wound healing,(56,58,59,67) and result in a significant rebound bacterial count at 48 hours.(56,58,67)

Recommendation

If irrigation is performed, additives should not be used (except for rabies-prone wounds as discussed previously). Recommendation grade: 1A.

Debridement

Wounds closed with devitalized tissue present have a higher incidence of infection.(68) However, debridement requires surgical skill and knowledge and must balance tissue loss with function and ability to achieve closure without undue tension, if closure is part of the goal. If there is significant question regarding tissue viability, it is better to minimize debridement in favor of delayed primary closure.

Recommendation

Wounds with significant devitalized tissue should be left open. Recommendation grade: 1C.

Hair

Hair is probably not a significant source of wound contamination in itself. However, hair removal may be necessary to facilitate wound care or closure. Shaving has been shown to increase the wound infection rate by damaging the epithelium and hair follicles, resulting in a dermatitis.(69–71) The use of clippers is not associated with an increase in infection rate.(72)

Recommendation

If hair removal is required to facilitate wound care or closure, clipping rather than shaving should be used. Recommendation grade: IC.

LACERATIONS

Lacerations are one of the most common types of wounds encountered in the wilderness setting. Effective management of lacerations can be accomplished in the outdoor setting with basic supplies and technique. Primary vs delayed wound closure Although traditional teaching favors delayed primary closure (DPC), or closure by secondary intention, in many clinical situations, little data exist to demonstrate a higher incidence of wound infection with immediate primary closure. Primary wound closure has not been associated with a higher incidence of wound infection in several high-risk wounds including mammalian bites,(73,74) below-knee amputations resulting from land mines,(75) open fractures,(76–78) complicated appendicitis,(79,80) complex open abdominal military wounds,(81) and pilonidal sinus.(82) Conservatively, however, DPC results in the lowest risk of wound infection. DPC is a standard technique used in war wounds, all of which are considered to be markedly contaminated. Use of DPC has been shown to result in a very low risk of wound infection even in wounds in which infection would be likely. A study from the Vietnam War showed punji stake wounds treated with DPC to have an infection rate of only 2%, compared with nearly universal infection in those not treated with DPC.(83) Civilian observational studies have shown similar results with other contaminated wounds.(84)

Recommendation

Most wounds can be treated safely with acute primary closure. Grossly contaminated wounds should be packed open to allow for closure by secondary intention or delayed primary closure. Recommendation grade: 1B.

Table 2is a matrix with recommendations for wound care.

Timing of wound closure

In spite of the fact that most wounds are contaminated with bacteria, infection is unlikely to occur with less than 105 bacteria per gram of tissue.(85) An early animal model showed that a bacterial count greater than 10 (5) colony-forming units occurs in a traumatic wound within 5 hours of injury.(86) For many years, this period of 5 hours has been accepted as the“golden period,”after which the risk of infection is thought to dramatically increase. One study of hand and forearm wounds, with a small sample size, found significantly more infections in wounds presenting after 4 hours.(87) Another study found no difference in the frequency of hand wound infections regardless of time of presentation, up to 18 hours.(88) Similarly, another study showed no increase in wound failure when closed before 19 hours after injury.(89) Lammers et al (90) reported increased risk of infection after 10 hours (8 hours for hand wounds). A recent Cochrane review

attempted to compare primary closure vs DPC for nonbite traumatic wounds within 24 hours after injury, but no studies could be identified that met the inclusion criteria.(91)

Recommendation

Most clean wounds can be safely closed up to 6 hours after injury, up to 10 hours for face and scalp wounds. Recommendation grade: 1C.

Method of wound closure

The use of tissue glue (octyl cyanoacrylate) to close minor wounds is well supported by the literature.(92–95) Glue can be applied more quickly than sutures and causes less pain. Tissue adhesives are easy to transport and apply. High-tension wounds have a higher dehiscence rate when repaired with glue than with sutures.(96,97) Surgical tapes have the lowest tensile strength of any wound closure aid and therefore the highest rate of dehiscence.(98) In low-tension wounds, results are com- parable to the use of tissue adhesives alone.(99) Care should be taken to apply surgical tapes without exerting shear stress on the skin, particularly with the use of adhesive aids (eg, tincture of benzoin), as this may lead to blistering.

The“hair apposition technique”has been described and validated for the closure of scalp wounds.(100,101) Hair on either side of the wound can be knotted or twisted and secured with cyanoacrylate. This technique is most effective in small wounds, results in apposition of only the superficial skin layer, and does not provide much hemostasis.(102)

Staples and sutures probably yield equivalent rates of wound healing, surgical site infection, and dehiscence,(103) although one meta-analysis suggests a higher incidence of wound infection with the use of staples in orthopaedic surgery.(104) Wound stapling devices are easier to use than sutures but are bulkier to carry and result in an inferior cosmetic result.

Wound closure should always be performed with attention to cosmetic outcome. In areas of high visibility, such as the face, closure should be performed, when possible, with tissue adhesive, surgical tape, or fine sutures. Staples should never be used in these areas.

Recommendation

For most simple wounds, tissue adhesives provide an acceptable outcome, but with more complex wounds requiring closure under some tension, sutures or staples are preferred. Recommendation grade: 1A.

Table 2.Wound care matrix

Wound type	Irrigation pressure	Irrigation volume	Closure
Clean	Low	Low	Primary
Dirty	High	Low	Primary
Contaminated	High	High	DPC

If a question exists between 2wound types, consider it to be the “dirtier” of the choices.

Consider any wound presenting after 8–12 hours (24 hours if the face) to be contaminated.

All compression-induced stellate wounds (which occur when 2 bodies collide) should be considered dirty regardless of time or location.

Animal or human bites to any area other than the face should be considered contaminated. DPC, delayed primary closure.

Aftercare

When evaluation and treatment is complete, aftercare is important to protect the wound and assist the healing process. A moist wound environment (as opposed to a dry environment) has been a growing tenet in wound care and management since an article by Winter in 1962 showed that moisture promotes wound healing.^105 This has been supported by many subsequent studies.^106 –^108 This is best accomplished with the use of low-adherent dressings or semipermeablefilms. In the limited-resource environment, wet to dry gauze dressing or topical antibiotic ointment will provide reasonable alternatives. Although protecting the wound from contamination and using an appropriate dressing to help control exu- dates would seem to make practical sense, recent reviews have challenged these beliefs. A 2011 systematic review found no evidence to suggest that one dressing type was better than any other, that providing wound coverage was better at preventing infection than not providing coverage, or that any dressing type improved scarring, pain control, patient acceptance, or ease of removal.^109 Previous reviews have found no advantage for any specific type of dressing in traumatic wounds.^110 The downside risk, however, of covering a wound, particu- larly in the wilderness, with a clean bandage appears quite low. At the very least, a dressing facilitates management by protecting wounds from the intro- duction of noxious substances such as soil and organic material and absorbs exudate.

Recommendation

A moist wound environment is beneficial to healing. Recommendation grade: 1C.

Recommendation

A clean, protective wound bandage can be helpful in austere environments. Recommendation grade: 2C.

Summary recommendations for lacerations

Most lacerations can be safely closed in the wilderness. Grossly contaminated wounds should be left open and packed with wet to dry dressings. A clean, but not sterile, field is adequate for wound care. Obvious devitalized tissue should be debrided if it can be done so safely. Foreign debris should be removed, although the wound should not be extended for the purpose of retrieving foreign bodies not visible on gentle exploration of the wound. Wounds should be irrigated early with potable water. No additive should be added to the irrigation fluid or applied to the wound. Tissue adhesives are effective in wounds requiring a low-tension closure. Sutures or staples should be considered in more complex wounds requiring closure under tension. Staples should never be used on the face.

ABRASIONS

Recommendation

Abrasions should be managed with the same recommendations as lacerations. Rather than closure, these wounds should be covered with a nonadherent dressing. Recommendation grade: 1C.

FRICTION BLISTERS

Blisters are ubiquitous and disproportionately disabling wounds in the austere environment. A review of the medical risks of hiking the Appalachian Trail reported that foot blisters affected 64% of all hikers.(111) During a 12-month period of Operation Iraqi Freedom 1, the incidence of foot blisters was 33%.(112) Blisters are a prominent example of the adage that“an ounce of prevention is worth a pound of cure.”Single or double socks capable of wicking away moisture appear to be beneficial in preventing blisters.(113,114) One study showed a marked reduction in blisters in runners with the use of acrylic vs cotton socks.(115) Another study showed a reduction in the number and severity of blisters with a thin polyester undersock.(116) Little evidence exists to support a preventive role for moleskin, duoderm, adhesive tape, emollients, tincture of benzoin, or foot powders. In a double-blind, placebo-controlled study, aluminum-based antiperspirant (20% aluminum chloride hexahydrate solution without emollients or perfumes) applied to the soles of the feet for at least 3 consecutive days reduced blisters by 27% vs placebo.(117)

Small blisters (o5 mm in diameter) and hot spots should be protected with pressure relief, such as a donut- shaped pad (eg, moleskin), or covered with hydrogel or hydrocolloidal dressings. Blisters larger than 5 mm in diameter should be drained, but not unroofed, then covered with a hydrocolloid or hydrogel patch or equivalent (petrolatum or antibacterial ointment covered with gauze or moleskin).(118)

Little evidence exists to support the use of tissue adhesives for blister treatment. One study prospectively evaluated the use of 2-octyl cyanoacrylate compared with standard treatment and found a greater degree of procedural discomfort but no treatment advantage with the 2-octyl cyanoacrylate.(119)

Recommendation

Blister prevention is facilitated by the use of proper footwear and promoting a dry environment for the foot, including the use of a wicking sock system. Recommendation grade: 1C.

Recommendation

Small blisters (o5 mm in diameter) and hot spots should be protected with pressure relief, such as a donut-shaped pad (eg, moleskin), or covered with hydrogel or hydrocolloidal dressings. Recommendation grade: 1C.

Recommendation

Blisters larger than 5 mm in diameter should be drained, but not unroofed, then covered with a hydrocolloid or hydrogel patch or equivalent (petrolatum or antibacterial ointment covered with gauze or moleskin). Recommendation grade: 1C.

BURNS

A paucity of high-quality evidence exists to provide recommendations for wilderness burn wound care. Irrigation or submersion of the burned area in cool water has been shown to limit the extent of the burn and is helpful in controlling pain. Care must be taken to avoid tissue freezing and hypothermia.(120) A recent systematic review of dressings for superficial and partial-thickness burns demonstrated that silver sulfadiazine was consistently associated with poorer healing outcomes than biosynthetic and silicon-coated dressings, whereas hydrogel-treated burns had better healing outcomes than those treated with standard dressings.(121) Circumferential burns can result in compartment syndrome secondary to the constricting effect of the resulting eschar. Patients with circumferential burns should be watched for the development of compartment syndrome. In these patients, an escharotomy may be required.

Recommendation

Beyond routine wound care as described above, we are unable to provide specific recommendations regarding care of burn wounds based on existing evidence.

Recommendation

Escharotomy should be performed in circumferential burns with risk of compartment syndrome. Recommendation grade: 1A.

INFECTION PROPHYLAXIS AND PREVENTION

An overall principle of wound management is that whether or not prophylactic antibiotics are given, wounds should be monitored closely. Complications can develop rapidly or in an indolent manner. These include local secondary infection, undetected penetration of deeper structures, and systemic illnesses that can result from hematogenous seeding of organisms inoculated into the wound. With the exception of certain specific wound categories, there is scant evidence to support the routine use of systemic antibiotics for prophylaxis against wound infection. A notable exception is an open fracture, in which acute antibiotic administration significantly lowers the rate of infection.(54) ,(122) This is of particular significance given the substantial morbidity associated with subsequent osteomyelitis. Virtually all open wounds are colonized with micro- organisms, but this is usually without clinical consequences.^123 The presence of colonizing bacteria does not constitute infection.

A systematic review of mammalian bites showed a statistically significant reduction in the rate of infection with the use of prophylactic antibiotics after bites by humans but not after bites by cats or dogs, except bites of the hand.(124) There was a statistically significant reduction in the rate of infection with the use of prophylactic antibiotics after mammalian bites to the hand. Although dated, there is evidence to support the use of topical antibiotics to promote wound healing and decrease infection.(125–127)

Recommendation

Treatment with systemic antibiotics is indicated in the presence of open fractures. Recommendation grade: 1A.

Recommendation

Treatment with systemic antibiotics is indicated in the presence of human bites. Recommendation grade: 1B.

Recommendation

Treatment with systemic antibiotics is indicated in the presence of mammalian bites to the hand. Recommendation grade: 1B.

Recommendation

Use of topical antibiotics may promote wound healing and decrease the incidence of infection, with little downside risk in the nonallergic patient. Recommendation grade: 2C.

There is little compelling evidence to support the prophylactic use of antibiotics for burn wounds. One systematic review concluded that the use of topical silver sulfadiazine is associated with a significant increase in the rate of burn wound infections when compared with dressings or skin substitutes.(128) The same review concluded that there was not enough evidence to draw reliable

conclusions regarding the use of systemic antibiotics. Another systematic review concluded that there was insufficient evidence to support the use of silver-containing dressings or other topical agents in the prevention of infection.(129)

Recommendation

Treatment with systemic antibiotics is not indicated for prophylactic use in burn wounds. Recommendation grade: 1C.

Recommendation

Silver sulfadiazine may negatively affect wound healing and may increase infection rate. Recommendation grade: 1A.

WOUND INFECTIONS

Even with proper wound care, there is a 1% to 12% risk of infection.(130) If appropriate equipment and training are available, wounds with signs of infection after closure should be opened and any abscess collection should be drained. These maneuvers will increase the success of antibiotic treatment and will improve patient comfort. Elevation of the involved extremity may be helpful, particularly if there is a cellulitic component.(131) Because culture information will not be available and many infections are likely to be polymicrobial in nature, empiric therapy is indicated with a consideration of any unique environmental issues associated with the inoculum (marine environment, mammalian bites, etc). Anti- microbial selection may also be guided practically by which items are available in the first aid kit. Amoxicillin/ clavulanate is often a first choice for infected animal bites and other skin and soft tissue infections (SSTI).(132,133) Moxifloxacin is suitable for SSTI, includ- ing animal bites, in the penicillin-allergic patient and is a goodfirst-choice agent for infections caused by aquatic exposures,(134,135) which have a higher likelihood of a gram-negative microbial etiology.(136,137) Other suitable antibiotics may include oral second- or third-generation cephalosporins. These have better activ- ity against such relevant entities asPasteurellaspp, and oral anaerobes, and equal activity against staphylococci and streptococci compared withfirst-generation agents such as cephalexin, doxycycline, and trimethoprim-sulfamethoxazole. No single particular agent will be reliably effective in all scenarios. Furthermore, the data supporting the use of many antibiotics in specific clinical situations are supported less by randomized clinical trials and more by extrapolation of studies of the bacteriology of particular wounds or environments.

EVACUATION

Recommendation

Patients with the following wounds require care that is not available in a wilderness setting and should be evacuated. Recommendation grade: 1C:

- all complex wounds not closed primarily
- open fractures
- wounds with underlying tendon, joint, nerve, or vessel damage
- wounds secondary to mammalian bites
- any wound showing early signs of infection, if appropriate early antibiotics are not available
- wounds with progression of infection after administration of antibiotics
- wounds associated with a large foreign body, particularly if organic in nature
- wounds with symptoms of systemic toxicity (fever, alterations of consciousness, shock)
- wounds in the presence of hypothermia
- wounds with palpable gas in the soft tissues
- wounds with significant associated devitalized tissue
- tetanus-prone wounds requiring immunization
- bite wounds with any possibility of rabies inoculation
- burn wounds associated with any of the following:
    - airway inhalation injury
    - burns to the thorax that impair ventilation
    - significant burns to hands, feet, genitals, mucous membranes, or face
    - circumferential burns that are partial or full thickness
    - full-thickness burns 4 5% total body surface area
    - partial-thickness burns 4 10% to 20% total body surface area
    - infected burns
    - burns with uncontrolled pain
    - lightning injuries
    - electrical burns
    - chemical burns

Conclusions

Wounds represent a ubiquitous threat in the wilderness environment. Although many wounds in this environ- ment are relatively simple to treat, significant obstacles to management may present themselves when both the environment itself and lack of resources prove challeng- ing. Increasingly complex and severe wounds add to the challenge and can be life-threatening. The purpose of this review has been to provide evidence-based guide- lines for the basic management of wounds in the austere environment.

Supplementary tables

Supplementary ACCP Table 1 and Evidence Table 2 are available online at10.1016/j.wem.2014.08.015.

References

1. Leemon D, Schimelpfenig T. Wilderness injury, illness, and evacuation: National Outdoor Leadership School’s incident profiles, 1999–2002.Wilderness Environ Med. 2003;14:174– 182.

2. Singer AJ, Hollander JE, Quinn JV. Evaluation and management of traumatic laceration.N Engl J Med. 1997;337:1142– 1148.
3. Flores AH, Haileyesus T, Greenspan AI. National estimates of outdoor recreational injuries treated in emergency departments, United States, 2004–2005.Wil- derness Environ Med. 2008;19:91– 98.
4. McIntosh SE, Leemon D, Visitacion J, Schimelpfenig T, Fosnocht D. Medical incidents and evacuations on wilderness expeditions.Wilderness Environ Med. 2007; 18:298– 304. 5.Elliott TB, Elliot BA, Bixby MR. Risk factors associated with camp accidents.Wilderness Environ Med. 2003;14:2– 8.
5. Heggie TW, Heggie TM. Viewing lava safely: an epidemiology of hiker injury and illness in Hawaii Volcanoes National Park. Wilderness Environ Med. 2004;15:77– 81.
6. Heggie TW. Reported fatal and non-fatal incidents involving tourists in Hawaii Volcanoes National Park, 1992 – 2002.Travel Med Infect Dis. 2005;3:123–131.
7. Montalvo R, Wingard DL, Bracker M, Davidson TM. Morbidity and mortality in the wilderness.West J Med. 1998;168:248– 254.
8. Gentile DA, Morris JA, Schimelpfenig T, Bass SM, Auerbach PS. Wilderness injuries and illnesses. Ann Emerg Med. 1992;21:853– 861.
9. Fraga AM, Fraga GP, Noordenbos J, et al. Beach and campfire burns: a site of pleasure and tragedy.J Burn Care Res. 2010;31:184– 189.
10. Guyatt G, Gutterman D, Baumann M, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force.Chest. 2006;129:174–181.
11. Scarfone RJ, Jasani M, Gracely EJ. Pain of local anesthetics: rate of administration and buffering. Ann Emerg Med. 1998;31:36– 40.
12. Brogan GX, Singer AJ, Valentine SM, Thode HC Jr, Giarrusso E, Hollander JE. Comparison of wound infection rates using plain versus buffered lidocaine for anesthesia of traumatic wounds. Am J Emerg Med. 1997;15:25–28.
13. Thomson CJ, Lalonde DH, Denkler KA, Feicht AJ. A critical look at evidence for and against elective epi- nephrine use in thefinger.Plast Reconstr Surg. 2007; 119:260– 266.
14. Waterbook AL, Germann CA, Southall JC. Is epinephr- ine harmful when used with anesthetics for digital nerve blocks?Ann Emerg Med. 2007;50:472–475.
15. McCarron PA, Murphy DJ, Little C, McDonald J, Kelly OJ, Jenkins MG. Preliminary clinical assessment of polyvinyl alcohol–tetrahydroxyborate hydrogels as po- tential topical formulations for local anesthesia of lacerations.Acad Emerg Med. 2011;18:333– 339.
16. Hollander JE, Singer AJ. Laceration management.Ann Emerg Med. 1999;34:356– 367.
17. Zempsky WT, Cravero JP. American Academy of Pediatrics Committee on Pediatric Emergency Medicine and Section on Anesthesiology and Pain Medicine. Re- lief of pain and anxiety in pediatric patients in emergency medical systems.Pediatrics. 2004;114:1348– 1356.

18. Ferguson C, Loryman B, Body R. Best evidence topic report. Topical anaesthetic versus lidocaine infiltration to allow closure of skin wounds in children.Emerg Med J. 2005;22:507– 509.
19. Harmatz A. Local anesthetics: uses and toxicities.Surg Clin North Am. 2009;89:587– 598.
20. Chapman LE, Sullivent EE, Grohskopf LA, et al. Centers for Disease Control and Prevention (CDC). Recommendations for postexposure interventions to prevent infection with hepatitis B virus, hepatitis C virus, or human immunodeficiency virus, and tetanus in per- sons wounded during bombings and other mass-casualty events—United States, 2008: recommendations of the Centers for Disease Control and Prevention (CDC). MMWR Recomm Rep. 2008;57:1– 21.
21. McQuillan GM, Kruszon-Moran D, Deforest A, Chu SY, Wharton M. Serologic immunity to diphtheria and tetanus in the United States.AnnInternMed. 2002;136:660– 666.
22. Kretsinger K, Broder KR, Cortese MM, et al. Centers for Disease Control and Prevention; Advisory Committee on Immunization Practices; Healthcare Infection Control Practices Advisory Committee. Preventing tetanus, diph- theria, and pertussis among adults: use of tetanus toxoid,reduced diphtheria toxoid and acellular pertussis vaccine: recommendations of the Advisory Committee on Immu- nization Practices (ACIP) and recommendation of ACIP, supported by the Healthcare Infection Control Practices Advisory Committee (HICPAC), for use of Tdap among health-care personnel.MMWR Recomm Rep. 2006;55: 1 – 37.
23. Manning SE, Rupprecht CE, Fishbein D, et al. Advisory Committee on Immunization Practices Centers for Disease Control and Prevention (CDC). Human rabies prevention—United States, 2008: recommendations of the Advisory Committee on Immunization Practices. MMWR Recomm Rrep. 2008;57:1– 28.
24. Broughton G 2nd, Janis JE, Attinger CE. A brief history of wound care. Plast Reconstr Surg. 2006;117(7 suppl): 6S–11S. 26.Naimer SA, Anat N,Katif G, Rescue Team. Evaluation of techniques for treating the bleeding wound.Injury. 2004;35:974– 979.
25. Lehmann KG, Heath-Lange SJ, Ferris ST. Randomized comparison of hemostasis techniques after invasive cardiovascular procedures.Am Heart J. 1999;138:1118– 1125.
26. Koreny M, Riedmüller E, Nikfardjam M, Siostrzonek P, Müllner M. Arterial puncture closing devices compared with standard manual compression after cardiac catheter- ization: systematic review and meta-analysis. JAMA. 2004;291:350– 357.
27. Naimer SA, Chemla F. Elastic adhesive dressing treat- ment of bleeding wounds in trauma victims.Am J Emer Med. 2000;18:816– 819.
28. Markenson D, Ferguson JD, Chameides L, et al. First Aid Chapter Collaborators. Part 13: first aid: 2010 American Heart Association and American Red Cross International Consensus on First Aid Science With Treatment Recommendations.Circulation. 2010;122(suppl 2):S582–S605.
29. Swan KG Jr, Wright DS, Barbagiovanni SS, Swan BC, Swan KG. Tourniquets revisited.J Trauma. 2009;66: 672 – 675.

30. Caterson EJ, Carty MJ, Weaver MJ, Holt EF. Boston bombings: a surgical view of lessons learned from combat casualty care and the applicability to Boston's terrorist attack.J Craniofac Surg. 2013;24:1061– 1067.
31. Kragh JF Jr, Walters TJ, Baer DG, et al. Practical use of emergency tourniquets to stop bleeding in major limb trauma.J Trauma. 2008;64(2 suppl):S38–S50.
32. Kragh JF Jr, Walters TJ, Baer DG, et al. Survival with emergency tourniquet use to stop bleeding in major limb trauma.Ann Surg. 2009;249:1– 7.
33. Beekley AC, Sebesta JA, Blackbourne LH, et al. 31st Combat Support Hospital Research Group. Prehospital tourniquet use in Operation Iraqi Freedom: effect on hemorrhage control and outcomes.J Trauma. 2008; (2 suppl):S28–S37.
34. Doyle GS, Tailac PP. Tourniquets: a review of current use with proposals for expanded prehospital use.Pre- hosp Emerg Care. 2008;12:241– 256.
35. Kragh JF, O'Neill ML, Beebe DF, et al. Survey of the indications for use of emergency tourniquets.J Spec Oper Med. 2011;11:30– 38.
36. Tourtier JP, Palmier B, Tazarourte K, et al. The concept of damage control: extending the paradigm in the prehospital setting.Ann Fr Anesth Reanim. 2013;32:520– 526.
37. Ostman B, Michaelsson K, Rahme H, Hillered L. Tourniquet-induced ischemia and reperfusion in human skeletal muscle. Clin Orthop Relat Res. 2004;418: 260 – 265.
38. Klenerman L. Tourniquet time—how long?Hand. 1980; 12:231– 234.
39. King DR, van der Wilden G, Kragh JF Jr, Blackbourne LH. Forward assessment of 79 prehospital battlefield tourniquets used in the current war.J Spec Oper Med. 2012;12:33– 38.
40. Pedowitz RA, Gershuni DH, Fridén J, Garfin SR, Rydevik BL, Hargens AR. Effects of reperfusion intervals on skeletal muscle injury beneath and distal to a pneumatic tourniquet.JHandSurgAm. 1992;17:245– 255.
41. Jacobsen MD, Pedowitz RA, Oyama BK, Tryon B, Gershuni DH. Muscle functional deficits after tourniquet ischemia.Am J Sports Med. 1994;22:372–377.
42. Howe N, Cherpelis B. Obtaining rapid and effective hemostasis: part I. Update and review of topical hemosta- tic agents.JAmAcadDermatol. 2013;69: 659.e1–659.e17.
43. Wedmore I, McManus JG, Pusateri AE, Holcomb JB. A special report on the chitosan-based hemostatic dressing: experience in current combat operations. J Trauma. 2006;60:655– 658.
44. Brown MA, Daya MR, Worley JA. Experience with chitosan dressings in a civilian EMS system.J Emerg Med. 2009;37:1– 7.
45. Kheirabadi B. Evaluation of topical hemostatic agents for combat wound treatment.US Army Med Dep J. 2011:25– 37.
46. Lairet JR, Bebarta VS, Burns CJ, et al. Prehospital interventions performed in a combat zone: a prospective multicenter study of 1,003 combat wounded.J Trauma Acute Care Surg. 2012;73(2 suppl 1):S38–S42.
47. Rhee P, Brown C, Martin M, et al. QuikClot use in trauma for hemorrhage control: case series of 103 documented uses.J Trauma. 2008;64:1093–1099.

48. Rodeheaver GT, Pettry D, Turnbull V, Edgerton MT, Edlich RF. Identification of wound infection-potentiation factors in soil.Am J Surg. 1974;128:8– 14.
49. Perelman VS, Francis GJ, Rutledge T, Foote J, Martino F, Dranitsaris G. Sterile versus nonsterile gloves for repair of uncomplicated lacerations in the emergency department: a randomized controlled trial.Ann Emerg Med. 2003;43:362– 370.
50. Creamer J, Davis K, Rice W. Sterile gloves: do they make a difference?Am J Surg. 2012;204:976– 980.
51. Rhinehart MB, Murphy MM, Farley MF, Albertini JG. Sterile versus nonsterile gloves during Mohs micro- graphic surgery: infection rate is not affected.Dermatol Surg. 2006;32:170– 176.
52. Anglen JO. Wound irrigation in musculoskeletal injury. J Am Acad Orthop Surg. 2001;9:219– 226.
53. Quinn RH, Macias DJ. The management of open frac- tures.Wilderness Environ Med. 2006;17:41– 48.
54. Bhandari M, Schemitsch EH, Adili A, Lachowki R, Shaughnessy SG. High and low pressure pulsatile lavage of contaminated tibial fractures: an in vitro study of bacterial adherence and bone damage.J Orthop Trauma. 1999;13:526– 533.
55. Owens BD, White DW, Wenke JC. Comparison of irrigation solutions and devices in a contaminated mus- culoskeletal wound survival model.J Bone Joint Surg Am. 2009;91:92– 98.
56. Longmire AW, Broom LA, Burch J. Wound infection following high-pressure syringe and needle irrigation. Am J Emerg Med. 1987;5:179– 181.
57. FLOW Investigators. Petrisor B, Sun X, Bhandari M, et al. Fluid lavage of open wounds (FLOW): a multi- center, blinded, factorial pilot trial comparing alternative irrigating solutions and pressures in patients with open fractures.J Trauma. 2011;71:596– 606.
58. Gainor BJ, Hockman DE, Anglen JO, Christensen G, Simpson WA. Benzalkonium chloride: a potential disinfect- ing irrigation solution.JOrthopTrauma. 1997;11:121– 125. 61.Resende M, Hochman B, Gragnani A, et al. Tap water has no influence on microbial colonization of skin wounds in rats.Wounds. 2012;24:275– 282.
59. Valente JH, Forte RJ, Fruendlich JF, Zandieh SO, Crain EF. Wound irrigation in children: saline solution or tap water?Ann Emerg Med. 2003;41:609– 616.
60. Griffiths RD, Fernendez RS, Ussia CA. Is tap water a safe alternative to normal saline for wound irrigation in the community setting?JWoundCare. 2001;10:407– 411.
61. Moscati RM, Mayrose J, Reardon RF, Janicke DM, Jehle DV. A multicenter comparison of tap water versus sterile saline for wound irrigation.Acad Emerg Med. 2007;14: 404 – 409.
62. Svoboda SJ, Owens BD, Gooden HA, Melvin ML, Baer DG, Wenke JC. Irrigation of potable water versus normal saline in a contaminated musculoskeletal wound model.J Trauma. 2008;64:1357– 1359. 66.Fernandez R, Griffiths R. Water for wound cleansing. Cochrane Database Syst Rev. 2012;2:CD003861.
63. Anglen JO. Comparison of soap and antibiotic solutions for irrigation of lower-limb open fracture wounds. A prospective, randomized study.J Bone Joint

Surg Am. 2005;87:1415– 1422.
64. Haury B, Rodeheaver G, Vensko J, Edgerton MT, Edlich RF. Debridement: an essential component of traumatic wound care.Am J Surg. 1978;135:238–242.
65. Dineen P, Dursin L. Epidemics of postoperative wound infections associated with hair carriers.Lancet. 1973;2: 1157 – 1159.
66. Seropian R, Reynolds BM. Wound infections after preoperative depilation versus razor preparation.Am J Surg. 1971;121:251– 254.
67. Cruse PJE, Foord R. Afive-year prospective study of 23,649 surgical wounds.Arch Surg. 1973;107:206– 210.
68. Alexander JW, Fischer JE, Boyajian M, Palmquist J, Morris MJ. The influence of hair-removal methods on wound infections.Arch Surg. 1983;118:347– 351.
69. Chen E, Hornig S, Shepherd SM, Hollander JE. Primary closure of mammalian bites.Acad Emerg Med. 2000;7: 157 – 161.
70. Paschos NK, Makris EA, Gantsos A, Georgoulis AD. Primary closure versus non-closure of dog bite wounds. A randomised controlled trial. Injury. 2014;45:237– 240.
71. Ateşalp AS, Erler K, Gür E, Solakoglu C. Below-knee amputations as a result of land-mine injuries: comparison of primary closure versus delayed primary closure.J Trauma. 1999;47:724– 727.
72. Hohmann E, Tetsworth K, Radziejowski MJ, Wiesniewski TF. Comparison of delayed and primary wound closure in the treatment of open tibial fractures.Arch Orthop Trauma Surg. 2007;127:131– 136.
73. Rajasekaran S, Dheenakhayalan J, Babu JN, Sundarajan SR, Venkatramani H, Sabapathy SR. Immediate primary skin closure in type-III A and B open fractures.J Bone Joint Surg Br. 2009;91:217– 224.
74. Weitz-Marshall AD, Bosse MJ. Timing of closure of open fractures.J Am Acad Orthop Surg. 2002;10:379– 384.
75. Henry MCW, Moss RL. Primary versus delayed wound closure in complicated appendicitis: an international systematic review and meta-analysis.Pediatr Surg Int. 2005;21:625– 630.
76. Duttaroy DD, Jitendra J, Duttaroy B, et al. Management strategy for dirty abdominal incisions: primary or delayed primary closure? A randomized trial.Surg Infect (Larchmt). 2009;10:129– 136.
77. Vertrees A, Greer L, Pickett C, et al. Modern management of complex open abdominal wounds of war: a 5-year experience.J Am Coll Surg. 2008;207:801– 809.
78. Al-Khamis A, McCallum I, King PM, Bruce J. Healing by primary versus secondary intention after surgical treatment for pilonidal sinus.Cochrane Database Syst Rev. 2010;1:CD006213.
79. Shepard GH, Rich NM, Dimond F. Punji stick wounds: experience with 342 wounds in 324 patients.Ann Surg. 1967;166:902– 907.
80. Shepard GH. Wounds treated by healing of delayed primary closure: a clinical study.Mil Med. 1981;146:473– 475.
81. Cooney WP 3rd, Fitzgerald RH Jr, Dobyns JH, Washington JA 2nd. Quantitative wound cultures in upper extremity trauma.J Trauma. 1982;22:112– 117.

82. Robson MC, Duke WF, Krizek TJ. Rapid bacterial screening in the treatment of civilian wounds.J Surg Res. 1973;14:426– 430.
83. Morgan WJ, Hutchinson D, Johnson HM. The delayed treatment of wounds to the hand and forearm under antibiotic cover.Br J Surg. 1980;67:140– 141.
84. Nylén S, Carlsson B. Time factor, infection frequency and quantitative microbiology in hand injuries: a prospective study.Scand J Plast Reconstr Surg. 1980;14:185– 189.
85. Berk WA, Osborne DD, Taylor DD. Evaluation of the “golden period”for wound repair: 204 cases from a Third World emergency department.Ann Emerg Med. 1988;17:496– 500.
86. Lammers RL, Hudson DL, Seaman ME. Prediction of traumatic wound infection with a neural network-derived decision model.Am J Emerg Med. 2003;21:1– 7.
87. Eliya MC, Banda GW. Primary closure versus delayed closure for non bite traumatic wounds within 24 hours post injury. Cochrane Database Syst Rev. 2013;10: CD008574.
88. Coulthard P, Esposito M, Worthington HV, van der Elst M, van Waes OJ, Darcy J. Tissue adhesives for closure of surgical incisions. Cochrane Database Syst Rev. 2010;5:CD004287.
89. Coulthard P, Worthington H, Esposito M, Elst M, Waes OJ. Tissue adhesives for closure of surgical incisions. Cochrane Database Syst Rev. 2004;2:CD004287.
90. Singer AJ, Quinn JV, Clarke RE, Hollander JE. Trau- maSeal Study Group Closure of lacerations and incisions with octylcyanoacrylate: a multicenter randomized con- trolled trial.Surgery. 2002;131:270– 276.
91. Farion KJ, Osmond MH, Hartling L, et al. Tissue adhe- sives for traumatic lacerations: a systematic review of randomized controlled trials.Acad Emerg Med. 2003;10: 110 – 118.
92. Bernard L, Doyle J, Friedlaender SF, Eichenfield LF, Gibbs NF, Cunningham BB. A prospective comparison of octyl cyanoacrylate tissue adhesives (Dermabond) and suture for the closure of excisional wounds in children and adolescents. Arch Dermatol. 2001;137: 1177 – 1180.
93. Switzer EF, Dinsmore RC, North JH Jr. Subcuticular closure versus Dermabond: a prospective randomized trial.Am Surg. 2003;69:434– 436.
94. Rodeheaver GT, Spengler MD, Edlich RF. Performance of new wound closure tapes.JEmergMed. 1987;5:451– 462.
95. Zempsky WT, Parrotti D, Grem C, Nichols J. Random- ized controlled comparison of cosmetic outcomes of simple facial lacerations closed with Steri Strip Skin Closures or Dermabond tissue adhesive.Pediatr Emerg Care. 2004;20:519– 524.
96. Davies MJ. Scalp wounds: an alternative to suture. Injury. 1988;19:375– 376.
97. Hock MOE, Ooi SBS, Saw SM, Lim SH. A randomized controlled trial comparing hair apposition technique with tissue glue to standard suturing in scalp lacerations (HAT study).Ann Emerg Med. 2002;40:19– 26.
98. Singer AJ. Hair apposition for scalp lacerations.Ann Emerg Med. 2002;40:27– 29.
99. Biancari F, Tiozzo V. Staples versus sutures for closing leg wounds after vein graft harvesting for coronary artery bypass surgery.Cochrane Database Syst

Rev. 2010;5: CD008057.
100. Smith TO, Sexton D, Mann C, Donnell S. Sutures versus staples for skin closure in orthopaedic surgery: meta- analysis.BMJ. 2010;340:c1199.
101. Winter G. Formation of the scab and rate of epithelization of superficial wounds in the skin of the young domestic pig.Nature. 1962;193:293– 294.
102. Wiechula R. The use of moist wound-healing dressings in the management of split-thickness skin graft donor sites: a systematic review.Int J Nurs Pract. 2003;9:S9–S17.
103. Hackl F, Kiwanuka E, Philip J, et al. Moist dressing coverage supports proliferation and migration of trans- planted skin micrografts in full-thickness porcine wounds.Burns. 2014;40:274– 280.
104. SvensjöT, Pomahac B, Yao F, Slama J, Ericksson E. Accelerated healing of full-thickness skin wounds in a wet environment.Plast Reconstr Surg. 2000;106:602– 614.
105. Dumville JC, Walter CJ, Sharp CA, Page T. Dressings for the prevention of surgical site infection.Cochrane Database Syst Rev. 2011;7:CD003091.
106. Chaby G, Senet P, Vaneau M, et al. Dressings for acute and chronic wounds: a systematic review.Arch Derma- tol. 2007;143:1297– 1304.
107. Boulware DR, Forgey WW, Martin WJ 2nd. Medical risks of wilderness hiking.Am J Med. 2003;114:288– 293.
108. Brennan FH Jr, Jackson CR, Olsen C, Wilson C. Blisters on the battlefield: the prevalence of and factors associated with foot friction blisters during Operation Iraqi Freedom 1.Mil Med. 2012;177:157– 162.
109. Knapik JJ, Reynolds KL, Duplantis KL, Jones BH. Friction blisters: pathophysiology, prevention and treat- ment.Sports Med. 1995;20:136– 147.
110. Van Tiggelen D, Wickes S, Coorevits P, Dumalin M, Witvrouw E. Sock systems to prevent foot blisters and the impact on overuse injuries of the knee joint.Mil Med. 2009;174:183– 189.
111. Herring KM, Richie DH Jr. Comparison of cotton and acrylic socks using a generic cushion sole design for runners.J Am Podiatr Med Assoc. 1993;83:515– 522.
112. Knapik JJ, Hamlet MP, Thompson KJ, Jones BH. Influence of boots-sock systems on frequency and severity of foot blisters.Mil Med. 1996;161:594– 598.
113. Knapik JJ, Reynolds KL, Barson J. Influence of an antiperspirant on foot blister incidence during cross- country hiking.J Am Acad Dermatol. 1998;39:202– 206.
114. Cortese TA Jr, Fukuyama K, Epstein W, Sulzberger MB. Treatment of friction blisters: an experimental study. Arch Dermatol. 1968;97:717– 721.
115. Levy PD, Hile DC, Hile LM, Miller MA. A prospective analysis of the treatment of friction blisters with 2-octylcyanoacrylate.J Am Podiatr Med Assoc. 2006;96: 232 – 237.
116. Cuttle L, Kravchuk O, Wallis B, Kimble RM. An audit of first-aid treatment of pediatric burns patients and their clinical outcome.J Burn Care Res. 2009;30:1028– 1034.
117. Wasiak J, Cleland H, Campbell F, Spinks A. Dressings for superficial and partial thickness burns. Cochrane Database Syst Rev. 2013;3:CD002106.

118. Gosselin RA, Roberts I, Gillespie WJ. Antibiotics for preventing infection in open limb fractures.Cochrane Database Syst Rev. 2004;1:CD003764.
119. Lipsky BA, Hoey C. Topical antimicrobial therapy for treat- ing chronic wounds.Clin Infect Dis. 2009;49:1541– 1549.
120. Medeiros I, Saconato H. Antibiotic prophylaxis for mammalian bites.Cochrane Database Syst Rev. 2001;2: CD001738.
121. Dire DJ, Coppola M, Dwyer DA, Lorette JJ, Karr JL. Prospective evaluation of topical antibiotics for prevent- ing infections in uncomplicated soft-tissue wounds repaired in the ED.Acad Emerg Med. 1995;2:4– 10.
122. Leyden JJ, Bartlett NM. Comparison of topical anti- biotic ointments, a wound protectant, and antiseptics for the treatment of human blister wounds contaminated withStaphylococcus aureus.J Fam Pract. 1987;24: 601 – 604.
123. Maddox JS, Ware JC, Dillon HC Jr. The natural history of streptococcal skin infection: prevention with topical antibiotics.J Am Acad Dermatol. 1985;13:207– 212.
124. Barajas-Nava LA, López-Alcalde J, Roqué i Figuls M, Solà I, Bonfill Cosp X. Antibiotic prophylaxis for preventing burn wound infection.Cochrane Database Syst Rev. 2013;6:CD008738.
125. Storm-Versloot MN, Vos CG, Ubbink DT, Vermeulen H. Topical silver for preventing wound infection.Cochrane Database Syst Rev. 2010;3:CD006478.
126. Cummings P, Del Beccarro MA. Antibiotics to prevent infection of simple wounds: a meta-analysis of random- ized studies.Am J Emerg Med. 1995;13:396– 340.
127. Armstrong DG, Nguyen HC. Improvement in healing with aggressive edema reduction after debridement of foot infection in diabetes.Arch Surg. 2000;135:1405– 1409.
128. Brakenbury PH, Muwanga C. A comparative double blind study of amoxycillin/clavulanate vs placebo in the prevention of infection after animal bites.Arch Emerg Med. 1989;6:251– 256.
129. Goldstein EJ, Reinhardt JF, Murray PM, Finegold SM. Outpatient therapy of bite wounds. Demographic data, bacteriology, and a prospective, randomized trial of amoxicillin/clavulanic acid versus penicillinþ/- diclox- acillin.Int J Dermatol. 1987;26:123– 127.
130. Vick-Fragoso R, Hernández-Oliva G, Cruz-Alcázar J, et al. STIC Study Group, Efficacy and safety of sequential intravenous/oral moxifloxacin vs intravenous/ oral amoxicillin/clavulanate for complicated skin and skin structure infections.Infection. 2009;37:407– 417.
131. Giordano P, Song J, Pertel P, Herrington J, Kowalsky S. Sequential intravenous/oral moxifloxacin versus intra- venous piperacillin-tazobactam followed by oral amoxicillin-clavulanate for the treatment of complicated skin and skin structure infection.Int J Antimicrob Agents. 2005;26:357– 365.
132. Auerbach PS, Yajko DM, Nassos PS, Kizer KW, Morris JA Jr, Hadley WK. Bacteriology of the freshwater environment: implications for clinical therapy. Ann Emerg Med. 1987;16:1016– 1022.
133. Auerbach PS, Yajko DM, Nassos PS, et al. Bacteriology of the marine environment: implications for clinical therapy.Ann Emerg Med. 1987;16:643–649.

ANZCOR Guideline 9.2.1 - Recognition and First Aid Management of Suspected Heart Attack

Summary

Who does this guideline apply to?

This guideline applies to adults.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. First aiders should send for an ambulance if symptoms are severe, get worse quickly or last longer than 10 minutes. [Good Practice Statement]
2. First aiders should stay with the person until the ambulance or on-site resuscitation team takes over care. [Good Practice Statement]
3. We suggest that first aiders give aspirin (300 mg orally) to adults with non-traumatic chest pain unless the person has known anaphylaxis to aspirin.[1] [CoSTR 2020: weak recommendation, very low certainty evidence]

Abbreviations

ANZCOR: Australian and New Zealand Committee on Resuscitation CoSTR: Consensus on Science with Treatment Recommendations (from International Liaison Committee on Resuscitation - ILCOR) AED: Automated External Defibrillator

Guideline

1 Introduction

A person experiences a heart attack when there is a sudden partial or complete blockage of one of the coronary arteries that supply the heart muscle. As a result of the interruption to the blood supply, there is an immediate risk of life-threatening changes to the heart rhythm. If not corrected quickly there is also a risk of serious, permanent heart muscle damage. To reduce the chance of sudden death from heart attack, urgent medical care is required - '*every minute counts*'.

Heart attack is different from, but may lead to, cardiac arrest. Cardiac arrest is cessation of heart action.

Survival after heart attack can be improved by current treatments[1] and clot-dissolving medications that clear the blocked artery, restore blood supply to the heart muscle and limit damage to the heart. These therapies are most effective if administered as soon as possible following the onset of symptoms with these benefits declining with delays in treatment.

2 Recognition

For some people, sudden cardiac arrest may occur as the first sign of heart attack - however most experience some warning signs. It is important to note:

- a heart attack can occur in a person without chest pain or discomfort. The most common symptom of heart attack in a person without chest pain is shortness of breath
- a person who experiences a heart attack may pass off their symptoms as 'just indigestion'

2.1 Warning signs/Red Flags

If the warning signs are severe, get worse quickly, or last longer than 10 minutes, **act immediately**. The person may experience one or more of these symptoms:

- pain or discomfort in the chest, neck, jaw or arms
- pale skin
- shortness of breath
- nausea or vomiting
- sweating
- feeling dizzy or light-headed.

Discomfort or pain in the centre of the chest may start suddenly or come on slowly over minutes. It may be described as tightness, heaviness, fullness or squeezing. The pain may be severe, moderate or mild. The pain may be limited to, or spread to, the neck, throat, jaw, either or both shoulders, the back, either or both arms and into the wrists and hands.

Atypical chest pain is defined as pain that does not have a heaviness or squeezing sensation (typical angina symptoms), precipitating factors (e.g., exertion), or usual location.

Some people are more likely to describe atypical[2] or minimal symptoms[3] and include:

- the elderly
- women
- people with diabetes and/or chronic inflammatory conditions (eg Rheumatoid arthritis)
- Aboriginal and Torres Straight Islanders, Māori and Pasifika people

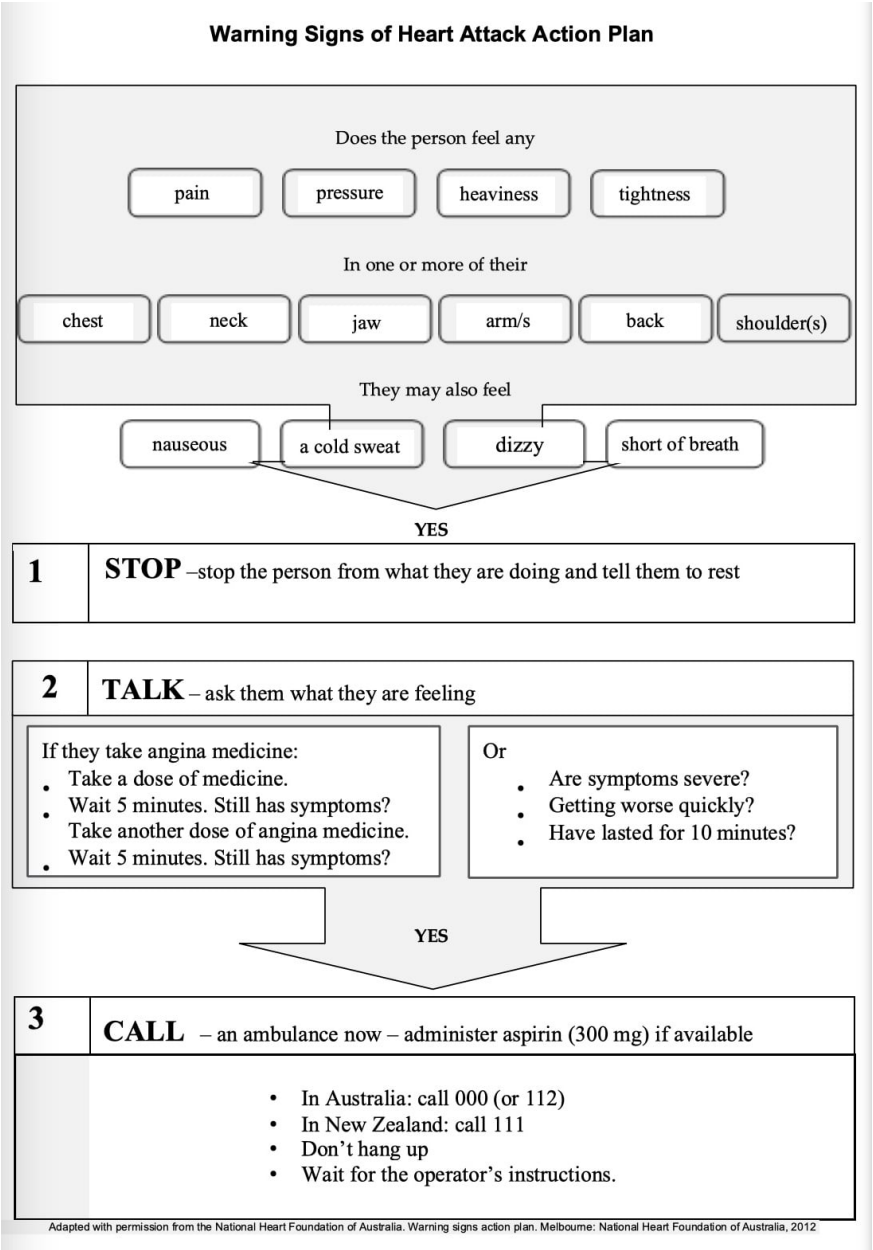
These people should seek urgent assessment by a health care professional if they have any warning signs of heart attack, no matter how mild.

3 Management

- Encourage the person to stop what they are doing and to rest in a comfortable position.
- If the person has been prescribed medication such as a tablet or oral spray to treat episodes of chest pain or discomfort associated with angina, assist them to take this as they have been directed.
- Send for an ambulance if symptoms are severe, get worse quickly or last longer than 10 minutes.
- Follow the instructions of the ambulance call taker/operator who will advise you what to do.
- Stay with the person until the ambulance or on-site resuscitation team arrives.
- ANZCOR suggests to give aspirin (300 mg orally) to adults with non-traumatic chest pain unless the person has known anaphylaxis to aspirin [CoSTR 2020: weak recommendation, very low certainty evidence]
- ANZCOR suggests against the routine administration of oxygen in persons with suspected heart attack. [COSTR 2015, weak recommendation, very-low certainty evidence] Administer oxygen only if there are obvious signs of shock or evidence of low oxygen saturation according to [Refer to ANZCOR Guideline 9.2.10][5],
- If practical and resources allow, locate the closest AED and bring it to the person.

If the person is unresponsive and not breathing normally, commence resuscitation following the Basic Life Support Flowchart [Refer to ANZCOR Guideline 8].

Warning Signs of Heart Attack Action Plan



References

1. Djarv T, Swain J M, Chang W, et al. (February 03, 2020) Early or First Aid Administration Versus Late or In-hospital Administration of Aspirin for Non-traumatic Adult Chest Pain: A Systematic Review. Cureus 12(2): e6862. doi:10.7759/cureus.
2. 2011 Addendum to the National Heart Foundation/Cardiac Society of Australia and New Zealand Guidelines for the Management of Acute Coronary Syndromes (ACS) 2006. Heart, Lung and Circulation Volume 20, Issue 8, Aug 2011, Pages 487–
3. Patel H, Rosengren A, Ekman I, et al. (2004) Symptoms in acute coronary syndromes: Does sex make a difference? American Heart Journal. 148(1):27-33.

4. Canto J, Rogers W, Goldberg R, et al. (2012) Association of Age and Sex With Myocardial Infarction Symptom Presentation and In-Hospital Mortality. Journal of the American Medical Association. 307(8):813- 822
5. Nikolaou NI, Welsford M, Beygui F, et al. Part 5: Acute coronary syndromes 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2015; **95** : e121-e46.
6. Chew D, Scott I, Cullen L, et al. National Heart Foundation of Australia and Cardiac Society of Australia and New Zealand: Australian clinical guidelines for the management of acute coronary syndromes 2016. *Med J Aust* 2016; **205** (3): 128

Further Reading

- National Heart Foundation (Australia). Heart Attack Facts. http://www.heartattackfacts.org.au
- National Heart Foundation (Australia) Warning signs of heart attack action plan. http://www.heartattackfacts.org.au/action_plans/HeartAttackActionPlan-english.pdf
- ANZCOR Guideline 7 External Automated Defibrillation in Basic Life Support
- ANZCOR Guideline 8 Cardiopulmonary Resuscitation
- ANZCOR Guideline 14 ACS: Overview & summary
- ANZCOR Guideline 14.1 ACS: Presentation with ACS
- ANZCOR Guideline 14.2 ACS: Initial Medical Therapy

About this Guideline:

- Search date/s: October 2020
- Question/PICO:
  - Population: Adults who experience non-traumatic chest pain
  - Intervention: early or first aid administration of aspirin
  - Comparators: late or in-hospital administration of aspirin
  - Outcomes: Survival, complications and incidence of cardiac arrest were ranked as critical outcomes. Cardiac functional outcome, infarct size and chest pain resolution were ranked as important outcomes.
- Study Designs: Randomized controlled trials (RCTs) and non- randomized studies (non-randomized controlled trials, interrupted time series, controlled before-and-after studies, cohort studies), case series of 5 or more subjects were eligible for inclusion. Unpublished studies (e.g., conference abstracts, trial protocols) were excluded.
- Timeframe: All years and all languages were included; unpublished studies (e.g., conference abstracts, trial protocols) were excluded. Literature search updated to October 22, 2019.
- Method: Systematic Review (ILCOR First Aid Task Force, CoSTR) search in 2019
- Primary reviewers: Natalie Hood, Finlay Macneil
- Other consultation: Dion Stub

- Worksheet: See https://www.ilcor.org/
- Approved: April 2021
- Guideline superseded: ANZCOR Guideline 9.2. 1 - August 2016

ANZCOR Guideline 9.2.2 - Stroke

Summary

This guideline has been updated based on updates to the 2020 International Liaison Committee on Resuscitation (ILCOR) evidence review,[1,2] the National Stroke Foundation Clinical Guidelines[3] and the Thoracic Society of Australia and New Zealand Oxygen Guidelines for Acute Oxygen Use in Adults.[4]

Who does this guideline apply to?

This guideline applies to adults and children.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. If the person is unresponsive and not breathing normally, commence resuscitation following the Basic Life Support Flowchart [Refer to ANZCOR Guideline 8].
2. If the person becomes unconscious but is breathing to lay the person on their side and ensure airway is clear [Refer to ANZCOR Guideline 3].
3. We recommend using a validated stroke assessment system to assist stroke recognition.[1] [CoSTR 2020, strong recommendation, low certainty evidence]
4. We suggest the use of the Facial drooping, Arm weakness, Speech difficulties and Time to call emergency services (FAST) stroke assessment for individuals with suspected acute stroke when blood glucose measurement is not feasible.[2] [CoSTR 2020, weak recommendation, low certainty of evidence]
5. We suggest that when blood glucose measurement is feasible, the use of a stroke assessment tool that includes blood glucose measurement, such as the Melbourne Ambulance Stroke Screen (MASS) or the Los Angeles Prehospital Stroke Screen (LAPPS).[2] [CoSTR 2020, weak recommendation, low certainty evidence]
6. Send for an ambulance immediately if stroke is suspected, even if short duration of symptoms or if symptoms have resolved. [Good Practice

Statement]

7. Do not routinely administer oxygen to persons with stroke.[2] Administer oxygen only if there are obvious signs of shock or evidence of low oxygen saturation according to Use of Oxygen in Emergencies [Refer to ANZCOR Guideline 9.2.10].
8. Do not give the person anything to eat or drink, as swallowing may be impaired. [Good Practice Statement] If the blood sugar is measured and low, treat according to ANZCOR Guideline 9.2.9.

Abbreviations

- ANZCOR: Australian and New Zealand Committee on Resuscitation
- CoSTR: Consensus on Science with Treatment Recommendations (from International Liaison Committee on Resuscitation - ILCOR)
- FAST:
  - **F**acial weakness - ask the person to smile. Is their mouth droopy on one side?
  - **A**rm weakness - ask the person raise both arms. Can they only raise one arm or is one arm weaker?
  - **S** peech difficulty - ask the person to repeat a phrase. Is their speech slurred and can they understand what you say?
  - **T**ime to act fast (Take Action) - if any of these signs are present send for an ambulance immediately
- MASS: Melbourne Ambulance Stroke Screen
- LAPSS: Los Angeles Prehospital Stroke Screen

Guideline

1 Introduction

Stroke is a common cause of death and disability.[5] A stroke occurs when the supply of blood to part of the brain is suddenly disrupted. Blood flow can stop through the artery when it gets blocked by a blood clot or when an artery ruptures. Without the oxygen that the blood supplies, surrounding brain cells are quickly damaged and die. A quick response is needed because 'Time is Brain'. If treatment is provided quickly, some of these damaged brain cells can survive. This is why it is so important to recognise stroke quickly and to send for an ambulance immediately if stroke symptoms are present.

A person with the symptoms of stroke should be transported by ambulance because paramedics can start the management for stroke and make sure the person is taken to the most appropriate hospital for specialist stroke management. Paramedics can also notify the receiving hospital, reducing time to the start of treatment.

2 Recognition

- ANZCOR recommends using a validated stroke assessment system to assist stroke recognition. ([1]) [CoSTR 2020, strong recommendation, low certainty evidence] When there is doubt over the diagnosis, the person should be managed as having a stroke until proven otherwise.
- ANZCOR suggests the use of the FAST stroke assessment for individuals with suspected acute stroke when blood glucose measurement is not possible.[1] [CoSTR 2020, weak recommendation, low certainty of evidence. **FAST**[6] is a simple way for remembering the most common signs of stroke.
    - **F**acial weakness - ask the person to smile. Is their mouth droopy on one side?
    - **A**rm weakness - ask the person raise both arms. Can they only raise one arm or is one arm weaker?
    - **S** peech difficulty - ask the person to repeat a phrase. Is their speech slurred and can they understand what you say?
    - **T**ime to act fast (Take Action) - if any of these signs are present send for an ambulance immediately

Other less common symptoms and signs of stroke include:

- numbness of the face, arm or leg
- difficulty swallowing
- dizziness, loss of balance or an unexplained fall
- loss of vision, sudden blurred or decreased vision in one or both eyes
- headache, usually severe and of abrupt onset or unexplained change in the pattern of headaches
- drowsiness
- confusion
- reduced level of consciousness.

- ANZCOR suggests, that when blood glucose level measurement is feasible, use a validated stroke assessment tool that includes blood glucose level measurement[1] such as the Melbourne Ambulance Stroke Screen (MASS)[7] or the Los Angeles Prehospital Stroke Screen (LAPSS).[8] [CoSTR 2020, weak recommendation, low certainty evidence]

Symptoms of stroke may also be caused by other conditions such as epilepsy, migraine or low blood glucose level (BGL). The measurement of a blood glucose level may improve the recognition of stroke from other conditions when used in conjunction with a stroke assessment tool.[1] Importantly, early recognition of a low blood glucose level enables early treatment according to ANZCOR Guideline 9.2.9 improving the outcome of this condition as well.

3 Management

- If the person is unresponsive and not breathing normally, commence resuscitation following the Basic Life Support Flowchart [Refer to ANZCOR Guideline 8].
- If the person becomes unconscious but is breathing, lay the person on their side and ensure airway is clear [Refer to ANZCOR Guideline 3].

- Send for an ambulance for any person who has shown signs of stroke, no matter how brief or if symptoms have resolved. [Good Practice Statement].
- ANZCOR suggests against the routine administration of oxygen to persons with stroke. [2,10,11] [CoSTR 2020, weak recommendation, low to moderate certainty of evidence] Administer oxygen only if there are obvious signs of shock or evidence of low oxygen saturation according to ANZCOR Guideline 9.2.10.
- Do not give anything to eat or drink, as swallowing may be impaired. [Good Practice Statement].
- If the blood glucose level is measured and low and the person is fully conscious and able to swallow, treat according to ANZCOR Guideline 9.2.9.

References

1. Meyran D, Cassan P, Singletary E, Zideman D on behalf of the International Liaison Committee on Resuscitation (ILCOR) First Aid Task Forces. First Aid Stroke Recognition Consensus on Science with Treatment Recommendations [Internet] Brussels, Belgium: International Liaison Committee on Resuscitation (ILCOR) First Aid Task Force, 2020 January 2 available from: http://ilcor.org
2. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science With Treatment Recommendations. *Circulation* 2020; **142** (16_suppl_1): S284-S334.
3. National Stroke Foundation. Clinical Guidelines for Stroke Management (2020). Melbourne, Australia. http://www.strokefoundation.com.au (Accessed 29/07/2020).
4. Beasley, R., Chien, J., Douglas, J., Eastlake, L., Farah, C., King, G., Moore, R., Pilcher, J., Richards, M., Smith, S. and Walters, H. (2015), Thoracic Society of Australia and New Zealand oxygen guidelines for acute oxygen use in adults: 'Swimming between the flags'. Respirology, 20: 1182-1191. doi: 10.1111/resp.12620.
5. AIHW, Cause of death Australia, https://www.aihw.gov.au/reports/life-expectancy- death/deaths-in-australia/contents/multiple-causes-of-death. Accessed 18 July 2020
6. Nor AM, McAllister S, Louw J, Dyker G, Davis M, Jenkinson G, Ford A. Agreement Between Ambulance Paramedic and Physician-Recorded Neurological Signs With Face Arm Speech Test (FAST) in Acute Stroke Patients. Stroke 2004; 35(6): 1355-1359.
7. Bray, J.E, Martin, J, Cooper, G, Barger, B, Bernard, S, and Bladin, C. Paramedic identification of stroke: community validation of the Melbourne Ambulance Stroke Screen (MASS). Cerebrovascular Diseases 2005;20:28-33.
8. Kidwell CS, Starkman S, Eckstein M, Weems K, Saver JL: Identifying stroke in the field. Prospective validation of the Los Angeles prehospital stroke screen (LAPSS). Stroke 2000; 31: 71-76.
9. Chang WT, Sakamoto T, Lee CC, Singletary EM, Zideman D, Schmölzer G, Bendall J, Berry DC, Borra V, Carlson JN, Cassan P, Charlton NP, Djarv T, Douma M, Epstein J, Hood NA, Markenson D, Meyran D, Orkin A, Swain J,

Woodin JA, Morley P: First Aid Supplementary Oxygen in Acute Stroke Consensus on Science with Treatment Recommendations [Internet] Brussels, Belgium: International Liaison Committee on Resuscitation (ILCOR) First Aid Task Force, 1 Jan 2020. Available from http://ilcor.org.

10. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science With Treatment Recommendations. *Resuscitation* 2020; **156** : A240-A
11. National Stroke Foundation. Clinical Guidelines for Stroke Management 2017. Chapter 3 of 8: Acute medical and surgical management. Melbourne. Retrieved 20 November 2020 from https://informme.org.au/en/Guidelines/Clinical-Guidelines-for-Stroke-Management-2017: National Stroke Foundation

Further Reading

- ANZCOR Guideline 2 Managing an Emergency
- ANZCOR Guideline 3 Recognition and First Aid Management of the Unconscious Victim
- ANZCOR Guideline 4 Airway
- ANZCOR Guideline 5 Breathing
- ANZCOR Guideline 8 Cardiopulmonary Resuscitation
- ANZCOR Guideline 9.2.9 First Aid Management of a Diabetic Emergency
- ANZCOR Guideline 9.2.10 The Use of Oxygen in Emergencies

About this Guideline

- Search date/s: September and October 2019
- Question/PICO 1:
  - Population: Adults with suspected acute stroke
  - Intervention: Use of a rapid stroke scoring system or scale (or test) (as FAST, LAPSS, CPSS, OPSS, KPSS, MASS or others)
  - Comparison: Basic first aid assessment without the use of a scale
  - Outcomes:
    - Change time to treatment (e.g. symptom onset to hospital/emergency department arrival or hospital admission (9-Critical)
    - Recognition of stroke: (5-Important) high number considered beneficial for observational study; high sensitivity and high specificity considered beneficial for diagnosis study
    - Discharge with favourable neurologic status (increase considered beneficial) (5-Important)
    - Survival with favourable neurologic outcome (increase considered beneficial) (5-Important)
    - Increased public/layperson recognition of stroke signs (5- Important)
- Population: Adults with suspected acute stroke
- Intervention: Use of supplementary oxygen
- Comparators: No use of supplementary oxygen

- Outcomes: Clinical outcomes such as survival, neurological outcomes (e.g. NIHSS, Scandinavian stroke scale, modified Rankin scale score, etc.), and neurological recovery in the acute phase were ranked as critical outcomes. Quality of life (e.g. Barthel index, EuroQol, Nottingham ADL score, etc.) and hospital length of stay were ranked as important outcomes. Adverse effects and complications (pneumonia, pulmonary edema, necessity of non-invasive positive pressure ventilation, intubation with mechanical ventilation, etc.) were listed as important outcomes. Imaging outcomes such as magnetic resonance imaging (MRI) indicators (diffusion-weighted imaging, lesion volume, diffusion/perfusion mismatch, magnetic resonance spectroscopic indicators, etc.) and reperfusion rate were ranked as important outcomes. Laboratory outcomes such as oxygen saturation (highest, lowest, incidence or duration of oxygen saturation < 90% or 95%, etc.) were listed as good-to-know outcomes.
- Study Designs: Randomized controlled trials (RCTs) and non- randomized studies (non-randomized controlled trials, interrupted time series, controlled before-and-after studies, cohort studies) are eligible for inclusion. Unpublished studies (e.g., conference abstracts, trial protocols) were excluded.
- Timeframe: All years and all languages were included; unpublished studies (e.g., conference abstracts, trial protocols) were excluded. Literature search updated to Oct 16, 2019.
- Method: Systematic Reviews (ILCOR First Aid Task Force, CoSTR)
- Primary reviewers: Natalie Hood, Finlay Macneil
- Other consultation** Kevin Nation, Janet Bray
- Worksheet: See https://www.ilcor.org/
- Approved: April 2021
- Guideline superseded: ANZCOR Guideline 9.2.2 - August 2016

ANZCOR GUIDELINE 9.2.3 – SHOCK

Who does this guideline apply to?

This guideline applies to adult, child and infant victims.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid providers.

1 Introduction

Shock is a loss of effective circulation resulting in impaired tissue oxygen and nutrient delivery^1 and causes life threatening organ failure.

2 Causes

Some conditions which may cause shock include^2 :

2.1 Loss of circulating blood volume (hypovolaemic shock), e.g.:

- severe bleeding (internal and / or external)
- major or multiple fractures or major trauma
- severe burns or scalds
- severe diarrhoea and vomiting
- severe sweating and dehydration.

2.2 Cardiac causes (cardiogenic shock), e.g.:

- heart attack
- dysrhythmias (abnormal heart rhythm).

2.3 Abnormal dilation of blood vessels (distributive shock), e.g.:

- severe infection
- allergic reactions
- severe brain / spinal injuries
- fainting.

2.4 Blockage of blood flow in or out of heart (obstructive shock), e.g.:

- tension pneumothorax
- cardiac tamponade
- pulmonary embolus
- in pregnancy, compression of large abdominal blood vessels by the uterus.

3 Recognition

The symptoms, signs and rate of onset of shock will vary widely depending on the nature and severity of the underlying cause^3. Shock is a condition that may be difficult to identify.

3.1 Symptoms may include:

- dizziness
- thirst
- anxiety
- restlessness
- nausea
- breathlessness
- feeling cold.

3.2 Signs may include:

- collapse
- rapid breathing
- rapid pulse which may become weak or slow
- cool, sweaty skin that may appear pale
- confusion or agitation
- decreased or deteriorating level of consciousness
- vomiting.

4 Management

1. Place the victim in the supine position. If victim is unconscious place victim on side (Guideline 3).
2. Control any bleeding promptly (Guideline 9.1.1).
3. **Call an ambulance.**
4. Administer treatments relevant to the cause of the shock.
5. Administer oxygen if available and trained to do so (Guideline 10.4).
6. Maintain body temperature (prevent hypothermia).
7. Reassure and constantly re-check the victim’s condition for any change.
8. If the victim is unresponsive and not breathing normally, follow the Basic Life Support Flowchart (ANZCOR Guideline 8).

4.1 Positioning of victims with shock

Place individuals with shock in the supine position as opposed to the upright position^4 (CoSTR 2015, weak recommendation, low-quality evidence).

For individuals with shock who are in the supine position and with no evidence of trauma, the use of PLR (passive leg raise) may provide a transient (less than 7 minutes) improvement.

The clinical significance of this transient improvement is uncertain; however, no study reported adverse effects due to PLR^4. Because improvement with PLR is brief and its clinical significance uncertain, ANZCOR recommends the supine position without leg raising for victims in shock^4 (CoSTR 2015, values and preferences statement).

References

1. Skinner, B. & Joans, M. (2007). Causes and Management of Shock. Anesthesia and Intensive Care Medicine. **8** (12): 520-524.
2. Graham, C.A. & Parke, T.R. (2005). Critical care in the emergency department: shock and circulatory support. Emergency Medicine Journal. **22** :17-21.
3. Moore, F.A., McKinley, B.A. & Moore, E.E. (2004). The next generation in shock resuscitation. The Lancet **363:** 1988-96.
4. Zideman, D. A., Singletary, E. M., De Buck, E.,et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. *Resuscitation, 95* , e225. http://www.cprguidelines.eu/assets/downloads/costr/S0300- 9572(15)00368-8_main.pdf Accessed 19/11/2015.

AUSTRALIAN RESUSCITATION COUNCIL GUIDELINE 9.2.4 - FIRST AID MANAGEMENT OF A SEIZURE

INTRODUCTION

A seizure is a sign of abnormal brain activity, which can be caused by many problems. Up to 10% of the population is likely to experience a seizure at some time in their life.^2 A seizure may occur when the normal pattern of electrical activity of the brain is disrupted. This can cause changes in sensation, awareness and behaviour, or sometimes convulsions, muscle spasms or loss of consciousness. Seizures vary greatly and most are over in less than 5 minutes. Not all seizures are considered epilepsy. 1, 2

A seizure may be associated with:

- lack of oxygen (hypoxia);
- onset of cardiac arrest;
- medical conditions affecting the brain, e.g. low blood sugar, low blood pressure, head injury, neurological diseases, epilepsy;
- trauma to the head;
- some poisons and drugs;
- withdrawal from alcohol and other substances of dependence;
- fever in children under six years.

RECOGNITION

Seizures may affect all or part of the body.^2 Seizure activity may take many forms, and symptoms may include:

- sudden spasm of muscles producing rigidity. If standing the victim will fall down;
- jerking movements of the head, arms and legs;
- Shallow breathing or breathing may stop temporarily;
- dribbling from the mouth; the tongue may be bitten leading to bleeding;
- incontinence of urine and/or faeces;
- changes in conscious state from being fully alert to confused, drowsy, or loss of consciousness;
- changes in behaviour where the victim may make repetitive actions like fiddling with their clothes.

Generalised seizures usually involve the entire body and cause a loss or marked alteration in consciousness. Some generalized seizures result in life-threatening problems with airway or breathing, or risk of trauma from muscle spasms or loss of normal control of posture and movement. During partial seizures, usually only part of the body is affected and the person retains consciousness but may be frightened or confused.

Febrile convulsions are associated with fever and usually resolve without treatment. They occur in approximately 3% of children at some stage between the age of six months and six years. 3, 4 Children who suffer from a febrile convulsion are not at increased risk of epilepsy as a result of experiencing febrile convulsions.3, 4

MANAGEMENT OF A SEIZURE

If the victim is unresponsive and not breathing normally, follow Australian Resuscitation Council and New Zealand Resuscitation Council Basic Life Support Flowchart (ARC Guideline 8).

If the victim is unconscious and actively seizing, the rescuer should:

- follow the victim’s seizure management plan, if there is one in place;
- manage the victim according to (ARC Guideline 3);
- call an ambulance.

The rescuer should:

- manage the victim as for any unconscious person (ARC Guideline 3);
- remove the victim from danger or remove any harmful objects which might cause secondary injury to the victim;
- note the time the seizure starts;
- protect the head;
- avoid restraining the victim during the seizure unless this is essential to avoid injury;
- lay the victim down and turn the victim on the side when practical;
- maintain an airway;
- reassure the victim who may be dazed, confused or drowsy;
- call an ambulance;
- frequently reassess the victim.

Do not:

- put a child in a bath (to lower their temperature) during a convulsion as this is dangerous;
- do not force the victim’s mouth open nor attempt to insert any object into the mouth.

A seizure in water is a life threatening situation. If the seizure occurs in water:

- support the victim in the water with the head tilted so the face is out of the water^2 ;
- remove the victim from the water as soon it is safe to do so^2 ;
- call an ambulance;

- if the victim is unresponsive and not breathing normally, follow Australian Resuscitation Council and New Zealand Resuscitation Council Basic Life Support Flowchart (ARC Guideline 8).

REFERENCES

1. Epilepsy Action Australia (2012) Fact sheet: Seizure Smart: Basic facts on Epilepsy. Epilepsy Action Australia
2. Epilepsy Foundation (2012) Understanding Epilepsy. [online at http://www.epinet.org.au/articles/understanding_epilepsy/seizures__epilepsy]
3. Gratten-Smith P. Textbook of Paediatric Emergency Medicine. Philadelphia: Churchill Livingstone Elsevier; c2006. Chapter 8.3, Seizures and non-epileptic events; pp.229- 33
4. Royal Children's Hospital (2008) Fact sheet: Febrile convulsions. Royal Children's Hospital, Melbourne. [online at http://www.rch.org.au/kidsinfo/fact_sheets/Febrile_Convulsions/]

LEVEL OF EVIDENCE

Expert Consensus Opinion

CLASS OF RECOMMENDATION Class A - Recommended

FURTHER READING

ARC Guideline 2 Priorities in an Emergency ARC Guideline 3 Unconsciousness ARC Guideline 4 Airway ARC Guideline 8 Cardiopulmonary Resuscitation ARC Guideline 9.1.4 Head Injury ARC Guideline 9.2.2 Stroke

ARC Worksheet 9.4.2a:

- In adults and children (P), who exhibit seizure activity (I) compared with victims who do not have a seizure (C) what proportion are related to cardiac arrest (O)?
- In adults and children (P), who exhibit seizure activity (I) compared with victims who do not have a seizure (C) what is the risk of cardiac arrest / sudden death (O)?

ARC Worksheet 9.4.2b:

- In adults and children having a seizure (P), does giving oxygen (I) compared with not giving oxygen (C) improve outcome (O = mortality, seizure duration, incidence of post-seizure hypoxaemia)?

ANZCOR GUIDELINE 9.2.5 – FIRST AID FOR ASTHMA

Guideline

Who does this guideline apply to?

This guideline applies to adult and child victims.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid providers.

1 Introduction

Asthma is a disorder of the smaller airways of the lungs. People with asthma have sensitive airways which can narrow when exposed to certain ‘triggers’, leading to difficulty in breathing.

Three main factors cause the airways to narrow:

1. The muscle around the airway tightens (bronchoconstriction).
2. The inside lining of the airways becomes swollen (inflammation)
3. Extra mucus (sticky fluid) may be produced.

In asthma, symptoms are made worse by 'triggers'. Every person's asthma is different and not all people will have the same triggers. Triggers can include:

- Respiratory infection
- Irritants (e.g. cigarette, wood fire or bushfire smoke, occasionally perfumed or cleaning products)
- Inhaled allergens (e.g. dust mite, mould spores, animal danders, grass/tree pollen)
- Cold air, exercise, laughing/crying
- Non steroidal anti-inflammatory agents (e.g. aspirin, ibuprofen)
- Sulfite additives (food preservatives) – more common in those with poorly controlled asthma
- Food allergy – while usually accompanied by other symptoms such as rash or vomiting, isolated severe asthma may occur as the only presentation and may result in death
- Food colours and flavours

- Emotional triggers such as stress.

Most fatal cases of food-induced anaphylaxis occur in those with asthma. In patients with asthma known to be at risk from anaphylaxis, if it is uncertain whether the patient is suffering from asthma or anaphylaxis, it is appropriate to administer an adrenaline autoinjector first, followed by asthma reliever medication. No harm is likely to occur by doing so in a patient having asthma without anaphylaxis.

2 Recognition

Asthma can be recognised by the following symptoms and signs:

- A dry, irritating, persistent cough, particularly at night, early morning, with exercise or activity
- Chest tightness
- Shortness of breath
- Wheeze (high pitched whistling sound during breathing).

2.1 Symptoms and signs of a severe asthma attack include some or all of the following:

- Gasping for breath (may have little or no wheeze due to little movement of air)
- Severe chest tightness
- Inability to speak more than one or two words per breath
- Feeling distressed and anxious
- Little or no improvement after using “reliever” medication
- ‘Sucking in’ of the throat and rib muscles, use of shoulder muscles or bracing with arms to help breathing
- Blue discolouration around the lips (can be hard to see if skin colour also changes)
- Pale and sweaty skin
- Symptoms rapidly getting worse or using reliever more than every two hours.[2]

As well as the above symptoms, young children appear restless, unable to settle or become drowsy. A child may also ‘suck’ in muscles around the ribs and may have problems eating or drinking due to shortness of breath. A child also may have severe coughing and vomiting.

An asthma attack can take anything from a few minutes to a few days to develop.

3 Managing An Asthma Attack

If the victim has a personal written asthma action plan then that plan should be followed.

If there is no action plan in place then use the following Asthma First Aid plan.

3.1 Asthma First Aid Plan

If a victim has any signs of a severe asthma attack, call an ambulance straight away and follow the Asthma First Aid Plan while waiting for the ambulance to arrive.

Step	Australia (4 x 4 x 4)	New Zealand (6 x 6 x 6)
1	Sit the person comfortably upright. Be calm and reassuring. Do not leave the person alone.	Sit the person comfortably upright. Be calm and reassuring. Do not leave the person alone.
2	Without delay give four separate puffs of a “reliever”. The medication is best given one puff at a time via a spacer device. Ask the person to take four breaths from the spacer after each puff of medication.	Without delay give six separate puffs of a “reliever”. The medication is best given one puff at a time via a spacer device. Ask the person to take six breaths from the spacer after each puff of medication.
3	Wait four minutes. If there is little or no improvement give another four puffs.	Wait six minutes. If there is little or no improvement give another six puffs.
4	If there is still no improvement, call an ambulance immediately. Keep giving four puffs every four minutes until the ambulance arrives.	If there is still no improvement, call an ambulance immediately. Keep giving six puffs every six minutes until the ambulance arrives.

If a spacer is not available, simply use the inhaler. Use the victim’s own inhaler if possible. If not,use the first aid kit inhaler if available or borrow one from someone else.

No harm is likely to result from giving a “reliever” inhaler to someone without asthma.[2] *[LOE: Expert Consensus Opinion].*

If oxygen is available, it should be administered by a person trained in its use, following Use of Oxygen in Emergencies (ANZCOR Guideline 10.4). [LOE: Expert Consensus Opinion].

If a severe allergic reaction is suspected, follow Anaphylaxis – First Aid Management (ANZCOR Guideline 9.2.7)

If victim becomes unresponsive and not breathing normally, commence resuscitation following the Basic Life Support Flowchart (ANZCOR Guideline 8).

4 Regional differences in recommended dose and intervals

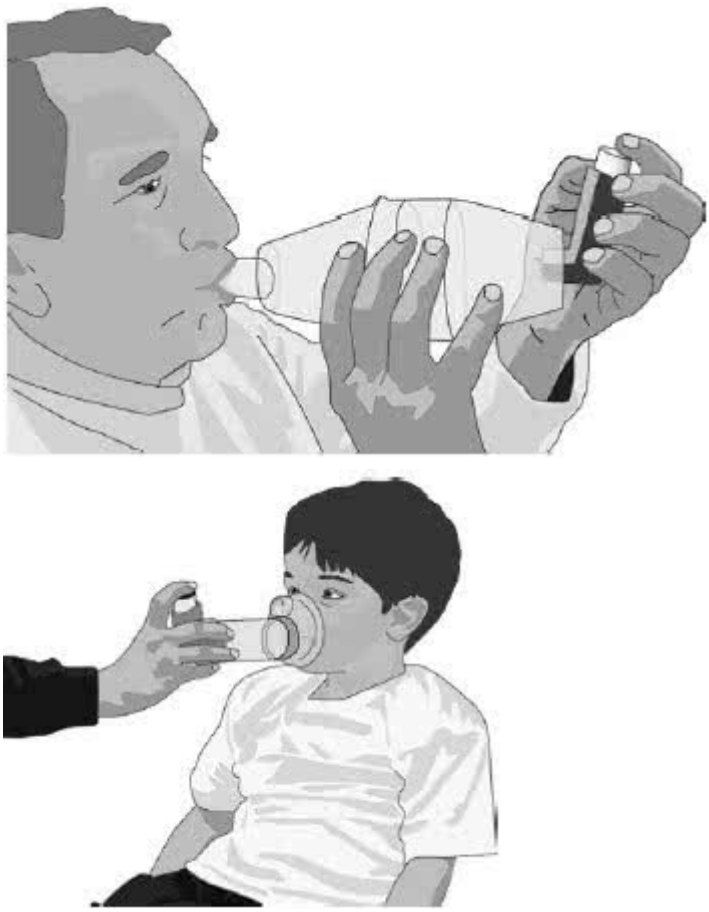
There are differences in first aid treatment recommendations (e.g. doses and timing of relievers) between Australia and New Zealand. Additionally treatment recommendations for the clinical management of acute asthma by health professionals are different again.

In Australia, the National Asthma Council Australia recommends taking 4 puffs every 4 minutes (4 x 4 x 4) 4, 5 whereas in New Zealand, the Asthma and Respiratory Foundation NZ recommend taking 6 puffs every 6 minutes[6] (6 x 6 x 6).

The 2015 International Consensus on First Aid Science did not provide any update on dosage or interval between doses. This guideline is not intended to contradict current recommendations of peak asthma bodies in Australia or in New Zealand – the ANZCOR recommended treatment in 3.1 accounts for these regional differences. This guideline does not seek to alter first aid practice (with respect to dosage or timing of reliever medication)in either Australia or New Zealand.

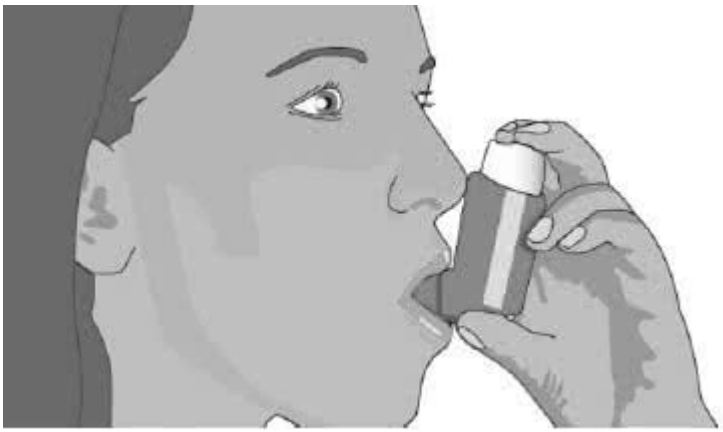
WITH SPACER

Assemble the spacer. Remove inhaler cap and shake well. Place the inhaler upright into the spacer. Place the spacer mouthpiece into the victim's mouth, between the teeth with the lips sealed around it. Press firmly on the inhaler to fire one puff into the spacer. Ask the victim to breathe in and out normally for four to six breaths via the spacer. Repeat this promptly until four to six puffs have been given. Remember to shake the inhaler before each puff.



WITHOUT SPACER

When a spacer is unavailable, shake the inhaler. Place the mouthpiece into the victim's mouth, between the teeth with the lips sealed around it. Press firmly on the inhaler to administer one puff as the victim inhales slowly and steadily. Slip the inhaler out of the victim's mouth. Ask the victim to hold their breath for four seconds or as long as comfortable. Breathe out slowly away from the inhaler. Repeat this promptly until four to six puffs have been given. Remember to shake the inhaler before each puff.



The most common reliever medication is salbutamol. Victim's own reliever medication may be used as an alternative.

References

1. Asthma Australia. About asthma. https://www.asthmaaustralia.org.au/national/about-asthma Accessed 05/12/
2. National Asthma Council Australia. Asthma Action Plans http://www.nationalasthma.org.au/health-professionals/asthma-action-plans Accessed 19/11/
3. Zideman, D. A., Singletary, E. M., De Buck, E.,et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. *Resuscitation, 95* , e225.http://www.cprguidelines.eu/assets/downloads/costr/S0300-9572(15)00368-8_main.pdf Accessed 19/11/
4. National Asthma Council. First Aid for Asthma http://www.nationalasthma.org.au/first-aid Accessed 19/11/
5. National Asthma Council Australia. *Australian Asthma Handbook*, Version 1.1. National Asthma Council Australia, Melbourne, 2015. Website. Available from: http://www.asthmahandbook.org.au Accessed 19/11/
6. Asthma and Respiratory Foundation of New Zealand. Controlling your asthma [https://s3-ap-southeast-2.amazonaws.com/assets.asthmafoundation.org.nz/documents/Controlling-Your-Asthma- Resource.pdf] Accessed 6/7/

Further Reading

ANZCOR Guideline 5 Breathing

ANZCOR Guideline 8 Cardiopulmonary Resuscitation

ANZCOR Guideline 9.2.7 Anaphylaxis – First Aid Management

ANZCOR Guideline 10.4 The Use of Oxygen in Emergencies

ANZCOR GUIDELINE 9.2.7 – FIRST AID MANAGEMENT OF ANAPHYLAXIS

Guideline

Who does this guideline apply to?

This guideline applies to adults, children and infant victims.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid providers.

1 Introduction

Anaphylaxis is the most severe form of allergic reaction and is potentially life threatening. It must be treated as a medical emergency, requiring immediate treatment and urgent medical attention. Anaphylaxis is a generalised allergic reaction, which often involves more than one body system. A severe allergic reaction usually occurs within 20 minutes of exposure to the trigger.^1 Severe allergic reactions may occur without prior exposure to a trigger. It is characterised by rapidly developing airway and/or breathing and/or circulation problems usually associated with swelling, redness or itching of the skin, eyes, nose, throat or mouth. Many substances can cause anaphylaxis but more common causes include:

- foods (especially peanuts, tree nuts, cow's milk, eggs, wheat, seafood, fish, soy, sesame)^2
- drugs (e.g. penicillin^3)
- venom from bites (ticks) or stings (e.g. bees, wasps or ants).

2 Recognition

Anaphylaxis encompasses a variety of symptoms and signs. Diagnosis is largely based on history and physical findings. Onset can range from minutes to hours of exposure to a substance.^2 Symptoms and signs are highly variable and may include^4 one or more of the following:

- difficult / noisy breathing

- wheeze or persistent cough
- swelling of face and tongue
- swelling / tightness in throat
- difficulty talking and /or hoarse voice
- persistent dizziness / loss of consciousness and / or collapse
- pale and floppy (young children)
- abdominal pain and vomiting
- hives, welts and body redness.

3 Management

People with diagnosed allergies should avoid all trigger agents / confirmed allergens and have a readily accessible anaphylaxis action plan and medical alert device. Whenever possible, this information should be sought and implemented provided this does not delay emergency treatment and seeking medical assistance.

3.1 Emergency Treatment

The injection of adrenaline (epinephrine) is the first line drug treatment in life threatening anaphylaxis.4,5,6,^

Adrenaline (epinephrine) autoinjectors are safe and effective management of anaphylaxis. People who have had a prior episode of anaphylaxis often have prescribed medication including adrenaline (epinephrine) in the form of an autoinjector and the early administration of adrenaline (epinephrine) is the priority in the emergency treatment.

If the victim’s symptoms and signs suggest anaphylaxis the following steps should be followed.^4

1. Lay the victim flat; do not stand or walk. If breathing is difficult, allow to sit (if able).
2. Prevent further exposure to the triggering agent if possible.
3. Administer adrenaline (epinephrine) via intramuscular injection4,6 (Class A; LOE 4) preferably into lateral thigh:
4. Child less than 5 years - 0.15 mg
5. Older than 5 years - 0.3mg. **4. Call an ambulance.**
6. Administer oxygen, if available and trained to do so (Class B LOE Expert Consensus Opinion. ANZCOR Guideline 10.4).
7. Give asthma medication for respiratory symptoms.
8. A second dose of adrenaline (epinephrine) should be administered by autoinjector to victims with severe anaphylaxis whose symptoms are not relieved by the initial dose (CoSTR 2015: weak recommendation/very low quality evidence)^8. The second dose is given if there is no response 5 minutes after the initial dose^5.
9. If allergic reaction or anaphylaxis has occurred from an insect bite or sting follow Envenomation- Tick Bites And Bee, Wasp And Ant Stings (ANZCOR Guideline 9.4.3).

10. If victim becomes unresponsive and not breathing normally, give resuscitation following the Basic Life Support Flowchart (ANZCOR Guideline 8).

Acknowledgement

Instructional information regarding auto injectors can be accessed via the ASCIA (Australian Society of Clinical Immunology and Allergy) webpage: http://www.allergy.org.au/health-professionals/anaphylaxis-resources

References

1. Anaphylaxis Australia. What is anaphylaxis. https://www.allergyfacts.org.au/allergy- anaphylaxis/what-is-anaphylaxis Accessed 19/
2. Sampson, H.A. Anaphylaxis and emergency treatment. Pediatrics 2003. **111** : 1601- 8
3. Fisher, M. Anaphylaxis to anaesthetic drugs. Novartis Found. Symp. 2004. **257** : 193-
4. Australasian Society of Clinical Immunology and Allergy. Action Plan for Anaphylaxis. http://www.allergy.org.au/health-professionals/anaphylaxis-resources/ascia-action-plan-for- anaphylaxis Accessed 19/11/2015.
5. Australasian Society of Clinical Immunology and Allergy. First Aid for Anaphylaxis. http://www.allergy.org.au/images/stories/anaphylaxis/2015/ASCIA_FIRST_AID_FOR_ANAP HYLAXIS_2015.pdf Accessed 19/11/2015.
6. Sheikh, A, Shehata, Y.A, Brown, S.G.A., Simons, F.E.R. Adrenaline (epinephrine) for the treatment of anaphylaxis with and without shock. Cochrane Database of Systematic Reviews 2008, Issue 4. Art. No.: CD006312. DOI: 10.1002/14651858.CD006312.pub2.
7. Brown, S.G.A, Mullins, R.J., Gold, M.S. Anaphylaxis: diagnosis and management. Med. J. Aust. 2006. **185** (5):283- 9
8. Zideman, D. A., Singletary, E. M., De Buck, E.,et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. *Resuscitation, 95* , e225. http://www.cprguidelines.eu/assets/downloads/costr/S0300-9572(15)00368-8_main.pdf00368-8_main.pdf) Accessed 19/11/

Further Reading

ANZCOR Guideline 4 Airway

ANZCOR Guideline 8 Cardiopulmonary Resuscitation

ANZCOR Guideline 9.4.3 Envenomation – Bee, Wasp and Ant Stings

ANZCOR Guideline 10.4 The Use of Oxygen by First Aiders

AUSTRALIAN RESUSCITATION COUNCIL GUIDELINE 9.2.8 - THE FIRST AID MANAGEMENT OF HYPERVENTILATION SYNDROME

INTRODUCTION

Hyperventilation syndrome is the term used to describe the symptoms and signs resulting from over-breathing.

In this condition, the rate and depth of breathing exceed that required to maintain normal levels of carbon dioxide in the blood. Consequently the carbon dioxide level in the arterial blood falls, resulting in a range of symptoms and signs as below. Anxiety is usually present.

Not every person who is breathing deeply or rapidly has hyperventilation syndrome. Other more serious conditions which could cause this include:

- asthma attack
- heart failure
- pulmonary embolus
- heart attack
- spontaneous pneumothorax
- some poisoning incidents
- uncontrolled diabetes.

If any of the above conditions are suspected, call for an ambulance (Dial Triple Zero – 000)

RECOGNITION

Symptoms may include:

- lightheadedness
- shortness of breath
- being unable to get enough breath in
- chest discomfort
- a feeling of panic and impending death
- blurred vision
- tingling of fingers, toes and lips
- palpitations

- a feeling of detachment and not being in full control of the body (depersonalisation).

Signs may include:

- rapid breathing
- occasional deep, sighing breaths
- rapid pulse
- altered level of consciousness e.g. fainting
- hand and finger spasm (carpo-pedal spasm) in advanced attacks. The fingers and wrists become claw-like with the thumb held stiffly across the palm.

MANAGEMENT

- Reassure the victim and encourage the person to slow down their breathing.
- Follow the ARC Basic Life Support Flow Chart Guideline 8.
- If the symptoms of hyperventilation are unresolved, call for an ambulance (Dial Triple Zero – 000).

DO NOT USE ANY BAG FOR RE-BREATHING

RATIONALE

This practice may be dangerous and should only be used under medical direction.

LEVEL OF EVIDENCE

Expert Consensus Opinion

CLASS OF RECOMMENDATION

Class A

FURTHER READING

ARC Guideline 8 Cardiopulmonary Resuscitation

ARC Guideline 9.2.1 Chest Pain

ARC Guideline 9.2.5 First Aid for Asthma

ARC Guideline 9.5.1 Emergency Management of a Victim Who has been Poisoned

ARC Guideline 9.5.2 Emergency Management of Victims of Inhalational Incidents

ANZCOR Guideline 9.2.9 - First Aid Management of a Diabetic Emergency

Summary

Who does this guideline apply to?

This guideline applies to adults and children.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. When available, and trained to do so, use a blood glucometer to check the person's blood glucose level. [Good Practice Statement]
2. We recommend the use of oral glucose (swallowed) for individuals with suspected hypoglycaemia who are conscious and able to swallow.[1] [strong recommendation, very low certainty of evidence]
3. We suggest against buccal glucose administration compared with oral glucose administration for individuals with suspected hypoglycaemia who are conscious and able to swallow.[1] [weak recommendation, very low certainty of evidence]
4. If oral glucose (tablet) is not immediately available, we suggest a combined oral + buccal glucose (glucose gel) administration for individuals with suspected hypoglycaemia who are conscious and able to swallow.[1] [weak recommendation, very low certainty of evidence]
5. We suggest the use of sublingual glucose administration for suspected hypoglycaemia for children who may be uncooperative with the oral (swallowed) glucose administration route.[1] [weak recommendation, very low certainty of evidence]
6. When available, and trained to do so, use a glucagon injection to manage suspected hypoglycaemia in an unconscious or seizing person. [Good Practice Statement]
7. If unsure of the blood glucose level, manage the person as having suspected hypoglycaemia. [Good Practice Statement].

Guideline

1 Introduction

Diabetes is a chronic, lifelong medical condition which occurs when the pancreas fails to produce sufficient insulin or the body develops a resistance to the action of its own insulin. Untreated, the absolute or relative lack of insulin will lead to a high blood glucose level. There are two main types of diabetes. Type 1 diabetes is an auto-immune disease that often develops in childhood, and requires lifelong treatment with insulin. Type 2 diabetes is more commonly recognised in adulthood, and requires a treatment combination of diet, exercise, oral medication, and sometimes insulin. Gestational diabetes is a relatively common condition specific to pregnancy, and diabetes can also occur as a consequence of another disease or as a side effect of medication.

Normally the body tightly controls its blood glucose level within a 'normal' range. Having diabetes interferes with this control system, and people living with diabetes need to manage their own blood glucose levels by monitoring what they eat, adjusting their insulin or other medication doses, and frequently testing their own blood glucose levels.

When blood glucose levels become too high or too low, people with diabetes (and some other people without diabetes) may become unwell and need first aid, or treatment at a medical facility.

2 Low blood glucose (hypoglycaemia or ‘a hypo’)

2.1 Introduction

People with diabetes may develop low blood glucose levels as a result of:^2

- too much insulin or other blood glucose lowering medication;
- inadequate or delayed carbohydrate intake after their usual insulin or oral medication dose;
- exercise without adequate carbohydrate intake; possibly delayed for up to 12 hours or more after exercise.
- in the setting of other illness; or
- excessive alcohol intake.

Competitors in ultra-marathon endurance events, who do not have diabetes, can also become energy deplete and develop low blood glucose levels requiring first aid management.

Hypoglycaemic events range from those that can be self-managed, to severe episodes, where medical help is needed.

2.2 Recognition

The brain requires a continuous supply of glucose to function normally. When blood glucose levels fall below normal levels symptoms and signs may include:

- sweating,
- pallor (pale skin), especially in young children[1]
- a rapid pulse;
- shaking, trembling or weakness;
- hunger;
- light headedness or dizziness;
- headache;
- mood or behavioural changes, confusion, inability to concentrate;
- slurred speech;
- inability to follow instructions;
- unresponsive; or
- seizure

2.3 Management

If a person with diabetes has a diabetes management plan, then that plan should be followed. If a person with diabetes reports low blood glucose level or exhibits symptoms or signs of *hypoglycaemia* :

- Stop any exercise, rest and reassure;
- If the person is able to follow simple commands and swallow safely, we recommend that first aid providers administer 15-20 grams glucose tablets (4 - 5 x 4 gram glucose tablets) for treatment of symptomatic hypoglycaemia.[1,3,4,5] ~~[ILCOR CoSTR 2020 strong recommendation, very low certainty of evidence] If glucose tablets are not available, we suggest administering:~~
- Confectionary including:
  - jelly beans (5 to 20 beans depending on the brand)
  - Skittles® (20 to 25 candies)
  - Mentos® (5 to 10 mints)[3] [ILCOR CoSTR 2015, weak recommendation, very low certainty of evidence]
- Sugary drinks or sugar-sweetened beverages (approx. 200 mL), but DO NOT administer 'diet' or 'low-cal' or 'zero' or 'sugar free' beverages;
- Fruit juices (approx. 200 mL);
- Honey or sugar (3 teaspoons);
- Glucose gels (15 g of glucose gel)[6]; and
- Monitor for improvement. Resolution of symptoms would be expected within 15 minutes.

If symptoms or signs of hypoglycaemia persist after 10 to 15 minutes, and the person is still able to follow simple commands and swallow safely, administer a further 4 x 4g glucose tablets or alternatives as listed above. Once recovered, give a snack with longer acting carbohydrate, for example: 1 slice of bread OR 1 glass of milk OR 1 piece of fruit OR 2 to pieces of dried fruit OR 1 snack size tub of yoghurt (not diet or 'sugar free' yogurt). If it is a usual meal time, then eat that meal.

If the person deteriorates, does not improve with treatment, is seizing or is unconscious, call for an ambulance.

- If the person is unresponsive and not breathing normally, commence resuscitation following the Basic Life Support Flowchart [Refer to ANZCOR Guideline 8].
- If the person is unconscious but breathing, lie the person on their side and ensure the airway is clear [Refer to ANZCOR Guideline 3]

Insulin Pumps

If the person is wearing an insulin pump, then they themselves may 'suspend' their own pump if part of a personal diabetes management plan.

First aiders should ***not*** touch any insulin pump being worn by the person. They should manage and provide treatment for hypoglycaemia as listed above.

2.4 Use of glucagon to treat severe hypoglycaemia

Family members of, and carers for, people with diabetes may be trained in the use of the GlucaGen® HypoKit®. These kits contain an injection of glucagon, which works by triggering the liver to release stored glucose, resulting in raised blood glucose levels. The glucagon is administered by injection.

If trained to do so, give Glucagon in the case of a severe hypoglycaemic event, when the person is unconscious or seizing, and/or is unable to swallow safely.[4]

3 High blood glucose (hyperglycaemia)

3.1 Introduction

Hyperglycaemia means having a high blood glucose level. Common causes of hyperglycaemia include inadequate levels of insulin or incorrect doses of diabetes oral medications, infections, excess carbohydrate intake, and stressful situations. Hyperglycaemia can develop over hours or days, and many people do not experience symptoms from hyperglycaemia until their blood glucose levels are extremely high. Hyperglycaemia can also occur at the time of initial diagnosis of diabetes, and may go unrecognised until the person is clearly unwell. If untreated, the person gradually deteriorates, and can go into a coma.

3.2 Recognition

When blood glucose levels remain above normal levels symptoms and signs may include:

- excessive thirst;
- frequent urination;
- dry skin and mouth, with sunken eyes (signs of dehydration);
- recent weight loss;
- rapid pulse;
- nausea and vomiting;

- abdominal pain;
- rapid breathing;
- fruity sweet smell of acetone on the breath (similar to paint thinner or nail polish remover); and
- confusion, a deteriorating level of consciousness, or unresponsiveness.

3.3 Management

If a person with diabetes has a diabetes management plan then that plan should be followed. If the person has no management plan and has symptoms or signs of *hyperglycaemia* they should be assessed by a health care professional.

- If the person is unresponsive and not breathing normally, commence resuscitation following the Basic Life Support Flowchart [Refer to ANZCOR Guideline 8]
- If the person is unconscious but breathing, lie the person on their side and ensure the airway is clear [Refer to ANZCOR Guideline 3].

4 Management when unsure if the blood glucose

level is high or low

When unsure if the person has a high or low blood glucose level, the safest option is to treat as for *hypoglycaemia* (low blood glucose level). Treatment may lead to a marked improvement if the blood glucose level is low, and is unlikely to do harm if the blood glucose level is high. [Refer to ANZCOR Diabetes fact sheet - appendix]

4.1 Use of blood glucose measuring devices (Glucometers)

If trained to do so and a glucometer is available, checking the person's blood glucose level will guide management, and can confirm *hypoglycaemia* or *hyperglycaemia*. Normal blood glucose concentrations are between 4.0 mmol/L and 7.8 mmol/L.

A blood glucose level between 3.0 mmol/L and 4.0 mmol/L is an 'alert value', meaning that to prevent progression to a more serious, clinically important hypoglycaemia, it is time for a normal food intake, either a snack or meal, depending on the time of day and usual food intake habits.[5]

Clinically important *hypoglycaemia* is defined as a blood glucose level less than 3.0 mmol/L, where there is decreased neuro-cognitive function (reasoning ability or orientation) and increased morbidity (illness) and mortality.[5]

Symptoms of hypoglycaemia may be mimicked by other conditions such as stroke, epilepsy, or migraine. If trained, checking a blood glucose will improve the accuracy of diagnosing hypoglycaemia. We suggest that if the blood glucose concentration is normal, and symptoms and signs of hypoglycaemia persist, other diagnoses need to be considered.[3] [CoSTR 2015: weak recommendation/low quality evidence]

Hyperglycaemia is defined as a blood glucose level above the normal reference range. Severe hyperglycaemia is defined as a blood glucose level greater than 15 mmol/L.

References

1. Singletary EM, Zideman DA, Bendall JC, Berry DC, Borra V, Carlson JN, Cassan P, Chang WT, Charlton NP, Djärv T, Douma MJ. 2020 International Consensus on First Aid Science With Treatment Recommendations. Circulation. 2020 Oct 20;142(16_suppl_1):S284- 334.
2. International Hypoglycaemia Study Group. Minimizing Hypogylcemia in Diabetes. Diabetes Care. August 2015. Volume 38: 1583 -
3. Zideman, D. A., Singletary, E. M., De Buck, E., et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. Resuscitation, 95, e225.
4. Carlson JN, Schunder-Tatzber S, Neilson CJ, Hood N. Dietary sugars versus glucose tablets for first-aid treatment of symptomatic hypoglycaemia in awake patients with diabetes: a systematic review and meta-analysis. Emerg Med J 2016; 34:100-106.
5. Villani M, de Courten B, Zoungas S. Emergency treatment of hypoglycaemia: a guideline and evidence review. Diabet Med. 2017 Sep;34(9):1205-1211.
6. International Hypoglycaemia Study Group. Glucose Concentrations of Less Than 3.0mmol/L (54mg/dL) Should Be Reported in Clinical Trials: A Joint Position Statement of the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care. Jan 2017, 40 (1) 155-157; DOI: 10.2337/dc16- 2215

Further Reading

- ANZCOR Guideline 2 - Managing an Emergency
- ANZCOR Guideline 3 - Recognition and First Aid Management of the Unconscious Victim
- ANZCOR Guideline 8 - Cardiopulmonary Resuscitation
- ANZCOR Guideline 9.2.2 - Stroke

About this Guideline

- Search date/s: August 2018
- Question/PICO:

    - Population: Adults and children with suspected hypoglycaemia^ (out-of-hospital, including healthy volunteers). Neonates are excluded, as we believe the identification of hypoglycaemia in this age group is a specialized diagnostic and treatment process well beyond First Aid.
    - Intervention: Administration of glucose by any enteral route appropriate for use by first aid providers
    - Comparators: Administration of glucose by another enteral route appropriate for use by first aid providers
- Outcomes:
    - Resolution of symptoms (critical) - defined as the reversal of the initial symptoms (dichotomous outcome; yes/no).
    - Time to resolution of symptoms (critical) - defined as the time from the administration of the sugar containing solution until the symptoms resolved (continuous outcome).
    - Blood or plasma glucose concentration at 20 minutes (critical) - defined as the glucose level as measured 20 minutes after the administration of the sugar substrate (continuous outcome) or as evidence of blood or plasma glucose elevation at 20 minutes (dichotomous outcome; yes/no).
    - Resolution of hypoglycaemia (Important) - defined as elevation of the blood glucose level to rise above the authors' threshold for determining hypoglycaemia (dichotomous outcome; yes/no).
    - Time to resolution of hypoglycaemia (Important) - defined as the time from the administration of the sugar containing solution until the blood glucose concentration rose above the threshold for the authors' definition of hypoglycaemia (continuous outcome).
    - Any adverse event (Important) - any event resulting from the administration as defined by the study authors (e.g. aspiration).
    - Administration delay (Important) - defined as the delay in administering the sugar containing solution as a result of the administration arm (dichotomous outcome; yes/no).
- Study Designs: Randomized and nonrandomized clinical trials, observational studies were included. Unpublished studies (e.g., conference abstracts, trial protocols, methods papers) were excluded.
- Timeframe: All years and all languages were included provided there was an English abstract from inception to December 22, 2017 with an update performed on July 11, 2018.
- Method: Systematic Review by ILCOR with CoSTR available at ILCOR.org
- Primary reviewers: Natalie Hood, Finlay Macneil
- Other consultation: Nil
- Worksheet: See https://www.ilcor.org/
- Approved: April 2021
- Guidelines superseded: ANZCOR GL 9.2.9 - November 2017

DIABETES FACT SHEET FOR FIRST AIDERS

Diabetes is a disorder of regulation and use of sugar. It is due to a lack of insulin production or resistance of the body to insulin, one of the hormones that controls the use of glucose (a simple sugar used by the body to provide energy for various functions). Untreated, the absolute or relative lack of insulin will lead to a high blood glucose level. There are two main types of diabetes. ‘Type 1 diabetes’ is an auto-immune disease (the body attacks and destroys part of itself) that often develops in childhood, and requires lifelong treatment with insulin. ‘Type 2 diabetes’ is more commonly recognised in adulthood, and requires a treatment combination of diet, exercise, medication, and sometimes insulin. Less commonly, ‘gestational diabetes’ may develop in pregnancy, and diabetes can also occur as a consequence of another disease or as a side effect of medication.

Normally the body tightly controls its blood glucose level within a ‘normal’ range. Having diabetes interferes with this control system, and people living with diabetes need to manage their own blood glucose levels by monitoring what they eat, adjusting their insulin or medication doses, and frequently testing their own blood glucose levels.

The reasoning behind the practice of assuming hypoglycaemia for a diabetic unwell due to the diabetes is based on the CoSTR (consensus on science and treatment recommendations) published in 20181 which made the following recommendations:

- We recommend the use of oral glucose (swallowed) for individuals with suspected hypoglycemia who are conscious and able to swallow (strong recommendation, very low certainty of evidence).
- We suggest against buccal glucose administration compared with oral glucose administration for individuals with suspected hypoglycemia who are conscious and able to swallow (weak recommendation, very low certainty of evidence).
- If oral glucose (e.g. tablet) is not immediately available, we suggest a combined oral + buccal glucose (e.g. glucose gel) administration for individuals with suspected hypoglycemia who are conscious and able to swallow (weak recommendation, very low certainty of evidence).
- We suggest the use of sublingual glucose administration for suspected hypoglycaemia for children who may be uncooperative with the oral (swallowed) glucose administration route (weak recommendation, very low certainty of evidence).

The problem is that most first aiders do not have a glucometer and are not trained in the use of one.

If a diabetic is unwell due to the diabetes, there are 2 possibilities, hypoglycaemia or hyperglycaemia. If oral glucose or glucagon is given to an unwell diabetic person with:

- hypoglycaemia and able to swallow, they will improve.
- hyperglycaemia, they will not get much worse in the short term

If oral glucose or glucagon is withheld from an unwell diabetic person with:

- hypoglycaemia, they will continue to deteriorate rapidly and may not be in a position to swallow safely in a short time
- hyperglycaemia, they will continue to deteriorate slowly till IV rehydration and insulin administration.

Thus there is no short term problems with giving oral glucose or glucagon to a person with diabetic emergency, but major problems if glucose or glucagon is withheld from some persons with a diabetic emergency. Hence ANZCOR recommends that first aiders treat an unwell person with diabetes for hypoglaemia.

Reference:

1. Borra V, Carlson JN, De Buck E, Djärv T, Singletary EM, Zideman D, Bendall J, Berry DC, Cassan P, Chang WT, Charlton NP, Hood NA, Meyran D, Woodin JA, Swain J. Glucose administration routes for first aid in case of symptomatic hypoglycemia. Consensus on Science and Treatment Recommendations [Internet] Brussels, Belgium: International Liaison Committee on Resuscitation (ILCOR) First Aid Task Force, 2018 Aug 27. Available from: http://ilcor.org

ANZCOR Guideline 9.2.10 - The Use of Oxygen in Emergencies

Summary

Who does this guideline apply to?

This guideline applies to adults, children and infants.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. Basic Life Support measures should never be delayed whilst waiting for oxygen or other equipment. [Good Practice Statement]
2. The administration of supplemental oxygen should be limited to individuals with specific training in oxygen administration.[1,2] [Good Practice Statement]
3. When bag-valve-mask oxygen resuscitation is used by trained but occasional operators, a minimum of two trained rescuers are required to provide ventilation for a non-breathing person. [Good Practice Statement]
4. Persons who require supplemental oxygen in a first aid setting be further evaluated by a health care professional. [Good Practice Statement].

Abbreviations

ANZCOR- Australian and New Zealand Committee on Resuscitation CoSTR- Consensus on Science with Treatment Recommendations (from International Liaison Committee on Resuscitation - ILCOR) ANZTS- Australia and New Zealand Thoracic Society

Guideline

1 Introduction

Administration of supplemental oxygen is traditionally considered essential for individuals presenting with shortness of breath, difficulty breathing, or hypoxemia (low oxygen level in the blood). In certain circumstances, oxygen supplementation might have adverse effects that complicate the disease course or even worsen clinical outcomes.[1] The administration of supplemental oxygen should be limited to individuals with specific training in oxygen administration.[1,2] [Good practice statement]

There is evidence to support the use of oxygen as part of first aid management of decompression illness[1] and for shortness of breath (dyspnoea) in cancer patients with hypoxaemia.[1]

The use of oxygen delivery devices, such as bag-valve-mask equipment and oxygen powered resuscitation equipment, should also only be undertaken by those who are trained in their use (with current training and certification). [Good Practice Statement]

2 Equipment

There are many types of oxygen delivery devices available, ranging from the simple oxygen mask, which can be used with very little training, to the more complex bag-valve-mask ventilation equipment.

It is recommended that when bag-valve-mask oxygen resuscitation is used, a minimum of two trained people are required to provide ventilation for a non-breathing person: one to manage the airway, mask and seal, and the second to operate the bag.[3] [Good Practice Statement]

If two trained people are not available to provide ventilation for a non-breathing person then mouth- to-mask breathing using a resuscitation face mask with supplemental oxygen will provide adequate oxygenation and ventilation.[4] [Refer to ANZCOR Guideline 5].

3 Management

Basic Life Support measures should never be delayed whilst waiting for oxygen or other equipment.[6] [Good Practice Statement]

In the non-breathing person, oxygen may be used if available by mouth-to-mask, bag-valve- mask or positive pressure oxygen delivery system, if the appropriate equipment and personnel with current training and certification in its use are available. [Good Practice Statement]

A person who requires supplemental oxygen in a first aid setting requires further assessment by a health care professional so an ambulance must always be sent for.

3.1 Use of pulse oximetry

It is best practice that the use of supplemental oxygen is guided by pulse oximetry [2,5] [TSANZ Grade C recommendation: 'Body of evidence provides some support for recommendation(s) but care should be taken in its application'].[5]

Oxygen should be administered to persons with an oxygen saturation of less than 92%.[5]

Oxygen should be given to persons signs with cyanosis (blue colouration of skin), shock including from major injury,[2] decompression illness[6] or a situation suggesting carbon monoxide poisoning[7] (eg. house fire) irrespective of their oxygen saturation level or whether pulse oximetry is available.

3.2 Oxygen administration in specific circumstances

Conditions where oxygen is recommended include:

- during cardiopulmonary resuscitation [Refer to ANZCOR Guideline 11.1.1 and ANZCOR Guideline 12.2]
- bleeding [Refer to ANZCOR Guideline 9.1.1]
- burns [Refer to ANZCOR Guideline 9.1.3]
- shock [Refer to ANZCOR Guideline 9.2.3]
- asthma [Refer to ANZCOR Guideline 9.2.5]
- anaphylaxis [Refer to ANZCOR Guideline 9.2.7]
- drowning [Refer to ANZCOR Guideline 9.3.2]
- decompression illness [Refer to ANZCOR Guideline 9.3.5]
- poisoning [Refer to ANZCOR Guideline 9.5.1].

Oxygen use in persons with stroke [Refer to ANZCOR Guideline 9.2.2] and heart attack [Refer to ANZCOR Guideline 9.2.1] who do not have signs of shock should be guided by pulse oximetry as excessive oxygen may be harmful in these conditions.[2,5,8-10] ANZCOR suggests against the routine administration of oxygen in persons with stroke.[11,12] [2020 CoSTR, weak recommendation, low- to- moderate certainty evidence] ANZCOR suggests that for persons with stroke, the routine use of oxygen is not recommended if the oxygen saturation is >92% on room air [National Stroke Foundation: weak recommendation, moderate-to-high certainty evidence].[8]

ANZCOR suggests against the routine administration of oxygen in persons with chest pain.[13] [COSTR, weak recommendation, very-low certainty evidence] For persons with heart attack, routine use of oxygen is not recommended if the oxygen saturation is >93% [National Heart Foundation of Australia & Cardiac Society of Australia and New Zealand: practice advice].[9]

References

1. Zideman DA, Singletary EM, De Buck EDJ, et al. Part 9: First aid 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2015; **95** : e225-e61.

2. O'Driscoll BR, Howard L, Earis J, et al. BTS guideline for oxygen use in adults in healthcare and emergency settings. *Thorax* 2017; **72** (Suppl 1): ii1-ii90.
3. Jesudian MC, Harrison RR, Keenan RL, Maull KI. Bag-valve-mask ventilation; two rescuers are better than one: preliminary report. *Crit Care Med* 1985; **13** (2): 122-3.
4. Harrison RR, Maull KI, Keenan RL, Paul Boyan C. Mouth-to-mask ventilation: A superior method of rescue breathing. *Annals of emergency medicine* 1982; **11** (2): 74-6.
5. Beasley R, Chien J, Douglas J, et al. Thoracic Society of Australia and New Zealand oxygen guidelines for acute oxygen use in adults: 'Swimming between the flags'. *Respirology* 2015; **20** (8): 1182- 91.
6. Longphre J, Denoble P, Moon R, Vann R, Freiberger J. First aid normobaric oxygen for the treatment of recreational diving injuries. *Undersea & hyperbaric medicine : journal of the Undersea and Hyperbaric Medical Society, Inc* 2007; **34** (1): 43-9.
7. Wolf SJ, Maloney GE, Shih RD, Shy BD, Brown MD. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute carbon monoxide poisoning. *Annals of emergency medicine* 2017; **69** (1): 98-107.
8. National Stroke Foundation. Clinical Guidelines for Stroke Management 2017. Chapter 3 of 8: Acute medical and surgical management. Melbourne. Retrieved 20 November 2020 from https://informme.org.au/en/Guidelines/Clinical-Guidelines-for-Stroke-Management-2017: National Stroke Foundation, 2017.
9. Chew D, Scott I, Cullen L, et al. National Heart Foundation of Australia and Cardiac Society of Australia and New Zealand: Australian clinical guidelines for the management of acute coronary syndromes 2016. *Med J Aust* 2016; **205** (3): 128.
10. Stub D, Smith K, Bernard S, et al. Air versus oxygen in ST-segment-elevation myocardial infarction. Circulation. 2015 Jun 16;131(24):2143-50.
11. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science With Treatment Recommendations. *Circulation* 2020; **142** (16_suppl_1): S284-S334.
12. Singletary EM, Zideman DA, Bendall JC, et al. 2020 International Consensus on First Aid Science With Treatment Recommendations. *Resuscitation* 2020; **156** : A240-A82.
13. Nikolaou NI, Welsford M, Beygui F, et al. Part 5: Acute coronary syndromes 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Resuscitation* 2015; **95** : e121-e46.

About this Guideline

- **Search date/s** November 2020
- **Method:** Evidence update based on British Thoracic Society and Thoracic Society of Australia and New Zealand Guidelines

- **Primary reviewers:** Julie Considine
- **Other consultation:** Finlay Macneil
- **Worksheet:** See BTS and TSANZ publications
- **Approved:** April 2021
- **Guideline superseded:** ANZCOR Guideline 9.2. 10 - January 2016

ANZCOR Guideline 9.2.11 - First Aid Management of The Agitated Person

Summary

Who does this guideline apply to?

This guideline applies to adults, adolescents and children

Who is the audience for this guideline?

This guideline is for bystanders, first aiders and first aid training providers

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) make the following recommendations:

1. Ensuring your safety and the safety of others; send for help early. [Ungraded, Good practice statement]
2. Attempt de-escalation strategies/techniques only if experienced in these techniques. [Ungraded, Good practice statement]
3. Identify and treat any medical conditions and/or injuries when safe to do so. [Ungraded, Good practice statement]
4. Avoid physical restraint for an aggressive, agitated/behaviourally disturbed person. [Ungraded, Good practice statement]

Guideline

1 Introduction

First aiders and first aid providers may encounter persons presenting with abnormal behaviour including agitation, aggression and abnormal thinking or thoughts. Behavioural disturbances can range from mild to life threatening. Professional healthcare assessment is needed to determine the most likely cause of the abnormal behaviour to guide appropriate treatment. The immediate goals of first aid for the agitated/behaviourally disturbed person is keeping yourself, others

and the person safe from harm. Severe behavioural disturbance is behaviour that puts the disturbed person or others at immediate risk of serious harm and may include threatening or aggressive behaviour, extreme distress, and serious self-harm which could cause major injury or death.[1]

2 Causes

Agitation or behavioural disturbance can have many causes and may or may not be related to a mental health disorder or other illness. There are many causes of agitation / behavioural disturbance including:

- medical conditions e.g. head injury, hypoxia (low oxygen levels in blood), infections (e.g. meningitis), seizures, metabolic derangements (e.g. low blood sugar, electrolyte disturbance), organ failures (e.g. liver & kidney), dementia and delirium
- intoxication / withdrawal e.g. alcohol, hallucinogens, stimulants (e.g. amphetamine type substances, cocaine), cannabis, synthetics, opioids, sedatives
- mental health conditions e.g. psychotic disorders (e.g. schizophrenia), anxiety disorders, and personality disorders
- others: developmental disorders e.g. intellectual disability, autism spectrum disorders, grief, situational stress and pain.

These causes may be applicable to the person requiring first aid and/or others at the scene such as a parent, friend, partner or family member. The first aid management of agitation / behavioural disturbance may apply to one or more people at the same time[2].

3 Recognition

Agitation / behavioural disturbance encompasses a variety of symptoms and signs alone or in combination. Diagnosis is largely based on history and physical findings. Symptoms and signs are highly variable but include:

- increased arousal (e.g. agitation, excitation, restlessness, pacing, tearful, wringing hands, screaming, yelling, frightened, frantic)
- a rigid body language (an indicator of an intense effort to control themselves)
- abnormal or unusual thinking, perception or ideas (e.g. hallucinations)
- inappropriate clothing for the climate or context
- altered conscious state
- aggressive / violent / argumentative / bizarre behaviour.

A severe and potentially life-threatening form of behavioural disturbance is present when the person has:

- an elevated body temperature, is hot to touch or is sweating profusely
- insensitivity to pain (e.g. may be walking with a broken leg or severe injury)
- a rapid respiratory rate and rapid pulse rate
- extreme arousal with aggression or violence.

4 Management

The initial approach to a person with agitation / behavioural disturbance should be focused on safety. De-escalation strategies are extremely difficult without training and experience. They can exacerbate the situation if not performed properly. They should not be attempted unless trained and skilled at the technique. The important point is to stay safe and seek help. The points below are given as information on how to avoid further danger to first aiders and bystanders.[1-5]

Principles

- Ensure your safety and the safety of others - seek appropriate support and assistance early (e.g. ambulance, security services, police, mental health professionals).
- Reassure - empathise and listen actively, if it is safe and trained to do so. Listen closely and non-judgementally to what the person is saying and feeling.
- Seek advice or assessment from a healthcare professional.
- If the person deteriorates or becomes unconscious, manage the person according to ANZCOR Guideline 3.
- If the person becomes unresponsive and not breathing normally, give resuscitation following the Basic Life Support Flowchart (ANZCOR Guideline 8).

5 Staying safe

Staying safe is a priority. Be aware of the potential danger and ensure safety of first aiders, others and the behaviourally disturbed person. If you are unsure or feel threatened in any way, remove yourself

and others from the situation, seek a safe space and send for appropriate support and assistance.

- Avoid being alone with the person and when possible keep at least two arm lengths away.
- Always face the person, maintain visual contact and never turn your back.
- Be vigilant for signs of violence or escalation.
- Make sure there is access to two exits if possible and avoid blocking exits.
- Remove any object that could be used as weapons.
- People who are calming may be of assistance and try to keep conflict partners away from the person.
- Speak politely and with non-threatening body language.
- Reduce external stimuli such as noise, odour, light, and background movement.

- Be aware of the person's cultural background to avoid words or actions that are taboo or could shame the person.
- It is helpful to find someone the person knows and trusts to help with their care, but do not leave the scene to find that person.
- Be aware the person may act on a delusion or hallucination and this may not make sense to the first aider.
- If a person does become violent or you feel unsafe; stop managing them, move to safety and send for appropriate support and assistance.
- Watch for decreasing level of consciousness.

6 Behavioural disturbance in children

The effective management of behavioural disturbance in children is difficult and requires specialised training and extensive experience, increasing the need to seek appropriate support and assistance early. The same general principles of management apply as described above modified to the child's age and needs. The initial focus of management should be on your safety, the safety of others and the safety of the child. Involvement of the child's family/ carer (if available and appropriate) will generally be helpful in the first aid setting. The child's family / carer will be able to provide advice and assistance to determine the most likely effective de-escalation strategies such as age appropriate distraction techniques (e.g. toys).

7 Physical restraint

Physical restraint is associated with potential harm to both the aggressive or agitated / behaviourally disturbed person and the care provider. The risks of physical restraint typically outweigh the benefits. ANZCOR recommends avoiding the physical restraint of aggressive, agitated/behaviourally disturbed persons by bystanders / first aiders and first aid providers.

References

1. New South Wales Health 2015, Management of patients with Acute Severe BehaviouralDisturbance in Emergency Departments, viewed 15 March 2019, https://www1.health.nsw.gov.au/pds/ActivePDSDocuments/GL2015_007.pdf
2. Iroku-Malize, T. & Grisson, M. The agitated patient: Steps to take, how to stay safe. *The Journal* of Family Practice. 2018 March; 67(3): 136 -147.
3. Zeller S, Nordstrom K, Wilson M, The Diagnosis and management of Agitation. 2017 Cambridge: Cambridge University Press
4. Richmond JS, Berlin JS, Fishkind AB, et al. Verbal De-escalation of the Agitated Patient: Consensus Statement of the American Association for Emergency Psychiatry Project BETA De- escalation Workgroup. West J Emerg Med. 2012;13(1):17–25. doi:10.5811/westjem.2011.9.
5. Robyn L. Langlands, Anthony F. Jorm, Claire M. Kelly, and Betty A. Kitchener First aid recommendations for psychosis: using the Delphi method to gain

consensus between mental health consumers, carers, and clinicians. Schizophr Bull. 2008 May;34(3):435-43.

Further Reading

- ANZCOR Guideline 2 - Managing an emergency
- ANZCOR Guideline 8 - Cardiopulmonary Resuscitation
- ANZCOR Guideline 9.5.1 - Emergency management of a person who has been poisoned
- Kitchener BA, Jorm AF, Kelly CM. Mental Health First Aid Manual. 4th ed. Melbourne: Mental Health First Aid Australia; 2017

About this Guideline

- Search date/s: November 2019
- Question/PICO: (Aggression or agitation) (and (in out of hospital setting)) - used as later filter (P), does any intervention (I) vs none [C], affect outcome (O)
- Method: Scoping literature review plus consensus of experts on ANZCOR and Red Cross consulted by first author and advice in Kitchener, Iorm and Kelly. Mental Health First Aid Manual. 4th ed. Melbourne: Mental Health First Aid Australia; 2017
- Primary reviewers: Tom Clark, Jason Bendall, Finlay Macneil
- Other consultation: NSW Health Policy Document PD2015_004 (accessed 1 November 2019) Hugh Grantham, Tracy Kidd and Ella Tyler
- Approved: 20 November 2020
- Guidelines superseded: N/A new guideline

ANZCOR Guideline 9.2.12 - Recognition and

First Aid Management of the Seriously Ill Person including Sepsis

Summary

Who does this guideline apply to?

This guideline applies to adults, children and infants.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. First aiders learn to recognise a seriously ill person [Good Practice Statement]
2. If serious illness is suspected, send for an ambulance - delayed treatment can quickly lead to more serious illness and death. [Good Practice Statement]

Abbreviations

ANZCOR: Australian and New Zealand Committee on Resuscitation

Guideline

1 Introduction

There are many conditions that present in the community that need urgent assessment and treatment by health care professionals. The nature of most of them is obvious but some are difficult to diagnose even under ideal circumstances in hospital. One frequent example is sepsis. There are a number of other conditions that are hard to distinguish from sepsis. However, the diagnosis of the exact condition is not important for the first aider because these conditions share a common set of symptoms and signs. It is more important to recognise that a person needs urgent medical care than to diagnose the nature of the illness. There is very little published about the first aid management or recognition of these conditions. There is a growing body of evidence about how health care professionals should 'recognise the deteriorating patient.' The purpose of this guideline is to help the first aider recognise the person in need of urgent medical care but is not intended for the diagnosis of illnesses.

More information on sepsis can be found on the Australian Sepsis Network website (https://www.australiansepsisnetwork.net.au)

2 Causes

Anyone can deteriorate quickly with a serious illness, but certain people are at higher risk including[1] :

- children under 10[1]
- people over 65 years of age[2,3,4]
- people with chronic diseases
- people with weakened immune systems
- Aboriginal and Torres Strait Islander Peoples, Maori and Pasifika

3 Recognition

Early recognition of serious illness is critical as early treatment improves outcomes. These symptoms and signs set out below may indicate serious illness. These symptoms and signs are common to many conditions and it is their combination that alerts health professionals^ to the possibility of serious illness and prompts further investigation and treatment. The more signs and symptoms in combination, the higher the risk that the underlying problem is a serious illness. Perhaps the most important indicator is that the person with a serious illness feels 'not right' or say they might feel they are 'going to die'.[9] This is even more significant if the people that know this person have noticed a change in their behaviour.[6]

3.1 Red Flags for serious illness.

The more red flags present, the greater the concern that the person is seriously ill.

The bulk of the evidence related to serious illness in adults is from in-hospital studies, so the indicators of serious illness in adults are extrapolated from that evidence. The indicators of serious illness in adults include:[8-13]

- rapid breathing (breathing rate ≥22 / minute) is the most reliable indicator of serious illness in adults
- breathlessness or feeling short of breath
- restlessness, agitation, dizziness, decreased level of consciousness, confusion, slurred speech or disorientation
- shivering or shaking, fever or feeling very cold
- unexplained muscle pain or discomfort
- passing little or no urine
- rapid heart rate
- nausea and or vomiting
- new rash or blotchy, pale, or discoloured (often described as mottled) skin;
- person may say they 'don't feel right' or they might say they feel like they 'are going to die'.

3.2 Serious illness in children and infants

Children and infants with serious illness can deteriorate quickly. Symptoms and signs of serious illness in infants and children may include:[1][4]

- rapid breathing, weak cry or grunting
- hard to wake, lethargic or floppy
- seizure or fits
- a rash that doesn't fade when pressed
- discoloured, mottled, very pale or bluish skin
- fever, feeling cold or cold to touch
- vomiting repeatedly
- not passing urine (or no wet nappy) for several hours
- not feeding or drinking.

Children often cannot express how they feel so look for the combination of an infection with any of the signs and symptoms listed.

In children, parental concern that this illness is more severe or different and care providers thinking “something is wrong” are predictive of the presence of sepsis.[5]

4 Management

Serious illness is a medical emergency and typically requires in-hospital management and the prompt administration of medications or an operation which targets the infection or other illness.

Send for an ambulance if:

- you suspect sepsis or other serious illness;
- an infection related illness is not improving;
- carer is concerned that this illness is more severe or different;
- care providers (including first aiders) think “something is wrong”.

4.1 Those that are unresponsive, unconscious or fitting

Send for an ambulance.

- if the person is unresponsive and not breathing normally, commence resuscitation following the Basic Life Support Flowchart [Refer to ANZCOR Guideline 8];
- if the person is unconscious but breathing, lie them on their side, ensure the airway is clear [Refer to ANZCOR Guideline 3] and keep them under observation;
- if the person is having a seizure, lie them on their side, ensure the airway is clear [Refer to ANZCOR Guideline 3 and Guideline 9.2.4] and keep them under observation;
- administer oxygen only if there are obvious signs of shock or evidence of low oxygen saturation according to use of Oxygen in Emergencies [Refer to ANZCOR Guideline 9.2.10].

4.2 Those that are conscious

Send for an ambulance.

- lie the person down if comfortable lying down;
- treat shock if present [Refer to ANZCOR Guideline 9.2.3], but do not cover with a blanket if the person already feels hot to touch;
- consider administering oxygen if indicated as per ANZCOR Guideline 9.2.10;
- reassure and constantly re-check the person's condition for any change.

References

1. Melanie K Prusakowski MD and Audrey P Chen PNP (2017). “Pediatric Sepsis.” Emergency Medicine Clinics of North America Volume 35, Issue 1, February 2017, Pages 123- 138
2. Fernando SM, Reardon PM, McIsaac DI, et al. (2018) Outcomes of older hospitalized patients requiring rapid response team activation for acute deterioration. *Critical Care Medicine* 2018; **46** (12): 1953- 60.
3. Considine J, Street M, Hutchinson AM, et al. (2020) Vital sign abnormalities as predictors of clinical deterioration in subacute care patients: a prospective case-time-control study. *International Journal of Nursing Studies* 2020; **108** : 103612.
4. Jayasundera, R., Neilly, M., Smith, T. O., & Myint, P. K. (2018). Are early warning scores useful predictors for mortality and morbidity in hospitalised acutely unwell older patients? A systematic review. *Journal of clinical medicine* , *7* (10), 309.
5. Harley A, Latour JM, Schlapbach LJ. (2019) The Role of Parental Concerns in the Recognition of Sepsis in Children: A Literature Review. Front Pediatr. 2019;7:161.
6. Kumar A, Roberts D, Wood KE, Light B, Parrillo JE, Sharma S et al. (2006) Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock. Crit Care Med 2006;34:1589-96.

7. Fenella J. Gill, Gavin D. Leslie, Andrea P. Marshall, (2016) The Impact of Implementation of Family- Initiated Escalation of Care for the Deteriorating Patient in Hospital: A Systematic Review. Worldviews on Evidence-Based Nursing, 2016; 13:4, 303–313.
8. Knight, C. and L. Glennie (2010). "Early recognition of meningitis and septicaemia." The journal of family health care **20** (1): 6-8.
9. Olander A, Andersson H, Sundler AJ, Bremer A, Ljungström L, Hagiwara MA. Prehospital characteristics among patients with sepsis: a comparison between patients with or without adverse outcome. BMC emergency medicine. 2019 Dec;19(1):1-8.
10. Singer M, Deutschman CS, Seymour CW, et al. The third international consensus definitions for sepsis and septic shock (sepsis-3). (2016) *Journal Of The American Medical Association.* 2016;315(8):801-810.
11. Cretikos M, Chen J, Hillman K, Bellomo R, Finfer S, Flabouris A. (2007) The objective medical emergency team activation criteria: a case-control study. *Resuscitation.* 2007;73(1):62-72.
12. Burrell AR, McLaws M-L, Fullick M, Sullivan RB, Sindhusake D. (2016) SEPSIS KILLS: early intervention saves lives. *Med J Aust.* 2016;204(1):1.e1 - 1.e7.
13.
    i. Bleyer AJ, Vidya S, Russell GB, et al. (2011) Longitudinal analysis of one million vital signs in patients in an academic medical center. Resuscitation. 2011;82(11):1387-1392.
14. Van den Bruel A, Haj-Hassan T, Thompson M, Buntinx F, Mant D, European Research Network on Recognising Serious Infection investigators. Diagnostic value of clinical features at presentation to identify serious infection in children in developed countries: a systematic review. The Lancet. 2010 Mar 6;375(9717):834-45.

About this Guideline

- Search date/s: November 2019
- Question/PICO: In adults, children and infants, is there any reliable method of recognizing sepsis available to a first aider in out of hospital setting?
- Method: Scoping review
- Primary reviewers: Jason Bendall, Finlay Macneil, Natalie Hood, Hugh Grantham
- Other consultation Australian Sepsis Network
- Approved: April 2021
- Guidelines superseded: N/A - new guideline

Wilderness Medical Society Practice Guidelines for the Treatment of Acute Pain in Remote Environments: 2014 Update

Katie W. Russell, MD; Courtney L. Scaife, MD; David C. Weber, BS; Jeremy S. Windsor, MB ChB; Albert R. Wheeler, MD; William R. Smith, MD; Ian Wedmore, MD; Scott E. McIntosh, MD, MPH; James R. Lieberman, MD

From the Department of Surgery, University of Utah School of Medicine, Salt Lake City, UT (Drs Russell and Scaife); Denali National Park & Preserve Rescue, Talkeetna, AK (Mr Weber); the Chesterfield Royal Hospital, Calow, Derbyshire, United Kingdom (Dr Windsor); St. John's Medical Center, Jackson, WY (Drs Wheeler and Smith); the Clinical Faculty, University of Washington School of Medicine, Seattle, WA (Dr Smith); the University of Washington School of Medicine, Madigan Army Medical Center, Ft. Lewis, WA (Dr Wedmore); the Division of Emergency Medicine, University of Utah School of Medicine, Salt Lake City, UT (Dr McIntosh); and the the Department of Anesthesia, Swedish Medical Center, Seattle, WA (Dr Lieberman).

The Wilderness Medical Society convened an expert panel to develop evidence-based guidelines for the management of pain in austere environments. Recommendations are graded on the basis of the quality of supporting evidence as defined by criteria put forth by the American College of Chest Physicians. This is an updated version of the original WMS Practice Guidelines for the Treatment of Acute Pain in Remote Environments published inWilderness & Environmental Medicine2014;25(1):41–49. Key words:pain control, analgesia, sedation, local anesthesia, wilderness medicine, austere, remote, oligoanalgesia

Introduction

Evidence suggests that oligoanalgesia, the under treatment of acute pain, is a recurring issue in the management of patients in the prehospital setting.[1,2] A recent study evaluating helicopter transfers of 1200 trauma patients found that analgesia was inadequate in 43% of those transported.[2] Similar trends are likely to occur in austere environments where medical personnel and supplies are often limited.

Practitioners often report a reluctance to provide adequate pain management because of a wide range of different factors. These include inappropriate estimation of pain by the provider, a lack of medication or the means to administer the necessary analgesics, lack of pharmacologic knowledge, a fear of addiction,

concern of masking potential clinical deterioration, and life-threatening side effects such as respiratory depression, hemodynamic instability, and aspiration.[3,4] Acute untreated pain is not the only consequence of inadequate analgesia. Failure to adequately manage pain may also cause a significant stress response as well as an increase in the risk of developing posttraumatic stress disorder.[5] Patients may also become increasingly sensitive to painful stimuli the longer pain remains uncontrolled, making their pain more difficult to control.[6]

Pain management is exceedingly important in the austere environment as practitioners are often faced with the difficulty of providing prolonged care or dealing with technical extrications. Efficient analgesia reduces both physical and psychological stress and helps to facilitate the comfortable evacuation of these patients to definitive care.[7] The following are qualities of an ideal pain medication for wilderness use, and should be kept in mind when used in these environments:[8]

- Compact and lightweight
- Durable
- Nonsedating
- Wide spectrum of use
- Biochemically and environmentally stable
- Multiple routes of administration
- Minimal side effects

The purpose of these guidelines is to provide a literature-based review and simple algorithm for the treatment of acute pain in austere environments. Although an ideal medication does not exist, these guidelines seek to follow such a set of requirements as closely as possible when making recommendations. These guidelines do not encompass all analgesic medications, and the committee recognizes the usefulness of other medications not fully described in this paper. Given potential adverse complications of oligoanalgesia, together with the plethora of options now available, we believe that every effort should be made to obtain optimal pain control.

Methods

A panel was convened during the 2013 Annual Winter Meeting of the Wilderness Medical Society in Park City, UT. Invitations were based on the individual's extensive clinical or research experience, and included representatives from emergency medicine, anesthesiology, surgery, military medicine, and the field of prehospital emergency medical services (EMS). Relevant articles were identified through the PUBMED database using a key word search of the following terms: wilderness pain control, prehospital pain, prehospital narcotics, prehospital opioids, prehospital regional anesthesia, fentanyl vs morphine, acetaminophen trauma, ibuprofen trauma, ketamine efficacy, anxiolysis pain, and empathy pain. Searches were initially limited to randomized controlled trials and then expanded to include a broader spectrum of research. This literature review was further supplemented by a hand search of selected articles. The majority of information has been extrapolated from EMS and hospital literature, and very limited evidence is derived directly from the wilderness setting. For the purpose of this paper, the

terms remote, austere, tactical, disaster, and wilderness are used interchangeably to describe the varied settings defined by extended patient care times and delayed or difficult access to definitive care. All articles were reviewed and the level of evidence assessed. The panel used a consensus approach to develop recommendations regarding each modality and graded the recommendations according to the criteria developed by the American College of Chest Physicians (ACCP) (see online Supplementary ACCP Table).[9]

Table 1.Verbal numeric rating scale for assessment of pain [10]

Numeric rating scale	Pain assessment
0	No pain
1 – 3	Mild pain
4 – 6	Moderate pain
7 – 10	Severe pain

Overview of Pain Control

Indications for pain control in austere environments are typically directed at musculoskeletal injuries including strains, sprains, dislocations, and fractures. Other circumstances that may require similar management include acute medical ailments and environmental injury such as cold injury, bites, stings, and burns. Mechanisms requiring detailed assessment before pain control include traumatic brain injury, spinal cord injury, or airway- compromised patients. These guidelines do not address specific logistical evacuation issues, but they do aim to make evacuations, when required, more comfortable for patients through improved analgesia. Although narcotics are frequently used for analgesia,the committee recognizes that several other options are available and may be used first and in combination with other medications. Pain scales are extensively used throughout the medical community. Although visual aids may not be available in the backcountry, a numeric rating scale (NRS;Table 1)[10] can still be used. These help to provide an initial assessment and aid caregivers wishing to quantify any response to treatment.

The Figure outlines the recommended approach to escalating analgesic care for the typical backcountry patient. The authors created this pyramid for wilderness use, based on widely adopted pain algorithms that have previously been shown to be effective.^11 By beginning care at the base of the pyramid, providers can focus their attention first on using the safest and most accessible interventions before any escalation of care. As care is escalated up the pyramid, more-invasive modalities are incorporated into the patient's treatment. Potent drugs with potentially harmful side effects will thus be reserved for those in extreme pain, and only after safer, less-invasive therapies have been considered. It is important that providers practice within their scope of practice or licensure. Scope of practice is a terminology used by national, state, and provincial licensing boards for various professions that seeks to define the procedures and actions that are permitted for the licensed individual. Providers should not administer treatments if they are not qualified by licensure, or through ability. In the United States,

protocols exist that allow some non-physician prehospital providers to practice above what urban licensure allows. This is termed an “expanded scope of practice.” This can allow non- physician providers, for example, to administer prescription medications when indicated.

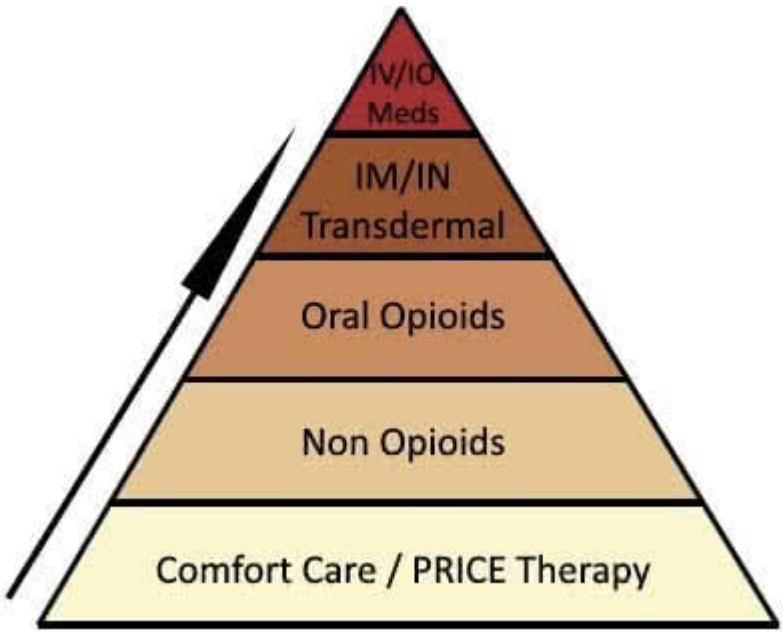


Figure.Pain management pyramid. IM, intramuscular; IN, intranasal; IO, intraosseous; IV, intravenous; PRICE, protection, rest, ice, compression, and elevation.

Recommendation

Wilderness providers should receive appropriate education and practice in various pain management techniques, commensurate with their scope of practice,in order to provide optimal pain control for patients in wilderness environments. Recommendation grade: 1C.

COMFORT CARE

Healthcare teams often overlook the utility of comforting those patients with pain and anxiety. Simple techniques, such as regularly using the patient’s name and reviewing the plan for pain management with the patient, can be very comforting, thereby decreasing their anxiety and often improving their perception of pain. This is particularly important if pain medications are not available. Sambo et al[12] studied visual analog scales and empathy and noted anxious individuals experienced“less pain in the presence of an‘empathetic’ other.”They also found lower heart rate responses to pain when individuals were not alone.[12] Designating an individual as the primary caregiver, especially for prolonged extrications, can be helpful in maximizing patient comfort and minimizing miscommunication. Because there are virtually no negative side effects to providing comfort care, this is a valuable technique that should always be included in patient management.

Recommendation

Attention to empathy should be used as first-line treatment of pain in all wilderness patient care. Recommendation grade: 1B.

PRICE TREATMENT PROTOCOL

Treatment for acute injuries has traditionally involved some variation of the RICE acronym (rest, ice, compression, and elevation). The physiological basis underpinning this approach is to reduce the formation of edema that results from injury. Extensive edema can be both painful and detrimental to the process of tissue healing.[13] Currently, PRICE (including protection of the injury) is considered the optimal treatment regimen for muscular injury and fracture,[13] whereas the MEAT (movement, exercise, analgesics, and treatments) protocol has been proposed for managing ligament and tendon injury because some inflammation actually promotes healing in these circumstances.[14] In a remote setting, it is difficult to differentiate between these injury patterns, and for this reason PRICE is still the recommended therapy for all acute soft tissue injuries. If an isolated ligament or tendon injury is highly suspected, MEAT therapy can be considered.

The following treatment strategies are recommended for optimal outcomes when enlisting the PRICE protocol:

1. **P**rotection from further injury and providing stability in the form of taping, bracing, or splinting.
2. **R**est can reduce further inflammation and pain. This said, patients can still be encouraged to self-ambulate during evacuation whenever appropriate.
3. **I**ce,when available, can decrease skin temperature to 15 deg. C, at which point nerve conduction is inhibited and pain decreases.[13] It also reduces edema formation. This should be performed at 10-minute intervals, or whenever practical, for the first 24 to 48 hours after injury.[14] If this is logistically feasible during an evacuation, Algafly et al[15] recommend 10 minutes on followed by 10 minutes off, and repeat, taking care to avoid frostbite or hypothermia, especially if the injured area becomes numb. When ice or snow is not available, cold water may be substituted for this therapy.
4. **C**ompression, with an elastic bandage, aims to reduce the swelling secondary to the acute inflammatory process. The bandage should be close fitting without compromising the circulation and should still allow adequate muscle expansion and sufficient blood flow. Bandages should be checked periodically to avoid over compression.
5. **E**levation of the injured area above the level of the heart will increase venous return to the systemic circulation and thus reduce potential swelling and aid the removal of waste products.

Recommendation

The PRICE therapy should be used for acute injury and pain concurrently with empathy as first-line treatment of pain in all wilderness patient care. Recommendation grade: 1B.

NONOPIOID ANALGESIA

Nonopioid medications have fewer side effects than opioids and often prove very effective in the management of acute pain. Therefore, when medications are required for pain control in remote environments, these should be considered first. In multiple prospective, randomized controlled trials, the combination of a nonsteroidal anti-inflammatory (NSAID) medication with acetaminophen has been demonstrated to provide superior pain control to either drug alone or in combination with an oral narcotic. The side effects have been shown to be reduced, and patient satisfaction was reported as being higher. These benefits have been seen across different injury patterns and in postoperative patients.[16–18] A systematic review of 21 studies over 10 years found that combining acetaminophen and an NSAID was superior to either drug pre- scribed alone.[19] Military applications of both NSAIDs and acetaminophen have also been successful.[20,21] In fact, some US military units give all soldiers a“combat pill pack”to be taken immediately after a penetrating extremity wound of any type. These packs contain acetaminophen, an NSAID, and an antibiotic.[8] Acute pain can be treated, in part or completely, with appropriately dosed nonnarcotics before opioids are introduced. For severe pain, the initial dose should be simultaneous, after which the medications can be administered in a simultaneous fashion or at staggered intervals.

Ketorolac is another commonly used NSAID that is often given IV or intramuscularly (IM). As an analgesic, its opioid-sparing effects have been demonstrated in numerous studies.[22,23] It is particularly useful when oral NSAIDs cannot be administered, although studies show no difference in reduction of pain scores when IM ketorolac is compared with oral ibuprofen.[24] NSAIDs do have adverse effects. They can cause renal injury, especially in dehydrated patients, and inhibit platelet function, which can increase bleeding. An association between gastrointestinal injury and long-term use of NSAIDs is also well known. Little data exist about assessing acute bleeding risk after trauma, but to extrapolate from surgical literature, multiple studies have shown that no increase in postoperative bleeding occurs with short-term use of NSAIDs.[25,26] Although prolonged use can cause peptic ulcer disease, short-term use for up to 10 days of over-the-counter NSAIDs with standard dosing has been shown to be extremely safe and well tolerated.[27–29] An ibuprofen dose of 1200 mg/d or less or naproxen at 660 mg/d or less has no greater risk than placebo.[27] Giving NSAIDs with food can alleviate gastrointestinal symptoms, and if significant abdominal pain develops, the medication should be stopped. The most important side effect related to acetaminophen use is the possibility of liver failure related to overdose. Prescription-dose acetaminophen up to 4 g/d in an adult is still appropriate for short-term use. It is considered safe in intoxicated patients, but care should be taken in chronic alcoholics and patients with hepatic dysfunction.[30,31]

As the basis of medical therapy for acute injury, these medications should be regularly dosed throughout the acute injury period.

Recommendation

Combining acetaminophen with an NSAID should be used as the first-line medication treatment of acute pain in the wilderness, unless there is a specific patient allergy or other contraindication. Recommendation grade: 1A.

OPIOID ANALGESIA

If additional medication is required, as indicated by a patient's NRS score, a move to the next level of the pyramid is recommended. When there is concern about the patient's level of consciousness, regular monitoring is required and caution should be applied when providing any analgesic agent that could have a sedative effect. Providing opioid analgesia can be challenging in the backcountry setting and may be associated with a wide range of side effects, including dysphoria, euphoria, pruritus, nausea and vomiting, sedation, loss of airway reflexes, and constipation. Nonetheless, opioids have a clear role in the treatment of moderate-to-severe pain. Universal standards for the type, dose, and mode of administration of opioid in the prehospital environment remain unclear. Compared with parenteral preparations, oral formulations of opioids have the benefit of being easier to carry, store, and administer. Therefore, these should be the most common opioid preparation carried on wilderness expeditions. Frequently, individuals, other than the providers, will carry their own opioids. Combination drugs such as those containing hydrocodone and acetaminophen (Vicodin) or oxycodone and acetaminophen (Percocet) can also be used provided that the daily maximum recommended dose of acetaminophen is not exceeded.

Medical providers in the United States and certain other countries are typically restricted to prescribing and administering medications only to the specific recipient; third-party prescribing and administering is not legally allowed in most states. A common method to comply with these laws while still carrying pain medications is for each participant to bring a supply of medications prescribed by their personal doctor.

Recommendation

Providers with appropriate licensing should carry at least 1 form of an oral opioid into the wilderness for the treatment of acute moderate and severe pain. Recommendation grade: 2B.

Transmucosal narcotics

Oral transmucosal fentanyl citrate (OTFC) is an effective means of delivering noninvasive rapid-onset pain control. Several military studies, including one with 286 patients, have concluded that OTFC provides safe and effective analgesia in the prehospital combat setting.[10,32] Literature on burn patients also supports its use.[33] This application of fentanyl has not yet become widespread in the backcountry, but has promising potential as it harbors many of the ideal qualities discussed previously—good efficacy, rapid onset, noninvasive administration, and relative ease of packing.

Recommendation

Transmucosal opioids should be used as a safe option for acute moderate-to-severe pain in remote environments. Recommendation grade: 1B.

Intranasal narcotics

Intranasal (IN) administration of narcotics provides an easy to administer, noninvasive, low-cost, rapid-onset route for providing analgesia to the injured patient. Multiple prospective, randomized, controlled trials have shown intranasal fentanyl (INF) to be equivalent in terms of efficacy compared with IV morphine. Advantages are rapid onset of pain relief without the need for IV access.[34,35] Bioavailability is estimated at 71% for INF, necessitating a conversion to 1.4 IV doses.[35] Sufentanil requires a lower volume because of its higher concentration and has been shown to be safe and effective in reducing pain in a ski clinic.[36] There are several key concepts to consider when administering IN medications: 1) the drug concentration must be maximized in the smallest volume; 2) the dose must be sufficient to overcome bioavailability limitations of the nasal mucosa; and 3) both nostrils should be used to maximize absorptive surface.[37] Based on these concepts,the most concentrated narcotic will be the easiest to use. In the United States, sufentanil would be ideal. In some countries fentanyl has been concentrated to 300µg/mL, making adequate nasal dosing easily achievable with minimal volume.

Recommendation

Intranasal opioids should be considered as highly effective safe options for acute moderate-to-severe pain control in the wilderness. Recommendation grade: 1B.

Intramuscular narcotics

Intramuscular injection of pain medication has been an important route of administration in many prehospital and hospital environments. Although the onset and efficacy may be inferior to alternative routes,^38 most narcotics can be given IM and it remains an option for patients without vascular access or when other routes of administration are inappropriate or unavailable. Of note, variations in muscle blood flow during hypoperfusion from cold temperatures or shock may make absorption rates less predictable.[39]

Newer administration techniques, such as IN and transmucosal routes, are replacing the reliance on IM injection for delivery of pain medications.

Recommendation

Intramuscular injections of narcotics can be efficacious for acute moderate-to-severe pain in the wilderness; however, supporting evidence is lacking. Recommendation grade: 2B.

Intravenous narcotics

Intravenous medications provide fast and effective analgesia. However, this may be more difficult in austere environments as IV access requires considerable experience and suitable equipment. Intraosseous access (IO) is a viable alternative, and should be considered when IV access cannot be readily obtained.^40

Intravenous analgesia has several advantages. It provides rapid pain relief and reliable drug delivery and is a familiar mode of administration for healthcare professionals. In terms of drug choice, fentanyl and morphine have been compared prospectively and proven equivalent in terms of analgesia and side effects.[41] Fentanyl has been recommended as the drug of choice for prehospital IV pain control.[42] It has a rapid onset (within 5 minutes) and a hemodynamically stable profile that is advantageous in trauma patients. Although the extra equipment and invasive nature of an IV medication makes it unreasonable for many austere settings, once IV or IO access is established these medications are still considered the gold standard for pain control. The availability of IV medications may be more reasonable in base camp scenarios where prolonged stays and delayed evacuations are more likely to occur.

Recommendation

Either IV or IO opioids, when available, should be used for acute moderate-to-severe pain in the wilderness environment when repeated doses of medications are required and need to be titrated continually. Recommendation grade: 1A.

Transdermal narcotics

Transdermal opioids can be effective in treating subacute pain for extended patient care scenarios. Fentanyl is the most common narcotic available for this route. This noninvasive method may provide consistent medication administration, making it ideal when longer-term or subacute pain control is warranted, such as for a long evacuation after stabilization. Its small size and low cost also make it an attractive wilderness analgesic. Disadvantages include its slower onset, erratic absorption, and complications, especially respiratory depression.

Recommendation

The committee has no recommendations regarding transdermal opioids. Although they can be useful in certain stable evacuation situations, there are other excellent alternatives available.

Reversal agents

Respiratory failure caused by opioid administration should always be a concern, and the ability to treat these side effects should always be available. However, with careful titration of opioid medications, this problem should be avoided.

Recommendation

All providers who administer opioid analgesics should be proficient in managing the patient's airway and respiratory status. Naloxone may be carried as an adjunct. Recommendation grade: 1C.

KETAMINE

Ketamine is a dissociative anesthetic agent and analgesic that has a number of applications in the wilderness setting. It can be used alone at higher dissociative doses for moderate to deep sedation during painful procedures. In smaller doses it can be used alone to treat pain, or in combination with opioids to enhance pain control. Ketamine is well tolerated, and has been shown to be safe when administered by nonphysician practitioners.^43 However, caution should be used to stay within a practitioner's scope of practice and experience. In appropriately titrated doses, patients can maintain their own airway because pharyngeal reflexes and spontaneous ventilation are preserved. Patients are usually amnestic after the event, which is beneficial if a prolonged evacuation ensues. Important side effects are related to increasing sympathetic discharge, resulting in increased heart rate and blood pressure, as well as increased salivation. Therefore, in higher doses adequate airway management skills are required. Ketamine is also a suitable choice when cardiovascular compromise is present. There is growing evidence that ketamine may be safely used with nonperiorbital injuries. Halstead et al[44] demonstrated no rise in intraocular pressure with ketamine for procedural sedation in 80 pediatric patients with nonperiorbital injuries. However, further clinical studies are required, and caution is advised with ocular trauma or glaucoma.

Ketamine is particularly useful in procedures that may cause extreme pain but are of short duration, including fracture reduction, manipulation in a confined space, wound repair, and amputations. It has been used successfully for procedures at high altitudes (4243 m).[45] Additionally, ketamine can be given through many routes: IM, IV, or less commonly IN, oral, sublingual, and rectal.

Recommendation

Ketamine should be used as an advanced-tier method for controlling acute moderate-to-severe pain in the wilderness setting. Recommendation grade: 1B.

LOCAL AND REGIONAL ANESTHESIA

Local anesthesia can provide a safe and effective means of analgesia for the injured patient in the wilderness. Nerve blocks can be used for analgesia for fractures, soft tissue trauma (including lacerations and crush injuries), and procedures including removal of foreign bodies and suturing. With appropriate training, regional anesthesia may be a feasible option for well-trained and practiced providers. Prehospital femoral nerve blocks for femur fracture have been shown to be a safe and effective method of acute pain control when performed by experienced physicians, typically anesthesiologists.[46,47] Attention needs to be paid to avoid intravascular injection by aspiration before injection and to stay within recommended dosing to avoid life-threatening toxicity. Regional

blocks that can be provided in the wilderness with appropriate expertise include digital block, hematoma block, intercostal block, and distal nerve block.[48] If a practitioner is prepared to suture wounds, local anesthetic should be strongly considered. Full discussion of the techniques of regional blocks is beyond the scope of this article.

Recommendation

Local anesthetics are a safe and effective option for controlling acute pain in the remote or wilderness setting. Recommendation grade: 1B.

A summary of these medications is provided in Table 2.

ADJUNCT MEDICATIONS

Anxiety can play an important role in elevating a patient's pain. It affects not only the patient but also providers. Although benzodiazepines are commonly used as an adjunct to anesthesia and analgesia in the hospital environment, there has been no significant research that evaluates the use of anxiolytics in the prehospital setting. Some physicians promote the use of benzodiazepines for their muscle-relaxant properties, but the choice to carry these must be left to the individual provider. Given the lack of research, the potential for side effects, and the practical considerations of weight and space, the committee makes no formal recommendations for adjunctive anxiolytics at this time.

Conclusions

This article provides a summary of available evidence for treatment of pain in remote settings. Most evidence is taken from the EMS, hospital, and military literature. Although minimal literature exists that is directly related to wilderness pain control, many similarities exist with other specialties, and some conclusions can be drawn. A baseline algorithm and additional options for advanced practitioners have been described in these guidelines. The goal is to better prepare providers who venture into the backcountry, and ensure that better treatment is available for those suffering from pain.

Table 2.Recommended drug doses for adult and pediatric patients

Drug	Adult dose	Pediatric dose	Route	Frequency	
Intravenous agents					
Fentanyl	1 – 2 µg/kg (50–100 µg)	0.5– 2 µg/kg	IV	5 – 15 minutes	
Ketamine	0.3–4 mg/kg (20 mg IV for pain)	0.3–4 mg/kg	IV	5 – 15 minutes	
Morphine	1 – 5 mg	0.1 mg/kg	IV	15 – 30 minutes	
IM/IN/transdermal agents					
Fentanyl	200 – 1600 µg lozenge	N/A	OTFC	15 – 30 minutes	
Fentanyl	2 µg/kg (100µg)	0.5–1.5µg/kg	IM/IN	15 – 30 minutes	
Ketamine	0.5–4 mg/kg (50–100mg)	0.3–4 mg/kg	IM/IN	15 – 30 minutes	
Morphine	5 – 10 mg	0.1 mg/kg	IM	30 minutes	
Oral opioid agents					
Hydromorphone	2 – 4 mg	0.05–0.1 mg/kg	PO	4 – 6 hours	
Oxycodone	5 – 15 mg	0.1 mg/kg	PO	4 – 6 hours	
Oral nonopioid					
Acetaminophen	650 – 1000 mg	15 mg/kg **Do not exceed 75mg/kg per day**	PO	6 hours	

Ibuprofen (substitute NSAIDs per package recommendations)|600 – 800 mg|10 mg/kg per dose|PO|6 hours|2400 mg adult| Supplementary tables

IM, intramuscular; IN, intranasal; NSAID, nonsteroidal anti-inflammatory drug; OTFC, oral transmucosal fentanyl citrate; PO, orally (per os).

Supplementary ACCP Table 1 and Evidence Table 2 are available online at doi:10.1016/j.wem.2014.07.016.

References

1. Middleton PM, Simpson PM, Sinclair G, Dobbins TA, Math B, Bendall JC. Effectiveness of morphine, fentanyl, and methoxyflurane in the prehospital setting.Prehosp Emerg Care. 2010;14:439– 447.
2. Albrecht E, Taffe P, Yersin B, Schoettker P, Decosterd I, Hugli O. Undertreatment of acute pain (oligoanalgesia) and medical practice variation in prehospital analgesia of adult trauma patients: a 10 yr retrospective study.Br J Anaesth. 2013;110:96– 106.
3. Rupp T, Delaney KA. Inadequate analgesia in emergency medicine.Ann Emerg Med. 2004;43:494– 503.
4. Davidson EM, Ginosar Y, Avidan A. Pain management and regional anaesthesia in the trauma patient.Curr Opin Anaesthesiol. 2005;18:169– 174.
5. Holbrook TL, Galarneau MR, Dye JL, Quinn K, Dougherty AL. Morphine use after combat injury in Iraq and post-traumatic stress disorder. N Engl J Med. 2010; 362:110– 117.
6. Ducharme J. Acute pain and pain control: state of the art. Ann Emerg Med. 2000;35:592– 603.
7. Chambers JA, Guly HR. The need for better pre-hospital analgesia.Arch Emerg Med. 1993;10:187– 192.
8. Wedmore IS, Johnson T, Czarnik J, Hendrix S. Pain management in the wilderness and operational setting. Emerg Med Clin North Am. 2005;23:585–601, xi–xii.
9. Guyatt G, Gutterman D, Baumann MH, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force.Chest. 2006;129:174–181.
10. Wedmore IS, Kotwal RS, McManus JG, et al. Safety and efficacy of oral transmucosal fentanyl citrate for prehospital pain control on the battlefield.J Trauma Acute Care Surg. 2012;73(6 suppl 5):S490–S495.
11. Zech DF, Grond S, Lynch J, Hertel D, Lehmann KA. Validation of World Health Organization Guidelines for cancer pain relief: a 10-year prospective study. Pain. 1995;63:65– 76.
12. Sambo CF, Howard M, Kopelman M, Williams S, Fotopoulou A. Knowing you care: effects of perceived empathy and attachment style on pain perception.Pain. 2010;151:687– 693.
13. Bleakley CM, O'Connor S, Tully MA, Rocke LG, MacAuley DC, McDonough SM. The PRICE study (Protection Rest Ice Compression Elevation): design of a randomised controlled trial comparing standard versus cryokinetic ice applications in the management of acute ankle sprain [ISRCTN13903946]. BMC Musculoskelet Disord. 2007;8:125.
14. Bleakley CM, O'Connor SR, Tully MA, et al. Effect of accelerated rehabilitation on function after ankle sprain: randomised controlled trial.BMJ. 2010;340:c1964.
15. Algafly AA, George KP, Harrington L. The effect of cryotherapy on nerve conduction velocity, pain thresh- old and pain tolerance.Br J Sports Med. 2007;41:365– 369.
16. Sniezek PJ, Brodland DG, Zitelli JA. A randomized controlled trial comparing acetaminophen, acetaminophen and ibuprofen, and acetaminophen and

codeine for post- operative pain relief after Mohs surgery and cutaneous reconstruction.Dermatol Surg. 2011;37:1007– 1013.
17. Mitchell A, van Zanten SV, Inglis K, Porter G. A randomized controlled trial comparing acetaminophen plus ibuprofen versus acetaminophen plus codeine plus caffeine after outpatient general surgery.J Am Coll Surg. 2008;206: 472 – 479.
18. Clark E, Plint AC, Correll R, Gaboury I, Passi B. A randomized, controlled trial of acetaminophen, ibuprofen, and codeine for acute pain relief in children with muscu- loskeletal trauma.Pediatrics. 2007;119:460– 467.
19. Ong CKS, Seymour RA, Lirk P, Merry AF. Combining paracetamol (acetaminophen) with nonsteroidal antiinflam- matory drugs: a qualitative systematic review of analgesic efficacy for acute postoperative pain. Anesth Analg. 2010;110:1170– 1179.
20. Black IH, McManus J. Pain management in current combat operations.Prehosp Emerg Care. 2009;13:223– 227.
21. Aldington DJ, McQuay HJ, Moore RA. End-to-end military pain management.Philos Trans R Soc Lond B Biol Sci. 2011;366:268– 275.
22. Wheatley RG. Analgesic efficacy of ketorolac. Acta Anaesthesiol Belg. 1995;47:135– 142.
23. Gillis JC, Brogden RN. Ketorolac. A reappraisal of its pharmacodynamic and pharmacokinetic properties and therapeutic use in pain management. Drugs. 1997;53: 139 – 188.
24. Arora S, Wagner JG, Herbert M. Myth: parenteral ketor- olac provides more effective analgesia than oral ibuprofen. CJEM. 2007;9:30– 32.
25. Cardwell ME, Siviter G, Smith AF. Nonsteroidal anti- inflammatory drugs and perioperative bleeding in paedi- atric tonsillectomy. Chichester, UK: Wiley. The Cochrane Library Issue 10, 2010.
26. Forrest JB, Heitlinger EL, Revell S. Ketorolac for post- operative pain management in children. Drug Saf. 1997;16:309– 329.
27. Ong CKS, Lirk P, Tan CH, Seymour RA. An evidence- based update on nonsteroidal anti-inflammatory drugs. Clin Med Res. 2007;5:19– 34.
28. Rampal P, Moore N, Van Ganse E, et al. Gastrointestinal tolerability of ibuprofen compared with paracetamol and aspirin at over-the-counter doses. J Int Med Res. 2002;30:301– 308.
29. Le Parc JM, Van Ganse E, Moore N, Wall R, Schneid H, Verrière F. Comparative tolerability of paracetamol, aspirin and ibuprofen for short-term analgesia in patients with musculoskeletal conditions: results in 4291 patients. Clin Rheumatol. 2002;21:28– 31.
30. Zimmerman HJ, Maddrey WC. Acetaminophen (para- cetamol) hepatotoxicity with regular intake of alcohol: analysis of instances of therapeutic misadventure.Hep- atology. 1995;22:767– 773.
31. Schmidt LE, Dalhoff K, Poulsen HE. Acute versus chronic alcohol consumption in acetaminophen-induced hepato- toxicity.Hepatology. 2002;35:876– 882.
32. Kotwal RS, O'Connor KC, Johnson TR, Mosely DS, Meyer DE, Holcomb JB. A novel pain management strategy for combat casualty care. Ann Emerg Med. 2004;44:121– 127.

33. Robert R, Brack A, Blakeney P, et al. A double-blind study of the analgesic efficacy of oral transmucosal fentanyl citrate and oral morphine in pediatric patients undergoing burn dressing change and tubbing.J Burn Care Rehabil. 2003;24:351– 355.
34. Rickard C, O'Meara P, McGrail M, Garner D, McLean A, Le Lievre P. A randomized controlled trial of intranasal fentanyl vs intravenous morphine for analgesia in the prehospital setting.Am J Emerg Med. 2007;25:911– 917.
35. Borland M, Jacobs I, King B, O'Brien D. A randomized controlled trial comparing intranasal fentanyl to intra- venous morphine for managing acute pain in children in the emergency department.Ann Emerg Med. 2007;49: 335 – 340.
36. Steenblik J, Goodman M, Davis V, et al. Intranasal sufentanil for the treatment of acute pain in a winter resort clinic.Am J Emerg Med. 2012;30:1817– 1821.
37. Wolfe TR, Bernstone T. Intranasal drug delivery: an alternative to intravenous administration in selected emer- gency cases.J Emerg Nurs. 2004;30:141– 147.
38. Bourke M, Hayes A, Doyle M, McCarroll M. A compar- ison of regularly administered sustained release oral morphine with intramuscular morphine for control of postoperative pain.Anesth Analg. 2000;90:427– 427.
39. De Paepe P, Belpaire FM, Buylaert WA. Pharmacokinetic and pharmacodynamic considerations when treating patients with sepsis and septic shock.Clin Pharmacokinet. 2002;41:1135– 1151.
40. Buck ML, Wiggins BS, Sesler JM. Intraosseous drug administration in children and adults during cardiopulmonary resuscitation.Ann Pharmacother. 2007;41:1679– 1686.
41. Galinski M, Dolveck F, Borron SW, et al. A randomized, double-blind study comparing morphine with fentanyl in prehospital analgesia. Am J Emerg Med. 2005;23: 114 – 119.
42. Thomas SH, Rago O, Harrison T, Biddinger PD, Wedel SK. Fentanyl trauma analgesia use in air medical scene transports.J Emerg Med. 2005;29:179– 187.
43. Bisanzo M, Nichols K, Hammerstedt H, et al. Nurse- administered ketamine sedation in an emergency depart- ment in rural Uganda.Ann Emerg Med. 2012;59:268– 275.
44. Halstead SM, Deakyne SJ, Bajaj L, Enzenauer R, Roose- velt GE. The effect of ketamine on intraocular pressure in pediatric patients during procedural sedation.Acad Emerg Med. 2012;19:1145– 1150.
45. Grocott MPW, Johannson L. Ketamine for emergency anaesthesia at very high altitude (4243 m above sea-level). Anaesthesia. 2007;62:959– 962.
46. Gros T, Viel E, Ripart J, Delire V, Eledjam JJ, Sebbane M. Prehospital analgesia with femoral nerve block following lower extremity injury. A 107 cases survey [in French]. Ann Fr Anesth Reanim. 2012;31:846– 849.
47. Wu JJ, Lollo L, Grabinsky A. Regional anesthesia in trauma medicine.Anesthesiol Res Pract. 2011;2011:1– 7.
48. Simpson PM, McCabe B, Bendall JC, Cone DC, Mid- dleton PM. Paramedic-performed digital nerve block to facilitatefield reduction of a dislocatedfinger.Prehosp Emerg Care. 2012;16:415– 417.

Wilderness Medical Society Clinical Practice Guidelines for Diabetes Management

Karin D.Van Baak MD, Laura M.Nally MD, Ryan T. Finigan MD, Carrie L. Jurkiewicz MD, Andre M. Burnier MD, Barry P.Conrad MPH, RD, Morteza Khodaee MD, MPH, Grant S. Lipman MD

The Wilderness Medical Society convened an expert panel in 2018 to develop a set of evidence-based guidelines for the treatment of type 1 and 2 diabetes, as well as the recognition, prevention, and treatment of complications of diabetes in wilderness athletes. We present a review of the classifications, pathophysiology, and evidence-based guidelines for planning and preventive measures, as well as best practice recommendations for both routine and urgent therapeutic management of diabetes and glycemic complications. These recommendations are graded based on the quality of supporting evidence and balance between the benefits and risks or burdens for each recommendation.

Introduction

In 2015, the Centers for Disease Control & Prevention estimated that 30 million individuals in the United States (9% of the population) have diabetes.1 Athletes with diabetes are undertaking ever-expanding wilderness challenges. People self-identified as having diabetes represented 7% of 3000 surveyed ultramarathon runners,[2] and [19] individuals with type 1 diabetes have entered or completed the high altitude Leadville 161 km (100 mi) ultramarathon trail and mountain bike race.[3] To date, at least 3 individuals with diabetes have successfully summited Mount Everest.4

Hypoglycemia and hyperglycemia can be catastrophic in resource-limited environments, and glycemic control is more challenging in extreme conditions, requiring additional monitoring, treatment adjustments, and careful pre-trip planning.[4] Athletes with diabetes have been shown to have a lower maximum oxygen consumption ,[5] impaired heat loss,[6,7] higher risk of acute kidney injury,[8] and higher risk of infections,[9] among other complications.9 Athletes with diabetes may also be at increased risk of altitude-related illness.10 However, the benefits of exercise for people with diabetes are numerous and well documented, including improved HbA1c,[11,12] lower body mass index,[12] improved blood pressure levels,[13] improved lipid profiles,[13] and decreased all-cause mortality.[14]

The Wilderness Medical Society convened an expert panel to develop a set of evidence-based clinical guidelines for the recognition, prevention, and treatment of diabetes and its complications in the wilderness athlete. These clinical practice guidelines define a “wilderness athlete with diabetes” as an individual with type 1 or type 2 diabetes who participates in mild- to vigorous-intensity exercise in a wilderness environment with limited medical access, at altitudes greater than 2500 m (8250 ft), in climatic extremes, and/or with limited access to immediate medical care and supplies.

Methods

Specialists in emergency medicine, primary care, sports medicine, endocrinology, and nutrition convened in the spring of 2018. Relevant articles were identified through the PubMed database using the following keywords: diabetes, insulin, wilderness, expedition, mountaineering, and ultramarathon. The literature search was supplemented by a manual search of articles referenced by articles found in the initial PubMed search. Studies in these categories included randomized controlled trials, observational studies, retrospective studies, case series, and case studies. Abstract-only reports were not included. Conclusions from review articles and recommendations from related practice guidelines were cited to provide background information, but only primary sources were used in the formulation of recommendation grades. When no relevant studies were identified, the panel recommendation was based on perceptions of risk and benefit derived from clinical experience. The panel used a consensus approach to develop recommendations for the wilderness athlete with diabetes, with level of evidence assigned according to the methodology stipulated by the American College of Chest Physicians for grading of evidence and recommendations (see online Supplementary Table). These recommendations are graded on the basis of the quality of supporting evidence and balance between the benefits and risks or burdens for each modality or intervention.

Background

Diabetes mellitus is a metabolic disease characterized by elevated blood glucose resulting from a lack of insulin secretion, reduced insulin sensitivity, or both. Type 2 diabetes is the most common form (90% of individuals with diabetes have type 2 diabetes)[15] and is caused by a combination of reduced insulin sensitivity and impaired insulin secretion. Type 2 diabetes has classically been diagnosed in adults, although the rate of diagnosis in adolescents is increasing, and it is often accompanied by hypertension, hyperlipidemia, and obesity. Type 1 diabetes is typically diagnosed in children or young adults and is caused by autoimmune destruction of islet cells in the pancreas, resulting in insulin deficiency. People with type 1 diabetes require treatment with subcutaneous insulin, whereas those with type 2 diabetes may or may not require insulin treatment.

Strenuous exercise and wilderness environments can complicate glycemic control. In managing type 1 and type 2 diabetes, exercise and environmental stressors may also have a complex interaction with commonly used medications, glucose monitoring, and medication delivery. The effects of exercise alone on diabetes have been discussed in detail elsewhere,[16,17] so this guide will briefly review physical activity and environmental considerations in the pathophysiology of diabetes.

Glycemic Pathophysiology in Physical Activity

Physical activity in wilderness environments often involves prolonged activity and a combination of aerobic, anaerobic, and high-intensity exercise, so it is important to understand how neurohormonal responses differ with each type of exercise. Different types of physical activity cause distinct metabolic responses in individuals without diabetes. Aerobic activity involves repetitive glucose uptake by large muscle groups. To balance exercise-induced increases in glucose uptake from muscle, insulin secretion decreases. At the same time, counterregulatory hormones, including glucagon cortisol, growth hormone, and catecholamines, are released, promoting endogenous glucose production through glycogenolysis and gluconeogenesis.[17, 18, 19] In athletes with diabetes, these counterregulatory hormones may lead to hyperglycemia if adjustments in carbohydrate consumption and insulin dose are not made.

In individuals without diabetes, insulin levels in anaerobic and high-intensity exercise (>80% of) do not decrease as significantly as in aerobic activities. Rather, catecholamines play a more prominent role in glucose metabolism, although growth hormone and cortisol are important as well.[17,20] After anaerobic and high-intensity exercise in individuals without diabetes, insulin levels rise to compensate for the effects of elevations in counterregulatory hormones[18,21] (Figure 1).

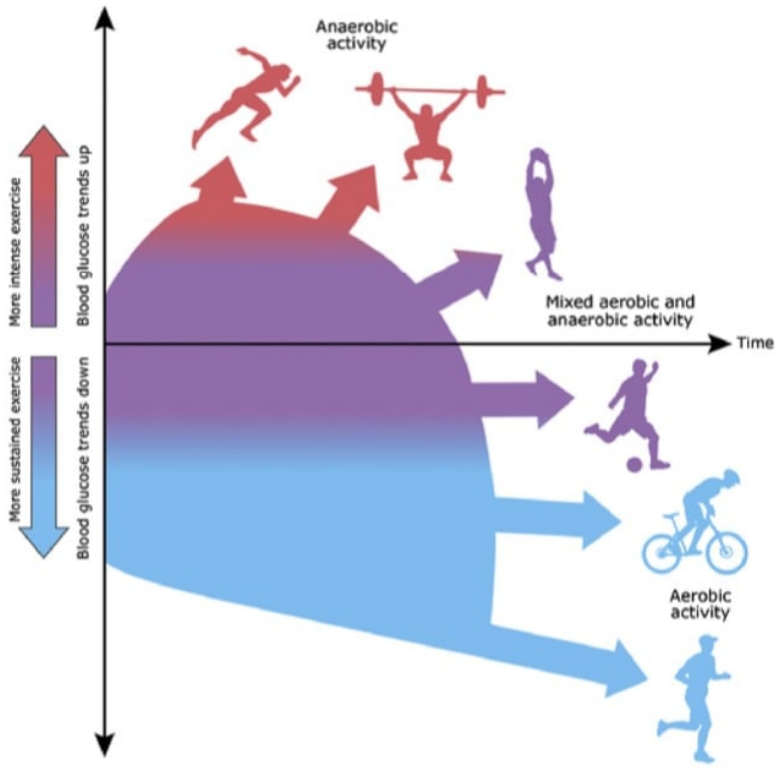


Figure 1. Illustration of different types of exercise including mutual differences in intensities and the way this affects glucose levels. Illustration by Anne Greene, Senior Medical Illustrator, reproduced with permission from UpToDate, Inc. Copyright © 2017 Duration and intensity. Reproduced with permission from: Riddell MC. Management of exercise for children and adolescents with type 1 diabetes mellitus.

After exercise, glucose uptake remains elevated by both insulin-dependent and insulin-independent processes. If exercise is prolonged, insulin-dependent mechanisms can continue up to 48 h after exercise to replenish muscle glycogen stores.16 Exercise lasting greater than 60 min has been shown to increase peripheral insulin sensitivity for up to 48 h post exercise.[22, 23, 24, 25] The basic management strategy for type 1 diabetes of decreasing basal insulin rate while increasing carbohydrate intake during exercise to match energy utilization is based on these principles.[15,26,27] It is important to understand the increased risk of hypoglycemia that occurs after exercise, as frequent hypoglycemic events in individuals with diabetes have been linked with long-term morbidity and mortality.[28] The glycemic response to exercise changes with time of day, and risk for nocturnal hypoglycemia is highest when exercise is completed in the evening.[29]

In summary, brief high-intensity exercise has been shown to cause hyperglycemia,30 whereas long-duration aerobic exercise is more likely to cause hypoglycemia31 (Figure 1). The glycemic response to exercise is complex and varies from individual to individual.[32] For example, muscle damage, such as in an abrupt increase in the level of exercise, may decrease insulin sensitivity.[33] Additionally, the effects of various environmental factors on glycemic control will

be discussed in detail. Although we attempt to provide evidence-based guidance on insulin management in athletes with diabetes, the care of each individual must be managed thoughtfully.

Pretrip Planning

Preparation is an essential component of any expedition, and even more so for the wilderness athlete with diabetes. Pretrip preparations should generally include appropriate medical screening; attention to physical fitness; planning of insulin regimen, diet, and hydration; and education and discussion on the chosen activity and how it may alter or interfere with an individual's typical diabetes care. Lastly, it is important to ensure packing of all essential diabetes-specific medical supplies and consideration of contingency and emergency planning.

PREPARTICIPATION MEDICAL EVALUATION AND COUNSELING

The American Diabetes Association does not recommend routine medical clearance for asymptomatic individuals with diabetes before low- to moderate-intensity physical activity.[16] However, given the nature of intense physical activity in resource-limited environments, it is prudent for wilderness athletes with diabetes to discuss their plans with their primary care provider and/or endocrinologist prior to travel. Attention to regular follow-up and annual screening tests including ophthalmologic examinations is recommended.

Recommendation

1. Diabetes-specific healthcare maintenance should be up to date prior to wilderness activity. Athletes with diabetes may need to undergo additional and more frequent specialty evaluations (Evidence grade: 1C).
2. Athletes with diabetes should meet with their primary care provider and/or endocrinologist prior to wilderness travel (Evidence grade: 1C).

Cardiovascular screening

Individuals with diabetes are at increased risk of ischemic heart disease,34 and individuals with type 1 diabetes have higher rates of silent myocardial ischemia and coronary artery disease than the general population.[34] Extrapolation is limited because the majority of studies on those with type 1 diabetes in wilderness settings have excluded any patient with electrocardiogram (ECG) abnormalities.[35,36] Some groups have recommended baseline ECG testing prior to wilderness activity,[37] and consensus guidelines from the Undersea and Hyperbaric Medical Society and Divers Alert Network have proposed routine preparticipation exercise ECG in adults with diabetes over the age of 40 y prior to scuba diving.[38]

However, there are differing opinions regarding the utility of ECG or exercise stress testing in asymptomatic individuals to prevent sudden cardiac death during exercise.[39, 40, 41, 42] The American College of Sports Medicine recommends a

visit with a medical provider for asymptomatic individuals with diabetes prior to vigorous activity. Neither the American Diabetes Association nor the American College of Sports Medicine recommends routine ECG or exercise stress test to screen for cardiovascular disease.41,43 The United States Preventive Services Task Force recommends assessment of 10 y cardiovascular disease risk to guide decisions on the utility of further testing for cardiovascular disease.42 The American Heart Association recognizes possible value of exercise testing in people with diabetes before starting an exercise program, despite conflicting evidence regarding its efficacy for prevention of adverse cardiovascular outcomes.[39,40] The need for additional cardiac testing should be based on individual risk factors.39,41,42 Tools such as the atherosclerotic cardiovascular disease Risk Estimator from the American College of Cardiology—which takes into account risk factors such as presence of diabetes, age, and sex—can be useful for cardiovascular risk assessment and shared decision making.[44]

Recommendations

1. Individuals with diabetes should undergo comprehensive risk assessment for cardiovascular disease with their primary care provider and/or endocrinologist prior to wilderness travel (Evidence grade: 1B).
2. Routine pre-participation ECG screening of wilderness athletes with diabetes is not recommended (Evidence grade: 2C).
3. Routine exercise ECG to screen for coronary artery disease in asymptomatic wilderness athletes with diabetes is not recommended (Evidence grade: 1B).

Diabetes-specific medical conditions

Special attention should be paid to those with diabetes and peripheral neuropathy, nephropathy, or retinopathy because these conditions pose additional risks in wilderness activities.[37,45] Those with peripheral neuropathy should be aware of the increased risk of developing cold-induced injury and should be screened for wounds before a wilderness excursion. Foot care should be reviewed prior to activity.[37] People with diabetes living at high altitude may have higher rates of albuminuria, though there is no evidence to suggest that short-term altitude exposure is nephrotoxic.[37] Individuals with a history of nephropathy should be counseled regarding appropriate medication use—including the renal risk of nonsteroidal anti-inflammatory drug use[46,47]—and appropriate hydration. Wilderness sports carry a risk of ocular injuries.48 Although corneal injuries and edema are most often reported in the wilderness athlete,[49,50] high altitude retinal hemorrhages are also known to occur,[51] but there have been no studies on high altitude's effect on diabetic retinopathy.[48,52] Regardless, any retinal damage carries additional importance in individuals with diabetes, and it is recommended that individuals with diabetes have a dilated fundoscopic examination prior to ascent to high altitude.48 To avoid further complications while in the backcountry, it is important to ensure optimal control of diabetes complications prior to high-risk wilderness activities.

Recommendations

1. Individuals with pre-existing diabetes complications (including nephropathy, peripheral neuropathy, and retinopathy) should be counseled on minimizing additional risks to these organ systems with wilderness activity (Evidence grade: 1C).
2. All individuals with diabetes planning high altitude travel should be up to date on yearly dilated fundoscopy. If any degree of retinopathy is present, ophthalmologic risks of wilderness travel should be discussed (Evidence grade: 1C).

SUPPLY PREPARATION

Appropriate supplies and medical equipment should be brought on any wilderness activity.[15,53] Table 1 lists specific supplies that should be considered essential for wilderness athletes with diabetes.

Table 1. Medical kit preparation

Category	Supply	Notes
Glucose monitoring	Glucometer with case	
	Backup glucometer supplies	Extra meter batteries and/or charging cable
	Test strips	Twice as many as anticipated for trip
	Lancets	Twice as many as anticipated for trip
	CGM and supplies	If using this glucose monitoring method
Treatment	Insulin	Twice as much as anticipated for trip
	Extra prescription for medications/supplies	
	Needles/Syringes	If not using an insulin pump
	Pump with case and needles/syringes	If using this method, should bring needles and syringes in case of pump failure
	Extra pump supplies	Batteries and/or charging cable, extra infusion sites, extra pump reservoirs
	Backup insulin pump supplies	Needles/Syringes in case of pump failure, extra pump batteries and/or charging cable, extra infusion sites, extra pump reservoirs
	Alcohol swabs	Twice as many as anticipated for trip
	Sharps container	
Emergency supplies	Glucagon emergency kit	
	Urine ketone strips	Can use as primary method of ketone monitoring, should bring as backup if using serum ketone meter
	Ketone meter with case	Extra meter batteries and/or charging cable
	Prepackaged snacks	
Other	Letter from physician stating need to carry supplies	
	Insulin management and emergency plan	Developed in conjunction with endocrinologist

Category	Supply	Notes
	Medical identification card/bracelet with emergency contact information	
	Health insurance card	
	CGM	continuous glucose monitor.

Recommendation

1. Wilderness athletes should be counseled on a complete packing list of routine and emergency diabetes supplies (Evidence grade: 1C).
2. Wilderness athletes should carry documentation of their medical history, basic diabetes management plan, and basic emergency action plan (Evidence grade: 1C).

GLUCOMETERS

Glucometers can fail in the wilderness owing to extreme environmental conditions, water exposure, or physical damage from high-impact activity. Hypobaric hypoxia at high altitude may alter the reaction used to estimate blood glucose in glucometers that use a glucose oxidase reaction (GOX) or a glucose dehydrogenase reaction (GDH).54,55 GDH and GOX systems have been tested in hypobaric chambers up to 4500 m (14,850 ft), with some tending to overestimate blood glucose (0.8 to 15%)56 and some tending to underestimate blood glucose.57 At 2000 m (6560 ft), GDH systems overestimated blood glucose by 3.5 to 8%, whereas GOX systems showed no significant difference from reference values.54 Although high altitude studies have found statistically significant inaccuracies in blood glucometers, the differences were small enough to be of questionable clinical significance. When tested at high (25°C) and low (8°C) temperatures, GOX and GDH systems performed similarly, with tendencies toward both overestimation and underestimation in some systems, with errors of 5 to 10% or less.56,58

Similar to high altitude, temperature appears to affect glucometer function in a clinically insignificant way, although the errors appear to be brand specific and are unpredictable. There are no studies to define the temperature at which a glucometer will fail despite warming, but caution should be taken and the manual for a device reviewed. In cold environments, glucometers should be kept inside a jacket pocket close to the body during the day and in a sleeping bag at night. Each product guide should be reviewed to determine the altitude level for which a glucometer is approved. Athletes should have at least 1 backup glucometer in case of glucometer failure or loss.

Recommendations

1. For glucometers and other monitoring equipment, the product guide should be reviewed carefully before an expedition. Individuals should carry a backup monitor and battery supply (Evidence grade: 1C).

INSULIN Insulin requires safe use and storage in wilderness environments. Each insulin package insert should be read carefully to ensure proper transportation and storage, and insulin should be discarded after an appropriate amount of time.[59,60]

Insulin is a protein that is denatured by freezing or extreme heat and thus should not be used if exposed to extreme temperatures. Insulin should not be exposed to extreme temperatures (<2 or="">30°C). Likewise, light exposure can potentially reduce insulin's efficacy. Excess agitation should be avoided because this can lead to clumping and decreased potency.60 In extreme ambient temperatures, measures should be taken to keep insulin at a stable cool temperature.[59] This may be accomplished by storing insulin in a well-insulated container (cooler or thermos) with a hard case to prevent vials/pens from breaking. Likewise, using a gel or evaporative cooling pack in warm temperatures can help maintain potency. In hot environments, cooling systems may be used, and the insulin should be stored in the middle of the pack to minimize heat exposure. There are commercially available insulin storage products which use evaporation of cold water to keep vials and pens cold. In cold temperatures, insulin should not only be kept in an insulated container, but should also be carried close to the body during the day and kept inside a sleeping bag at night to prevent freezing. A backup supply of insulin should be kept in an insulated container in the middle of the backpack to prevent exposure to extreme temperatures. It is prudent for any wilderness athlete to have a backup supply of insulin on any expedition. The type of excursion will dictate the safest way to store insulin.

Recommendation

1. In the wilderness, insulin should be protected from environmental extremes, such as high or low temperatures, light exposure, and physical agitation. Any method of physical and/or temperature protection should be tested in a low-risk environment prior to use in the wilderness. A contingency supply of insulin should be kept in a separate location (Evidence grade: 1B).

Environmental Considerations

One of the most challenging aspects of diabetes care in the wilderness athlete is to predict and respond to the effects of extreme environmental conditions. The basic effect of different environmental conditions on diabetes is summarized in Table 2.

Table 2. Environmental effects on diabetes

Environmental factor	Effect on blood glucose	Other effects and risks for wilderness athletes with diabetes
Extreme altitude (>4000 m)	Increases	Capillary blood glucose testing overestimation
Moderate altitude (>1500 – <4000 m)	Unknown	
Extreme heat	No direct effect	Increased heat illness, insulin becomes nonviable, increased insulin absorption
Extreme cold	Variable	Possible poor insulin absorption, increased cold injuries, insulin freezing

HIGH ALTITUDE

Change in insulin requirements

High altitude, especially extreme altitude (>5000 m [16,400 ft]), adds greater complexity and difficulty to glucose control in type 1 diabetes. Studies of exercise at sea level showed improved glycemia in individuals with type 1 diabetes who were exposed to a hypoxic environment when compared to a normoxic environment.61 Although there is conflicting evidence on glycemic control at altitudes between 1500 m (4,920 ft) and 4000 m (13,120 ft),[36] several studies have observed increased insulin requirements above 4000 m (13,120 ft) in those with type 1 diabetes.[35,36] Relative hyperglycemia [62]and insulin resistance at extremely high altitudes in people without diabetes have been reported as well.[36,63,64] It is unclear whether this observed insulin resistance is a result of acute mountain sickness or exposure to hypobaric hypoxia. Ketosis is an additional risk at high altitude; it is related to suboptimal carbohydrate intake, leading to less insulin administration and higher levels of counterregulatory hormones.[35]

Recommendation

1. Those with insulin-dependent diabetes traveling to high altitude should be counseled on the potential for increased insulin requirements. Athletes should consider close monitoring on shorter trips to learn about their own glycemic trends prior to a major high altitude expedition (Evidence grade: 2C).

High altitude illness

The spectrum of high altitude illnesses includes acute mountain sickness (AMS), high altitude pulmonary edema (HAPE), high altitude cerebral edema (HACE), and high altitude retinal hemorrhage. AMS is defined as a constellation of symptoms including headache, gastrointestinal disturbances, weakness, and dizziness in an individual who has ascended to >2500 m (8,200 ft) and has no

other obvious explanation for these symptoms.[65] Increased risk of high altitude illness has not been reported in those with diabetes when compared to individuals without diabetes.35,36,66 Although type 1 diabetes itself may not increase the risk of AMS, metabolic decompensation may occur in persons with diabetes who develop AMS, and there have been a few case reports of diabetic ketoacidosis (DKA) in the setting of AMS.[67] The symptoms of AMS may confound or mimic symptoms of hypoglycemia or hyperglycemia and make appropriate diagnosis and treatment of type 1 diabetes and ketoacidosis more difficult.[37,52] Therefore, if AMS occurs in an individual with diabetes, descent is often the safest option. Although there is limited data on the incidence of HAPE and HACE in those with type 1 diabetes, various case studies have showed no increased incidence for these conditions.[35,36,52,68]

Recommendations

1. Individuals with diabetes should be counseled on symptoms and management options for high altitude illness and dysglycemia. More frequent blood glucose and ketone checks are recommended if symptoms of high altitude illness occur (Evidence grade: 1C).

Acetazolamide

Commonly used in the prophylaxis and treatment of AMS,[69,70] acetazolamide is a carbonic anhydrase inhibitor that limits bicarbonate reabsorption in the renal tubules, producing a bicarbonate diuresis and metabolic acidosis.[71] Theoretically, this could trigger or worsen acidosis and dehydration and contribute to ketoacidosis.[35,68] Although data on the incidence of DKA are inconsistent, there are reports of increased insulin requirements in individuals with type 1 diabetes on acetazolamide, with insulin requirements abruptly returning to normal once acetazolamide was discontinued.[67] Also important to consider, paresthesia is a common side effect of acetazolamide and may be confused with hypoglycemic symptoms.[35,52]

Recommendation

1. Acetazolamide should be used with caution in individuals with diabetes (Evidence grade: 2C).

Dexamethasone

Dexamethasone is an oral corticosteroid commonly used in the treatment of AMS, HACE, and HAPE and occasionally is used for prophylaxis of these conditions.[69] Dexamethasone is known to cause hyperglycemia and increased insulin requirements.[72,73] If considering use of dexamethasone, the risk and severity of high altitude illness needs to be balanced with the risk of and ability to manage resultant hyperglycemia. In particular, it should be noted that in the case of acutely life-threatening conditions such as HACE, the benefits of corticosteroids outweigh the risks of hyperglycemia.

Recommendation

1. In wilderness athletes with diabetes, oral corticosteroids should be used with caution in light of the risk of hyperglycemia (Evidence grade: 1C).

COLD ENVIRONMENTS

Extremely cold air temperatures are common in high altitude and other wilderness pursuits and may complicate insulin use in wilderness athletes with diabetes. In individuals without diabetes, metabolic rate increases in response to cold exposure, although no change has been observed in plasma glucose or insulin response to an oral glucose tolerance test.[74] Cold acclimation for 10 d has been shown to improve insulin sensitivity in type 2 diabetes.[75] However, reduced skin temperatures have been shown to cause decreased insulin absorption within the first 90 min after insulin injection in both type 1 and type 2 diabetes.[76] Clinical data are insufficient to accurately predict how cold environments will affect insulin requirements in the field. As discussed, careful consideration needs to be taken with medication storage in cold environments.

Recommendation

1. There are insufficient data to describe the effect that cold exposure has on diabetes management (no recommendation).

Cold illness

Individuals with preexisting neuropathy or peripheral vascular disease may be at increased risk for cold-related skin and soft tissue injuries,[37,45] and these individuals are at higher risk of frostbite.[77]

Recommendation

Wilderness athletes with diabetic peripheral neuropathy and peripheral vascular disease are at increased risk of frostbite (Evidence grade: 2C).

HOT ENVIRONMENTS

Extremely hot environments present unique challenges to wilderness athletes with diabetes. In subjects with and without diabetes, higher plasma blood glucose has been observed in response to an oral glucose load during exposure to a hot environment as compared to a neutral or cold environment.[78, 79, 80] Extreme heat exposure can lead to increased insulin absorption.81 In the setting of elevated surface temperatures, serum insulin levels were observed to be approximately double for the first 90 min after subcutaneous injection.[76] Clinical data are insufficient to accurately predict how hot environments will affect insulin requirements in the field. Insulin itself may denature if exposed to high temperatures (>30°C). As previously discussed, careful consideration needs to be taken with medication storage in hot environments. For expeditions that require repeated bouts of exercise, it is also important to recognize that glycogen resynthesis is impaired in hot environments.[82]

Recommendation

1. There are insufficient data to describe the effect that heat exposure has on diabetes management (no recommendation).

Heat illness

During exercise, cutaneous vascular flow and sweating increase to release excess endogenous heat and maintain core body temperature. Individuals with type 1 diabetes may have impaired sweating during high-intensity exercise.[7,83,84] Both type 1 and 2 diabetes may result in impaired exertional heat loss in hot environments.[6,7,84] It is theorized that those with diabetes have a blunted sensitivity to elevated ambient temperatures and an inadequate cutaneous vascular perfusion or sweat production response. Age, long-term hyperglycemia, and neuropathy further impair blood flow to the skin, putting individuals with diabetes at higher risk of heat illness.[85,86] The ADA recommends that older adults with diabetes avoid exercising outdoors on very hot and/or humid days.[16] For relatively young adults, close blood glucose monitoring for hyperglycemia is important during heat exposure to avoid exacerbating potential existing impairments in the body's cooling mechanisms.

Recommendation

1. Wilderness athletes with diabetes are at increased risk for heat illness (Evidence grade: 1C).

MONITORING AND TREATMENT

Glucose monitoring

Prior recommendations on athletic participation for individuals with diabetes have provided reasonable suggestions for blood glucose monitoring and involvement.[16,53] Ideally, pre-exercise blood glucose monitoring should be performed 2 to 3 times at 30 min intervals to determine a blood glucose trend; during exercise, every 30 min; and after exercise, every 2 to 4 h owing to potential delayed hypoglycemia.[53] It has been suggested that the ideal blood glucose level for exercise is between 130 and 180 $mg \cdot dL^{-1}$,[16,26,53] though it is preferable to have individualized targets based on an athlete's personal history. Because such time-intensive glucose monitoring is difficult in the wilderness, it is prudent for an individual to establish trends in insulin and carbohydrate requirements during exercise and in similar environmental conditions prior to the expedition. Patients with well-established exercise insulin requirements may check blood glucose prior to exercise and every few hours during prolonged periods of exertion. Continuous glucose monitors (CGM), which will be discussed separately, may be a useful adjunctive tool for close monitoring of blood glucose during wilderness activities, though robust clinical data are lacking.

Recommendation

1. In insulin-dependent diabetes, blood glucose should be monitored before, during, and after intense and/or prolonged exercise (Evidence grade: 1B).

Carbohydrate intake

The goal of euglycemia for health and performance in individuals with diabetes requires managing both carbohydrate intake and insulin treatment. Increased carbohydrate intake and/or reduction in insulin dose is typically required to reach the goal of euglycemia during exercise. Past studies suggest 10 to 15 g of carbohydrate is needed to prevent hypoglycemia for low- to moderate-intensity aerobic activity lasting 30 to 60 min.[87] A similar amount of activity following an insulin bolus may require 30 to 60 g of carbohydrate to maintain euglycemia.[88] A composite of specific guidelines for carbohydrate intake from several organizations are shown in Table 3.[16,53,87] Specifically regarding travel in the wilderness, frequent snacks are recommended to maintain energy needs for prolonged physical activity, and we generally recommend increased carbohydrate intake to help mitigate the risk of altitude illness.

Table 3. Blood glucose monitoring and treatment strategy around exercise

Pre-exercise blood glucose level (mg·dL^{-1})	Carbohydrate intake recommendation
<90	For typical activities, consume 10–30 g of fast-acting CHO prior to start of exercise
	For brief or very high-intensity activities, consider no additional CHO intake
	For prolonged activities consume an additional 0.5-1.0g per kg body mass per h of exercise based on BG monitoring
	Or, delay exercise until blood glucose is >90 mg·dL^{-1} and monitor closely for hypoglycemia
90–124	Consume 10 g of glucose before starting aerobic exercise
	Can start anaerobic or high-intensity interval exercise right away
125–150 (alternate 125–180)	Start consuming 0.5-1.0 g per kg body mass per h of exercise at onset of exercise
	May need to adjust based on type of exercise and insulin
150–250 (alternate 180–270)	Delay carbohydrate intake until BG levels are below 150 mg·dL^{-1}
	Consume non-CHO-containing fluid to prevent dehydration
250–350	Test for ketones
	Delay intense exercise until BG levels are below 250 mg·dL^{-1}
>350 with no ketones	Consider conservative insulin correction (eg, 50%) before exercise
	Initiate mild- to moderate-intensity exercise
	Delay intense exercise until BG levels are below 250 mg·dL^{-1}
>250+ ketosis	Avoid exercise if moderate to large amounts of ketones are present

BG, blood glucose; CHO, carbohydrate.

Guidelines from different groups have recommended different ranges for blood glucose monitoring in exercise.[16,53,87]

Recommendation

1. Those planning protocols for glucose monitoring and carbohydrate intake in exercise should understand how to adjust carbohydrate intake based on blood glucose and exercise. This plan should be individualized based on patients' medical and exercise history and the environmental stressors to which they are exposed (Evidence grade:1B).

Hydration

Adequate hydration is important before, during, and after exercise. Hyperglycemia may predispose to dehydration and electrolyte (potassium, magnesium, phosphorous) loss through osmotic diuresis. In general, wilderness athletes are able to participate safely if they drink “according to thirst,”[89,90] but people with diabetes should hydrate more frequently, especially in the setting of hyperglycemia, ketosis, and illness related to environmental stressors (eg, altitude illness, heat illness).[15,91] Specific medications that may affect hydration should be discussed with a medical provider prior to travel. There are no evidence-based protocols for managing hydration in individuals with diabetes during exercise. Similar to insulin and carbohydrate management, it is prudent to base the hydration strategy of an individual with diabetes on experience in similar environmental conditions prior to an expedition.

Recommendation

1. Individual hydration strategies should be developed prior to embarking on wilderness activities and should be adjusted based on real-time factors, including environmental temperature, altitude, and exercise type and duration (Evidence grade: 1C).

Insulin management

It has been suggested that individuals using multiple daily injections (MDI) of insulin should have their basal insulin rate reduced by 20% for doses before and after exercise. With continuous subcutaneous insulin infusion (CSII), athletes can reduce or suspend basal insulin infusion at the start of exercise, or even 30 to 60 min before exercise, to prevent hypoglycemia. Reduction in insulin boluses by 25 to 75% 2 to 3 h prior to activity may limit hypoglycemia.[16,91] Although guidelines are helpful, it is important to consider the variables of an individual and trip; not everyone will need to decrease their insulin during wilderness pursuits. Owing to factors related to environmental extremes and prolonged physical activity, insulin needs may increase.

Multiple attempts have been made to develop uniform guidelines for glucose monitoring, carbohydrate intake, and insulin dosing during exercise. Generalized management recommendations can be unreliable owing to variable individual glycemic responses to physical activity as well as environmental conditions. Individuals with type 1 diabetes should discuss individual management strategies with their endocrinologist prior to wilderness activities, and an individual management plan should be based on one’s previous glycemic responses to similar exercise and/or environmental conditions. Individuals using an insulin pump should carry a backup method of insulin delivery. Additionally, it is important for wilderness athletes with diabetes to be at an appropriate level of physical fitness and to develop an action plan regarding potential complications prior to an expedition.

Recommendation

1. Wilderness athletes with type 1 diabetes should understand how to adjust insulin doses via either MDI or CSII. This should be individualized based on their medical and exercise history and the environment to which they are exposed. This should be discussed in detail with their primary care provider and/or endocrinologist prior to embarking on wilderness activities. Any device should be explained thoroughly prior to an expedition (Evidence grade: 1B).

Noninsulin medications

Although the majority of this clinical practice guideline discusses insulin-treated type 1 and type 2 diabetes, special consideration should be given to common noninsulin medications used in the treatment of diabetes and their unique side effects and potential complications (Table 4).

Table 4. Noninsulin diabetes medications

Medication	Important side effects	Incidence
Metformin	Diarrhea	10–30%
	Nausea	10–15%
	Lactic acidosis	2–5/100,000
Sulfonylureas	Hypoglycemia	Frequency varies by degree of hypoglycemia
Glinides	Hypoglycemia	Frequency varies by degree of hypoglycemia
Acarbose	Abdominal pain	19%
	Diarrhea	31%
DPP4 inhibitors	Nausea	10–24%
	Diarrhea	10–18%
SGLT2 inhibitors	Diuresis (hypotension, dehydration)	1%
	Increased risk of UTIs	<1% (19 cases of urosepsis reported over 1 year)
Euglycemic diabetic ketoacidosis	<1% (73 cases reported over 2 years)	
GLP-1 receptor agonists Nausea/Vomiting 10–30%		

Diarrhea 10–15% AP, artificial pancreas; BG, blood glucose; CGM, continuous glucose monitoring; DPP4, dipeptidyl peptidase-4; GLP-1, glucagon-like peptide 1; HCL, hybrid closed loop; MDI, multiple daily injection; SGLT2, sodium-glucose transport protein 2; UTI, urinary tract infection.

Metformin is a commonly used medication, prescribed to 84% of people with type 2 diabetes in the United Kingdom.92 Metformin is a biguanide class antihyperglycemic that improves glycemia by reducing hepatic gluconeogenesis and increasing peripheral insulin-mediated glucose utilization. Metformin may cause diarrhea, and 25% experience some form of digestive tract disturbance upon starting the medication. Metformin-induced diarrhea may predispose to dehydration and hypokalemia.[93] There is a known risk of metformin-induced lactic acidosis, found at a rate of 2 to 5 per 100,000,[94] with elevated serum creatinine being the greatest predisposing risk factor. Although concurrent use of metformin and acetazolamide (a diuretic carbonic anhydrase inhibitor commonly used for AMS prophylaxis) has not been directly assessed, there is concern that concurrent use of a diuretic with metformin could precipitate lactic acidosis.[94,95] Exercise alone is known to be safe while using metformin alone,[16] and no dose adjustments are recommended.

Both sulfonylurea and glinide class medications carry a risk of hypoglycemia, and blood glucose should be monitored following initiation or changes in these medications. Acarbose and DPP4 inhibitor medications including sitagliptin and saxagliptin can cause vomiting and diarrhea.96,97 Hypoglycemia appears to be a minimal risk with the DPP4 medications.[97] SGLT2 inhibitors block glucose reuptake in renal tubules and so have diuretic-like effects that could theoretically worsen dehydration, though there is no supporting clinical evidence.[98] SGLT2 inhibitors pose a known increased risk of urinary tract infections and euglycemic DKA, carrying a specific Food and Drug Administration warning.98,99 Glucagon-like peptide-1 receptor agonists are a relatively new class of injectable antihyperglycemic medication, with nausea, vomiting, and diarrhea being the most frequently reported gastrointestinal side effects.[100]

Noninsulin diabetes medications should ideally be stored in a dark environment at room temperature, although compared with insulin, they are not as sensitive to environmental perturbations.

Recommendation

Use of noninsulin diabetes medications should not be considered a contraindication to wilderness athletic involvement, though participants should be cautious regarding side effects. Particular attention should be paid to the individual risks of each specific class of medication (Evidence grade: 1C).

GLYCEMIC COMPLICATIONS

In addition to proper pre-trip planning and understanding the basics of carbohydrate intake and insulin management, several specific complications and medical issues deserve unique consideration for the wilderness athlete with diabetes.

Hypoglycemia

Hypoglycemia is defined as a plasma glucose level less than 70 mg· dL^{-1}, is a well-recognized hindrance to athletic performance, and most importantly poses an acute health risk in diabetes. Typical symptoms of hypoglycemia include drowsiness, confusion, dizziness, nausea, palpitations, tremor, sweating, and anxiety. As a result of hormonal counterregulatory mechanisms, hypoglycemia is also accompanied by hypokalemia, which can last for several hours after blood glucose levels have returned to normal101 and can cause impaired skeletal muscle contraction, weakness, and potential cardiac arrhythmias.[91]

The primary treatment option for hypoglycemia is glucose repletion. Although common options typically are oral, rectal, and intravenous (IV) repletion, IV repletion should not be relied on as an option in the wilderness setting. Therefore, it is important to assess the severity of a hypoglycemic episode to decide on the safety and utility of oral treatment or whether evacuation is necessary. The basic principles of hypoglycemia management in the wilderness are cessation of insulin treatment and glucose repletion. Glucose repletion can be accomplished either directly or by administration of glucagon, which stimulates gluconeogenesis and glycogenolysis[102] (Table 5).[103,104] High glycemic index carbohydrates are best for oral glucose repletion, and16 in the wilderness, low-weight options such as sugar packets, sugar cubes, glucose gel, honey, corn syrup, and glucose tablets can be considered. For severe hypoglycemia—defined as severe cognitive impairment requiring external assistance for recovery—glucagon is the first-line treatment. Any glucagon treatment should be followed by carbohydrate intake to prevent rebound hypoglycemia, especially in the setting of depleted glycogen stores.[105,106] Similar to insulin, glucagon should be stored with insulation to avoid extremes of heat and cold. Individuals accompanying those with diabetes should know where glucagon is stored and how and when to administer it. It is important to consider the risk of ketosis if insulin therapy is suspended for more than 2 to 3 h.[107]

Table 5. Treatment of hypoglycemia in the backcountry

Medication Dose Route Timing Side effects Special considerations Glucose 15–20 g PO, PR, IV Repeat dose q15 min PRN for persistent hypoglycemia Hyperglycemia Patient must immediately consume meal or carbohydrate snack after resolution of hypoglycemia. Glucagon 1 mg IM, “mini-dose” subcutaneous, intranasal Effective in 10–15 min Nausea, vomiting Only if able to take oral glucose. Effectiveness limited in glycogen-depleted patients. IM, intramuscular; IV, intravenous; PO, oral administration; PR, per rectum; PRN, as needed.

Individuals with type 1 diabetes are at risk for delayed nocturnal hypoglycemia following daytime exercise. There are multiple reasons for this phenomenon, including a rise in insulin sensitivity after exercise, increased glucose uptake by skeletal muscles to replenish glucose stores, and impaired counterregulation in response to hypoglycemia.22 For those with MDI, the risk of nocturnal hypoglycemia may be minimized through approximately 20% reduction of daily basal insulin dose, reduced prandial bolus insulin, and low glycemic index carbohydrate feeding after evening exercise. For those using CSII, basal rate reductions of approximately 20% at bedtime for 6 h after afternoon exercise may

limit nocturnal hypoglycemia. Other strategies include a bedtime snack, glucose checks overnight, and/or use of CGM with alarms and automatic pump suspension.91

Recommendation Wilderness athletes with diabetes should have a plan and carry supplies for treating hypoglycemia. They should be prepared to use a glucose repletion and glucagon strategy (Evidence grade: 1C).

Wilderness athletes with diabetes should have experience with individualized methods for managing nocturnal hypoglycemia prior to wilderness activity (Evidence grade: 1C).

Hyperglycemia If someone with diabetes is found to be hyperglycemic (plasma glucose level >250 mg· dL-1), it is important to determine whether the individual is in an acute hyperglycemic crisis, including hyperosmolar hyperglycemic state (HHS) or DKA. To help differentiate between hyperglycemia and HHS/DKA, individuals with type 1 diabetes should be able to test for blood or urine ketones if they have unexplained hyperglycemia.

Hyperosmolar hyperglycemic state HHS is a state of progressive hyperglycemia and hyperosmolarity typically seen in individuals with poorly controlled or undiagnosed type 2 diabetes, limited access to water, and a precipitating medical event. The development of HHS is attributed to insulin resistance, deficiency, or both, in addition to increased hepatic gluconeogenesis, osmotic diuresis, and dehydration. The hyperglycemia in HHS is profound, with serum glucose level usually >600 mg· dL-1 and extreme dehydration with a fluid deficit of 8 to 12 L.108

Diabetic ketoacidosis DKA occurs predominantly in type 1 diabetes and results from absolute insulin deficiency. Alternate fuel stores are broken down, leading to hyperglycemia, ketosis, dehydration, and acidosis. Without insulin to correct the acidosis, DKA ensues. DKA is defined by a blood glucose level >250 mg· dL-1, an anion gap >10, pH <7.3, and="" ketonemia.108</sup>

Both ketosis and DKA have been reported in the wilderness setting.35,109 DKA and HHS may be precipitated by infections, heat illness, dehydration, cardiac ischemia, major trauma, and medications including steroids and diuretics. Thus, if one of these conditions is diagnosed, it is important to consider the underlying trigger.108 Both DKA and HHS are potentially life-threatening situations, and hospital-based treatment includes IV fluids and insulin, hourly blood tests, electrolyte replacement, close cardiovascular and neurological monitoring, and slow correction of acidosis and dehydration. In the wilderness environment, those interventions are unavailable, and so focus should be on close monitoring and prevention of worsening ketosis. Our panel has experience managing ketosis in the backcountry with oral hydration and subcutaneous insulin. However, given the high mortality potential of both conditions, one should have a low threshold for evacuation if there is clinical suspicion for true HHS or DKA. In the wilderness setting, it is important to prevent potentially severe or life-threatening conditions with careful planning.

Ketone monitoring and management Symptoms of ketosis, including nausea, vomiting, fatigue, tachycardia, and lethargy, can mimic dehydration, gastroenteritis, heat illness, exercise-associated hyponatremia, or altitude illness.

Signs and symptoms of ketosis should be reviewed carefully prior to wilderness activities. Individuals with diabetes in the wilderness should have some method for monitoring ketones. Monitoring options for ketones include urine ketone strips and serum ketone monitoring using a fingerstick monitor similar to a blood glucometer. Serum ketone measurement may be preferred over urine ketone testing84 because this method provides real-time levels of beta-hydroxybutyrate in the blood. Urine ketones measure bladder ketone levels, which may reflect ketone levels that were previously higher.108 Both modalities are small, light, and reasonable to carry in a medical kit. Expiration dates for ketone strips should be checked carefully prior to travel because strips may be inaccurate after their use-by date.107 Temperature guidelines for ketone monitors are similar to glucometers, and it is important to verify the temperature and altitude limitation for each type of ketone monitor (ie, some are approved from –25 to 55°C, and up to 7100 m). Ketone monitoring strategies should be discussed with anyone with type 1 or type 2 diabetes with insulin deficiency, type 2 diabetes requiring insulin injections, or someone who has known or suspected ketosis-prone type 2 diabetes.[110]

Recommendation Those with insulin-dependent diabetes should know the signs and symptoms of ketosis, carry a serum and/or urine ketone testing kit, and know how to treat ketones during wilderness activities. It may be prudent to carry both as a contingency in the event of failure due to environmental conditions (Evidence grade: 1B).

DKA and HHS are medical emergencies, but in the wilderness setting, it may be possible to prevent progression to advanced illness with early recognition of ketosis and treatment with subcutaneous insulin and oral hydration/glucose repletion. Symptoms of ketosis, particularly vomiting or a change in mental status,110 or blood glucose level elevated above 250 mg· dL-1 should prompt ketone monitoring. In general, any ketosis is a sign of insulin deficiency, and in the setting of hyperglycemia, moderate or large amounts of ketones on serum or urine monitoring reflects actual or impending DKA. The general strategy for management of ketosis involves increased insulin availability along with attention to maintaining carbohydrate availability, generous oral hydration, and modification or cessation of physical activity.87 Figure 2 outlines a proposed guideline for treatment of ketosis in the backcountry, with the goal of early recognition and prevention of progression to ketoacidosis and subsequent need for evacuation. Prior to a wilderness expedition, the wilderness athlete should discuss a plan for insulin adjustment, treatment of ketosis, and criteria for evacuation with their endocrinologist.

Figure 2 Download : Download high-res image (1MB)Download : Download full-size image Figure 2. Algorithm for management of hyperglycemia and ketosis in the backcountry. EDD, estimated daily dose; PO, oral intake.

Recommendation Ketosis may be safely managed in the wilderness if an athlete with diabetes and the athlete’s healthcare provider are comfortable with a treatment protocol and if the patient is able to take oral hydration and nutrition and shows no signs of altered mental status (Evidence grade: 2C).

Both HHS and DKA should be considered medical emergencies managed by emergent removal or evacuation to definitive care (Evidence grade: 1A).

Healthcare providers covering events or expeditions in the wilderness should have the ability to monitor blood glucose and ketones and have a basic familiarity with how to treat and triage glucose abnormalities (Evidence grade: 1C).

There should be a plan for evacuation in the case of a hyperglycemic emergency (Evidence grade: 1A).

Exercise-induced hyperglycemia Exercise-induced hyperglycemia is common in type 1 and type 2 diabetes. In wilderness athletes specifically, there seems to be increased insulin requirements at some environmental extremes.[35,36] Decreased insulin doses before exercise can promote a rise in blood glucose, as can malfunctioning insulin pump infusion sets. Overconsumption of carbohydrates before or during exercise, along with aggressive insulin reduction, can result in hyperglycemia during any exercise. The risk of hyperglycemia during exercise may be mitigated if intense activities are interspersed between moderate-intensity aerobic activities. Similarly, combining resistance training (first) with aerobic training (second) optimizes glucose stability in those with type 1 diabetes. Options to correct postexercise hyperglycemia include a conservative insulin correction (50% of usual) and/or an aerobic cool down.16 It is important to remember that excessive insulin corrections after exercise may increase the risk of nocturnal hypoglycemia. As previously discussed, attention should also be paid to pre-trip level of fitness to mitigate glycemic complications from exercise.

Recommendation Those with insulin-dependent diabetes should understand how to adjust insulin doses when hyperglycemia occurs during activity. This should be based on their individual experiences during exercise, training, and previous exposures to environmental stressors. This should be discussed in detail with their endocrine provider prior to embarking on a wilderness adventure (Evidence grade: 1B).

EMERGING TECHNOLOGY The management of diabetes is on the brink of large-scale change as new technologies become available. CGM and hybrid closed loop insulin delivery systems have ever-growing evidence favoring their use and the potential to alter the standard of diabetes care. The end goal of these developments is a completely autonomous technological system that functions as normal human pancreatic endocrine cells. These technologies offer the possibility of sophisticated glycemic monitoring and treatment in the wilderness, decreased risk of hypoglycemia, and maintenance of optimal glycemia for performance. Technological innovations may allow access to wilderness pursuits (eg, big wall rock climbing, scuba diving) that have historically been off limits to those with diabetes given excessive risks or difficulty in glycemic monitoring.[111] Important considerations for any electronic device include maintaining functionality in extreme environments, limited access to power sources, cleaning, and servicing in case of malfunction.

Multiple studies in individuals with type 1 diabetes comparing either CSII or MDI have validated positive clinical results of CGM, including reduction in HbA1c, reduction in time spent in hypoglycemia, and improvement in quality of life.[112,

[113, 114, 115] CGM use reduces glycemic anxiety given real-time tracking and hypoglycemia alarm systems, an effect that could be beneficial for the wilderness athlete.[116]

The US Federal Drug Administration approved a hybrid closed loop insulin delivery system (also known as the artificial pancreas) in October 2016. In this system, a CGM is paired with an insulin pump, and insulin delivery rate is adjusted based on CGM data, directed by an onboard algorithm independent of patient input. Preliminary data shows promise for safety and improved glycemic control.[114,117, 118, 119] However, operator input is still required for meal bolus insulin because it remains difficult to autonomously alert an artificial system of an impending meal.

For various reasons, exercise poses a challenge to any automated insulin delivery system.[120] To date, only 1 study has evaluated such a system in the wilderness athlete. Those randomized to a single-hormone artificial pancreas versus sensor augmented pump therapy had more time in ideal blood glucose control, reduced time in hypoglycemia, and higher satisfaction with therapy.[121] There was no documented device failure for either system in the study's alpine skiing environment.

A future target of research and development is the addition of other hormones to the artificial pancreas system. Glucagon has been studied most, as diabetes results in impaired secretion.[122] One study found improved glycemic control with less time in hypoglycemia with a dual-hormone artificial pancreas, as compared to sensor augmented therapy.[123] The future expectation may be that all individuals with diabetes will use CGM, and all insulin-dependent people will be encouraged to use a hybrid closed loop insulin delivery system, which would be a great benefit for the wilderness athlete undergoing high energy expenditure in wilderness environments. Still, resource-limited scenarios with extreme environmental conditions will limit complete technologic dependence on technology, and a backup analog management plan will likely always be necessary (Table 6).

Table 6. Emerging technology

Treatment system Description Pros Cons 4 hormone AP Insulin, glucagon, amylin, C-peptide infusion
• Complete replacement of hormones lost in type 1 diabetes mellitus

• Theoretical reduction in micro/macrovascular complications

• Only conceptual

• No data to support efficacy of quad hormone replacement

• Not clinically available

Dual hormone AP Pump automatically adjusts basal insulin based on CGM data, user input for meals. Glucagon infusions automatically given for hypoglycemia.
• Improved control reduced hypoglycemia

• More medication to store/carry/manage

• More complicated software and technology

• Not clinically available

Single hormone AP Pump automatically adjusts basal rate based on CGM data, user input for meals
• Improved control

• Reduced hypoglycemia

• Requires technological literacy

• Frequent calibration

CGM + insulin pump (“sensor augmented pump”) Pump and CGM do not communicate. Basal rate and meal bolus manually adjusted (some have low glucose suspend feature).
• If one system fails the other is still functional

• More input required

• Less control compared to AP

CGM + MDI CGM with bolus and basal insulin given as MDI
• One less device

• Multiple medications needed

• Less control than above

Insulin pump + capillary BG Pump with capillary BG monitoring only
• No calibration required

• Greater risk of hypoglycemia

MDI + capillary BG MDI for bolus and basal insulin with capillary BG monitoring only
• No device contained on body

• Lowest level of control and monitoring

AP, artificial pancreas; BG, blood glucose; CGM, continuous glucose monitoring; MDI, multiple daily injection.

Recommendations

1. Although there is insufficient in vivo data on continuous glucose monitoring or novel hybrid closed loop insulin delivery systems to recommend their use for wilderness athletes with diabetes, the use of such technology may be considered after discussion with an individual’s endocrine provider (Evidence grade: 1C).

Conclusions

As the number of athletes in wilderness and remote environments increase, the number of wilderness athletes with diabetes will also increase. There is strong evidence that exercise and outdoor activities have health benefits for individuals with diabetes. Individuals with diabetes and the healthcare providers who care for

them should be fully equipped to support the pursuit of exercise through wilderness activities. During pre-trip planning, addressing the daily management of diabetes, acute medical issues, and considerations unique to the wilderness environment is a good start; there are so many variables that it is impossible to come up with a single set of guidelines that can be uniformly applied to all wilderness athletes with diabetes.

It is important to tailor and personalize the medical care of each individual based on personal history and input. Attention should be paid to physical preparation for an excursion, including muscular and aerobic fitness, as well as prior exposure to environmental conditions. Insulin doses and diet plans should be adjusted according to the type and degree of activity that will be performed, an individual's baseline level of fitness, the individual's athletic and disease history, and the environment to which the athlete will be exposed. Wilderness athletes with diabetes should carry a basic written plan developed with their endocrinologist describing their usual insulin regimen, a plan for basic adjustments in the backcountry, basics of hypo-/hyperglycemia management, and an emergency action plan. It is also important to consider the comfort level of an athlete's travel companions, available resources, and emergency evacuation options.

We recommend that healthcare professionals and athletes with diabetes approach wilderness activities through shared decision making. When preparing for wilderness pursuits, it may be most important to consider the athlete's own attitudes toward disease management, awareness of the complexities of their individual disease, and their comfort with daily adjustments. With thoughtful and thorough preparation and planning, the wilderness can be accessible and safe for individuals with diabetes.

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Appendix A. Supplementary materials The following is the Supplementary data to this article: Download : Download Word document (13KB) Supplementary Table 1.

References

1. Centers for Disease Control and Prevention National Diabetes Statistics Report, 2017 Centers for Disease Control and Prevention, Atlanta, GA (2017)
2. M.D. Hoffman, E. Krishnan Health and exercise-related medical issues among 1,212 ultramarathon runners: baseline findings from the Ultrarunners Longitudinal TRAcking (ULTRA) Study PLoS One, 9 (1) (2014), Article e83867 CrossRef

3. M. Khodaee, M. Riederer, K. VanBaak, J.C. Hill Ultraendurance athletes with type 1 diabetes: Leadville 100 experience Wilderness Environ Med, 26 (2) (2015), pp. 273-275 ArticleDownload PDF

4. P. de Mol, S.T. de Vries, E.J. de Koning, R.O. Gans, H.J. Bilo, C.J. Tack Physical activity at altitude: challenges for people with diabetes: a review Diabetes Care, 37 (8) (2014), pp. 2404-2413

5. K.J. Nadeau, J.G. Regensteiner, T.A. Bauer, M.S. Brown, J.L. Dorosz, A. Hull, et al. Insulin resistance in adolescents with type 1 diabetes and its relationship to cardiovascular function J Clin Endocrinol Metab, 95 (2) (2010), pp. 513-521

6. G.P. Kenny, J.M. Stapleton, J.E. Yardley, P. Boulay, R.J. Sigal Older adults with type 2 diabetes store more heat during exercise Med Sci Sports Exerc, 45 (10) (2013), pp. 1906-1914

7. M.R. Carter, R. McGinn, J. Barrera-Ramirez, R.J. Sigal, G.P. Kenny Impairments in local heat loss in type 1 diabetes during exercise in the heat Med Sci Sports Exerc, 46 (12) (2014), pp. 2224-2233

8. D. Patschan, G.A. Muller Acute kidney injury in diabetes mellitus Int J Nephrol, 2016 (2016), p. 6232909

9. W. Abu-Ashour, L.K. Twells, J.E. Valcour, J.M. Gamble Diabetes and the occurrence of infection in primary care: a matched cohort study BMC Infect Dis, 18 (1) (2018), p. 67

10. C. Leal, J. Admetlla, G. Viscor, A. Ricart Diabetic retinopathy at high altitude High Alt Med Biol, 9 (1) (2008), pp. 24-27

11. D. Umpierre, P.A. Ribeiro, C.K. Kramer, C.B. Leitao, A.T. Zucatti, M.J. Azevedo, et al. Physical activity advice only or structured exercise training and association with HbA1c levels in type 2 diabetes: a systematic review and meta-analysis JAMA, 305 (17) (2011), pp. 1790-1799

12. L. Avery, D. Flynn, A. van Wersch, F.F. Sniehotta, M.I. Trenell Changing physical activity behavior in type 2 diabetes: a systematic review and meta-analysis of behavioral interventions Diabetes Care, 35 (12) (2012), pp. 2681-2689

13. L. Chen, J.H. Pei, J. Kuang, H.M. Chen, Z. Chen, Z.W. Li, et al. Effect of lifestyle intervention in patients with type 2 diabetes: a meta-analysis Metabolism, 64 (2) (2015), pp. 338-347

14. D. Sluik, B. Buijsse, R. Muckelbauer, R. Kaaks, B. Teucher, N.F. Johnsen, et al. Physical activity and mortality in individuals with diabetes mellitus: a prospective study and meta-analysis Arch Intern Med, 172 (17) (2012), pp. 1285-1295

15. G.D. Harris, R.D. White Diabetes in the competitive athlete Curr Sports Med Rep, 11 (6) (2012), pp. 309-315

16 S.R. Colberg, R.J. Sigal, J.E. Yardley, M.C. Riddell, D.W. Dunstan, P.C. Dempsey, et al. Physical activity/exercise and diabetes: a position statement of the American Diabetes Association Diabetes Care, 39 (11) (2016), pp. 2065-2079

17 W. Kindermann, A. Schnabel, W.M. Schmitt, G. Biro, J. Cassens, F. Weber Catecholamines, growth hormone, cortisol, insulin, and sex hormones in anaerobic and aerobic exercise Eur J Appl Physiol Occup Physiol, 49 (3) (1982), pp. 389-399

18 R.C. Camacho, P. Galassetti, S.N. Davis, D.H. Wasserman Glucoregulation during and after exercise in health and insulin-dependent diabetes Exerc Sport Sci Rev, 33 (1) (2005), pp. 17-23

19 P. Galassetti, D. Tate, R.A. Neill, S. Morrey, S.N. Davis Effect of gender on counterregulatory responses to euglycemic exercise in type 1 diabetes J Clin Endocrinol Metab, 87 (11) (2002), pp. 5144-5150

20 A. Eliakim, D. Nemet, G. Most, N. Rakover, M. Pantanowitz, Y. Meckel Effect of gender on the GH-IGF-I response to anaerobic exercise in young adults J Strength Cond Res, 28 (12) (2014), pp. 3411-3415

21 E.B. Marliss, M. Vranic Intense exercise has unique effects on both insulin release and its roles in glucoregulation: implications for diabetes Diabetes, 51 (Suppl 1) (2002), pp. S271-S283

22 M.J. MacDonald Postexercise late-onset hypoglycemia in insulin-dependent diabetic patients Diabetes Care, 10 (5) (1987), pp. 584-588

23 F. Magkos, Y. Tsekouras, S.A. Kavouras, B. Mittendorfer, L.S. Sidossis Improved insulin sensitivity after a single bout of exercise is curvilinearly related to exercise energy expenditure Clin Sci (Lond), 114 (1) (2008), pp. 59-64

24 X. Wang, B.W. Patterson, G.I. Smith, J. Kampelman, D.N. Reeds, S.A. Sullivan, et al. A ~60-min brisk walk increases insulin-stimulated glucose disposal but has no effect on hepatic and adipose tissue insulin sensitivity in older women J Appl Physiol (1985), 114 (11) (2013), pp. 1563-1568

25 K.J. Mikines, B. Sonne, P.A. Farrell, B. Tronier, H. Galbo Effect of physical exercise on sensitivity and responsiveness to insulin in humans Am J Physiol, 254 (3 Pt 1) (1988), pp. E248-E259

26 R.D. White Insulin pump therapy (continuous subcutaneous insulin infusion) Prim Care, 34 (4) (2007), pp. 845-871 ArticleDownload PDF 27 D.P. Zaharieva, M.C. Riddell Prevention of exercise-associated dysglycemia: a case study-based approach Diabetes Spectr, 28 (1) (2015), pp. 55-62

28 B.M. Frier Hypoglycaemia in diabetes mellitus: epidemiology and clinical implications Nat Rev Endocrinol, 10 (12) (2014), pp. 711-722

29 J.J. Ruegemer, R.W. Squires, H.M. Marsh, M.W. Haymond, P.E. Cryer, R.A. Rizza, et al. Differences between prebreakfast and late afternoon glycemic responses to exercise in IDDM patients Diabetes Care, 13 (2) (1990), pp. 104-110

30 T.H. Mitchell, G. Abraham, A. Schiffrin, L.A. Leiter, E.B. Marliss Hyperglycemia after intense exercise in IDDM subjects during continuous subcutaneous insulin infusion Diabetes Care, 11 (4) (1988), pp. 311-317

31 M.J. Tansey, E. Tsalikian, R.W. Beck, N. Mauras, B.A. Buckingham, S.A. Weinzimer, et al. The effects of aerobic exercise on glucose and counterregulatory hormone concentrations in children with type 1 diabetes Diabetes Care, 29 (1) (2006), pp. 20-25

32 S.A. Biankin, A.B. Jenkins, L.V. Campbell, K.L. Choi, Q.G. Forrest, D.J. Chisholm Target-seeking behavior of plasma glucose with exercise in type 1 diabetes Diabetes Care, 26 (2) (2003), pp. 297-301

33 J.C. Tee, A.N. Bosch, M.I. Lambert Metabolic consequences of exercise-induced muscle damage Sports Med, 37 (10) (2007), pp. 827-836

34 M.C. Brindisi, B. Bouillet, B. Verges, S. Halimi Cardiovascular complications in type 1 diabetes mellitus Diabetes Metab, 36 (5) (2010), pp. 341-344 ArticleDownload PDF 35 K. Moore, N. Vizzard, C. Coleman, J. McMahon, R. Hayes, C.J. Thompson Extreme altitude mountaineering and type 1 diabetes; the Diabetes Federation of Ireland Kilimanjaro Expedition Diabet Med, 18 (9) (2001), pp. 749-755

36 P. Pavan, P. Sarto, L. Merlo, D. Casara, A. Ponchia, R. Biasin, et al. Metabolic and cardiovascular parameters in type 1 diabetes at extreme altitude Med Sci Sports Exerc, 36 (8) (2004), pp. 1283-1289

37 S. Mohajeri, B.A. Perkins, P.L. Brubaker, M.C. Riddell Diabetes, trekking and high altitude: recognizing and preparing for the risks Diabet Med, 32 (11) (2015), pp. 1425-1437

38 N.W. Pollock, D.M. Uguccioni, G. de Lisle Dear Diabetes and recreational diving: guidelines for the future Diving Hyperb Med, 36 (1) (2006), pp. 29-34

39 R.J. Gibbons, G.J. Balady, J.T. Bricker, B.R. Chaitman, G.F. Fletcher, V.F. Froelicher, et al. ACC/AHA 2002 guideline update for exercise testing: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines) Circulation, 106 (14) (2002), pp. 1883-1892

40 M. Lauer, E.S. Froelicher, M. Williams, P. Kligfield Exercise testing in asymptomatic adults: a statement for professionals from the American Heart Association Council on Clinical Cardiology, Subcommittee on Exercise, Cardiac Rehabilitation, and Prevention Circulation, 112 (5) (2005), pp. 771-776

41 D. Riebe, B.A. Franklin, P.D. Thompson, C.E. Garber, G.P. Whitfield, M. Magal, et al. Updating ACSM's recommendations for exercise preparticipation health screening Med Sci Sports Exerc, 47 (11) (2015), pp. 2473-2479

42 S.J. Curry, A.H. Krist, D.K. Owens, M.J. Barry, A.B. Caughey, K.W. Davidson, et al. Screening for cardiovascular disease risk with electrocardiography: US Preventive Services Task Force recommendation statement JAMA, 319 (22) (2018), pp. 2308-2314

43

1. Cardiovascular Disease and Risk Management: Standards of Medical Care in Diabetes-2018 Diabetes Care, 41 (Suppl 1) (2018), pp. S86-S104

44 T.J. Gluckman, R.J. Kovacs, N.J. Stone, D. Damalas, J.B. Mullen, W.J. Oetgen The ASCVD risk estimator app: from concept to the current state J Am Coll Cardiol, 67 (3) (2016), pp. 350-352 ArticleDownload PDF 45 G.P. Kenny, R.J. Sigal, R. McGinn Body temperature regulation in diabetes Temperature (Austin), 3 (1) (2016), pp. 119-145

46 J.R. Bruso, M.D. Hoffman, I.R. Rogers, L. Lee, G. Towle, T. Hew-Butler Rhabdomyolysis and hyponatremia: a cluster of five cases at the 161-km 2009 Western States Endurance Run Wilderness Environ Med, 21 (4) (2010), pp. 303-308 ArticleDownload PDF 47 J. Baker, J.D. Cotter, D.F. Gerrard, M.L. Bell, R.J. Walker Effects of indomethacin and celecoxib on renal function in athletes Med Sci Sports Exerc, 37 (5) (2005), pp. 712-717

48 T.H. Mader, G. Tabin Going to high altitude with preexisting ocular conditions High Alt Med Biol, 4 (4) (2003), pp. 419-430

49 J.A. Ellerton, I. Zuljan, G. Agazzi, J.J. Boyd Eye problems in mountain and remote areas: prevention and onsite treatment--official recommendations of the International Commission for Mountain Emergency Medicine ICAR MEDCOM Wilderness Environ Med, 20 (2) (2009), pp. 169-175 ArticleDownload PDF 50 T.A. Cope, A. Kropelnicki Eye injuries in the extreme environment ultra-marathon runner BMJ Case Rep, 2015 (2015)

51 M.M. Bosch, D. Barthelmes, K. Landau High altitude retinal hemorrhages--an update High Alt Med Biol, 13 (4) (2012), pp. 240-244

52 P.L. Brubaker Adventure travel and type 1 diabetes: the complicating effects of high altitude Diabetes Care, 28 (10) (2005), pp. 2563-2572

53 C.C. Jimenez, M.H. Corcoran, J.T. Crawley, W. Guyton Hornsby, K.S. Peer, R.D. Philbin, et al. National athletic trainers' association position statement: management of the athlete with type 1 diabetes mellitus J Athl Train, 42 (4) (2007), pp. 536-545

54 H. Bilen, A. Kilicaslan, G. Akcay, F. Albayrak Performance of glucose dehydrogenase (GDH) based and glucose oxidase (GOX) based blood glucose meter systems at moderately high altitude J Med Eng Technol, 31 (2) (2007), pp. 152-156

55 B.H. Ginsberg Factors affecting blood glucose monitoring: sources of errors in measurement J Diabetes Sci Technol, 3 (4) (2009), pp. 903-913

56 D. Oberg, C.G. Ostenson Performance of glucose dehydrogenase-and glucose oxidase-based blood glucose meters at high altitude and low temperature Diabetes Care, 28 (5) (2005), p. 1261

57 J.F. Gautier, A.X. Bigard, P. Douce, A. Duvallet, G. Cathelineau Influence of simulated altitude on the performance of five blood glucose meters Diabetes Care, 19 (12) (1996), pp. 1430-1433

58 K. Nerhus, P. Rustad, S. Sandberg Effect of ambient temperature on analytical performance of self-monitoring blood glucose systems Diabetes Technol Ther, 13 (9) (2011), pp. 883-892

59 M.M. Grajower, C.G. Fraser, J.H. Holcombe, M.L. Daugherty, W.C. Harris, M.R. De Felippis, et al. How long should insulin be used once a vial is started? Diabetes Care, 26 (9) (2003), pp. 2665-2666

60 Insulin administration Diabetes Care, 27 (Suppl 1) (2004), pp. S106-S109 61 A. Zebrowska, B. Hall, A. Kochanska-Dziurowicz, G. Janikowska The effect of high intensity physical exercise and hypoxia on glycemia, angiogenic biomarkers and cardiorespiratory function in patients with type 1 diabetes Adv Clin Exp Med, 27 (2) (2018), pp. 207-216

62 N.E. Hill, K. Deighton, J. Matu, S. Misra, N.S. Oliver, C. Newman, et al. Continuous glucose monitoring at high altitude-effects on glucose homeostasis Med Sci Sports Exerc, 50 (8) (2018), pp. 1679-1686

63 G.L. Peltonen, R.L. Scalzo, M.M. Schweder, D.G. Larson, G.J. Luckasen, D. Irwin, et al. Sympathetic inhibition attenuates hypoxia induced insulin resistance in healthy adult humans J Physiol, 590 (11) (2012), pp. 2801-2809

64 J.J. Larsen, J.M. Hansen, N.V. Olsen, H. Galbo, F. Dela The effect of altitude hypoxia on glucose homeostasis in men J Physiol, 504 (Pt 1) (1997), pp. 241-249

65 R.C. Roach, P.H. Hackett, O. Oelz, P. Bartsch, A.M. Luks, M.J. MacInnis, et al. The 2018 Lake Louise Acute Mountain Sickness Score High Alt Med Biol, 19 (1) (2018), pp. 4-6

66 P. de Mol, S.T. de Vries, E.J. de Koning, R.O. Gans, C.J. Tack, H.J. Bilo Increased insulin requirements during exercise at very high altitude in type 1 diabetes Diabetes Care, 34 (3) (2011), pp. 591-595

67 S.C. Miller Diabetic ketoacidosis and acute mountain sickness: case report and review of treatment options in type 1 diabetes mellitus Wilderness Environ Med, 26 (2) (2015), pp. 185-188 ArticleDownload PDF 68 N.S. Kalson, A.J. Davies, S. Stokes, H. Frost, A.G. Whitehead, I. Tyrrell-Marsh, et al. Climbers with diabetes do well on Mount Kilimanjaro Diabet Med, 24 (12) (2007), p. 1496

69 A.M. Luks, S.E. McIntosh, C.K. Grissom, P.S. Auerbach, G.W. Rodway, R.B. Schoene, et al. Wilderness Medical Society practice guidelines for the prevention and treatment of acute altitude illness: 2014 update Wilderness Environ Med, 25 (4 Suppl) (2014), pp. S4-S14 ArticleDownload PDF 70 B. Basnyat, J.H. Gertsch, E.W. Johnson, F. Castro-Marin, Y. Inoue, C. Yeh Efficacy of low-dose acetazolamide (125 mg BID) for the prophylaxis of acute mountain sickness: a prospective, double-blind, randomized, placebo-controlled trial High Alt Med Biol, 4 (1) (2003), pp. 45-52

71 L. Filippi, F. Bagnoli, M. Margollicci, E. Zammarchi, M. Tronchin, F.F. Rubaltelli Pathogenic mechanism, prophylaxis, and therapy of symptomatic acidosis induced by acetazolamide J Investig Med, 50 (2) (2002), pp. 125-132

72 A. Wajngot, A. Giacca, V. Grill, M. Vranic, S. Efendic The diabetogenic effects of glucocorticoids are more pronounced in low- than in high-insulin responders Proc Natl Acad Sci U S A, 89 (13) (1992), pp. 6035-6039

73 J.L. Hwang, R.E. Weiss Steroid-induced diabetes: a clinical and molecular approach to understanding and treatment Diabetes Metab Res Rev, 30 (2) (2014), pp. 96-102

74 A.L. Vallerand, J. Frim, M.F. Kavanagh Plasma glucose and insulin responses to oral and intravenous glucose in cold-exposed humans J Appl Physiol (1985), 65 (6) (1988), pp. 2395-2399

75 M.J. Hanssen, J. Hoeks, B. Brans, A.A. van der Lans, G. Schaart, J.J. van den Driessche, et al. Short-term cold acclimation improves insulin sensitivity in patients with type 2 diabetes mellitus Nat Med, 21 (8) (2015), pp. 863-865

76 M. Berger, H.J. Cuppers, H. Hegner, V. Jorgens, P. Berchtold Absorption kinetics and biologic effects of subcutaneously injected insulin preparations Diabetes Care, 5 (2) (1982), pp. 77-91

77 I. Harirchi, A. Arvin, J.H. Vash, V. Zafarmand Frostbite: incidence and predisposing factors in mountaineers Br J Sports Med, 39 (12) (2005), pp. 898-901

78 C.L. Dumke, D.R. Slivka, J.S. Cuddy, W.S. Hailes, S.M. Rose, B.C. Ruby The effect of environmental temperature on glucose and insulin after an oral glucose tolerance test in healthy young men Wilderness Environ Med, 26 (3) (2015), pp. 335-342 ArticleDownload PDF 79 R.G. Moses, M.J. Patterson, J.M. Regan, R. Chaunchaiyakul, N.A. Taylor, A.B. Jenkins A non-linear effect of ambient temperature on apparent glucose tolerance Diabetes Res Clin Pract, 36 (1) (1997), pp. 35-40 ArticleDownload PDF 80 A.O. Akanji, R.N. Oputa The effect of ambient temperature on glucose tolerance Diabet Med, 8 (10) (1991), pp. 946-948

81 V.A. Koivisto Sauna-induced acceleration in insulin absorption from subcutaneous injection site Br Med J, 280 (6229) (1980), pp. 1411-1413

82 M. Naperalsky, B. Ruby, D. Slivka Environmental temperature and glycogen resynthesis Int J Sports Med, 31 (8) (2010), pp. 561-566

83 J.M. Stapleton, J.E. Yardley, P. Boulay, R.J. Sigal, G.P. Kenny Whole-body heat loss during exercise in the heat is not impaired in type 1 diabetes Med Sci Sports Exerc, 45 (9) (2013), pp. 1656-1664

84 L.M. Laffel, C. Limbert, H. Phelan, A. Virmani, J. Wood, S.E. Hofer ISPAD Clinical Practice Consensus Guidelines 2018: sick day management in children and adolescents with diabetes Pediatr Diabetes, 19 (Suppl 27) (2018), pp. 193-204

85 J.E. Yardley, J.M. Stapleton, R.J. Sigal, G.P. Kenny Do heat events pose a greater health risk for individuals with type 2 diabetes? Diabetes Technol Ther, 15 (6) (2013), pp. 520-529

86 J.E. Yardley, J.M. Stapleton, M.R. Carter, R.J. Sigal, G.P. Kenny Is whole-body thermoregulatory function impaired in type 1 diabetes mellitus? Curr Diabetes Rev, 9 (2) (2013), pp. 126-136

87 M.C. Riddell, I.W. Gallen, C.E. Smart, C.E. Taplin, P. Adolfsson, A.N. Lumb, et al. Exercise management in type 1 diabetes: a consensus statement Lancet Diabetes Endocrinol, 5 (5) (2017), pp. 377-390 ArticleDownload PDF 88 M.P. Francescato, G. Stel, E. Stenner, M. Geat Prolonged exercise in type 1 diabetes: performance of a customizable algorithm to estimate the carbohydrate supplements to minimize glycemic imbalances PLoS One, 10 (4) (2015), Article e0125220 CrossRef 89 B.L. Bennett, T. Hew-Butler, M.D. Hoffman, I.R. Rogers, M.H. Rosner Wilderness Medical Society practice guidelines for treatment of exercise-associated hyponatremia: 2014 update Wilderness Environ Med, 25 (4 Suppl) (2014), pp. S30-S42 ArticleDownload PDF 90 M.D. Hoffman, T. Hew-Butler, K.J. Stuempfle Exercise-associated hyponatremia and hydration status in 161-km ultramarathoners Med Sci Sports Exerc, 45 (4) (2013), pp. 784-791

91 J.E. Yardley, S.R. Colberg Update on management of type 1 diabetes and type 2 diabetes in athletes Curr Sports Med Rep, 16 (1) (2017), pp. 38-44

92 I. Pernicova, M. Korbonits Metformin--mode of action and clinical implications for diabetes and cancer Nat Rev Endocrinol, 10 (3) (2014), pp. 143-156

93 A.J. Garber, T.G. Duncan, A.M. Goodman, D.J. Mills, J.L. Rohlf Efficacy of metformin in type II diabetes: results of a double-blind, placebo-controlled, dose-response trial Am J Med, 103 (6) (1997), pp. 491-497 ArticleDownload PDF 94 F.J. Pasquel, R. Klein, A. Adigweme, Z. Hinedi, R. Coralli, J.L. Pimentel, et al. Metformin-associated lactic acidosis Am J Med Sci, 349 (3) (2015), pp. 263-267 ArticleDownload PDF 95 C.T. Supuran Drug interaction considerations in the therapeutic use of carbonic anhydrase inhibitors Expert Opin Drug Metab Toxicol, 12 (4) (2016), pp. 423-431

96 G. Derosa, P. Maffioli Efficacy and safety profile evaluation of acarbose alone and in association with other antidiabetic drugs: a systematic review Clin Ther, 34 (6) (2012), pp. 1221-1236 ArticleDownload PDF 97 S. Subbarayan, M. Kipnes Sitagliptin: a review Expert Opin Pharmacother, 12 (10) (2011), pp. 1613-1622

98 A.J. Scheen SGLT2 inhibitors: benefit/risk balance Curr Diab Rep, 16 (10) (2016), p. 92

99 US Food & Drug Administration FDA revises labels of SGLT2 inhibitors for diabetes to include warnings about too much acid in the blood and serious urinary tract infections Drug Safety Communications (2015)

100 K. Bettge, M. Kahle, M.S. Abd El Aziz, J.J. Meier, M.A. Nauck Occurrence of nausea, vomiting and diarrhoea reported as adverse events in clinical trials studying glucagon-like peptide-1 receptor agonists: a systematic analysis of published clinical trials Diabetes Obes Metab, 19 (3) (2017), pp. 336-347

101 A. Caduff, H.U. Lutz, L. Heinemann, G. Di Benedetto, M.S. Talary, S. Theander Dynamics of blood electrolytes in repeated hyper- and/or hypoglycaemic events in patients with type 1 diabetes Diabetologia, 54 (10) (2011), pp. 2678-2689

102 Glucagon rDNA origin (GlucaGen) and recombinant LH. (no. 12 in a series of articles to promote a better understanding of the use of genetic engineering) J Biotechnol, 79 (2) (2000), pp. 185-189

103 M.W. Haymond, M.J. Redondo, S. McKay, M.J. Cummins, B. Newswanger, J. Kinzell, et al. Nonaqueous, mini-dose glucagon for treatment of mild hypoglycemia in adults with type 1 diabetes: a dose-seeking study Diabetes Care, 39 (3) (2016), pp. 465-468

104 M.R. Rickels, K.J. Ruedy, N.C. Foster, C.A. Piche, H. Dulude, J.L. Sherr, et al. Intranasal glucagon for treatment of insulin-induced hypoglycemia in adults with type 1 diabetes: a randomized crossover noninferiority study Diabetes Care, 39 (2) (2016), pp. 264-270

105 N. Kedia Treatment of severe diabetic hypoglycemia with glucagon: an underutilized therapeutic approach Diabetes Metab Syndr Obes, 4 (2011), pp. 337-346

106 G. Harrism, A. Diment, M. Sulway, M. Wilkinson Glucagon administration – underevaluated and undertaught Practl Diabetes Intern, 18 (1) (2001), pp. 22-25 CrossRef 107 National Collaborating Centre for Women’s and Children’s Health (UK) Management of type 1 diabetes - targets for and monitoring of glycaemic control Diabetes (Type 1 and Type 2) in Children and Young People: Diagnosis and Management, Vol 18, NICE Guideline (2015)

108 D.M. Cline, MJ, R.K. Cydulka, G.D. Meckler, D.A. Handel, S.H. Thomas Tintinalli's Emergency Medicine Manual (7th ed.), McGraw-Hill Companies, Inc. (2012)

109 B. Matejko, A. Gawrecki, M. Wrobel, J. Hohendorff, T. Benbenek-Klupa, M.T. Malecki, et al. Type 1 diabetes at high altitude: performance of personal insulin pumps and patient metabolic control Diabetes Technol Ther, 19 (10) (2017), pp. 600-602

110

1. Glycemic Targets: Standards of medical care in diabetes-2018 Diabetes Care, 41 (Suppl 1) (2018), pp. S55-S64

111 R.M. Bergenstal, W.V. Tamborlane, A. Ahmann, J.B. Buse, G. Dailey, S.N. Davis, et al. Effectiveness of sensor-augmented insulin-pump therapy in type 1 diabetes N Engl J Med, 363 (4) (2010), pp. 311-320

112 R.W. Beck, T. Riddlesworth, K. Ruedy, A. Ahmann, R. Bergenstal, S. Haller, et al. Effect of continuous glucose monitoring on glycemic control in adults with type 1 diabetes using insulin injections: the DIAMOND randomized clinical trial JAMA, 317 (4) (2017), pp. 371-378

113 M. Lind, W. Polonsky, I.B. Hirsch, T. Heise, J. Bolinder, S. Dahlqvist, et al. Continuous glucose monitoring vs conventional therapy for glycemic control in adults with type 1 diabetes treated with multiple daily insulin injections: the GOLD randomized clinical trial JAMA, 317 (4) (2017), pp. 379-387

114 T. Battelino, I. Conget, B. Olsen, I. Schutz-Fuhrmann, E. Hommel, R. Hoogma, et al. The use and efficacy of continuous glucose monitoring in type 1 diabetes treated with insulin pump therapy: a randomised controlled trial Diabetologia, 55 (12) (2012), pp. 3155-3162

115 W.V. Tamborlane, R.W. Beck, B.W. Bode, B. Buckingham, H.P. Chase, R. Clemons, et al. Continuous glucose monitoring and intensive treatment of type 1 diabetes N Engl J Med, 359 (14) (2008), pp. 1464-1476

116 J.J. Chamberlain, D. Dopita, E. Gilgen, A. Neuman Impact of frequent and persistent use of continuous glucose monitoring (CGM) on hypoglycemia fear, frequency of emergency medical treatment, and SMBG frequency after one year J Diabetes Sci Technol, 10 (2) (2015), pp. 383-388

117 J. Soupal, L. Petruzelkova, M. Flekac, T. Pelcl, M. Matoulek, M. Dankova, et al. Comparison of different treatment modalities for type 1 diabetes, including sensor-augmented insulin regimens, in 52 weeks of follow-up: a COMISAIR study Diabetes Technol Ther, 18 (9) (2016), pp. 532-538

118 B.A. Buckingham, G.P. Forlenza, J.E. Pinsker, M.P. Christiansen, R.P. Wadwa, J. Schneider, et al. Safety and feasibility of the OmniPod hybrid closed-loop system in adult, adolescent, and pediatric patients with type 1 diabetes using a personalized model predictive control algorithm Diabetes Technol Ther, 20 (4) (2018), pp. 257-262

119 E. Bekiari, K. Kitsios, H. Thabit, M. Tauschmann, E. Athanasiadou, T. Karagiannis, et al. Artificial pancreas treatment for outpatients with type 1 diabetes: systematic review and meta-analysis BMJ, 361 (2018), p. k1310 CrossRef 120 M.C. Riddell, D.P. Zaharieva, L. Yavelberg, A. Cinar, V.K. Jamnik Exercise and the development of the artificial pancreas: one of the more difficult series of hurdles J Diabetes Sci Technol, 9 (6) (2015), pp. 1217-1226

121 M.D. Breton, D.R. Chernavvsky, G.P. Forlenza, M.D. DeBoer, J. Robic, R.P. Wadwa, et al. Closed-loop control during intense prolonged outdoor exercise in adolescents with type 1 diabetes: the artificial pancreas ski study Diabetes Care, 40 (12) (2017), pp. 1644-1650

122 A. Siafarikas, R.J. Johnston, M.K. Bulsara, P. O'Leary, T.W. Jones, E.A. Davis Early loss of the glucagon response to hypoglycemia in adolescents with type 1 diabetes Diabetes Care, 35 (8) (2012), pp. 1757-1762

123 P.G. Jacobs, J. El Youssef, R. Reddy, N. Resalat, D. Branigan, J. Condon, et al. Randomized trial of a dual-hormone artificial pancreas with dosing adjustment during exercise compared with no adjustment and sensor-augmented pump therapy Diabetes Obes Metab, 18 (11) (2016), pp. 1110-1119

AUSTRALIAN RESUSCITATION COUNCIL GUIDELINE 9.3.2 - RESUSCITATION OF THE DROWNING VICTIM

INTRODUCTION

Drowning is the process of experiencing respiratory impairment from submersion/immersion in liquid. Drowning outcomes are classified as death, morbidity and no morbidity – the latter two now referred to as “non-fatal drownings”.(1)

The most important consequence of drowning is interruption of the oxygen supply to the brain. Early rescue and resuscitation by trained first responders or first aiders at the scene offer the victim the best chance of survival.

POSSIBLE SEQUENCE OF EVENTS

- Immersion of the face in water (or other liquid). Water entering the mouth is spat out, swallowed or aspirated.
- Breath-holding, usually lasting no more than a minute.
- Vigorous breathing efforts. These may continue, even after loss of consciousness. Some amount of water is aspirated into the airways causing coughing and sometimes laryngeal spasm, which temporarily prevents further water entering the lungs.
- Swallowing of air and water, often in large amounts. This usually causes vomiting or regurgitation of stomach contents, which may be aspirated into the lungs.
- Respiratory impairment causes brain hypoxia, leading to unconsciousness and cessation of breathing efforts.
- The heart rate initially increases with exercise and panic. With hypoxia, the heart rate and blood pressure begin to fall, progressing finally to a cardiac arrest, requiring CPR.

MANAGEMENT

- Remove the victim from the water as soon as possible but do not endanger your own safety. Throw a rope or something to provide buoyancy to the victim. Call for help; plan and effect a safe rescue.
- In minor incidents, removal from the water is often followed by coughing and spontaneous resumption of breathing.

- In more serious incidents, assess the victim. If unconscious or not breathing normally, commence resuscitation following the ANZCOR Basic Life Support Flowchart.
- Assess the victim on the back with the head and the body at the same level, rather than in a head down position. This decreases the likelihood of regurgitation and vomiting and is associated with increased survival.(2)
- The victim should not be routinely rolled onto the side to assess airway and breathing. Assessing the airway of the victim without turning onto the side (i.e. leaving the victim on the back or in the position in which they have been found) has the advantages of simplified teaching, taking less time to perform and avoids movement ANZCOR Guideline 4 Airway.
- The exceptions to this would be where the airway is obstructed with fluid (water or blood) or particulate matter (sand, debris, vomit). In this instance the victim should be promptly rolled onto the side to clear the airway. The mouth should be opened and turned slightly downwards to allow any foreign material to drain using gravity ANZCOR Guideline 4 Airway.
- Vomiting and regurgitation often occur during the resuscitation of a drowned victim. If the victim has been rolled to the side to clear the airway, then reassess their condition. If breathing commences, the victim can be left on the side with appropriate head tilt. If not breathing normally, the victim should be promptly rolled onto the back and resuscitation recommenced as appropriate (ARC Guideline 4).
- Avoid delays or interruptions to CPR. Do not empty a distended stomach by applying external pressure. Do not attempt to expel or drain clear water or frothy fluid that may re-accumulate in the upper airway during resuscitation.
- Victims who appear to have been successfully rescued and resuscitated require close monitoring to detect a relapse into cardiopulmonary arrest. This can occur in the minutes or hours following return of spontaneous circulation and breathing, due to persisting lung damage and hypoxic injury to the heart. (3)
- Call an ambulance for all victims of an immersion event, even if seemingly minor or the victim appears recovered.

NOTES

Oxygen

The administration of oxygen is beneficial in the resuscitation of drowned victims, but resuscitation efforts should not be delayed while waiting for oxygen equipment to become available.

Medical conditions leading to sudden incapacitation in the water

Not all water related deaths are primary drowning. Sudden incapacitation leading to swim failure, unconsciousness and subsequent water in the airway can occur due to heart attacks, cardiac rhythm disturbances, seizure disorders, hyperventilation, drugs and alcohol, dementia, frailty and other conditions causing

loss of consciousness, e.g. low blood sugar in a diabetic. These conditions should be suspected in known competent swimmers found drowned unexpectedly. In some victims these medical conditions can be aggravated by the shock of sudden immersion in cold water.

Spinal injuries occurring in the water

Spinal injury occurring concurrently with drowning is rare, estimated at less than 0.5% but should be suspected if the victim dived into shallow water, is found in an area of dumping surf, rocks or after an accident involving a boat or other aquatic craft.(4)Remove the victim from the water taking care to keep the airway clear of wave splash while minimising movement of the spine in any direction. Airway management takes precedence over a suspected spinal injury and an unconscious, non-breathing victim should be removed immediately from the water by whatever means possible.

Concurrent hypothermia

There is no evidence that drowning in colder water has an increased survival rate compared with warmer water, yet the literature yields many case reports of successful outcomes of victims rescued from icy waters, even after prolonged resuscitation efforts. Hypothermia is more likely due to prolonged immersion time and ongoing cooling during resuscitation at the scene, in a wet, open environment.

In-water resuscitation

In-water resuscitation may improve survival of victims who are in the initial stages of the drowning sequence but delays time to full assessment and CPR.3,5 Remove the victim from the water as soon as possible, and only begin in water rescue breathing if immediate removal from the water is delayed or impossible. Rescue breathing in deep water requires an appropriately trained rescuer and floatation aid such as a rescue board, tube or buoyancy vest. In water, chest compressions are ineffective and should not be attempted.

Use of the AED

If available, the AED should be attached and the prompts followed. Dry the victim’s chest before applying pads. Although the rhythm deterioration in drowning is usually to a non-shockable rhythm, the AED may be lifesaving in ~6% of drowning victims who, on initial assessment, are found to have a shockable cardiac rhythm.(6)

Compression-only CPR is not the recommended resuscitation method

The primary cause of cardiac arrest in drowning is a lack of breathing. Compression-only CPR circulates oxygen-poor blood and fails to address the victim’s need for immediate ventilation. It is not the recommended resuscitation

method in a victim of drowning and should only be used temporarily if the rescuer is unable or unwilling to perform rescue breathing before the arrival of a barrier device, face mask or bag-valve-mask device.

LEVEL OF EVIDENCE III CLASS OF RECOMMENDATION A

REFERENCES

1. World Health Organization 2013 http://www.who.int/violence_injury_prevention/other_injury/drowning/en/
2. Szpilman, D, Idris, A, Cruz Filho, F 2002, 'Position of Drowning Resuscitation Victims on a Sloping Beach', abstract presented at the World Conference on Drowning, Amsterdam, The Netherlands, 26-28 June.
3. Manolis, N & Mackie, I 1988, 'Drowning and near-drowning on Australian beaches patrolled by life-savers: a 10-year study, 1973-1983'. Medical Journal of Australia, vol 148, pp 165-171.
4. Watson, RS, Cummings, P, Quan, L, Bratton s, Weiss, NS 2001, 'Cervical spine injuries among submersion victims'. Journal of Trauma, vol 51, pp 656-662.
5. Szpilman, D, Soares, M 2004, 'In-water resuscitation – is it worthwhile?' Resuscitation, vol 63, pp 25-31.
6. Dyson, K, Morgans, A, Bray, J, Matthews, B, Smith, K 2013, 'Drowning related out-of- hospital cardiac arrests: Characteristics and outcomes'. Resuscitation, vol 84, pp 1114- 1118.

ADDITIONAL RESOURCES

- Surf Life Saving Australia: Public Safety and Aquatic Rescue Manual 33rd Edition Revised November 2011
- Royal Life Saving Society Australia: Lifeguarding 4th Edition

FURTHER READING

ARC Guideline 3 Unconsciousness

ANZCOR Guideline 4 Airway

ANZCOR Guideline 5 Breathing

ANZCOR Guideline 8: Cardiopulmonary Resuscitation

ANZCOR Guidelines 9.1.6 Management of Suspected Spinal Injury

ANZCOR Guideline 9.3.3 Hypothermia: First-Aid Management

ANZCOR Guidelines 9.2.10: Use of Oxygen in Emergencies

AUSTRALIAN RESUSCITATION COUNCIL GUIDELINE 9.3.3 HYPOTHERMIA: FIRST AID MANAGEMENT

INTRODUCTION:

For normal function of most body systems and organs the human body's temperature is kept controlled between narrow limits (about 37 deg.C). Hypothermia is when the body's temperature is below 35 deg.C. As the body's temperature falls, systems and organs progressively fail until death occurs, usually from cardiac arrest. Infants and elderly people are at greater risk. Hypothermia may develop acutely (eg: falling into icy water) or be a gradual and insidious process.

COMMON CAUSES:

- ENVIRONMENTAL: exposure to cold, wet, or windy conditions; cold water immersion/submersion; exhaustion.
- TRAUMA: trauma, immobility and burns.
- DRUGS: alcohol and / or sedatives.
- NEUROLOGICAL: stroke and altered consciousness.
- ENDOCRINE: impaired metabolism.
- SYSTEMIC ILLNESS: severe infections, malnutrition.

RECOGNITION:

- **Mild hypothermia:**
    - victim shivering,
    - pale, cool skin,
    - impaired coordination,
    - slurred speech,
    - responsive but with apathy or confusion.
- **Moderate to severe hypothermia:**
    - absence of shivering,
    - increasing muscle stiffness,
    - progressive decrease in consciousness,
    - slow irregular pulse
    - hypotension.

In more severe cases there may be dangerous cardiac arrhythmias and cardiac arrest, fixed dilated pupils. The victim may appear dead, particularly if they have a weak slow pulse.

MANAGEMENT

- Call an ambulance (Dial Triple Zero - 000).
- Remove from cold environment.
- General and supportive treatment with application of BLS where appropriate; this must continue until the victim is rewarmed.
- Remove sources of heat loss such as wet clothing, contact with cold surfaces and windy environment. Do not remove wet clothing if there is no dry blanket or other suitable cover. Apply insulation between body and the environment (eg. blanket).
- Dry the victim if wet.
- Give warm oral fluids (not alcohol) and only if the victim is fully conscious.
- If the victim is in a remote location and not shivering the rescuer should initiate active rewarming(1).
- Cautiously apply a source of external heat such as heat pack or body to body contact.
- To avoid burns ensure that any heat source is warm or tepid but not hot.
- Do not place the victim in a warm bath.

LEVEL OF EVIDENCE Level III

CLASS OF RECOMMENDATION Class A - Recommended

REFERENCES:

1. First Aid Science Advisory Board. Part 10: First Aid. Circulation, 2005.112:115-125.

FURTHER READING

ANZCOR Guideline 8: Cardiopulmonary Resuscitation

ANZCOR GUIDELINE 9.3.4 – HEAT INDUCED ILLNESS (HYPERTHERMIA) GUIDELINE

Who does this guideline apply to?

This guideline applies to adult, child and infant victims.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid providers.

1 Introduction

Heat induced illness may be caused by:

- excessive heat absorption from a hot environment
- excessive heat production from metabolic activity
- failure of the cooling mechanisms
- an alteration in the body's set temperature.

Factors which may contribute to heat induced illness include:

- excessive physical exertion
- hot climatic conditions with high humidity
- inadequate fluid intake
- infection (particularly a viral illness)
- inappropriate environments (e.g. unventilated hot buildings)
- wearing unsuitably heavy, dark clothing on hot days
- drugs which affect heat regulation.

The very young1, 2, 3 and very old(4) are more prone to heat induced illness.

2 Prevention

On warm, humid or hot days:

- keep infants and the elderly in cool, ventilated areas and provide ample oral fluids
- wear light coloured, loose-fitting clothing during physical exertion and hats during outside activities
- take adequate fluids during exertion on hot days – thirst is a useful guide to required fluid intake.

For participants in, and organisers of sporting events:

- allow six weeks for acclimatisation with progressive exercise before competition
- avoid vigorous exercise during a viral illness
- plan to conduct events in the early morning or late evening or in the cooler months of the year
- provide regular drink stations
- follow the support guidelines relevant to specific activities.

At no time should children or the elderly be left unattended in parked cars.

For workers in outdoor or potentially hot environments, refer to occupational health guidelines relevant to the particular environment. Work environments that may be particularly prone to precipitating hyperthermia and heat induced illness include those in which there is a high ambient temperature with reduced air movement, the worker is exposed to radiant heat and there is difficulty in maintaining adequate hydration.

3 Recognition

Mild elevation in body temperature is normally controlled with sweating, which allows cooling by evaporation. Once the individual becomes too dehydrated to sweat, body temperature can rise rapidly and dramatically.

3.1 Heat Exhaustion

Heat exhaustion is recognized by fatigue associated with headache, nausea, vomiting malaise and dizziness, which may be accompanied by collapse. Body temperature will be less than 40°C and conscious state will become normal once the victim is lying down(5).

3.2 Heat Stroke

Heat stroke is the most serious form of heat related illness and may lead to unconsciousness and death. All body organs may be affected. Heat stroke may be recognized by lack of sweating, temperature above 40°C, an altered conscious state, hot dry skin (however, in some victims profuse sweating is common) and collapse(5).

4 Management

The management of heat induced illness is aimed at removing the cause and assisting the normal cooling mechanisms of evaporation, conduction, radiation and convection.

4.1 Heat Exhaustion

- lie the victim down in a cool environment or in the shade
- loosen and remove excessive clothing

- moisten the skin with a moist cloth or atomizer spray
- cool by fanning
- give water to drink if fully conscious
- call for an ambulance if not quickly improving.

4.2 Heat Stroke

Heat stroke is a life threatening condition:(6,7)

- call for an ambulance
- resuscitate following the Basic Life Support Flow Chart (ANZCOR Guideline 8)
- place the victim in a cool environment
- moisten the skin with a moist cloth or atomizer spray and fan repeatedly
- apply wrapped ice packs to neck, groin and armpits.

ANZCOR suggest a 3-8% carbohydrate electrolyte fluid [any commercially available “sports drink”] for the treatment of exertion related dehydration (CoSTR 2015, weak recommendation, very low quality evidence)(8). If carbohydrate electrolyte fluid is unavailable, water is an acceptable alternative.

4.3 Febrile Convulsions

- Follow First Aid Management of a Seizure (ANZCOR Guideline 9.2.4).

References

1. Falk B. Effects of thermal stress during rest and exercise in the paediatric population, Sports Med. 1998. **25:** 221 –240.
2. Harpin V.A., Chellappah G. & Rutter N. Responses of the newborn infant to overheating, Biol. Neonate 1983. **44:** 65–75.
3. Mellor M.F.A., Heat-induced illnesses. In: R.M. Barkin, Editor, Pediatric Emergency Medicine (2nd ed), St Louis, MO. Mosby. 1997: 496–499.
4. Woodruff R., Hales S., Butler C., McMichael A. (2005) “Climate Change Health Impacts in Australia”, joint report for the Australian Conservation Foundation and the Australian Medical Association, http://www.ama.com.au.
5. Rogers I. & Williams A. Heat-related illness. In: Cameron P., Jelinek G., Kelly A., Murray L., Heyworth J., editors. Adult Textbook of Emergency Medicine. 2nd ed. Sydney: Churchill Livingstone. 2004: 748-751.
6. Bouchama A., Dehbi M., Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical recommendations. Critical Care 2007, **11** (3):54.
7. Ramsey C.B., Watson W.A., Robinson W.A. Effect of cooling time on survival in classical heatstroke. Journal of Wilderness Medicine 1993. **4** (1): 27–31.
8. Zideman, D. A., Singletary, E. M., De Buck, E.,et al. (2015). Part 9: First aid: 2015 International Consensus on First Aid Science with Treatment Recommendations. *Resuscitation, 95* , e225. http://www.cprguidelines.eu/assets/downloads/costr/S0300-9572(15)00368-8_main.pdf00368-8_main.pdf) Accessed 19/11/

Further Reading

ANZCOR Guideline 2 Priorities in an Emergency

ANZCOR Guideline 8: Cardiopulmonary Resuscitation

ANZCOR Guideline 9.2.4 First-Aid Management of a Seizure

ANZCOR Guideline 9.3.5 - First Aid and Resuscitation for Divers who have Breathed Compressed Gas

Summary

Who does this guideline apply to?

This guideline applies to adults and children over 8 years old who have dived while breathing compressed gas during the previous 24 hours or have travelled to altitude (e.g. by aircraft) within 24 hours of diving and have developed symptoms and signs which could be related to the diving.

Who is the audience for this guideline?

This guideline is for use by divers, dive professionals, bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. Send for an ambulance early in the treatment of suspected decompression illness or pulmonary barotrauma and promptly contact the nearest public hospital Diving and Hyperbaric Medicine Unit (in Australia) or Diving Emergency Service Hotline (New Zealand) for specialist diving medical advice. [Good Practice Statement]
2. Provide near-100% oxygen to the person as soon as possible and continue oxygen administration until relieved by medical personnel. (Near-100% oxygen should be administered even if pulse oximetry indicates a high oxygen saturation). [Good Practice Statement]
3. Manage the person in a horizontal position if early onset decompression illness is suspected. Otherwise, and in the event of breathing difficulty, the person can be managed in a position of comfort. [Good Practice Statement]
4. Record details of the dive(s), the symptoms and signs, first aid provided and response. [Good Practice Statement]
5. An alert and stable person suspected of having decompression illness may drink non-alcoholic fluids as advised by the diving doctor. [Good Practice

Statement]
6. Keep the person thermally comfortable. [Good Practice Statement]

Abbreviations

Abbreviation Meaning/Phrase

ANZCOR- Australian and New Zealand Committee on Resuscitation SSBA- Surface Supplied Breathing Apparatus DCI- Decompression Illness PBT- Pulmonary Barotrauma DCS- Decompression Sickness CPR- Cardiopulmonary Resuscitation CAGE- Cerebral Arterial Gas Embolism CoSTR- Consensus on Science with Treatment Recommendations (from International Liaison Committee on Resuscitation - ILCOR)

Guideline

1 Introduction

'Compressed gas' divers breathe gas (usually air) while underwater. Most commonly, divers use self-contained underwater breathing apparatus (scuba) and breathe from cylinders carried underwater. However, the breathing gas can also be supplied from the surface via a surface supplied breathing apparatus (SSBA). Divers are vulnerable to a variety of potential injuries and illnesses which include ear injuries, drowning, carbon monoxide poisoning, and heart attack, among others. These can be managed by the usual first aid and resuscitation protocols outlined in various ANZCOR Guidelines. However, breathing compressed gas underwater can lead to several unique medical problems, the most significant being decompression illness (DCI) and pressure damage of the lungs called pulmonary barotrauma (PBT). Australian hyperbaric units treat an average of 125 cases of DCI a year.[1] Twenty- nine percent of calls to an Australian based diving emergency hotline were due to suspected DCI, compared to 1% from PBT.[2]

Decompression illness and pulmonary barotrauma require special first aid considerations, including the prompt and continued administration of near-100% oxygen. This is different from most other first aid uses of oxygen as detailed in ANZCOR Guideline 9.2.10, which should be read in conjunction with this guideline.

2 Background

2.1 Decompression Sickness

During an air dive, oxygen and nitrogen from the inhaled gas is dissolved in the diver's blood and body tissues. The oxygen is used by the body but the nitrogen remains dissolved in the diver's blood and tissues. Unless the diver ascends slowly enough to allow excess nitrogen to leave the body gradually through the lungs, nitrogen comes out of solution and may form bubbles in the diver's blood

and body tissues. The physical effects of these bubbles, and biochemical changes incited by them, can cause lack of blood supply (ischaemic) and inflammatory tissue damage. This is known as decompression sickness (DCS). Some deep divers breathe mixtures of gas containing helium and may face the same problems due to helium bubbles.

2.2 Pulmonary Barotrauma

As a diver ascends, the gas in the lungs expands with reducing ambient pressure and, unless expanding gas is adequately exhaled, the diver's lungs can distend and tear. This can result in a collapsed lung (pneumothorax) and/or trapping of gas in the mediastinum (mediastinal emphysema), or under the skin (subcutaneous emphysema). Escaped gas may also enter the arterial circulation and distribute to the cerebral circulation causing cerebral arterial gas embolism (CAGE), causing stroke-like symptoms and signs.

2.3 Decompression Illness

The term decompression illness (DCI) is used to collectively describe both DCS and CAGE. In the emergency setting it is unnecessary to differentiate between these as the first aid and more advanced treatment is the same for both conditions.

3 Recognition

3.1 Decompression Illness

DCI is associated with a wide range of potential symptoms and signs, from minor to rapidly fatal.

Common symptoms and signs include:

- pain (often at or near joints)
- numbness/tingling
- extreme fatigue/feeling unwell
- dizziness/vertigo
- muscle weakness in one or both arms and/or legs
- mottled skin rash
- poor co-ordination
- altered consciousness

3.2 Pulmonary barotrauma

- chest pain
- difficulty breathing
- coughing
- blueness of lips and tongue (cyanosis)
- voice changes
- difficulty swallowing

- 'crackly' skin around neck (crepitus)
- symptoms and signs of decompression illness may also be present.

4 Management

- If the person is not responding and is not breathing normally, commence resuscitation following the ANZCOR Basic Life Support flowchart [Refer to ANZCOR Guideline 8]. [Good Practice Statement] A person with DCI may regain consciousness and appear to have recovered but still needs to be managed for suspected DCI due to the possibility of relapse.
- Promptly provide as close to 100% oxygen as possible if available and trained to do so and continue to do so until the ambulance/evacuation personnel arrives and takes over management.[3,4,5] Near 100% oxygen should be administered even if pulse oximetry indicates a high oxygen saturation. [Good Practice Statement]
- If early-onset DCI is suspected (within 30 minutes of surfacing), lay the person flat if possible.[3,6] [Good Practice Statement]
- Seek immediate diving medical advice by calling the nearest public hospital diving and hyperbaric medicine unit (in Australia), or the Diver Emergency Service (0800 4 DES 111 in New Zealand).[3] [Good Practice Statement]
- Assist with any transfer to a recompression chamber if requested to do so.
- The consulting diving doctor may advise that an alert and stable person thought to be suffering from DCI may freely drink non-alcoholic fluids, such as water or isotonic/electrolyte fluids (as long as they have no stomach cramps, nausea, urinary retention or paralysis).[3,9,10] [Good Practice Statement]
- The person should be kept thermally comfortable (warm but not hyperthermic).[3,6,11] [Good Practice Statement]
- Record details of the dive(s), the symptoms and signs (and their timing) the first aid given and the response to the first aid. [Good Practice Statement]

Rationale

A flat (horizontal) posture without leg elevation is recommended in persons suspected of DCI as it has been shown to increase the rate of inert gas elimination.[6] It may also reduce the likelihood of arterial bubbles distributing to the brain.[8] An unconscious diver should be managed in the 'recovery position'. However, a conscious person having increased difficulty breathing when supine can be placed in a position of comfort.

Administration of 100% oxygen reduces the size and number of gas bubbles in the blood and tissues by helping to eliminate the inert gas.[5,12]

References

1. Historical decompression illness data - Australia. Available from: https://www.adsf.org.au/r/general-reports. [cited 2020 October 18].

2. Wilkinson D, Goble S. A review of 17 years of telephone calls to the Australian Diver Emergency Service (DES). Diving Hyperb Med. 2012; 42(3):137-145.
3. Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR, Kot J, Lafère P. Pre- hospital management of decompression illness: expert review of key principles and controversies. Diving Hyperb Med. 2018 Mar 31;48(1):45-55.
4. Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiberger JJ. First aid normobaric oxygen for the treatment of recreational diving injuries. Undersea Hyperb Med. 2007 Jan-Feb;34(1):43-9.
5. Hyldegaard O, Møller M, Madsen J. Effect of He-O2, O2, and N2O-O2 breathing on injected bubbles in spinal white matter. Undersea Biomed Res. 1991 Sep-Nov;18(5-6):361-71.
6. Balldin UI. Effects of ambient temperature and body position on tissue nitrogen elimination in man. Aerosp Med. 1973 Apr;44(4):365-70.
7. Dutka , AJ ; Polychronidis , J; Mink, RB; Hallenbeck, JM. Head-down position after air embolism impairs recovery of brain function as measured by the somatosensory evoked response in canines. Undersea Biomed res. 1990; 17(Suppl):64.
8. Van Allen CM, Hrdina LS, Clark J. Air embolism from the pulmonary vein. Arch Surg 1929;19:567-99.
9. Williams ST, Prior FG, Bryson P. Hematocrit change in tropical scuba divers. Wilderness Environ Med. 2007 Spring;18(1):48-53.
10. Gempp E, Blatteau JE, Pontier JM, Balestra C, Louge P. Preventive effect of pre-dive hydration on bubble formation in divers. Br J Sports Med. 2009 Mar;43(3):224-8.
11. Pendergast DR, Senf CJ, Fletcher MC, Lundgren CE. Effects of ambient temperature on nitrogen uptake and elimination in humans. Undersea Hyperb Med. 2015 Jan-Feb;42(1):85-94.
12. Hyldegaard O, Møller M, Madsen J. Protective effect of oxygen and heliox breathing during development of spinal decompression sickness. Undersea Hyperb Med. 1994 Jun;21(2):115-28.

Further Reading

- Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR, Kot J, Lafère P. Pre-hospital management of decompression illness: expert review of key principles and controversies. Diving Hyperb Med. 2018 Mar 31;48(1):45-55.
- Edmonds C, Bennett M, Lippmann J, Mitchell S, editors. Diving & Subaquatic Medicine. 5th ed. Boca Raton, FL: Taylor & Francis; 2016.
- Blake DF, Crowe M, Lindsay D, Brouff A, Mitchell SJ, Leggat PA, Pollock NW. Comparison of tissue oxygenation achieved breathing oxygen using different delivery devices and flow rates. Diving Hyperb Med. 2020 Mar 31;50(1):34-42.
- Lippmann J. Oxygen First Aid for Divers. Melbourne: Submariner Publications; 2016.

- Lippmann J, Bugg S. The Diving Emergency Handbook. 8 th ed. Melbourne: Submariner Publications; 2020.

About this Guideline

- Search date/s: 18 September 2020
- Question/PICO: P: Divers using compressed gas requiring (resuscitation OR first aid)
  - I: Any first aid or first responder intervention (out of hospital) other than standard CPR
  - C: Standard CPR
  - O: (Survival to discharge neurologically intact) OR (survival to admission to hospital) OR (return of spontaneous circulation)
  - S: Any study
  - T: 2000 - present
- Method: Evidence update following systematic review Mitchell et al 2018
- Primary reviewers: John Lippmann, Simon Mitchell, Michael Bennett
- Secondary review: Finlay Macneil
- Worksheet Hereco spreadsheet
- Approved: April 2021
- Guidelines superseded: ARC Guideline 9.3.5 - November 2011

AUSTRALIAN RESUSCITATION COUNCIL GUIDELINE 9.3.6 - COLD INJURY

Exposure to cold conditions can lead to generalised cooling of the body (hypothermia) or localised cold injury. The latter may be either Freezing Cold Injury (frostbite) or Non- Freezing Cold Injury (NFCI or trench foot).

FREEZING COLD INJURY

Frostbite results from the freezing of tissues causing ice crystal formation and blocking of small blood vessels. The areas most commonly affected are those exposed to cold, windy conditions (e.g. the face, inc. ears), and those with the most peripheral blood supply (e.g. fingers and toes). Frostbite can most simply and usefully be classified into superficial frostbite in which only the skin is frozen and can still be moved in relation to the underlying tissue; and deep frostbite which involves deeper tissues. The vast majority of cases that occur in Australia are of the superficial type.

Management of Superficial Frostbite

- Seek shelter. Get out of the cold and wind.
- DO NOT rub the frozen tissue.
- DO NOT use radiant heat to rewarm the part.
- Rewarm the affected part immediately by gently placing the affected fingers in the opposite armpit, or by placing a warm hand over a frostnipped cheek or ear. Feet can be reheated on the warm abdomen (under clothing) of a companion. Rewarming can be very painful.
- Ensure that re-freezing does not occur. Once the colour and consistency of the skin have been restored the person can safely resume normal activity provided they increase their insulation and are especially vigilant against recurrence.

Management of Deep Frostbite

- Seek shelter. Get out of the cold and wind.
- Remove constrictive or damp clothing if dry replacement clothing is available.
- Wrap in warm blankets and give warm fluids by mouth.
- If tissue is still frozen at time of presentation, the best tissue survivability results from placing the injured part in a warm water bath with circulating water (40-42°C, that is, comfortably hot to the back of a rescuer's hand) until

the affected part thaws. This may take 30 minutes or more. Such management is best achieved under hospital conditions where infection-control and adequate pain-relief can be provided.

- If tissue has spontaneously thawed by time of presentation (as is often the case) the 40- 42°C water bath is not required, but affected tissue can be cleaned and bathed at a more comfortable temperature (30-35°C). Rewarming can be very painful.
- Elevate the affected part.
- DO NOT use radiant heat to rewarm the part.
- DO NOT break blisters.
- NEVER thaw a part if there is any likelihood of it being refrozen. Thawing and refreezing results in far more tissue damage than leaving the tissue frozen for even several hours.

NON-FREEZING COLD INJURY

Prolonged exposure of limbs to low temperatures above freezing may lead to “trench foot” or “immersion foot”. When first seen, the injured part is pale, anaesthetic, pulseless and immobile, but not frozen. The pathophysiology is not fully understood. Although there is no formation of ice crystals in the tissue, the cold temperature alone appears to cause damage to nerves and to the lining of small blood vessels, leading to occlusion and stasis of blood flow.

Management of Non-Freezing Cold Injury

The treatment of Non-Freezing Cold Injury is still controversial, as no single treatment method has been demonstrated to result in better tissue survivability than other treatments.

- Dry the foot well. Keep the body warm but the foot cool.
- Do not let the victim walk on affected feet. Rather they should lie down with the feet elevated.
- DO NOT use radiant heat to rewarm the part.

Useful References:

1. Burr R. Trench Foot. J Wild Med 1993;4:348-352.
2. Francis TJR, Golden FStC. Non-freezing cold injury: the pathogenesis. J Roy Nav Med Serv 1985;71;3-8.
3. Granberg PO. Cold Injury. In: Chant AD, D’Sa AA, eds. Emergency Vascular Practice. London: Arnold, 1997:119-134.
4. McCauley RL, Smith DJ, Robson MC, Heggers JP. Frostbite and other cold-induced injuries. In Auerbach PS,ed. Wilderness Medicine, 3rd ed. St Louis: Mosby, 1995:129-145.
5. Mills WJ. Summary of the treatment of the cold injured patient-frostbite. 1993;Vol 35.1:61-66.
6. Riddell DI. A practical guide to local cold injuries. J Roy Nav Med Serv 1986;72:20-25.

7. Swedish National Board of Health and Welfare. Hypothermia - Cold-induced injuries. Stockholm: 1997: 1-110.

FURTHER READING

ANZCOR Guideline 9.3.3 Hypothermia: First-Aid Management

ANZCOR Guideline 9.4.1 - First Aid Management of Australian Snake Bite

Summary

Who does this guideline apply to?

This guideline applies to adults, children and infants.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. Send for an ambulance. [Good Practice Statement]
2. Keep the person immobilised (still), reassured and under constant observation. [Good Practice statement]
3. Apply pressure bandaging with immobilisation. [Good Practice Statement)
4. Sudden collapse with cardiac arrest requires immediate CPR. [Good Practice Statement]

Abbreviations

Abbreviation Meaning/Phrase

ANZCOR- Australian and New Zealand Committee on Resuscitation CPR- Cardiopulmonary Resuscitation CoSTR- Consensus on Science with Treatment Recommendations (from International Liaison Committee on Resuscitation - ILCOR)

Guideline

1 Introduction

Many of the snakes found in Australia are capable of lethal bites to humans. These include Taipans, Brown snakes, Tiger snakes, Death Adders, Black snakes, Rough Scaled snakes and many Sea snakes.[1]

There are no snakes native to New Zealand, but snake bite may rarely occur in New Zealand for example, in zoos or at ports.

Snakes produce venom in modified salivary glands and the venom is forced out under pressure through paired fangs in the upper jaw. Snake venoms are complex mixtures of many toxic substances which can cause a range of effects in humans.

The greatest threat to life and cause of over half of deaths is early cardiovascular collapse.[2] In the 16 years to 2016, 16 Australians were recorded as dying of snake bite in Australia.[3]

Other significant effects include:

- major bleeding due to inability to clot blood;
- nerve paralysis leading to respiratory muscle paralysis;
- muscle damage;
- kidney failure due to microscopic blood clots.

2 Recognition

The bite may be painless and without visible marks. Other symptoms and signs of a snake bite may include:

- paired fang marks, but often only a single mark or a scratch mark may be present; (localised redness and bruising are uncommon in Australian snake bite)
- headache;
- nausea and vomiting;
- abdominal pain;
- blurred or double vision, or drooping eyelids;
- difficulty in speaking, swallowing or breathing;
- swollen tender glands in the groin or armpit of the **bitten** limb;
- limb weakness or paralysis;
- respiratory weakness or respiratory arrest.

The most common cause of death from snake bite is collapse with cardiac arrest[2]. This can occur within 10 to 60 minutes of a bite with envenomation, is most often pre-hospital, and requires immediate CPR.

An occasional feature of a brown snake bite is initial collapse or confusion followed by apparent partial or complete recovery. It often occurs as the only finding after a bite from a brown snake and may be the only evidence of envenomation. This information may be useful when providing handover to the treating health practitioner who is considering administration of antivenom.

3 Management

If the person is unresponsive and not breathing normally, follow the ANZCOR Basic Life Support Flowchart and ANZCOR Guideline 8. If the person is unconscious and breathing normally, follow ANZCOR Guideline 3.

- Send for an ambulance for any person with a suspected snake bite;
- Keep the person immobilised (still), reassured and under constant observation;
- Apply pressure bandaging with immobilisation [Refer to ANZCOR Guideline 9.4.8];
- Commence CPR [Refer to ANZCOR Guideline 8] if person is unresponsive and not breathing normally. There is no risk of transmission of venom to rescuer by providing CPR.

Note:

- DO NOT cut or incise the bite
- DO NOT use an arterial tourniquet
- DO NOT wash or suck the bite

Snake identification

Many of Australia's snakes are protected species. It is strongly recommended that no attempts be made to kill the snake due to the risk of multiple bites or another person being bitten. A digital photograph of the snake may be helpful in identification if safe to do so.

Antivenom is available for all venomous snakes native to Australia, but must be given under health professional supervision in a properly equipped medical facility. Antivenom is not routinely available in New Zealand.

References

1. Sutherland SK, Tibballs J. Australian Animal Toxins. Melbourne: Oxford University Press 2001.
2. Johnston CI, Ryan NM, Page CB, Buckley NA, Brown SG, O'Leary MA, Isbister GK. The Australian Snakebite Project, 2005-2015 (ASP-20). MJA. 2017;207: 119-125.
3. Welton, RE, Liew, D and Braitberg, G. (2017). Incidence of fatal snake bite in Australia: A coronial based retrospective study (2000-2016). Toxicon. 131:11-15.
4. Avau B, Borra V, Vandekerckhove P, De Buck E. The treatment of snake bites in a first aid setting: a systematic review. PLoS neglected tropical diseases. 2016 Oct 17;10(10):e0005079.

Further Reading

- ANZCOR Guideline 8 Cardiopulmonary Resuscitation
- ANZCOR Guideline 3 Recognition and First Aid Management of the Unconscious Victim
- ANZCOR Guideline 9.4.8 Envenomation - Pressure Immobilisation Technique

- 'A Clinician's Guide to Australian Venomous Bites and Stings' (Prof Julian White) [BioCSL 2013] Available at https://biomedicalsciences.unimelb.edu.au/__data/assets/pdf_file/0004/3216739/A-Clinicians-Guide-to-Venomous-Bites-and-Stings-2013.pdf

Rationale For Pressure Bandaging with Immobilisation

Most snake venom reaches the blood stream via the lymphatic system. Laboratory research has shown that very little venom reaches the circulation, even after several hours, if the pressure bandaging with immobilisation (PIB) is applied immediately and maintained. The correct application of the pressure bandage is difficult to achieve,^4 but its use is supported for the snakes encountered in the wild in Australia.

About this Guideline

Search date/s May 2020

Question/PICO: For the population (P), studies concerning people with snakebites or healthy volunteers with 'mock' snakebites were included. The interventions (I) that were included in this systematic review were interventions for the first aid management of snakebites that can be applied by lay- people without medical background. We excluded interventions for the management of snake-bites that are not feasible to be performed in a first aid setting where laypeople are the first aid providers. We selected studies that compared (C) the interventions to any other first aid intervention or no intervention. Concerning the outcomes (O), we included (1) survival, functional recovery, pain, complications, time to resumption of usual activity, restoration of the pre-expo- sure condition, time to resolution of symptoms or other health outcome measures (including adverse effects) for studies involving snakebite victims, (2) spread of mock venom for studies investigating the efficacy of pressure immobilization and (3) quality of the bandage applied and tension generated for studies investigating the feasibility of pressure immobilization.

Method: Evidence update, not scoping or systematic review. The PICO shown above was rerun in Pubmed, Cochrane, Embase and Medline databases in May 2020, but search extended to include papers using viper instead of snake and snake bite as well, yielding an extra 186 papers since 2016. The papers found and notes are shown in the attached Excel spreadsheet. The details of the searches are in the attached worksheet.

Primary reviewers: Natalie Hood, Finlay Macneil

Other consultation: Geoff Isbister, Jim Tibballs

Worksheet Evidence update from SR by Avau et al, 2016 - May 2020

Approved: April 2021

Guideline superseded: ANZCOR Guideline 9.4.1 - March 2020

ANZCOR Guideline 9.4.2 - First Aid Management of Spider Bite

Summary

Who does this guideline apply to?

This guideline applies to adults, children and infants.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid training providers.

Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

1. If serious symptoms or signs develop from any spider bite, transport the person to hospital.
2. If funnel-web spider bite is suspected:
    - Send for an ambulance. [Good Practice Statement]
    - Keep the person immobilised (still), reassured and under constant observation. [Good Practice Statement]
    - Apply pressure bandaging with immobilisation. [Good Practice Statement)
    - Sudden collapse with cardiac arrest requires immediate CPR. [Good Practice Statement]
3. If other spider bite, apply ice pack for pain relief, transport to medical care if unwell. [Good Practice Statement]

Abbreviations

Abbreviation Meaning/Phrase

- ANZCOR- Australian and New Zealand Committee on Resuscitation
- CPR- Cardiopulmonary Resuscitation

Guideline

1 Introduction

The bites of many different Australian spiders may cause pain but only bites from some funnel-web spiders are an immediate threat to life, although the Redback spider bite may be a threat to life in the very young or very old. There are no spiders native to New Zealand that are considered a threat to life, however, Australian Redback spiders may be encountered in some areas of New Zealand.

If serious symptoms or signs develop from any spider bite, transport the person to hospital.

2 Funnel-web Spiders

A bite from a large (> 2cm), dark-coloured spider ('big black spider'), especially in the regions of Sydney, Blue Mountains, central, northern, southern highlands or south coast of NSW, or south-eastern Queensland, should be considered a dangerous bite and immediate treatment given.[1,2,3]

Sydney funnel web spider, Atrax robustus, Photo copyright Prof Julian White

Recognition

Symptoms and signs of funnel Web spider bite may include:

- pain at the bite site, but little local reaction
- tingling around the mouth
- profuse sweating
- copious secretion of saliva
- abdominal pain
- muscular twitching (called fasciculation)
- breathing difficulty
- confusion leading to unconsciousness

Note: Life threatening effects may occur within 10 minutes.

Management

The rescuer should:

- Send for an ambulance [Good Practice Statement]
- Apply pressure bandage with immobilisation and immobilize the person immediately [see Refer to ANZCOR Guideline 9.4.8] [Good Practice Statement]

Attempts to capture the spider may result in further bites. Do not bring a live spider to hospital. A photo on a phone may help in identification but should not delay the first two steps of management. [Good Practice Statement]

If the person is unresponsive and not breathing normally, follow Australian Resuscitation Council and New Zealand Resuscitation Council Basic Life Support Flowchart [Refer to ANZCOR Guideline 8].

Note: Antivenom is available for treatment of funnel-web spider envenomation in areas where these spiders are encountered in Australia.

3 Redback Spider

This spider (approximately 1cm body length) has a characteristic red, orange or pale stripe on the back of its abdomen. A bite may threaten the life of a child, but apart from pain, is rarely serious for an adult.[1]

Red back spider, Latrodectus hasseltii, Photo copyright Prof Julian White

Recognition

Symptoms and signs may include:

- immediate pain at the bite site which becomes hot, red and swollen
- intense local pain which increases and spreads
- nausea, vomiting and abdominal pain
- profuse sweating, especially at the bite site
- swollen tender glands in the groin or armpit of the envenomated limb.

Management

The first aider should:

- keep the person under constant observation
- apply an ice or cold compress to lessen the pain (for periods of no longer than 20mins)
- transport the person to a medical facility, preferably by ambulance, if the person is a young child or collapse occurs or pain is severe. [Good Practice Statement]

Note:

Local pain develops rapidly at the bite site and may become widespread, but the venom acts slowly so a serious illness is unlikely in less than 3 hours. Pain can be treated with antivenom3,4,5,8 in a hospital where resuscitation facilities are available. [Good Practice Statement] A related species, the Cupboard Spider (resembles the redback spider without the stripe) may be treated with the Redback spider antivenom.1,5 [Good Practice Statement]. The Pressure Immobilisation Technique is **not** used because the venom acts slowly and any attempt to slow its movement tends to increase local pain.

4 White-Tailed Spider Bite

Although the bite of the White-tailed spider may cause severe inflammation,[7] contrary to popular opinion it has caused very few cases of severe local tissue destruction.[1,7] An ice pack may be used to relieve pain. [Good Practice Statement]

White-tailed spider, Photo copyright Prof Julian White

The Pressure Immobilisation Technique should **not** be used. [Good Practice Statement]

5 Other Australian Spider Bites

All other spider bites should be treated symptomatically: apply ice or cold compress to lessen the pain. [Good Practice Statement]

6 Spider Bites in New Zealand

Spider bites in New Zealand are much rarer than Australia and only the Katipo spider is considered dangerous to people; but this spider is not aggressive and rarely bites humans.[9] However, the more aggressive Australian redback spider has been introduced into New Zealand and populations of the spider are described in Central Otago and New Plymouth and these bites may be encountered. Spider bites in New Zealand should be treated symptomatically: apply ice or cold compress to lessen the pain.[9] If redback spider bite is suspected in the young or elderly, send for an ambulance. [Good Practice Statement]

References

1. Sutherland SK, Tibballs J. Australian Animal Toxins, Oxford University Press, Melbourne. 2001
2. Isbister GK, Gray MR, Balit CR et al. Funnel-web spider bite: a systematic review of recorded clinical cases. Med J Aust 2005; 182: 407-11.
3. White J and Limited C. (2013). A clinician's guide to Australian venomous bites and stings: Incorporating the Updates CSL Antivenom Handbook. CSL Limited; 2013
4. Ellis RM, Sprivulis PC, Jelinek GA, et al. A double-blind, randomized trial of intravenous versus intramuscular antivenom for Red-back spider envenoming. Emerg Med Aus 2005; 17: 152-156.4.
5. Isbister GK, Brown SGA, Miller M. et al. A randomised controlled trial of intramuscular vs. intravenous antivenom for lactrodectism- the RAVE study. Q J Med 2008; 101: 557-565.
6. Isbister GK, Gray MR. Effects of envenoming by comb-footed spiders of the genera *Steatoda* and *Achaearanea* (family Theridiidae: Araneae) in Australia. J Toxicol - Clin Toxicol 2003; 41: 809-819.
7. Isbister GK, Gray MR. White-tail spider bite: a prospective study of 130 definite bites by *Lampona* species. MJA 2003;179: 199- 203
8. Isbister GK, Page CB, Buckley NA, et al. Randomized controlled trial of intravenous antivenom versus placebo for latrodectism: the second Redback Antivenom Evaluation (RAVE-II) study. Annals of emergency medicine. 2014 Dec 1;64(6):620-8.
9. Derraik, JG, Sirvid, PJ and Rademaker, M. (2010). The first account of a bite by the New Zealand native spider Trite planiceps (Araneae: Salticidae). The New Zealand Medical Journal (Online). 123(1314).

Further Reading

- ANZCOR Guideline 8 Cardiopulmonary Resuscitation
- ARC Guideline 9.4.8 Envenomation - Pressure Immobilisation Technique

ANZCOR GUIDELINE 9.4.3 – ENVENOMATION FROM TICK BITES AND BEE, WASP AND ANT STINGS

Who does this guideline apply to?

This guideline applies to adults, children and infant victims.

Who is the audience for this guideline?

This guideline is for use by bystanders, first aiders and first aid providers.

1 Introduction

Single stings from a bee, wasp or ant, while painful, seldom cause serious problems except for persons who have an allergy to the venom. Multiple insect stings can cause severe pain and widespread skin reaction. Stings around the face can cause serious envenomation and difficulty breathing even if the person is not known to be allergic.

It is important to remember that bee stings with the venom sac attached continue to inject venom into the skin, whilst a single wasp or ant may sting multiple times.

Ticks can inject a toxin that may cause local skin irritation or a mild allergic reaction, however most tick bites cause few or no symptoms.

In susceptible people tick bite or other bites/stings may cause a severe allergic reaction or anaphylaxis, which can be life threatening. This can also occur in victims with no previous exposure or apparent susceptibility.

2 Recognition

Symptoms and signs may include:

2.1 Minor

- Immediate and intense local pain.
- Local redness and swelling.

2.2 Major/Serious

- Allergic reaction/anaphylaxis ANZCOR Guideline 9.2.7 First-Aid Management of Anaphylaxis.
- Abdominal pain and vomiting in the case of allergic reaction to insect venom ANZCOR Guideline 9.2.7 First-Aid Management of Anaphylaxis.

Airway obstruction may result from swelling of the face and tongue due to anaphylaxis, or from insect stings in or around the mouth. This may occur immediately or over several hours and always requires urgent medical care.

3 Management

- If the victim is unresponsive and not breathing normally, commence resuscitation, follow ANZCOR Guideline 8: Cardiopulmonary Resuscitation.
- If the victim has signs of anaphylaxis, follow ANZCOR Guideline 9.2.7 First-Aid Management of Anaphylaxis.
- In the case of a bee sting, remove the sting, by any means, as quickly as possible (1,2).
- In the case of tick bite, if there is no history of tick allergy, immediately remove the tick (3).
- If the victim has a history of tick allergy, the tick must be killed where it is, rather than removed (3).
- If in a remote location, consultation with healthcare professionals is recommended.
- Move victim to a safe place.
- Apply a cold compress to help reduce pain and swelling.
- Monitor the victim for signs of allergic reaction (difficulty speaking, breathing difficulties, collapse and generalized rash).
- Refer the victim to hospital if sting is to the face or tongue.

Instructional information regarding auto injectors can be accessed from the Australian Society of Clinical Immunology and Allergy's (ASCIA) webpage: http://www.allergy.org.au/health- professionals/anaphylaxis-resources.

Instructional information regarding killing and removing ticks can be accessed from the web link: Tick-induced Allergies Research and Awareness (TiARA) http://www.tiara.org.au

Acknowledgements

Australian Society of Clinical Immunology and Allergy (ASCIA)

Further Reading

ANZCOR Guideline 8: Cardiopulmonary Resuscitation

ANZCOR Guideline 9.2.7 First-Aid Management of Anaphylaxis

References

1. Australian Society of Clinical Immunology and Allergy (ASCIA) Allergic Reaction to Bites and Stings: http://www.allergy.org.au/patients/insect-allergy-bites-and-stings/allergic-reactions-to- bites-and-stings?highlight=WyJiZWUiXQ== Accessed 19 September 2016
2. Visscher PK, Vetter RS, Camazine S. Removing bee stings. *Lancet* 1996; **348** :301-02.
3. http://www.tiara.org.au/how-to-remove-a-tick/ Accessed 24 July 2018

GUIDELINE 9.4.5 ENVENOMATION - JELLYFISH STINGS

INTRODUCTION

The mechanism of jellyfish envenomation (1,2

Stinging by jellyfish is caused by the simultaneous discharge of many thousands of microscopic stinging capsules called nematocysts. These are located on the surface of tentacles and in some species on the body of a jellyfish. Nematocysts contain coiled threads (tubules) loaded with venom. Upon contact, the nematocysts 'discharge' their tubules into the victim's skin like mini-harpoons. The more tentacles which make skin contact, the more venom is injected.

Stings cause immediate, sharp pain and an acute inflammatory skin reaction at the sting site consisting of redness, wheal and swelling which may progress to local skin destruction. Some stings cause rapid collapse. In Australia, life-threatening stings generally occur in tropical areas, with few in southern regions. Because of their smaller body size, children are greater risk of the effects of envenomation.

Most stings are not serious and over-treatment of minor stings should be avoided. Wearing a full-body Lycra suit or equivalent provides good protection from stings. (4)

Tropical envenomations

Potentially fatal envenomation is caused by two jellyfish types in Australian waters.

1. Box Jellyfish. The Australian Box jellyfish, Chironex fleckeri, has a large (box-like) bell up to 20 x 30 cm and multiple tentacles. It inhabits estuarine and on-shore coastal waters. Contact with tentacles causes severe immediate pain and whip-like marks on the skin. A sting with several metres of tentacles can cause respiratory and cardiac arrest within a few minutes. Approximately 80 deaths have been recorded.
2. Jellyfish causing Irukandji syndrome Approximately 10 small to medium-sized offshore and onshore jellyfish [including Carukia barnes and species of the Carybdea, Malo , Alatina , Gerongia and Morbakka genera] are known or suspected to produce an "Irukandji syndrome". (5-8) These jellyfish have only 4 tentacles and some are too small to be seen by the victim.

A minor sting on the skin with no tentacle visible, is followed in 5-40 (typically 20-30) minutes by severe generalised pain (often cramping in nature), nausea and vomiting, difficulty breathing, sweating, restlessness and a feeling of “impending doom”. Victims may develop heart failure, pulmonary oedema and hypertensive stroke.

Prevention of further stinging by nematocyst inhibition

When a sting occurs, pieces of tentacles and non-discharged nematocysts may be left on the victim’s skin. In large or life-threatening stings it is important to inhibit non-discharged nematocysts so that subsequent handling or treatment does not cause further envenomation.

Nematocysts from different species of jellyfish are either inhibited or stimulated to discharge by different substances used for first-aid (1,2,3).

Vinegar

- Vinegar (4-6% acetic acid) inhibits nematocyst discharge of Box jellyfish(10) but does not provide pain relief from the venom already injected.
- Although not proven to inhibit nematocyst discharge of all jellyfish causing Irukandji syndrome, its use is considered good first-aid practice (1,2,3)
- Vinegar causes nematocyst discharge of some other jellyfish, including Physalia (“Bluebottle”)(5) and is therefore recommended only for tropical areas where Box jellyfish and Irukandji stings occur.

RECOGNITION

Since it is usually difficult to recognise which species of jellyfish has caused a sting, management is based on the risk of serious stings in the known geographical distribution of dangerous species. Jellyfish able to cause life-threatening stings primarily occur along the tropical coastline of Australia i.e. from Bundaberg (Queensland) northwards, across the northern coastline and down to Geraldton (Western Australia)(1,2).

Tentacles on the skin

- Long lengths of easily-visible large tentacles on the skin in association with severe pain should be regarded as Box jellyfish tentacles.
- In the setting of large numbers of blue jellyfish washed up on the beach or floating on the surface of the water, tentacles are probably from a Physalia species (“Bluebottle”).
- Tentacles from hundreds of other species of jellyfish in Australian waters are difficult to identify. Often no tentacles remain.

Skin markings

A variety of skin markings are associated with the stings of various jellyfish species and could include the following:

- an inconspicuous mark which may develop a red flare

- an inconspicuous mark with goose pimples or an orange-peel appearance
- an inconspicuous mark with profuse sweating only at the sting site
- an irregularly shaped blotchy wheal
- white wheals with a surrounding red flare
- multiple whip-like wheals on the skin or a “frosted ladder pattern” suggest a sting by a box jellyfish
- later blistering or darkening of the sting pattern.

Pain(

- skin pain is generally immediate and varies in intensity from mild irritation to very severe sharp or burning pain
- generalised muscle aches
- severe muscle cramps in the limbs, chest and abdomen.

Symptoms and signs of severe stings

- difficulty or cessation of breathing
- cardiac arrest
- severe pain
- restlessness and irrational behaviour
- nausea and vomiting, headache
- physical collapse
- profuse sweating, sometimes only in the sting area.

FIRST AID MANAGEMENT

- No one nationwide recommendation for first-aid can be made because of differences between jellyfish species around Australia.
- In most cases, first-aid providers are unlikely to be able to identify the jellyfish.
- In the tropics, because of the risk (even if small) that the victim has been stung by a potentially lethal jellyfish, the priority must be to preserve life. If the species causing the sting cannot clearly be identified as harmless, or due to a “Bluebottle”, it is safer to treat the victim with vinegar.
- Outside the tropics, where huge numbers of non-life-threatening stings occur, the primary objective is pain relief with heat or cold.

TROPICAL AUSTRALIA

- Remove the victim from the water and restrain if necessary.
- If victim has more than a localized single sting, or who looks/feels unwell, call an Ambulance (Dial Triple Zero - 000) and seek assistance from a lifesaver/lifeguard if available.
- Assess victim and commence resuscitation as necessary following the ARC BLS flow chart (Guideline 8).
- Liberally douse/spray the stung area with vinegar for 30 seconds to neutralise invisible stinging cells,(4) then pick off remaining tentacles.

- If the victim has clearly been stung by a “Bluebottle” (see above) and is assessed as having a localized sting, is stable and not requiring ambulance, vinegar should not be applied(9) and victim managed as per stings in non-tropical Australia.
- If vinegar is unavailable, pick off any tentacles (this is not harmful to the rescuer) and rinse the sting well with seawater.
- Apply a cold pack or ice in a dry plastic bag for analgesia.(12) Do not allow or apply fresh water directly onto the sting because it may cause discharge of undischarged nematocysts.
- Antivenom is available for Chironex fleckeri and other multi-tentacled box jellyfish stings. In tropical coastal areas, hospitals keep and ambulances carry antivenom.
- Patients who initially appear stable but experience severe symptoms in the following 30 minutes may be suffering Irukandji syndrome and need urgent medical care.

NON-TROPICAL AUSTRALIA

- Keep the victim at rest, reassure and keep under constant observation.
- Do not allow rubbing of the sting area.
- Pick off any tentacles (this is not dangerous to the rescuer) and rinse sting area well with seawater to remove invisible nematocysts.
- Place the victim’s stung area in hot water (no hotter than the rescuer can comfortably tolerate) for 20 minutes.(11)
- If local pain is unrelieved by heat, or if hot water is not available, apply a cold pack or ice in a dry plastic bag.(13)
- If pain persists or is generalised, if the sting area is large (half of a limb or more), or involves sensitive areas (eg the eye) call an ambulance (Dial Triple Zero – 000) and seek assistance from a lifesaver/lifeguard if available.

For advice concerning any marine envenomation contact Australian Venom Research Unit 1300 760 451 or Poisons Information Centre 13 11 26

LEVEL OF EVIDENCE

Level II (Hot water for non tropical blue bottle stings) and Level IV

CLASS OF RECOMMENDATION

Class B - Recommended

REFERENCES

1. Williamson JA, Callanan VI, Hartwick RF. (1980). Serious envenomation by the Northern Australian Box Jellyfish (Chironex fleckeri) Med J Aust 1 :13-15.
2. Sutherland SK, Tibballs J. Australian Animal Toxins, Oxford University Press, Melbourne, 2001.
3. Tibballs J (2006). Australian venomous jellyfish, envenomation syndromes, toxins and therapy. Toxicon 48: 830-859.

4. Fenner P (1991). Cubozoan jellyfish envenomation syndromes and their medical treatment in northern Australia. Hydrobiologia 216/217: 637-640.
5. Flecker H. (1952). Irukandji stings to North Queensland bathers without symptoms of wheals but with severe general symptoms. Med J Aust 2: 89-91.
6. Fenner PJ, Williamson JA, Burnett JW et al (1988). The “Irukandji syndrome” and acute pulmonary oedema. Med J Aust 149:150-155.
7. Fenner PJ. Hadok JC (2002). Fatal envenomation by jellyfish causing Irukandji syndrome. Med J Aust 177(7):362-3.
8. Little M. Pereira P. Mulcahy R. Cullen P. Carrette T. Seymour J. Severe cardiac failure associated with presumed jellyfish sting. Irukandji syndrome? (2003) Anaesth Intens Care. 31(6):642-7.
9. Birsa LM, Verity PG, Lee RF. (2010). Evaluation of the effects of various chemicals on discharge of and pain caused by jellyfish nematocysts. Comp Biochem Physiol, Part C: 151: 426-430.
10. Hartwick RF, Callanan VI, Williamson JA (1980). Disarming the box jellyfish. Nematocyst inhibition in Chironex fleckeri. Med J Aust 1: 15-20.
11. Loten C, Stokes B, Worsley D, Seymour JE, Simon Jiang S, Isbister GK. (2006). A randomised controlled trial of hot water (45° C) immersion versus ice packs for pain relief in bluebottle stings. Med J Aust 184: 329-333.
12. Currie BJ, Jacups SP. (2005). Prospective study of Chironex fleckeri and other box jellyfish stings in the “Top End” of Australia’s Northyern Territory. Med J Aust 183: 631-636.
13. Exton DR, Fenner PJ, Williamson JA. (1989). Cold packs: effective topical analgesia in the treatment of painful stings by Physalia and other jellyfish. Med J Aust 151: 625- 626.

FURTHER READING

ANZCOR Guideline 8: Cardiopulmonary Resuscitation

ANZCOR Guideline 9.4.6 Envenomation - Blue Ringed Octopus & Cone Shell

ANZCOR Guideline 9.4.7 Envenomation - Fish Stings

ANZCOR Guideline 9.4.8 Envenomation - Pressure Immobilisation Technique

GUIDELINE 9.4.6 ENVENOMATION - BLUE-RINGED OCTOPUS AND CONE SHELL

INTRODUCTION

Blue-ringed octopuses (Hapalochlaena spp) inhabit all Australian coastal waters and are often found in tidal pools. If handled, these small animals may inflict a potentially fatal bite, injecting venom stored in salivary glands.

Many species of cone shell (Conus spp) are found in tropical waters. They fire a dart-like barb to deliver venom when handled.

Although different, venoms from both these creatures can cause paralysis and death from respiratory failure within 30 minutes. This can be treated with Basic Life Support.

RECOGNITION

Symptoms and signs may include:

- a painless bite: a spot of blood visible
- numbness of lips and tongue
- the progressive weakness of muscles of respiration leading to inadequate or cessation of breathing.

MANAGEMENT

- Call an ambulance
- Keep the victim at rest, reassured and under constant observation.
- Use the Pressure Immobilisation Technique Guideline 9.4.8 if possible (Class B, Expert Consensus Opinion)
- Transport the victim to a medical facility, preferably by ambulance.

If the victim is unresponsive and not breathing normally, follow ANZCOR Guideline 8: Cardiopulmonary Resuscitation.

NOTE: Despite being unable to move, the victim may be able to hear spoken comments.

AUSTRALIAN VENOM RESEARCH UNIT

For urgent advice concerning any marine envenomation, call the Australian Venom Research Unit 24 hour advisory line. 1300 760 451

FURTHER READING

ANZCOR Guideline 5 Breathing

ANZCOR Guideline 6: Compressions

ANZCOR Guideline 8: Cardiopulmonary Resuscitation

ANZCOR Guideline 9.4.8 Envenomation - Pressure Immobilisation Technique

RATIONALE

Following envenomation by either the blue ringed octopus or cone shell, prompt application of the Pressure Immobilisation Technique will trap most of the venom at the bite site. When absorbed, the venoms cause muscle paralysis leading to breathing failure without direct effects on the heart. Paralysis my be long lasting (hours) and cardiopulmonary resuscitation must be continued until the patient is in the care of a health professional.

GUIDELINE 9.4.7 ENVENOMATION - FISH STINGS

INTRODUCTION

Many fish have spines with attached venom glands. When trodden upon, the spines of the marine Stonefish (Synanceia spp) and the freshwater Bullrout (Notesthes robusta) penetrate deeply and deposit venom causing excruciating pain. General cardiovascular toxic effects can occur but are rare. Handling these or similar fish is also potentially dangerous.

The barbed spines on the tails of stingrays can inflict a serious gash or penetrating stab injury with subsequent venom-induced tissue death. Organs and blood vessels may be damaged and fragments of spine may remain in the wound requiring surgical removal. Injuries usually occur when the victim stands on an unseen fish, pulls a captured fish into a boat or swims too closely over a fish on the sea-floor.

RECOGNITION

Symptoms and signs may include:

- intense pain, leading to irrational behaviour
- swelling
- sometimes a local grey/blue discolouration
- an open wound
- bleeding

MANAGEMENT

- Call an ambulance
- If the sting is to the trunk (chest, abdomen), assess the victim for signs of bleeding and treat as per ARC Guideline 9.1.1 Principles of Control of Bleeding for First Aiders
- If there is an embedded object (eg. a barb from a stingray sting), do not remove it as it may be plugging the wound and restricting bleeding. Place padding around or above and below the object and apply pressure over the pads.
- If the sting is to a limb, place the victim's stung hand or foot in hot water (no hotter than the rescuer can comfortably tolerate)1-8 (Class A, LOE IV).
- Transport the victim to a medical facility.

If the victim is unresponsive and not breathing normally, follow ANZCOR Guideline 8: Cardiopulmonary Resuscitation.

Note: DO NOT use the Pressure Immobilisation Technique. Antivenom is available for stonefish envenomation. (LOE IV, CLASS A)

AUSTRALIAN VENOM RESEARCH UNIT

For urgent advice concerning any marine envenomation you can call the Australian Venom Research Unit 24 hour advisory line 1300 760 451

RATIONALE

Whilst the mechanism is not understood, the local application of heat decreases pain in the majority of cases. The Pressure Immobilisation Technique is not used for fish stings because the venom remains localized at the wound

REFERENCES

1. Clark RF, Girard RH, Rao D, Ly BT, Davis DP. Stingray envenomations: a retrospective review of clinical presentation and treatment in 119 cases. J Emerg Med 2007; 33: 33-37.
2. Atkinson PR, Boyle A, Hartin D, McAuley D. Is hot water immersion an effective treatment for marine envenomation? Emerg Med J 2006; 23: 503-508.
3. Isbister GK. Venomous fish stings in tropical northern Australia. Am J Emerg Med 2001; 19: 561-565.
4. Aldred B, Erickson T, Lipscomb J. Lionfish envenomations in an urban wilderness. Wilderness Environ Med 1996; 7: 291-296.
5. Kizer KW, McKinney HE, Auerbach PS. Scorpaenidae envenomation. A five-year poison center experience. JAMA 1985; 253: 807-810.
6. Ngo SY, Ong SH, Ponampalam R. Stonefish envenomation presenting to a Singapore hospital. Singapore Mewd J 2009; 50: 506-9.
7. Satora L, Kuciel M, Gawlikowski T. Catfish stings and the venom apparatus of the African catfish Clarias gariepinus (Burchell, 1822), and stinging catfish Heteropneustes fossilis (Bloch, 1794). Ann Agric Environ Med 2008; 15: 163-8.
8. Grandcolas N, Galéa J, Ananda R. et al. Stonefish stings : difficult analgesia and notable risk of complications. [Article in French] Presse Med 2008; 37: 395-400.

FURTHER READING

ARC Guideline 8 Cardiopulmonary Resuscitation

ARC Guideline 9.1.1 Principles of Control of Bleeding for First Aiders

GUIDELINE 9.4.8 ENVENOMATION - PRESSURE IMMOBILISATION TECHNIQUE

INTRODUCTION

The pressure immobilisation technique (PIT) was introduced for the treatment of Australian snake bites(1) and is suitable for other elapid snake bites.(2) It is also recommended for envenomation by a number of other animals. The PIT retards the flow of lymph(3) by which venoms gain access to the circulation.

It has also been shown that there may be inactivation of certain venoms and venom components when the injected venom remains trapped in the tissues by the pressure bandage.(4)

USE OF THE PRESSURE IMMOBILISATION TECHNIQUE

The Pressure Immobilisation Technique (PIT) is recommended for application to bites and stings by the following creatures:

- All Australian venomous snakes, including sea snakes [Class A; LOE III]
- Funnel Web spider [Class A; LOE IV]
- Blue-ringed octopus [Class B; LOE Expert Consensus Opinion]
- Cone shell [Class B; LOE Expert Consensus Opinion]

The Pressure Immobilisation Technique is **NOT** recommended for the first aid management of:

- other spider bites including redback;
- jellyfish stings;
- fish stings including stonefish bites
- stings by scorpions, centipedes or beetles.

The evidence for PIT evolved from small-scale animal studies, and tracer studies in humans. No field based direct comparisons of first aid techniques in humans affected by snakebite exist. Where field based studies have reported outcomes after application of PIT no clear benefit has been shown. This may be confounded by poor quality PIT application, as most observational studies report inadequately applied PIT in the field.(5) It has not been shown clearly which component of the PIT inadequately applied (the local pressure, the full limb bandaging or the limb immobilisation) is potentially causative of the lack of observed clinical efficacy, if

not all three. There is insufficient evidence to determine which technique or method of bandage application is most effective in the field in minimizing venom absorption. Techniques reported include local pressure first then a fully limb encircling pressure bandage, or a limb encircling bandage only. Furthermore whilst commencing the encircling bandage distally and moving proximally may improve comfort and tolerance of the bandage, it may act to increase venom movement. Starting proximally and working distally may further minimise venom movement but may cause distal oedema /fluid retention and make the bandage too uncomfortable for prolonged use. Training (using manometer feedback) has been shown to improve the pressure achieved with PIT, and the use of elasticised bandages may also improve the pressure obtained in PIT application.5, 6 [Class A; LOE: III-2]

MANAGEMENT

If resuscitation is needed it takes precedence over the PIT (refer to ARC Guideline 8). However the resuscitation team should apply PIT as soon as possible to potentially minimise further venom flow.

If on a limb, apply a broad pressure bandage over the bite site as soon as possible. Elasticised bandages (10-15cm wide) are preferred over crepe bandages, if neither are available, clothing or other material should be used.(5) [Class A; LOE: III-2]The bandage should be firm and tight, you should be unable to easily slide a finger between the bandage and the skin.

In order to further restrict lymphatic flow and to assist in immobilisation of the limb, apply a further pressure bandage, commencing at the fingers or toes of the bitten limb and extending upward covering as much of the limb as possible.(3) [Class A; LOE: III-2] The bandage should be applied over existing clothing if possible. The purpose of this bandage is to further restrict lymphatic flow and assist immobilisation. (Alternatively, a single bandage may be used to achieve both pressure on the bite site and immobilisation of the limb. In this method, the bandage is initially applied to the fingers or toes and extended up the limb as far as possible including the bite site).4, 8 [Class A; LOE: Expert Consensus Opinion].

Splint the limb including joints on either side of the bite, to restrict limb movement. The splint material can be incorporated under the layers of the bandage. For the upper limb, use a sling. [Class A; LOE: Expert Consensus Opinion].

Keep the victim and the limb completely at rest. Bring transport to the victim if possible. Transport the victim to medical care, preferably by ambulance. If alone, the victim should apply the pressure immobilisation bandage as completely as possible over the bite site and affected limb. They should keep immobile until assistance arrives. If they are unable to obtain urgent help to come to them, then apply local pressure if possible, immobilisation is contraindicated and they should move themselves to seek urgent help. Do not remove the bandages or splints before evaluation in an appropriate hospital environment. [Class A; LOE: Expert Consensus Opinion]

If the bite is not on the limb, firm direct pressure on the bite site may be useful. Do not restrict breathing or chest movement and do not apply firm pressure to the neck or head. [Class A; LOE: Expert Consensus Opinion]

Note:

- DO NOT cut or excise the bitten area, or attempt to suck venom from the bite site.
- DO NOT wash the bitten area.
- DO NOT apply an arterial tourniquet. (Arterial tourniquets that cut off circulation to the limb, are potentially dangerous and are not recommended for any type of bite or sting in Australia)

REFERENCES

1. Sutherland SK, Coulter AR, Harris RD. Rationalisation of first-aid measures for elapid snakebite. Lancet 1979; 1 : 183-185.
2. Gauthier N, Thomas S. Consensus on ECC & CPR Science and Treatment Recommendations. First Aid worksheet 270: What is the safety, efficacy and feasibility of compressive wrapping for coral snake (elapid) envenomation? 2005: Retrieved 18 July 2011 from http://circ.ahajournals.org/content/2112/2022_suppl/b2011/suppl/DC2443.
3. Howarth DM, Southee AE, Whyte IM. Lymphatic flow rates and first-aid in simulated peripheral snake or spider envenomation. Medical Journal of Australia 1994; 161 : 695-700.
4. Sutherland SK, Tibballs J. Australian Animal Toxins. Melbourne: Oxford University Press 2001.
5. Canale E, Isbister GK, Currie BJ. Investigating pressure bandaging for snakebite in a simulated setting: bandage type, training and the effect of transport. Emergency Medicine Australasia 2009; 21 : 184-190.
6. Norris RL, Ngo J, Nolan K, Hooker G. Physicians and lay people are unable to apply pressure immobilization properly in a simulated snakebite scenario. Wilderness & Environmental Medicine 2005; 16 : 16-21.
7. Simpson ID, Tanwar PD, Andrade C, Kochar DK, Norris RL. The Ebbinghaus retention curve: training does not increase the ability to apply pressure immobilisation in simulated snake bite--implications for snake bite first aid in the developing world. Transactions of the Royal Society of Tropical Medicine & Hygiene 2008; 102 : 451- 459.
8. Sutherland SK, Hawdon GM, Winkel KD. First aid for snake bite in Australia Melbourne: Australian Venom Research Unit. The University of Melbourne 1999.

FURTHER READING

ARC Guideline 8 - Cardiopulmonary Resuscitation

Ireland G, Brown SGA, Buckley NA, Stormer J, Currie BJ, White J, et al. Changes in serial laboratory test results in snakebite patients: when can we safely exclude envenoming? Medical Journal of Australia, 2010;193:285-90.

Anker RL, Straffon WG, Loiselle DS and Anker KM, Retarding the uptake of "mock venom" in humans: comparison of three first-aid treatments. Medical Journal of Australia 1982: 1(5);212-

Bush SP, Green SM, Laack TA, Hayes WK, Cardwell MD and Tanen DA, Pressure immobilization delays mortality and increases intra-compartmental pressure after artificial intramuscular rattlesnake envenomation in a porcine model. Annals of Emergency Medicine 2004: 44(6);599-

German BT, Hack JB, Brewer K and Meggs WJ, Pressure-immobilization bandages delay toxicity in a porcine model of eastern coral snake (Micrurus fulvius fulvius) envenomation. Annals of Emergency Medicine 2005: 45(6);603-

Meggs WJ, Courtney C, O'Rourke D and Brewer KL, Pilot studies of pressure-immobilization bandages for rattlesnake envenomations. Clinical Toxicology: The Official Journal of the American Academy of Clinical Toxicology & European Association of Poisons Centres & Clinical Toxicologists 2010: 48(1);61-

Pe T, Mya S, Myint AA, Aung NN, Kyu KA and Oo T, Field trial of efficacy of local compression immobilization first-aid technique in Russell's viper (Daboia russelii siamensis) bite patients. Southeast Asian Journal of Tropical Medicine & Public Health 2000: 31(2);346-

Sutherland SK and Coulter AR, Early management of bites by the eastern diamondback rattlesnake (Crotalus adamanteus): studies in monkeys (Macaca fascicularis). American Journal of Tropical Medicine & Hygiene 1981: 30(2);497-

Sutherland SK, Coulter AR and Harris RD, Rationalisation of first-aid measures for elapid snakebite. Lancet 1979: 1(8109);183-

Pe T, Muang Muang T, Myint Myint T, Aye Aye M, Kyaw M and Thein T, The efficacy of compression immobilization technique in retarding spread of radio-labeled Russell's viper venom in rhesus monkeys and 'mock venom' NaI131 in human volunteers. Southeast Asian Journal of Tropical Medicine & Public Health 1994: 25(2);349-

Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Treatment of Acute Altitude Illness: 2019 Update

Andrew M.LuksMD[1] Paul S.AuerbachMD, MS[2] LuanneFreerMD[345] Colin K.GrissomMD[67] Linda E.KeyesMD[89] Scott E.McIntoshMD, MPH[10] George W.RodwayPhD, APRN[11] Robert B.SchoeneMD[12] KenZafrenMD[213] Peter H.HackettMD[14]

[1] *Division of Pulmonary, Critical Care and Sleep Medicine, University of Washington, Seattle, WA*

[2] *Department of Emergency Medicine, Stanford University School of Medicine, Stanford, CA*

[3] *Yellowstone National Park, WY*

[4] *Midway Atoll National Wildlife Refuge, Honolulu, HI*

[5] *Everest ER, Himalayan Rescue Association, Kathmandu, Nepal*

[6] *Division of Pulmonary and Critical Care Medicine, Intermountain Medical Center, Salt Lake City, UT*

[7] *Division of Pulmonary and Critical Care Medicine, University of Utah, Salt Lake City, UT*

[8] *Department of Emergency Medicine, University of Colorado, Denver, CO*

[9] *Boulder Community Health, Boulder, CO*

[10] *Division of Emergency Medicine, University of Utah, Salt Lake City, UT*

[11] *University of California, Davis School of Nursing, Sacramento, CA*

[12] *Division of Pulmonary and Critical Care Medicine, Sound Physicians, St. Mary's Medical Center, San Francisco, CA*

[13] *Himalayan Rescue Association, Kathmandu, Nepal*

[14] *Altitude Research Center, Division of Pulmonary Sciences and Critical Care Medicine, Department of Medicine, University of Colorado Anschutz Medical Campus, Aurora, CO*

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To provide guidance to clinicians about best preventive and therapeutic practices, the Wilderness Medical Society (WMS) convened an expert panel to develop evidence-based guidelines for prevention and treatment of acute mountain sickness, high altitude cerebral edema, and high altitude pulmonary edema. Recommendations are graded based on the quality of supporting evidence and the balance between the benefits and risks/burdens according to criteria put forth by the American College of Chest Physicians. The guidelines also provide suggested approaches to prevention and management of each form of acute altitude illness that incorporate these recommendations. This is an updated version of the original WMS Consensus Guidelines for the Prevention and Treatment of Acute Altitude Illness published in 2010 and subsequently updated as the WMS Practice Guidelines for the Prevention and Treatment of Acute Altitude Illness in 2014.

Keywords

high altitude
acute mountain sickness
high altitude pulmonary edema
high altitude cerebral edema
acetazolamide
dexamethasone
nifedipine

Introduction

Travel to elevations above 2500 m is associated with risk of developing 1 or more forms of acute altitude illness: acute mountain sickness (AMS), high altitude cerebral edema (HACE), and high altitude pulmonary edema (HAPE). Because large numbers of people travel to such elevations, many clinicians are faced with questions from patients about the best means to prevent these disorders. In addition, clinicians working at facilities in high altitude regions or as members of expeditions traveling to such areas can expect to see persons who are experiencing these illnesses and must be familiar with prophylactic regimens and proper treatment protocols.

To provide guidance to clinicians and disseminate knowledge about best practices, the Wilderness Medical Society (WMS) convened an expert panel to develop evidence-based guidelines for prevention and treatment of acute altitude illness. Preventive and therapeutic modalities are presented and recommendations made for each form of acute altitude illness. Recommendations are graded based on the quality of supporting evidence and consideration of benefits and risks/burdens associated with each modality. These recommendations are intended to apply to all travelers to high altitude, whether they are traveling to high altitude for work, recreation, or various activities including hiking, skiing, trekking, and mountaineering.

Methods

The original expert panel was convened at the 2009 annual meeting of the WMS in Snowmass, Colorado. Members were selected by the WMS based on their clinical and/or research experience. Relevant articles were identified through the MEDLINE database by keyword search using the terms acute mountain sickness, high altitude pulmonary edema, high altitude cerebral edema, treatment, prevention, acetazolamide, dexamethasone, ibuprofen, nifedipine, tadalafil, sildenafil, and salmeterol. English-language, peer-reviewed studies including adults and/or children that were related to prevention and treatment of acute altitude illnesses, including randomized controlled trials, observational studies, and case series, were reviewed, and the level of evidence supporting various preventive and treatment modalities was assessed. Animal studies and abstract-only studies were not included. Conclusions from review articles were not considered in the formulation of recommendations but are cited to provide background information on the acute altitude illnesses and their management. The panel used a consensus approach to develop recommendations and graded each recommendation according to criteria stipulated in the American College of Chest Physicians statement on grading recommendations and strength of evidence in clinical guidelines (online Supplementary Table 1).[1]

This set of guidelines is an updated version of the original Wilderness Medical Society Consensus Guidelines for the Prevention and Treatment of Acute Altitude Illness published in 2010[2] and the update as the Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Acute Altitude Illness published in 2014.[3] As for the 2014 update, the panel used the approach described to identify relevant studies, adding additional search terms to reflect updates in the literature. The new search terms for the current version included budesonide, acetaminophen, continuous positive airway pressure (CPAP), and hypoxic tents.

Defining the threshold for “high altitude” and when to apply these guidelines

Unacclimatized individuals are at risk of high altitude illness when ascending to altitudes above 2500 m. Prior studies and extensive clinical experience, however, suggest that susceptible individuals can develop AMS, and potentially HAPE, at elevations as low as 2000 m.[4, 5, 6] HACE is typically encountered at higher elevations but has also been reported at around 2500 m in patients with concurrent HAPE.[7] Part of the difficulty in defining a specific threshold at which altitude illness can develop is the fact that the symptoms and signs of AMS, the most common form of altitude illness, are nonspecific, as demonstrated in several studies in which participants met criteria for the diagnosis of AMS despite no gain in altitude.[8, 9, 10] As a result, studies assessing AMS incidence at modest elevations may label individuals as having altitude illness when, in fact, symptoms are related to some other process, thereby falsely elevating the reported incidence of AMS at that elevation.

Recognizing the difficulty in defining a clear threshold, the expert panel recommends an approach to preventing and treating acute altitude illness that does not depend strictly on the altitude to which an individual is traveling. Preventive measures should be considered based on the altitude to which the individual is traveling and also account for factors such as history of performance at high altitude, rate of ascent, and availability of acclimatization days (described in greater detail later). Diagnoses of AMS, HAPE, or HACE should not be excluded based on the fact that an ill individual is below 2500 m. These diagnoses should be strongly considered in the presence of compatible clinical features, with careful attempts to exclude other entities such as severe dehydration, hyponatremia, pneumonia, carbon monoxide poisoning, and hypoglycemia.

Acute mountain sickness and high altitude cerebral edema

Information on the epidemiology, clinical presentation, and pathophysiology of AMS and HACE is provided in several extensive reviews.[11], [12], [13], [14] From a clinical standpoint, HACE represents an extremely severe form of AMS; therefore, preventive and treatment measures for the 2 disorders can be addressed simultaneously.

PREVENTION

Measures considered for prevention of AMS and HACE include the following.

Gradual ascent

Controlling the rate of ascent, in terms of the number of meters gained per day, is a highly effective means of preventing acute altitude illness; however, aside from 2 recent prospective studies,[15], [16] this strategy has largely been evaluated retrospectively.[17] In planning the rate of ascent, the altitude at which someone sleeps is considered more important than the altitude reached during waking hours.

Recommendation. Gradual ascent, defined as a slow increase in sleeping elevation, is recommended for AMS and HACE prevention. A specific approach is described further later in the text. Recommendation Grade: 1B

Acetazolamide

Multiple trials have established a role for acetazolamide in prevention of AMS.[18], [19], [20], [21]

Acetazolamide contains a sulfa moiety but carries an extremely low risk of inciting an allergic reaction in persons with sulfonamide allergy. As a result, persons with known allergy to sulfonamide medications can consider a supervised trial of acetazolamide before the trip, particularly if planning travel to a location remote

from medical resources.[22] Prior anaphylaxis to a sulfonamide medication or a history of Stevens-Johnson syndrome should be considered a contraindication to acetazolamide.

Some studies suggest that acetazolamide may have an adverse effect on maximum exercise capacity,[23] perceived dyspnea during maximal exercise tests,[24] and respiratory muscle function at high levels of work.[25] The small observed changes, however, are unlikely to affect overall exercise performance for the majority of activities in which high altitude travelers engage (hiking, skiing) or the chance of summit success for climbers at moderate and even extreme elevations. These changes should not be viewed as a reason to avoid acetazolamide.

The recommended adult dose for prophylaxis is 125 mg every 12 h (Table 1). Although doses up to 750 mg daily are effective at preventing AMS compared to placebo, they are associated with more frequent and/or pronounced side effects, do not convey greater efficacy, and are not recommended for prevention. A recent, small study suggested that 62.5 mg every 12 h was noninferior to 125 mg every 12 h,[26] but further research with greater numbers of participants in different high altitude settings should be completed before a change in dose can be recommended. The pediatric dose of acetazolamide is 2.5 $mg \cdot kg^{-1} \cdot dose^{-1}$ (maximum 125 $mg \cdot dose^{-1}$) every 12 h.[27]

Table 1. Recommended dosages for medications used in the prevention and treatment of altitude illness

Medication	*Indication*	*Route*	*Dosage*
Acetazolamide	AMS, HACE prevention	Oral	125 mg every 12 h[a] Pediatrics: 2.5 mg·kg^{-1} every 12 h
	AMS treatment[b]	Oral	250 mg every 12 h Pediatrics: 2.5 mg·kg^{-1} every 12 h (maximum: 125 mg per dose)
Dexamethasone	AMS, HACE prevention	Oral	2 mg every 6 h or 4 mg every 12 h[a] Pediatrics: Should not be used for prophylaxis
	AMS, HACE treatment	Oral, IV, IM	AMS: 4 mg every 6 h HACE: 8 mg once, then 4 mg every 6 h Pediatrics: 0.15 mg·kg^{-1}·dose^{-1} every 6 h (Maximum: 4 mg per dose)
Ibuprofen	AMS prevention	Oral	600 mg every 8 h
Nifedipine	HAPE prevention	Oral	30 mg ER version, every 12 h or 20 mg ER version every 8 h[c]
	HAPE treatment	Oral	30 mg ER version, every 12 h or 20 mg ER version every 8 h
Tadalafil	HAPE prevention	Oral	10 mg every 12 h[c]
Sildenafil	HAPE prevention	Oral	50 mg every 8 h[c]

AMS, acute mountain sickness; HACE, high altitude cerebral edema; IM, intramuscularly; ER, extended release; HAPE, high altitude pulmonary edema.

a

For individuals ascending to and remaining at a given elevation, after arrival at the target elevation, the medication should be continued for 2 d in individuals adhering to the recommended ascent rate and 2 to 4 d in individuals ascending faster than recommended rates. Individuals who ascend to a target elevation and immediately descend can stop the medication once descent is initiated.

b

Acetazolamide can also be used at this dose as an *adjunct* to dexamethasone in HACE treatment, but dexamethasone remains the primary treatment for HACE.

c

For individuals ascending to and remaining at a given elevation, after arrival at the target elevation, the medication should be continued for 4 d in individuals adhering to the recommended ascent rate and 4 to 7 d in individuals

ascending faster than recommended rates. Individuals who ascend to a target elevation and immediately descend can stop the medication once descent is initiated.

Recommendation. Acetazolamide should be strongly considered in travelers at moderate or high risk of AMS with ascent to high altitude. Recommendation Grade: 1A.

Recommendation. Acetazolamide can be used in children for prevention of AMS. Recommendation Grade: 1C.

Dexamethasone

Although dexamethasone does not facilitate acclimatization like acetazolamide, prospective trials have established a benefit for dexamethasone in AMS prevention.[28], [29] The recommended adult doses are 2 mg every 6 h or 4 mg every 12 h. Very high doses (4 mg every 6 h) may be considered in very high-risk situations, such as military or search and rescue personnel being airlifted to altitudes >3500 m with immediate performance of physical activity, but should not be used except in these limited circumstances. Prolonged use carries a risk of adrenal suppression. Although some resources state that use of less than 2-wk duration does not require a taper,[30] in remote mountain environments a more conservative approach is warranted. If used for longer than 10 d, the medication should be tapered over a 1-wk period rather than stopped abruptly. Given the absence of data on the use of dexamethasone for AMS prevention in children and the availability of other safe alternatives—specifically, graded ascent and acetazolamide—dexamethasone is not recommended for AMS prevention in children.

Recommendation. Dexamethasone can be used as an alternative to acetazolamide for adult travelers at moderate or high risk of AMS. Recommendation Grade: 1A.

Inhaled budesonide

Two studies indicated that inhaled budesonide 200 micrograms twice daily was effective at preventing AMS when compared to placebo.[31], [32] These studies were limited by methodological issues such as timing of the assessment for AMS[31] and number of participants in each study arm.[32] A clear mechanism of action was not apparent in these studies, but small improvements in spirometry and oxygen saturation—both of little clinical significance—were suggested as evidence that the benefit might derive from a direct lung effect. More recent, well-designed randomized controlled trials failed to replicate these results.[33], [34]

Recommendation. Inhaled budesonide should not be used for altitude illness prophylaxis. Recommendation Grade: 1C

Ginkgo biloba

Although 2 trials demonstrated a benefit of *Ginkgo* in AMS prevention,[35, 36] 2 other negative trials have also been published.[37, 38] This discrepancy may result from differences in the source and composition of the *Ginkgo* products.[39] *Ginkgo* should be avoided in pregnant women[40] and used with caution in people taking anticoagulants.[41] Acetazolamide is considered far superior for AMS prevention.

Recommendation. *Ginkgo biloba* should not be used for AMS prevention. Recommendation Grade: 1C

Ibuprofen

Two trials demonstrated that ibuprofen (600 mg 3 times daily) is more effective than placebo at preventing AMS,[42, 43] while a third, smaller study showed no benefit.[44] Another study claimed to show benefit, but the trial did not include a placebo arm and instead compared the incidence of AMS with ibuprofen with historically reported rates from the region in which the study was conducted.[45] Although no studies have compared ibuprofen with dexamethasone, 2 studies have compared ibuprofen with acetazolamide. The first found an equal incidence of high altitude headache and AMS in the acetazolamide and ibuprofen groups, with both showing significant protection compared to placebo.[46] A more recent trial failed to show that ibuprofen was noninferior to acetazolamide (ie, ibuprofen is inferior to acetazolamide for AMS prophylaxis).[47] The aforementioned trials all used the medication for a short duration (~24 to 48 h). As a result, efficacy and safety (eg, the risk of gastrointestinal bleeding or renal dysfunction) over longer periods of use at high altitude remain unclear. For these reasons, as well as more extensive clinical experience with acetazolamide and dexamethasone, ibuprofen cannot be recommended over these medications for AMS prevention for rapid ascent.

Recommendation. Ibuprofen can be used for AMS prevention in persons who do not wish to take acetazolamide or dexamethasone or have allergies or intolerance to these medications. Recommendation Grade: 2B.

Acetaminophen

A single study demonstrated that acetaminophen 1000 mg 3 times daily was as effective as ibuprofen at preventing AMS in trekkers travelling between 4370 and 4940 m in elevation.[45] Rather than including a placebo arm, the study attempted to establish the benefit of acetaminophen by comparing the incidence rates in the study with those of untreated trekkers from prior studies that used the same ascent profile. Based on these data, acetaminophen is not recommended for use as a preventive agent over acetazolamide or dexamethasone.

Recommendation. Acetaminophen should not be used for AMS prevention. Recommendation Grade: 1C

Staged ascent and preacclimatization

Two studies showed that spending 6 to 7 d at moderate altitude (~2200 to 3000 m) before proceeding to higher altitude (referred to as “staged ascent”) decreases the risk of AMS, improves ventilation and oxygenation, and blunts the pulmonary artery pressure response after subsequent ascent to 4300 m.[16], [48] Many travelers to high altitude visit mountain resorts at more moderate elevations between 2500 and 3000 m. The value of short stays at intermediate elevations of ~1500 m for decreasing the risk of AMS during such ascents makes sense from a physiologic standpoint. However, this approach has not been studied in a randomized fashion, aside from 1 cross-sectional study finding a decreased risk of AMS in travelers who spent 1 night at 1600 m before ascent to resort communities between 1920 and 2950 m.[5]

A larger number of studies examining the effects of repeated exposures to hypobaric or normobaric hypoxia in the days and week preceding high altitude travel (referred to as “preacclimatization”) showed mixed results, with some studies finding benefit in terms of decreased AMS incidence or severity[49], [50], [51] and others showing no effect.[52], [53], [54], [55] A significant challenge in interpreting the literature on preacclimatization is the variability among the hypoxic exposure protocols used, as well as the fact that not all studies include evidence that their protocols induced physiologic responses consistent with acclimatization.

Implementation of either staged ascent or preacclimatization may be logistically difficult for many high altitude travelers. In general, short-term exposures (eg, 15 to 60 min of exposure to hypoxia, or a few hours of hypoxia a few times before ascent) are unlikely to aid acclimatization, whereas longer exposures (eg, >8 h daily for >7 d) are more likely to yield benefit. Hypobaric hypoxia is more effective than normobaric hypoxia in facilitating preacclimatization and preventing AMS.[56] Because the optimal methods for preacclimatization and staged ascent have not been fully determined, the panel recommends consideration of these approaches but does not endorse a particular protocol.

Recommendation. When feasible, staged ascent and preacclimatization can be considered as a means for AMS prevention. Recommendation Grade: 1C

Hypoxic tents

Commercial products are available that allow individuals to sleep or exercise in hypoxic conditions for the purpose of facilitating acclimatization before a trip to high altitude. Only 1 placebo-controlled study has examined their utility.[57] Although this study demonstrated a lower incidence of AMS in persons who slept in simulated high altitude conditions compared to normoxia, technical difficulties with the system resulted in a substantial number of study participants not receiving the intended hypoxic dose. Although the systems are marketed to be of benefit and anecdotal reports suggest they are widely used by climbers and other athletes competing at high altitude, there are no data indicating increased likelihood of summit success or improved physical performance. As with the preacclimatization approaches previously described, any benefit that may accrue from these systems is more likely with long hypoxic exposures (>8 h per day) for at least several weeks before planned high altitude travel. Short and/or infrequent exposures, including exercise training, are likely of no benefit. In addition to the

cost of the systems and power needed to run them, individuals face the risk of poor sleep, which over a long period of time could have deleterious effects on performance during an expedition.

Recommendation. Hypoxic tents can be used for facilitating acclimatization and preventing AMS, provided sufficiently long exposures can be undertaken regularly over an appropriate number of weeks and other factors, such as sleep quality, are not compromised. Recommendation Grade: 2B

Other options

Chewed coca leaves, coca tea, and other coca-derived products are commonly recommended for travelers in the Andes mountains for AMS prevention. Their utility in prevention of altitude illness has not been properly studied, so they should not be substituted for other established preventive measures described in these guidelines. Multiple studies have sought to determine whether other agents, including antioxidants,[58] iron,[59] dietary nitrates,[60] leukotriene receptor blockers,[61, 62] phosphodiesterase inhibitors,[63] salicylic acid,[64] spironolactone,[65] and sumatriptan[66] can prevent AMS, but the current state of evidence does not support their use. “Forced” or “over” hydration has never been found to prevent altitude illness and might increase the risk of hyponatremia; however, maintenance of adequate hydration is important because symptoms of dehydration can mimic those of AMS. Nocturnal expiratory positive airway pressure (EPAP) administered via a single-use nasal strip during sleep is not effective for AMS prophylaxis,[67] nor is a regimen of remote ischemic preconditioning.[68]

No studies have examined short-term oxygen use in the form of either visits to oxygen bars or over-the-counter oxygen delivery systems by which individuals inhale oxygen-enriched gas from a small prefilled canister. Due to the small volume of gas (2 to 10 L/canister) and short duration of administration, these interventions are unlikely to be of benefit and, as a result, have no role in AMS/HACE prevention. Other over-the-counter products, such as powdered drink mixes, also lack any evidence of benefit.

SUGGESTED APPROACH TO AMS/HACE PREVENTION

Because the rates of acclimatization and physiologic responses to high altitude vary considerably between individuals, clinicians must recognize that the recommendations that follow, although generally effective, do not guarantee successful prevention in all high altitude travelers.

The approach to prevention of AMS and HACE should be a function of the risk profile of the individual traveling to high altitude (Table 2). The first priority should be ensuring gradual ascent to the target elevation. Travelers can lower their risk by sleeping 1 night at an intermediate altitude. For example, sea-level residents traveling to Colorado resort areas over 2800 m can spend 1 night in Denver (1600 m). It should be recognized that a large number of people will travel directly by car or plane to commonly visited mountain high altitude locations, often located

between 2500 and 3000 m, and may be unable to ascend gradually because of various logistical factors. In such situations, pharmacologic prophylaxis can be considered. Such individuals should also take care to slow the rate of further ascent beyond the altitude achieved at the start of their visit.

Table 2. Risk categories for acute mountain sickness

Risk category	***Description***
Low	• Individuals with no history of altitude illness and ascending to ≤2800 m
	• Individuals taking ≥2 d to arrive at 2500–3000 m with subsequent increases in sleeping elevation <500 $m \cdot d^{-1}$ and an extra day for acclimatization every 1000 m
Moderate	• Individuals with history of AMS and ascending to 2500–2800 m in 1 d
	• No history of AMS and ascending to >2800 m in 1 d
	• All individuals ascending >500 $m \cdot d^{-1}$ (increase in sleeping elevation) at altitudes above 3000 m but with an extra day for acclimatization every 1000 m
High	• Individuals with a history of AMS and ascending to >2800 m in 1 d
	• All individuals with a history of HACE or HAPE
	• All individuals ascending to >3500 m in 1 d
	• All individuals ascending >500 $m \cdot d^{-1}$ (increase in sleeping elevation) above >3000 m without extra days for acclimatization
	• Very rapid ascents (eg, <7 d ascents of Mt. Kilimanjaro)

AMS, acute mountain sickness; HACE, high altitude cerebral edema; HAPE, high altitude pulmonary edema.

Altitudes listed in the table refer to the altitude at which the person sleeps.

Ascent is assumed to start from elevations <1200 m.

The risk categories described pertain to unacclimatized individuals.

With travel above 3000 m, individuals should not increase their sleeping elevation by more than 500 $m \cdot d^{-1}$ and should include a rest day (ie, no ascent to higher sleeping elevation) every 3 to 4 d. The increase in sleeping elevation should be less than 500 m for any given day of a trip. In many areas, terrain and other logistical factors prevent strict adherence to this approach and mandate larger gains in sleeping elevation over a single day. In such cases, acclimatization days should be strongly considered before and/or after these large gains in elevation and elsewhere in the itinerary to ensure—at the very least and as an approximation of properly controlled ascent—that the overall ascent rate averaged over the entire trip (ie, total elevation gain divided by the number of days of ascent during the trip) is below the 500 $m \cdot d^{-1}$ threshold.

Prophylactic medications are not necessary in low-risk situations but should be considered in addition to gradual ascent for use in moderate- to high-risk situations (Table 2). Acetazolamide is the preferred medication; dexamethasone may be used as an alternative in individuals with a history of intolerance of or allergic reaction to acetazolamide. In rare circumstances (eg, military or rescue teams that must ascend rapidly to and perform physical work at >3500 m), consideration can be given to concurrent use of acetazolamide and dexamethasone. This strategy should be avoided except in these particular or other emergency circumstances that mandate very rapid ascent.

Acetazolamide and dexamethasone should be started the day before ascent but still have beneficial effects if started on the day of ascent. For individuals ascending to and staying at the same elevation for more than several days, prophylaxis may be stopped after 2 d at the highest altitude. Individuals ascending faster than the recommended ascent rates may consider continuing preventive medication for 2 to 4 d after arrival at the target altitude, but there are no data to support this approach. For individuals ascending to a high point and then descending toward the trailhead (eg, descending from the summit of Mt. Kilimanjaro), in the absence of AMS/HACE symptoms, preventive medications should be stopped when descent is initiated.

TREATMENT

Potential therapeutic options for AMS and HACE include the following.

Descent

Descent remains the single best treatment for AMS and HACE, but it is not necessary in all circumstances (discussed further later in the text). Individuals should descend until symptoms resolve unless terrain, weather, or injuries make descent impossible. Symptoms typically resolve after descent of 300 to 1000 m, but the required decrease in altitude varies among individuals. Individuals should not descend alone, particularly if they are experiencing HACE.

Recommendation. Descent is effective for any degree of AMS/HACE and is indicated for individuals with severe AMS, AMS that fails to resolve with other measures, or HACE. Recommendation Grade: 1A

Supplemental oxygen

Oxygen delivered by nasal cannula or mask at flow rates sufficient to relieve symptoms provides a suitable alternative to descent. A peripheral capillary oxygen saturation (S_pO_2) >90% is usually adequate. Use of oxygen is not required in all circumstances and is generally reserved for mountain clinics and hospitals where supply is abundant. It should also be used when descent is recommended but not feasible or during descent in severely ill individuals. The inspired oxygen fraction will vary significantly between oxygen delivery systems, including nasal cannula, simple facemasks, Venturi masks, or non-rebreather masks. In addition, because of interindividual variability in inspiratory flow rates and minute ventilation, the inspired fractional concentration of oxygen (F_IO_2) can

vary significantly between patients for any given common oxygen delivery system, with the exception of high flow systems. For this reason, supplemental oxygen should be administered to target an S_pO_2 of >90% rather than a specific F_IO_2. Oxygen supply may be limited at remote high altitude clinics or on expeditions, necessitating judicious use. Short-term oxygen use in the form of visits to oxygen bars or use of over-the-counter oxygen canisters has not been studied for AMS treatment and should not be relied on for this purpose.

Recommendation. When available, ongoing supplemental oxygen sufficient to raise S_pO_2 to >90% or to relieve symptoms can be used while waiting to initiate descent or when descent is not practical. Recommendation Grade: 1A

Portable hyperbaric chambers

Portable hyperbaric chambers are effective for treating severe altitude illness[69, 70] but require constant tending by care providers and are difficult to use with claustrophobic or vomiting patients. Symptoms may recur when individuals are removed from the chamber,[71] but this should not preclude use of the chamber when indicated. In many cases, ill individuals may improve sufficiently to enable them to assist in their evacuation and descend once symptoms improve. Use of a portable hyperbaric chamber should not delay descent in situations where descent is required.

Recommendation. When available, portable hyperbaric chambers should be used for patients with severe AMS or HACE when descent is infeasible or delayed and supplemental oxygen is not available. Recommendation Grade: 1B

Acetazolamide

Only 1 study has examined acetazolamide for AMS treatment. The dose studied was 250 mg every 12 h; whether a lower dose might suffice is unknown.[72] No studies have assessed AMS treatment with acetazolamide in pediatric patients, but anecdotal reports suggest it has utility. The pediatric treatment dose is 2.5 $mg \cdot kg^{-1} \cdot dose^{-1}$ every 12 h up to a maximum of $250\ mg \cdot dose^{-1}$.

Recommendation. Acetazolamide should be considered for treatment of AMS. Recommendation Grade: 1C

Dexamethasone

Dexamethasone is very effective for treating AMS.[73, 74, 75] The medication does not facilitate acclimatization, so further ascent should be delayed until the patient is asymptomatic while off the medication. Although systematic studies have not been conducted, extensive clinical experience supports using dexamethasone in patients with HACE. It is administered as an 8 mg dose (intramuscularly, IV, or orally) followed by 4 mg every 6 h until symptoms resolve. The pediatric dose is 0.15 $mg \cdot kg^{-1} \cdot dose^{-1}$ every 6 h.[27]

Recommendation. Dexamethasone should be considered for treatment of AMS. Recommendation Grade 1B.

Recommendation. Dexamethasone should be administered to patients with HACE. Recommendation Grade: 1B

Acetaminophen

Acetaminophen has been found to relieve headache at high altitude[76] but has not been found to improve the full spectrum of AMS symptoms or effectively treat HACE.

Recommendation. Acetaminophen can be used to treat headache at high altitude. Recommendation Grade: 1C.

Ibuprofen

Ibuprofen has been found to relieve headache at high altitude[76] but has not been shown to improve the full spectrum of AMS symptoms or effectively treat HACE.

Recommendation. Ibuprofen can be used to treat headache at high altitude. Recommendation Grade: 1C.

Continuous positive airway pressure

Rather than affecting barometric pressure, CPAP works by increasing transmural pressure across alveolar walls, thereby increasing alveolar volume and improving ventilation-perfusion matching and gas exchange. Two reports have demonstrated the feasibility of administering CPAP to treat AMS,[77, 78] but this has not been studied in a systematic manner. Logistical challenges to use in field settings include access to power and the weight and bulk of these systems.

Recommendation. Because of lack of data, no recommendation can be made regarding use of CPAP for AMS treatment.

SUGGESTED APPROACH TO AMS/HACE TREATMENT

Care should be taken to exclude disorders whose symptoms and signs resemble those seen with AMS and HACE, such as carbon monoxide poisoning, dehydration, exhaustion, hypoglycemia, hypothermia, and hyponatremia. Persons with AMS of any severity or HACE should cease ascending and may need to consider descent, depending on the severity of illness and the circumstances (Table 3).[11] Patients with AMS can remain at their current altitude and use nonopioid analgesics for headache and antiemetics for nausea and vomiting. These individuals should be closely observed for signs of progression of altitude illness. Descent should be initiated for AMS if symptoms worsen or fail to improve after 1 to 2 d of appropriate interventions.

Table 3. Acute mountain sickness classification

Category	*Mild AMS*	*Moderate–Severe AMS*	*High altitude cerebral edema (HACE)*
Symptoms	Headache plus 1 or more other symptoms (nausea/vomiting, fatigue, lassitude, dizziness)	Headache plus 1 or more other symptoms (nausea/vomiting, fatigue, lassitude, dizziness)	Worsening of symptoms seen in moderate to severe AMS
	All symptoms of mild intensity	All symptoms of moderate–severe intensity	
Signs	None	None	Ataxia, severe lassitude, altered mental status, encephalopathy
Lake Louise AMS Score[a]	3–5	6–12	Not applicable

AMS, acute mountain sickness.

a

Self-report AMS score. Roach et al.[103]

Although acetazolamide facilitates acclimatization and is somewhat effective for treating mild illness, it is likely better for prevention than for treatment. Dexamethasone is considered to be a more reliable treatment for moderate to severe AMS, which often also requires descent. Individuals with AMS may resume ascending once symptoms resolve. Further ascent or reascent to a previously attained altitude should never be undertaken if there are ongoing symptoms. After resolution of AMS, taking acetazolamide at preventive doses during reascent is prudent.

HACE is differentiated from severe AMS by neurological signs such as ataxia, confusion, or altered mental status in the setting of acute ascent to high altitude and may follow AMS or occur concurrently with HAPE. Individuals developing HACE in locations with access to hospitals or specialized clinics should be started on dexamethasone and supplemental oxygen sufficient to achieve an S_pO_2 >90%. In remote areas away from medical resources, descent should be initiated in any suspected cases of HACE or if symptoms of AMS are worsening despite treatment with acetazolamide or dexamethasone. If descent is not feasible, supplemental oxygen or a portable hyperbaric chamber should be used. Persons with HACE should also be started on dexamethasone. There are no systematic data or case reports about reascent during the same trip or expedition after resolution of HACE. The prudent course is to avoid reascent in this situation, but if it is to be attempted, at a minimum the individual should be asymptomatic and no longer taking dexamethasone for at least 2 to 3 d before reascent.

High altitude pulmonary edema

Information on the epidemiology, clinical presentation, and pathophysiology of HAPE, the majority of which comes from studies in adults, is provided in extensive reviews.[13], [14], [79], [80] Although some of the prophylactic and therapeutic modalities are the same for HAPE as for AMS and HACE, important differences in the underlying pathophysiology mandate certain alternative prevention and treatment approaches.

PREVENTION

Potential preventive measures for HAPE include the following.

Gradual ascent

No studies have prospectively assessed whether limiting the rate of increase in sleeping elevation prevents HAPE; however, there is a clear relationship between rate of ascent and disease incidence.[17], [81], [82]

Recommendation. Gradual ascent is recommended to prevent HAPE. Recommendation Grade: 1B

Nifedipine

A single, randomized, placebo-controlled study[83] and extensive clinical experience have established a role for nifedipine in HAPE prevention in susceptible individuals. The recommended dose is 30 mg of the extended-release preparation administered every 12 h. Hypotension was not noted in the study[83] and is generally not a concern with the extended-release version of the medication but may occur in a limited number of individuals.

Recommendation. Nifedipine is recommended for HAPE prevention in HAPE-susceptible people. Recommendation Grade: 1B

Salmeterol

In a single randomized, placebo-controlled study, the long-acting inhaled beta-agonist salmeterol decreased the incidence of HAPE by 50% in susceptible individuals.[84] Very high doses (125 micrograms twice daily) that are often associated with side effects, including tremor and tachycardia, were used in the study. Clinical experience with salmeterol at high altitude is limited.

Recommendation. Salmeterol is not recommended for HAPE prevention. Recommendation Grade: 2B.

Tadalafil

In a single, randomized placebo-controlled trial, 10 mg of tadalafil every 12 h was effective in preventing HAPE in susceptible individuals.[85] The number of individuals in the study was small, and 2 developed incapacitating AMS. Clinical experience with tadalafil is lacking compared to nifedipine. As a result, further data are necessary before tadalafil can be recommended over nifedipine.

Recommendation. Tadalafil can be used for HAPE prevention in known susceptible individuals who are not candidates for nifedipine. Recommendation Grade: 1C

Dexamethasone

In the same study that assessed the role of tadalafil in HAPE prevention, dexamethasone (8 mg every 12 h) was also found to prevent HAPE in susceptible individuals.[85] The mechanism for this effect is not clear, and there is very little clinical experience in using dexamethasone for this purpose. Further data are necessary before it can be recommended for HAPE prevention.

Recommendation. Dexamethasone can be used for HAPE prevention in known susceptible individuals who are not candidates for nifedipine and tadalafil. Recommendation Grade: 1C

Acetazolamide

Because acetazolamide hastens acclimatization, it should be effective at preventing all forms of acute altitude illness. It has also been shown to blunt hypoxic pulmonary vasoconstriction, a key factor in HAPE pathophysiology, in animal models[86, 87, 88] and in a single study in humans,[89] but there are no data specifically supporting a role in HAPE prevention. Clinical observations suggest acetazolamide may prevent reentry HAPE,[90] a disorder seen in individuals who reside at high altitude, travel to lower elevation, and then develop HAPE upon rapid return to their residence.

Recommendation. Because of lack of data, no recommendation can be made regarding use of acetazolamide for HAPE prevention.

Recommendation. Acetazolamide can be considered for prevention of reentry HAPE in people with a history of the disorder. Recommendation Grade: 1C

Preacclimatization and staged ascent

No study has examined whether preacclimatization strategies are useful for HAPE prevention. Staged ascent, with 7 d of residence at moderate altitude (~2200 m), has been found to blunt the hypoxia-induced increase in pulmonary artery pressure.[48] However, uncertainty remains as to the magnitude and duration of moderate altitude exposure necessary to yield benefit, and no study has specifically investigated whether the strategy is of benefit in HAPE-susceptible individuals. Although the risks of preacclimatization and staged ascent are likely low, feasibility is a concern for many high altitude travelers. Because the optimal methods for preacclimatization and staged ascent have not been fully determined, the panel recommends consideration of these approaches but cannot endorse a particular protocol for implementation.

Recommendation. When feasible, staged ascent and preacclimatization can be considered as a means for HAPE prevention. Recommendation Grade: 1C

SUGGESTED APPROACH TO HAPE PREVENTION

As noted earlier, because the rates of acclimatization and physiologic responses to high altitude vary considerably among individuals, the recommendations that follow, although generally effective, do not guarantee prevention in all high altitude travelers. A gradual ascent profile is the primary method for preventing HAPE; the recommendations provided for AMS and HACE prevention also apply to HAPE prevention. Pharmacologic prophylaxis should only be considered for individuals with a history of HAPE, especially multiple episodes. Nifedipine is the preferred drug in such situations; it should be started the day before ascent and continued either until descent is initiated or the individual has spent 4 d at the highest elevation, perhaps up to 7 d if the individual's rate of ascent was faster than recommended. Note that these durations are longer than use of acetazolamide for AMS prevention. For individuals ascending to a high point and then descending toward the trailhead (eg, descending from the summit of Kilimanjaro), prophylactic medications should be stopped when descent is initiated. Further research is needed before tadalafil or dexamethasone can be recommended over nifedipine for prevention. Acetazolamide facilitates acclimatization in general but should not be relied upon as the sole preventive agent in known HAPE-susceptible individuals.

TREATMENT

Therapeutic options for HAPE include the following.

Descent

As with AMS and HACE, descent remains the single best treatment for HAPE. Individuals should try to descend at least 1000 m or until symptoms resolve. They should exert themselves as little as possible while descending (eg, travel without a pack or via motor vehicle, helicopter, or animal transportation) because exertion can further increase pulmonary artery pressure and exacerbate edema formation.

Recommendation. Descent is indicated for individuals with HAPE.
Recommendation Grade: 1A

Supplemental oxygen

Oxygen delivered by nasal cannula or mask at flow rates sufficient to achieve an S_pO_2 >90% provides a suitable alternative to descent, particularly when patients can access healthcare facilities and be closely monitored.[91, 92, 93] As noted earlier in the section on AMS/HACE treatment, providers should target an S_pO_2 of >90% rather than a particular F_IO_2. Short-term use in the form of visits to oxygen bars or use of over-the-counter oxygen canisters has no role in HAPE treatment.

Recommendation. When available, supplemental oxygen sufficient achieve an S_pO_2 of >90% or to relieve symptoms should be used while waiting to initiate descent when descent is infeasible and during descent in severely ill patients.
Recommendation Grade: 1A

Portable hyperbaric chambers

As for AMS and HACE, portable hyperbaric chambers can be used for HAPE treatment. They have not been systematically studied for this purpose, but their use for HAPE has been reported in the literature.[94] Use of a portable hyperbaric chamber should not delay descent in situations where descent is feasible.

Recommendation. When descent is infeasible or delayed or supplemental oxygen is unavailable, a portable hyperbaric chamber may be used to treat HAPE. Recommendation Grade: 1C

Nifedipine

A single, nonrandomized, unblinded study demonstrated utility of nifedipine (10 mg of the short-acting version followed by 20 mg slow-release every 6 h) for HAPE treatment when oxygen or descent was not available.[95] Although participants in this study received a loading dose of the short-acting version of the medication, this initial dose is no longer employed because of concerns about provoking systemic hypotension. Although hypotension is less common with the extended-release preparation, it may develop when nifedipine is given to patients with intravascular volume depletion. A prospective, cross-sectional study of individuals with HAPE demonstrated that addition of nifedipine (30 mg sustained release every 12 h) to descent, oxygen, and rest offered no additional benefit in terms of time to resolution of hypoxemia and radiographic opacities or hospital length of stay.[96]

Recommendation. Nifedipine should be used for HAPE treatment when descent is impossible or delayed and reliable access to supplemental oxygen or portable hyperbaric therapy is unavailable. Recommendation Grade: 1C

Beta-agonists

Although there are reports of beta-agonist use in HAPE treatment[97] and the risks of use are likely low, no data support a benefit from salmeterol or albuterol in patients experiencing HAPE.

Recommendation. No recommendation can be made regarding beta-agonists for HAPE treatment due to lack of data.

Phosphodiesterase inhibitors

By virtue of their ability to cause pulmonary vasodilation and decrease pulmonary artery pressure, there is a strong physiologic rationale for using phosphodiesterase inhibitors in HAPE treatment. Although reports document their use for this purpose,[97, 98] no systematic study has examined the role of tadalafil or sildenafil in HAPE treatment as either mono- or adjunctive therapy. Combined use of nifedipine and sildenafil or tadalafil should be avoided because of risk of hypotension.

Recommendation. Tadalafil or sildenafil can be used for HAPE treatment when descent is impossible or delayed, access to supplemental oxygen or portable hyperbaric therapy is impossible, and nifedipine is unavailable. Recommendation Grade: 2C

Continuous positive airway pressure

As noted earlier, positive airway pressure works by increasing transmural pressure across alveolar walls, thereby increasing alveolar volume and improving ventilation-perfusion matching and, as a result, gas exchange. A small study demonstrated that EPAP, in which a mask system is used to increase airway pressure during exhalation only, improved gas exchange in patients with HAPE.[99] However, although several reports document use of CPAP for management of HAPE in hospital and field settings,[6, 78] there is no systematic evidence that CPAP or EPAP improves patient outcomes compared to oxygen alone or in conjunction with medications. Given the low risks associated with the therapy, CPAP can be considered an adjunct to oxygen administration in a medical facility, provided the patient has normal mental status and can tolerate the mask. Although lithium battery–powered devices and decreased size and weight of CPAP machines have increased feasibility of field use, logistical challenges remain and currently limit overall utility in this setting.

Recommendation. CPAP or EPAP may be considered for treatment of HAPE when supplemental oxygen or pulmonary vasodilators are not available or as adjunctive therapy in patients not responding to supplemental oxygen alone. Recommendation Grade: 2C

Diuretics

Although their use is documented in older reports,[100] diuretics have no role in HAPE treatment, particularly because many patients with HAPE have intravascular volume depletion.

Recommendation. Diuretics should not be used for treatment of HAPE. Recommendation Grade: 1C.

Acetazolamide

Although clinical reports document use of acetazolamide for treatment of HAPE,[97, 98] there are no systematic studies examining its role in HAPE treatment. The diuretic effect might provoke hypotension in the intravascularly depleted patient, and the added stimulus to ventilation might worsen dyspnea.

Recommendation. Acetazolamide should not be used for treatment of HAPE. Recommendation Grade: 1C

Dexamethasone

Considering its potential role in HAPE prevention noted earlier and studies demonstrating effects on maximum exercise capacity,[101] pulmonary inflammation, and ion transporter function in hypoxia,[102] dexamethasone may have a role in HAPE treatment. Although reports document clinical use in this regard,[98] no study has established whether it is effective for this purpose.

Recommendation. Because of insufficient evidence, no recommendation can be made regarding dexamethasone for HAPE treatment.

SUGGESTED APPROACH TO HAPE TREATMENT

Before initiating treatment, consideration should be given to other causes of respiratory symptoms at high altitude, such as asthma, bronchospasm, mucous plugging, pneumonia, pneumothorax, pulmonary embolism, viral upper respiratory tract infection, or myocardial infarction. If HAPE is suspected or diagnosed, oxygen should be started if available, and descent to lower elevation should be initiated. If descent is infeasible or delayed, supplemental oxygen should be continued or the individual should be placed in a portable hyperbaric chamber. Patients who have access to supplemental oxygen and can be adequately monitored in a medical setting (eg, urgent care clinic or emergency department) may not need to descend to lower elevation and can be treated with oxygen alone at the current elevation. Descent should be initiated, however, if oxygenation fails to improve with supplemental oxygen and/or CPAP, if the patient's condition deteriorates despite achieving an oxygen saturation >90%, or if the patient fails to show signs of improvement with appropriate interventions for HAPE. In more remote settings, early descent should be considered.

Addition of nifedipine may not yield additional benefit in well-monitored settings.93, 96 In the field setting, where resources are limited, nifedipine can be used as an adjunct to descent, supplemental oxygen, or portable hyperbaric therapy. It should only be used as primary therapy if none of these other measures is available. A phosphodiesterase inhibitor may be used if nifedipine is not available, but concurrent use of multiple pulmonary vasodilators is not recommended. In the hospital setting, CPAP can be considered as an adjunct to supplemental oxygen and nifedipine can be added if the patient fails to respond to oxygen therapy alone. There is no established role for beta-agonists, diuretics, acetazolamide, or dexamethasone in the treatment of HAPE, although, as noted below, dexamethasone should be considered when concern is raised for concurrent HACE.

Selected patients (able to achieve an oxygen saturation >90%, with adequate support from family or friends, with adequate housing or lodging arrangements) may be discharged from direct medical care if they can continue using supplemental oxygen rather than being admitted to a healthcare facility. Individuals treated in this manner should be admitted to the hospital if they develop worsening symptoms and/or oxygen saturation while on supplemental oxygen. Descent to lower elevation should be pursued if oxygenation or other aspects of their condition worsen despite appropriate interventions for HAPE, as this suggests they may have alternative pathology that requires further evaluation and management.

Individuals who develop HAPE may consider further ascent to higher altitude or reascent only when symptoms of HAPE have completely resolved and they maintain stable oxygenation at rest and with mild exercise while off supplemental oxygen and/or vasodilator therapy. Consideration may be given to using nifedipine or another pulmonary vasodilator upon resuming ascent.

SUGGESTED APPROACH FOR PATIENTS WITH CONCURRENT HAPE AND HACE

Dexamethasone should be added to the treatment regimen of patients with concurrent HAPE and HACE at the doses described earlier for patients with HACE. Some patients with HAPE may have neurologic dysfunction caused by hypoxic encephalopathy rather than caused by HACE, but making the distinction between hypoxic encephalopathy and HACE in the field can be difficult. Therefore, dexamethasone should be added to the treatment regimen for patients with HAPE with neurologic dysfunction that does not resolve rapidly with administration of supplemental oxygen and improvement in oxygen saturation. If supplemental oxygen is not available, dexamethasone should be started in addition to the medications for HAPE in patients with altered mental status and/or suspected concurrent HACE. Nifedipine or other pulmonary vasodilators may be used in patients with concurrent HAPE and HACE, with care to avoid lowering mean arterial pressure, as this may decrease cerebral perfusion pressure and thus increase the risk for cerebral ischemia.

Conclusions

We have provided evidence-based guidelines for prevention and treatment of acute altitude illnesses, including the main prophylactic and therapeutic modalities for AMS, HACE, and HAPE, and recommendations regarding their role in disease management. Although these guidelines cover many of the important issues related to prevention and treatment of altitude illness, several important questions remain to be addressed and should serve as a focus for future research. Such research includes determining the optimal rate of ascent to prevent altitude illness, the role of acetazolamide in HAPE prevention and treatment, proper dosing regimens for prevention and treatment of altitude illness in the pediatric population, and the role of staged ascent, preacclimatization, and hypoxic tents in altitude illness prevention.

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Appendix. Supplementary materials

Download : Download Word document (14KB)

Supplementary Table 1.

Recommended articles | Citing articles (4)

References

1

G. Guyatt, D. Gutterman, M.H. Baumann, D. Addrizzo-Harris, E.M. Hylek, B. Phillips, *et al.***Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force**
Chest, 129 (1) (2006), pp. 174-181
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

2

A.M. Luks, S.E. McIntosh, C.K. Grissom, P.S. Auerbach, G.W. Rodway, R.B. Schoene, *et al.***Wilderness Medical Society consensus guidelines for the prevention and treatment of acute altitude illness**
Wilderness Environ Med, 21 (2) (2010), pp. 146-155
ArticleDownload PDFView Record in ScopusGoogle Scholar

3

A.M. Luks, S.E. McIntosh, C.K. Grissom, P.S. Auerbach, G.W. Rodway, R.B. Schoene, *et al.***Wilderness Medical Society Practice Guidelines for the prevention and treatment of acute altitude illness: 2014 update**
Wilderness Environ Med, 25 (4S) (2014), pp. S4-S14
ArticleDownload PDFView Record in ScopusGoogle Scholar

4

A.B. Montgomery, J. Mills, J.M. Luce**Incidence of acute mountain sickness at intermediate altitude**
JAMA, 261 (5) (1989), pp. 732-734
CrossRefView Record in ScopusGoogle Scholar

5

B. Honigman, M.K. Theis, J. Koziol-McLain, R. Roach, R. Yip, C. Houston, *et al.***Acute mountain sickness in a general tourist population at moderate altitudes**
Ann Intern Med, 118 (8) (1993), pp. 587-592
CrossRefView Record in ScopusGoogle Scholar

6

A.L. Gabry, X. Ledoux, M. Mozziconacci, C. Martin**High-altitude pulmonary edema at moderate altitude (<2,400 m; 7,870 feet): a series of 52 patients**
Chest, 123 (1) (2003), pp. 49-53
ArticleDownload PDFView Record in ScopusGoogle Scholar

7

P.H. Hackett, P.R. Yarnell, D.A. Weiland, K.B. Reynard**Acute and evolving MRI of high-altitude cerebral edema: microbleeds, edema, and pathophysiology**
AJNR Am J Neuroradiol, 40 (3) (2019), pp. 464-469
View Record in ScopusGoogle Scholar

8

A. Southard, S. Niermeyer, M. Yaron**Language used in Lake Louise Scoring System underestimates symptoms of acute mountain sickness in 4- to 11-year-old children**
High Alt Med Biol, 8 (2) (2007), pp. 124-130

CrossRefView Record in ScopusGoogle Scholar

9

J.M. Muhm, P.B. Rock, D.L. McMullin, S.P. Jones, I.L. Lu, K.D. Eilers, *et al.***Effect of aircraft-cabin altitude on passenger discomfort**
N Engl J Med, 357 (1) (2007), pp. 18-27
View Record in ScopusGoogle Scholar

10

M.K. Theis, B. Honigman, R. Yip, D. McBride, C.S. Houston, L.G. Moore**Acute mountain sickness in children at 2835 meters**
Am J Dis Child, 147 (2) (1993), pp. 143-145
CrossRefView Record in ScopusGoogle Scholar

11

P.H. Hackett, R.C. Roach**High-altitude illness**
N Engl J Med, 345 (2) (2001), pp. 107-114
View Record in ScopusGoogle Scholar

12

P.H. Hackett, R.C. Roach**High altitude cerebral edema**
High Alt Med Biol, 5 (2) (2004), pp. 136-146
CrossRefView Record in ScopusGoogle Scholar

13

P. Bartsch, E.R. Swenson**Clinical practice: acute high-altitude illnesses**
N Engl J Med, 368 (24) (2013), pp. 2294-2302
CrossRefView Record in ScopusGoogle Scholar

14

P.H. Hackett, A.M. Luks, J.S. Lawley, R.C. Roach**High altitude medicine and pathophysiology. In: Auerbach PS, ed**
Auerbach's Wilderness Medicine, Elsevier, Philadelphia (2017)
Google Scholar

15

K.E. Bloch, A.J. Turk, M. Maggiorini, T. Hess, T. Merz, M.M. Bosch, *et al.***Effect of ascent protocol on acute mountain sickness and success at Muztagh Ata, 7546 m**
High Alt Med Biol, 10 (1) (2009), pp. 25-32
CrossRefView Record in ScopusGoogle Scholar

16

B.A. Beidleman, C.S. Fulco, S.R. Muza, P.B. Rock, J.E. Staab, V.A. Forte, *et al.***Effect of six days of staging on physiologic adjustments and acute mountain sickness during ascent to 4300 meters**
High Alt Med Biol, 10 (3) (2009), pp. 253-260
CrossRefView Record in ScopusGoogle Scholar

17

P.H. Hackett, D. Rennie, H.D. Levine**The incidence, importance, and prophylaxis of acute mountain sickness**
Lancet, 2 (7996) (1976), pp. 1149-1155

ArticleDownload PDFView Record in ScopusGoogle Scholar

18

S.A. Forward, M. Landowne, J.N. Follansbee, J.E. Hansen**Effect of acetazolamide on acute mountain sickness**
N Engl J Med, 279 (16) (1968), pp. 839-845
View Record in ScopusGoogle Scholar

19

B. Basnyat, J.H. Gertsch, P.S. Holck, E.W. Johnson, A.M. Luks, B.P. Donham, *et al.***Acetazolamide 125 mg BD is not significantly different from 375 mg BD in the prevention of acute mountain sickness: the prophylactic acetazolamide dosage comparison for efficacy (PACE) trial**
High Alt Med Biol, 7 (1) (2006), pp. 17-27
CrossRefView Record in ScopusGoogle Scholar

20

M.C. van Patot, G. Leadbetter 3rd, L.E. Keyes, K.M. Maakestad, S. Olson, P.H. Hackett**Prophylactic low-dose acetazolamide reduces the incidence and severity of acute mountain sickness**
High Alt Med Biol, 9 (4) (2008), pp. 289-293
CrossRefView Record in ScopusGoogle Scholar

21

E.V. Low, A.J. Avery, V. Gupta, A. Schedlbauer, M.P. Grocott**Identifying the lowest effective dose of acetazolamide for the prophylaxis of acute mountain sickness: systematic review and meta-analysis**
BMJ, 345 (2012), p. e6779
CrossRefGoogle Scholar

22

A.M. Luks, E.R. Swenson**Medication and dosage considerations in the prophylaxis and treatment of high-altitude illness**
Chest, 133 (3) (2008), pp. 744-755
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

23

E. Elisabeth, G. Hannes, B. Johannes, F. Martin, P. Elena, B. Martin**Effects of low-dose acetazolamide on exercise performance in simulated altitude**
Int J Physiol Pathophysiol Pharmacol, 9 (2) (2017), pp. 28-34
View Record in ScopusGoogle Scholar

24

A.R. Bradwell, S.D. Myers, M. Beazley, K. Ashdown, N.G. Harris, S.B. Bradwell, *et al.***Exercise limitation of acetazolamide at altitude (3459 m)**
Wilderness Environ Med, 25 (3) (2014), pp. 272-277
ArticleDownload PDFView Record in ScopusGoogle Scholar

25

J.U. Gonzales, B.W. Scheuermann**Effect of acetazolamide on respiratory muscle fatigue in humans**
Respir Physiol Neurobiol, 185 (2) (2013), pp. 386-392

ArticleDownload PDFView Record in ScopusGoogle Scholar

26

S.E. McIntosh, M. Hemphill, M.C. McDevitt, T.Y. Gurung, M. Ghale, J.R. Knott, *et al.***Reduced acetazolamide dosing in countering altitude illness: a comparison of 62.5 vs 125 mg (the RADICAL Trial)**
Wilderness Environ Med, 30 (1) (2019), pp. 12-21
ArticleDownload PDFView Record in ScopusGoogle Scholar

27

A.J. Pollard, S. Niermeyer, P. Barry, P. Bartsch, F. Berghold, R.A. Bishop, *et al.***Children at high altitude: an international consensus statement by an ad hoc committee of the International Society for Mountain Medicine, March 12, 2001**
High Alt Med Biol, 2 (3) (2001), pp. 389-403
CrossRefGoogle Scholar

28

A.J. Ellsworth, E.B. Larson, D. Strickland**A randomized trial of dexamethasone and acetazolamide for acute mountain sickness prophylaxis**
Am J Med, 83 (6) (1987), pp. 1024-1030
ArticleDownload PDFView Record in ScopusGoogle Scholar

29

A.J. Ellsworth, E.F. Meyer, E.B. Larson**Acetazolamide or dexamethasone use versus placebo to prevent acute mountain sickness on Mount Rainier**
West J Med, 154 (3) (1991), pp. 289-293
View Record in ScopusGoogle Scholar

30

G. Chrousos, A.N. Pavlaki, M.A. Magiakou, *et al.***Glucocorticoid therapy and adrenal suppression**
L.J. De Groot, G. Chrousos, K. Dungan (Eds.), Endotext, MDText.com, South Dartmouth, MA (2011)
Google Scholar

31

C.R. Zheng, G.Z. Chen, J. Yu, J. Qin, P. Song, S.Z. Bian, *et al.***Inhaled budesonide and oral dexamethasone prevent acute mountain sickness**
Am J Med, 127 (10) (2014), pp. 1001-1009
e2
View Record in ScopusGoogle Scholar

32

G.Z. Chen, C.R. Zheng, J. Qin, J. Yu, H. Wang, J.H. Zhang, *et al.***Inhaled budesonide prevents acute mountain sickness in young Chinese men**
J Emerg Med, 48 (2) (2015), pp. 197-206
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

33

M.M. Berger, F. Macholz, M. Sareban, P. Schmidt, S. Fried, D. Dankl, *et al.***Inhaled budesonide does not prevent acute mountain sickness after rapid ascent to 4559 m**
Eur Respir J, 50 (3) (2017)
Google Scholar

34

G.S. Lipman, D. Pomeranz, P. Burns, C. Phillips, M. Cheffers, K. Evans, *et al.***Budesonide versus acetazolamide for prevention of acute mountain sickness**
Am J Med, 131 (2) (2018), pp. e9-e16
Google Scholar

35

J.P. Roncin, F. Schwartz, P. D'Arbigny**EGb 761 in control of acute mountain sickness and vascular reactivity to cold exposure**
Aviat Space Environ Med, 67 (5) (1996), pp. 445-452
View Record in ScopusGoogle Scholar

36

F.A. Moraga, A. Flores, J. Serra, C. Esnaola, C. Barriento***Ginkgo biloba* decreases acute mountain sickness in people ascending to high altitude at Ollague (3696 m) in northern Chile**
Wilderness Environ Med, 18 (4) (2007), pp. 251-257
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

37

J.H. Gertsch, B. Basnyat, E.W. Johnson, J. Onopa, P.S. Holck**Randomised, double blind, placebo controlled comparison of *Ginkgo biloba* and acetazolamide for prevention of acute mountain sickness among Himalayan trekkers: the prevention of high altitude illness trial (PHAIT)**
BMJ, 328 (7443) (2004), p. 797
View Record in ScopusGoogle Scholar

38

T. Chow, V. Browne, H.L. Heileson, D. Wallace, J. Anholm, S.M. Green***Ginkgo biloba* and acetazolamide prophylaxis for acute mountain sickness: a randomized, placebo-controlled trial**
Arch Intern Med, 165 (3) (2005), pp. 296-301
CrossRefView Record in ScopusGoogle Scholar

39

G. Leadbetter, L.E. Keyes, K.M. Maakestad, S. Olson, M.C. Tissot van Patot, P.H. Hackett***Ginkgo biloba* does—and does not—prevent acute mountain sickness**
Wilderness Environ Med, 20 (1) (2009), pp. 66-71
ArticleDownload PDFView Record in ScopusGoogle Scholar

40

J.J. Dugoua, E. Mills, D. Perri, G. Koren**Safety and efficacy of ginkgo (*Ginkgo biloba*) during pregnancy and lactation**
Can J Clin Pharmacol, 13 (3) (2006), pp. e277-e284

View Record in ScopusGoogle Scholar

41

G.J. Stoddard, M. Archer, L. Shane-McWhorter, B.E. Bray, D.F. Redd, J. Proulx, *et al.* ***Ginkgo*** **and warfarin interaction in a large veterans administration population**
AMIA Annu Symp Proc, 2015 (2015), pp. 1174-1183
View Record in ScopusGoogle Scholar

42

J.H. Gertsch, B. Corbett, P.S. Holck, A. Mulcahy, M. Watts, N.T. Stillwagon, *et al.* **Altitude sickness in climbers and efficacy of NSAIDs trial (ASCENT): randomized, controlled trial of ibuprofen versus placebo for prevention of altitude illness**
Wilderness Environ Med, 23 (4) (2012), pp. 307-315
ArticleDownload PDFView Record in ScopusGoogle Scholar

43

G.S. Lipman, N.C. Kanaan, P.S. Holck, B.B. Constance, J.H. Gertsch **Ibuprofen prevents altitude illness: a randomized controlled trial for prevention of altitude illness with nonsteroidal anti-inflammatories**
Ann Emerg Med, 59 (6) (2012), pp. 484-490
ArticleDownload PDFView Record in ScopusGoogle Scholar

44

J. Lundeberg, J.R. Feiner, A. Schober, J.W. Sall, H. Eilers, P.E. Bickler **Increased cytokines at high altitude: lack of effect of ibuprofen on acute mountain sickness, physiological variables, or cytokine levels**
High Alt Med Biol, 19 (3) (2018), pp. 249-258
CrossRefView Record in ScopusGoogle Scholar

45

N.C. Kanaan, A.L. Peterson, M. Pun, P.S. Holck, J. Starling, B. Basyal, *et al.* **Prophylactic acetaminophen or ibuprofen results in equivalent acute mountain sickness incidence at high altitude: a prospective randomized trial**
Wilderness Environ Med, 28 (2) (2017), pp. 72-78
ArticleDownload PDFView Record in ScopusGoogle Scholar

46

J.H. Gertsch, G.S. Lipman, P.S. Holck, A. Merritt, A. Mulcahy, R.S. Fisher, *et al.* **Prospective, double-blind, randomized, placebo-controlled comparison of acetazolamide versus ibuprofen for prophylaxis against high altitude headache: the Headache Evaluation at Altitude Trial (HEAT)**
Wilderness Environ Med, 21 (3) (2010), pp. 236-243
ArticleDownload PDFView Record in ScopusGoogle Scholar

47

P. Burns, G.S. Lipman, K. Warner, C. Jurkiewicz, C. Phillips, L. Sanders, *et al.* **Altitude sickness prevention with ibuprofen relative to acetazolamide**
Am J Med, 132 (2) (2019), pp. 247-251
ArticleDownload PDFView Record in ScopusGoogle Scholar

48

A.L. Baggish, C.S. Fulco, S. Muza, P.B. Rock, B. Beidleman, A. Cymerman, *et al.***The impact of moderate-altitude staging on pulmonary arterial hemodynamics after ascent to high altitude**
High Alt Med Biol, 11 (2) (2010), pp. 139-145
CrossRefView Record in ScopusGoogle Scholar

49

T.P. Lyons, S.R. Muza, P.B. Rock, A. Cymerman**The effect of altitude pre-acclimatization on acute mountain sickness during reexposure**
Aviat Space Environ Med, 66 (10) (1995), pp. 957-962
View Record in ScopusGoogle Scholar

50

B.A. Beidleman, S.R. Muza, C.S. Fulco, A. Cymerman, D. Ditzler, D. Stulz, *et al.***Intermittent altitude exposures reduce acute mountain sickness at 4300 m**
Clin Sci (Lond), 106 (3) (2004), pp. 321-328
View Record in ScopusGoogle Scholar

51

M. Wille, H. Gatterer, K. Mairer, M. Philippe, H. Schwarzenbacher, M. Faulhaber, *et al.***Short-term intermittent hypoxia reduces the severity of acute mountain sickness**
Scand J Med Sci Sports, 22 (5) (2012), pp. e79-e85
CrossRefView Record in ScopusGoogle Scholar

52

R.L. Burse, V.A. Forte Jr**Acute mountain sickness at 4500 m is not altered by repeated eight-hour exposures to 3200–3550 m normobaric hypoxic equivalent**
Aviat Space Environ Med, 59 (10) (1988), pp. 942-949
View Record in ScopusGoogle Scholar

53

K. Schommer, N. Wiesegart, E. Menold, U. Haas, K. Lahr, H. Buhl, *et al.***Training in normobaric hypoxia and its effects on acute mountain sickness after rapid ascent to 4559 m**
High Alt Med Biol, 11 (1) (2010), pp. 19-25
CrossRefView Record in ScopusGoogle Scholar

54

C.S. Fulco, S.R. Muza, B.A. Beidleman, R. Demes, J.E. Staab, J.E. Jones, *et al.***Effect of repeated normobaric hypoxia exposures during sleep on acute mountain sickness, exercise performance, and sleep during exposure to terrestrial altitude**
Am J Physiol Regul Integr Comp Physiol, 300 (2) (2011), pp. R428-R436
CrossRefView Record in ScopusGoogle Scholar

55

M. Faulhaber, E. Pocecco, H. Gatterer, M. Niedermeier, M. Huth, T. Dunnwald, *et al.***Seven passive 1-h hypoxia exposures do not prevent AMS in**

susceptible individuals
Med Sci Sports Exerc, 48 (12) (2016), pp. 2563-2570
View Record in ScopusGoogle Scholar

56
C.S. Fulco, B.A. Beidleman, S.R. Muza**Effectiveness of preacclimatization strategies for high-altitude exposure**
Exerc Sport Sci Rev, 41 (1) (2013), pp. 55-63
View Record in ScopusGoogle Scholar

57
C. Dehnert, A. Bohm, I. Grigoriev, E. Menold, P. Bartsch**Sleeping in moderate hypoxia at home for prevention of acute mountain sickness (AMS): a placebo-controlled, randomized double-blind study**
Wilderness Environ Med, 25 (3) (2014), pp. 263-271
ArticleDownload PDFView Record in ScopusGoogle Scholar

58
J.K. Baillie, A.A. Thompson, J.B. Irving, M.G. Bates, A.I. Sutherland, W. Macnee, *et al.***Oral antioxidant supplementation does not prevent acute mountain sickness: double blind, randomized placebo-controlled trial**
QJM, 102 (5) (2009), pp. 341-348
CrossRefView Record in ScopusGoogle Scholar

59
N.P. Talbot, T.G. Smith, C. Privat, A.H. Nickol, M. Rivera-Ch, F. Leon-Velarde, *et al.***Intravenous iron supplementation may protect against acute mountain sickness: a randomized, double-blinded, placebo-controlled trial**
High Alt Med Biol, 12 (3) (2011), pp. 265-269
CrossRefView Record in ScopusGoogle Scholar

60
G.M.K. Rossetti, J.H. Macdonald, L.J. Wylie, S.J. Little, V. Newton, B. Wood, *et al.***Dietary nitrate supplementation increases acute mountain sickness severity and sense of effort during hypoxic exercise**
J Appl Physiol (1985), 123 (4) (2017), pp. 983-992
CrossRefView Record in ScopusGoogle Scholar

61
C.K. Grissom, L.D. Richer, M.R. Elstad**The effects of a 5-lipoxygenase inhibitor on acute mountain sickness and urinary leukotriene e4 after ascent to high altitude**
Chest, 127 (2) (2005), pp. 565-570
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

62
A.M. Luks, W.R. Henderson, E.R. Swenson**Leukotriene receptor blockade does not prevent acute mountain sickness induced by normobaric hypoxia**
High Alt Med Biol, 8 (2) (2007), pp. 131-138
CrossRefView Record in ScopusGoogle Scholar

63

M.G. Bates, A.A. Thompson, J.K. Baillie, A.I. Sutherland, J.B. Irving, N. Hirani, *et al.***Sildenafil citrate for the prevention of high altitude hypoxic pulmonary hypertension: double blind, randomized, placebo-controlled trial**

High Alt Med Biol, 12 (3) (2011), pp. 207-214

CrossRefView Record in ScopusGoogle Scholar

64

B. Kayser, R. Hulsebosch, F. Bosch**Low-dose acetylsalicylic acid analog and acetazolamide for prevention of acute mountain sickness**

High Alt Med Biol, 9 (1) (2008), pp. 15-23

CrossRefView Record in ScopusGoogle Scholar

65

B. Basnyat, P.S. Holck, M. Pun, S. Halverson, P. Szawarski, J. Gertsch, *et al.***Spironolactone does not prevent acute mountain sickness: a prospective, double-blind, randomized, placebo-controlled trial by SPACE Trial Group (spironolactone and acetazolamide trial in the prevention of acute mountain sickness group)**

Wilderness Environ Med, 22 (1) (2011), pp. 15-22

ArticleDownload PDFView Record in ScopusGoogle Scholar

66

S. Jafarian, F. Gorouhi, S. Salimi, J. Lotfi**Sumatriptan for prevention of acute mountain sickness: randomized clinical trial**

Ann Neurol, 62 (3) (2007), pp. 273-277

CrossRefView Record in ScopusGoogle Scholar

67

G.S. Lipman, N.C. Kanaan, C. Phillips, D. Pomeranz, P. Cain, K. Fontes, *et al.***Study looking at end expiratory pressure for altitude illness decrease (SLEEP-AID)**

High Alt Med Biol, 16 (2) (2015), pp. 154-161

CrossRefView Record in ScopusGoogle Scholar

68

M.M. Berger, F. Macholz, L. Lehmann, D. Dankl, M. Hochreiter, B. Bacher, *et al.***Remote ischemic preconditioning does not prevent acute mountain sickness after rapid ascent to 3,450 m**

J Appl Physiol (1985), 123 (5) (2017), pp. 1228-1234

CrossRefView Record in ScopusGoogle Scholar

69

P. Bartsch, B. Merki, D. Hofstetter, M. Maggiorini, B. Kayser, O. Oelz**Treatment of acute mountain sickness by simulated descent: a randomised controlled trial**

BMJ, 306 (6885) (1993), pp. 1098-1101

CrossRefView Record in ScopusGoogle Scholar

70

H.R. Keller, M. Maggiorini, P. Bartsch, O. Oelz**Simulated descent v dexamethasone in treatment of acute mountain sickness: a randomised trial**
BMJ, 310 (6989) (1995), pp. 1232-1235
CrossRefView Record in ScopusGoogle Scholar

71

R.L. Taber**Protocols for the use of portable hyperbaric chambers for the treatment of high altitude disorders**
J Wild Med, 1 (1990), pp. 181-192
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

72

C.K. Grissom, R.C. Roach, F.H. Sarnquist, P.H. Hackett**Acetazolamide in the treatment of acute mountain sickness: clinical efficacy and effect on gas exchange**
Ann Intern Med, 116 (6) (1992), pp. 461-465
CrossRefView Record in ScopusGoogle Scholar

73

G. Ferrazzini, M. Maggiorini, S. Kriemler, P. Bartsch, O. Oelz**Successful treatment of acute mountain sickness with dexamethasone**
Br Med J (Clin Res Ed), 294 (6584) (1987), pp. 1380-1382
CrossRefView Record in ScopusGoogle Scholar

74

P.H. Hackett, R.C. Roach, R.A. Wood, R.G. Foutch, R.T. Meehan, D. Rennie, *et al.***Dexamethasone for prevention and treatment of acute mountain sickness**
Aviat Space Environ Med, 59 (10) (1988), pp. 950-954
View Record in ScopusGoogle Scholar

75

B.D. Levine, K. Yoshimura, T. Kobayashi, M. Fukushima, T. Shibamoto, G. Ueda**Dexamethasone in the treatment of acute mountain sickness**
N Engl J Med, 321 (25) (1989), pp. 1707-1713
View Record in ScopusGoogle Scholar

76

N.S. Harris, R.P. Wenzel, S.H. Thomas**High altitude headache: efficacy of acetaminophen vs. ibuprofen in a randomized, controlled trial**
J Emerg Med, 24 (4) (2003), pp. 383-387
ArticleDownload PDFView Record in ScopusGoogle Scholar

77

P.L. Johnson, C.C. Johnson, P. Poudyal, N. Regmi, M.A. Walmsley, B. Basnyat**Continuous positive airway pressure treatment for acute mountain sickness at 4240 m in the Nepal Himalaya**
High Alt Med Biol, 14 (3) (2013), pp. 230-233
CrossRefView Record in ScopusGoogle Scholar

78

M. Walmsley**Continuous positive airway pressure as adjunct treatment of acute altitude illness**
High Alt Med Biol, 14 (4) (2013), pp. 405-407
CrossRefView Record in ScopusGoogle Scholar

79
J.O. Stream, C.K. Grissom**Update on high-altitude pulmonary edema: pathogenesis, prevention, and treatment**
Wilderness Environ Med, 19 (4) (2008), pp. 293-303
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

80
P. Bartsch, H. Mairbaurl, M. Maggiorini, E.R. Swenson**Physiological aspects of high-altitude pulmonary edema**
J Appl Physiol, 98 (3) (2005), pp. 1101-1110
CrossRefView Record in ScopusGoogle Scholar

81
P. Bartsch, M. Maggiorini, H. Mairbaurl, P. Vock, E.R. Swenson**Pulmonary extravascular fluid accumulation in climbers**
Lancet, 360 (9332) (2002), p. 571
author reply –2
ArticleDownload PDFView Record in ScopusGoogle Scholar

82
I. Singh, C.C. Kapila, P.K. Khanna, R.B. Nanda, B.D. Rao**High-altitude pulmonary oedema**
Lancet, 191 (1965), pp. 229-234
ArticleDownload PDFView Record in ScopusGoogle Scholar

83
P. Bartsch, M. Maggiorini, M. Ritter, C. Noti, P. Vock, O. Oelz**Prevention of high-altitude pulmonary edema by nifedipine**
N Engl J Med, 325 (18) (1991), pp. 1284-1289
View Record in ScopusGoogle Scholar

84
C. Sartori, Y. Allemann, H. Duplain, M. Lepori, M. Egli, E. Lipp, *et al.***Salmeterol for the prevention of high-altitude pulmonary edema**
N Engl J Med, 346 (21) (2002), pp. 1631-1636
View Record in ScopusGoogle Scholar

85
M. Maggiorini, H.P. Brunner-La Rocca, S. Peth, M. Fischler, T. Bohm, A. Bernheim, *et al.***Both tadalafil and dexamethasone may reduce the incidence of high-altitude pulmonary edema: a randomized trial**
Ann Intern Med, 145 (7) (2006), pp. 497-506
CrossRefView Record in ScopusGoogle Scholar

86
C. Hohne, M.O. Krebs, M. Seiferheld, W. Boemke, G. Kaczmarczyk, E.R. Swenson**Acetazolamide prevents hypoxic pulmonary vasoconstriction in**

conscious dogs
J Appl Physiol, 97 (2) (2004), pp. 515-521
View Record in ScopusGoogle Scholar

87

L.A. Shimoda, T. Luke, J.T. Sylvester, H.W. Shih, A. Jain, E.R. Swenson**Inhibition of hypoxia-induced calcium responses in pulmonary arterial smooth muscle by acetazolamide is independent of carbonic anhydrase inhibition**
Am J Physiol Lung Cell Mol Physiol, 292 (4) (2007), pp. L1002-L1012
CrossRefView Record in ScopusGoogle Scholar

88

P.A. Pickerodt, R.C. Francis, C. Hohne, F. Neubert, S. Telalbasic, W. Boemke, *et al.***Pulmonary vasodilation by acetazolamide during hypoxia: impact of methyl-group substitutions and administration route in conscious, spontaneously breathing dogs**
J Appl Physiol (1985), 116 (7) (2014), pp. 715-723
CrossRefView Record in ScopusGoogle Scholar

89

L.J. Teppema, G.M. Balanos, C.D. Steinback, A.D. Brown, G.E. Foster, H.J. Duff, *et al.***Effects of acetazolamide on ventilatory, cerebrovascular, and pulmonary vascular responses to hypoxia**
Am J Respir Crit Care Med, 175 (3) (2007), pp. 277-281
View Record in ScopusGoogle Scholar

90

D.R. Liptzin, S.H. Abman, A. Giesenhagen, D.D. Ivy**An approach to children with pulmonary edema at high altitude**
High Alt Med Biol, 19 (1) (2018), pp. 91-98
CrossRefView Record in ScopusGoogle Scholar

91

E. Marticorena, H.N. Hultgren**Evaluation of therapeutic methods in high altitude pulmonary edema**
Am J Cardiol, 43 (2) (1979), pp. 307-312
ArticleDownload PDFView Record in ScopusGoogle Scholar

92

K. Zafren, J.T. Reeves, R. Schoene**Treatment of high-altitude pulmonary edema by bed rest and supplemental oxygen**
Wilderness Environ Med, 7 (2) (1996), pp. 127-132
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

93

U. Yanamandra, V. Nair, S. Singh, A. Gupta, D. Mulajkar, S. Yanamandra, *et al.***Managing high-altitude pulmonary edema with oxygen alone: results of a randomized controlled trial**
High Alt Med Biol, 17 (4) (2016), pp. 294-299
CrossRefView Record in ScopusGoogle Scholar

94

K. Freeman, M. Shalit, G. Stroh**Use of the Gamow bag by EMT-basic park rangers for treatment of high-altitude pulmonary edema and high-altitude cerebral edema**
Wilderness Environ Med, 15 (3) (2004), pp. 198-201
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

95

O. Oelz, M. Maggiorini, M. Ritter, U. Waber, R. Jenni, P. Vock, *et al.***Nifedipine for high altitude pulmonary oedema**
Lancet, 2 (8674) (1989), pp. 1241-1244
ArticleDownload PDFView Record in ScopusGoogle Scholar

96

R. Deshwal, M. Iqbal, S. Basnet**Nifedipine for the treatment of high altitude pulmonary edema**
Wilderness Environ Med, 23 (1) (2012), pp. 7-10
ArticleDownload PDFView Record in ScopusGoogle Scholar

97

P.J. Fagenholz, J.A. Gutman, A.F. Murray, N.S. Harris**Treatment of high altitude pulmonary edema at 4240 m in Nepal**
High Alt Med Biol, 8 (2) (2007), pp. 139-146
CrossRefView Record in ScopusGoogle Scholar

98

B.E. Jones, S. Stokes, S. McKenzie, E. Nilles, G.J. Stoddard**Management of high altitude pulmonary edema in the Himalaya: a review of 56 cases presenting at Pheriche medical aid post (4240 m)**
Wilderness Environ Med, 24 (1) (2013), pp. 32-36
ArticleDownload PDFView Record in ScopusGoogle Scholar

99

R.B. Schoene, R.C. Roach, P.H. Hackett, G. Harrison, W.J. Mills Jr**High altitude pulmonary edema and exercise at 4,400 meters on Mount McKinley. Effect of expiratory positive airway pressure**
Chest, 87 (3) (1985), pp. 330-333
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

100

I. Singh, M. Lal, P.K. Khanna, N.T. Mathew**Augmentation of frusemide diuresis by morphine in high altitude pulmonary oedema**
Br Heart J, 29 (5) (1967), pp. 709-713
CrossRefView Record in ScopusGoogle Scholar

101

C. Siebenmann, K.E. Bloch, C. Lundby, Y. Nussbamer-Ochsner, M. Schoeb, M. Maggiorini**Dexamethasone improves maximal exercise capacity of individuals susceptible to high altitude pulmonary edema at 4559 m**
High Alt Med Biol, 12 (2) (2011), pp. 169-177
CrossRefView Record in ScopusGoogle Scholar

102

M. Urner, I.K. Herrmann, C. Booy, B.R. Z'Graggen, M. Maggiorini, B. Beck-Schimmer**Effect of hypoxia and dexamethasone on inflammation and ion transporter function in pulmonary cells**

Clin Exp Immunol, 169 (2) (2012), pp. 119-128

CrossRefView Record in ScopusGoogle Scholar

103

R.C. Roach, P.H. Hackett, O. Oelz, P. Bartsch, A.M. Luks, M.J. MacInnis, *et al.***The 2018 Lake Louise acute mountain sickness score**

High Alt Med Biol, 19 (1) (2018), pp. 4-6

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Wilderness Medical Society Clinical Practice Guidelines for the Prevention and Treatment of Frostbite: 2019 Update

Scott E.McIntosh MD, MPH[1] Luanne Freer MD[23] Colin K.Grissom MD[4] Paul S.Auerbach MD, MS[5] George W.Rodway PhD, APRN[6] Amalia Cochran MD[7] Gordon G. Giesbrecht MD[8] Marion McDevitt DO, MPH[9] Christopher H.Imray MD[10] Eric L.Johnson MD[11] Prativa Pandey MD[12] Jennifer Dow MD[13] Peter H.Hackett MD[1415]

The Wilderness Medical Society convened an expert panel to develop a set of evidence-based guidelines for prevention and treatment of frostbite. We present a review of pertinent pathophysiology. We then discuss primary and secondary prevention measures and therapeutic management. Recommendations are made regarding each treatment and its role in management. These recommendations are graded on the basis of the quality of supporting evidence and balance between the benefits and risks or burdens for each modality according to methodology stipulated by the American College of Chest Physicians. This is an updated version of the guidelines published in 2014.

Keywords

hypothermia
rewarming
aloe vera
thrombolysis
tPA
iloprost

Introduction

The Wilderness Medical Society (WMS) convened an expert panel to develop a set of evidence-based guidelines for prevention and treatment of frostbite to guide clinicians and first responders and disseminate knowledge about best practices in this area of clinical care. We present the main prophylactic and therapeutic modalities and make recommendations about their role in injury management. Recommendations are graded on the basis of the quality of supporting evidence and balance between the benefits and risks or burdens for each modality. We then provide suggested approaches for prevention and management that incorporate these recommendations.

The original expert panel was convened at the 2010 Annual Winter Meeting of the WMS in Park City, UT. Members were selected on the basis of their clinical or research experience. Relevant articles were identified through the MEDLINE database using the search terms frostbite, frostbite management, prehospital frostbite treatment, prehospital frostbite management, frostbite prevention, first aid frostbite treatment, and first aid frostbite and were restricted to the English language. Studies in these categories were reviewed, and level of evidence was assessed. The panel used a consensus approach to develop recommendations regarding each modality and graded each recommendation according to criteria stipulated by the American College of Chest Physicians statement on grading recommendations and strength of evidence in clinical guidelines (online Supplementary Table 1).[1] This is an updated version of the guidelines published in 2014.[2]

Pathophysiology of frostbite

Frostbite is a freezing injury that may be divided into 4 overlapping pathologic phases: prefreeze, freeze–thaw, vascular stasis, and late ischemic. The prefreeze phase consists of tissue cooling with accompanying vasoconstriction and ischemia and without actual ice crystal formation. Neuronal cooling and ischemia produce hyperesthesia or paresthesia. In the freeze–thaw phase, ice crystals form intracellularly (during a more rapid-onset freezing injury) or extracellularly (during a slower freeze), causing protein and lipid derangement, cellular electrolyte shifts, cellular dehydration, cell membrane lysis, and cell death.[3] The thawing process may initiate ischemia, reperfusion injury, and an inflammatory response. In the vascular stasis phase, vessels fluctuate between constriction and dilation; blood may leak from vessels or coagulate within them.[4, 5, 6] The late ischemic phase results from progressive tissue ischemia and infarction from a cascade of events, including inflammation mediated by thromboxane A_2, prostaglandin F_2alpha, bradykinin, and histamine; intermittent vasoconstriction of arterioles and venules; continued reperfusion injury; showers of emboli coursing through the microvessels[7, 8]; and thrombus formation in larger vessels.[9] Destruction of the microcirculation is the main factor leading to cell death.[10] The initial cellular damage caused by ice crystals and the subsequent postthawing processes are made worse if refreezing follows thawing of injured tissues.[11, 12]

Classification of frostbite

Frostnip is superficial nonfreezing cold injury associated with intense vasoconstriction on exposed skin, usually cheeks, ears, or nose. Ice crystals, appearing as frost, form on the skin surface. Frostnip is distinct from and may precede frostbite. With frostnip, ice crystals do not form within the tissue and tissue loss does not occur. Numbness and pallor resolve quickly after warming the skin with appropriate clothing, direct contact, breathing with cupped hands over the nose, or gaining shelter. No long-term damage occurs. Frostnip signals conditions favorable for frostbite; appropriate action should be undertaken immediately to prevent injury.

Frostbite has historically been divided into 4 tiers or “degrees” of injury following the classification scheme for thermal burn injury. These classifications are based on acute physical findings and advanced imaging after rewarming.[13] The classifications can be difficult to assess in the field before rewarming because the still-frozen tissue is hard, pale, and anesthetic. An alternate 2-tiered classification more appropriate for field use (after rewarming but before imaging) is suggested with the following the 4-tier classification:

- First-degree frostbite causes numbness and erythema. A white or yellow, firm, and slightly raised plaque develops in the area of injury. No gross tissue infarction occurs; there may be slight epidermal sloughing. Mild edema is common.
- Second-degree frostbite injury causes superficial skin vesiculation; a clear or milky fluid is present in the blisters, surrounded by erythema and edema.
- Third-degree frostbite causes deeper hemorrhagic blisters, indicating that the injury has extended into the reticular dermis and beneath the dermal vascular plexus.
- Fourth-degree frostbite extends completely through the dermis and involves the comparatively avascular subcutaneous tissues, with necrosis extending into muscle and bone.

For field classification, after spontaneous or formal rewarming but before imaging, we favor the following 2-tier classification scheme:

- *Superficial*—no or minimal anticipated tissue loss, corresponding to first- and second-degree injury.
- *Deep*—anticipated tissue loss, corresponding to third- and fourth-degree injury.

Severity of frostbite may vary within a single extremity.

Once thawing occurs and a patient reaches a field clinic or hospital, one can further classify or characterize the frostbite injury via 2 additional methods. The Hennepin score[14] uses a system similar to that for measuring burns by total body surface area. The effect of treatment can then be quantified retrospectively. The Cauchy classification method[13] measures extent of frostbite anatomically using the following grades: 0—no lesion; 1—lesion on the distal phalanx; 2—lesion on the middle phalanx or proximal phalanx for the thumb/big toe; 3—lesion on the proximal phalanx except for the thumb/big toe; 4—lesion on the metacarpal/metatarsal; 5—lesion on the carpal/tarsal. Although not validated, grades correlate well with bone scans and clinical outcomes and may assist caregivers in predicting tissue loss. The Cauchy classification method may assist caregivers in predicting amputation risk, which helps to inform evacuation

decisions. For example, a necrotic fingertip (labeled grade 4 by the 4-tiered system but unlikely to involve significant amputation) would be designated a grade 1 on the Cauchy classification method, designating lower severity. Higher grades in the Cauchy classification method designate more proximal injuries with greater risk for functionally important amputation.

Prevention

The adage that “prevention is better than treatment” is especially true for frostbite, which is typically preventable and often not improved by treatment. Underlying medical problems may increase risk of frostbite, so prevention must address both environmental and health-related aspects. Frostbite injury occurs when tissue heat loss exceeds the ability of local tissue perfusion to prevent freezing of soft tissues (blood flow delivers heat). One must both ensure adequate perfusion and minimize heat loss to prevent frostbite. The adventurer should recognize cold-induced “numbness” as a warning that frostbite injury may be imminent if protective or avoidance measures are not taken to decrease tissue cooling. Subsequent loss of sensation does not mean the situation has improved; rather, receptors and nerves are not conducting pain/cold signals because they are nearing the freezing point.

MAINTAINING PERIPHERAL PERFUSION

Preventive measures to ensure local tissue perfusion include: 1) maintaining adequate core temperature and body hydration; 2) minimizing the effects of known diseases, medications, and substances (including awareness and symptoms of alcohol and drug use) that might decrease perfusion; 3) covering all skin and the scalp to insulate from the cold; 4) minimizing blood flow restriction, such as occurs with constrictive clothing, footwear, or immobility; 5) ensuring adequate nutrition; and 6) using supplemental oxygen in severely hypoxic conditions (eg, >7500 m). ***Recommendation Grade:*** 1C.

EXERCISE

Exercise is a specific method to maintain peripheral perfusion. Exercise enhances the level and frequency of cold-induced peripheral vasodilation. In one study, exercise resulted in cold-induced peripheral vasodilation in the toes of 58% of exercising subjects vs 28% in nonexercising subjects.[15] Another study found increased skin temperature in the hands during exercise.[16] However, using exercise to increase warmth can lead to exhaustion, with subsequent profound systemic heat loss should exhaustion occur. Recognizing this caveat, exercise and its associated elevation in core and peripheral temperatures can be protective in preventing frostbite. ***Recommendation Grade:*** 1B.

PROTECTION FROM COLD

Measures should be taken to minimize exposure of tissue to cold. These measures include the following: 1) avoiding environmental conditions that predispose to frostbite, specifically below -15°C, even with low wind speeds[17]; 2) protecting skin from moisture, wind, and cold; 3) avoiding perspiration or wet extremities; 4) increasing insulation and skin protection (eg, by adding clothing layers, changing from gloves to mitts); 5) ensuring beneficial behavioral responses to changing environmental conditions (eg, not being under the influence of illicit drugs, alcohol, or extreme hypoxemia)[18]; 6) using chemical hand and foot warmers and electric foot warmers to maintain peripheral warmth (note: warmers should be close to body temperature before being activated and must not be placed directly against skin or constrict flow if used within a boot); 7) regularly checking oneself and the group for extremity numbness or pain and warming the digits and/or extremities as soon as possible if there is concern that frostbite may be developing; 8) recognizing frostnip or superficial frostbite before it becomes more serious; and 9) minimizing duration of cold exposure. Emollients do not protect against—and might even increase—risk of frostbite.[19] The time that a digit or extremity can remain numb before developing frostbite is unknown; thus, digits or extremities with paresthesia should be warmed as soon as possible. An extremity at risk for frostbite (eg, numbness, poor dexterity, pale color) should be warmed with adjacent body heat from the patient or a companion, using the axilla or abdomen. ***Recommendation Grade:*** 1C.

Field treatment and secondary prevention

If a body part is frozen in the field, the frozen tissue should be protected from further damage. Remove jewelry or other constrictive extraneous material from the body part. Do not rub or apply ice or snow to the affected area.[20]

REFREEZING INJURY

A decision must be made whether to thaw the tissue. If environmental conditions are such that thawed tissue could refreeze, it is safer to keep the affected part frozen until a thawed state can be maintained. Prostaglandin and thromboxane release associated with the freeze–thaw cycle[20, 21, 22] causes vasoconstriction, platelet aggregation, thrombosis, and, ultimately, cellular injury. Refreezing thawed tissue further increases release of these mediators, and significant morbidity may result. One must absolutely avoid refreezing if field thawing occurs. ***Recommendation Grade:*** 1B.

SPONTANEOUS OR PASSIVE THAWING

Most frostbite thaws spontaneously and should be allowed to do so if rapid rewarming (described in the following) cannot be readily achieved. Do not purposefully keep tissue below freezing temperatures because this will increase the duration that the tissue is frozen and might result in more proximal freezing

and greater morbidity. If environmental and situational conditions allow for spontaneous or slow thawing, tissue should be allowed to thaw. ***Recommendation Grade:*** 1C.

Strategies for 2 scenarios are presented:

Scenario 1: The frozen part has the potential for refreezing and is not actively thawed.

Scenario 2: The frozen part is thawed and kept warm without refreezing until evacuation is completed.

THERAPEUTIC OPTIONS FOR BOTH SCENARIOS

Many of these guidelines parallel the State of Alaska cold injuries guidelines.[23] Therapeutic options include the following:

Treatment of hypothermia

No studies examine concurrent hypothermia and frostbite. Hypothermia frequently accompanies frostbite and causes peripheral vasoconstriction that impairs blood flow to the extremities. Mild hypothermia may be treated concurrently with frostbite injury. Moderate and severe hypothermia should be treated effectively before treating frostbite injury. ***Recommendation Grade:*** 1C.

Hydration

Vascular stasis can result from frostbite injury. No studies have specifically examined the effect of hydration status on frostbite outcomes, but it is believed that appropriate hydration and avoidance of hypovolemia are important for frostbite recovery. Oral fluids may be given if the patient is alert, capable of purposeful swallowing, and not vomiting. If the patient is nauseated or vomiting or has an altered mental status, IV normal saline should be given to maintain normal urine output. Intravenous fluids should optimally be warmed (minimally to 37°C but preferably to 40 to 42°C with a method that has been proven to be effective in the present environmental conditions) before infusion and be infused in small (eg, 250 mL), rapid boluses because slow infusion will result in fluid cooling and even freezing as it passes through the tubing. Fluid administration should be optimized to prevent clinical dehydration. ***Recommendation Grade:*** 1C.

Low molecular weight dextran

Intravenous low molecular weight dextran (LMWD) decreases blood viscosity by preventing red blood cell aggregation and formation of microthrombi and can be given in the field once it has been warmed. In some animal studies, the extent of tissue necrosis was found to be significantly less than in control subjects when LMWD was used[24, 25, 26, 27] and was more beneficial if given early.[28] In one animal trial,[28] tissue in the LMWD group thawed slightly more rapidly, but overall tissue loss was no different from that of control animals. Give a test dose before administration because of the low risk of anaphylaxis. This low risk of anaphylaxis

should not deter administration. The slight risk of bleeding is minimal, and benefits seem to outweigh this risk; however, availability is limited in the United States. The use of LMWD has not been evaluated in combination with other treatments such as thrombolytics. LMWD should be given if the patient is not being considered for other systemic treatments, such as thrombolytic therapy. ***Recommendation Grade:*** 2C.

Ibuprofen

Nonsteroidal anti-inflammatory drugs (NSAIDs) block the arachidonic acid pathway and decrease production of prostaglandins and thromboxanes.[29] These mediators can lead to vasoconstriction, dermal ischemia, and further tissue damage. No studies have demonstrated that any particular anti-inflammatory agent or dosing is clearly related to outcome. Aspirin has been proposed as an option and is used in many parts of the world for anti-inflammatory and platelet inhibition effects. One rabbit ear model study showed 23% tissue survival with aspirin vs 0% in the control group.[30] However, aspirin theoretically blocks production of certain prostaglandins that are beneficial to wound healing,[31] and the authors of the rabbit ear model study recommend ibuprofen in their treatment algorithm. No studies specifically compare aspirin with ibuprofen in frostbite. Ibuprofen should be started in the field at a dose of 12 $mg \cdot kg^{-1}$ per day divided twice daily (minimum to inhibit harmful prostaglandins[29]) to a maximum of 2400 $mg \cdot d^{-1}$ divided 4 times daily. ***Recommendation Grade:*** 2C.

SPECIFIC RECOMMENDATIONS—SCENARIO 1

Therapeutic options for frostbite in Scenario 1 (no active thawing) include the following:

Dressings

No evidence supports applying a dressing to a frostbitten part intended to remain frozen until rewarming can safely be achieved. If this is considered, it should only be done if practical and will not interfere with mobility. Bulky, clean, and dry gauze or sterile cotton dressings should be applied to the frozen part and between the toes and fingers. ***Recommendation Grade:*** 2C.

Ambulation and protection

If at all possible, a frozen extremity should not be used for walking, climbing, or other maneuvers until definitive care is reached. If use of the frozen extremity for mobility is considered, a risk-benefit analysis must consider the potential for further trauma and possible poorer outcome. Although it is reasonable to walk on a foot with frostbitten toes for evacuation purposes, it is inadvisable to walk on an entirely frostbitten foot because of the potential for resulting morbidity. This risk is theoretical and based on the panel's opinion. Mills described frostbite patients who ambulated on frozen extremities for days and sustained no or limited

amputation.[32] If using a frozen extremity for locomotion or evacuation is unavoidable, the extremity should be padded, splinted, and kept as immobile as possible to minimize additional trauma. ***Recommendation Grade:*** 2C.

SPECIFIC RECOMMENDATIONS—SCENARIO 2

Therapeutic options for frostbite in Scenario 2 (thawing and continued warming) include the following:

Rapid field rewarming of frostbite

Field rewarming by warm water bath immersion can and should be performed if the proper resources are available and definitive care is more than 2 h distant. Other heat sources (eg, fire, space heater, oven, heated rocks) should be avoided because of the risk of thermal burn injury. Rapid rewarming by water bath has been found to result in better outcomes than slow rewarming.[20, 27, 32] Field rewarming should only be undertaken if the frozen part can be kept thawed and warm until the victim arrives at definitive care. Water should be heated to 37 to 39°C (98.6 to 102.2°F), using a thermometer to maintain this range.[33] If a thermometer is not available, a safe water temperature can be determined by placing a caregiver's uninjured hand in the water for at least 30 s to confirm that the water temperature is tolerable and will not cause burn injury. Circulation of water around the frozen tissue will help maintain correct temperature.[34, 35] Because the water may cool quickly after the rewarming process is started, the water should be continuously and carefully warmed to the target temperature. If the frozen part is being rewarmed in a pot, skin should not press against the bottom or sides. Rewarming is complete when the involved part takes on a red or purple appearance and becomes soft and pliable to the touch. This is usually accomplished in approximately 30 min, but the time is variable depending on the extent and depth of injury. The affected tissues should be allowed to air dry or be gently dried with a blotting technique (not rubbing) to minimize further damage. Under appropriate circumstances, this method of field rewarming is the first definitive step in frostbite treatment. ***Recommendation Grade:*** 1B.

Antiseptic solution

Most injuries do not become infected, but adding an antiseptic solution (eg, povidone-iodine, chlorhexidine) to the rewarming water has theoretical benefits of reducing skin bacteria. Evidence for this practice does not exist for frostbite care, however. Adding an antiseptic solution to the water while rewarming is unlikely to be harmful and might reduce the risk for cellulitis if severe edema is present in the affected extremity. ***Recommendation Grade:*** 2C.

Pain control

During rewarming, pain medication (eg, NSAIDs or an opiate analgesic) should be given to control symptoms as dictated by individual patient situation. ***Recommendation Grade:*** 1C.

Spontaneous or passive thawing

According to the foregoing guidelines, rapid rewarming is strongly recommended. If field rewarming is not possible, spontaneous or slow thawing should be allowed. Slow rewarming is accomplished by moving to a warmer location (eg, tent or hut) and warming with adjacent body heat from the patient or a caregiver, as previously described. The expert panel agrees that slow thawing is a reasonable course of action to initiate the rewarming process if it is the only means available. ***Recommendation Grade:*** 1C.

Debridement of blisters

Debridement of blisters should not be routinely performed in the field. If a clear, fluid-filled blister is tense and at high risk for rupture during evacuation, blister aspiration and application of a dry gauze dressing should be performed in the field to minimize infection risk. Hemorrhagic bullae should not be aspirated or debrided in the field. These recommendations are common practice but lack evidence beyond case series.[29] ***Recommendation Grade:*** 2C.

Topical aloe vera

Aloe vera ointment has been shown in an observational study[36] and an animal model[30] to improve frostbite outcome by reducing prostaglandin and thromboxane formation. Topical agents do not penetrate far into tissues, however, so aloe vera is theoretically only beneficial for superficially injured areas. The study supporting the benefit of aloe vera examined its application on unroofed blebs where it would be able to penetrate underlying tissue. Topical aloe vera should be applied to thawed tissue before application of dressings. ***Recommendation Grade:*** 2C.

Dressings

Bulky, dry gauze dressings should be applied to the thawed parts for protection and wound care. Substantial edema should be anticipated, so circumferential dressings should be wrapped loosely to allow for swelling without placing pressure on the underlying tissue. ***Recommendation Grade:*** 1C.

Ambulation and protection

A risk-benefit analysis must consider the potential for further trauma and, ultimately, potentially higher morbidity if a thawed part is used for ambulation. For example, it might be reasonable to walk on a foot with thawed toes for evacuation purposes, but it is inadvisable to walk on a recently thawed frostbitten foot because of the potential resulting morbidity. Very little evidence is available to guide recommendations. In one study, mobilization within 72 h after thawing did not affect tissue loss, complications, or hospital length of stay.[37] After the rewarming process, swelling should be anticipated. If passive thawing has occurred, boots (or inner boots) may need to be worn continuously to compress swelling. Boots that were removed for active rewarming may not be able to be re-

donned if tissue swelling has occurred during the warming process. The panel's clinical experience supports the concept that a recently thawed extremity should ideally not be used for walking, climbing, or other maneuvers and should be protected to prevent further trauma.[36], [38]***Recommendation Grade:*** 2C.

Elevation of extremity

If possible, the thawed extremity should be elevated above the level of the heart, which might decrease formation of dependent edema. ***Recommendation Grade:*** 1C.

Oxygen

Recovery of thawed tissue partly depends on the level of tissue oxygenation in the postfreezing period. One small study that measured hand temperature at normobaric hypoxia found decreased skin temperatures with decreasing F_IO_2.[39] However, hyperoxia has been found to cause vasoconstriction in the extremities[40]; therefore, oxygen should not be applied routinely to patients who are not hypoxic. Although evidence is lacking to support use of supplemental oxygen for frostbite, oxygen may be delivered by face mask or nasal cannula if the patient is hypoxic (oxygen saturation <88%) or at high altitude above 4000 m. ***Recommendation Grade:*** 2C.

For a summary of the suggested approach to the field treatment of frostbite, see Table 1.

Table 1. Summary of field treatment of frostbite (>2 h from definitive care)

Treat hypothermia or serious trauma
1. Remove jewelry or other extraneous material from the body part.
2. Rapidly rewarm in water heated and maintained between 37 and 39°C (98.6 and 102.2°F) until area becomes soft and pliable to the touch (approximately 30 min); allow spontaneous or passive thawing if rapid rewarming is not possible.
3. Ibuprofen (12 mg·kg^{-1} per day divided twice daily) if available.
4. Pain medication (eg, opiate) as needed.
5. Air dry (ie, do not rub at any point).
6. Protect from refreezing and direct trauma.
7. Apply topical aloe vera cream or gel if available.
8. Dry, bulky dressings.
9. Elevate the affected body part if possible.
10. Systemic hydration.
11. Avoid ambulation on thawed lower extremity (unless only distal toes are affected).

Immediate medical therapy—hospital (or high-level field clinic)

Once the patient reaches the hospital or field clinic, a number of treatments should be initiated. After reaching the hospital or field clinic, potential therapeutic options for frostbite include the following:

TREATMENT OF HYPOTHERMIA

Similar recommendations apply to hospital or field clinic treatment of hypothermia before frostbite treatment (see previous). ***Recommendation Grade:*** 1C.

HYDRATION

Similar recommendations apply in the hospital or field clinic regarding hydration (see previous). ***Recommendation Grade:*** 1C.

LOW MOLECULAR WEIGHT DEXTRAN

Similar recommendations apply in the hospital or field clinic regarding LMWD (see previous). ***Recommendation Grade:*** 2C.

RAPID REWARMING OF FROZEN TISSUES

Frozen tissue should be assessed to determine whether spontaneous thawing has occurred. If tissue is completely thawed, further rewarming will not be beneficial. Rapid rewarming should be undertaken according to the field protocol described previously if the tissue remains partially or completely frozen. ***Recommendation Grade:*** 1B.

MANAGEMENT OF BLISTERS

Clear or cloudy blisters contain prostaglandins and thromboxanes that may damage underlying tissue. Hemorrhagic blisters are thought to signify deeper tissue damage extending into the dermal vascular plexus. Common practice is to drain clear blisters (eg, by needle aspiration) while leaving hemorrhagic blisters intact.[34, 36, 38, 41, 42] Although this approach to frostbite blister management is recommended by many authorities, comparative studies have not been performed and data are insufficient to make absolute recommendations. Some authors argue that unroofing blisters might lead to the desiccation of exposed tissue and that blisters should only be removed if they are tense, likely to break or be infected, or interfere with the patient's range of motion.[43] In a remote field situation, draining or unroofing blisters may not be under control of the provider. Blisters most often will have been broken by the patient's boots. In this case, the most important treatment is applying aloe vera and a sterile dressing to the unroofed blister. Debridement or aspiration of clear, cloudy, or tense blisters is at the provider's discretion, with consideration of patient circumstances, until better evidence becomes available. ***Recommendation Grade:*** 2C.

TOPICAL ALOE VERA

Topical aloe vera cream or gel should be applied to the thawed tissue before application of dressings. Aloe vera is reapplied at each dressing change or every 6 h.[36] ***Recommendation Grade:*** 2C.

SYSTEMIC ANTIBIOTICS

Frostbite is not an inherently infection-prone injury. Therefore, antibiotic administration specifically for preventing infection during or after frostbite injury is not supported by evidence. Some authorities reserve antibiotics for situations when edema occurs after thawing because of the notion that edema increases skin susceptibility to infection by gram-positive bacteria.[38] However, this practice is not based on evidence. Systemic antibiotics, either oral or parenteral, should be administered to patients with significant trauma, other potential infectious sources, or signs and symptoms of cellulitis or sepsis. ***Recommendation Grade:*** 1C.

TETANUS PROPHYLAXIS

Tetanus prophylaxis should be administered according to standard guidelines. ***Recommendation Grade:*** 1C.

IBUPROFEN

If NSAIDs have not been initiated in the field, ibuprofen should be administered at a dose of 12 mg· kg^{-1} divided twice daily (to inhibit harmful prostaglandins but remain less injurious to the gastrointestinal system[29]) until the frostbite wound is healed or surgical management occurs (typically for 4 to 6 wk).
Recommendation Grade: 2C.

THROMBOLYTIC THERAPY

The goal of thrombolytic therapy in frostbite injury is to lyse and clear microvascular thromboses. For deep frostbite injury with potential significant morbidity, angiography and use of either IV or intra-arterial tissue plasminogen activator (tPA) within 24 h of thawing may salvage some or all tissue at risk. A retrospective, single-center review by Bruen et al[44] demonstrated reduction in digital amputation rates from 41% in those patients who did not receive tPA to 10% in patients receiving tPA within 24 h of injury. The 20-y series presented by the Regions Hospital group found that two-thirds of patients who received intra-arterial tPA responded well and that amputation rate correlated closely with angiographic findings.[45] The Massachusetts General Hospital group has proposed a screening and treatment tool for thrombolytic management of frostbite based on a case report and their evaluation of the Utah and Minneapolis experiences.[46] Twomey et al[47] from Hennepin County Medical Center have developed a specific protocol based on a small group of good outcomes with intravenous tPA. Further study is needed to compare intra-arterial vs IV tPA on tissue salvage and functional outcome. Animal studies demonstrate benefit from thrombolytics.[48]

When considering using a thrombolytic, a risk-benefit analysis should be performed. Only deep injuries with potential for significant morbidity (eg, extending into the proximal interphalangeal joints of digits) should be considered for thrombolytic therapy. Potential risks of tPA include systemic and catheter site bleeding, compartment syndrome, and failure to salvage tissue. The long-term, functional consequences of digit salvage using tPA have not been fully evaluated.

Thrombolytic treatment should be undertaken in a facility familiar with the technique and with intensive care monitoring capabilities. If a frostbite patient is being cared for in a remote area, transfer to a facility with tPA administration and monitoring capabilities should be considered if tPA can be started within 24 h of tissue thawing. Time to thrombolysis appears to be very important, with best outcomes within 12 h and ideally as soon as possible. Recent work from Hennepin County has found that each hour of delay of thrombolytic therapy results in a 28% decrease in salvage.[49] Rare use of tPA in the field has shown variable success[50] and should only be undertaken with extreme caution because bleeding complications may be impossible to detect and treat. If other treatment options are limited or unavailable, tPA should be considered for field treatment only of severe frostbite extending to the proximal interphalangeal joint or more proximally (eg, Cauchy classification grade 3 to 5).

Method of administration

Dosing is typically a 3 mg bolus (30 mL of 0.1 mg·mL^{-1} solution) followed by infusion of 1 mg·mL^{-1} (10 mL·h^{-1}) until specialists (eg, vascular, burn, radiology) recommend discontinuation. Heparin is administered concurrently: 500 units·h^{-1}.[51]

Intra-arterial angiography or IV pyrophosphate scanning should be used to evaluate the initial injury and monitor progress after tPA administration as directed by local protocol and resources. As of the end of 2018,[52] the following have been published on tPA use in frostbite: 1 randomized controlled prospective trial (tPA plus iloprost, 16 patients),[53] 3 retrospective cohort studies (59 patients),[44, 49, 54] 8 retrospective case series (130 patients),[47,55, 56, 57, 58, 59, 60, 61] and 3 case reports.[46, 62, 63] Although further studies are needed to determine the absolute efficacy of tPA for frostbite injury and to compare intra-arterial tPA to IV prostacyclin, we recommend IV or intra-arterial tPA within 24 h of injury as a reasonable choice in an environment with appropriate monitoring capabilities. ***Recommendation Grade:*** 1C.

IMAGING

In patients with delayed presentation (4-24 h from the time of the frostbite thawing), noninvasive imaging with technetium pyrophosphate[13] or magnetic resonance angiography[64] can be used at an early stage to predict the likely levels of tissue viability for amputation. Cauchy et al[13] described the combination of a clinical scoring system and technetium scanning to successfully predict subsequent level of amputation on day 2 after frostbite rewarming. Single photon emission computed tomography (CT)/CT combines the anatomic precision of CT with the functional vascular information obtained from multiphase bone

scintigraphy. Kraft et al used single photon emission CT/CT for 7 patients with frostbite and found it improved surgical planning for deep frostbite injuries by enabling early and precise anatomic localization of nonviable tissues.[65, 66]

If available, appropriate imaging should be used to assess tissue viability and guide timing and extent of amputation. ***Recommendation Grade:*** 1C.

Other potential useful imaging techniques include Doppler ultrasound[55]; triple phase technitium[58, 67]; indocyanine green microangiography[68]; and thermal imaging.[39] Although some of these techniques show potential, further studies are required to determine their exact role.

ILOPROST

Iloprost, a prostacyclin (PGI2) analogue, is a potent vasodilator that also inhibits platelet aggregation, down-regulates lymphocyte adhesion to endothelial cells,[69] and may have fibrinolytic activity.[70] Intravenous iloprost was first used for treatment of frostbite by Groechenig in 1994, in 5 patients with second- and third-degree frostbite. He infused iloprost daily, starting at 0.5 ng·kg^{-1} and increasing to 2.0 ng·kg^{-1} total dose over 3 d, and then continued for between 14 and 42 d.[71] Recovery without amputation was achieved in all patients.

A randomized trial by Cauchy et al assessed the efficacy of aspirin plus: 1) buflomedil, an alpha-blocker vasodilator; 2) iloprost; or 3) intravenous tPA plus iloprost.[53] Forty-seven patients with severe frostbite, with 407 digits at risk, were randomly assigned to 8 d of treatment with the 3 different regimens. Iloprost alone (0% amputation rate) was found superior to tPA plus iloprost (19%) and buflomedil (60%) groups. A limitation of this study was that ischemia was not documented with angiography or technetium scanning before treatment; groups were randomized according to clinical severity.

A Canadian study documented full recovery of grade 3 frostbite when iloprost was started within 48 h of injury in 2 long distance runners.[72] In a Finnish study, iloprost was partially beneficial with digit salvage rate of 78% in 4 persons: 2 with contraindication for tPA, 1 with failed tPA therapy, and 1 with vasospasm without thrombosis on angiography.[55] One patient with minimal response to tPA had complete reperfusion with iloprost.

Despite the limitations of these initial studies, iloprost has shown consistently favorable effects.[73] Extending the treatment window, Pandey et al[74] reported good results with iloprost therapy up to 72 h after injury. In 5 Himalayan climbers with 34 digits at risk, 5 d of daily iloprost infusion produced excellent outcomes in 4 of 5 patients. Treatment delayed beyond 72 h has not been beneficial except in 1 patient.[74, 75] No serious side effects have been noted in these studies.

Intravenous iloprost should be considered first-line therapy for grade 3 and 4 frostbite <72 h after injury, when tPA is contraindicated, and in austere environments where tPA infusion is considered risky or evacuation to a treatment facility will be delayed. Field use of both iloprost and IV tPA has been advocated to reduce delay in treatment for mountaineers who will invariably take >48 h for evacuation to a hospital.[50] In these situations, iloprost may be the safer

alternative. The IV form of iloprost is not approved by the US Food and Drug Administration, however. Consider iloprost for deep frostbite to or proximal to the proximal interphalangeal joint; within 48 h after injury, especially if angiography is not available; or with contraindications to thrombolysis. Expedition physicians should consider adding iloprost to their medical armamentarium, especially if it can be safely sourced and when treatment is occurring outside of the United States. ***Recommendation Grade:*** 1B.

Method of administration

Iloprost dosage is given IV via controlled infusion or syringe pump. Iloprost is mixed with normal saline or dextrose in water. On days 1 through 3, start at an initial rate of 0.5 $ng \cdot kg^{-1} \cdot min^{-1}$, then gradually increase by 0.5 $ng \cdot kg^{-1} \cdot min^{-1}$ at 30-min intervals to a maximum dose of 2.0 $ng \cdot kg^{-1} \cdot min^{-1}$. If intolerable side effects (nausea, headache, flushing) emerge or blood pressure or heart rate are outside normal limits, reduce the rate by 0.5 $ng \cdot kg^{-1} \cdot min^{-1}$ until side effects are tolerable or vital signs normalize. Mild and tolerable side effects can be treated symptomatically, whereas hypotension or severe symptoms require dose reduction. Continue the highest dose achieved or a maximum of 2.0 $ng \cdot kg^{-1} \cdot min^{-1}$ for 6 h total. For days 4 through 5, start directly at the highest/optimum rate or a maximum of 2.0 $ng \cdot kg^{-1} \cdot min^{-1}$ for 6 h daily.[51] Some protocols recommend up to 8 d of treatment; the first dose is considered the most important.

HEPARIN

No evidence supports use of low molecular weight heparin or unfractionated heparin for initial management of frostbite in the field or hospital, although climbers and practitioners in many regions use these medications. Evidence supports use of heparin as adjunctive therapy in tPA protocols, as described previously. Heparin has been used in conjunction with iloprost as well; the 5 patients in the 1994 Groechenig iloprost study,[71] the 1 Israeli traveler with excellent outcome in the Kathmandu study,[74] and 4 patients in the Finnish study[55] were treated with low molecular weight heparin (enoxaparin) in addition to iloprost. Whether low molecular weight heparin offers additional benefit when combined with iloprost requires further investigation; currently data are insufficient for a recommendation on this combination. ***Recommendation Grade:*** Not recommended as monotherapy owing to insufficient data.

OTHER VASODILATOR THERAPY

Vasodilators, such as prostaglandin E_1,[76] nitroglycerin,[46] pentoxifylline,[77, 78] phenoxybenzamine, nifedipine, reserpine,[79, 80] and buflomedil,[53, 81, 82] have been used as primary and adjunctive therapies for treatment of frostbite. In addition to vasodilation, some of these agents might also prevent platelet aggregation and microvascular occlusion. Sheridan et al[46] recommend intra-arterial infusion of nitroglycerin during angiography before tPA infusion. A study in rabbits that did not undergo rapid rewarming found benefit from intra-arterial administration of prostaglandin E1.[76] Buflomedil is an alpha-adrenolytic agent that is used widely in Europe with preliminary and anecdotal evidence of good

results[53, 82]; however, animal models have not replicated these findings.[81] This medication is not approved by the US Food and Drug Administration. Intra-arterial reserpine studied in a controlled trial was found not to be effective.[79]

Pentoxifylline, a methylxanthine-derived phosphodiesterase inhibitor, has been widely used for treatment of peripheral vascular disease and yielded promising results in animal[78, 83, 84] and human frostbite.[77] Hayes et al[77] recommend pentoxifylline in the controlled-release form of one 400 mg tablet 3 times a day with meals, continued for 2 to 6 wk. Controlled studies of pentoxifylline in management of frostbite have not been performed.

Certain vasodilators have the potential to improve outcomes and can be used with minimal risk. However, as discussed earlier, data demonstrating benefit are limited. Iloprost is the only vasodilator with reasonable scientific evidence supporting its use.

For a summary of the suggested approach to hospital or advanced field clinic treatment of frostbite, see Table 2.

Table 2. Summary of initial hospital management of frostbite

1. Treat hypothermia or serious trauma.
2. Rapidly rewarm in water heated and maintained between 37 and 39°C (98.6 and 102.2°F) until area becomes soft and pliable to the touch (approximately 30 min).
3. Ibuprofen (12 mg·kg^{-1} per day divided twice daily).
4. Pain medication (eg, opiate) as needed.
5. Tetanus prophylaxis.
6. Air dry (ie, do not rub at any point).
7. Debridement: selectively drain (eg, by needle aspiration) clear blisters and leave hemorrhagic blisters intact.
8. Topical aloe vera every 6 h with dressing changes.
9. Dry, bulky dressings.
10. Elevate the affected body part if possible.
11. Systemic hydration.
12. Thrombolytic therapy: consider for deep frostbite at the distal interphalangeal joint or proximal if less than 24 h after thawing; use angiography for prethrombolytic intervention and monitoring of progress. Consider intravenous thrombolysis if angiography is not available.
13. Iloprost therapy: consider for deep frostbite to or proximal to the proximal interphalangeal joint, within 48 h after injury, especially if angiography is not available or with contraindications to thrombolysis.
14. Clinical examination (plus angiography or technetium-99 bone scan if necessary) to assist determination of surgical margins. Evaluation by an experienced surgeon for possible intervention.

Other post-thaw medical therapy

Once the patient has received initial frostbite therapy, long-term management is initiated to reduce long-term sequelae. Therapeutic options for frostbite after thawing include the following:

HYDROTHERAPY

Daily or twice-daily hydrotherapy at 37 to 39°C (98.6 to 102.2°F) has been recommended in the post-thaw period.[32,34, 35, 36,85] Hydrotherapy theoretically increases circulation, removes superficial bacteria, and debrides devitalized tissue.[38] No trials support improved outcomes, but the practice has few negative consequences and has the potential to benefit recovery. Data are insufficient to recommend specific temperature, timing, or duration of therapy. ***Recommendation Grade:*** 1C.

HYPERBARIC OXYGEN THERAPY

Many types of nonfrostbite wounds show accelerated or more complete healing as a result of increased tissue oxygenation from hyperbaric oxygen therapy (HBOT).[86] Because oxygen under pressure increases oxygen tension in the blood, HBOT is typically effective only if blood supply to distal tissues is competent and, therefore, may not be successful in frostbite. However, HBOT may have other effects such as making erythrocytes more malleable and decreasing bacterial load. Despite anecdotal success in extremely limited case series,[87, 88, 89, 90] controlled studies have not been conducted. The time, expense, and availability of HBOT also limit its use. At this time, data are insufficient to recommend HBOT for frostbite treatment. ***Recommendation Grade:*** Not recommended owing to insufficient data.

SYMPATHECTOMY

Because blood flow is partly determined by sympathetic tone, chemical or surgical sympathectomy has been proposed in the immediate postexposure phase to reduce tissue loss. In a rat lower limb model, early surgical denervation (within 24 h of exposure) reduced tissue loss but had no effect if performed after 24 h.[91] In a rabbit ear model, procaine-induced sympathectomy had no demonstrable beneficial effect.[92] Frostbite patients often experience long-term delayed symptoms, such as pain, paresthesia, and numbness. Chemical or surgical sympathectomy to treat these symptoms has been performed with variable results. In some studies, surgical sympathectomy has been found to reduce duration of pain and expedite demarcation of tissue necrosis. However, it has not been found to reduce the ultimate extent of tissue loss.[41, 93] Acute treatment success with IV guanethidine has been reported[94] but was not beneficial in another case report.[95] Sympathectomy may have a role in preventing certain long-term sequelae of frostbite such as pain (putatively caused by vasospasm), paresthesia, and hyperhidrosis.[96, 97] Despite many years of study, the data on

surgical sympathectomy are limited and conflicting; therefore, a recommendation for their use cannot be made. ***Recommendation Grade:*** Not recommended owing to insufficient data.

HOSPITALIZATION

Hospital admission and discharge are determined on an individual basis. Factors should include severity of the injury, coexisting injuries, comorbidities, and need for hospital-based interventions (tPA, vasodilators, surgery) or supportive therapy, as well as ease of access to appropriate community medical and nursing support. Significant swelling should prompt evaluation for compartment syndrome and admission for observation. Patients with superficial frostbite can usually be managed as outpatients or with brief inpatient stays followed by wound care instructions. Initially, deep frostbite should be managed in an inpatient setting. ***Recommendation Grade:*** 1C.

FASCIOTOMY

Thawing results in reperfusion of ischemic tissue and, in turn, sometimes results in elevated pressures within closed soft-tissue compartments. Compartment syndrome clinically manifests as tense, painful distention with reduced movement and sensation. Urgent attention is necessary to evaluate compartment pressures. If elevated compartment pressures are present, prompt surgical decompression is indicated for limb salvage.[20] ***Recommendation Grade:*** 1C.

SURGICAL TREATMENT OR AMPUTATION

After frostbite occurs, complete demarcation of tissue necrosis may take 1 to 3 mo. Angiography, technetium-99 bone scan, or magnetic resonance imaging may be used to assist determination of surgical margins[42, 64, 98] in conjunction with clinical findings. If the patient exhibits signs and symptoms of sepsis attributed to infected frostbitten tissue, amputation should be performed expeditiously.[85] Otherwise, amputation should be delayed until definitive demarcation occurs. The affected limb is often insensate. Therefore, an approach that addresses footwear and orthotics is essential to provide optimal function. Our experience has found that early involvement of a multidisciplinary rehabilitation team produces better long-term functional results. Telemedicine or electronic consultation with a surgical frostbite expert to guide local surgeons should be considered when no local expert is available. Because significant morbidity may result from unnecessary or premature surgical intervention, a surgeon with experience evaluating and treating frostbite should assess the need for and the timing of any amputation. ***Recommendation Grade:*** 1C.

Conclusions

This summary provides evidence-based guidelines for prevention and treatment of frostbite. Many important questions remain and should serve as a focus for future research. This includes elucidation of pathophysiology, medications to

assist in the prevention of frostbite, perithawing procedures to reduce injury and decrease morbidity, and post-thaw therapies that might improve long-term outcomes.

Author Contributions

Drafting of the manuscript (SEM, LF, CKG, PSA, GWR, AC, GGG, MM, CHI, ELJ, PP, JD, PHH); critical revision of the manuscript (SEM, LF, CKG, PSA, GWR, AC, GGG, MM, CHI, ELJ, PP, JD, PHH); and approval of final manuscript (SEM, LF, CKG, PSA, GWR, AC, GGG, MM, CHI, ELJ, PP, JD, PHH).

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Appendix. Supplementary materials

Download : Download Word document (13KB)

Supplementary Table 1.

References

1

G. Guyatt, D. Gutterman, M.H. Baumann, D. Addrizzo-Harris, E.M. Hylek, B. Phillips, *et al.***Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians Task Force**
Chest, 129 (1) (2006), pp. 174-181
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

2

S.E. McIntosh, M. Opacic, L. Freer, C.K. Grissom, P.S. Auerbach, G.W. Rodway, *et al.***Wilderness Medical Society practice guidelines for the prevention and treatment of frostbite: 2014 update**
Wilderness Environ Med, 25 (4 suppl) (2014), pp. S43-S54
ArticleDownload PDFView Record in ScopusGoogle Scholar

3

P. Mazur**Causes of injury in frozen and thawed cells**
Fed Proc, 24 (1965), pp. S175-S182
View Record in ScopusGoogle Scholar

4

K. Lange, L.J. Boyd, L. Loewe**The functional pathology of frostbite and the prevention of gangrene in experimental animals and humans**
Science, 102 (2641) (1945), pp. 151-152
View Record in ScopusGoogle Scholar

5

H.T. Meryman**Tissue freezing and local cold injury**
Physiol Rev, 37 (2) (1957), pp. 233-251
CrossRefView Record in ScopusGoogle Scholar

6

R. Quintanilla, F.H. Krusen, H.E. Essex**Studies on frost-bite with special reference to treatment and the effect on minute blood vessels**
Am J Physiol, 149 (1) (1947), pp. 149-161
CrossRefView Record in ScopusGoogle Scholar

7

M.C. Robson, J.P. Heggers**Evaluation of hand frostbite blister fluid as a clue to pathogenesis**
J Hand Surg Am, 6 (1) (1981), pp. 43-47
ArticleDownload PDFView Record in ScopusGoogle Scholar

8

S. Zacarian**Cryogenics: The cryolesion and the pathogenesis of cryonecrosis**
S. Zacarian (Ed.), Cryosurgery for Skin Cancer and Cutaneous Disorders, Mosby, St. Louis (1985)
Google Scholar

9

J.P. Kulka**Microcirculatory impairment as a factor in inflammatory tissue damage**
Ann N Y Acad Sci, 116 (1964), pp. 1018-1044
View Record in ScopusGoogle Scholar

10

P.S. Daum, W.D. Bowers Jr., J. Tejada, M.P. Hamlet**Vascular casts demonstrate microcirculatory insufficiency in acute frostbite**
Cryobiology, 24 (1) (1987), pp. 65-73
ArticleDownload PDFView Record in ScopusGoogle Scholar

11

A. Bhatnagar, B.B. Sarker, K. Sawroop, M.K. Chopra, N. Sinha, R. Kashyap**Diagnosis, characterisation and evaluation of treatment response of frostbite using pertechnetate scintigraphy: a prospective study**
Eur J Nucl Med Mol Imaging, 29 (2) (2002), pp. 170-175
View Record in ScopusGoogle Scholar

12

P. Petrone, E.J. Kuncir, J.A. Asensio**Surgical management and strategies in the treatment of hypothermia and cold injury**
Emerg Med Clin North Am, 21 (4) (2003), pp. 1165-1178
ArticleDownload PDFView Record in ScopusGoogle Scholar

13

E. Cauchy, E. Chetaille, V. Marchand, B. Marsigny**Retrospective study of 70 cases of severe frostbite lesions: a proposed new classification scheme**
Wilderness Environ Med, 12 (4) (2001), pp. 248-255
ArticleDownload PDFView Record in ScopusGoogle Scholar

14

R.M. Nygaard, A.B. Whitley, R.M. Fey, A.L. Wagner**The Hennepin score: quantification of frostbite management efficacy**
J Burn Care Res, 37 (4) (2016), pp. e317-e322
CrossRefView Record in ScopusGoogle Scholar

15

U. Dobnikar, S.N. Kounalakis, I.B. Mekjavic**The effect of exercise-induced elevation in core temperature on cold-induced vasodilatation response in toes**
Eur J Appl Physiol, 106 (3) (2009), pp. 457-464
CrossRefView Record in ScopusGoogle Scholar

16

C.L. Geurts, G.G. Sleivert, S.S. Cheung**Local cold acclimation during exercise and its effect on neuromuscular function of the hand**
Appl Physiol Nutr Metab, 31 (6) (2006), pp. 717-725
CrossRefGoogle Scholar

17

U. Danielsson**Windchill and the risk of tissue freezing**
J Appl Physiol, 81 (6) (1996), pp. 2666-2673
CrossRefView Record in ScopusGoogle Scholar

18

J.D. Urschel, J.W. Urschel, W.C. Mackenzie**The role of alcohol in frostbite injury**
Scand J Soc Med, 18 (4) (1990), p. 273
CrossRefView Record in ScopusGoogle Scholar

19

E. Lehmuskallio**Emollients in the prevention of frostbite**
Int J Circumpolar Health, 59 (2) (2000), pp. 122-130
View Record in ScopusGoogle Scholar

20

W.J. Mills Jr.**Frostbite. A discussion of the problem and a review of the Alaskan experience. 1973**
Alaska Med, 35 (1) (1993), pp. 29-40
View Record in ScopusGoogle Scholar

21

W.J. Mills Jr.**Summary of treatment of the cold injured patient: frostbite. 1983**
Alaska Med, 35 (1) (1993), pp. 61-66
View Record in ScopusGoogle Scholar

22
W.J. Mills Jr.**Summary of treatment of the cold injured patient. 1980**
Alaska Med, 35 (1) (1993), pp. 50-53
View Record in ScopusGoogle Scholar

23
K. McLeron**State of Alaska Cold Injury Guidelines**
(7th ed.), Department of Health and Social Services Division of Public Health Section of Community Health and EMS, Juneau, AK (2003)
Google Scholar

24
G. Martinez Villen, G. Garcia Bescos, V. Rodriguez Sosa, J.R. Morandeira Garcia**Effects of haemodilution and rewarming with regard to digital amputation in frostbite injury: an experimental study in the rabbit**
J Hand Surg Br, 27 (3) (2002), pp. 224-228
View Record in ScopusGoogle Scholar

25
J.R. Talwar, S.M. Gulati, B.M. Kapur**Comparative effects of rapid thawing, low molecular dextran and sympathectomy in cold injury in the monkeys**
Indian J Med Res, 59 (2) (1971), pp. 242-250
View Record in ScopusGoogle Scholar

26
R.C. Weatherley-White, B. Sjostrom, B.C. Paton**Experimental studies in cold injury. II. The pathogenesis of frostbite**
J Surg Res, 4 (1964), pp. 17-22
ArticleDownload PDFView Record in ScopusGoogle Scholar

27
D.R. Webster, G. Bonn**Low-molecular-weight dextran in the treatment of experimental frostbite**
Can J Surg, 8 (4) (1965), pp. 423-427
View Record in ScopusGoogle Scholar

28
B.M. Kapur, S.M. Gulati, J.R. Talwar**Low molecular dextran in the management of frostbite in monkeys**
Indian J Med Res, 56 (11) (1968), pp. 1675-1681
View Record in ScopusGoogle Scholar

29
K.D. Rainsford**Ibuprofen: pharmacology, efficacy and safety**
Inflammopharmacology, 17 (6) (2009), pp. 275-342
CrossRefView Record in ScopusGoogle Scholar

30

J.P. Heggers, M.C. Robson, K. Manavalen, M.D. Weingarten, J.M. Carethers, J.A. Boertman, *et al.***Experimental and clinical observations on frostbite**
Ann Emerg Med, 16 (9) (1987), pp. 1056-1062
ArticleDownload PDFView Record in ScopusGoogle Scholar

31

M.C. Robson, E.J. DelBeccaro, J.P. Heggers, G.L. Loy**Increasing dermal perfusion after burning by decreasing thromboxane production**
J Trauma, 20 (9) (1980), pp. 722-725
CrossRefView Record in ScopusGoogle Scholar

32

W.J. Mills Jr.**Frostbite. A method of management including rapid thawing**
Northwest Med, 65 (2) (1966), pp. 119-125
View Record in ScopusGoogle Scholar

33

M.S. Malhotra, L. Mathew**Effect of rewarming at various water bath temperatures in experimental frostbite**
Aviat Space Environ Med, 49 (7) (1978), pp. 874-876
View Record in ScopusGoogle Scholar

34

C. Imray, A. Grieve, S. Dhillon**Cold damage to the extremities: frostbite and non-freezing cold injuries**
Postgrad Med J, 85 (1007) (2009), pp. 481-488
CrossRefView Record in ScopusGoogle Scholar

35

W.J. Mills, R. Whaley**Frostbite: experience with rapid rewarming and ultrasonic therapy. 1960-1**
Wilderness Environ Med, 9 (4) (1998), pp. 226-247
ArticleDownload PDFView Record in ScopusGoogle Scholar

36

R.L. McCauley, D.N. Hing, M.C. Robson, J.P. Heggers**Frostbite injuries: a rational approach based on the pathophysiology**
J Trauma, 23 (2) (1983), pp. 143-147
CrossRefView Record in ScopusGoogle Scholar

37

M. Dole, F.W. Endorf, J. Gayken, R. Fey, R.M. Nygaard**Early mobilization in lower extremity frostbite injury: preliminary experience at a single burn center**
J Burn Care Res, 39 (3) (2018), pp. 339-344
View Record in ScopusGoogle Scholar

38

B.V. Reamy**Frostbite: review and current concepts**
J Am Board Fam Pract, 11 (1) (1998), pp. 34-40
CrossRefView Record in ScopusGoogle Scholar

39

D. Jones, S.F. Covins, G.E. Miller, K.I. Morrison, A.G. Clark, S.D. Calcott, *et al.***Infrared thermographic analysis of surface temperature of the hands during exposure to normobaric hypoxia**
High Alt Med Biol, 19 (4) (2018), pp. 388-393
CrossRefView Record in ScopusGoogle Scholar

40
A. Rousseau, I. Steinwall, R.D. Woodson, F. Sjoberg**Hyperoxia decreases cutaneous blood flow in high-perfusion areas**
Microvasc Res, 74 (1) (2007), pp. 15-22
ArticleDownload PDFView Record in ScopusGoogle Scholar

41
A. Golant, R.M. Nord, N. Paksima, M.A. Posner**Cold exposure injuries to the extremities**
J Am Acad Orthop Surg, 16 (12) (2008), pp. 704-715
CrossRefView Record in ScopusGoogle Scholar

42
J.V. Murphy, P.E. Banwell, A.H. Roberts, D.A. McGrouther**Frostbite: pathogenesis and treatment**
J Trauma, 48 (1) (2000), pp. 171-178
View Record in ScopusGoogle Scholar

43
J. Biem, N. Koehncke, D. Classen, J. Dosman**Out of the cold: management of hypothermia and frostbite**
CMAJ, 168 (3) (2003), pp. 305-311
View Record in ScopusGoogle Scholar

44
K.J. Bruen, J.R. Ballard, S.E. Morris, A. Cochran, L.S. Edelman, J.R. Saffle**Reduction of the incidence of amputation in frostbite injury with thrombolytic therapy**
Arch Surg, 142 (6) (2007), pp. 546-551
discussion 551–3
CrossRefView Record in ScopusGoogle Scholar

45
K. Jenabzadeh, W. Mohr, D. Ahrenholz**Frostbite: a single institution's twenty year experience with intraarterial thrombolytic therapy**
J Burn Care Research, 30 (2) (2006), p. S103
View Record in ScopusGoogle Scholar

46
R.L. Sheridan, M.A. Goldstein, F.J. Stoddard Jr, T.G. Walker**Case records of the Massachusetts General Hospital. Case 41-2009. A 16-year-old boy with hypothermia and frostbite**
N Engl J Med, 361 (27) (2009), pp. 2654-2662
View Record in ScopusGoogle Scholar

47

J.A. Twomey, G.L. Peltier, R.T. Zera**An open-label study to evaluate the safety and efficacy of tissue plasminogen activator in treatment of severe frostbite**
J Trauma, 59 (6) (2005), pp. 1350-1354
discussion 1354–5
CrossRefView Record in ScopusGoogle Scholar

48

T.A. Zdeblick, G.A. Field, J.W. Shaffer**Treatment of experimental frostbite with urokinase**
J Hand Surg Am, 13 (6) (1988), pp. 948-953
ArticleDownload PDFView Record in ScopusGoogle Scholar

49

R.M. Nygaard, A.M. Lacey, A. Lemere, M. Dole, J.R. Gayken, A.L. Lambert Wagner, *et al.***Time matters in severe frostbite: assessment of limb/digit salvage on the individual patient level**
J Burn Care Res, 38 (1) (2017), pp. 53-59
CrossRefView Record in ScopusGoogle Scholar

50

E. Cauchy, C.B. Davis, M. Pasquier, E.F. Meyer, P.H. Hackett**A new proposal for management of severe frostbite in the austere environment**
Wilderness Environ Med, 27 (1) (2016), pp. 92-99
ArticleDownload PDFView Record in ScopusGoogle Scholar

51

C. Handford, P. Buxton, K. Russell, C.E. Imray, S.E. McIntosh, L. Freer, *et al.***Frostbite: a practical approach to hospital management**
Extrem Physiol Med, 3 (2014), p. 7
CrossRefGoogle Scholar

52

R.L. Hutchison, H.M. Miller, S.K. Michalke**The use of tPA in the treatment of frostbite: a systematic review**
Hand (NY), 14 (1) (2019), pp. 13-18
CrossRefView Record in ScopusGoogle Scholar

53

E. Cauchy, B. Cheguillaume, E. Chetaille**A controlled trial of a prostacyclin and rt-PA in the treatment of severe frostbite**
N Engl J Med, 364 (2) (2011), pp. 189-190
CrossRefView Record in ScopusGoogle Scholar

54

N. Patel, D.R. Srinivasa, R.N. Srinivasa, J.J. Gemmete, V. Krishnamurthy, N. Dasika, *et al.***Intra-arterial thrombolysis for extremity frostbite decreases digital amputation rates and hospital length of stay**
Cardiovasc Intervent Radiol, 40 (12) (2017), pp. 1824-1831
CrossRefView Record in ScopusGoogle Scholar

55

A. Lindford, J. Valtonen, M. Hult, H. Kavola, K. Lappalainen, R. Lassila, *et al.***The evolution of the Helsinki frostbite management protocol**
Burns, 43 (7) (2017), pp. 1455-1463
ArticleDownload PDFView Record in ScopusGoogle Scholar

56

A. Wexler, S. Zavala**The use of thrombolytic therapy in the treatment of frostbite injury**
J Burn Care Res, 38 (5) (2017), pp. e877-e881
CrossRefView Record in ScopusGoogle Scholar

57

A.R. Johnson, H.L. Jensen, G. Peltier, E. DelaCruz**Efficacy of intravenous tissue plasminogen activator in frostbite patients and presentation of a treatment protocol for frostbite patients**
Foot Ankle Spec, 4 (6) (2011), pp. 344-348
CrossRefView Record in ScopusGoogle Scholar

58

L.M. Jones, R.A. Coffey, M.P. Natwa, J.K. Bailey**The use of intravenous tPA for the treatment of severe frostbite**
Burns, 43 (5) (2017), pp. 1088-1096
ArticleDownload PDFView Record in ScopusGoogle Scholar

59

A.E. Ibrahim, J. Goverman, K.A. Sarhane, J. Donofrio, T.G. Walker, S.P. Fagan**The emerging role of tissue plasminogen activator in the management of severe frostbite**
J Burn Care Res, 36 (2) (2015), pp. e62-e66
CrossRefView Record in ScopusGoogle Scholar

60

S. Tavri, S. Ganguli, R.G. Bryan Jr, J. Goverman, R. Liu, Z. Irani, *et al.***Catheter-directed intraarterial thrombolysis as part of a multidisciplinary management protocol of frostbite injury**
J Vasc Interv Radiol, 27 (8) (2016), pp. 1228-1235
ArticleDownload PDFView Record in ScopusGoogle Scholar

61

T. Gonzaga, K. Jenabzadeh, C.P. Anderson, W.J. Mohr, F.W. Endorf, D.H. Ahrenholz**Use of intra-arterial thrombolytic therapy for acute treatment of frostbite in 62 patients with review of thrombolytic therapy in frostbite**
J Burn Care Res, 37 (4) (2016), pp. e323-e334
View Record in ScopusGoogle Scholar

62

A.M. Saemi, J.M. Johnson, C.S. Morris**Treatment of bilateral hand frostbite using transcatheter arterial thrombolysis after papaverine infusion**
Cardiovasc Intervent Radiol, 32 (6) (2009), pp. 1280-1283
CrossRefView Record in ScopusGoogle Scholar

63

C. Wagner, C.J. Pannucci**Thrombolytic therapy in the acute management of frostbite injuries**
Air Med J, 30 (1) (2011), pp. 39-44
ArticleDownload PDFView Record in ScopusGoogle Scholar

64
J.R. Barker, M.J. Haws, R.E. Brown, J.O. Kucan, W.D. Moore**Magnetic resonance imaging of severe frostbite injuries**
Ann Plast Surg, 38 (3) (1997), pp. 275-279
CrossRefView Record in ScopusGoogle Scholar

65
J.D. Millet, R.K. Brown, B. Levi, C.T. Kraft, J.A. Jacobson, M.D. Gross, *et al.***Frostbite: spectrum of imaging findings and guidelines for management**
Radiographics, 36 (7) (2016), pp. 2154-2169
CrossRefView Record in ScopusGoogle Scholar

66
C. Kraft, J.D. Millet, S. Agarwal, S.C. Wang, K.C. Chung, R.K. Brown, *et al.***SPECT/CT in the evaluation of frostbite**
J Burn Care Res, 38 (1) (2017), pp. e227-e234
CrossRefView Record in ScopusGoogle Scholar

67
C. Handford, O. Thomas, C.H.E. Imray**Frostbite**
Emerg Med Clin North Am, 35 (2) (2017), pp. 281-299
ArticleDownload PDFView Record in ScopusGoogle Scholar

68
T. Masters, S. Omodt, J. Gayken, C. Logue, B. Westgard, S. Hendriksen, *et al.***Microangiography to monitor treatment outcomes following severe frostbite injury to the hands**
J Burn Care Res, 39 (1) (2018), pp. 162-167
Google Scholar

69
S. Della Bella, M. Molteni, C. Mocellin, S. Fumagalli, P. Bonara, R. Scorza**Novel mode of action of iloprost: in vitro down-regulation of endothelial cell adhesion molecules**
Prostaglandins Other Lipid Mediat, 65 (2-3) (2001), pp. 73-83
ArticleDownload PDFView Record in ScopusGoogle Scholar

70
M. Candela, A. Pansoni, L. Jannino, V.G. Menditto, M. Natalini, F. Ravaglia, *et al.***Coagulative modifications in patients with systemic sclerosis treated with iloprost or nifedipine**
Ann Ital Med Int, 16 (3) (2001), pp. 170-174
View Record in ScopusGoogle Scholar

71
E. Groechenig**Treatment of frostbite with iloprost**

Lancet, 344 (8930) (1994), pp. 1152-1153
ArticleDownload PDFView Record in ScopusGoogle Scholar

72
A. Poole, J. Gauthier**Treatment of severe frostbite with iloprost in northern Canada**
CMAJ, 188 (17-18) (2016), pp. 1255-1258
CrossRefView Record in ScopusGoogle Scholar

73
M. Kaller**BET 2: treatment of frostbite with iloprost**
Emerg Med J, 34 (10) (2017), pp. 689-690
CrossRefView Record in ScopusGoogle Scholar

74
P. Pandey, R. Vadlamudi, R. Pradhan, K.R. Pandey, A. Kumar, P. Hackett**Case report: severe frostbite in extreme altitude climbers—the Kathmandu iloprost experience**
Wilderness Environ Med, 29 (3) (2018), pp. 366-374
ArticleDownload PDFView Record in ScopusGoogle Scholar

75
S. Irarrazaval, P. Besa, E. Cauchy, P. Pandey, J. Vergara**Case report of frostbite with delay in evacuation: field use of iloprost might have improved the outcome**
High Alt Med Biol, 19 (4) (2018), pp. 382-387
CrossRefView Record in ScopusGoogle Scholar

76
R.A. Yeager, T.W. Campion, J.C. Kerr, R.W. Hobson 2nd, T.G. Lynch**Treatment of frostbite with intra-arterial prostaglandin E1**
Am Surg, 49 (12) (1983), pp. 665-667
View Record in ScopusGoogle Scholar

77
D.W. Hayes Jr, V.J. Mandracchia, C. Considine, G.E. Webb**Pentoxifylline. Adjunctive therapy in the treatment of pedal frostbite**
Clin Podiatr Med Surg, 17 (4) (2000), pp. 715-722
CrossRefGoogle Scholar

78
M.B. Miller, P.J. Koltai**Treatment of experimental frostbite with pentoxifylline and aloe vera cream**
Arch Otolaryngol Head Neck Surg, 121 (6) (1995), pp. 678-680
CrossRefView Record in ScopusGoogle Scholar

79
D.L. Bouwman, S. Morrison, C.E. Lucas, A.M. Ledgerwood**Early sympathetic blockade for frostbite—is it of value?**
J Trauma, 20 (9) (1980), pp. 744-749
CrossRefView Record in ScopusGoogle Scholar

80

G.A. Espinosa**Management of frostbite injuries**
J Natl Med Assoc, 73 (12) (1981), pp. 1125-1131
View Record in ScopusGoogle Scholar

81

P.S. Daum, W.D. Bowers Jr, J. Tejada, D. Morehouse, M.P. Hamlet**An evaluation of the ability of the peripheral vasodilator buflomedil to improve vascular patency after acute frostbite**
Cryobiology, 26 (1) (1989), pp. 85-92
ArticleDownload PDFView Record in ScopusGoogle Scholar

82

J. Foray, P.E. Baisse, J.P. Mont, C. Cahen**Treatment of frostbites. Analysis of results in 20 patients with buflomedil chlorhydrate (author's transl) [in French]**
Sem Hop, 56 (9-10) (1980), pp. 490-497
View Record in ScopusGoogle Scholar

83

S.S. Purkayastha, A. Roy, S.K. Chauhan, S.S. Verma, W. Selvamurthy**Efficacy of pentoxifylline with aspirin in the treatment of frostbite in rats**
Indian J Med Res, 107 (1998), pp. 239-245
View Record in ScopusGoogle Scholar

84

S.S. Purkayastha, G. Bhaumik, S.K. Chauhan, P.K. Banerjee, W. Selvamurthy**Immediate treatment of frostbite using rapid rewarming in tea decoction followed by combined therapy of pentoxifylline, aspirin & vitamin C**
Indian J Med Res, 116 (2002), pp. 29-34
View Record in ScopusGoogle Scholar

85

G.J. Jurkovich**Environmental cold-induced injury**
Surg Clin North Am, 87 (1) (2007), pp. 247-267
viii
ArticleDownload PDFView Record in ScopusGoogle Scholar

86

S.R. Thom**Hyperbaric oxygen: its mechanisms and efficacy**
Plast Reconstr Surg, 127 (suppl 1) (2011), pp. 131S-141S
CrossRefGoogle Scholar

87

Z. Finderle, K. Cankar**Delayed treatment of frostbite injury with hyperbaric oxygen therapy: a case report**
Aviat Space Environ Med, 73 (4) (2002), pp. 392-394
View Record in ScopusGoogle Scholar

88

L.R. Folio, K. Arkin, W.P. Butler**Frostbite in a mountain climber treated with hyperbaric oxygen: case report**
Mil Med, 172 (5) (2007), pp. 560-563
CrossRefView Record in ScopusGoogle Scholar

89
D. von Heimburg, E.M. Noah, U.P. Sieckmann, N. Pallua**Hyperbaric oxygen treatment in deep frostbite of both hands in a boy**
Burns, 27 (4) (2001), pp. 404-408
ArticleDownload PDFView Record in ScopusGoogle Scholar

90
C.A. Lansdorp, G.R. Roukema, O. Boonstra, J. Dokter**Delayed treatment of frostbite with hyperbaric oxygen: a report of two cases**
Undersea Hyperb Med, 44 (4) (2017), pp. 365-369
CrossRefView Record in ScopusGoogle Scholar

91
P.L. Gildenberg, E. Hardenbergh**The effect of immediate sympathectomy on tissue survival following experimental frostbite**
Ann Surg, 160 (1964), pp. 160-168
CrossRefView Record in ScopusGoogle Scholar

92
F.A. Fuhrman, J.M. Crismon**Studies on gangrene following cold injury; the effect of rutin and other chemical agents on the course of experimental frostbite in rabbits**
J Clin Invest, 27 (3 Pt 1) (1948), pp. 364-371
CrossRefView Record in ScopusGoogle Scholar

93
C. Christenson, C. Stewart**Frostbite**
Am Fam Physician, 30 (6) (1984), pp. 111-122
View Record in ScopusGoogle Scholar

94
R. Kaplan, P. Thomas, H. Tepper, B. Strauch**Treatment of frostbite with guanethidine**
Lancet, 2 (8252) (1981), pp. 940-941
View Record in ScopusGoogle Scholar

95
O. Engkvist**The effect of regional intravenous guanethidine block in acute frostbite. Case report**
Scand J Plast Reconstr Surg, 20 (2) (1986), pp. 243-245
CrossRefView Record in ScopusGoogle Scholar

96
M.I. Khan, M. Tariq, A. Rehman, A. Zafar, S.N. Sheen**Efficacy of cervicothoracic sympathectomy versus conservative management in patients suffering from incapacitating Raynaud's syndrome after frostbite**

J Ayub Med Coll Abbottabad, 20 (2) (2008), pp. 21-24
View Record in ScopusGoogle Scholar

97

M.S. Taylor**Lumbar epidural sympathectomy for frostbite injuries of the feet**
Mil Med, 164 (8) (1999), pp. 566-567
View Record in ScopusGoogle Scholar

98

E. Cauchy, B. Marsigny, G. Allamel, R. Verhellen, E. Chetaille**The value of technetium 99 scintigraphy in the prognosis of amputation in severe frostbite injuries of the extremities: a retrospective study of 92 severe frostbite injuries**
J Hand Surg Am, 25 (5) (2000), pp. 969-978
ArticleDownload PDFView Record in ScopusGoogle Scholar

Wilderness Medical Society Practice Guidelines for Treatment of Exercise-Associated Hyponatremia: 2014 Update

Brad L. Bennett, PhD; Tamara Hew-Butler, DPM, PhD; Martin D. Hoffman, MD; Ian R. Rogers, MD; Mitchell H. Rosner, MD

From the Military & Emergency Medicine Department, F. Edward Hebert School of Medicine, Uniformed Services University of the Health Sciences, Bethesda, MD (Dr Bennett); Oakland University, Rochester, MI (Dr Hew-Butler); the Department of Physical Medicine & Rehabilitation, Department of Veterans Affairs, Northern California Health Care System, and University of California Davis Medical Center, Sacramento, CA (Dr Hoffman); St. John of God Murdoch Hospital & University of Notre Dame, Murdoch, Western Australia (Dr Rogers); and the Division of Nephrology, University of Virginia, Charlottesville, VA (Dr Rosner).

Exercise-associated hyponatremia (EAH) is defined by a serum or plasma sodium concentration below the normal reference range of 135 mmol/L that occurs during or up to 24 hours after prolonged physical activity. It is reported to occur in individual physical activities or during organized endurance events conducted in austere environments in which medical care is limited and often not available, and patient evacuation to definitive care is often greatly delayed. Rapid recognition and appropriate treatment are essential in the severe form to ensure a positive outcome. Failure in this regard is a recognized cause of event-related fatality. In an effort to produce best practice guidelines for EAH in the austere environment, the Wilderness Medical Society convened an expert panel. The panel was charged with the development of evidence-based guidelines for management of EAH. Recommendations are made regarding the situations when sodium concentration can be assessed in the field and when these values are not known. These recommendations are graded on the basis of the quality of supporting evidence and balance between the benefits and risks/burdens for each parameter according to the methodology stipulated by the American College of Chest Physicians. This is an updated version of the original WMS Practice Guidelines for Treatment of Exercise-Associated Hyponatremia published in Wilderness & Environmental Medicine 2013;24(3):228–240.

Key words:hyponatremia, exercise-associated hyponatremia, arginine vasopressin, SIADH, exercise

Introduction

Nearly 3 decades after the first report of exercise-associated hyponatremia (EAH),[1] great strides are taking place in an effort to prevent what is now recognized as a leading cause of preventable morbidity and mortality in endurance activities throughout the world. To date, review articles and international consensus statements have documented risk factors, pathophysiology, signs and symptoms, prevention, and patient management strategies.[2–8] These reports have primarily focused on incidences of EAH in organized endurance events that are conducted in the front country where medical tents and local emergency medical services are available on site to assist these participants and to transport as needed to a local hospital for appropriate management. Beyond front country triathlons and marathons, many prolonged individual activities, ultramarathons, and multiple-day endurance events take place in the back-country. EAH has been documented in hikers, trekkers, climbers, and cold climate endurance athletes.[9–14] Furthermore, it is likely that many individuals with symptomatic or asymptomatic EAH go under reported in the literature.[15] The lessons learned from current evidence- based EAH guidelines can be extended to those providing care in the backcountry in a limited-resource environment. It was the intent of this panel to develop evidence-based practice guidelines for EAH for use in austere environments, during transport by emergency medical services, and for immediate care by the receiving hospital.

This set of guidelines is an updated version of the original Wilderness Medical Society Practice Guidelines for Treatment of Exercise-Associated Hyponatremia published in Wilderness & Environmental Medicine 2013;24(3):228–240.

Methods

The expert panel was convened at the Wilderness Medical Society annual meeting in Whistler, British Columbia, Canada, July 2012. Members were selected on the basis of clinical interest or research experience. Relevant articles were identified by a search of MED- LINE as the primary database, US National Library of Medicine, National Institutes of Health (http://www.ncbi.nlm.nih.gov/pubmed/). Key search terms used were hyponatremia, exercise-associated hyponatremia, arginine vasopressin, syndrome of inappropriate antidiuretic hormone (SIADH), hyponatremic encephalopathy, and 3% hypertonic saline. Peer-reviewed studies related to EAH, including randomized controlled trials, observational studies, and case series, were reviewed, and the level of evidence supporting the conclusions was assessed. Abstract-only studies were not included. Conclusions from review articles were not considered in the formulation of recommendations but are cited below in an effort to provide context. When no relevant studies were identified, the panel recommendation was based on risk vs benefit perceptions derived from patient-care experience. The panel used a consensus approach to develop recommendations regarding management of EAH in the wilderness. These recommendations have been graded on the basis of clinical strength as outlined by the American College of Chest Physicians (ACCP; see the onlineSupplementary ACCP Table 1).[16]

Scope of the Problem

EAH is defined by a serum or plasma sodium concentration below the normal reference range of 135 mmol/L that occurs during or up to 24 hours after prolonged physical activity.[7] The reported incidence of EAH varies widely, in part because the diagnosis is based solely on an abnormal biochemical result in an appropriate clinical setting. Many cases of EAH may be asymptomatic and are largely detected from blood samples taken from consenting athletes participating in research screening protocols, with reported incidence ranging from 0% to 51%. The highest reported incidence of"asymptomatic" hyponatremia has been noted in ultramarathon races covering 161 km (100 miles) in North America, in which the incidence of EAH has ranged between 30% and 51%.[13,17–19]

The incidence of asymptomatic EAH is greater than the incidence of "symptomatic" EAH, which refers to a biochemical diagnosis of EAH combined with clinical symptoms and signs. Severe EAH manifests as significant mental status changes resulting from cerebral edema (termed exercise-associated hyponatremic encephalopathy [EAHE]), at times associated with non-cardiogenic pulmonary edema.[5,6]Twelve confirmed deaths of public record have been directly attributed to complications associated with EAHE.[20–24] The overall incidence of symptomatic EAH in all marathon participants is typically less than 1%,[24,25] but the percentage of EAH seen in all symptomatic athletes seeking medical care has been reported to be as high as 23% in an Ironman Triathlon[26] and 38% in runners participating in a marathon and ultramarathon in Asia.[27] An increasing trend is that symptomatic EAH is now being reported in much shorter distance events, such as a half marathon[28] and sprint triathlon taking approximately 90 minutes to complete.[29]

Symptomatic cases of EAH have been reported with increased frequency in both hikers and military infantry personnel. The reported incidence of hyponatremia in Grand Canyon hikers seeking medical care from exercise-associated collapse or exhaustion from May 31, 1993, through September 31, 1993, was 16% with an estimated incidence rate between 2.0 and 4.0 per 100,000 persons.[9,30] Furthermore, suspected hyponatremia was found to account for 19% of nonfatal heat related incidents in Grand Canyon National Park from April through September during 2004 through 2009.[31] US military services have reported an increased trend of EAH cases primarily in Marine Corps and Army infantry personnel of the past decade.[32,33] However, new data (1999 to 2012) released in March 2013 shows an EAH incidence rate of 6.7 cases/100,000 person-years in US military services. These new data suggest that the annual incidence of EAH may have decreased by almost 50% from 2010 to 2012.[34] It is important to note that one previously published paper entitled "Death by Water Intoxication" details 4 fatal cases of dilutional hyponatremia in military personnel.[32] Whether or not all of these fatalities were primarily associated with EAH has been called into question. Thus, although it has been widely propagated that there have been 4 military deaths associated with EAH as the primary pathogenic mechanism, the number of EAH military deaths may be as low as 1 or 2. Nevertheless, modest hiking and marching activities in young and healthy individuals have led to documented EAHE morbidity and mortality.[32,35]

Pathogenesis of EAH

Two major pathologic mechanisms largely account for the development of EAH:

1. excessive fluid intake, and
2. impaired urinary water excretion, largely as a result of persistent secretion of arginine vasopressin (AVP), also referred to as antidiuretic hormone or ADH.[4,5]

EXCESSIVE FLUID INTAKE

Overhydration appears to be the primary risk factor for the development of EAH. This is reflected in the weight gains seen in the majority of, but not all, athletes who become symptomatic with EAH. Individuals with normal renal function, ingesting a regular diet, can excrete between 500 and 1000 mL/h of water.[36] With the additional nonrenal losses of water as a result of sweat and insensible fluid losses, athletes should be able to consume as much as 1000 to 1500 mL/h before developing water retention and dilutional hyponatremia. Thus, although fluid ingestion is necessary to develop EAH, it is likely not sufficient except in those circumstances in which water intake is very excessive (> 1500 mL/h).

INAPPROPRIATE AVP SECRETION

Failure to suppress AVP can markedly reduce the ability of the kidneys to excrete a water load. Under normal circumstances, ingestion of excessive water should suppress AVP, leading to production of dilute, high-volume urine (urine osmolality as low as 50 mOsm/kg and a volume of 500 to 1000 mL/h). If AVP is not suppressed appropriately with water loading, then the ability to produce dilute urine is markedly impaired (for instance, a low-level persistence of AVP can result in a fixed urine osmolality of 150 mOsm/kg and a decrease in the rate of water excretion by two-thirds as compared with a urine osmolality of 50 mOsm/kg). In fact, the available data support the concept that many athletes who experience EAH have submaximal suppression of AVP and an inappropriately high urine sodium and osmolality.[23,35,37] This is similar to SIADH. There are a number of nonosmotic stimuli that lead to secretion of AVP that may be operable in endurance athletes: intense exercise itself, nausea or vomiting, hypoglycemia, and nonspecific stresses such as pain and emotion.[38-40] Not all AVP release in athletes may be inappropriate, as excessive sweat losses may induce volume depletion and appropriate secretion of AVP. This appropriate AVP secretion may be important in those athletes who experience EAH along with net weight loss (Figure 1).

OTHER FACTORS

Although the combination of excessive water intake and inappropriate AVP secretion will clearly lead to hyponatremia, other factors may be operable in endurance athletes. In a study of endurance athletes running for a mean of 6 hours with ad libitum fluid intake, it was noted that even with a mean 3.8-kg mass loss, serum sodium was maintained at normal levels. Despite the loss in plasma volume in these subjects, there were elevations in the levels of brain natriuretic peptide (NT-BNP).[35,40] The elevation in NT-BNP may lead to excessive losses of

urine sodium and raise the risk of hyponatremia. Indeed, elevated NT-BNP concentrations and increased urinary sodium concentrations have been observed in EAH.[41] A possible mechanism for maintenance of a normal serum sodium level despite weight gain is the release of sodium from internal stores.[42] Up to 25% of body sodium is bound in bone (to negatively charged proteoglycan matrix) and, although not osmotically active, ispotentially recruitable into an osmotically active form.[43,44] Thus, this pool could minimize the fall in serum sodium induced by overhydration or exacerbate hyponatremia if not mobilized. Whether or not impairment of this system might play a role in the development of EAH in some individuals is not clear.[19] The absorption of water retained in the gastrointestinal tract at the end of a race has been suggested as a cause for an acute drop in serum sodium concentration.[22,25] This may account for a transient lucid period after finishing a race followed by the acute development of clinical signs of EAHE within about 30 minutes after a competition. The breakdown of glycogen into smaller, more osmotically active molecules, such as lactate, during exercise initially increases cellular osmolality and shifts water into cells, leading to a rise in serum sodium. This may then reverse within 5 minutes after the cessation of exercise and transiently lower the serum sodium.[45,46] Changes in potassium balances that serve as effective osmoles may also affect the serum sodium such that hypokalemia will lead to or exacerbate hyponatremia.

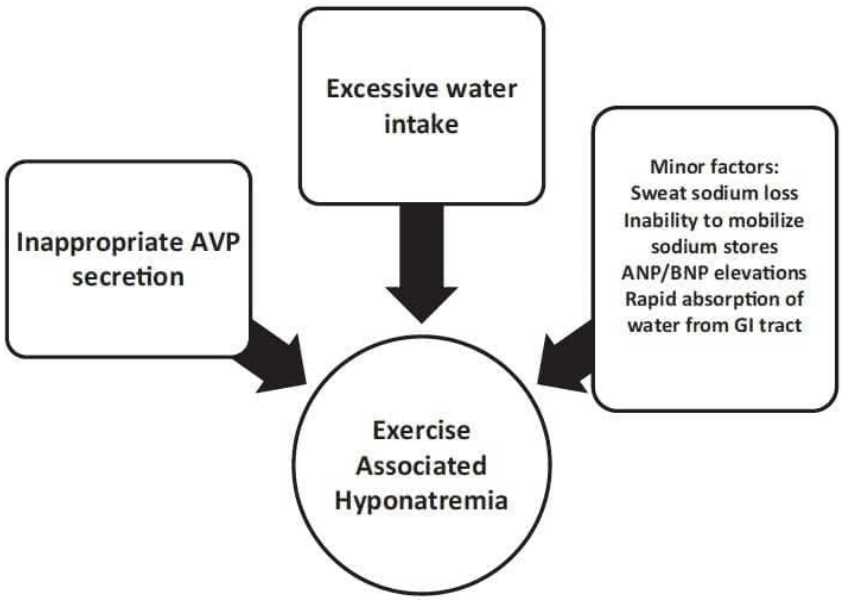


Figure 1.Exercise-associated hyponatremia pathogenesis.

ANP, atrial natriuretic peptide; AVP, arginine vasopressin; BNP, brain natriuretic peptide; GI, gastrointestinal.

The issue of whether sweat sodium loss contributes to the development of EAH remains controversial. There is a highly variable degree of sodium loss from sweat (ranging from15to65mmol/L),and compared with the general population, endurance athletes generally have lower sweat sodium levels.[47,48] The direct effect of losing hypotonic sweat would be to raise the serum sodium. However, sweat loss could contribute to the development of hyponatremia if the degree of fluid loss were sufficient to produce significant volume depletion and provide a

stimulus to AVP release, thereby impairing excretion of water. In this case, there would also have to be ingestion of hypotonic fluids. This scenario may explain the finding of EAH developing in some athletes with net weight loss.[17-19]

Risk Factors

As stated above, the major risk factor for developing EAH is excessive water intake beyond the capacity for renal water excretion.[49,50] Other independent risk factors include longer race times (continuous endurance exercise lasting 4 4 hours)[7,51] and a low[23,25,51] or high[49] body mass index (BMI). Practically speaking, smaller athletes (low BMI) who are following fluid intake guidelines designed for larger individuals and slower, unfit athletes (high BMI) drinking generous amounts of fluids while exercising at a lower intensity are at increased risk for experiencing hyponatremia during exercise. Although the incidence of women experiencing symptomatic hyponatremia appears to be greater than that of men in some environments,[22,24,26,51] when adjusted for BMI and racing time, the apparent sex difference has not been shown to be statistically significant.[51] Along with other nonosmotic stimuli to AVP secretion,[40,52-57] nonsteroidal anti-inflammatory drugs (NSAIDs) have been implicated as a risk factor in the development of EAH[22,25,58] by potentiating the water retention effects of AVP at the kidney.[59,60] However, data are still conflicting,[24,49] and further investigation is necessary to determine whether NSAID usage — with respect to both classification and dosages — is a clear risk factor for the development of EAH. Other medications associated with SIADH, such as selective serotonin reuptake inhibitors, may also increase the risk for EAH, but data are not conclusive.

Prevention

AVOID OVERHYDRATION

The primary strategy to prevent EAH is to avoid over- drinking during exercise. Because fluid losses through sweat and urine are highly dynamic and variable across individuals participating in a variety of outdoor activities, recommending fixed ranges of fluid intake are not appropriate. Using the sensation of thirst as a real-time guide to fluid ingestion during exercise appears safe and effective and eliminates both of the detrimental extremes of fluid balance (dehydration and overhydration).(7,61-64) Therefore, participant education on this approach to hydration during exercise is an important prevention strategy. Another strategy that has been shown to reduce the incidence of hyponatremia during endurance events is to reduce the availability of fluids along the routes of exercise.[65]

Recommendation: Participants should focus on avoid- ing overdrinking during exercise by drinking according to thirst, and race organizers might consider reducing the excess availability of fluids (> 3 km apart) along routes of exercise. Recommendation grade: 1B.

AVOID EXCESSIVE SODIUM SUPPLEMENTATION

Sodium supplementation during exercise has not been shown to prevent the development of hyponatremia during physical activity lasting less than 18 hours.[66-69] In athletes who drink beyond thirst or fully replace 100% of body weight losses during exercise, supplemental sodium may attenuate the decline in blood sodium concentration[69,70] but will not prevent the development of hyponatremia if overdrinking were to continue.[67] Supplemental sodium has no effect on blood sodium concentration when athletes drink according to thirst.[66-68] However, exercisers who drink insufficient amounts of fluid during exercise will often finish races with elevated blood sodium concentrations.[71-74] Collectively, these results demonstrate that it is the amount of fluid ingested rather than the amount of sodium ingested during exercise that has a more pronounced effect on blood sodium concentrations as mathematically predicted else- where.[75] Adverse effects associated with abnormal water retention from excessive sodium intake have been reported.[76,77]

Recommendation: Excessive sodium supplementation is not recommended during physical activity lasting less than 18 hours. Recommendation grade: 2B.

MONITORING BODY WEIGHT

Because over consumption of hypotonic fluids beyond the capacity to excrete any fluid excess is often key in the pathophysiology of EAH, the monitoring of body weight change is one strategy commonly used in 161-km ultramarathons to help prevent overhydration. As a result of the combination of substrate losses and the liberation of glycogen-bound water during exertion, some weight loss is appropriate during exercise. Furthermore, EAH has been reported with substantial weight loss in some environments,[17-19] so weight loss is not a reliable approach for excluding the diagnosis of EAH. On the other hand, it appears as though those with EAH who have not lost weight during exercise are the most likely to become symptomatic.[42,76] Therefore, in the presence of weight gain during exercise,fluid intake should be reduced, and if sodium supplementation has been taking place, this should also be curtailed until body weight returns to an appropriate level. If feasible, weight scales can be made available at organized athletic events for this purpose, but care should be taken to assure proper scale calibration and placement on solid level surfaces, and participants should be educated in proper use of body weight information.

Recommendation: Body weight can be monitored in organized events, and in the presence of weight gain during exercise, fluid and sodium intake should be reduced until weight returns to 2% to 4% of body weight loss from baseline level. Recommendation grade: 1B.

EDUCATE EVENT SUPPORT AND MEDICAL PERSONNEL

Event support staff should have a basic understanding of EAH to avoid the provision of improper hydration advice to participants because it has been previously shown that runners have a poor understanding of the relationship between drinking habits and hyponatremia.[78,79] On-site medical personnel should be aware of proper treatment of EAH. This should include the recognition

that hypotonic fluid replacement (intravenous or oral) should be avoided when the diagnosis of EAH is under consideration to prevent further declines in blood sodium concentration. Such education can be provided by event medical directors via prerace briefings and the use of suggested reading material or educational videotapes.

Recommendation: Event support staff should be knowledgeable so they can provide proper hydration advice, and on-site medical and emergency medical service (EMS) personnel should be educated about proper recognition and treatment of EAH. Recommendation grade: 1B.

Field Treatment

Appropriate management of EAH depends first on correctly diagnosing the condition. EAH must be routinely considered in the differential diagnosis of an individual presenting for medical attention during or shortly after exercise or strenuous activity. EAH can easily be mistaken for dehydration, heat illness, or acute altitude illnesses[80,81] because of overlapping signs and symptoms if the diagnosis is not considered (Table 1).

Differentiation between dehydration and EAH is critical as provision of isotonic or hypotonic fluids is appropriate for the dehydrated athlete,[82] whereas such treatment could be detrimental for an athlete with EAH, in whom the administration of these hypotonic or isotonic fluids may worsen symptoms or delay recovery.[14,22,23,25,35,76,83,90] A conclusion of the Second International Exercise- Associated Hyponatremia Consensus Development Conference was that“medical directors should ensure the availability of onsite serum sodium concentration analysis.”[7] When EAH is routinely considered in the differential diagnosis of a collapsed athlete and point-of-care serum sodium concentration analysis is available, the field diagnosis of EAH becomes straightforward. The reality is that on-site analysis of serum sodium concentration is not widely available at organized endurance competitions, nor is it currently feasible to widely implement. Even relatively large and established events often have no capacity for on-site blood analysis. This is also the case with most wilderness activities.

Therefore, we provide strategies for the following 2 scenarios (Figure 2).

Scenario 1: An EAH diagnosis has been made by point-of-care sodium analysis.

Scenario 2: Point-of-care sodium analysis is not available, and the diagnosis of EAH is presumed.

THERAPEUTIC OPTIONS FOR BOTH SCENARIOS

Fluids

An important element in the treatment of EAH is to avoid exacerbating the condition with improper fluid management. If EAH is clinically suspected, an assessment of volume status should be completed before treatment with intravenous (IV) fluids. It must be made clear that inappropriate IV fluid

administration risks exacerbating hyponatremia with potentially devastating consequences.[14,22,23,25,35,76,83–90] Thus, clear indications (such as hypotension or unstable blood pressure) should be present to support administration of IV fluids. The use of hypotonic IV fluids should be avoided. If the patient does not have clear indications for IV fluids and EAH is suspected, then fluid restriction while the patient is being transported to a medical center should be instituted.

Recommendation: Hypotonic or isotonic fluid intake should be restricted in known or suspected EAH until urination begins. Recommendation grade: 1A.

Supplemental oxygen

Hypoxemia from pulmonary edema has been reported in EAH.[22] As a supportive intervention, supplemental oxygen (flow rate 2–4 L/min nasal) should be provided to treat any respiratory distress if available.

Recommendation: Respiratory symptoms should be supported with supplemental oxygen if available. Recommendation grade: 1B.

Appropriate transfer of care

The intent of field management is to stabilize the patient until they can be transferred to a definitive care medical facility. Unfortunately, EAH recognition is challenging,[91] and appropriate management is not universally understood. Therefore, when transferring care, it is critical to relay the potential diagnosis of EAH and to caution the transport team about the dangers of aggressive IV hydration with isotonic or hypotonic fluids. Ideally, an IV saline lock should be placed for EMS transport, and the provision of IV fluids should be based on clear indications of marked hypovolemia such as sustained hypotension. However, if the transport team insists on provision of isotonic or hypotonic fluids, they should be cautioned that a patient with EAH could experience worsening symptoms with this intervention. If symptoms worsen in this scenario, IV fluids should be stopped, and consideration of immediate hypertonic (3%) saline administration should occur.

Recommendation: When transferring care, receiving caregivers should be alerted to the potential diagnosis of EAH and appropriate fluid management. Recommendation grade: 1B.

SPECIFIC RECOMMENDATIONS—SCENARIO 1 (BLOOD SODIUM ESTIMATION IS AVAILABLE)

Clinical assessment

The portability of point-of-care testing devices means that they may be available to confirm a diagnosis of EAH, for instance at a mass-participation wilderness sporting event or on a well-equipped expedition.[30,92–94] In general, a sodium level of 130 mmol/L or higher will be minimally symptomatic or asymptomatic, whereas levels below this are increasingly likely to be

symptomatic.[14,22,23,25,26,35,76,83,85–90,92,93] Symptoms and signs of EAHE (ranging from headache and nausea or vomit- ing to confusion and lethargy) are key elements in making the diagnosis. Although the early symptoms of EAH may be nonspecific, the presence of altered mental status, coma, seizures, or respiratory distress (suggesting pulmonary edema)supports the diagnosis of EAHE[7,22,25,83] and should be promptly recognized.

Recommendation: A rapid assessment for signs and symptoms of cerebral edema or noncardiogenic pulmonary edema should be made in all patients with possible EAH. Recommendation grade: 1B.

Table 1. Signs and symptoms of exercise-associated hyponatremia and heat illness or altitude illness

General

Sign/Symptom	EAH	Heat illness	AMS, HACE, or HAPE
Fatigue/weakness	Possible	Possible	Likely
Increased thirst	Possible	Likely	Possible
Temperature: Elevated	Possible	Present	Not present
Tachycardia	Possible	Likely	Possible
Orthostasis	Possible	Likely	Possible
Nausea/vomiting	Possible	Possible	Possible
Headache/dizziness	Possible	Possible	Present
Blurred vision	Possible	Possible	Possible
Confusion/disorientation	Possible	Possible	Possible
Obtundation	Possible	Possible	Possible
Seizure	Possible	Possible	Possible
Coma	Possible	Possible	Possible
Respiratory distress	Possible	Not present	Possible
Oliguria	Possible	Likely	Possible
Diuresis	Possible	Not present	Possible

AMS, acute mountain sickness; EAH, exercise-associated hyponatremia; HACE, high altitude cerebral edema; HAPE, high altitude pulmonary edema.

Developing signs of cerebral or pulmonary edema (non-cardiogenic) signify an urgent medical condition requiring emergent care. In such situations, urgent blood sodium measurement is invaluable in guiding initial therapy.[94]

Recommendation: When point-of-care sodium analysis is available in the field and EAH is suspected, blood sodium measurement should be obtained as rapidly as possible. Recommendation grade: 1B.

Hypertonic saline

It is possible to commence and indeed even complete treatment for EAH in the field.[92-94] Individuals with EAH who are neurologically stable can be advised to limit fluid intake and consume salty snacks, soups or bouillon, or a small volume of hypertonic fluid until the onset of urination. They should be observed for at least 60 minutes during the initial post exercise period because water remaining in the gastrointestinal tract can be quickly absorbed at the cessation of exercise and result in rapid development of symptoms from EAH.[22,25,83] More urgent medical attention, including planning for transfer to definitive care, is required if signs or symptoms of EAH develop. Once any neurological symptoms more serious than headache develop, regardless of the degree of hyponatremia, treatment with hypertonic saline is indicated. When able to tolerate oral intake, a hypertonic (approximately 9% saline) solution of concentrated broth (3–4 bouillon cubes in 125 mL [1/2 cup] of water) would be an appropriate initial treatment.[92–94]

Recommendation: Oral hypertonic saline solutions are an appropriate intervention in the field for cases of EAH when oral intake is possible. Recommendation grade: 1B.

If the individual is unable to tolerate oral intake, or when there is no improvement or symptoms worsen with oral hypertonic saline, the recommended treatment is a 100-mL bolus of 3% hypertonic saline infused through a peripheral vein in less than 60 seconds. This can be repeated 2 additional times at 10-minute intervals if there is no clinical improvement.[7] Experience has proven this treatment to be without untoward symptoms at the infusion site (no burning, phlebitis, or residual discomfort) and no risk of osmotic demyelination or central pontine myelinolysis.[7,22,23,25,35,76,83,85–87,92,93]

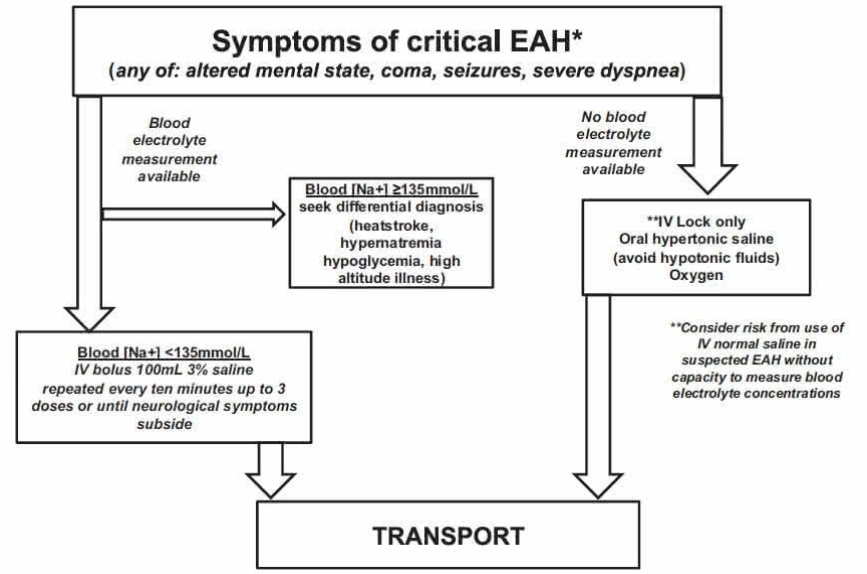
Recommendation: Symptomatic biochemically confirmed EAH can be treated in the field with a 100-mL bolus of 3% hypertonic saline, which can be repeated twice at 10-minute intervals (3 doses in total) with the aim of acutely increasing serum sodium concentration by about 4 to 5 mmol/L and reversing cerebral edema in the setting of acute hyponatremia. Recommendation grade: 1B.

SPECIFIC RECOMMENDATIONS—SCENARIO 2 (BLOOD SODIUM ESTIMATION IS NOT AVAILABLE)

When the capacity for on-site serum sodium measurement is not available, the decision-making process becomes challenging. Unfortunately, the possible signs and symptoms that can be present with EAH are quite similar to those present with heat illness, dehydration, or acute mountain sickness (Table 1). Furthermore, there were no differences between those experiencing mild EAH and those not experiencing EAH after a 161-km ultramarathon in terms of various individual characteristics, signs, and symptoms.[91] Even oliguria, which would be typical of the dehydrated state, is also commonly seen with EAH when AVP secretion is part of the pathophysiological mechanism leading to a highly concentrated, low-volume urine output.[23]

In some environments in which overhydration is a key feature in the underlying etiology of EAH, those experiencing EAH have been shown to be more likely to lose less weight or to gain weight during the exercise when compared with those not experiencing EAH.[42] However, in other environments, it is not at all uncommon for those with EAH to have considerable weight loss, suggesting other mechanisms in the development of EAH.[17-19] Therefore, changes in body weight are not universally helpful in making the diagnosis of EAH. This unfortunately means that the only reliable method of diagnosing EAH at present is through measurement of serum sodium concentration. On the other hand, it now appears that those developing symptomatic EAH generally have inadequate weight loss during the exercise.[42,76] So, if it is known that a symptomatic athlete has gained weight or lost little weight during the exercise, clinical suspicion of EAH may be raised.

Figure 2.Algorithm for exercise-associated hyponatremia (EAH) field management.



Asymptomatic EAH is generally not seen unless blood tests are obtained for other reasons. When only mild symptoms are present, treatment can be with either fluid restriction or oral hypertonic solutions (if tolerated) until the onset of urination.

Fluid restriction

A high clinical suspicion of symptomatic EAH necessitates fluid restriction and salt supplementation. Certainly in events such as long ultramarathon races in which the incidence of EAH may be high (30%–51%),[13,17-19,92] one should resist treating athletes with IV hypotonic or isotonic saline without certainty that they do not have EAH.[14,22,23,25,35,76,83,85-90] However, fluid restriction is contraindicated in the case of dehydration and rhabdomyolysis with impending acute kidney injury.[82] Therefore, in the situation in which the diagnosis of EAH is uncertain, the potential benefits of fluid restriction if the individual has EAH must be weighed against the potential harm that could result when the individual might have dehydration, rhabdomyolysis, and impending acute renal failure.

Recommendation: Hypotonic or isotonic fluids should be restricted in suspected EAH with consideration of the potential harm that could result from fluid restriction if the diagnosis is incorrect. Recommendation grade: 1C.

Hypertonic saline

In the event of neurological deterioration without access to rapid determination of serum sodium concentration, the use of IV hypertonic saline, if available, should be considered for presumed EAH. Such an intervention carries minimal potential risks, and the benefit that could be derived from a bolus of hypertonic saline of approximately 51 mmol of sodium for fluid volume expansion and the limited effect it would have on increasing blood sodium concentration suggest that the risk is low, even under conditions of dehydration and hypernatremia. When the patient is neurologically stable, oral sodium with limited fluid has been demonstrated to be an appropriate treatment.[92-94] In the field, this could be prepared by dissolving 3 to 4 bouillon cubes in 125 mL (1/2 cup) of water (approximately 9% saline).

Recommendation: IV hypertonic saline (100-mL bolus of 3% hypertonic saline, which can be repeated twice at 10-minute intervals) is an appropriate consideration in suspected EAH with neurological deterioration, whereas an oral hypertonic saline solution would be an appropriate consideration in suspected mild EAH. Recommendation grade: 1C.

Emergency transport

When point-of-care serum sodium concentration cannot be determined and any attempted field treatment has been insufficiently successful, emergency transport to a definitive care facility should be expedited. Organized endurance exercise events that do not have the on-site capacity for measurement of serum sodium concentration and treatment with hypertonic saline should have pre- arranged emergency transport systems. Local emergency department physicians and transport personnel should also be educated about EAH in advance of the event.

Recommendation: The assurance that an emergency transport system is in place is critical when point-of-care serum sodium measurement will not be available or treatment with hypertonic saline will not be feasible. Recommendation grade: 1C.

IMMEDIATE MEDICAL CARE IN-HOSPITAL ASSESSMENT

The medical care of suspected EAH in hospital is presumed to occur in a facility that has the capacity to measure an urgent sodium level either by point-of-care testing or in a hospital laboratory. The sodium level and a clinical assessment for signs of cerebral edema are the key factors that will determine urgent treatment. The primary intervention (when indicated) is the use of IV hypertonic saline, usually as a 3% solution, to acutely increase serum sodium and reduce cerebral edema, whereas the role of other therapeutic agents such as vasopressin receptor antagonists (vaptans), urea, oral saline solutions, and diuretics in the

treatment of acute symptomatic cases of EAH has not been firmly established.[8] See Table 2 for a summary of critical steps for immediate medical care in a receiving hospital.

Urgent sodium estimation

The in-hospital diagnosis of EAH is made in an appropriate clinical context, whether or not signs or symptoms are present, by the determination of a sodium level below the normal reference range. In general, a sodium level of 130 mmol/L or higher will be minimally symptomatic or asymptomatic,[7,8,92,93] whereas levels below this are likely to be symptomatic.[14,22,23,25,35,76,83,85–90] Developing signs of cerebral edema signify an urgent medical condition requiring emergent care.[7,22,23,25,76,83,94]

Recommendation: With suspected EAH, and particularly in those with altered mental status, sodium estimation should be obtained as rapidly as possible after hospital arrival. Recommendation grade: 1B.

Assessment for cerebral and pulmonary edema

Symptoms and signs of cerebral edema are a key element in making the diagnosis of severe or clinically significant EAH. Although the early symptoms of EAH may be nonspecific, the presence of altered mental state, coma, seizures, or respiratory distress (suggesting pulmonary edema) indicates severe EAH.[7,22,23,25,76,83] Such an assessment is made clinically at the bedside. It does not require imaging or scans and should never delay the use of IV hypertonic saline when indicated (see sub-sequent sections).

Recommendation: A rapid assessment for signs and symptoms of cerebral edema or noncardiogenic pulmonary edema should be made in all patients with possible EAH. Recommendation grade: 1B.

Other laboratory testing

Although not essential to guide initial therapy, there are other laboratory tests that can help to better delineate the pathophysiology of EAH in an individual patient and help guide subsequent treatment if more prolonged in-hospital care is required.[4] These tests may help to differentiate euvolemic from hypovolemic EAH and the role of AVP and brain natriuretic peptide in its pathogenesis.[7,8,23,41,95] Such tests are best taken before therapy is commenced even if they are only stored for subsequent analysis. However, such testing should never delay the use of IV hypertonic saline when indicated (see subsequent sections).

Recommendation: When possible, urine for sodium and osmolality and blood for osmolality should be obtained before commencement of treatment. Recommendation grade: 2C.

Table 2.Summary of acute hospital assessment and management of EAH

Assessment

- Urgent measurement of blood sodium by the most rapidly available means
- Assess for clinical signs suggestive of developing cerebral edema
- Obtain and store specimens if possible for later analysis of blood serum osmolality and urine sodium and osmolality

Management

- Supplemental oxygen to maintain oxygen saturation above 95%
- Restrict fluids (both IV and oral) until onset of urination
- Avoid IV normal saline until sodium correction is initiated
- Thereafter normal saline may be required for hypovolemic shock or in renal protection therapy for rhabdomyolysis
- In severe EAH (signs of cerebral edema or serum sodium < 125 mmol/L) administer IV 3% hypertonic saline as a 100-mL bolus repeated twice at 10-minute intervals aiming to reverse cerebral edema
- Aim to increase serum sodium by approximately 4 to 5 mmol/L or until neurological symptoms are reversed by active treatment, then allow the remaining correction to occur spontaneously via urinary free water excretion

Fluid restriction

Mild or asymptomatic EAH (essentially a biochemical- only diagnosis) will usually resolve without treatment during a period of observation. Hypotonic fluids, whether taken orally or given IV, will generally worsen the situation, especially before the onset of urination. IV normal saline will worsen EAH acutely in the presence of osmotically inappropriate (nonosmotic) AVP secretion,[14,22,23,25,35,76,83,85-90] but may be required later in specific clinical contexts such as the prevention of renal injury in rhabdomyolysis or the treatment of hypovolemic shock.

Recommendation: Oral and IV hypotonic or isotonic hydration should be avoided early in the management of EAH although it may be appropriate in certain clinical contexts once sodium correction has been initiated or hypovolemia is biochemically confirmed (by elevated blood urea nitrogen and urine sodium less than 30 mmol/ L). Recommendation grade: 1B.

Hypertonic saline

The most commonly available form of IV hypertonic saline is a 3% solution.[4,25,76,83,92-94] Hypertonic saline will acutely raise the serum sodium, resulting in a fluid shift that will decrease cerebral edema. A 100-mL solution of 3% hypertonic saline contains 51 mmol of sodium and in the average adult would be expected to increase the serum sodium by 1 to 2 mmol/L. It is used whenever there are signs of significant cerebral edema in EAH.[7,22,76,83,94] It may also be indicated in severe biochemical hyponatremia (o125 mmol/L) when initially presenting as asymptomatic or mildly symptomatic, as these cases have been known to progress to EAHE.[23,76] When using IV hypertonic saline, the aim is not to normalize the serum sodium concentration but rather to reverse cerebral edema while preventing or treating the life-threatening consequences of EAHE.[4,7,94] In general, this will require an increase in the sodium level of about

4 to 5 mmol/L. Thereafter, further normalization of sodium is not urgent and may be best allowed to occur spontaneously through suppression of nonosmotic AVP secretion and resultant urinary free water excretion. The use of IV hypertonic saline in EAH appears to be safe,[7,92,93] with no recorded cases of osmotic demyelination known to have occurred as opposed to the situation with rapid correction of chronic hyponatremia.[7,8]

Recommendation: In hospital, severe biochemically confirmed or symptomatic EAH should be treated with a 100-mL bolus of 3% hypertonic saline, which can be repeated twice at 10-minute intervals (3 doses in total), with the aim of acutely increasing serum sodium concentration by about 4 to 5 mmol/L and reversing cerebral edema. Recommendation grade: 1A.

Supplemental oxygen

Although the major manifestation of EAH is cerebral, pulmonary manifestations can occur.[22] Hypoxemia, which may worsen cerebral injury, should be avoided, but hyperoxia may also have detrimental effects.[96]

Recommendation: Supplemental oxygen to maintain an oxygenation saturation of 95% should be provided to treat hypoxemia from pulmonary edema when evident. Recommendation grade: 1B.

Conclusions

Exercise-associated hyponatremia has a complex pathogenesis and multifactorial etiology. It can result in devastating outcomes to participants in both organized or individual endurance activities in urban or in remote backcountry environments.

Preventing EAH is the key factor in protecting participants in endurance events and other wilderness activities. Currently, there is no one recommendation that fits all individuals for fluid and salt consumption during endurance events, although prudent general guidelines include drinking to thirst and specifically avoiding excessive fluid intake. There is an ongoing need for education to ensure that participants understand the risk of overhydration. Furthermore, a knowledge gap persists internationally among practitioners and prehospital EMS personnel about the assessment and treatment of EAH, which is compounded by the nonspecific nature of many of the signs and symptoms of EAH. The typical field response is to administer rapid isotonic IV fluids to endurance activity participants in the suspicion they are dehydrated. However, such universal treatment may result in increased morbidity and mortality in the EAH patient. A Supplementary Evidence Table 2is available online.

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Supplementary tables

Supplementary ACCP Table 1 and Evidence Table 2 are available online atdoi:10.1016/j.wem.2014.08.009.

References

1. Noakes TD, Goodwin N, Rayner BL, Branken T, Taylor RK. Water intoxication: a possible complication during endurance exercise.Med Sci Sports Exerc. 1985;17:370– 375.
2. Montain SJ, Sawka MN, Wenger CB. Hyponatremia associated with exercise: risk factors and pathogenesis. Exerc Sport Sci Rev. 2001;29:113– 117.
3. Speedy DB, Noakes TD, Schneider C. Exercise-associated hyponatremia: a review. Emerg Med (Fremantle). 2001;13:17– 27.
4. Rosner MH, Kirven J. Exercise-associated hyponatremia. Clin J Am Soc Nephrol. 2007;2:151– 161.
5. Rosner MH. Exercise-associated hyponatremia. Semin Nephrol. 2009;29:271– 281.
6. Rosner MH, Bennett B, Hew-Butler T, Hoffman MD. Exercise-associated hyponatremia. In: Simon EE, ed. Hyponatremia: Evaluation and Treatment. New York, NY: Springer; 2013:175– 192.
7. Hew-Butler T, Ayus JC, Kipps C, et al. Statement of the Second International Exercise-Associated Hyponatremia Consensus Development Conference, New Zealand, 2007.Clin J Sport Med. 2008;18:111– 121.
8. Hew-Butler T, Almond C, Ayus JC, et al. Exercise- Associated Hyponatremia (EAH) Consensus Panel. Consensus statement of the 1st International Exercise- Associated Hyponatremia Consensus Development Con- ference, Cape Town, South Africa 2005.Clin J Sport Med. 2005;15:208– 213.
9. Backer HD, Shopes E, Collins SL. Hyponatremia in recreational hikers in Grand Canyon National Park. J Wilderness Med. 1993;4:391– 406.
10. Basnyat B, Sleggs J, Spinger M. Seizures and delirium in a trekker: the consequences of excessive water drinking? Wilderness Environ Med. 2000;11:69– 70.
11. Rothwell SP, Rosengren DJ. Severe exercise-associated hyponatremia on the Kokoda Trail, Papua New Guinea. Wilderness Environ Med. 2008;19:42– 44.
12. Zafren K. Hyponatremia in a cold environment.Wilder- ness Environ Med. 1998;9:54– 55.
13. Stuempfle KJ, Lehmann DR, Case HS, et al. Hypona- tremia in a cold weather ultraendurance race.Alaska Med. 2002;44:51– 55.
14. Coler C, Hoffman MD, Towle G, Hew-Butler T. Hypo- natremia in an 85-year-old hiker: when depletion plus dilution produces delirium. Wilderness Environ Med. 2012;23:153– 157.
15. Rogers IR, Hew-Butler T. Exercise-associated hyponatre- mia: overzealousfluid consumption.Wilderness Environ Med. 2009;20:139– 143.

16. Guyatt G, Gutterman D, Baumann MH, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force.Chest. 2006;129:174–181.
17. Lebus DK, Casazza GA, Hoffman MD, Van Loan MD. Can changes in body mass and total body water accurately predict hyponatremia after a 161-km running race?Clin J Sport Med. 2010;20:193– 199.
18. Hoffman MD, Stuempfle KJ, Rogers IR, Weschler LB, Hew-Butler T. Hyponatremia in the 2009 161-km Western States Endurance Run. Int J Sports Physiol Perform. 2012;7:6– 10.
19. Hoffman MD, Hew-Butler T, Stuempfle KJ. Exercise-associated hyponatremia and hydration status in 161-km ultramarathoners.Med Sci Sports Exerc. 2013;45:784– 791.
20. Noakes T.Waterlogged: The Serious Problem of Over-hydration in Endurance Sports.Champaign, IL: Human Kinetics; 2012.
21. Kipps C, Sharma S, Pedoe DT. The incidence of exercise- associated hyponatraemia in the London marathon.Br J Sports Med. 2011;45:14– 19.
22. Ayus JC, Varon J, Arieff AI. Hyponatremia, cerebral edema, and noncardiogenic pulmonary edema in marathon runners.Ann Intern Med. 2000;132:711– 714.
23. Siegel AJ, Verbalis JG, Clement S, et al. Hyponatremia in marathon runners due to inappropriate arginine vasopres- sin secretion.Am J Med. 2007;120:461.e11–e17.
24. Hew TD, Chorley JN, Cianca JC, Divine JG. The incidence, risk factors, and clinical manifestations of hyponatremia in marathon runners.Clin J Sport Med. 2003;13:41– 47.
25. Davis DP, Videen JS, Marino A, et al. Exercise-associated hyponatremia in marathon runners: a two-year experience. J Emerg Med. 2001;21:47– 57.
26. Speedy DB, Noakes TD, Rogers IR, et al. Hyponatremia in ultradistance triathletes. Med Sci Sports Exerc. 1999;31:809– 815.
27. Lee JK, Nio AQ, Ang WH, et al. First reported cases of exercise-associated hyponatremia in Asia. Int J Sports Med. 2011;32:297– 302.
28. Glace B, Murphy C. Severe hyponatremia develops in a runner following a half-marathon.JAAPA. 2008;21:27– 29.
29. Shapiro SA, Ejaz AA, Osborne MD, Taylor WC. Moder- ate exercise-induced hyponatremia. Clin J Sport Med. 2006;16:72– 73.
30. Backer HD, Shopes E, Collins SL, Barkan H. Exertional heat illness and hyponatremia in hikers.Am J Emerg Med. 1999;17:532– 539.
31. Noe RS, Choudhary E, Cheng-Dobson J, Wolkin AF, Newman SB. Exertional heat-related illnesses at the Grand Canyon National Park, 2004–2009.Wilderness Environ Med. 2013;24:422– 428.
32. Gardner JW. Death by water intoxication. Mil Med. 2002;167:432– 434.
33. Garigan TP, Ristedt DE. Death from hyponatremia as a result of acute water intoxication in an Army basic trainee. Mil Med. 1999;164:234– 238.
34. O'Donnell FL, ed. Army Medical Surveillance Activity. Update: exertional hyponatremia, active component, U.S. Armed Forces, 1999–2012.Medical Surveillance Monthly Report. 2013:20;23–28.

35. Zelingher J, Putterman C, Ilan Y, et al. Case series: hyponatremia associated with moderate exercise.Am J Med Sci. 1996;311:86– 91.
36. Rose BD, Post TW.Clinical Physiology of Acid-Base and Electrolyte Disorders. 5th ed. New York, NY: McGraw Hill; 2001.
37. Hew-Butler T, Dugas JP, Noakes TD, Verbalis JG. Changes in plasma arginine vasopressin concentrations in cyclists participating in a 109-km cycle race.Br J Sports Med. 2010;44:594– 597.
38. Rowe JW, Shelton RL, Helderman JH, Vestal RE, Robertson GL. Influence of the emetic reflex on vaso- pressin release in man.Kidney Int. 1979;16:729–735.
39. Baylis PH, Zerbe RL, Robertson GL. Arginine vasopressin response to insulin-induced hypoglycemia in man.J Clin Endocrinol Metab. 1981;53:935–940.
40. Hew-Butler T, Jordaan E, Stuempfle KJ, et al. Osmotic and nonosmotic regulation of arginine vasopressin during prolonged endurance exercise.J Clin Endocrinol Metab. 2008;93:2072– 2078.
41. Harris G, Reid S, Sikaris K, McCrory P. Hyponatremia is associated with higher NT-proBNP than normonatremia after prolonged exercise.Clin J Sport Med. 2012;22:488– 494.
42. Noakes TD, Sharwood K, Speedy D, et al. Three independent biological mechanisms cause exercise- associated hyponatremia: evidence from 2,135 weighed competitive athletic performances.Proc Natl Acad Sci USA. 2005;102:18550– 18555.
43. Edelman IS, James AH, Brooks L, Moore FD. Body sodium and potassium. IV. The normal total exchangeable sodium; its measurement and magnitude.Metabolism. 1954;3:530– 538.
44. Edelman IS, James AH, Baden H, Moore FD. Electrolyte composition of bone and the penetration of radiosodium and deuterium oxide into dog and human bone.J Clin Invest. 1954;33:122– 131.
45. Halperin ML, Kamel KS, Sterns R. Hyponatremia in marathon runners.N Engl J Med. 2005;353:427– 428.
46. Lindinger MI, Heigenhauser GJ, McKelvie RS, Jones NL. Blood ion regulation during repeated maximal exercise and recovery in humans. Am J Physiol. 1992;262(1 Pt 2): R126–R136.
47. Buono MJ, Ball KD, Kolkhorst FW. Sodium ion concen- tration vs. sweat rate relationship in humans. J Appl Physiol. 2007;103:990– 994.
48. Buono MJ, Sjoholm NT. Effect of physical training on peripheral sweat production.J Appl Physiol. 1988;65:811– 814.
49. Speedy DB, Noakes TD, Boswell T, Thompson JM, Rehrer N, Boswell DR. Response to afluid load in athletes with a history of exercise induced hyponatremia.Med Sci Sports Exerc. 2001;33:1434– 1442.
50. Noakes TD, Wilson G, Gray DA, Lambert MI, Dennis SC. Peak rates of diuresis in healthy humans during oralfluid overload.S Afr Med J. 2001;91:852– 857.
51. Almond CS, Shin AY, Fortescue EB, et al. Hyponatremia among runners in the Boston Marathon.N Engl J Med. 2005;352:1550– 1556.
52. Hew-Butler T, Hoffman MD, Stuempfle KJ, Rogers IR, Morgenthaler NG, Verbalis JG. Changes in copeptin and bioactive vasopressin in runners with

and without hypo- natremia.Clin J Sport Med. 2011;21:211– 217.
53. Schmidt W, Rojas J, Böning D, Bernal H, Garcia S, Garcia O. Plasma-electrolytes in natives tohypoxia after marathon races at different altitudes.Med Sci Sports Exerc. 1999;31:1406– 1413.
54. Hellmann K, Weiner JS. Antidiuretic substance in urine following exposure to high temperatures.J Appl Physiol. 1953;6:194– 198.
55. Grant SM, Green HJ, Phillips SM, Enns DL, Sutton JR. Fluid and electrolyte hormonal responses to exercise and acute plasma volume expansion.J Appl Physiol. 1996;81: 2386 – 2392.
56. Stebbins CL, Symons JD, McKirnan MD, Hwang FF. Factors associated with vasopressin release in exercising swine.Am J Physiol. 1994;266(1 Pt 2):R118–R124.
57. Siegel AJ. Exercise-associated hyponatremia: role of cytokines.Am J Med. 2006;119(7 Suppl 1):S74–S78.
58. Wharam PC, Speedy DB, Noakes TD, Thompson JM, Reid SA, Holtzhausen LM. NSAID use increases the risk of developing hyponatremia during an Ironman triathlon. Med Sci Sports Exerc. 2006;38:618– 622.
59. Baker J, Cotter JD, Gerrard DF, Bell ML, Walker RJ. Effects of indomethacin and celecoxib on renal function in athletes.Med Sci Sports Exerc. 2005;37:712– 717.
60. Walker RJ, Fawcett JP, Flannery EM, Gerrard DF. Indome- thacin potentiates exercise-induced reduction in renal hemody- namics in athletes.Med Sci Sports Exerc. 1994;26:1302– 1306.
61. Hew-Butler T, Verbalis JG, Noakes TD, International Marathon Medical Directors Association. Updated fluid recommendation: position statement from the International Marathon Medical Directors Association (IMMDA).Clin J Sport Med. 2006;16:283– 292.
62. Cheuvront SN, Haymes EM. Ad libitumfluid intakes and thermoregulatory responses of female distance runners in three environments.J Sports Sci. 2001;19:845– 854.
63. Armstrong LE, Maresh CM, Gabaree CV, et al. Thermal and circulatory responses during exercise: effects of hypohydration, dehydration, and water intake. J Appl Physiol. 1997;82:2028– 2035.
64. Hoffman MD, Stuempfle KJ. Hydration strategies, weight change and performance in a 161-km ultramarathons.Res Sports Med. 2014;22:213–225.
65. Speedy DB, Rogers IR, Noakes TD, et al. Diagnosis and prevention of hyponatremia at an ultradistance triathlon. Clin J Sport Med. 2000;10:52– 58.
66. Speedy DB, Thompson JM, Rodgers I, Collins M, Shar- wood K, Noakes TD. Oral salt supplementation during ultradistance exercise.Clin J Sport Med. 2002;12:279– 284.
67. Hew-Butler TD, Sharwood K, Collins M, Speedy D, Noakes T. Sodium supplementation is not required to maintain serum sodium concentrations during an Ironman triathlon.Br J Sports Med. 2006;40:255– 259.
68. Twerenbold R, Knechtle B, Kakebeeke TH, et al. Effects of different sodium concentrations in replacementfluids during prolonged exercise in women.Br J Sports Med. 2003;37:300– 303.

69. Barr SI, Costill DL, Fink WJ. Fluid replacement during prolonged exercise: effects of water, saline, or nofluid. Med Sci Sports Exerc. 1991;23:811– 817.
70. Vrijens DM, Rehrer NJ. Sodium-free fluid ingestion decreases plasma sodium during exercise in the heat.J Appl Physiol. 1999;86:1847– 1851.
71. Röcker L, Kirsch KA, Heyduck B, Altenkirch HU. Influence of prolonged physical exercise on plasma volume, plasma proteins, electrolytes, and fluid-regulating hormones.Int J Sports Med. 1989;10:270– 274.
72. Beckner GL, Winsor T. Cardiovascular adaptations to prolonged physical effort.Circulation. 1954;9:835– 846.
73. Riley WJ, Pyke FS, Roberts AD, England JF. The effect of long-distance running on some biochemical variables.Clin Chim Acta. 1975;65:83– 89.
74. Åstrand PO, Saltin B. Plasma and cell volume after prolonged severe exercise.J Appl Physiol. 1964;19:829– 832.
75. Weschler LB. Exercise-associated hyponatraemia: a math- ematical review.Sports Med. 2005;35:899– 922.
76. Hoffman MD, Stuempfle KJ, Sullivan K, Weiss RH. Exercise-associated hyponatremia with exertional rhabdo- myolysis: importance of proper treatment.Clin Nephrol. 2014 Jun 16. [Epub ahead of print].
77. Luks AM, Robertson HT, Swenson ER. An ultracyclist with pulmonary edema during the Bicycle Race Across America.Med Sci Sports Exerc. 2007;39:8–12.
78. Williams J, Tzortziou Brown V, Malliaras P, Perry M, Kipps C. Hydration strategies of runners in the London Marathon.Clin J Sport Med. 2012;22:152–156.
79. Winger JM, Dugas JP, Dugas LR. Beliefs about hydration and physiology drive drinking behaviours in runners.Br J Sports Med. 2011;45:646– 649.
80. Ayus JC, Moritz ML. Exercise-associated hyponatremia masquerading as acute mountain sickness: are we missing the diagnosis?Clin J Sport Med. 2008;18:383– 386.
81. Sucholeiki R. Heatstroke.Semin Neurol. 2005;25:307– 314.
82. Patel DR, GyamfiR, Torres A. Exertional rhabdomyolysis and acute kidney injury.Phys Sportsmed. 2009;37:71– 79.
83. Frizzell RT, Lang GH, Lowance DC, Lathan SR. Hypona- tremia and ultramarathon running.JAMA. 1986;255:772– 774.
84. Bennett BL, Hew-Butler T, Hoffman MD, Rogers IR, Rosner MH. In reply to Clinical practice guidelines for treatment of exercise-associated hyponatremia.Wilderness Environ Med. 2013;24:468– 471.
85. Hew-Butler TD, Boulter J, Bhorat R, Noakes TD. Avoid adding insult to injury —correct management of sick female endurance athletes.S Afr Med J. 2012;102:927– 930.
86. Kormann R, Philippart F, Hubert S, Bruel C. Marathon runner with acute hyponatremia: a neurological disorder. Case Rep Emerg Med. 2012;2012:342760. doi: 10.1155/ 2012/342760. Epub 2012 May 31.
87. Reynolds CJ, Cleaver BJ, Finlay SE. Exercise associated hyponatraemia leading to tonic-clonic seizure.BMJ Case Rep. 2012 Aug 27;2012.
88. Severac M, Leplatois T, Ichai C. A near-fatal case of exercise-associated hyponatremia. Am J Emerg Med. 2014;32:813.e1–e2.

89. Spano SJ, Reagle Z, Evans T. Symptomatic hypotonic hyponatremia presenting at high altitude. Wilderness Environ Med. 2014;25:69– 74.
90. Thompson J-A, Wolff AJ. Hyponatremic encephalopathy in a marathon runner.Chest. 2003;124:313S.
91. Hoffman MD, Fogard K, Winger J, Hew-Butler T, Stuempfle KJ. Characteristics of 161-km ultramarathon finishers developing exercise-associated hyponatremia.Res Sports Med. 2013;21:164– 175.
92. Rogers IR, Hook G, Stuempfle KJ, Hoffman MD, Hew-Butler T. An intervention study of oral versus intra- venous hypertonic saline administration in ultramarathon runners with exercise-associated hyponatremia: a prelimi- nary randomized trial. Clin J Sport Med. 2011;21: 200 – 203.
93. Owen BE, Rogers IR, Hoffman MD, et al. Efficacy of oral versus intravenous hypertonic saline in runners with hyponatremia.J Sci Med Sport. 2014;17:457– 462.
94. Siegel AJ, d'Hemecourt P, Adner MM, Shirey T, Brown JL, Lewandrowski KB. Exertional dysnatremia in col- lapsed marathon runners: a critical role for point-of-care testing to guide appropriate therapy.Am J Clin Pathol. 2009;132:336– 340.
95. Verbalis JG, Goldsmith SR, Greenberg A, Schrier RW, Sterns RH. Hyponatremia treatment guidelines 2007: expert panel recommendations.Am J Med. 2007;120(Suppl 1):S1–S21.
96. Iscoe S, Beasley R, Fisher JA. Supplementary oxygen for nonhypoxemic patients: O2 much of a good thing?Crit Care. 2011;15:305.

Wilderness Medical Society Clinical Practice Guidelines for the Out-of-Hospital Evaluation and Treatment of Accidental Hypothermia: 2019 Update

Jennifer Dow MD, MHA[1] Gordon G.Giesbrecht PhD[2] Daniel F.Danzl MD[3] Hermann Brugger MD[45] Emily B.Sagalyn MD, MPH[6] Beat Walpoth MD[7] Paul S.Auerbach MD [8] Scott E.McIntosh MD, MPH[9] MáriaNémethy MD[10]Marion McDevittDO, MPH[10]Robert B.Schoene MD[11]George W.Rodway PhD, APRN[12]Peter H.Hackett MD[1314]KenZafren MD[48]Brad L.Bennett PhD[15]Colin K.Grissom MD[16]

To provide guidance to clinicians, the Wilderness Medical Society convened an expert panel to develop evidence-based guidelines for the out-of-hospital evaluation and treatment of victims of accidental hypothermia. The guidelines present the main diagnostic and therapeutic modalities and provide recommendations for the management of hypothermic patients. The panel graded the recommendations based on the quality of supporting evidence and a balance between benefits and risks/burdens according to the criteria published by the American College of Chest Physicians. The guidelines also provide suggested general approaches to the evaluation and treatment of accidental hypothermia that incorporate specific recommendations. This is the 2019 update of the Wilderness Medical Society Practice Guidelines for the Out-of-Hospital Evaluation and Treatment of Accidental Hypothermia: 2014 Update.

Keywords

rewarming
resuscitation
wilderness medicine
cold
shivering

Introduction

Accidental hypothermia is defined as an unintentional drop in core temperature to 35°C or lower. Accidental hypothermia due to environmental exposure can occur during any season and in most climates, with cold and wet environments posing

the greatest risk. Throughout history, it has been a disease of war and disasters, but those who work and recreate outside, especially in the wilderness, place themselves at risk for hypothermia.

In addition to occurring in wilderness environments, hypothermia is associated with urban homelessness, particularly with the use of alcohol and other intoxicating substances. Hypothermia can occur during resuscitation in emergency settings (iatrogenic hypothermia); is notably associated with trauma; and may be a feature of sepsis, diseases that decrease metabolic rate (including hypoendocrine states), and diseases that affect thermoregulation. Therapeutic hypothermia is beyond the scope of this review.

Hypothermia is the result of net heat loss from the body. Heat can be lost or gained by conduction, convection, and radiation and lost through evaporation. Conduction is direct transfer of heat from warmer to cooler objects that are in contact. Convection is transfer of heat to or from a gas or a liquid that is in motion. Radiation is transfer of heat in the form of electromagnetic energy between 2 objects that are visible to each other. Evaporation is loss of heat by vaporizing liquid (usually water from sweat or external sources) from the skin, clothing that is in contact with the skin, or respiration.

The human body attempts to maintain a core temperature at or near 37°C. The thermoregulatory control center in the hypothalamus receives input from central and peripheral thermal receptors. The integrated thermal signal triggers autonomic reflexes that control the initiation of cooling responses such as vasodilation or sweating (heat loss) or warming responses such as vasoconstriction (heat retention) or shivering (heat production).[1] Peripheral blood flow is also partly regulated by local skin temperature.

Humans originated in the tropics and have limited physiologic means to avoid developing hypothermia. Exercise and shivering can raise the metabolic rate to prevent hypothermia if nutritional reserves and insulation are adequate, but the benefit may be limited by environmental conditions. Prevention of hypothermia in humans mostly depends on behavior, chiefly wearing insulating clothing and using shelter.

Methods

The Wilderness Medical Society (WMS) convened an expert panel to develop evidence-based clinical guidelines for prevention and out-of-hospital diagnosis and treatment of victims of accidental hypothermia to update the previous WMS Practice Guidelines for the Out-of-Hospital Evaluation and Treatment of Accidental Hypothermia: 2014 Update.[2] Panelists were selected by the WMS based on clinical and/or research experience and generated a set of questions (Figure 1) to define the most significant areas of interest. As part of the update process, the current panel identified additional questions not previously considered. A literature search identified relevant articles with a key word search of the MEDLINE database. Keywords were hypothermia, accidental hypothermia, wilderness hypothermia, shivering, rewarming, core temperature, and resuscitation. The panel considered only peer-reviewed randomized controlled

trials, observational studies, case series, and case reports related to evaluation and treatment of accidental hypothermia. Although all articles were considered, those published between 2013 and March 2019 were the focus of this review.

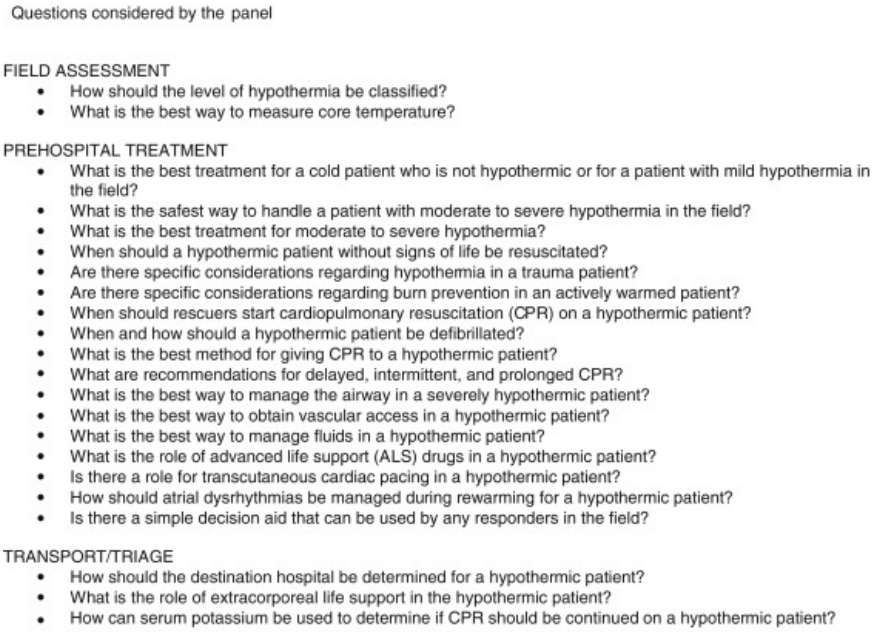

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Figure 1. Questions considered by the authors for the development of these practice guidelines.

The panel assessed the level of evidence supporting each diagnostic and therapeutic modality. Conclusions from review articles were not used in the formulation of recommendations, but the guidelines cite review articles when necessary to provide background information.

The panel used a consensus approach to develop recommendations regarding each evaluation technique and intervention and its role in management. The panel graded each recommendation based on the quality of supporting evidence and balance between the benefits and risks/burdens, according to the criteria of the American College of Chest Physicians (see online Supplementary Table).[3]

PATHOPHYSIOLOGY OF HYPOTHERMIA

The primary physiologic effects of tissue cooling are decreased resting metabolism and inhibition of central and peripheral neurologic function. During the initial stages of cooling of a neurologically intact victim, secondary responses to skin cooling predominate.[1] Shivering thermogenesis, triggered by skin cooling, results in increased metabolism due to the work of shivering and increased ventilation, cardiac output, and mean arterial pressure.[4] These physiologic parameters initially increase as core temperature decreases to approximately 32°C. The parameters then decrease with a further drop in core temperature.[1] Shivering ceases at and below a core temperature of approximately 30°C.[5] Once this occurs, metabolism decreases with further decreases in core temperature.

Clinical manifestations of accidental hypothermia relate predominantly to cerebral and cardiorespiratory effects. Brain activity begins to decline at a core temperature of approximately 33 to 34°C and continues to decline with further cooling.[6,7] Cooling of the brain leads to irritability, confusion, apathy, poor decision making, lethargy, somnolence, and eventually coma. Brain cooling decreases cerebral oxygen requirements.[8] This provides temporary protection during anoxic conditions such as cold-induced cardiac standstill and cold-water drowning. Cold stress reduces circulating blood volume due a combination of cold-induced diuresis, extravascular plasma shift, and inadequate fluid intake.[9] As the heart cools below 30°C, cardiac output decreases markedly, and bradycardia usually occurs. Abnormalities in electrical conduction lead to dysrhythmias such as premature atrial and ventricular contractions, atrial fibrillation, and ventricular fibrillation (VF).[10] Below 28°C, the heart is susceptible to VF, which can be triggered by acidosis, hypocarbia, hypoxia, or movement.[1] Decreased ventilatory response to carbon dioxide leads to hypoventilation and respiratory acidosis.[11]

FIELD ASSESSMENT

Classification of hypothermia

Most guidelines use a standard classification of hypothermia based on core temperature. Hypothermia is classified as mild at 35 to 32°C; moderate at 32 to 28°C; or severe at <28°C.[12, 13, 14] Some experts advocate a further category, profound hypothermia, at <24°C[12] or <20°C.[1] The chance of survival appears to be much lower in this range, probably because of a high likelihood of cardiac arrest. In cases of hypothermia secondary to cold water immersion, loss of airway protection and drowning may also contribute to causes of death. Although core temperature is used to classify hypothermia, individual variation to core temperature is wide, as is true of other physiologic parameters. Measuring core temperature is not always feasible in the out-of-hospital environment.[15]

Factors to guide treatment

The standard classification of hypothermia by core temperature correlates with the status of the thermoregulatory system. From 35 to 32°C (mild hypothermia) thermoregulatory shivering control is functional and increases as core temperature decreases.[16] With further cooling, shivering generally becomes less effective, although it can still be strong at 31°C.[5] Below 32°C (moderate hypothermia), thermoregulation becomes less effective and rewarming is possible only with addition of exogenous heat. As the core temperature decreases below 32°C, level of consciousness decreases. Below 28°C (profound/severe hypothermia), most patients are unconscious and not shivering, and the risk of VF or asystole is high.[17]

Recommendation

The key factors guiding hypothermia treatment are level of consciousness, alertness, shivering intensity, physical performance, and cardiovascular stability, which is based on blood pressure and cardiac rhythm (Figure 2). Core

temperature can provide additional helpful information, but it is difficult to accurately obtain in the field, and the panel recommends that this should not be the sole basis for treatment (**Evidence grade:** 1C).

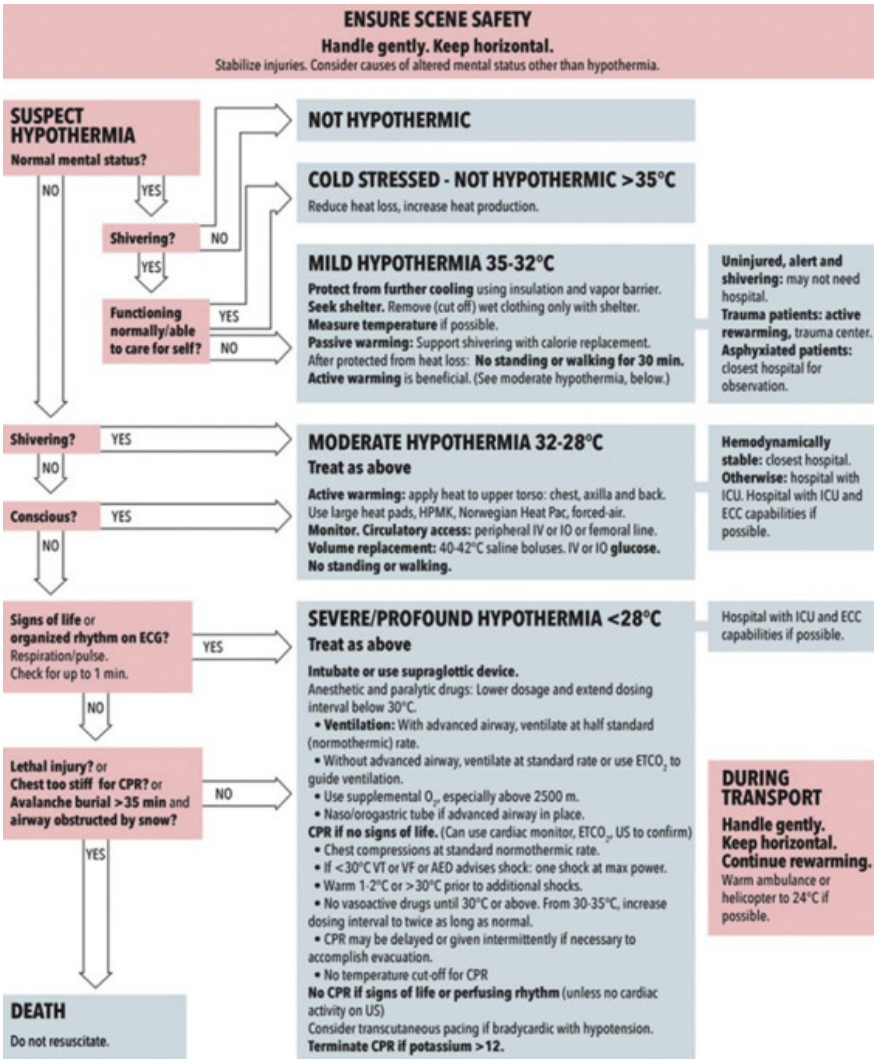

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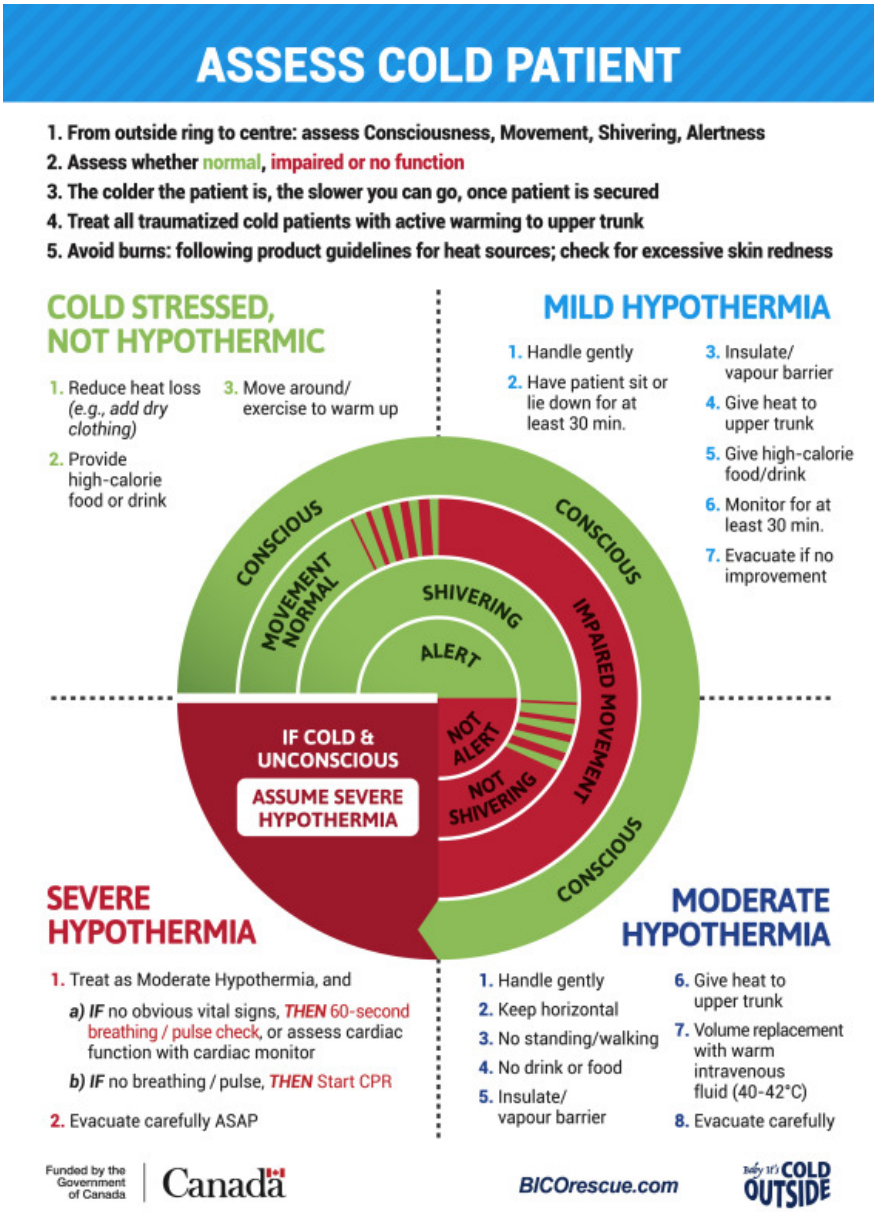
Figure 2. Recommendations for out-of-hospital evaluation and treatment of accidental hypothermia. *Abbreviations:* AED = automatic external defibrillator, CPR = cardiopulmonary resuscitation, ECC = extracorporeal circulation, ECG = electrocardiogram, $ETCO_2$ = end-tidal carbon dioxide, HPMK = Hypothermia Prevention Management Kit, ICU = intensive care unit, IV = intravenous, IO = intraosseous, O_2 = oxygen, PEA = pulseless electrical activity, US = ultrasound, VT = ventricular tachycardia, VF = ventricular fibrillation. From Zafren et al.[2] Reprinted with permission from the Wilderness Medical Society. ©2014 Wilderness Medical Society.

Simplified decision aid for field use

We have developed a simplified “Cold Card”[18] (Figure 3) corresponding to the more technical flowchart in Figure 2.



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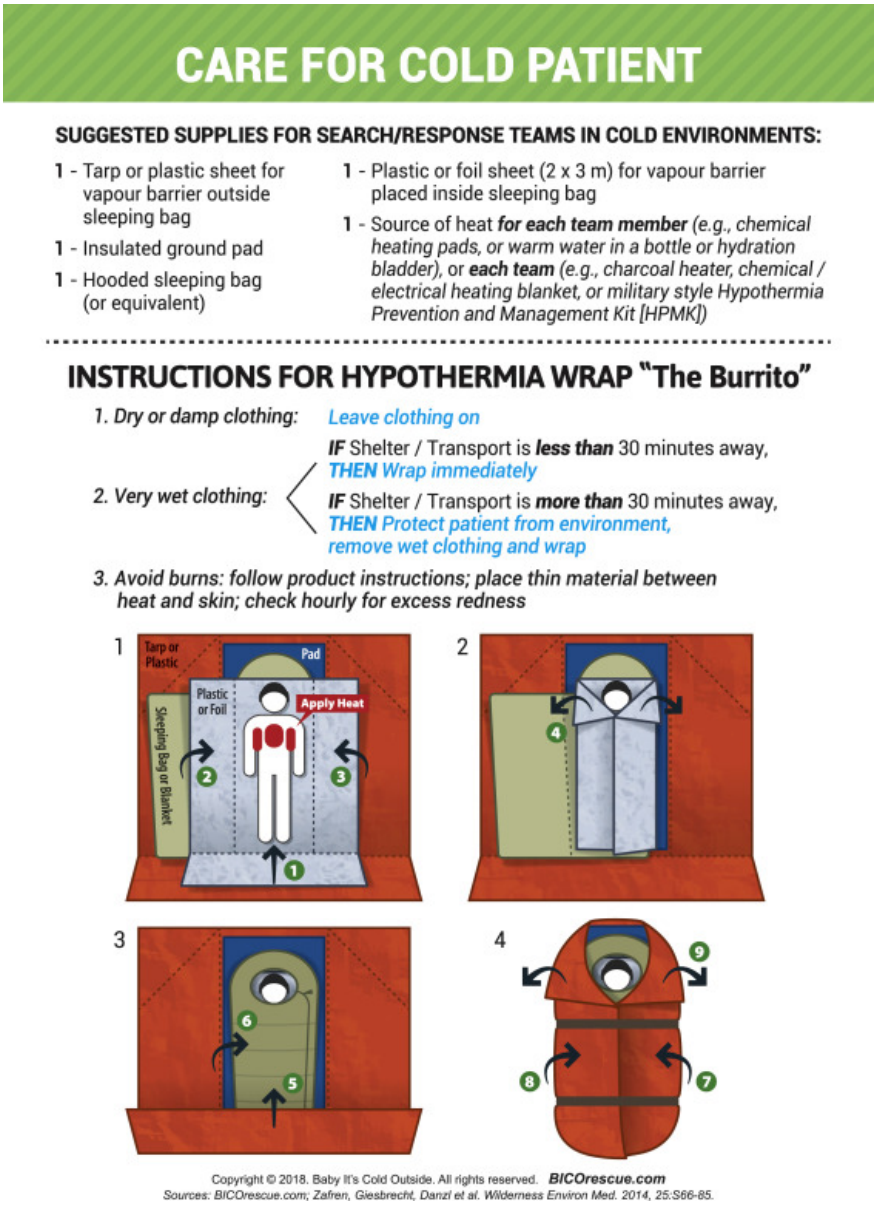

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Figure 3. (A) Front of cold card — “assess cold patient”. (B) Back of cold card — “care for cold patient.” From Giesbrecht.[18] Reprinted with permission from the Wilderness Medical Society. ©2018 Wilderness Medical Society

Recommendation

It is the recommendation of the working group that this decision aid be considered to facilitate evaluation and treatment of accidental hypothermia in the out-of-hospital setting for responders with varying levels of medical training.

Some patients are cold, but not hypothermic

Patients can be cold and shivering, but not hypothermic. Shivering is triggered by skin cooling as a mechanism for preventing hypothermia. A shivering patient with a core temperature >35°C is cold-stressed, but not hypothermic. If temperature

measurement is not possible, clinical judgment may be helpful to distinguish whether a patient is hypothermic or cold-stressed. For example, a patient who was not cold before being briefly immersed in cold water may be shivering but will not be hypothermic (Figure 2, Figure 3). Many alert, shivering patients who are well nourished and exhausted are not hypothermic.

Recommendation

It is the recommendation of the panel that a patient who is shivering but able to function well and care for him- or herself be closely observed because this patient is unlikely to be hypothermic. A patient who is shivering, becoming incapacitated, and having difficulty caring for him- or herself is likely to be hypothermic. If there is any doubt, assume that the patient is hypothermic and treat accordingly.

Alternate classification of hypothermia

The American Heart Association (AHA) 2010 Guidelines propose an alternate classification of hypothermia: mild (>34°C); moderate (34 to 30°C); and severe (<30°C).[19] Defibrillation is less likely to be successful at temperature below 30°C than above 30°C.

Recommendation

The panel recommends that the AHA scheme should not be used as the standard classification for out-of-hospital treatment of hypothermia because it changes the widely accepted definition of hypothermia and emphasizes response to defibrillation rather than physiologic changes.

Field classification of hypothermia: the “Swiss” system

The “Swiss” hypothermia classification was developed to help rescuers estimate core temperature by observing clinical signs.[12] Because individuals have variability in response to cold, estimating core temperature on the basis of clinical signs is only an approximation. The stages of the “Swiss” hypothermia (abbreviated “HT”) grading system with descriptions and estimated core temperature are as follows:

- HT I—clear consciousness with shivering: 35 to 32°C
- HT II—impaired consciousness without shivering: 32 to 28°C
- HT III—unconscious: 28 to 24°C
- HT IV—apparent death: 24 to 13.7°C
- HT V—death due to irreversible hypothermia: <13.7°C? (<9°C?)[12]

A limitation of this system is that individuals vary in physiologic response to hypothermia. Shivering may be maximal at 32 to 33°C but may continue at 31°C and may not cease until core temperature drops to approximately 30°C. A shivering patient with impaired consciousness should be treated for moderate, not mild, hypothermia. Temperature ranges for hypothermia stages should not be considered absolute but rather correlated with clinical observations. An analysis of reported cases of hypothermia revealed clinically significant temperature overlap with respect to hypothermia staging. The lowest recorded temperature was 28.1°C for HT I, 22°C for HT II, and 19.3°C for HT III.[20] It is advised that rescuers focus on the entire clinical picture rather than just shivering. Many case reports describe hypothermic patients with vital signs who had core temperatures below 24°C.[21, 22, 23, 24, 25] Individuals with core temperatures below 24°C are very susceptible to VF. Although the Swiss HT system can help guide rescuers in certain situations, we prefer to use the terms mild, moderate, severe, and profound hypothermia.

Recommendation

Rescuers should classify hypothermia as mild, moderate, severe, and profound on the basis of clinical observations, remembering that shivering can occur below 32°C, usually with altered mental status, and that patients can have detectable vital signs with core temperatures below 24°C. Furthermore, rescuers should be aware of core temperature overlap between classification categories[20,25] (**Evidence grade:** 1C).

Associated conditions complicating the field classification of hypothermia

In addition to hypothermia, many conditions can cause altered mental status and decreased level of consciousness. Conditions such as sepsis and severe trauma can decrease physiologic reserves and may decrease or abolish shivering.[26] Many drugs and medications[27] suppress shivering.[28]

Recommendation

Clinicians should consider causes other than hypothermia to explain altered mental status or lack of shivering that do not correlate with the measured core temperature or are associated with a history of minimal cold exposure (**Evidence grade:** 1B).

MEASUREMENT OF CORE TEMPERATURE

Esophageal temperature

The most accurate minimally invasive method of measuring core temperature is esophageal temperature, with the probe inserted into the lower third of the esophagus.[29] The degree of accuracy afforded by esophageal temperature monitoring is helpful to guide treatment of patients with moderate or severe hypothermia. Placement of an esophageal probe via the pharynx may cause vomiting and aspiration. The airway must be protected with an endotracheal tube

or supraglottic device that allows passage of a gastric tube before placement of an esophageal probe. Heated humidified oxygen does not significantly raise the temperature measured by a properly inserted esophageal probe.[30, 31, 32] If the esophageal probe is not inserted into the lower third of the esophagus, an average of 24 cm below the larynx in adults,[1] use of heated humidified oxygen may result in a falsely elevated esophageal temperature. Esophageal probes that do not have markings can be measured visually against the patient and marked to ensure the correct depth of insertion. Field conditions will rarely allow for placement of an esophageal probe; however, transporting air or ground medical services that have this capacity should place a probe as soon as possible.

Recommendation

If available, an esophageal temperature probe should be placed in a patient whose airway has been protected and secured. Esophageal temperature is the preferred method of core temperature measurement (**Evidence grade:** 1C).

Epitympanic temperature

Epitympanic (ear canal) temperature, measured using a soft probe with a thermistor in proximity to the tympanic membrane, reflects carotid artery temperature.[33] Epitympanic thermometers should not be confused with the more common and much less accurate infrared “tympanic” thermometers. In patients with adequate cardiac output, epitympanic temperature reflects core temperature. Epitympanic temperature can be lower than esophageal temperature during low flow (decreased cardiac output) or no-flow (cardiac arrest) states.[28] In out-of-hospital settings care must be taken to insulate the ear canal from the environment. With cold ambient temperature, epitympanic temperature may be falsely low, especially if the external auditory canal is blocked by cerumen, filled with snow, or not adequately sealed and properly covered with an isolating “cap.”[33] Epitympanic temperature is much lower than esophageal temperature during initial cooling of the head in normothermic subjects. After 10 min of isolated head cooling, the mean difference between epitympanic and esophageal temperatures is about 1 to 2°C.[34] Epitympanic thermometers for use in the operating room are not suitable for field use; they are not designed for use in cold environments.

Recommendation

Use an epitympanic thermometer designed for field conditions with an isolating ear cap in a patient whose airway has not been secured by endotracheal intubation or a supraglottic airway, or in a patient with a secured airway if an esophageal probe is not available (**Evidence grade:** 1C). Infrared tympanic thermometers should never be used to measure core temperature in a hypothermic patient[15] (**Evidence grade:** 1A).

Rectal temperature in the field

The use of rectal thermometers is not advisable before the patient has been removed from the cold environment because the patient must be further exposed, increasing heat loss and potentially worsening hypothermia.

Recommendation

Rectal temperature measurement should not be used unless the patient is in a warm environment (**Evidence grade:** 1C).

Rectal and bladder temperatures during rewarming

Rectal and bladder temperatures lag behind core temperature changes by as much as an hour and can give the false impression that the patient is still cooling.[4,9,35] Rectal and bladder temperature overestimate heart temperature during cooling and underestimate heart temperature during rewarming.

Recommendation

Monitor rectal or bladder temperature during rewarming of an unconscious patient only if an esophageal or epitympanic probe is not available. If rectal or bladder temperature is used for monitoring during rewarming, allow for inaccuracy due to the time lag behind core temperature changes (**Evidence grade:** 1A).

Oral temperature

Oral temperatures are useful only to rule out hypothermia. Nonelectronic thermometers are typically unable to measure temperatures below 35.6°C. If mercury or alcohol thermometers are used to diagnose hypothermia, they must be special “low reading” thermometers.[1]

Recommendation

Oral temperature measurement with a thermometer that cannot read below 35°C should not be used to diagnose hypothermia (**Evidence grade:** 1A).

“Temporal artery” thermometer

“Temporal artery” thermometers, used on the skin surface, do not provide accurate temperature measurements in hypothermia.[36]

Recommendation

Do not use a temporal artery thermometer in a possibly hypothermic patient (**Evidence grade:** 1C).

Zero-heat flux thermometer

A noninvasive heat flux or “double sensor” thermometer is currently under development.[37] This technology, which combines a skin temperature sensor with a heat flux sensor, correlates well with esophageal temperature in operative and intensive care unit settings.[38,39]

Recommendation

Because this technology has not been validated in field settings, no recommendation can be made at this time.

OUT-OF-HOSPITAL TREATMENT

Safety of the rescuers

Rescuer safety is the first priority during rescue. The scene may be unsafe to enter, or the safety officer in a rescue may allow only brief entry. Unless there are obvious fatal injuries, rescuers might need to move the patient to a safe place before deciding to resuscitate.

Recommendation

The decision to rescue or resuscitate a patient can only be made after the scene is secure and safe for the rescuers to enter and make an evaluation (**Evidence grade:** 1A). After rescuer safety has been assured, the priorities for out-of-hospital treatment of a hypothermic patient who is not in cardiac arrest are to avoid causing cardiovascular collapse during rescue, prevent further decrease in core temperature (afterdrop), and rewarm the patient in a safe manner. If a hypothermic patient is in cardiac arrest, rescuers should, if indicated, initiate resuscitation.

Core temperature afterdrop

Core temperature afterdrop refers to continued core cooling after removal from a cold environment. Afterdrop is caused by a combination of conductive heat loss from the warmer core to cooler peripheral tissue and convective heat loss from blood due to increased flow to cooler tissue and subsequent return to the central circulation and heart.[40, 41, 42] The convective component transfers more heat, and, unlike the conductive component, is affected by the method of rewarming. In a hypothermia victim, peripheral tissue is colder than the heart. Therefore, any action that increases blood flow to cold peripheral tissue (eg, hoisting or holding victim in a vertical position, allowing the victim to stand or walk, active or passive limb movement, immersion in warm water) will increase the volume of cold blood returning to the heart. This increases cardiac work and further decreases core temperature.

Afterdrop may be clinically important in victims who are at the threshold of moderate to severe hypothermia because they are susceptible to cardiovascular instability with a small further drop in core (heart) temperature. Afterdrop of as much as 5 to 6°C has been reported in hypothermic patients.[21,42, 43, 44] Therefore, care should be taken to prevent increased blood flow to the limbs during and after rescue.

Circumrescue collapse

Circumrescue collapse refers to light-headedness, collapse, syncope, or sudden death occurring in victims of cold-water immersion just before, during, or after rescue and removal from water.[45] Circumrescue collapse can be caused by mental relaxation and decreased catecholamine output causing life-threatening hypotension or by sudden onset of cardiac dysrhythmia, likely VF.[4]

The act of removing a victim from water decreases hydrostatic pressure,[4] which is normally greatest around the legs. Removing hydrostatic pressure allows blood to pool in dependent areas, causing decreased blood return with resultant hypotension or cardiovascular collapse. A cold heart may not be able to compensate for decreasing blood pressure by increasing cardiac output. Blood that does return from dependent areas will be cooled and will contribute to core temperature afterdrop. Afterdrop is increased if the victim has to perform work to assist in rescue (eg, having to climb a ladder onto a boat).[45,46] Mechanical stimulation of the heart during rescue and extrication—combined with hypotension, afterdrop, and acidosis—may precipitate fatal dysrhythmias.[47]

When rescue is imminent, mental relaxation in conscious patients may be associated with decreased catecholamine release, causing decreased blood pressure with loss of consciousness and subsequent drowning.[47] Circumrescue collapse has also been described in terrestrial rescue situations.[23,48]

Handling of a hypothermic patient during rescue

Keeping the patient in a horizontal position mitigates the effects of decreased hydrostatic pressure during rescue.[45] Avoiding physical effort protects against afterdrop.[46] Continued mental stimulus may help maintain catecholamine stimulus.

Recommendation

Rescuers should keep a hypothermic patient horizontal, especially during rescue from water or a crevasse (**Evidence grade:** 1B) and should limit physical effort by the patient during rescue (**Evidence grade:** 1B). A conscious patient should be encouraged to be attentive and focus on survival (**Evidence grade:** 1C).

Gentle handling to prevent ventricular fibrillation

Hypothermia lowers the threshold for VF, especially at core temperatures below 28°C.[1] Movement or significant warming of the extremities, as with warm water immersion, increases blood flow to colder tissues. Blood flows to the periphery, is cooled, and has the potential to cool the heart upon return to the core, increasing the risk of VF.[17,49,50] Additional blood return may also cause increased load on a heart that is already pumping ineffectively.

Recommendation

Handle a hypothermic patient gently and continue to keep the patient horizontal (**Evidence grade:** 1B). Avoid any disturbance, especially movement of the extremities that might precipitate VF (**Evidence grade:** 1B). In an effort to

minimize movement, clothes should be cut off of a patient once in a warm environment (**Evidence grade:** 1B).

Protection from further heat loss

After rescue, the next priority for care of a hypothermic patient in the out-of-hospital setting is to maintain core temperature by preventing further heat loss.

Insulation protects from heat loss. Insulating materials include extra clothing, blankets, quilts, sleeping bags, insulated pads, and bubble wrap.[51,52] A sleeping bag should not be used like a blanket; rather the patient should be placed inside, and the enclosure should be completely zipped up. Multiple sleeping bags, if available, can be placed within each other to create a multilayered enclosure. Any available insulation (eg, spare parkas) should be incorporated within or outside the enclosure in such a way as to not compromise loft. Bubble wrap is an effective vapor barrier, but it provides less insulation than the other materials.[51,52] A large amount of heat can be lost to the ground by conduction.[53] Significant heat can also be lost from the head and neck due to necessary exposure of the face to allow breathing.[53,54]

Vapor barriers protect against convective and evaporative cooling (substantially reducing heat loss) and importantly also keep the insulation dry and more effective. Barriers can be made from bubble wrap, tarps, sheets of plastic, reflective blankets, or garbage bags with a hole cut out for the face. The vapor barrier(s) may be placed inside the insulation (to keep the insulation dry if the patient is packaged wet) and/or outside the insulation to protect the insulation from a wet environment.[52,55]

Extra insulation can compensate for the absence of a windproof layer or vapor barrier.[55] A combined method using 2 vapor barriers (1 against the patient and 1 outside the insulating layers) will protect the insulation from becoming wet from all sources.[52]

Recommendations

Protect from further cooling by using insulation and vapor barriers until the patient has reached a warm environment, such as the warmed interior of an ambulance. Remove wet clothes, preferably by cutting them off, only when the patient has been protected from the cold (**Evidence grade:** 1C). Insulate the patient from the ground (eg, with sleeping pads) to protect from conductive heat loss. Minimize heat loss from the head and neck by covering these areas as effectively as possible (eg, toque, watch cap, hood, jacket) (**Evidence grade:** 1C).

Protection from windy conditions

In windy conditions, a windproof layer, ideally a vapor barrier, provides substantial protection from convective heat loss.[51]

Recommendation

An outer windproof layer should be used to protect the patient from wind and especially from rotor wash when loading or unloading from a helicopter. If possible, add a second vapor barrier against the victim to protect the insulating layers (**Evidence grade:** 1C).

FIELD REWARMING

Once a hypothermic patient has been protected from further heat loss, the next priority is to rewarm the patient. The rewarming methods described in this section achieve the safe rewarming rate of 1 to 2°C·h^{-1} and minimize the risk of afterdrop. Afterdrop can lead to hemodynamic instability and VF. The risk of afterdrop is reduced by limiting limb movement and by keeping the patient horizontal. Most patients with altered consciousness will require active rewarming.

It is important to recognize that the optimal rate of rewarming may not be the fastest rate. Even profound hypothermia may require slow, controlled rewarming. Only patients with hemodynamic instability should be considered for rapid rewarming via extracorporeal life support (ECLS).

Shivering

Vigorous shivering can increase heat production by 5 to 6 times the resting metabolic rate and up to 50% of maximal metabolic rate ($\dot{V}O_{2max}$).[5,56] Shivering can raise core temperature by 3 to 4°C·h^{-1},[16,28,57] but it uses a large amount of energy, stresses the cardiovascular system, and causes patient discomfort.[17]

Recommendation

Shivering is an effective method of rewarming in a patient who is cold-stressed or mildly hypothermic. The patient must be adequately insulated from the environment to retain the generated heat (**Evidence grade:** 1A). An alert patient who is shivering, and who is not at risk for aspiration, should receive high-carbohydrate liquids and/or food. Liquids and food may be warmed but should not be hot enough to burn the esophagus (**Evidence grade:** 1C).

Delay exercise to protect against afterdrop

Once a patient is protected from further heat loss and has adequate energy reserves, the most effective means of rescue may be for the patient to walk. Allowing the patient to shiver and rewarm, while insulated and before exercise, should help minimize afterdrop.[46] This time period should last 30 min but will depend upon the situation. Standing upright increases blood flow to and from the legs, worsening afterdrop and potentially decreasing blood pressure.[29] Walking or other exercise generates additional heat, but if initiated immediately after rescue, may cause a greater afterdrop in core temperature than if the patient remains at rest.[46] There may be situations when immediate movement is necessary to relocate a patient to a safer environment. When this is necessary, close monitoring is prudent.

Recommendation

A shivering patient who may be hypothermic should be kept as warm as possible, given calorie replacement, and observed before exercising. After this period of observation, the alert patient may be allowed to stand. If the patient can stand without difficulty, exercise intensity should start low and increase gradually as tolerated. The patient should be closely monitored; if the condition worsens, the patient should stop exercising and be treated accordingly (**Evidence grade:** 1C).

Active external rewarming

Field methods of external rewarming are useful in both shivering and nonshivering patients. Active (exogenous) rewarming methods, such as large electric heat pads or blankets,[58] large chemical heat pads,[59,60] warm water bottles,[61] and the Norwegian charcoal-burning Heat Pac,[17,58,62] all provide significant external heat. In a shivering patient, the added heat attenuates shivering heat production. This results in a rate of core rewarming similar to that produced by shivering but has the advantages of increased comfort and decreased energy use with lower cardiac workload. In a nonshivering patient, added heat will warm the core, even if slowly, in a patient lacking capacity for endogenous heat production. The Heat Pac should be used with caution because it can generate potentially toxic levels of carbon monoxide (CO).[32]

To maximize total body net heat gain, active heating will be more effective in conjunction with insulation and vapor barrier(s) to create an effective hypothermia enclosure system. Five such systems were compared on normothermic subjects in -20°C conditions.[63] The systems (all including active heating and vapor barrier) included 3 commercial systems using heavy insulation enclosures, a user-assembled system using a 3-season sleeping bag, and the Hypothermia Prevention Management Kit (HPMK).[63] Initially evaluated for effectiveness between other rewarming systems,[64] the HPMK was commercially developed for the Department of Defense-Joint Trauma System (JTS) as a lightweight, compact kit designed for field use that combines an oxygen-activated self-heating liner with a vapor barrier. The HPMK was part of the JTS theater-wide strategy for battlefield casualties and has been used extensively in military operations to decrease mortality from trauma-induced hypothermia.[65, 66, 67] System effectiveness (net body heat gain) generally depended on the mass of the insulation enclosure.[63] The 3 commercial systems were heavy and bulky and therefore only applicable at a point of care or if they could be delivered by sled or vehicle. The user-assembled and HPMK systems could both be transported by backpack; however, the HPMK was smaller and lighter and therefore more portable, but at the expense of providing less net body heat gain. Therefore, factors such as mass, volume, effectiveness, and cost will affect the type of system used in the field. Search and rescue teams, which usually consist of 2 to 4 persons, could realistically carry a more-effective user-assembled system because the items can be separated and dispersed to multiple backpacks (Figure 3). Alternatively, the HPMK should be complemented by incorporating a sleeping bag (or blankets) over the heated liner and inside the vapor barrier.

Recommendations

Active sources of heat should be used (**Evidence grade:** 1B). Rewarming devices should be used in conjunction with vapor barriers and insulation (**Evidence grade:** 1B). The Heat Pac should only be used outdoors or with proper ventilation that is carefully monitored to prevent CO accumulation (**Evidence grade:** 1B). The single-package, small, and light HPMK is a practical system for transport in a single backpack and useful for military operations and should be used with an added layer of insulation if possible (**Evidence grade:** 1C).

Body-to-body rewarming

Body-to-body rewarming of a shivering patient with a warm person in a sleeping bag blunts the increase in shivering thermogenesis, resulting in rewarming rates no greater than shivering alone.[57,58] Body-to-body rewarming may make the cold patient more comfortable due to decreased shivering, but at the cost of delaying evacuation.

Recommendation

Body-to-body rewarming can be used in mild hypothermia to increase patient thermal comfort if enough personnel are available and it does not delay evacuation to definitive care (**Evidence grade:** 1B).

Applying heat to the axillae, chest, and back

External heat is most effective if concentrated on the axillae, chest, and back (in that order), which are the areas with the highest potential for conductive heat transfer.[53] Upper torso rewarming is more effective than extremity rewarming.[68] Some scenarios may. however, preclude applying heat to the chest (eg, cardiopulmonary resuscitation [CPR] in progress or treatment of chest injury). Applying heat to the head, although requiring more technique to insulate and apply the heat, has been shown to be equally effective in shivering and nonshivering subjects, thus providing an alternative warming approach in extenuating circumstances.[69]

Recommendations

Apply heat sources to the axillae, chest, and back. A large heat pad or blanket should be placed over the chest and, if large enough, extend into the axillae and under the back (**Evidence grade:** 1B). Additional heat can be applied to the neck if precautions are taken to prevent heat loss through any neck opening (**Evidence grade:** 1C). Avoid applying external heat to the extremities, although it is not necessary to insulate the arms from heat applied to the torso (**Evidence grade:** 1B). If application of heat to the chest is contraindicated (eg, CPR or some chest injuries), heat sources may be still be applied under the patient's upper back or to the head (**Evidence grade:** 1B). If applying heat sources to the back, rescuers must be able to observe for the development of burns on a regular basis (**Evidence grade:** 1C).

Protection of cold skin

Cold skin is very susceptible to injury from pressure or heat.[70] There have been reports of burns associated with use of a hot water bottle with lukewarm water applied directly to hypothermic skin,[71] the HPMK,[64] water-perfused warming blankets, a Heat Pac, and hot pads.[72] Burns have been reported both in controlled settings while researching rewarming methods and during rescue of hypothermic patients. It is important to visually inspect the heated skin at regular intervals (eg, 20 to 30 min) to observe for excess reddening or other signs of pending burns; in these cases, active heating should be stopped in the affected areas. This must be done segmentally and carefully to minimize heat loss. Heated pads should be applied with great caution to areas such as the back that are difficult to visualize or under constant pressure from body weight or immobilization systems.

Recommendation

Avoid localized pressure to cold skin. Apply heat sources according to manufacturer instructions; this often precludes direct contact with the skin by placing some thin insulating material between the skin and heat source to prevent burning the skin (**Evidence grade:** 1C). Skin should be assessed every 20 to 30 min for excess reddening or other signs of impending thermal burns when active heat sources are being applied (**Evidence grade:** 1C).

Do not use small chemical heat packs for rewarming

Small chemical heat packs (eg, those used for hand and foot warming) do not provide sufficient heat to affect core temperature. In addition, the high surface temperature of small chemical heat packs creates a risk of thermal burns.

Recommendation

Do not use small chemical heat packs for core rewarming of a hypothermic patient (**Evidence grade:** 1B). However, these small chemical heat packs can be used to prevent local cold injury to the hands and feet during treatment and transport (**Evidence grade:** 1C).

Heated humidified oxygen

Although heated humidified oxygen prevents respiratory heat loss, the respiratory tract allows limited heat exchange. Heated humidified oxygen is not effective as a solitary rewarming method,[30, 31, 32] but it can be used as an adjunct to other methods.[26] Heated humidified oxygen has the potential to cause facial burns.[32]

Recommendations

Heated humidified oxygen can be used in combination with other rewarming methods (**Evidence grade:** 2C), but it should not be relied on as the only rewarming method (**Evidence grade:** 1B).

Do not use warm showers or baths for rewarming

A warm shower or bath markedly increases peripheral blood flow, increasing afterdrop and potentially causing hypotension.[29,42] Using a warm shower or bath, even in a patient who is mildly hypothermic, may cause cardiovascular collapse. This method of warming may be considered for patients who are cold-stressed or after an initial period of rewarming for those with mild hypothermia.

Recommendation

A warm shower or bath should not be used for initial rewarming, even if a patient appears to be only mildly hypothermic (**Evidence grade:** 1C).

Distal limb warming

Distal limb warming in 42 to 45°C water to the elbows and knees is effective for warming alert, mildly hypothermic patients.[9] This method works by opening arteriovenous anastomoses in the hands and feet, causing increased return flow of warmed blood directly from the arms and legs to the core. This is an exception to the general rule that peripheral rewarming is contraindicated in hypothermic patients. Because the warmed superficial venous blood bypasses the cold arteries in the extremities, there is little countercurrent heat exchange. In the one laboratory study that used this method, the afterdrop was less than the afterdrop for shivering.[9] Distal limb rewarming in water was designed for use on watercraft and is difficult to apply for other out-of-hospital transport.

Recommendation

Distal limb warming to the elbows and knees in 42 to 45°C water can be used for rewarming a patient with mild hypothermia if the clinical setting is appropriate (**Evidence grade:** 1C).

Rewarming during transport

Continued rewarming is challenging during transport. A randomized, controlled study of care in helicopter and ground advanced life support units showed a small increase in core temperature with using large chemical heat pads but decreased core temperature with passive rewarming, reflective blankets, warm IV fluids, and warm IV fluids plus reflective blankets.[60]

Forced air warming, usually with an air-filled plastic baffled blanket with continuous heated airflow through perforations in the bottom of the blanket, is an effective way to rewarm a hypothermic patient.[30,73,74] In 1 study, afterdrop with forced air warming was less than with shivering.[75] Forced air warming is more effective and more practical than a liquid-filled heating blanket.

Recommendations

Forced air warming should be used during air or ground transport, if available (**Evidence grade:** 1A). If forced air warming is not available, use of other heat sources can be continued. Care must be taken to prevent CO buildup with the charcoal Heat Pac in a ground ambulance; this can be done by igniting the device

outside the vehicle, bringing it inside only after initial smoke production subsides, ventilating the vehicle compartment, and monitoring CO (**Evidence grade:** 1C). A charcoal Heat Pac should not be used in an aircraft (**Evidence grade:** 1C).

Temperature in air or ground ambulances

The temperature in patient compartments should ideally be 28°C, which is the temperature at which unclothed resting normothermic humans will neither gain nor lose heat.[1] Warming the patient compartment will protect patients from further heat loss when exposed for monitoring or other procedures. However, an air temperature of 28°C is usually uncomfortably hot for pilots, drivers, and medical providers. A slightly cooler temperature of 24°C will limit heat loss and is better tolerated by ambulance personnel.

Recommendation

Patient compartments in ground and air ambulances should be heated to at least 24°C, if possible, to decrease further heat loss (**Evidence grade:** 1C).

Treatment of cold stressed patients who are not hypothermic

A cold patient who is alert and shivering but who has adequate energy reserves and is not hypothermic is at low risk for afterdrop or circumrescue collapse.

Recommendation

It is the consensus of the panel that a cold-stressed patient who is not hypothermic need not be kept horizontal. The patient may be allowed to remove his or her own wet clothing and to put on dry clothing without shelter, if necessary. The patient may be allowed to rest in a sitting position, to eat and drink to maintain energy reserves and hydration, and to move or keep moving, if necessary. These patients will need close monitoring to ensure they do not become hypothermic.

RESUSCITATION OF HYPOTHERMIC PATIENTS

Decision to resuscitate hypothermic patients without signs of life

Hypothermic patients have survived with normal neurologic function even after cardiac arrest.[23,76, 77, 78] Many of the usual indicators of death, such as fixed, dilated pupils and apparent rigor mortis, are unreliable in hypothermic patients.[76,77] Dependent lividity is an unpredictable indicator of death in hypothermia.

Recommendations

Fixed, dilated pupils, apparent rigor mortis, and dependent lividity are not considered contraindications to resuscitation of a severely hypothermic patient (**Evidence grade:** 1A for fixed, dilated pupils and apparent rigor mortis).

Rescuers should attempt CPR and resuscitation unless contraindications exist (**Evidence grade:** 1A).

Contraindications to resuscitation of hypothermic patients

The dictum that “no one is dead until they are warm and dead” is based on the difficulty of diagnosing death in a hypothermic patient in the field. However, some patients really are cold and dead. General contraindications to attempted resuscitation in the field include obvious fatal injuries, such as decapitation, open head injury with loss of brain matter, truncal transection, incineration, or a chest wall that is so stiff that compressions are not possible.[79]

Recommendation

Do not attempt to resuscitate a patient with obvious fatal injuries or whose chest wall is too stiff for compressions (**Evidence grade:** 1A).

Indication for cardiopulmonary resuscitation

CPR is only indicated in cardiac arrest and is contraindicated if there are signs of life. In a hypothermic patient in the out-of-hospital setting, signs of life may be very difficult to detect. The heart rate can be very slow and pulses difficult to palpate. The traditional method of checking a pulse by trying to feel the pulse with a finger placed over the presumed location of an artery is limited by cold. Cold fingers have decreased sensitivity to tactile stimuli. Breathing can be very slow and shallow but may be detectible in the absence of palpable pulses.[22] If cardiac monitoring is not available, the diagnosis of cardiac arrest can be difficult.

Recommendation

Rescuers should make every effort to move the patient to a warm setting, such as a ground or air ambulance or a medical facility where cardiac monitoring is available to guide resuscitation and to start rewarming (**Evidence grade:** 1C). Prior to starting CPR, feel for a carotid pulse for 1 min. If a pulse is not palpated after 1 min, start CPR, including rescue breathing (**Evidence grade:** 1C).

No cut-off temperature for resuscitation

The lowest known core temperature from which a patient with accidental hypothermia has been successfully resuscitated is 13.7°C.[80] The lowest core temperature ever induced therapeutically is 9°C.[81] Both patients survived neurologically intact. Induced hypothermia for cardiac or vascular surgery is usually to 18°C and—unlike accidental hypothermia—a very controlled situation. The lowest temperature from which humans with accidental hypothermia can be successfully resuscitated is not known, and reports of recovery from extremely low core temperatures make establishing a temperature cut-off for resuscitation challenging.

Recommendation

Resuscitation attempts should be continued regardless of the measured core temperature (**Evidence grade:** 2C).

Electrocardiographic monitoring

Electrocardiographic monitoring is the best way to diagnose cardiac arrest in the field. An organized rhythm without detectible pulses may be pulseless electrical activity or may be a perfusing rhythm with very weak pulses. In hypothermic patients, the amplitude of the QRS complexes may be decreased.[10]

Starting CPR in a hypothermic patient with an organized cardiac rhythm carries a risk of causing VF that would convert a perfusing rhythm to a nonperfusing rhythm. If end-tidal CO_2 ($ETCO_2$) monitoring is available, lack of waveform indicates lack of circulation or absence of metabolism.[13] If ultrasound is available, echocardiography can be used to determine if cardiac contractions correspond to electrical activity.[13]

Recommendation

CPR should be started if a nonperfusing rhythm, including ventricular tachycardia, VF, or asystole, is detected. If there is a cardiac rhythm with organized QRS complexes, CPR should not be performed (**Evidence grade:** 1C) unless $ETCO_2$ monitoring confirms lack of perfusion or echocardiography shows that there are no cardiac contractions corresponding to electrical activity (**Evidence grade:** 1B). Use maximal amplification on the monitor to search for QRS complexes (**Evidence grade:** 1C).

Delaying CPR, intermittent CPR, and prolonged CPR

Cooling reduces resting oxygen consumption of most human tissue by about 6% per 1°C decrease, with a greater decrease in brain tissue. Hypothermia preferentially protects the brain from hypoxia. At a core temperature of 28°C, whole body oxygen consumption is about 50% of normal,[1] while brain oxygen consumption may be reduced to about 35% of normal.[8] Surgical procedures employing deep hypothermic circulatory arrest (DHCA) have demonstrated a 7% decrease in cerebral oxygen consumption for every 1°C decrease in core temperature. Sixty percent of patients with a core temperature less than 18°C demonstrate an isoelectric electroencephalogram. DHCA is a controlled, rapid, decrease of core temperature from 18 to 20°C. Patients over the age of 60 y undergoing DHCA only tolerated an estimated 25 min of cardiac arrest, based upon incidence of postprocedural cognitive injury. Children tolerate longer periods of time, but there is a dearth of information for young and middle-aged adults.[82,83] There are many documented cases of full neurologic recovery, even after extended periods of cardiac arrest as long as 9 h[84,85] in persons who did not have asphyxia before they became hypothermic. Severely hypothermic patients have been resuscitated with good neurologic status after as long as 6 h 30 min of CPR.[23,86, 87, 88] Prolonged cardiac arrest in severely hypothermic patients does not necessarily cause brain injury as it does in normothermic patients.

In cardiac arrest, the classic teaching is that CPR must be started promptly and continued without interruption until return of spontaneous circulation (ROSC) can be established or death is confirmed. This CPR strategy may not be possible or warranted in patients with severe hypothermia. Multiple case reports describe survival with neurologic recovery when initiation of CPR was delayed and performed intermittently.[85,89] In 1 case report, a hypothermic avalanche victim was successfully resuscitated with complete neurologic recovery, although CPR was not started for 15 min after a monitored cardiac arrest.[23] In another case report, an avalanche victim was extricated apneic and pulseless after a 5 h burial in a crevasse. No attempt was made to resuscitate the patient, but the patient was flown to a nearby hospital where ECG showed asystole. CPR was started 70 min after rescue. The patient made a full neurologic recovery.[48] A third case report described successful resuscitation with good neurologic recovery of a hypothermic patient in cardiac arrest who was treated during evacuation with CPR in a stationary litter for 1-min periods alternating with 1-min periods of being carried without CPR.[86]

Continuous compressions are ideal, but intermittent compressions may be necessary to successfully and safely evacuate the patient. With properly performed compressions, it takes an estimated 5 min of cerebral oxygenation to overcome the ischemic threshold.[90]

Recommendation

Immediate, high-quality CPR should be performed for a hypothermic patient in cardiac arrest. If it is impossible or unsafe to perform immediate and continuous CPR, rescuers should perform delayed or intermittent CPR. Ideally, compressions will not be delayed for longer than 10 min, a conservative interval based on the uncontrolled nature of out-of-hospital hypothermic cardiac arrest (**Evidence grade:** 1C). If CPR cannot be performed continuously, compressions should be performed for a minimum of 5 min, with interruptions between periods of compressions that should not exceed 5 min (**Evidence grade:** 1C).

CPR technique in hypothermia

A hypothermic patient will have a stiff chest wall that limits the effectiveness of chest compressions and bag-valve-mask ventilation. Myocardial and pulmonary compliance are also markedly reduced in severe hypothermia. During hypothermic cardiac arrest with CPR[91] in a swine model, cardiac output, cerebral blood flow, and myocardial blood flow averaged 50, 55, and 31%, respectively, of those achieved during normothermic closed-chest compressions. However, metabolic demands are also decreased. Although there are no data to support increased survival when a mechanical compression device is used,[92] mechanical devices reduce the incidence of rescuer fatigue and may permit longer periods of uninterrupted compressions, especially when bridging to ECLS.[86,93]

Recommendation

Patients in cardiac arrest should have chest compressions delivered at the same rate as for normothermic patients (**Evidence grade:** 1C). Using a mechanical compression device may decrease interruptions and reduce rescuer fatigue (**Evidence grade:** 1C).

Automated external defibrillator

If an automated external defibrillator (AED) with a cardiac monitor is available, it can be used for cardiac monitoring. An AED without a cardiac monitor can also be used for diagnosis. The cardiac rhythms that may benefit from cardioversion or defibrillation (shockable rhythms) are VT and VF. VT is rare during moderate or severe hypothermia. The instruction “shock is advised” means that the rhythm is VT or VF. The instruction “no shock advised” on an AED without monitoring capability can mean that the rhythm is asystole or an organized rhythm, which may be pulseless electrical activity. Current AHA guidelines recommend a single shock.[19]

Recommendation

If shock is advised by the AED, rescuers should attempt defibrillation and start CPR. If no shock is advised on an AED, no carotid pulse is found after palpating for at least 1 min, normal breathing or other signs of life are not observed, and ultrasound is not available to verify cardiac activity or pulse, start CPR (**Evidence grade:** 1C).

Initial defibrillation in hypothermia

Defibrillation is only indicated for a shockable rhythm (pulseless VT or VF). An AED will only advise a shock if the rhythm is VT or VF. Current resuscitation guidelines recommend a single shock at maximum power for a patient whose core temperature is below 30°C.[13,17,94]

Recommendation

If a monitor/defibrillator shows VT or VF in a patient whose core temperature is thought to be below 30°C, a single shock should be given at maximum power (**Evidence grade:** 1C).

Repeat defibrillation attempts in hypothermia

Patients have been successfully defibrillated at core temperatures below 26°C.[95, 96, 97, 98] If defibrillation below 30°C is unsuccessful, delay further shocks until the temperature is greater than 30°C; below 30°C, defibrillation is less likely to be successful. Defibrillation in a patient whose core temperature has reached 30°C should follow guidelines for normothermic patients.[13]

Recommendations

Wait until a patient has been rewarmed to 30°C before attempting further shocks (**Evidence grade:** 2C). Once the core temperature reaches 30°C, follow defibrillation guidelines for normothermic patients (**Evidence grade:** 1C).

Airway management in hypothermia

The principles of airway management are the same in a hypothermic patient as in a normothermic patient. In a patient who is not breathing spontaneously or who is breathing spontaneously but not protecting their airway owing to a decreased level of consciousness, advanced airway management with endotracheal intubation or a supraglottic airway device is indicated to provide adequate ventilation and to protect against aspiration.[13,99] Although there are case reports of VF occurring during endotracheal intubation of a hypothermic patient,[21,50,100,101] this is an uncommon complication. In a multicenter study, 117 hypothermic patients were intubated endotracheally after preoxygenation with 100% oxygen with no induced dysrhythmias.[102]

Recommendations

The advantages of advanced airway management outweigh the risk of causing VF (**Evidence grade:** 1C). A nasogastric or orogastric tube should be placed after the airway is secured to decompress the stomach (**Evidence grade:** 1C).

Practical considerations

Rapid-sequence intubation with paralysis may not be effective if the paralytic agent is unable to overcome the trismus produced by profound hypothermia. Fiber optic intubation or cricothyroidotomy may be required to place an endotracheal tube if cold-induced trismus prevents laryngoscopy. A supraglottic device may be preferable to endotracheal intubation in these conditions. Overinflation of an endotracheal tube or supraglottic device cuff with cold air should be avoided because the air inside the cuff will expand as the victim rewarms, potentially kinking the tube or rupturing the cuff.

Ventilation in hypothermia without an advanced airway

Hyperventilation has many potentially adverse effects in hypothermia, including decreased cerebral blood flow. As shown in the swine model, ventilation without an advanced airway is limited by decreased thoracic compliance.[91,103] If available, $ETCO_2$ monitoring can be used to prevent hyperventilation.

Recommendation

In the absence of $ETCO_2$ monitoring, ventilation should be delivered at the same rate recommended for a normothermic patient,[13,19] unless an advanced airway is in place (see below) (**Evidence grade:** 2C).

Ventilation in hypothermia with advanced airway

If the patient is intubated or has a supraglottic device, ventilation is more effective than in a patient without advanced airway management.

Recommendation

In a patient with an advanced airway, if $ETCO_2$ monitoring is not available, ventilation should be delivered at half the rate recommended for a normothermic patient to avoid hyperventilation (**Evidence grade:** 1C).

Management of $ETCO_2$

$ETCO_2$ monitoring can be used to keep $ETCO_2$ in the normal range. This range depends on altitude.

Recommendation

If $ETCO_2$ monitoring is available, $ETCO_2$ should be kept within the normal range. In rescues at altitudes above 1200 m, advanced life support personnel should be aware of the normal range of $ETCO_2$ at a given altitude (**Evidence grade:** 1C).

Anesthetic and neuromuscular blocking agents in hypothermia

At low core temperatures, drug metabolism is decreased; anesthesia and neuromuscular blockade are prolonged.[104], [105], [106]

Recommendation

In patients with core temperatures lower than 30°C, dosages of anesthetic and neuromuscular blocking agents should be decreased, and intervals should be extended according to the degree of hypothermia. Current data are insufficient to recommend specific protocols (**Evidence grade:** 1C).

Supplemental oxygen

Oxygen extraction is not a limiting factor in survival in hypothermia at sea level.[107]

Recommendation

A hypothermic patient should receive supplemental oxygen, especially at altitudes over 2500 m, because of potential benefits and no known harm (**Evidence grade:** 1C).

Circulatory access in hypothermia

Obtaining intravenous (IV) access is often difficult in hypothermic patients. Intraosseous (IO) access is fast and reliable. Because the myocardium is irritable in hypothermia, catheters that contact the heart may cause dysrhythmias. Internal jugular or subclavian central lines that extend into the right atrium are contraindicated unless a short catheter is inserted. There is a risk of causing VF if the wire used during placement of a central venous catheter using the Seldinger technique is advanced into the heart. The femoral vein approach allows central venous access without the danger of inducing dysrhythmias, but it may be difficult in the field. Unsuccessful attempts often cause hematomas.

Recommendations

If circulatory access cannot immediately be obtained with a peripheral IV catheter, access should be obtained by the IO method (**Evidence grade:** 1C). Central venous access can be obtained using a femoral line if no other option is available (**Evidence grade:** 1C).

Volume replacement in hypothermia

Circulating blood volume in moderate and severe hypothermia is reduced.[9,107] During rewarming, vasoconstriction that previously limited the vascular space is abolished. Volume should be replaced to avoid severe volume depletion with resultant shock, while avoiding administration of fluid sufficient to cause volume overload. To prevent further core temperature cooling, IV/IO fluid should be warmed to at least 40°C and preferably to 42°C. In the field, IV/IO bags and tubing should be insulated. Fluid warmers, preferably commercial products that have been proven effective, should be used. Because the effective perfused mass (thermal core) is decreased in hypothermia as a result of intense peripheral vasoconstriction,[9] administration of fluid warmed to 40 to 42°C may help increase core temperature. Because metabolism is depressed, glucose-containing fluid is not essential. The fluid of choice for volume replacement is normal saline. Lactated Ringer's solution should not be used in a hypothermic patient because the cold liver cannot metabolize lactate.[1] Some clinicians use a mixture of crystalloid and colloid.[1]

Recommendation

Resuscitate a hypothermic patient with normal saline warmed to 40 to 42°C given IV or IO. Use caution to prevent volume overload (**Evidence grade:** 1B).

Fluid management in hypothermia

Giving fluids in boluses, as rapidly as possible, rather than by continuous infusion will alleviate problems with cooling of fluid or freezing of lines, which can occur even if lines are insulated. The ideal method is to saline lock the line when there will be a long pause after a bolus. Boluses of 500 mL can be titrated to maintain adequate systolic blood pressure, depending on the degree of hypothermia. There is no available evidence to quantify a target systolic blood pressure.

Recommendations

When practical, fluids should be given as boluses rather than by continuous infusion (**Evidence grade:** 1C). The goal of fluid administration should be to maintain systolic blood pressure at a level that provides adequate perfusion, depending on the degree of hypothermia (**Evidence grade:** 1C).

Use of exogenous glucose and insulin in hypothermia

Hypo- and hyperglycemia have been reported in hypothermia.[96,108] Point-of-care glucose testing is routine in patients with an altered level of consciousness but may not be available in an out-of-hospital setting. Hyperglycemia has not been shown to be deleterious in hypothermic patients.[96]

Recommendation

Glucose should be administered to the hypothermic patient who is hypoglycemic (**Evidence grade:** 1A). Insulin is not initially indicated for hyperglycemia (**Evidence grade:** 1B). If glucose testing is not available, IV glucose can be administered empirically to the hypothermic patient with altered mental status (**Evidence grade:** 1C).

Effects of vasoactive and antidysrhythmic drugs in hypothermia

There is limited evidence regarding drug effects in hypothermic cardiac arrest in humans. Most of the evidence comes from animal studies.[109] The cold heart has long been considered to be unresponsive to vasopressor or antiarrhythmic medications, although some animal studies have suggested otherwise. In a study of hypothermic dogs, epinephrine increased coronary perfusion pressure and ROSC after defibrillation.[110] In a hypothermic pig study, vasopressin increased coronary perfusion pressure with active compression-decompression CPR using an impedance threshold valve, but not with standard CPR.[111] Vasopressin improved ROSC and 1-h survival after defibrillation in a study of hypothermic pigs.[112] There is a case report of ROSC with vasopressin following unsuccessful use of epinephrine (2 mg) in a hypothermic patient, but the patient subsequently died OF multisystem failure.[113]

The ideal pharmacologic approach to ventricular dysrhythmias remains unresolved. Class III agents, such as bretylium and amiodarone, are theoretically ideal because they act directly against fibrillation. Amiodarone is less effective in hypothermia than in normothermia and carries a risk of inducing torsades des pointes.[114] The safety of amiodarone in hypothermia is not known. In a study of hypothermic dogs, the combination of epinephrine and amiodarone increased ROSC after defibrillation following administration of epinephrine alone.[110] Bretylium failed to increase the incidence of ROSC in a study of hypothermic dogs.[115] In another dog study, neither amiodarone nor bretylium improved ROSC.[116] There are 2 clinical reports of resolution of VF following infusion of bretylium.[117,118]

Recommendation

The panel concurs that no recommendation can be made at this time owing to the limited evidence available.

Dosing of drugs in hypothermia

In hypothermia, drug metabolism is decreased and protein binding is increased.[11] Drugs given have little activity while the patient is hypothermic but may reach toxic levels with rewarming.

Recommendations

Do not administer vasoactive drugs until the patient has been rewarmed to 30°C (**Evidence grade:** 1C). To minimize the potential for toxic accumulation of medications, the usual dose can be given, but dosing intervals should be twice as long as usual when the core temperature is 30 to 35°C (**Evidence grade:** 2C).

Transcutaneous cardiac pacing in hypothermia

Two case reports suggest that transcutaneous pacing may be beneficial in the hypothermic patient.[119] In both cases, transcutaneous pacing was instituted to increase blood pressure to facilitate arteriovenous rewarming rather than to control heart rate.

Recommendation

It is the consensus of the panel that transcutaneous pacing may be beneficial in hypothermia in the setting of bradycardia with hypotension disproportionate to the core temperature (**Evidence grade:** 2C).

Management of atrial dysrhythmias during rewarming of a hypothermic patient

Atrial dysrhythmias in hypothermic patients during rewarming are common and resolve spontaneously once the patient has been sufficiently rewarmed.[120]

Recommendation

No treatment is indicated for atrial dysrhythmias in a hemodynamically stable patient during rewarming (**Evidence grade:** IB).

TRANSPORT/TRIAGE

Severe trauma

Core temperatures <35°C are associated with decreased survival in patients with severe trauma.[121,122] Severe trauma can cause acidosis and coagulopathy. In trauma patients with hemorrhagic shock, the “lethal triad” of acidosis, coagulopathy, and hypothermia is associated with multiorgan system dysfunction[123] and extremely high mortality.[124]

Recommendation

To prevent hypothermia, the severely injured patient should be treated early and aggressively with active rewarming during all phases of out-of-hospital care (**Evidence grade:** 1B).

Stabilizing injuries for transport

Stabilization of injuries for transport is the same in a hypothermic patient as in a normothermic patient.

Recommendations

When preparing a patient for transport, potential spinal injuries should be stabilized[125] (**Evidence grade:** 1C). Fractures and dislocations should be reduced as much as possible to normal anatomic configuration (**Evidence grade:** 1C). Open wounds should be covered (**Evidence grade:** 1C).

Patients with mild hypothermia who are alert

Alert patients with mild hypothermia can be treated in the field.

Recommendation

An uninjured patient who is completely alert and shivering may be treated without being transported to a hospital (**Evidence grade:** 1B).

Choice of destination hospital for hypothermic patients

Profoundly hypothermic patients (<28°C) and those with hemodynamic instability and witnessed out-of-hospital cardiac arrest may benefit from transport to centers capable of ECLS. ECLS includes the techniques of extracorporeal circulation, extracorporeal membrane oxygenation (ECMO), and coronary bypass. ECLS provides both oxygenation and hemodynamic support for unstable patients while allowing for controlled rewarming.

Patients who are not profoundly hypothermic or hemodynamically unstable should be transported to the nearest facility.[87,126, 127, 128, 129, 130, 131]

Hypothermic patients with hemodynamic instability

Hemodynamically unstable patients require critical care and may benefit from ECLS with ECMO or CPB. ECMO is preferred over CPB,[127] but both have been used successfully to rewarm severely hypothermic patients. Profoundly hypothermic patients with witnessed cardiac arrest, regardless of return to spontaneous circulation in the field, have a greater chance of survival if transferred to a center where ECLS can be initiated.[87,126, 127, 128, 129, 130, 131]

Many geographic areas do not have a hospital capable of ECLS. Bad weather or other factors may prevent transfer of a patient to a hospital with ECLS. Hemodynamically unstable hypothermic patients, including hypothermic patients in cardiac arrest, have been successfully resuscitated with complete neurologic recovery without using ECLS.[88,132, 133, 134]

Recommendations

A patient with moderate to severe hypothermia who is hemodynamically stable can be transferred to the closest hospital or other appropriate medical facility, such as a rural clinic (**Evidence grade:** 1C). A patient who is hemodynamically unstable or with a core temperature <28°C should be transferred to a hospital capable of providing critical care and ECLS. If this will require significant

additional time—generally more than an additional hour—of noncritical care transport, the patient should first be stabilized at a closer facility (**Evidence grade:** 1C). A patient in cardiac arrest should be transferred to a hospital capable of providing ECLS if possible. If all other factors are equal, ECMO is preferable over CPB (**Evidence grade:** 1B).

In geographic regions where there is no hospital capable of providing ECLS or when a hospital capable of providing ECLS is not accessible, transport a patient in cardiac arrest to the closest hospital where serum potassium can be measured and resuscitation methods not involving ECLS can be attempted for a patient whose serum potassium is <12 mmol·L^{-1} (**Evidence grade:** 1C). (Please see section for use of biochemical markers.)

Hypothermic patients who are alert but have comorbidities, including trauma or asphyxia

Hypothermic patients with injuries or other medical comorbidities should be transferred to a facility able to appropriately manage the patient. Comorbidities can alter the clinical presentation of hypothermia and could potentially delay recognition of severe hypothermia.[135] Asphyxiated patients (from avalanche or drowning) may appear stable but are at risk for delayed complications and are likely to require a higher level of care.[127]

Recommendations

A patient with injuries meeting trauma criteria should be transported to a trauma center (**Evidence grade:** 1B). The asphyxiated patient should be transported to a hospital for observation (**Evidence grade:** 1B).

Use of biochemical markers to determine if resuscitation should be continued in a hypothermic patient without vital signs

Increased serum potassium in a hypothermic patient usually indicates that hypothermia was preceded by hypoxia. As such, it is a marker of cell lysis and death. The highest potassium in a patient resuscitated from hypothermia was 11.8 mmol·L^{-1} in a 31-mo-old child. This level is questionable because the repeat potassium 25 min later was 4.8 mmol·L^{-1} without mention of therapeutic intervention.[136] The highest levels recorded in patients who were resuscitated were 9.5 mmol·L^{-1} in a 13-y-old[137] and 7.9 mmol·L^{-1} in a 34-y-old.[138]

Recommendation

If an adult hypothermic patient has a potassium >12 mmol·L^{-1}, CPR should be terminated (**Evidence grade:** 1B).

Conclusions

To assist medical providers caring for patients with accidental hypothermia in the out-of-hospital setting, we have provided evidence-based recommendations for evaluation and treatment. There are several important areas of uncertainty that

warrant future research. These areas include optimal methods for evaluating patients with accidental hypothermia, best treatments for patients with mild to moderate hypothermia, and optimal methods of resuscitating hypothermic patients in cardiac arrest.

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Appendix A. Supplementary materials

The following is the Supplementary data to this article:Download : Download Word document (13KB)

Supplementary Table 1.

References

1

D.F. Danzl**Accidental hypothermia**
P.S. Auerbach, T.A. Cushing, N.S. Harris (Eds.), Auerbach's Wilderness Medicine (7th ed.), Elsevier, Philadelphia, PA (2017), pp. 135-162
Google Scholar

2

K. Zafren, G.G. Giesbrecht, D.F. Danzl, H. Brugger, E.B. Sagalyn, B. Walpoth, *et al.***Wilderness Medical Society practice guidelines for the out-of-hospital evaluation and treatment of accidental hypothermia: 2014 update**
Wilderness Environ Med, 25 (4 Suppl) (2014), pp. S66-S85
Article Download PDFView Record in ScopusGoogle Scholar

3

G. Guyatt, D. Gutterman, M.H. Baumann, D. Addrizzo-Harris, E.M. Hylek, B. Phillips, *et al.***Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force**
Chest, 129 (1) (2006), pp. 174-181
Article Download PDFCrossRefView Record in ScopusGoogle Scholar

4

G.G. Giesbrecht**Cold stress, near drowning and accidental hypothermia: a review**
Aviat Space Environ Med, 71 (7) (2000), pp. 733-752
View Record in ScopusGoogle Scholar

5

G.K. Bristow, G.G. Giesbrecht**Contribution of exercise and shivering to recovery from induced hypothermia (31.2°C) in one subject**
Aviat Space Environ Med, 59 (6) (1988), pp. 549-552
View Record in ScopusGoogle Scholar

6

T. FitzGibbon, J.S. Hayward, D. Walker**EEG and visual evoked potentials of conscious man during moderate hypothermia**
Electroencephalogr Clin Neurophysiol, 58 (1) (1984), pp. 48-54
Article Download PDFView Record in ScopusGoogle Scholar

7

G.G. Giesbrecht, J.L. Arnett, E. Vela, G.K. Bristow**Effect of task complexity on mental performance during immersion hypothermia**
Aviat Space Environ Med, 64 (3 Pt 1) (1993), pp. 206-211
View Record in ScopusGoogle Scholar

8

J.D. Michenfelder, J.H. Milde**The relationship among canine brain temperature, metabolism, and function during hypothermia**
Anesthesiology, 75 (1) (1991), pp. 130-136
CrossRefView Record in ScopusGoogle Scholar

9

L. Vanggaard, D. Eyolfson, X. Xu, G. Weseen, G.G. Giesbrecht**Immersion of distal arms and legs in warm water (AVA rewarming) effectively rewarms mildly hypothermic humans**
Aviat Space Environ Med, 70 (11) (1999), pp. 1081-1088
View Record in ScopusGoogle Scholar

10

H. Duguid, R.G. Simpson, J.M. Stowers**Accidental hypothermia**
Lancet, 2 (7214) (1961), pp. 1213-1219
Article Download PDFView Record in ScopusGoogle Scholar

11

G.G. Giesbrecht**The respiratory system in a cold environment**
Aviat Space Environ Med, 66 (9) (1995), pp. 890-902
View Record in ScopusGoogle Scholar

12

B. Durrer, H. Brugger, D. Syme, International Commission for Mountain Emergency Medicine**The medical on-site treatment of hypothermia: ICAR-MEDCOM recommendation**
High Alt Med Biol, 4 (1) (2003), pp. 99-103
CrossRefView Record in ScopusGoogle Scholar

13

J. Soar, G.D. Perkins, G. Abbas, A. Alfonzo, A. Barelli, J.J. Bierens, *et al.***European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: electrolyte**

abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution
Resuscitation, 81 (10) (2010), pp. 1400-1433
Article Download PDFView Record in ScopusGoogle Scholar

14
K. Zafren, G.G. Giesbrecht**State of Alaska Cold Injury Guidelines**
Juneau: State of Alaska (2014)
Google Scholar

15
G. Strapazzon, E. Procter, P. Paal, H. Brugger**Pre-hospital core temperature measurement in accidental and therapeutic hypothermia**
High Alt Med Biol, 15 (2) (2014), pp. 104-111
CrossRefView Record in ScopusGoogle Scholar

16
P. Tikuisis, G.G. Giesbrecht**Prediction of shivering heat production from core and mean skin temperatures**
Eur J Appl Physiol Occup Physiol, 79 (3) (1999), pp. 221-229
View Record in ScopusGoogle Scholar

17
G.G. Giesbrecht**Emergency treatment of hypothermia**
Emerg Med (Fremantle), 13 (1) (2001), pp. 9-16
View Record in ScopusGoogle Scholar

18
G.G. Giesbrecht**"Cold Card" to guide responders in the assessment and care of cold-exposed patients**
Wilderness Environ Med, 29 (4) (2018), pp. 499-503
Article Download PDFView Record in ScopusGoogle Scholar

19
T.L. Vanden Hoek, L.J. Morrison, M. Shuster, M. Donnino, E. Sinz, E.J. Lavonas, *et al.***Part 12: cardiac arrest in special situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care**
Circulation, 122 (18 Suppl 3) (2010), pp. S829-S861
CrossRefView Record in ScopusGoogle Scholar

20
T. Deslarzes, V. Rousson, B. Yersin, B. Durrer, M. Pasquier**An evaluation of the Swiss staging model for hypothermia using case reports from the literature**
Scand J Trauma Resusc Emerg Med, 24 (2016), p. 16
Google Scholar

21
F.J. Baumgartner, M.T. Janusz, W.R. Jamieson, T. Winkler, L.H. Burr, J.A. Vestrup**Cardiopulmonary bypass for resuscitation of patients with**

accidental hypothermia and cardiac arrest
Can J Surg, 35 (2) (1992), pp. 184-187
View Record in ScopusGoogle Scholar

22

P. Feiss, C. Mora, B. Devalois, R. Gobeaux, C. Christides**[Accidental deep hypothermia and circulatory arrest. Treatment with extracorporeal circulation]**
Ann Fr Anesth Reanim, 6 (3) (1987), pp. 217-218
Article Download PDFView Record in ScopusGoogle Scholar

23

R. Oberhammer, W. Beikircher, C. Hormann, I. Lorenz, R. Pycha, L. Adler-Kastner, *et al.***Full recovery of an avalanche victim with profound hypothermia and prolonged cardiac arrest treated by extracorporeal re-warming**
Resuscitation, 76 (3) (2008), pp. 474-480
Article Download PDFView Record in ScopusGoogle Scholar

24

M. Papenhausen, L. Burke, A. Antony, J.D. Phillips**Severe hypothermia with cardiac arrest: complete neurologic recovery in a 4-year-old child**
J Pediatr Surg, 36 (10) (2001), pp. 1590-1592
Article Download PDFView Record in ScopusGoogle Scholar

25

M. Pasquier, N. Zurron, B. Weith, P. Turini, F. Dami, P.N. Carron, *et al.***Deep accidental hypothermia with core temperature below 24°C presenting with vital signs**
High Alt Med Biol, 15 (1) (2014), pp. 58-63
CrossRefView Record in ScopusGoogle Scholar

26

H.B. Stoner**Thermoregulation after injury**
Adv Exp Med Biol, 33 (0) (1972), pp. 495-499
View Record in ScopusGoogle Scholar

27

A. Jain, M. Gray, S. Slisz, J. Haymore, N. Badjatia, E. Kulstad**Shivering treatments for targeted temperature management: a review**
J Neurosci Nurs, 50 (2) (2018), pp. 63-67
CrossRefView Record in ScopusGoogle Scholar

28

G.G. Giesbrecht, M.S. Goheen, C.E. Johnston, G.P. Kenny, G.K. Bristow, J.S. Hayward**Inhibition of shivering increases core temperature afterdrop and attenuates rewarming in hypothermic humans**
J Appl Physiol (1985), 83 (5) (1997), pp. 1630-1634
CrossRefView Record in ScopusGoogle Scholar

29

J.S. Hayward, J.D. Eckerson, D. Kemna**Thermal and cardiovascular changes during three methods of resuscitation from mild hypothermia**
Resuscitation, 11 (1–2) (1984), pp. 21-33
Article Download PDFView Record in ScopusGoogle Scholar

30

M.S. Goheen, M.B. Ducharme, G.P. Kenny, C.E. Johnston, J. Frim, G.K. Bristow, *et al.***Efficacy of forced-air and inhalation rewarming by using a human model for severe hypothermia**
J Appl Physiol (1985), 83 (5) (1997), pp. 1635-1640
CrossRefView Record in ScopusGoogle Scholar

31

I.B. Mekjavic, O. Eiken**Inhalation rewarming from hypothermia: an evaluation in -20°C simulated field conditions**
Aviat Space Environ Med, 66 (5) (1995), pp. 424-429
View Record in ScopusGoogle Scholar

32

J.A. Sterba**Efficacy and safety of prehospital rewarming techniques to treat accidental hypothermia**
Ann Emerg Med, 20 (8) (1991), pp. 896-901
Article Download PDFView Record in ScopusGoogle Scholar

33

B.H. Walpoth, J. Galdikas, F. Leupi, W. Muehlemann, P. Schlaepfer, U. Althaus**Assessment of hypothermia with a new "tympanic" thermometer**
J Clin Monit, 10 (2) (1994), pp. 91-96
View Record in ScopusGoogle Scholar

34

S.C. Skaiaa, G. Brattebø, J. Aßmus, Ø. Thomassen**The impact of environmental factors in pre-hospital thermistor-based tympanic temperature measurement: a pilot field study**
Scand J Trauma Resusc Emerg Med, 23 (2015), p. 72
Google Scholar

35

C.K. Grissom, C.H. Harmston, J.C. McAlpine, M.I. Radwin, B. Ellington, E.L. Hirshberg, *et al.***Spontaneous endogenous core temperature rewarming after cooling due to snow burial**
Wilderness Environ Med, 21 (3) (2010), pp. 229-235
Article Download PDFView Record in ScopusGoogle Scholar

36

O. Kimberger, D. Cohen, U. Illievich, R. Lenhardt**Temporal artery versus bladder thermometry during perioperative and intensive care unit monitoring**
Anesth Analg, 105 (4) (2007), pp. 1042-1047
CrossRefView Record in ScopusGoogle Scholar

37

H.C. Gunga, A. Werner, A. Stahn, M. Steinach, T. Schlabs, E. Koralewski, *et al.***The double sensor - a non-invasive device to continuously monitor core temperature in humans on earth and in space**
Respir Physiol Neurobiol, 169 (Suppl 1) (2009), pp. S63-S68
Article Download PDFView Record in ScopusGoogle Scholar

38
O. Kimberger, R. Thell, M. Schuh, J. Koch, D.I. Sessler, A. Kurz**Accuracy and precision of a novel non-invasive core thermometer**
Br J Anaesth, 103 (2) (2009), pp. 226-231
Article Download PDFCrossRefView Record in ScopusGoogle Scholar

39
C. Dahyot-Fizelier, S. Lamarche, T. Kerforne, T. Benard, B. Giraud, R. Bellier, *et al.***Accuracy of zero-heat-flux cutaneous temperature in intensive care adults**
Crit Care Med, 45 (7) (2017), pp. e715-e717
View Record in ScopusGoogle Scholar

40
G.G. Giesbrecht, G.K. Bristow**A second postcooling afterdrop: more evidence for a convective mechanism**
J Appl Physiol (1985), 73 (4) (1992), pp. 1253-1258
CrossRefView Record in ScopusGoogle Scholar

41
F.S. Golden, G.R. Hervey**The mechanism of the after-drop following immersion hypothermia in pigs [proceedings]**
J Physiol, 272 (1) (1977), pp. 26P-27P
Google Scholar

42
T.T. Romet**Mechanism of afterdrop after cold water immersion**
J Appl Physiol (1985), 65 (4) (1988), pp. 1535-1538
CrossRefView Record in ScopusGoogle Scholar

43
J.B. Fox, F. Thomas, T.P. Clemmer, M. Grossman**A retrospective analysis of air-evacuated hypothermia patients**
Aviat Space Environ Med, 59 (11 Pt 1) (1988), pp. 1070-1075
View Record in ScopusGoogle Scholar

44
M.D. Stoneham, S.J. Squires**Prolonged resuscitation in acute deep hypothermia**
Anaesthesia, 47 (9) (1992), pp. 784-788
CrossRefView Record in ScopusGoogle Scholar

45
F.S. Golden, G.R. Hervey, M.J. Tipton**Circum-rescue collapse: collapse, sometimes fatal, associated with rescue of immersion victims**
J R Nav Med Serv, 77 (3) (1991), pp. 139-149

View Record in ScopusGoogle Scholar

46

G.G. Giesbrecht, G.K. Bristow**The convective afterdrop component during hypothermic exercise decreases with delayed exercise onset**
Aviat Space Environ Med, 69 (1) (1998), pp. 17-22
View Record in ScopusGoogle Scholar

47

G.G. Giesbrecht, J.S. Hayward**Problems and complications with cold-water rescue**
Wilderness Environ Med, 17 (1) (2006), pp. 26-30
Article Download PDFCrossRefView Record in ScopusGoogle Scholar

48

U. Althaus, P. Aeberhard, P. Schupbach, B.H. Nachbur, W. Muhlemann**Management of profound accidental hypothermia with cardiorespiratory arrest**
Ann Surg, 195 (4) (1982), pp. 492-495
CrossRefView Record in ScopusGoogle Scholar

49

C.H. Lee, C. Van Gelder, K. Burns, D.C. Cone**Advanced cardiac life support and defibrillation in severe hypothermic cardiac arrest**
Prehosp Emerg Care, 13 (1) (2009), pp. 85-89
View Record in ScopusGoogle Scholar

50

L. Osborne, A.S. Kamal El-Din, J.E. Smith**Survival after prolonged cardiac arrest and accidental hypothermia**
Br Med J (Clin Res Ed)., 289 (6449) (1984), pp. 881-882
CrossRefView Record in ScopusGoogle Scholar

51

O. Henriksson, J.P. Lundgren, K. Kuklane, I. Holmer, U. Bjornstig**Protection against cold in prehospital care-thermal insulation properties of blankets and rescue bags in different wind conditions**
Prehosp Disaster Med, 24 (5) (2009), pp. 408-415
View Record in ScopusGoogle Scholar

52

Ø. Thomassen, H. Faerevik, Ø. Osteras, G.A. Sunde, E. Zakariassen, M. Sandsund, *et al.***Comparison of three different prehospital wrapping methods for preventing hypothermia--a crossover study in humans**
Scand J Trauma Resusc Emerg Med, 19 (2011), p. 41
CrossRefGoogle Scholar

53

J.S. Hayward, M. Collis, J.D. Eckerson**Thermographic evaluation of relative heat loss areas of man during cold water immersion**
Aerosp Med, 44 (7) (1973), pp. 708-711
View Record in ScopusGoogle Scholar

54

T. Pretorius, G.K. Bristow, A.M. Steinman, G.G. Giesbrecht**Thermal effects of whole head submersion in cold water on nonshivering humans**
J Appl Physiol (1985), 101 (2) (2006), pp. 669-675
CrossRefView Record in ScopusGoogle Scholar

55

O. Henriksson, P. Lundgren, K. Kuklane, I. Holmer, P. Naredi, U. Bjornstig**Protection against cold in prehospital care: evaporative heat loss reduction by wet clothing removal or the addition of a vapor barrier--a thermal manikin study**
Prehosp Disaster Med, 27 (1) (2012), pp. 53-58
View Record in ScopusGoogle Scholar

56

P.F. Iampietro, J.A. Vaughan, R.F. Goldman, M.B. Kreider, F. Masucci, D.E. Bass**Heat production from shivering**
J Appl Physiol, 15 (1960), pp. 632-634
CrossRefView Record in ScopusGoogle Scholar

57

G.G. Giesbrecht, D.I. Sessler, I.B. Mekjavic, M. Schroeder, G.K. Bristow**Treatment of mild immersion hypothermia by direct body-to-body contact**
J Appl Physiol (1985), 76 (6) (1994), pp. 2373-2379
CrossRefView Record in ScopusGoogle Scholar

58

M.V. Hultzer, X. Xu, C. Marrao, G. Bristow, A. Chochinov, G.G. Giesbrecht**Pre-hospital torso-warming modalities for severe hypothermia: a comparative study using a human model**
CJEM, 7 (6) (2005), pp. 378-386
View Record in ScopusGoogle Scholar

59

D.D. Watts, M. Roche, R. Tricarico, F. Poole, J.J. Brown Jr., G.B. Colson, *et al.***The utility of traditional prehospital interventions in maintaining thermostasis**
Prehosp Emerg Care, 3 (2) (1999), pp. 115-122
CrossRefView Record in ScopusGoogle Scholar

60

P. Lundgren, O. Henriksson, P. Naredi, U. Bjornstig**The effect of active warming in prehospital trauma care during road and air ambulance transportation - a clinical randomized trial**
Scand J Trauma Resusc Emerg Med, 19 (2011), p. 59
CrossRefGoogle Scholar

61

J.P. Lundgren, O. Henriksson, T. Pretorius, F. Cahill, G. Bristow, A. Chochinov, *et al.***Field torso-warming modalities: a comparative study using a human model**

Prehosp Emerg Care, 13 (3) (2009), pp. 371-378
CrossRefView Record in ScopusGoogle Scholar

62

G.G. Giesbrecht, G.K. Bristow, A. Uin, A.E. Ready, R.A. Jones**Effectiveness of three field treatments for induced mild (33.0°C) hypothermia**
J Appl Physiol (1985), 63 (6) (1987), pp. 2375-2379
CrossRefView Record in ScopusGoogle Scholar

63

R. Dutta, K. Kulkarni, P. Gardiner, A. Steinman, G. Giesbrecht**PW 2321 Human responses to five heated hypothermia enclosure systems in a cold environment**
Injury Prevention, 24 (2018)
A216.1–A
Google Scholar

64

P.B. Allen, S.W. Salyer, M.A. Dubick, J.B. Holcomb, L.H. Blackbourne**Preventing hypothermia: comparison of current devices used by the US Army in an in vitro warmed fluid model**
J Trauma, 69 (Suppl 1) (2010), pp. S154-S161
CrossRefGoogle Scholar

65

Z. Arthurs, D. Cuadrado, A. Beekley, K. Grathwohl, J. Perkins, R. Rush, *et al.***The impact of hypothermia on trauma care at the 31st combat support hospital**
Am J Surg, 191 (5) (2006), pp. 610-614
Article Download PDFView Record in ScopusGoogle Scholar

66

K. Palm, A. Apodaca, D. Spencer, G. Costanzo, J. Bailey, L.H. Blackbourne, *et al.***Evaluation of military trauma system practices related to damage-control resuscitation**
J Trauma Acute Care Surg, 73 (6 Suppl 5) (2012), pp. S459-S464
View Record in ScopusGoogle Scholar

67

B.L. Bennett, J.B. Holcomb**Battlefield trauma-induced hypothermia: transitioning the preferred method of casualty rewarming**
Wilderness Environ Med, 28 (2S) (2017), pp. S82-S89
Article Download PDFView Record in ScopusGoogle Scholar

68

M.B. Ducharme, P. Tikuisis**Role of blood as heat source or sink in human limbs during local cooling and heating**
J Appl Physiol (1985), 76 (5) (1994), pp. 2084-2094
CrossRefView Record in ScopusGoogle Scholar

69

K. Kulkarni, E. Hildahl, R. Dutta, S.C. Webber, S. Passmore, G.K. McDonald, *et al.***Efficacy of head and torso rewarming using a human model for severe hypothermia**
Wilderness Environ Med, 30 (1) (2019), pp. 35-43
Article Download PDFView Record in ScopusGoogle Scholar

70
A.M. Steinman**Cardiopulmonary resuscitation and hypothermia**
Circulation, 74 (6 Pt 2) (1986), pp. IV29-IV32
Google Scholar

71
G.G. Giesbrecht**Immersion into cold water**
(7th ed.)P.S. Auerbach, T.A. Cushing, N.S. Harris (Eds.), Auerbach's Wilderness Medicine, Vol 1, Elsevier, Philadelphia, PA (2017), p. 162
View Record in ScopusGoogle Scholar

72
G.G. Giesbrecht, B. Walpoth**Risk of burns during active external rewarming for accidental hypothermia**
Wilderness Environ Med, 30 (4) (2019), pp. 431-436
Article Download PDFView Record in ScopusGoogle Scholar

73
M.B. Ducharme, G.G. Giesbrecht, J. Frim, G.P. Kenny, C.E. Johnston, M.S. Goheen, *et al.***Forced-air rewarming in -20°C simulated field conditions**
Ann NY Acad Sci, 813 (1997), pp. 676-681
CrossRefView Record in ScopusGoogle Scholar

74
M.T. Steele, M.J. Nelson, D.I. Sessler, L. Fraker, B. Bunney, W.A. Watson, *et al.***Forced air speeds rewarming in accidental hypothermia**
Ann Emerg Med, 27 (4) (1996), pp. 479-484
Article Download PDFView Record in ScopusGoogle Scholar

75
G.G. Giesbrecht, M. Schroeder, G.K. Bristow**Treatment of mild immersion hypothermia by forced-air warming**
Aviat Space Environ Med, 65 (9) (1994), pp. 803-808
View Record in ScopusGoogle Scholar

76
C.S. Ko, J. Alex, S. Jeffries, J.M. Parmar**Dead? Or just cold: profoundly hypothermic patient with no signs of life**
Emerg Med J, 19 (5) (2002), pp. 478-479
View Record in ScopusGoogle Scholar

77
K.H. Leitz, N. Tsilimingas, H.G. Guse, P. Meier, H.J. Bachmann**[Accidental drowning with extreme hypothermia--rewarming with extracorporeal circulation]**
Chirurg, 60 (5) (1989), pp. 352-355

View Record in ScopusGoogle Scholar

78

B.H. Walpoth, B.N. Walpoth-Aslan, H.P. Mattle, B.P. Radanov, G. Schroth, L. Schaeffler, *et al.***Outcome of survivors of accidental deep hypothermia and circulatory arrest treated with extracorporeal blood warming**
N Engl J Med, 337 (21) (1997), pp. 1500-1505
View Record in ScopusGoogle Scholar

79

P. Paal, M. Milani, D. Brown, J. Boyd, J. Ellerton**Termination of cardiopulmonary resuscitation in mountain rescue**
High Alt Med Biol, 13 (3) (2012), pp. 200-208
CrossRefView Record in ScopusGoogle Scholar

80

M. Gilbert, R. Busund, A. Skagseth, P.A. Nilsen, J.P. Solbo**Resuscitation from accidental hypothermia of 13.7°C with circulatory arrest**
Lancet, 355 (9201) (2000), pp. 375-376
Article Download PDFView Record in ScopusGoogle Scholar

81

S.A. Niazi, F.J. Lewis**Profound hypothermia in man; report of a case**
Ann Surg, 147 (2) (1958), pp. 264-266
CrossRefView Record in ScopusGoogle Scholar

82

B.R. Englum, N.D. Andersen, A.M. Husain, J.P. Mathew, G.C. Hughes**Degree of hypothermia in aortic arch surgery - optimal temperature for cerebral and spinal protection: deep hypothermia remains the gold standard in the absence of randomized data**
Ann Cardiothorac Surg, 2 (2) (2013), pp. 184-193
View Record in ScopusGoogle Scholar

83

L.G. Svensson, E.S. Crawford, K.R. Hess, J.S. Coselli, S. Raskin, S.A. Shenaq, *et al.***Deep hypothermia with circulatory arrest. Determinants of stroke and early mortality in 656 patients**
J Thorac Cardiovasc Surg, 106 (1) (1993), pp. 19-28
View Record in ScopusGoogle Scholar

84

M. Meyer, N. Pelurson, E. Khabiri, N. Siegenthaler, B.H. Walpoth**Sequela-free long-term survival of a 65-year-old woman after 8 hours and 40 minutes of cardiac arrest from deep accidental hypothermia**
J Thorac Cardiovasc Surg, 147 (1) (2014), pp. e1-e2
Article Download PDFView Record in ScopusGoogle Scholar

85

A. Forti, P. Brugnaro, S. Rauch, M. Crucitti, H. Brugger, G. Cipollotti, *et al.***Hypothermic cardiac arrest with full neurologic recovery after**

approximately nine hours of cardiopulmonary resuscitation: management and possible complications
Ann Emerg Med, 73 (1) (2019), pp. 52-57
Article Download PDFView Record in ScopusGoogle Scholar

86
Y. Boue, J. Lavolaine, P. Bouzat, S. Matraxia, O. Chavanon, J.F. Payen**Neurologic recovery from profound accidental hypothermia after 5 hours of cardiopulmonary resuscitation**
Crit Care Med, 42 (2) (2014), pp. e167-e170
CrossRefView Record in ScopusGoogle Scholar

87
J. Hilmo, T. Naesheim, M. Gilbert**"Nobody is dead until warm and dead": prolonged resuscitation is warranted in arrested hypothermic victims also in remote areas -- a retrospective study from northern Norway**
Resuscitation, 85 (9) (2014), pp. 1204-1211
Article Download PDFView Record in ScopusGoogle Scholar

88
K. Lexow**Severe accidental hypothermia: survival after 6 hours 30 minutes of cardiopulmonary resuscitation**
Arctic Med Res, 50 (Suppl 6) (1991), pp. 112-114
View Record in ScopusGoogle Scholar

89
L. Gordon, P. Paal, J.A. Ellerton, H. Brugger, G.J. Peek, K. Zafren**Delayed and intermittent CPR for severe accidental hypothermia**
Resuscitation, 90 (2015), pp. 46-49
Article Download PDFView Record in ScopusGoogle Scholar

90
R. Imberti, G. Bellinzona, F. Riccardi, M. Pagani, M. Langer**Cerebral perfusion pressure and cerebral tissue oxygen tension in a patient during cardiopulmonary resuscitation**
Intensive Care Med, 29 (6) (2003), pp. 1016-1019
CrossRefView Record in ScopusGoogle Scholar

91
P.A. Maningas, L.R. DeGuzman, S.J. Hollenbach, K.A. Volk, R.F. Bellamy**Regional blood flow during hypothermic arrest**
Ann Emerg Med, 15 (4) (1986), pp. 390-396
Article Download PDFView Record in ScopusGoogle Scholar

92
U. Pietsch, V. Lischke, C. Pietsch, K.H. Kopp**Mechanical chest compressions in an avalanche victim with cardiac arrest: an option for extreme mountain rescue operations**
Wilderness Environ Med, 25 (2) (2014), pp. 190-193
Article Download PDFView Record in ScopusGoogle Scholar

93

L. Wik, S. Kiil**Use of an automatic mechanical chest compression device (LUCAS) as a bridge to establishing cardiopulmonary bypass for a patient with hypothermic cardiac arrest**
Resuscitation, 66 (3) (2005), pp. 391-394
Article Download PDFView Record in ScopusGoogle Scholar

94

M.R. Ujhelyi, J.J. Sims, S.A. Dubin, J. Vender, A.W. Miller**Defibrillation energy requirements and electrical heterogeneity during total body hypothermia**
Crit Care Med, 29 (5) (2001), pp. 1006-1011
View Record in ScopusGoogle Scholar

95

T.S. DaVee, E.J. Reineberg**Extreme hypothermia and ventricular fibrillation**
Ann Emerg Med, 9 (2) (1980), pp. 100-102
Article Download PDFView Record in ScopusGoogle Scholar

96

R. Koller, T.W. Schnider, P. Neidhart**Deep accidental hypothermia and cardiac arrest--rewarming with forced air**
Acta Anaesthesiol Scand, 41 (10) (1997), pp. 1359-1364
CrossRefView Record in ScopusGoogle Scholar

97

R. Thomas, C.J. Cahill**Successful defibrillation in profound hypothermia (core body temperature 25.6°C)**
Resuscitation, 47 (3) (2000), pp. 317-320
Article Download PDFView Record in ScopusGoogle Scholar

98

P. Mair, L. Gasteiger, B. Mair, M. Stroehle, B. Walpoth**Successful defibrillation of four hypothermic patients with witnessed cardiac arrest**
High Alt Med Biol, 20 (1) (2019), pp. 71-77
CrossRefView Record in ScopusGoogle Scholar

99

L.J. Morrison, C.D. Deakin, P.T. Morley, C.W. Callaway, R.E. Kerber, S.L. Kronick, *et al.***Part 8: Advanced life support: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations**
Circulation, 122 (16 Suppl 2) (2010), pp. S345-S421
CrossRefView Record in ScopusGoogle Scholar

100

R. Binnema, A. van der Wal, C. Visser, R. Schepp, L. Jekel, P. Schroder**Treatment of accidental hypothermia with cardiopulmonary bypass: a case report**
Perfusion, 23 (3) (2008), pp. 193-196
CrossRefView Record in ScopusGoogle Scholar

101

M.G. Hauty, B.C. Esrig, J.G. Hill, W.B. Long**Prognostic factors in severe accidental hypothermia: experience from the Mt. Hood tragedy**
J Trauma, 27 (10) (1987), pp. 1107-1112
CrossRefView Record in ScopusGoogle Scholar

102

D.F. Danzl, R.S. Pozos, P.S. Auerbach, S. Glazer, W. Goetz, E. Johnson, *et al.***Multicenter hypothermia survey**
Ann Emerg Med, 16 (9) (1987), pp. 1042-1055
Article Download PDFView Record in ScopusGoogle Scholar

103

C.W. Deal, J.C. Warden, I. Monk**Effect of hypothermia on lung compliance**
Thorax, 25 (1) (1970), pp. 105-109
CrossRefView Record in ScopusGoogle Scholar

104

J.E. Caldwell, T. Heier, P.M. Wright, S. Lin, G. McCarthy, J. Szenohradszky, *et al.***Temperature-dependent pharmacokinetics and pharmacodynamics of vecuronium**
Anesthesiology, 92 (1) (2000), pp. 84-93
View Record in ScopusGoogle Scholar

105

T. Heier, J.E. Caldwell**Impact of hypothermia on the response to neuromuscular blocking drugs**
Anesthesiology, 104 (5) (2006), pp. 1070-1080
CrossRefView Record in ScopusGoogle Scholar

106

K. Leslie, D.I. Sessler, A.R. Bjorksten, A. Moayeri**Mild hypothermia alters propofol pharmacokinetics and increases the duration of action of atracurium**
Anesth Analg, 80 (5) (1995), pp. 1007-1014
View Record in ScopusGoogle Scholar

107

T.V. Kondratiev, K. Flemming, E.S. Myhre, M.A. Sovershaev, T. Tveita**Is oxygen supply a limiting factor for survival during rewarming from profound hypothermia?**
Am J Physiol Heart Circ Physiol, 291 (1) (2006), pp. H441-H450
CrossRefView Record in ScopusGoogle Scholar

108

G. Strapazzon, M. Nardin, P. Zanon, M. Kaufmann, M. Kritzinger, H. Brugger**Respiratory failure and spontaneous hypoglycemia during noninvasive rewarming from 24.7°C (76.5°F) core body temperature after prolonged avalanche burial**
Ann Emerg Med, 60 (2) (2012), pp. 193-196
Article Download PDFView Record in ScopusGoogle Scholar

109

C.R. Wira, J.U. Becker, G. Martin, M.W. Donnino**Anti-arrhythmic and vasopressor medications for the treatment of ventricular fibrillation in severe hypothermia: a systematic review of the literature**
Resuscitation, 78 (1) (2008), pp. 21-29
Article Download PDFView Record in ScopusGoogle Scholar

110

C. Wira, G. Martin, J. Stoner, K. Margolis, M. Donnino**Application of normothermic cardiac arrest algorithms to hypothermic cardiac arrest in a canine model**
Resuscitation, 69 (3) (2006), pp. 509-516
Article Download PDFView Record in ScopusGoogle Scholar

111

C. Raedler, W.G. Voelckel, V. Wenzel, L. Bahlmann, W. Baumeier, C.A. Schmittinger, *et al.***Vasopressor response in a porcine model of hypothermic cardiac arrest is improved with active compression-decompression cardiopulmonary resuscitation using the inspiratory impedance threshold valve**
Anesth Analg, 95 (6) (2002), pp. 1496-1502
View Record in ScopusGoogle Scholar

112

B. Schwarz, P. Mair, C. Raedler, D. Deckert, V. Wenzel, K.H. Lindner**Vasopressin improves survival in a pig model of hypothermic cardiopulmonary resuscitation**
Crit Care Med, 30 (6) (2002), pp. 1311-1314
View Record in ScopusGoogle Scholar

113

G. Sumann, A.C. Krismer, V. Wenzel, E. Adelsmayr, B. Schwarz, K.H. Lindner, *et al.***Cardiopulmonary resuscitation after near drowning and hypothermia: restoration of spontaneous circulation after vasopressin**
Acta Anaesthesiol Scand, 47 (3) (2003), pp. 363-365
CrossRefView Record in ScopusGoogle Scholar

114

J.N. Khan, N. Prasad, J. Glancy**Amiodarone use in therapeutic hypothermia following cardiac arrest due to ventricular tachycardia and ventricular fibrillation**
Europace, 11 (11) (2009), pp. 1566-1567
CrossRefView Record in ScopusGoogle Scholar

115

R.M. Elenbaas, K. Mattson, H. Cole, M. Steele, J. Ryan, W. Robinson**Bretylium in hypothermia-induced ventricular fibrillation in dogs**
Ann Emerg Med, 13 (11) (1984), pp. 994-999
Article Download PDFView Record in ScopusGoogle Scholar

116

J. Stoner, G. Martin, K. O'Mara, J. Ehlers, M. Tomlanovich**Amiodarone and bretylium in the treatment of hypothermic ventricular fibrillation in a canine model**
Acad Emerg Med, 10 (3) (2003), pp. 187-191
View Record in ScopusGoogle Scholar

117

D.F. Danzl, M.B. Sowers, S.J. Vicario, D.M. Thomas, J.W. Miller**Chemical ventricular defibrillation in severe accidental hypothermia**
Ann Emerg Med, 11 (12) (1982), pp. 698-699
Article Download PDFView Record in ScopusGoogle Scholar

118

E.L. Lloyd**Accidental hypothermia**
Resuscitation, 32 (2) (1996), pp. 111-124
Article Download PDFView Record in ScopusGoogle Scholar

119

J.D. Ho, W.G. Heegaard, D.D. Brunette**Successful transcutaneous pacing in 2 severely hypothermic patients**
Ann Emerg Med, 49 (5) (2007), pp. 678-681
Article Download PDFView Record in ScopusGoogle Scholar

120

A.C. Rankin, A.P. Rae**Cardiac arrhythmias during rewarming of patients with accidental hypothermia**
Br Med J (Clin Res Ed)., 289 (6449) (1984), pp. 874-877
CrossRefView Record in ScopusGoogle Scholar

121

G.J. Jurkovich, W.B. Greiser, A. Luterman, P.W. Curreri**Hypothermia in trauma victims: an ominous predictor of survival**
J Trauma, 27 (9) (1987), pp. 1019-1024
CrossRefView Record in ScopusGoogle Scholar

122

R.S. Martin, P.D. Kilgo, P.R. Miller, J.J. Hoth, J.W. Meredith, M.C. Chang**Injury-associated hypothermia: an analysis of the 2004 National Trauma Data Bank**
Shock, 24 (2) (2005), pp. 114-118
CrossRefView Record in ScopusGoogle Scholar

123

G.J. Beilman, J.J. Blondet, T.R. Nelson, A.B. Nathens, F.A. Moore, P. Rhee, *et al.***Early hypothermia in severely injured trauma patients is a significant risk factor for multiple organ dysfunction syndrome but not mortality**
Ann Surg, 249 (5) (2009), pp. 845-850
View Record in ScopusGoogle Scholar

124

B. Mitra, F. Tullio, P.A. Cameron, M. Fitzgerald**Trauma patients with the 'triad of death'**

Emerg Med J, 29 (8) (2012), pp. 622-625
CrossRefView Record in ScopusGoogle Scholar

125

R. Quinn, J. Williams, B. Bennett, G. Stiller, A. Islas, S. McCord, *et al.***Wilderness Medical Society practice guidelines for spine immobilization in the austere environment**
Wilderness Environ Med, 24 (3) (2013), pp. 241-252
Article Download PDFView Record in ScopusGoogle Scholar

126

G. Debaty, I. Moustapha, P. Bouzat, M. Maignan, M. Blancher, A. Rallo, *et al.***Outcome after severe accidental hypothermia in the French Alps: a 10-year review**
Resuscitation, 93 (2015), pp. 118-123
Article Download PDFView Record in ScopusGoogle Scholar

127

E. Ruttmann, A. Weissenbacher, H. Ulmer, L. Muller, D. Hofer, J. Kilo, *et al.***Prolonged extracorporeal membrane oxygenation-assisted support provides improved survival in hypothermic patients with cardiocirculatory arrest**
J Thorac Cardiovasc Surg, 134 (3) (2007), pp. 594-600
Article Download PDFCrossRefView Record in ScopusGoogle Scholar

128

E. Ruttmann, M. Dietl, T. Kastenberger, R. El Attal, M. Strohle, H. Ulmer, *et al.***Characteristics and outcome of patients with hypothermic out-of-hospital cardiac arrest: experience from a European trauma center**
Resuscitation, 120 (2017), pp. 57-62
Article Download PDFView Record in ScopusGoogle Scholar

129

M. Khorsandi, S. Dougherty, N. Young, D. Kerslake, V. Giordano, R. Lendrum, *et al.***Extracorporeal life support for refractory cardiac arrest from accidental hypothermia: a 10-year experience in Edinburgh**
J Emerg Med, 52 (2) (2017), pp. 160-168
Article Download PDFView Record in ScopusGoogle Scholar

130

T. Darocha, S. Kosinski, A. Jarosz, D. Sobczyk, R. Galazkowski, J. Piatek, *et al.***The chain of survival in hypothermic circulatory arrest: encouraging preliminary results when using early identification, risk stratification and extracorporeal rewarming**
Scand J Trauma Resusc Emerg Med, 24 (2016), p. 85
Google Scholar

131

A. Jarosz, T. Darocha, S. Kosinski, R. Galazkowski, P. Mazur, J. Piatek, *et al.***Profound accidental hypothermia: systematic approach to active recognition and treatment**
ASAIO J, 63 (3) (2017), pp. e26-e30

CrossRefView Record in ScopusGoogle Scholar

132

E. Gruber, W. Beikircher, R. Pizzinini, H. Marsoner, M. Pornbacher, H. Brugger, *et al.***Non-extracorporeal rewarming at a rate of 6.8°C per hour in a deeply hypothermic arrested patient**
Resuscitation, 85 (8) (2014), pp. e119-e120
Article Download PDFView Record in ScopusGoogle Scholar

133

E. Roggero, H. Stricker, P. Biegger**[Severe accidental hypothermia with cardiopulmonary arrest: prolonged resuscitation without extracorporeal circulation]**
Schweiz Med Wochenschr, 122 (5) (1992), pp. 161-164
View Record in ScopusGoogle Scholar

134

J. Turtiainen, J. Halonen, S. Syvaoja, T. Hakala**Rewarming a patient with accidental hypothermia and cardiac arrest using thoracic lavage**
Ann Thorac Surg, 97 (6) (2014), pp. 2165-2166
Article Download PDFView Record in ScopusGoogle Scholar

135

T. Woehrle, U. Lichtenauer, A. Bayer, S. Brunner, M. Angstwurm, S.T. Schafer, *et al.***Misleading symptoms and successful noninvasive rewarming of a patient with severe hypothermia (23.1°C)**
Anaesthesist, 67 (12) (2018), pp. 931-935
CrossRefView Record in ScopusGoogle Scholar

136

J.A. Dobson, J.J. Burgess**Resuscitation of severe hypothermia by extracorporeal rewarming in a child**
J Trauma, 40 (3) (1996), pp. 483-485
View Record in ScopusGoogle Scholar

137

L.K. von Segesser, E. Garcia, M. Turina**Perfusion without systemic heparinization for rewarming in accidental hypothermia**
Ann Thorac Surg, 52 (3) (1991), pp. 560-561
Article Download PDFView Record in ScopusGoogle Scholar

138

M. Farstad, K.S. Andersen, M.E. Koller, K. Grong, L. Segadal, P. Husby**Rewarming from accidental hypothermia by extracorporeal circulation. A retrospective study**
Eur J Cardiothorac Surg, 20 (1) (2001), pp. 58-64
CrossRefView Record in ScopusGoogle Scholar

Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Lightning Injuries: 2014 Update

Chris Davis, MD; Anna Engeln, MD; Eric L. Johnson, MD; Scott E. McIntosh, MD, MPH; Ken Zafren, MD; Arthur A. Islas, MD, MPH; Christopher McStay, MD; William R. Smith, MD; Tracy Cushing, MD, MPH

From the Department of Emergency Medicine, University of Colorado School of Medicine, Aurora, CO (Drs Davis, McStay, and Cushing); the Department of Emergency Medicine, Denver Health Medical Center/University of Colorado School of Medicine, Denver,Colorado (Dr Engeln); the Department of Wound Healing and Hyperbaric Medicine, St. Alphonsus Hospital, Boise, ID, and Emergency Services, Teton Valley Hospital, Driggs, ID (Dr Johnson); the Division of Emergency Medicine, University of Utah School of Medicine, Salt Lake City, UT (Dr McIntosh); the Division of Emergency Medicine, Stanford University Medical Center, Palo Alto, CA, the Department of Emergency Medicine, Alaska Native Medical Center, Anchorage, AK, and the International Commission for Mountain Emergency Medicine (Dr Zafren); the Department of Family & Community Medicine, Paul L. Foster School of Medicine (Dr Islas); and the Department of Emergency Medicine, University of Washington School of Medicine, Seattle, WA, Department of Emergency Medicine, St. John's Medical Center, Jackson, WY (Dr Smith).

To provide guidance to clinicians about best practices, the Wilderness Medical Society (WMS) convened an expert panel to develop evidence-based guidelines for the treatment and prevention of lightning injuries. These guidelines include a review of the epidemiology of lightning and recommendations for the prevention of lightning strikes, along with treatment recommendations organized by organ system. Recommendations are graded on the basis of the quality of supporting evidence according to criteria put forth by the American College of Chest Physicians. This is an updated version of the original WMS Practice Guidelines for Prevention and Treatment of Lightning Injuries published inWilderness & Environmental Medicine2012;23(3):260–269.

Key words:lightning, lightning strike, lightning injury, Lichtenberg, keraunoparalysis

Introduction

Lightning occurs nearly 50 times per second worldwide.^1 Approximately one-fifth of these flashes result in ground strikes. Internationally, an estimated 24,000 fatalities with 10 times as many injuries occur annually as a result of lightning.^2 ,^3 To provide guidance to clinicians and prehospital providers and to disseminate knowledge in this area, the Wilderness Medical Society (WMS) convened an expert panel to develop evidence-based guidelines for the treatment and prevention of lightning injuries. The WMS previously published guidelines on lightning injuries in 2006 and 2012.^4 ,^5 This is an updated version of the original WMS Practice Guidelines for Prevention and Treatment of Lightning Injuries published in Wilderness & Environmental Medicine 2012;23(3): 260 – 269. The goal of this review is to update the guidelines published in 2012 with relevant evidence- based information including a summary table of the best available literature. However, it must be recognized that the nature of lightning injuries often limits the available evidence to case reports and case series.

Methods

A panel was first convened at the 2011 Annual Meeting of the WMS in Snowmass, CO. Members were selected on the basis of clinical or research experience. The lead author identified articles through the PUBMED databases using a key word search with the terms lightning, lightning strike, lightning injury, Lichtenberg, and keraunoparalysis. This was supplemented by a hand search of these articles. The amassed evidence was then reviewed and graded for quality by the panel. In August 2014 these guidelines were updated using a key word search of PUBMED for lightning-related articles from 2011 to 2014. The same search terms were used. This supplement also incorporates suggestions from readers related to the original publication. The panel used the American College of Chest Physicians (ACCP) classification scheme for grading evidence and recommendations^6 (see onlineSupplementary ACCP Table 1). Injuries and recommended treatment strategies are organized by organ system.

Epidemiology

REGIONAL CONSIDERATIONS: WEATHER AND GEOGRAPHY

Lightning strikes are not uniformly distributed around the Earth (Figure 1). Regions with frequent thunderstorms have more lightning strikes. Thunderstorms are formed by 3 atmospheric elements: moisture, warm air on the surface of the earth, and a lifting wind. As the warm, moisture- laden air is pushed upward by vertical updraft, it condenses and cools, forming cumulonimbus clouds. Water freezes into ice particles near the top of this cloud. It is believed that the movement of these ice particles forms an electrical gradient (or differential) that is eventually dis- charged as lightning.^1

In addition to prevailing weather patterns, geography is also a determining factor in the location and frequency of thunderstorms. Central Africa has the greatest incidence of lightning strikes because of its mountainous terrain coupled with moist airflow from the Atlantic Ocean. This leads to year-round thunderstorms.^7

Worldwide, rural populations have been at greatest risk. Demographically this risk has been attributed to higher occupational exposure (rural farmers). These populations typically do not have access to substantial buildings that could provide shelter.^2 Though rare, lightning is possible even if the overlying sky is blue (so-called bolt from the blue).^8 This occurs in sunny conditions, usually after a storm,when strikes can return to areas from which the storm has passed, posing a risk to people who return to outdoor activity too soon. Lightning is also possible in snowstorms. Graupel (snow pellets) heralds weather favorable to lightning formation, as ice and snow pellets are believed to generate positive and negative charges as they collide, ultimately providing the electrical gradient that facilitates lightning formation.^9

TRENDS IN THE UNITED STATES

The incidence of lightning-related deaths in the United States has declined consistently during the past 50 years to approximately 40 deaths per year.^10 An estimated 400 lightning injuries occur annually based on data averaged over the last decade.^10 In comparison, approximately 70 flood-related deaths and 30 avalanche-related deaths occur yearly.^11 ,^12 A demographic study of lightning victims reveals that greater than 80% of victims are male.^13 Most deaths occur in individuals 20 to 45 year of age.^14 More than 90% of incidents occur between May and September.^14 Florida and Texas have accounted for nearly a quarter of all lightning-related deaths.^14 Lightning fatalities per state are reported inFigure 2. In the United States, the lifetime risk of being struck by lightning is estimated at 1:10,000.^10

Physics and Physiology

Lightning can be both negatively and positively charged and can take the form of both direct and alternating current depending on circumstance. However, lightning does not cause the muscle tetany seen with alternating currents of other electrical injuries. A bolt of lightning has a massive current ranging from 30,000 to 110,000 A, although such currents are only applied for 10 to 100 ms.^15 Energy transfer to the body is therefore limited.

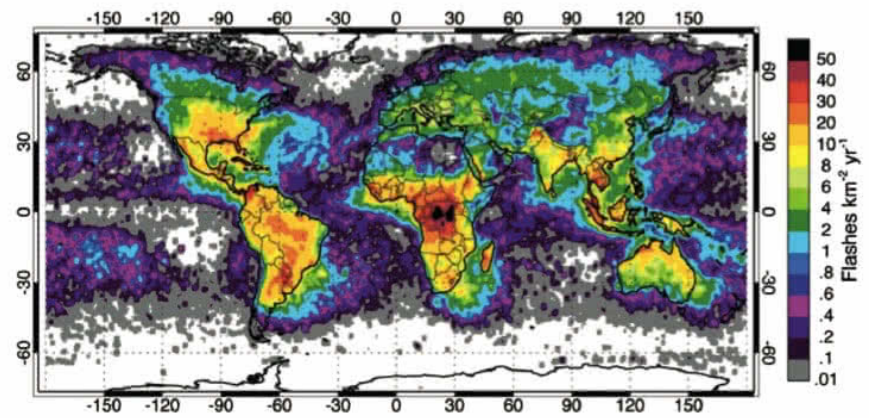

Figure 1.Worldwide density of lightning strikes.

Lightning injuries are classified as direct strike, contact injury, side splash, or ground current.^15 A direct strike occurs when there is an uninterrupted connection between a lightning bolt and an individual; direct strikes are relatively rare, accounting for approximately 5% of lightning strikes involving people.^15 Contact injury occurs when a person is touching an object that is struck. Side splash accounts for one-third of lightning injuries and occurs when the current“splashes”or jumps from a nearby object to the recipient’s body; such splashes follow the path of least resistance when compared with the initially struck object such as a tree. Ground current, also known as step voltage, occurs when lightning strikes an object or the ground near a person and travels through the ground from the strike point to the victim. This mechanism accounts for nearly half of lightning injuries.^15 A fifth mechanism of lightning injury has been recently reported. The“upward streamer” describes current passing up from the ground, through the victim, without a nearby ground strike; ultimately it is postulated that such a current does not become part of a completed lightning channel.^16 Lightning electricity, as with all electrical energy, will travel the path of least resistance. In body tissues, the order of least to greatest resistance is:

- nerve
- blood
- muscle
- skin
- fat
- bone.

Prevention

Evidence-based guidelines are limited regarding lightning prevention and safety. The following recommendations represent opinion from this panel or from previously published guidelines.^17 –^20

BEHAVIORAL STRATEGIES

No place is absolutely safe from lightning. However, individuals can choose safer places in an effort to reduce their risk of lightning strike.“When thunder roars, go indoors”is the currently recommended safety maxim of the National Weather Service. In essence, if one can hear thunder, then there is a risk of lightning strikes and one should seek shelter immediately. As substantial shelter is rarely available in the wilderness, hearing thunder in this setting should trigger an individual to immediately avoid or leave areas that are high risk for lightning strikes, such as ridgelines or summits, and to avoid tall objects such as ski lifts, cell phone towers, or isolated trees. One should observe for changing weather patterns that could indicate a developing thunderstorm: building cumulonimbus clouds, increasing winds, and darkening skies. Previous rules have relied on timing lightning flashes with thunder to estimate distance from an approaching storm. Such calculations may engender a false sense of security either from incorrect calculations or incorrect pairing of a given lightning flash with the correct thunderclap. Individuals should instead rely on observing signs of impending storms and seeking cover accordingly. Individuals should wait a minimum of 30

minutes after hearing the last thunderclap before resuming outdoor activity. Waiting 30 minutes should allow for the trailing edge of the thunder- storm to move the estimated 10 miles needed to establish an appropriate buffer zone. Recommendation grade: 1C.

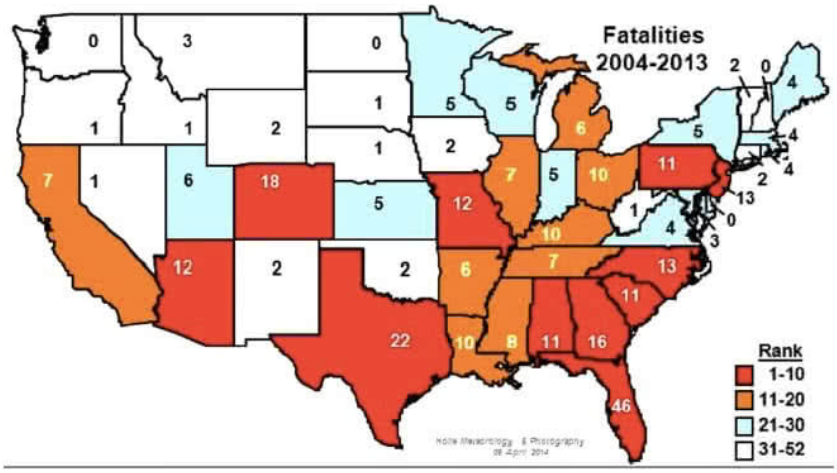

Figure 2. Lightning fatalities by state, 2004-2013.

SHELTER

There is no absolutely safe place from lightning—some locations are safer than others. When possible, shelter should be sought in the largest enclosed building available away from doors or windows. Another option is in a metal-topped vehicle with windows and doors closed; convertibles with fabric tops are not protective.^21 As this option is markedly limited in the wilderness setting, this panel recommends seeking a sheltered area inside a deep cave, far into a dense forest, or in a deep ravine; these features represent a safer alternative than remaining in an open, exposed area. Shallow caves, solitary trees, or open shelters (such as a picnic shelter, dugout, canopy, or lean-to) should be avoided because of the risk of side splash and ground current.^22 ,^23 Tents do not provide adequate protection from lightning.^24 When possible, the safest shelters are a building followed by a hardtop vehicle. Recommendation grade: 1C.

LIGHTNING POSITION

The lightning position involves sitting or crouching with knees and feet close together to create only one point of contact with the ground (Figure 3). If standing, have feet touching. If sitting, lift feet off the ground. Take this position only when a lightning strike is imminent and when all other lightning prevention strategies have failed. Signs of imminent strike include a blue haze around objects or individuals (St. Elmo's fire), static electricity over hair or skin, an ozone smell, or a nearby crackling sound. Attempt to minimize the risk of ground current injury by insulating oneself from the ground; sit on a pack (remove any metal from the pack), a dry coiled rope, or a rolled foam sleeping pad. This is a strategy of last resort, as it is

a difficult position to maintain for a long time, and should not be relied on as primary prevention but may reduce the risk of injury from an imminent lightning strike.^25 Recommendation grade: 2C.

GROUP SAFETY

This panel recommends the separation of group members by greater than 20 feet or more to limit potential mass casualties, as lightning can jump up to 15 feet between objects. Although each individual should be aware of lightning safety, groups should develop a specific light- ning safety plan. Such a plan accounts for local weather patterns, current weather forecast, local terrain, and predetermined available shelter and evacuation routes.^18 ,^19 A preestablished plan should mitigate the chaos of evacuating a crowd during a lightning storm. Further examples of lightning safety plans are available online through the National Lightning Safety Institute and the National Weather Service.^20 ,^26 Recommendation grade: 1C.

LIGHTNING DETECTION TECHNOLOGY

In the United States numerous commercial services are available that can provide automatic notifications when nearby lightning is detected by the National Lightning Detection Network (NLDN).^27 ,^28 Automatic notices of lightning activity are transmitted by e-mail, text, or cell phone to a predetermined individual. As cell phone reception is rarely available in the wilderness, personal lightning detection devices are an alternative option that does not rely on cellular technology. These devices are about the size of a pager, are easy to carry, and can detect lightning as far away as 75 miles. The device immediately signals the person of lightning activity and its distance by beeps, flashing lights, or a text message. This technology can be used to augment (but not supersede) a lightning safety plan. It should be noted that the available data on the efficacy of this technology is not peer reviewed and are largely based on manufacturer testimonials. Recommendation grade: 2C.

LIGHTNING IN A MOUNTAIN ENVIRONMENT

The panel strongly recommends the avoidance of peaks and ridgelines in the afternoon as thunderstorms are most frequent during this time period.^29 A common safety adage is “up by noon and down by two,” meaning that hikers and climbers should be off peaks and ridgelines by 2:00PM. If caught in a thunderstorm, climbers should tie-off individually as lightning is able to conduct over wet climbing ropes and may affect both climber and belayer. Individuals should discard metal objects such as ski poles or mountaineering axes to avoid contact burns.

Figure 3.Lightning position. Recommendation grade: 1C.

LIGHTNING IN A WATER ENVIRONMENT

This panel recommends that individuals exit the water and seek shelter expeditiously if caught swimming during a lightning storm.When rafting or kayaking, move to shore and away from the water's edge as soon as possible. When boating, seek shelter below deck after locking off the helm.^30 If no shelter is available below, tie into a lifeline. Recommendation grade: 1C.

Injuries and Treatment

TRIAGE AND RESUSCITATION

The mechanism of sudden death from lightning strike is simultaneous cardiac and respiratory arrest. The patho- physiology is classically described as an initial asystolic arrest caused by the simultaneous depolarization of all myocardial cells. Ventricular fibrillation may also be observed.^31 Cardiac automaticity, typically in the form of sinus bradycardia, precedes recovery of the respiratory system. As the medullary respiratory center remains paralyzed despite return of spontaneous circulation (ROSC), a second cardiac arrest may occur if ventilation is not supported. Animal models corroborate this paradigm.^32 Death is rare should a victim survive the initial lightning strike.^33

Reverse triage

As ROSC precedes resolution of respiratory arrest, a patient's ventilation should be supported as soon as possible. This highlights the need for a"reverse triage" system for lightning victims in which priority is initially given to those individuals without vital signs or spontaneous respirations.^31 In instances of multiple lightning casualties, we recommend using a reverse triage strategy. Recommendation grade 1C.

Resuscitation

Victims of lightning strike do not carry residual electrical charge; it is, therefore, safe to resuscitate these individuals immediately should the scene otherwise be deemed safe. Basic and advanced life support algorithms, including trauma when appropriate, remain the standard of care.^34 ,^35 There are numerous case reports of survival with intact neurologic function in lightning victims who received immediate resuscitation; mortality from cardiac arrest is lower in the lightning victims when compared with cardiac arrest in the general population.^31 ,^33 ,^36 ,^37 We recommend following current advanced life support guidelines for lightning victims requiring resuscitation.^34 ,^35 Recommendation grade: 1B.

CARDIOVASCULAR

The effect of a lightning strike on the cardiovascular system is variable ranging from benign electrocardiographic (ECG) changes to sudden death. Cardiovascular collapse is more commonly associated with direct strikes, whereas more transient ECG changes are seen with contact strikes or ground current.^38 Initial cardiovascular effects can include ST elevation, prolongation of the QT interval, cardiomyopathy, atrial fibrillation, and elevated cardiac markers.^38 –^40 Most of these findings resolve within 3 days, although pericarditis may recur several months after the initial injury.^38 Although ST elevation may suggest a localizing vascular lesion, coronary angiography may be normal.^41 In one instance, a victim experienced cardiogenic shock and required an intraaortic balloon pump. However, her cardiac function normalized after 72 hours.^42 It is important to note that delayed-onset symptoms and ECG changes have been reported as far out as 3 days.^38 ,^43 Labile blood pressures and autonomic instability are possible after lightning strikes and may persist for weeks to months.^44 ,^45

Initial cardiac evaluation

Once evacuated, we recommend that high-risk patients (Table),^46 including those experiencing a direct strike or those complaining of chest pain or dyspnea, receive a screening ECG and echocardiography. Recommendation grade: 1C

Cardiac markers

Although elevated cardiac markers are commonly reported after lightning strike, such abnormalities are not typically prognostic and do not correlate with anatomic lesions. Routine screening of cardiac markers, therefore, has limited clinical utility.^38 ,^41 ,^47 Recommendation grade: 2C.

Admission criteria

Patients experiencing a direct strike or those with an abnormal screening ECG or echocardiogram should be monitored with telemetry for a minimum of 24 hours.^38 ,^42 ,^43 ,^48 Recommendation grade: 1C.

Table.High-risk indicators in lightning strike victims

- Suspected direct strike
- Loss of consciousness
- Focal neurologic complaint
- Chest pain or dyspnea
- Major trauma defined by Revised Trauma Scoreo 444
- Cranial burns, leg burns or burns 4 10% TBSA
- Pregnancy

TBSA, total body surface area.

Return precautions

As delayed or recurring cardiac injuries such as pericarditis or cardiomyopathy are possible,^38 discharged patients should be counseled to return should they exhibit new chest pain or dyspnea. Recommendation grade: 1C.

NEUROLOGIC

Neurologic injuries are common after lightning strike and range from the transient and incidental to life threatening. These injuries have been categorized on the basis of symptom onset and duration.^49 As treatment strategies are limited for permanent neurologic injury resulting from lightning strikes, long-term neurorehabilitation is often the sole treatment option for those with permanent disability.^50 ,^51

Transient neurologic symptoms with immediate onset

This group accounts for the majority of neurologic manifestations of lightning injury. These include loss of consciousness, seizure, headache, paresthesia or weakness, confusion, and memory loss.

Keraunoparalysis

Transient paralysis after lightning strike has been documented in numerous case reports and is postulated to result from an overstimulation of the autonomic nervous system, leading to vascular spasm.^33 ,^49 ,^52 Typically, lower limbs are affected more than upper limbs. Signs and symptoms include lack of pulse, pallor or cyanosis, and motor and sensory loss in the affected extremities. Keraunoparalysis typically resolves within several hours. As keraunoparalysis may mimic a pulse- less victim, be vigilant to check a central pulse before starting cardiopulmonary resuscitation.We recommend hospital observation for keraunoparalysis. This phenomenon typically resolves spontaneously but may indicate more serious underlying trauma.^49 Recom- mendation grade: 1C.

Keraunoparalysis can mimic a spinal injury; thus, spinal precautions should be maintained and diagnostic imaging should be performed to rule out spinal cord injury if neurologic deficits persist despite resolution of pallor or pulselessness.^33 Recommendation grade: 1C.

Permanent neurologic symptoms with immediate onset

Permanent neurologic injury can manifest immediately after lightning strike, such as hypoxic encephalopathy resulting from cardiopulmonary arrest.^49 Lightning-induced intracranial hemorrhage may also occur instantly, most commonly affecting the basal ganglia or brainstem; this is believed to be attributable to preferential conduction of electricity through these areas of the brain.^49 ,^53 ,^54 Direct strikes to the head demonstrated higher fatality rates when compared with indirect strikes in one series.^55 Less common immediate-onset permanent neurologic injuries include peripheral nerve lesions, cerebral infarction, and cerebral salt-wasting syndrome.^55 –^57

Delayed neurologic syndromes

A multitude of delayed neurologic syndromes have been reported in victims struck by lightning. However, causal- ity to lightning strike has not been clearly established and the underlying pathophysiology is not yet understood.^54 ,^58 –^60 Progressive myelopathy has been described, resulting in weakness or sensory loss in the weeks to months after initial injury.^49 ,^59 Both animal models and human case studies have demonstrated the highest incidence of damage in the cervical and thoracic regions of the spinal cord.^59 ,^61 We recommend that anyone with delayed neurologic symptoms seek follow-up and treatment recommendations from a neurologist as soon as medically feasible. Recommendation grade: 2C.

Central nervous system injuries associated with secondary trauma and blast effect Any person having been struck by lightning should have a thorough examination for traumatic head injuries. All lightning strike victims with loss of consciousness or a persistently abnormal neurologic examination should receive a computerized tomography scan of the head.^49 ,^62 Recommendation grade: 1C.

DERMATOLOGIC

Lichtenbergfigure

A transient “ferning” or “feathering” pattern known as the Lichtenbergfigure is pathognomonic for lightning strike. It is not a burn, although its pathogenesis remains controversial.^63 This finding generally presents within 1 hour of lightning strike, and resolves in less than 24 hours. No histological change or damage has been found on biopsy, although pigment changes in the deeper layers of the skin may persist.^64 Treatment for these figures is not required, but their presence requires evaluation for other effects of lightning strike. Recommendation grade: 1C.

Burns

Burns associated with lightning injury include linear burns, punctate burns, and full-thickness burns. Linear burns are typically partial-thickness burns that result as sweat vaporizes into steam when lightning travels over the skin (also known as“flashover”). Areas that have heavy sweat concentration such as the underarms and beneath the breasts tend to be most affected.^65 Punctate burns are clustered circular burns believed to be the result of current passing out from the underlying deep tissue. An example is the“tip-toe”sign; these are small (usually < 1 cm) full-thickness burns found at the distal toes or sole of the foot. These burns are thought to result from current exiting the body. Punctate burns can also be caused by water droplets on the skin (from sweat or rain) becoming superheated and turning to steam from the energy of a lightning strike. Larger full-thickness burns are typically found in areas where the skin is in direct contact with synthetic fabric that melts onto skin or a metal object that is heated by the electrical energy of the lightning strike.^66 Full-thickness burns requiring skin grafting are uncommon; only 10% of lightning victims required skin grafting in a case series of 16 patients treated in a burn unit.^67 It is worth noting that the presence of cranial burns predicted a 3-fold increase in mortality in one series, and these patients were twice as likely to undergo cardiac arrest.^33

In limited case series, superficial burns related to lightning that involve less than 20% of total body surface area tend to heal quickly and may be treated with routine burn care.^67 –^69 Recommendation grade: 1C. If caught in a storm, remove metal objects such as watches, belt buckles, and necklaces in an effort to limit contact burns.^66 Recommendation grade: 1C.

EYE

Ocular injuries are common after lightning strike and may affect the anterior and posterior chambers. Damage may result from a number of mechanisms, including passage of current through the lens, blunt and blast trauma, vaso- constriction, or heat. The lens is commonly injured after lightning strike. Cataracts, often bilateral, comprise the majority of these injuries although their exact incidence is not reliably known.^70 Cataracts have been observed to develop between 2 days and 4 years after injury.^70 –^72 Visual prognosis is dependent on the extent of irreversible retinal damage and optic nerve injury as well as cataract formation. Ophthalmology evaluation is essential for all survivors of a high-risk (Table) lightning strike and for any victim who develops vision loss as soon as medically feasible. Recommendation grade: 1C.

EAR

The audiovestibular system is vulnerable to lightning, as it is a low-resistance pathway.^73 Tympanic membrane (TM) rupture was present in more than 60% of subjects in one case series in which 12 of 18 lightning victims had ruptured TMs.^74 Rupture may occur through a combination of blast trauma and electrical injury. Uncomplicated TM rupture usually heals spontaneously and can be managed conservatively. Otorrhea may be a sign of underlying basilar skull fracture and secondary trauma. Sensorineural deafness is also common after lightning strike and is usually transient. However, passage of current through the

temporal bone may cause microhemorrhages and microfractures to the deeper structures of the ear, resulting in permanent hearing loss.^74 Initial evaluation for TM integrity is necessary in all lightning strike victims; follow-up with an otolaryngologist is essential for victims with hearing loss. Recommendation grade: 1C.

PSYCHIATRIC AND NEUROCOGNITIVE

A number of poststrike psychiatric and cognitive dysfunctions are described in the literature.^50 ,^75 These are typically divided into functional or behavioral categories. Functional deficits include abnormalities in memory and concentration including a reduced capacity for problem solving. Behavioral problems include depression, sleep disturbances, emotional lability, and aggressive behavior. These syndromes typically develop in days to weeks after a lightning strike, usually after the individual has returned from the wilderness setting. Victims and their families can be referred to one of several lightning support networks that may provide further coun- seling on the long-term sequelae of lightning injury. (Lightning Strike and Electric ShockSurvivors International, Inc: http://www.lightning-strike.org;e-mail:info@lightning- strike.org; phone: (910) 346-4708.) The lightning strike victim and his or her family should be counseled by primary providers to watch for symptoms of neuropsychiatric dysfunction and should seek specialized care from a mental health professional should such symptoms manifest. Recommendation grade: 1C.

PREGNANCY

Lightning strikes in pregnancy are rare, with only 13 cases reported in the literature.^76 –^81 Among these vic- tims, maternal mortality is zero, although fetal mortality approaches 50%. The fetus is likely at higher risk than the mother because it is surrounded by highly conductive amnioticfluid.^78 In addition to primary electrical injury, lightning strikes have been reported to cause uterine rupture and induction of labor.^82 Pregnant women greater than 20 weeks'gestation who have been struck by lightning should be evacuated to a hospital for lightning-associated injury screening and fetal monitoring. In general, pregnancies less than 20 weeks are not considered viable and do not require fetal monitoring. Recommendation grade: 1C.

DISPOSITION AND EVACUATION

Individuals with high-risk indicators (Table) should be evacuated immediately after the scene is determined to be safe for rescuers. Lower-risk lightning injuries and other casualties should be triaged and evacuated based on their injuries and overall medical condition. Recommendation grade: 1C.

Conclusions

This article provides an updated summary of available evidence for the prevention and treatment of lightning injury. Although numerous case reports have been published since the original practice guidelines were released in 2012, the

summary recommendations remain largely unchanged. Most available data continue to be based on small, retrospective case reports or series because the prospective study of lightning injuries is not logistically and ethically possible. Although the strength of the overall evidence is limited, the authors still believe that many recommendations can be strongly supported [1C] as there is little risk of associated harm. Improved reporting to a national or international data- base could help with future epidemiological studies. Consensus on injury classification systems would also simplify the reporting process and allow data to be more easily combined for future study. (Also see the online Supplementary Evidence Table 2.)

Supplementary tables

Supplementary ACCP Table 1 and Evidence Table 2 are available online at10.1016/j.wem.2014.05.004.

References

1. National Oceanic and Atmospheric Administration. Light- ning Climatology. 2008. Available at: http://www.nssl. noaa.gov/primer/lightning/ltg_climatology.html. Accessed February 28, 2012.
2. Holle R. Annual rates of lightning fatalities by country. 20th Annual International Lightning Detection Confer- ence. Tucson, AZ; April 21–23, 2008. Available at: http://www.vaisala.com/Vaisala%20Documents/Scientific% 20papers/Annual_rates_of_lightning_fatalities_by_country.pdf. Accessed October 29, 2014.
3. Cherington M, Walker J, Boyson M, Glancy R, Hedegaard H, Clark S. Closing the gap on the actual numbers of lightning casualties and deaths. 11th Conference on Applied Climatology. Dallas, TX: American Meteorolog- ical Society; 1999:379–380.
4. Forgey WW, Wilderness Medical Society, Wilderness Medical Society Practice Guidelines for Wilderness Emergency Care. 5th ed. Guilford, CT: Falcon Guide; 2006.
5. Davis C, Engeln A, Johnson E, et al. Wilderness Medical Society practice guidelines for the prevention and treat- ment of lightning injuries. Wilderness Environ Med. 2012;23:260– 269.
6. Guyatt G, Gutterman D, Baumann MH, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force.Chest. 2006;129:174–181.
7. Christian HJ.Global Frequency and Distribution of Light- ning as Observed From Space.American Geophysical Union; 2001.
8. Cherington M, Krider EP, Yarnell PR, Breed DW. A bolt from the blue: lightning strike to the head.Neurology. 1997;48:683– 686.
9. Cherington M, Breed DW, Yarnell PR, Smith WE. Light- ning injuries during snowy conditions.Br J Sports Med. 1998;32:333– 335.
10. National Weather Service. Medical Aspects of Lightning. 2010. Available at: http://www.lightningsafety.noaa.gov/ medical.htm. Accessed February 15,

2012.
11. National Oceanic and Atmospheric Administration. Weather Fatalities. 2011. Available at:http://www.nws. noaa.gov/om/hazstats.shtml. Accessed March 2, 2012.
12. Colorado Avalanche Information Center. Avalanche Acci- dent Statistics. 2011. Available at:http://avalanche.state. co.us/acc/acc_stats.php. Accessed March 2, 2012.
13. Centers for Disease Control and Prevention (CDC), Lightning-associated deaths—United States, 1980–1995. MMWR Morb Mortal Wkly Rep. 1998;47:391– 394.
14. Adekoya N, Nolte KB. Struck-by-lightning deaths in the United States.J Environ Health. 2005;67(45–50):58.
15. Cooper MH, Holle RL. Mechanisms of lightning injury should affect lightning safety messages. 21st Annual International Lightning Detection Conference. Orlando, FL; April 19–20, 2010. Available at:http://www.vaisala.com/ Vaisala%20Documents/Scientific%20papers/11.Cooper,% 20Holle.pdf. Accessed October 29, 2014.
16. Cooper MA. Afifth mechanism of lightning injury.Acad Emerg Med. 2002;9:172– 174.
17. Zafren K, Durrer B, Herry JP, Brugger H, ICAR and UIAA MEDCOM. Lightning injuries: prevention and on- site treatment in mountains and remote areas. Official guidelines of the International Commission for Mountain Emergency Medicine and the Medical Commission of the International Mountaineering and Climbing Federation (ICAR and UIAA MEDCOM).Resuscitation. 2005;65: 369 – 372.
18. Zimmermann C, Cooper MA, Holle RL. Lightning safety guidelines.Ann Emerg Med. 2002;39:660– 664. 19.Cooper MA, Holle R, Lopez R. Recommendations for lightning safety.JAMA. 1999;282:1132– 1133.
19. National Lightning Safety Institute. Personal lightning safety. 2012. Available at:http://www.lightningsafety.com/ nlsi_pls.html. Accessed March 2, 2012.
20. Holle R. Lightning-caused deaths and injuries in the vicinity of vehicles. National Lightning Safety Insti- tute; 2009. Available at:http://www.lightningsafety.com/ nlsi_pls/lightning-caused-deaths-around-vehicles.pdf. Acc- essed October 29, 2014.
21. Bandara K. Lightning hazards: impacts and responses of the public. 18th Annual International Lightning Detection Conference. Helsinki, Finland: Vaisala; 2004.
22. Rakov VA. Lightning protection of structures and per- sonal safety. 2000 International Lightning Detection Conference. Tucson, AZ; November 7–8, 2000. Available at: http://www.w1npp.org/events/2011/2011-F~1/SAFETY/ LIGHTN~1.COM/LIGHTN~4.PDF. Accessed October 29, 2014.
23. Zack F, Hammer U, Klett I, Wegener R. Myocardial injury due to lightning.Int J Legal Med. 1997;110:326– 328.
24. Roeder W. Analysis of short notice outdoor lightning risk reduction and comments on why it should not be taught. Struck by Lightning. American Meteorological Society; 2008.
25. National Weather Service. Lightning risk reduction. 2011. Available at: http://www.lightningsafety.noaa.gov/out doors.htm. Accessed March 2, 2012.

26. Holle RH, Lopez RE, National Severe Storms Laboratory. Overview of Real-Time Lightning Detection Systems and Their Meteorological Uses.Norman, OK; Springfield, VA: U.S. Dept. of Commerce, National Oceanic and Atmospheric Administration, Environmental Research Laborato- ries, National Severe Storms Laboratory; for sale by the National Technical Information Service; 1993.
27. Kithil R. An overview of lightning detection equipment. 2011. Available at: http://www.lightningsafety.com/ nlsi_lhm/overview2002.html. Accessed March 3, 2012.
28. Vogt BJ. Visualizing summertime lightning patterns on Colorado fourteeners.Prof Geogr. 2012;66:41– 57.
29. Becker W.Boating: Lightning Protection.Gainesville, FL: University of Florida; 1992.
30. Taussig HB.“Death”from lightning and the possibility of living again.Am Sci. 1969;57:306– 316.
31. Andrews CDM. Effects of lightning on mammalian tissue. Proceedings of the International Conference of Lightning and Static Electricity. Bath, England; 1989:104.
32. Cooper MA. Lightning injuries: prognostic signs for death. Ann Emerg Med. 1980;9:134– 138.
33. 2005 American Heart Association Guidelines for Cardio- pulmonary Resuscitation and Emergency Cardiovascular Care.Circulation. 2005;112(24 Suppl):IV1–IV203.
34. Driscoll P, Wardrope J. ATLS: past, present, and future. Emerg Med J. 2005;22:2– 3.
35. Courtman SP, Wilson PM, Mok Q. Case report of a 13- year-old struck by lightning. Paediatr Anaesth. 2003; 13:76– 79.
36. Fahmy FS, Brinsden MD, Smith J, Frame JD. Lightning: the multisystem group injuries.J Trauma. 1999;46:937– 940.
37. Lichtenberg R, Dries D, Ward K, Marshall W, Scanlon P. Cardiovascular effects of lightning strikes. J Am Coll Cardiol. 1993;21:531– 536.
38. Jackson SH, Parry DJ. Lightning and the heart.Br Heart J. 1980;43:454– 457.
39. Dronacahrya L, Poudel R. Lightning induced atrialfibril- lation.Kathmandu Univ Med J (KUMJ). 2008;6:514– 515.
40. Saglam H, Yavuz Y, Yurumez Y, Ozkececi G, Kilit C. A case of acute myocardial infarction due to indirect light- ning strike.J Electrocardiol. 2007;40:527– 530.
41. Rivera J, Romero KA, González-Chon O, Uruchurtu E, Márquez MF, Guevara M. Severe stunned myocardium after lightning strike.Crit Care Med. 2007;35:280– 285.
42. Palmer AB. Lightning injury causing prolongation of the Q-T interval.Postgrad Med J. 1987;63:891– 894.
43. Weeramanthri TS, Puddey IB, Beilin LJ. Lightning strike and autonomic failure—coincidence or causally related?J R Soc Med. 1991;84:687– 688.
44. Grubb BP, Karabin B. New onset postural tachycardia syndrome following lightning injury.Pacing Clin Electro- physiol. 2007;30:1036– 1038.
45. Champion HR, Sacco WJ, Copes WS, Gann DS, Gennar- elli TA, Flanagan ME. A revision of the Trauma Score.J Trauma. 1989;29:623– 629.

46. Alyan O, Ozdemir O, Tufekcioglu O, Geyik B, Aras D, Demirkan D. Myocardial injury due to lightning strike—a case report.Angiology. 2006;57:219– 223.
47. Dundon BK, Puri R, Leong DP, Worthley MI. Takotsubo cardiomyopathy following lightning strike.Emerg Med J. 2008;25:460– 461.
48. Cherington M. Spectrum of neurologic complications of lightning injuries.NeuroRehabilitation. 2005;20:3– 8.
49. Cherington M. Neurorehabilitation of the multifaceted and complicated neurologic problems associated with lightning and electrical injuries.NeuroRehabilitation. 2005;20:1– 2.
50. Yarnell PR. Neurorehabilitation of cerebral disorders following lightning and electrical trauma.NeuroRehabili- tation. 2005;20:15– 18.
51. ten Duis HJ, Klasen HJ, Reenalda PE. Keraunoparalysis, a ‘specific’lightning injury.Burns. 1985;l2:54– 57. 53.Cherington M. Lightning injuries. Ann Emerg Med. 1995;25:517– 519.
52. Cherington M. Central nervous system complications of lightning and electrical injuries.Semin Neurol. 1995;15: 233 – 240.
53. Kleinschmidt-DeMasters BK. Neuropathology of lightning-strike injuries.Semin Neurol. 1995;15:323– 328.
54. Emet M, Caner I, Cakir M, Aslan S, Cakir Z. Lightning injury may cause abrupt cerebral salt wasting syndrome. Am J Emerg Med. 2010;28(640):e1– e3.
55. Frayne JH, Gilligan BS. Neurological sequelae of light- ning stroke.Clin Exp Neurol. 1987;24:195– 200.
56. Belsole RJ, Smith AA. Reflex sympathetic dystrophy: “gate”closed by lightning.J Hand Surg Am. 1990;15:523.
57. Davidson GS, Deck JH. Delayed myelopathy following lightning strike: a demyelinating process. Acta Neuro- pathol. 1988;77:104– 108.
58. O’Brien CF. Involuntary movement disorders following light- ning and electrical injuries.Semin Neurol. 1995;15:263– 267.
59. Lakshminarayanan S, Chokroverty S, Eshkar N, Grewal R. The spinal cord in lightning injury: a report of two cases. J Neurol Sci. 2009;276:199– 201.
60. Aslan S, Yilmaz S, Karcioglu O. Lightning: an unusual cause of cerebellar infarction.Emerg Med J. 2004;21:750– 751.
61. Cherington M, McDonough G, Olson S, Russon R, Yarnell PR. Lichtenbergfigures and lightning: case reports and review of the literature.Cutis. 2007;80:141– 143.
62. Resnik BI, Wetli CV. Lichtenbergfigures.Am J Forensic Med Pathol. 1996;17:99– 102.
63. Cooper MA. Emergent care of lightning and electrical injuries.Semin Neurol. 1995;15:268– 278.
64. Herrero F, García-Morato V, Salinas V, Alonso S. An unusual case of lightning injury: a melted silver necklace causing a full thickness linear burn.Burns. 1995;21:308– 309.
65. Maghsoudi H, Adyani Y, Ahmadian N. Electrical and lightning injuries.J Burn Care Res. 2007;28:255– 261.
66. Matthews MS, Fahey AL. Plastic surgical considerations in lightning injuries.Ann Plast Surg. 1997;39:561– 565.

67. Selvaggi G, Monstrey S, Van Landuyt K, Hamdi M, Blondeel P. Rehabilitation of burn injured patients follow- ing lightning and electrical trauma.NeuroRehabilitation. 2005;20:35– 42.
68. Sommer LK, Lund-Andersen H. Skin burn, bilateral iridocyclitis and amnesia following a lightning injury. Acta Ophthalmol Scand. 2004;82:596– 598.
69. Espaillat A, Janigian R Jr, To K. Cataracts, bilateral macular holes, and rhegmatogenous retinal detachment induced by lightning.Am J Ophthalmol. 1999;127:216– 217.
70. Cazabon S, Dabbs TR. Lightning-induced cataract.Eye (Lond). 2000;14(Pt 6):903– 904.
71. Jones DT, Ogren FP, Roh LH, Moore GF. Lightning and its effects on the auditory system. Laryngoscope. 1991;101:830– 834.
72. GluncićI, Roje Z, GluncićV, Poljak K. Ear injuries caused by lightning: report of 18 cases.J Laryngol Otol. 2001;115:4– 8.
73. Primeau M, Engelstatter GH, Bares KK. Behavioral consequences of lightning and electrical injury. Semin Neurol. 1995;15:279– 285.
74. Pierce MR, Henderson RA, Mitchell JM. Cardiopulmo- nary arrest secondary to lightning injury in a pregnant woman.Ann Emerg Med. 1986;15:597– 599.
75. García Gutiérrez JJ, Meléndez J, Torrero JV, Obregón O, Uceda M, Gabilondo FJ. Lightning injuries in a pregnant woman: a case report and review of the literature.Burns. 2005;31:1045– 1049.
76. Flannery DB, Wiles H. Follow-up of a survivor of intra- uterine lightning exposure. Am J Obstet Gynecol. 1982;142:238– 239.
77. Weinstein L. Lightning: a rare cause of intrauterine death with maternal survival.South Med J. 1979;72:632– 633.
78. Chan YF, Sivasamboo R. Lightning accidents in preg- nancy.J Obstet Gynaecol Br Commonw. 1972;79:761– 762.
79. Rees WD. Pregnant Woman Struck by Lightning.BMJ. 1965;1:103– 104.
80. Guha-Ray DK. Fetal death at term due to lightning.Am J Obstet Gynecol. 1979;134:103– 105.

Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Heat Illness: 2019 Update

Grant S. Lipman, MD1 ; Flavio G. Gaudio, MD2 ; Kurt P. Eifling, MD3 ; Mark A. Ellis, MD4 ; Edward M. Otten, MD5 ; Colin K. Grissom, MD6 (1) Department of Emergency Medicine, Stanford University School of Medicine, Stanford, CA; (2) Department of Emergency Medicine, New York- Presbyterian Hospital/Weill Cornell Medical College, New York, NY;3 Division of Emergency Medicine, Larner College of Medicine at the University of Vermont, Burlington, VT;4 Department of Emergency Medicine, Spartanburg Regional Healthcare System, Spartanburg, SC; (5) Department of Emergency Medicine, University of Cincinnati, Cincinnati, OH; (6) Pulmonary and Critical Care Division, Intermountain Medical Center and the University of Utah, Salt Lake City, UT

The Wilderness Medical Society convened an expert panel in 2011 to develop a set of evidence-based guidelines for the recognition, prevention, and treatment of heat illness. We present a review of the classifications, pathophysiology, and evidence-based guidelines for planning and preventive measures, as well as best practice recommendations for both field- and hospital-based therapeutic management of heat illness. These recommendations are graded based on the quality of supporting evidence and balance the benefits and risks or burdens for each modality.

This is an updated version of the original Wilderness Medical Society Practice Guidelines for the Treatment and Prevention of Heat-Related Illness published in 2013.

Keywords:heat stroke, hyperthermia, prevention, recognition, treatment

Introduction

Heat illness is a common occurrence worldwide. The European heat wave of 2003 resulted in at least 70, fatalities,[1] and in the last decade the United States averaged over 600 deaths annually associated with excessive heat exposure.[2] Currently, heat illness is the leading cause of morbidity and mortality among US high school athletes.[3] Heat stroke mortality approaches 10%4 and when presenting with hypotension increases to 33%.[5] Outcome is directly attributed to both the magnitude and duration of hyperthermia,[6,8] making early recognition and treatment a priority. The Wilderness Medical Society convened an expert

panel to develop a set of practice guidelines for the recognition, prevention, and treatment of heat illness. We present a review of the classifications, pathophysiology, and evidence-based guidelines for planning and preventive measures, as well as best practice recommendations for both field and hospital-based therapeutic management of heat illness. Although the spectrum of heat illness is discussed, this practice group's focus was on the exploration of exertional heat stroke (EHS), which is synonymous with the term "heat stroke" in this article unless otherwise specified.

Methods

Specialists in emergency medicine, primary care, and critical care from both civilian and military backgrounds were chosen based on their clinical or research experience. In 2011,[9] and for subsequent practice guideline updates,[10] relevant articles were identified through the PubMed database using the following key words: hyperthermia, heat stroke, heat illness, heat syncope, and heat exhaustion. This was supplemented by a hand search of articles from references in the initial PubMed search. Studies in these categories, including randomized con- trolled trials, observational studies, and case series, were reviewed. Abstract-only reports were not included. Conclusions from review articles were cited to provide background information but were not considered in the formulation of recommendation grades. The panel used a consensus approach to develop recommendations for the recognition and management of heat illness, with level of evidence assigned according to methodology stipulated by the American College of Chest Physicians for grading of evidence and recommendations (Supplementary Table 1). These recommendations are graded based on the quality of supporting evidence and balance between the benefits and risks or burdens for each modality or intervention.

Definition of heat illness

Heat illness can be manifested as a spectrum of disease from minor to severe, such as heat cramps, heat syncope, heat exhaustion, and life-threatening heat stroke. Hyperthermia is a deviation above the body's normal physiologic setpoint and should be considered separately from the concept of heat illness. It is an expected physiologic response when core body temperature rises as a result of exposure to elevated ambient temperatures or when internally generated heat from muscular activity accumulates faster than it can be dissipated. Hyperthermia is a natural outcome of exertion, and increased metabolic rate has been shown to be the most important factor in elevation of body temperature.[11] Studies of asymptomatic runners have found 15 to 56% with core temperatures 40 ℃ and 11%42 ℃.[12,13] As such, absolute temperature thresholds alone should not be routinely applied to asymptomatic individuals as a pathologic indicator of heat illness.

As with any syndrome, the terms used to define heat illness do not necessarily confer direct cause and effect but rather a strong contextual association that may have descriptive, prognostic, and epidemiological merit, if not pathophysiologic precision.14,15 Heat cramps were initially defined in the 1930s to describe the clinical phenomenon of involuntary diffuse large-muscle contractions associated with exertion in hot environments.16 These heat cramps are likely distinct from the focal muscle cramping in an athlete during repetitive exercises.[17] Dehydration and electrolyte disturbances have been associated with heat cramps,[18] and isotonic rehydration has been found to be restorative.[16] Heat edema is a benign self-limiting condition. Interstitial fluid accumulates in dependent extremities as a result of hydrostatic pressure, vascular leak, and cutaneous vasodilation. Heat syncope refers to a multifactorial syndrome involving transient loss of consciousness in the context of heat exposure with a relatively rapid return to normal function and baseline. Contributing factors may include peripheral vasodilation, orthostatic pooling of blood, prolonged standing, advanced age, dehydration, and coexisting medical conditions such as ischemic heart disease that reduces cardiac output. Although syncope can occur in both milder and more severe forms of heat illness, heat syncope generally refers to a more benign clinical condition that should resolve with rest and possibly rehydration at comfortable ambient temperatures.[19,21] Syncope may also occur during exertion from impaired baroreceptor reflex and lower extremity venous pooling in the absence of hyperthermia or dehydration, a syndrome known as exercise-associated collapse.[19] Heat exhaustion results from exposure to high ambient temperature or strenuous exertion. It manifests as a constellation of symptoms that range from uncomfortable to debilitating[6,21] and may limit continuation of exercise in the heat.[6] Symptoms are variable and may include weakness, fatigue, thirst, headache, nausea, dizziness, and muscle aches.[21] This mild to moderate heat illness may progress to heat stroke if left untreated or unrecognized in a hot environment, although heat stoke does occur as a fulminant illness without preceding heat exhaustion. Heat stroke is traditionally defined as a core temperature above 40 ℃ (104 ℉) with central nervous system involvement (eg, encephalopathy, seizures, or coma). Heat stroke is generally divided into 2 categories: classic heat stroke resulting from passive exposure to high environmental temperatures and EHS resulting from pathologic (non-physiologic) hyperthermia during strenuous exercise (Table 1).[22]

Table 1.Categories of heat illness |Condition|Definition| |---|---| |Hyperthermia|A rise in body temperature above the hypothalamic set point when heat-dissipating mechanisms are impaired (by clothing or insulation, drugs, or disease) or over-whelmed by external (environmental) or internal (metabolic) heat production.| |Heat edema|Dependent extremity swelling due to interstitial fluid pooling.| |Heat cramps|Exercise-associated painful involuntary muscle contractions during or immediately after exercise.| |Heat syncope|Transient loss of consciousness with spontaneous return to normal mentation.| |Heat exhaustion|Mild to moderate heat illness due to exposure to high environmental heat or strenuous physical exercise; signs and symptoms include intense thirst, weakness, discomfort, anxiety, dizziness, syncope; core temperature may be normal or slightly elevated >37 ℃ (98.6 ℉) but<40 ℃="" (104="" ℉).|="" |heat="" stroke|severe="" heat="" illness="" characterized="" by="" a="" core="" temperature="">40 ℃ (104 ℉) and central

nervous system abnormalities such as altered mental status (encephalopathy), seizure, or coma resulting from passive exposure to environmental heat (classic heat stroke) or strenuous exercise (exertional heat stroke).|

Heat dissipation and pathophysiology

Heat loss is controlled by peripheral centers in the skin and organs and the central nervous system via the hypo- thalamus, with a greater cooling response to temperature elevation via central sensors.[23] A temperature gradient exists between the body core and skin that promotes heat dissipation when core temperature is higher than surface temperature. A rise in blood temperature by less than 1℃ triggers hypothalamic thermoregulation to increase blood flow to the skin by up to 8 L/min via sympathetic cutaneous vasodilation, with sixfold blood flow increases to the forearms and the arteriovenous anastomotic microvascular structures deep to the glabrous skin areas, with their unique intrinsic heat transfer capabilities.[24,25] As blood is shunted to the periphery to facilitate heat loss, renal and splanchnic perfusion is reduced by 30%.[24] However, when the core temperature increases during exercise and skin temperature also rises as a result of the environment or internal heat production, heat dissipation is reduced. Similarly, when the body's metabolic heat production outpaces heat transfer, core temperature rises, and heat illness can occur.[26] Heat stroke occurs when internal core temperatures rise above a critical level, leading to a cascade of cellular and systemic responses. These responses include thermoregulatory dysfunction,an acute phase response, and a heat shock protein response. The acute phase response to heat stress involves an inflammatory reaction of interleukins, cytokines, and proteins that progresses in a sequence similar to that seen in sepsis. It is theorized that an exaggerated acute phase and inflammatory response mark the progression from heat stress to heat stroke, possibly incited by the hypoperfused gastrointestinal tract.[22] Increased mucosal permeability from inflammatory mediators allows endotoxins from the gut to enter the systemic circulation. This combination of endotoxemia and cascade of inflammatory cytokines leads to alterations in the microcirculation, further endothelial and tissue injury, and impaired thermoregulation, thus precipitating heat stroke and hypotension. An overlapping hypothesis presupposes that at a similar threshold temperature, the expression of protective heat-shock proteins is altered, decreasing their ability to prevent thermal denaturation of structural proteins and enzymes that start to fail at a cellular level, with ensuing end organ dysfunction.[22,27] At critical levels of hyperthermia, heat causes direct tissue injury and death via apoptosis or necrosis, with the severity of injury dictated by both the level and duration of thermal stress.[28,29] This complex constellation of overlapping events leads to thermoregulatory failure, heat stroke, and circulatory shock.

Prevention and planning

The proverb that *"an ounce of prevention is worth a pound of cure"* is especially apt in light of the potentially fatal nature of heat illness. Deliberate strategies for prevention should be included when planning for activities with a credible risk. Structured risk assessments can be built and validated for population-level use,[30,31] or the practitioner considering a particular scenario should consider the risk incurred by the individual participant's factors including physiology, the environment, and the planned athletic activity.[32]

INDIVIDUAL FACTORS

Any condition that limits heat loss through the skin may lead to heat retention, including hypohydrosis, extensive scars, and diminished cardiopulmonary reserve at the extremes of age. Small studies have linked acute sunburn with impaired sweating, which persisted for 7 d, considerably longer than the associated pain and erythema.[33] Impaired sweating is a risk factor for heat accumulation, but otherwise the risks of sunburn are of indeterminate clinical significance.

Certain drugs can predispose individuals to heat injury by 2 primary pathways, increased heat production resulting from drug actions and compromised function of thermoregulatory centers (Table 2).[35] Moderate caffeine intake appears to have no detrimental effect.[18] A large prospective study of military recruits found a significantly increased risk of heat illness among those who were obese or overweight compared with fitter individuals.[37]

Heat acclimatization, as induced by 1 to 2 h of heat-exposed exertion per day over 10 to 14 d, results in reproducible adaptations that increase the body's ability to tolerate and divest heat.[38,40] These adaptations may persist for up to a month.[41,42] Evidence suggests that a bout of heat stroke may acutely reset these thermoregulatory adaptations and cause elevated risk for subsequent heat injury for months after the initial event,[43] although case reports indicate that heat tolerance can be recovered fully.[44,45] Individuals with high levels of cardiopulmonary fitness tolerate more activity in heat-strained conditions and acclimatize to heat more rapidly because they have increased sweat volumes and higher subjective tolerance for activity when hyperthermic.

The most readily modifiable physiologic risk factor is hydration status. Although endurance athletes may comfortably tolerate weight losses of 3 to 4% during events,[46,47] fluid losses that result in a 2 to 3% decrease in body weight correlate with greater core temperatures at a given work load in the heat.[39,48,49] Dehydration has been found to increase physiologic strain, decrease sweat rates, increase perceived exertion, and increased core temperatures.[23,50]

Hyperhydration before activity has not been shown to have a significant effect on heat tolerance, nor has active body cooling before activity.[52] One investigation on the effect of sex as a risk factor on thermal recovery was confounded by body mass index differences, such that no conclusion could be reached.[53] The luteal phase of the menstrual cycle, which is associated with increased core temperatures, does not appear to induce heat intolerance in women on oral

contraceptive pills.[54] The physiology of pediatric and elderly populations differs enough from healthy adults to warrant special considerations that are outside this panel's scope but are discussed at length elsewhere.[55,57]

Recommendations.

1. Screen for significant pre-existing medical conditions (1B).
2. Minimize use of medications that could limit the thermoregulatory response (1C).
3. Recognize that an overweight body habitus is associated with greater risk of heat illness (1B).
4. Promote regular aerobic activity before heat exposure (1C).
5. Allow for acclimatization with 1 to 2 h per d of heat-exposed exertion for at least 8 days (1C).
6. Ensure euhydration before activity (1B).
7. Ensure ongoing rehydration with a "drink to thirst" approach sufficient to prevent >2% loss of body weight (1B).
8. Consider history of heat injury as a reversible risk factor for recurrence (1C).

Table 2. Medications and drugs that may contribute to heat illness

- Alcohol
- Alpha adrenergics
- Amphetamines
- Anticholinergics
- Antihistamines
- Antipsychotics
- Benzodiazepines
- Beta blockers
- Calcium channel blockers
- Clopidogrel
- Cocaine
- Diuretics
- Laxatives
- Neuroleptics
- Phenothiazines
- Thyroid agonists
- Tricyclic antidepressants

ENVIRONMENTAL CONSIDERATIONS

The body and the environment exchange heat through several mechanisms: conduction (heat transfer from the body to the surrounding environment along a temperature gradient by direct contact), evaporation (heat transfer from the body to sweat, resulting in transition of water from the liquid to vapor phase), thermal radiation (infrared rays given off by any mass as a function of the temperature of that mass), or convection (transfer of heat from the body to free fluids or gas moving across the skin surface).

As the environmental temperature increases, the body will eventually incur a net heat gain through conductive, convective, and radiative processes, leaving evaporation as the only cooling mechanism. The vaporization of 1.7 mL of sweat consumes 1 kcal of heat[58]; however, evaporative cooling is less effective in highly humid environments, which have a lower water vapor pressure difference between the sweat on the skin and the water in the surround- ing air. The wet-bulb globe temperature index (WBGT) is a composite index of temperature, humidity, and solar radiation that expresses the total thermal strain that an individual experiences. A series of WBGT values can be designated as cautionary warnings and triggers to activate guidelines for rehydration, active cooling, and limitations (or even cancellation) of physical activity.[34] An alternative to the WBGT that is more readily available is the heat index, which is a measure of the contribution that high temperature and high humidity (expressed as either relative humidity or dew point temperature) make in reducing the body's ability to cool itself. Although the wet bulb globe temperature is a metric likely not readily available to individual medical practitioners, its current use by military,[59] occupational,[60] and civilian groups[26,55] makes it the standard when discussing environmental thermal strain and choosing activity levels for ambient conditions. Guidelines that correlate the heat index with the risk of heat injury and outline parameters for limiting physical activity are readily available.[61]

Recommendations

1. WBGT should be used for the assessment of heat risk (1A).

ACTIVITY CONSIDERATIONS

The metabolic thermal output of an activity is the product of its intensity and duration. The accumulation of heat by the body is tempered in some circumstances by an activity that can enhance heat transfer with the environment (eg, water convection on a swimmer or wind past a cyclist). Occupational,[60,62] military,[59] and medical[24] guidelines recommend breaks in proportion to metabolic demand and ambient conditions, but there are few studies examining how to optimize the dosing of breaks.

Recommendations

1. Consider which mechanisms of heat accumulation or dissipation are dominant during an activity, and consider heat loss as a key feature of breaks (1C).

CLOTHING AND EQUIPMENT

Clothing or other equipment worn during an activity may limit or enhance the body's thermoregulatory efficiency. Of particular importance is equipment that occludes regions of skin, resulting in compromise of evaporative, convective, radiative, or conductive heat transfer. For example, the American football uniform prevents full heat exchange across much of the torso and head and can therefore contribute to heat accumulation,[63] similar to military helmets and body armor.64 Preventing heat exchange may be protective, as in the case of firefighting gear that prevents heat in a superheated environment from entering the body by

conduction or radiation. Sports medicine guidelines and military occupational guidelines have set examples of systematic reductions in clothing and equipment based on WBGT thresholds.[26]

Recommendations

1. Clothing and equipment for a given activity should be evaluated and modified as needed to optimize evaporative, convective, conductive, and radiative heat exchange or isolation (1C).

Field treatment

Optimal field management of heat illness may be challenging because of resource limitations or extreme settings. The ideal treatment, as emphasized in the previous section, is prevention via avoiding high-exertion activities in exposed or hot areas. The method and aggressiveness of cooling in the field depend on the type of heat illness encountered (Table 3). Regardless of the underlying cause, removal from the heat and rapid cooling is critical because the extent of morbidity is directly relate to both to the degree and duration of hyperthermia.[8,37,66,69] All treatment in the field is first directed to stabilization of the patient's airway, breathing, and circulation before proceeding to more specific cooling therapy. If no life-threatening complications exist, the implementation of on-site cooling before evacuation should be implemented (Figure 1).[26] In transitioning patient care to emergency medical service providers, it is important to communicate any cooling techniques begun in the field and to emphasize continued cooling of the patient by best available means en route to the destination.

MINOR HEAT ILLNESS TREATMENT

There is scant evidence supporting treatments of minor and moderate heat illness. Most treatments are anecdotal but effective and generalizable from the evidence-based treatment for more severe forms of heat illness (Table 3).

Heat cramps, which are historically described as generalized,[16] differ from the focal exercise-associated muscle cramps seen in endurance athletes. Heat cramps are relieved with oral salt solutions or electrolyte replacement that may be isotonic or hypertonic,[16] compared with exercise-associated muscle cramps that occur with neuromuscular fatigue and are relieved with passive stretch- ing.[70] Heat edema is reversed by extremity elevation or wearing of compression stockings. Diuretics are ineffective and may worsen volume depletion.[71] Heat syncope by definition is self-limiting. After consideration of other medical causes of syncope or resultant trauma from a fall, treatment consists of ensuring replacement of vascular volume with isotonic oral fluids and rest in a cool environment.19 Individuals at risk for heat syncope should move often and flex their larger leg muscles to prevent peripheral pooling of blood from cutaneous vasodilation. An individual with heat syncope is likely under acclimatized to the heat, and caution is warranted before immediate return to regular activity.

Table 3. Heat illness treatments

Severity of heat-related illness	Diagnosis	Treatment
Mild	Heat cramps	Oral isotonic or hypertonic fluid replacement
	Heat edema	Extremity elevation; Compression stockings
Moderate	Heat syncope	Remove from heat source; Passive cooling; Oral isotonic or hypertonic fluid hydration
	Heat exhaustion	Remove from heat source; Evaporative, convective, or conductive cooling; Oral or intravenous isotonic or hypertonic fluid hydration
Severe	Heat stroke	Remove from heat source; Supportive care of airway, breathing, and circulation; Cold water immersion; Whole-body conductive cooling; Intravenous hydration[a]; Evacuation[b]

a Intravenous hydration with isotonic (0.9% sodium chloride) or hypertonic (D5NS) fluids, with 3% sodium chloride indicated if concern for exercise-associated hyponatremia as cause of encephalopathy. b Initiate emergency medical services if unable to rapidly cool patient, prolonged encephalopathy, or concern of multiorgan dysfunction.

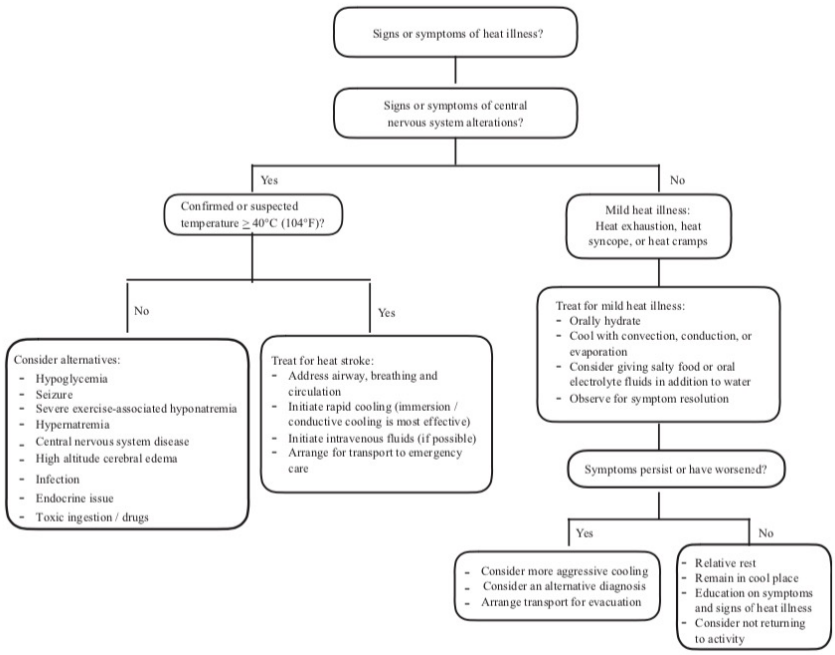

Figure 1. Heat illness treatment algorithm

Heat exhaustion, like heat stroke, results from a combination of both cardiovascular and thermal strain, and various forms of rest and whole-body cooling are dictated by severity of symptoms. Mild cases generally resolve with removing the patient from the hot environment, cessation of physical activity, and rehydration with oral isotonic fluids. Severe heat exhaustion typically has more

pronounced volume depletion and may require intravenous replacement of fluids as well as conductive and convective cooling. Because heat exhaustion can lead to cessation of physical activity and collapse,[6] it is important to actively reverse the process of heat exhaustion, which can progress to heat stroke without proper cooling techniques. Case reports have linked heat exhaustion or acute heat stress to precipitating electrocardiogram changes, symptomatic arrhythmias, and cardiac arrest with features of underlying Brugada syndrome.[72]

Recommendations

1. Heat syncope patients whose event is recurrent and inconsistent with exercise-associated collapse or other clear explanation should be referred for further cardiology diagnostics (2C).

TEMPERATURE MEASUREMENT

When possible, obtaining an accurate body temperature is an important diagnostic step in differentiating heat stroke from less severe heat illness. Rectal temperature is widely considered the standard field measurement,[25] because it is a reliable and practical measure of core temperature that is more accurate than temporal, axillary, oral, or aural thermometry.[74,75] Esophageal and ingestible thermistors have been validated and provide a more accurate measurement of core temperature but are impractical in the wilderness setting. Rectal temperature measurement carries implicit challenges in maintaining patient privacy and hygiene, so initial assessment and aggressive cooling should be implemented based on the clinical suspicion, regardless of the degree of hyperthermia or mode of measurement.

Recommendations

1. When available, rectal temperature should be considered the most accurate measurement of core hyperthermia compared to axillary, oral, or aural thermometry (1B).
2. In a hyperthermic individual with an altered sensorium, the initiation of empiric cooling for heat stroke should not be delayed by a measurement value that may be below the diagnostic threshold of 40℃ (1B).

PASSIVE COOLING

Simple measures can be easily taken to reduce the patient’s exposure to heat. Moving the victim into the shade can externally decrease the ambient temperature; however, this is most effective when temperatures are <20 ℃="" (68="" ℉).67</sup> Conduction of heat from the ground can be decreased by placing the victim on cool ground, if available, or on an insulating barrier such as sleeping pad or sleeping bag. Loosen- ing or removing any tight-fitting clothing to optimize air circulation aids in convective heat exchange.[76]

Recommendation

1. Passive cooling measures should be used to minimize thermal strain and maximize heat loss (1C).

HYDRATION

Euhydration is an important factor in reducing hyperthermia.[76,77] Body water losses through increased sweat rates, vomiting, diarrhea, or insufficient oral intake increase the risk of potential heat injury resulting from poor hydration status. Dehydration predisposes patients to hyperthermia by decreasing sweat rates and increases thermal strain at a given temperature.[50 ,77,79] Oral and intravenous hydration have been shown to be equally effective in replenishing water deficiencies related to heat stress.[77]

Recommendations

1. Dehydration should be minimized in heat illness (1C).

Optimizing hydration in heat stroke decreases both the cardiovascular and thermal strain. In a heat stroke victim with altered mental status and risk of seizure, the intravenous route minimizes aspiration risk and subsequent airway compromise. Few data exist on the optimal intravenous fluid type and amount relating specifically to heat illness. Because those with EHS may be volume depleted from insensible fluid losses, the reasonable choices of replenishment are 1 to 2 L of isotonic (0.9% normal saline or normal saline with 5% dextrose) fluids. Because heat stroke can occur in both euhydrated and dehydrated individuals, care should be taken to not over- hydrate patients (especially those with coronary comorbidity) because this may increase the risk of pulmonary edema.[79] Any effort to provide hydration in suspected heat stroke should not delay rapid whole-body cooling.[82] If intravenous hydration is provided, field monitoring of blood pressure, heart rate, lightening of urine color, and increase in urine output can help guide patient response and fluid status.

Recommendation

1. Intravenous fluids should be used for rehydration in EHS (1B).

Symptomatic exercise-associated hyponatremia may present similarly to heat exhaustion,[21] and the presence of altered mental status (eg, obtundation, coma, or seizures) without other explanation, such as hypoglycemia or trauma, in the absence of hyperthermia may indicate severe exercise-associated hyponatremia. This is a critical illness that ideally should be confirmed with serum sodium measurement, but in the absence of point-of-care testing the patient should empirically be resuscitated with up to 3, 100 mL 3% sodium chloride boluses given every 10 min, or until resolution of altered mental status, with rapid transfer to a medical facility.[83]

COLD WATER IMMERSION THERAPY

Cold water immersion therapy is the optimal field treatment to achieve rapid temperature reduction below critical levels in heat stroke. Immersion is a conductive method of cooling that takes advantage of water's high thermal conductivity, which is 24 times greater than that of air,[84] and the high thermal gradient that exists between cold water and skin,[85] which translates into a greater capacity for heat transfer. The colder the water, the faster the rate of cooling.[86] The theoretical concern that cold-water immersion causes peripheral vasoconstriction and shivering that slow cooling, or may even increase the core temperature, is a prevalent misconception, possibly stemming from a misinterpretation of the "Currie response."[87] This 18th century observation found an increase in the core temperature of shivering normothermic individuals by 0.1 to 0.2 ℃. Although shivering has been observed in immersions lasting longer than 10 min in healthy volunteers,[88,89] such shivering may be less problematic in actual heat stroke patients with failing thermoregulation.[90] In addition, the hindrance of cooling EHS by heat-generating shivering has been physiologically refuted.[86,91] Cold-water immersion is achieved by removing insulating clothes and equipment and submersing the patient's trunk and extremities in a bath of cold water or tub of ice water. Alternatively, water may be applied onto a patient covered in crushed ice and lying on a plastic sheet or tarp with its sides folded upright to keep the slurry in place ("tarp taco").[92] Ice water cooling has been shown to be twice as rapid in reducing core temperature as covering the body in soaked towels to enhance evaporative cooling (0.20 ℃-min^{-1} vs 0.11 ℃-min^{-1}).[66] In the field, using a natural body of water such as a stream, pond, river, or lake may be another treatment option. Special care should be taken to protect against currents and to ensure the head does not go underwater and the airway is protected; the patient should never be left alone owing to risk of aspiration and drowning. In lieu of a cold-water source, repeated dousing with cold water or snow, if available, is encouraged. Multiple military studies on immersion cooling of comparatively young and healthy EHS patients boast a 0% fatality rate,[93] strongly supporting that rapid treatment with this cooling modality has the best outcomes.

Recommendation

1. Cold water immersion is the optimal cooling method for heat stroke (1A).

EVAPORATIVE COOLING

If immersion or conductive cooling is unavailable, evaporative cooling measures should be initiated by loosening or removing clothing and dousing the patient with cold water to maximize the watervapor skin interface.[94] Convection is then facilitated with air movement by fanning. Cooling by evaporation plus convection has been studied predominantly in patients with non-EHS,[95] with mean cooling times of 40 to 68 min.[91,96] Limited studies on traditional evaporative cooling have been done with EHS or heat exhaustion, with reported cooling rates half as fast as immersion cooling.[97]

Recommendations

Evaporative or convective cooling can be considered as adjunct cooling methods if cold water immersion is unavailable (1C).

CHEMICAL COLD PACKS/ICE PACKS

There is a traditional advocacy for the use of ice packs or chemical cold packs strategically applied to the skin covering the neck, axillae, and groin to cool blood flow passing in the major vessels as an adjunctive cooling measure.[98] Limited studies show no benefit in heat reduction with ice packs or chemical cold packs applied to these areas.[99] Ice packs have been found to have greater cooling capacity than chemical cold packs,[101</su> and if used, are most efficacious when wet and covering the entire body (to optimize conductive cooling).[99] A small translational study applied chemical cold packs to the glabrous skin of the palms, soles, and cheeks and found twice the cooling rate over traditional major vascular locations,[102] using the high-capacity blood flow of the subcutaneous arteriovenous anastomoses.

Recommendations

1. Ice packs should be applied to cover the entire body (1C).
2. If chemical cold packs are used, they should be applied to the cheeks, palms, and soles rather than the skin covering the major vessels (1C).

ANTIPYRETICS

As clinicians, we generally treat elevated temperatures with antipyretics. This class of medications, such as ibuprofen and aspirin, works by inhibiting prostaglandin formation, and acetaminophen lowers the thermoregulatory setpoint.[103] Although this may be elevated in infectious causes of hyperthermia, this is not the case in exercise-induced hyperthermia. Antipyretic drugs are ineffectual and should be avoided.[104]

Recommendation.

1. Antipyretics should be avoided in heat illness (2B).

Hospital treatment

Generally, patients with heat stroke should be transported to a medical facility capable of critical care management of patients with multiple organ failure. Exceptions to this guideline have included soldiers or athletes treated in the field with ice-water immersion immediately at the onset of EHS with complete resolution of symptoms and subsequent observation in a medical station or infirmary.[106,107] In a hospital setting, the primary goals of treatment for heat stroke are rapidly lowering core body temperature and supporting organ system function[22] because patients may develop multiple organ failure with shock, acute respiratory failure, acute kidney injury, disseminated intravascular coagulopathy, and intestinal ischemia. Depending on the patient's clinical status, supportive treatment may include administering supplemental oxygen, performing intubation

and mechanical ventilation, establishing adequate intravascular access, restoring intravascular volume with intravenous isotonic crystalloid solution, placing a bladder catheter to monitor urine output, and initiating vasopressors to support blood pressure (after adequate volume resuscitation). The evidence on different cooling methods has involved a heterogeneous range of subjects experiencing EHS or classic heat stroke. Of the studies comparing different cooling methods, those involving randomized trials generally have been performed on healthy volunteers with exercise-induced hyperthermia and have enrolled relatively few subjects. The remaining studies on treating heat stroke patients have for the most part been case series reports or nonrandomized comparisons of treatment methods, with considerable variations in the base- line characteristics of subjects from one study to the next. Such heterogeneity and variation have reduced the comparative conclusions that can be drawn. Despite this limitation, the historical record has promoted 2 methods of cooling in a hospital setting: 1) conductive cooling via cold water immersion of the patient; and 2) evaporative and convective cooling via the application of sprayed water and forced air currents over the body.

CONDUCTIVE COOLING

Cold water immersion is safe and effective for young, athletic patients with EHS. A cooling protocol used for over 15 y involving an ice-water slurry has been applied effectively with no fatalities or adverse effects in hundreds of civilian and military individuals.[18,106,108] Agitation, intolerance, or combativeness may occur in encephalopathic heat stroke patients, and benefits of immersive cooling should be balanced with the theoretical concerns of impaired access to an immersed patient who may require advanced cardiac monitoring or resuscitation, especially among older patients.[90,94,109] Cold- water immersion may be considered in non-EHS, although this may not be practical for the typically older patients with multiple comorbidities in the critical care environment.[90,110] In the absence of cold-water immersion, wetted ice packs covering the entire body can cool through conduction.[99]

Recommendations.

1. Cold water immersion should be considered for EHS in the hospital setting (1A).
2. Cold water immersion can be considered for treatment of classic heat stroke patients (1C).

EVAPORATIVE AND CONVECTIVE COOLING

Evaporative cooling in elderly patients may offer several theoretical advantages, such as greater patient comfort and less agitation as well as easier access to patients who may need advanced monitoring or resuscitative procedures. In general, studies on evaporative and convective cooling have involved classic heat stroke patients and experimental volunteers with exercise-induced hyperthermia but not patients with actual EHS. The larger studies using a specially constructed device, termed a body cooling unit, have produced cooling rates ranging from 0.04 to 0.11 ℃-min^{1}, with an average cooling of time of 68 to 78 min and 10%

mortality.[94,95] No direct comparisons between the body cooling unit with cold water immersion are avail- able, but extrapolation of cooling rates suggests evaporative and convective cooling is an order of magnitude less efficacious. Because classic heat stroke patients are more likely to be older, obese, and with medical conditions such as diabetes, high blood pressure, and heart disease, the evidence suggests that the evaporative plus convective cooling technique by wetting and fanning the skin has a passable hospital-based role in the treatment of classic heat stroke but cools more slowly than conductive cooling and is not indicated in EHS.

Recommendations

1. Evaporative and convective cooling may be considered in classic heat stroke in the hospital setting, but cooling rates with this method are inferior to those with conductive cooling. Evaporative and convective cooling is not indicated in EHS, unless effective conductive cooling is not available (1C).

TARGET COOLING TEMPERATURES

The target cooling temperatures of EHS and exercise-induced hyperthermia to less than 39℃ by ice water immersion have been well tolerated, with no fatalities, adverse outcomes, or core temperature “afterdrop” resulting in hypothermia.[18,106,108,111] Practitioners should also be cautious of falsely elevated rectal temperature measurements in the recovery phase resulting from the insulating effect of body mass.[112]

Recommendations

Heat stroke patients should be cooled to a target temperature of no less than 39 ℃ (1B).

ADJUNCTIVE COOLING TREATMENTS

If intravenous fluids are available, it is beneficial to use cold fluids (4℃) whenever possible. These can decrease core temperature at a twofold rate compared with room temperature fluids but provide insufficient cooling as a primary treatment for heat stroke. More invasive techniques of body cavity lavage with cold isotonic fluid have been reported but have not been adequately studied.[113,114]Intravascular cooling catheters are suggested to decrease morbidity when added to evaporative and convective cooling.[115]

Recommendations

1. Cold intravenous fluids should be given for adjunctive cooling in heat stroke (1C).
2. Intra-vascular cooling catheters or cold water lavage are not recommended primary treatments for heat stroke (2C).

PHARMACOLOGIC TREATMENT

No pharmacologic agent has been shown to be helpful as a treatment for heat stroke. Dantrolene has been used for treatment of malignant hyperthermia. It acts by impairing calcium release from the sarcoplasmic reticulum, thereby reducing the muscular rigidity and hypertonicity typical of this condition. A well-designed randomized clinical trial of dantrolene vs placebo in classic heat stroke found no difference in cooling rates or outcome, concluding that this pharmacologic treatment should not be used in heat stroke patients.[116]

Recommendations

1. Dantrolene should be avoided for treatment of heat stroke patients (2B).

Conclusion

This article provides evidence-based guidelines for the prevention, recognition, and treatment of heat illness. Most of the available data are based on case series or extrapolation of results stemming from exercise-associated hyperthermia, which is an accepted research model, because randomized controlled trials for treatments of EHS are ethically challenging to justify. These guide- lines apply the strength of the evidence to 2 distinct populations of heat stroke patients, and although the patient with EHS is more likely to be found in the wilderness environment, the medical provider should be aware of all therapeutic modalities and their inherent risks and benefits. We recommend that patients with heat stroke be cooled by conductive means by whole-body ice water or cold-water immersion (the preferential method in EHS). Evaporative and convective cooling of classic heat stroke may be augmented with the addition of ice packs over the entire body to promote conductive cool- ing. Future areas of research should include direct comparisons of available cooling modalities in controlled models, as well as further evaluation of endovascular catheters and hospital-based systems for optimum cooling of critical patients.

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References

1. Robine JM, Cheung SL, Le Roy S, Van Oyen H, Griffiths C, Michel JP, et al. Death toll exceeded 70,000 in Europe during the summer of 2003.CRBiol. 2008;331(2):171–8.
2. Xu J. QuickStats: Number of heat-related deaths, by sexnational vital statistics system, United States, 1999 2010.MMWR Morb Mortal Wkly Rep. 2012; (36):729.
3. Gilchrist J, Haileyesus T, Murphy M, Comstock RD, Collins C, Yard E. Heat illness among high school athle- tesUnited States, 20052009.MMWR Morb Mortal Wkly Rep. 2010;59(32):1009–13.

4. Centers of Disease Control and Prevention. Heat-related illnesses and deathsUnited States, 19941995.MMWR Morb Mortal Wkly Rep. 1995;44(25):465–8.
5. Austin MG, Berry JW. Observations on one hundred cases of heatstroke.JAmMedAssoc. 1956;161(16): 1525–9.
6. Adolph EF. Tolerance to heat and dehydration in several species of mammals.Am J Physiol. 1947;151(2):564–75.
7. Overgaard J, Suit HD. Time-temperature relationship in hyperthermic treatment of malignant and normal tissue in vivo.Cancer Res. 1979;39(8):3248–53.
8. Vicario SJ, Okabajue R, Haltom T. Rapid cooling in classic heatstroke: effect on mortality rates.Am J Emerg Med. 1986;4(5):394–8.
9. Lipman GS, Eifling KP, Ellis MA, Gaudio FG, Otten EM, Grissom CK. Wilderness Medical Society practice guide- lines for the prevention and treatment of heat-related illness.Wilderness Environ Med. 2013;24(4):351–61.
10. Lipman GS, Eifling KP, Ellis MA, Gaudio FG, Otten EM, Grissom CK. Wilderness Medical Society practice guide- lines for the prevention and treatment of heat-related illness: 2014 update.Wilderness Environ Med. 2014; (4 Suppl):S55–65.
11. Noakes TD, Myburgh KH, du Plessis J, Lang L, Lambert M, van der Riet C, et al. Metabolic rate, not percent dehydra- tion, predicts rectal temperature in marathon runners.Med Sci Sports Exerc. 1991;23(4):443–9.
12. Byrne C, Lee JK, Chew SA, Lim CL, Tan EY. Continu- ous thermoregulatory responses to mass-participation dis- tance running in heat.Med Sci Sports Exerc. 2006; (5):803–10.
13. Veltmeijer MT, Eijsvogels TM, Thijssen DH, Hopman MT. Incidence and predictors of exertional hyperthermia after a 15-km road race in cool environmental conditions. J Sci Med Sport. 2015;18(3):333–7.
14. Lipman GS, Eifling KP, Ellis MA, Gaudio FG, Otten EM, Grissom CK. Keeping a broad perspective: reply regard- ing the Wilderness Medical Society practice guidelines for the prevention and treatment of heat-related illness. Wilderness Environ Med. 2014;25(2):251–2.
15. Nelson DA, Deuster PA, O'Connor FG, Kurina LM. Timing and predictors of mild and severe heat illness among new military enlistees.Med Sci Sports Exerc. 2018;50(8):1603–12.
16. Talbott JH, Michelsen J. Heat cramps. A clinical and chemical study.J Clin Invest. 1933;12(3):533–49.
17. Schwellnus MP. Cause of exercise associated muscle cramps (EAMC)altered neuromuscular control, dehydration or electrolyte depletion?Br J Sports Med. 2009;43(6):401–8.
18. Costrini AM, Pitt HA, Gustafson AB, Uddin DE. Cardio- vascular and metabolic manifestations of heat stroke and severe heat exhaustion.Am J Med. 1979;66(2):296–302.
19. Asplund CA, O'Connor FG, Noakes TD. Exercise-associ- ated collapse: an evidence-based review and primer for clinicians.Br J Sports Med. 2011;45(14):1157–62.

20. Casa DJ, DeMartini JK, Bergeron MF, Csillan D, Eichner ER, Lopez RM, et al. National Athletic Trainers' Associa- tion Position Statement: exertional heat illnesses.J Athl Train. 2015;50(9):986–1000.
21. Backer HD, Shopes E, Collins SL, Barkan H. Exertional heat illness and hyponatremia in hikers.Am J Emerg Med. 1999;17(6):532–9.
22. Bouchama A, Knochel JP. Heat stroke.N Engl J Med. 2002;346(25):1978–88.
23. Sawka MN, Wenger CB.Physiological Responses to Acute Exercise-Heat Stress. Indianapolis, IN: Benchmark Press; 1988.
24. Rowell LB. Cardiovascular aspects of human thermoreg- ulation.Circ Res. 1983;52(4):367–79.
25. Heller HC, Grahn DA. Enhancing thermal exchange in humans and practical applications.Disrupt Sci Technol. 2012;1(1):11–9.
26. Armstrong LE, Casa DJ, Millard-Stafford M, Moran DS, Pyne SW, Roberts WO. American College of Sports Medi- cine position stand. Exertional heat illness during training and competition.Med Sci Sports Exerc. 2007;39(3):556–72.
27. Leon LR, Helwig BG. Heat stroke: role of the systemic inflammatory response.J Appl Physiol (1985). 2010; (6):1980–8.
28. Sakaguchi Y, Stephens LC, Makino M, Kaneko T, Strebel FR, Danhauser LL, et al. Apoptosis in tumors and normal tissues induced by whole body hyperthermia in rats.Cancer Res. 1995;55(22):5459–64.
29. Buckley IK. A light and electron microscopic study of thermally injured cultured cells.Lab Invest. 1972; (2):201–9.
30. Eberman LE, Cleary MA. Development of a heat-illness screening instrument using the Delphi panel technique. J Athl Train. 2011;46(2):176–84.
31. Toloo GS, Fitzgerald G, Aitken P, Verrall K, Tong S. Are heat warning systems effective?Environ Health. 2013;12:27.
32. Pryor RR, Bennett BL, O'Connor FG, Young JM, Asplund CA. Medical evaluation for exposure extremes: heat.Wilderness Environ Med. 2015;26(4 suppl):S69–75.
33. Pandolf KB, Gange RW, Latzka WA, Blank IH, Kraning II KK, Gonzalez RR. Human thermoregulatory responses during heat exposure after artificially induced sunburn. Am J Physiol. 1992;262(4 Pt 2):R610–6.
34. Pandolf KB, Gange RW, Latzka WA, Blank IH, Young AJ, Sawka MN. Human thermoregulatory responses dur- ing cold water immersion after artificially induced sun- burn.Am J Physiol. 1992;262(4 Pt 2):R617–23.
35. Glazer JL. Management of heatstroke and heat exhaus- tion.Am Fam Physician. 2005;71(11):2133–40.
36. Bruning RS, Dahmus JD, Kenney WL, Alexander LM. Aspirin and clopidogrel alter core temperature and skin blood flow during heat stress.Med Sci Sports Exerc. 2013;45(4):674–82.
37. Bedno SA, Li Y, Han W, Cowan DN, Scott CT, Cavicchia MA, et al. Exertional heat illness among overweight US army recruits in basic training.Aviat Space Environ Med. 2010;81(2):107–11.
38. Brazaitis M, Skurvydas A. Heat acclimation does not reduce the impact of hyperthermia on central fatigue.Eur J Appl Physiol. 2010;109(4):771–8.
39. Cheung SS, McLellan TM. Heat acclimation, aerobic fit- ness, and hydration effects on tolerance during uncom- pensable heat stress.J Appl Physiol

(1985). 1998; (5):1731–9.
40. Garrett AT, Goosens NG, Rehrer NJ, Patterson MJ, Cotter JD. Induction and decay of short-term heat acclimation. Eur J Appl Physiol. 2009;107(6):659–70.
41. Weller AS, Linnane DM, Jonkman AG, Daanen HA. Quantification of the decay and re-induction of heat accli- mation in dry-heat following 12 and 26 days without exposure to heat stress.Eur J Appl Physiol. 2007; (1):57–66.
42. Daanen HA, Jonkman AG, Layden JD, Linnane DM, Weller AS. Optimising the acquisition and retention of heat acclimation.Intl J Sports Med. 2011;32(11):822–8.
43. Armstrong LE, De Luca JP, Hubbard RW. Time course of recovery and heat acclimation ability of prior exertional heat- stroke patients.Med Sci Sports Exerc. 1990;22(1):36–48.
44. Kazman JB, Heled Y, Lisman PJ, Druyan A, Deuster PA, O'Connor FG. Exertional heat illness: the role of heat tol- erance testing.Curr Sports Med Rep. 2013;12(2):101–5.
45. Johnson EC, Kolkhorst FW, Richburg A, Schmitz A, Martinez J, Armstrong LE. Specific exercise heat stress protocol for a triathlete's return from exertional heat stroke.Curr Sports Med Rep. 2013;12(2):106–9.
46. Sharwood KA, Collins M, Goedecke JH, Wilson G, Noakes TD. Weight changes, medical complications, and performance during an Ironman triathlon.Br J Sports Med. 2004;38(6):718–24.
47. Wharam PC, Speedy DB, Noakes TD, Thompson JM, Reid SA, Holtzhausen LM. NSAID use increases the risk of developing hyponatremia during an Ironman triathlon. Med Sci Sports Exerc. 2006;38(4):618–22.
48. Maughan RJ, Watson P, Shirreffs SM. Heat and cold: what does the environment do to the marathon runner? Sports Med. 2007;37(4-5):396–9.
49. Wall BA, Watson G, Peiffer JJ, Abbiss CR, Siegel R, Laursen PB. Current hydration guidelines are erroneous: dehydration does not impair exercise performance in the heat.Br J Sports Med. 2015;49(16):1077–83.
50. Sawka MN, Latzka WA, Matott RP, Montain SJ. Hydra- tion effects on temperature regulation.Int J Sports Med. 1998;19(Suppl 2):S108–10.
51. Sawka MN, Young AJ, Latzka WA, Neufer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: influence of hydration.J Appl Physiol (1985). 1992;73(1):368–75.
52. Duffield R, Steinbacher G, Fairchild TJ. The use of mixed-method, part-body pre-cooling procedures for team-sport athletes training in the heat.J Strength Cond Res. 2009;23(9):2524–32.
53. Lemire B, Gagnon D, Jay O, Dorman L, DuCharme MB, Kenny GP. Influence of adiposity on cooling efficiency in hyperthermic individuals.Eur J Appl Physiol. 2008; (1):67–74.
54. Tenaglia SA, McLellan TM, Klentrou PP. Influence of menstrual cycle and oral contraceptives on tolerance to uncompensable heat stress.Eur J Appl Physiol Occup Physiol. 1999;80(2):76–83.
55. Bergeron MF, Devore C, Rice SG. Policy statement— Climatic heat stress and exercising children and ado- lescents.Pediatrics. 2011;128(3):e741–e7.
56. Basu R, Samet JM. Relation between elevated ambient temperature and mortality: a review of the epidemiologic evidence.Epidemiol Rev.

2002;24(2):190–202.
57. Bouchama A, Dehbi M, Mohamed G, Matthies F, Shoukri M, Menne B. Prognostic factors in heat wave related deaths: a meta-analysis.Arch Intern Med. 2007;167(20):2170–6.
58. Nelson N, Eichna LW, Horvath SM, Shelley WB, Hatch TF. Thermal exchanges of man at high temperatures.Am J Physiol. 1947;151(2):626–52.
59. United States Department of the Navy, Bureau of Medi- cine and Surgery.Manual of Naval Preventive Medicine (NAVMED P-5010-3). Washington, DC: Department of the Navy, US Department of Defense; 2009.
60. Occupational Health and Safety Administration.OSHA Technical Manual (TED1-00-015). Washington, DC: Occupational Health and Safety Administration, US Department of Labor; 1999.
61. National Climactic Data Center.Heat Index Charts. Asheville, NC: National Oceanic and Atmospheric Administration, National Climactic Data Center; 2000.
62. Jackson LL, Rosenberg HR. Preventing heat-related illness among agricultural workers.J Agromedicine. 2010; (3):200–15.
63. Armstrong LE, Johnson EC, Casa DJ, Ganio MS, McDer- mott BP, Yamamoto LM, et al. The American football uniform: uncompensable heat stress and hyperthermic exhaustion.J Athl Train. 2010;45(2):117–27.
64. Porter AM. Heat illness and soldiers.Mil Med. 1993; (9):606–9.
65. Biery Jr JC, Blivin SJ, Pyne SW. Training in ACSM black flag heat stress conditions: how US marines do it.Curr Sports Med Rep. 2010;9(3):148–54.
66. Armstrong LE, Crago AE, Adams R, Roberts WO, Mar- esh CM. Whole-body cooling of hyperthermic runners: comparison of two field therapies.Am J Emerg Med. 1996;14(4):355–8.
67. Hadad E, Moran DS, Epstein Y. Cooling heat stroke patients by available field measures.Intensive Care Med. 2004;30(2):338.
68. Hadad E, Rav-Acha M, Heled Y, Epstein Y, Moran DS. Heat stroke: a review of cooling methods.Sports Med. 2004;34(8):501–11.
69. Shapiro Y, Seidman DS. Field and clinical observations of exertional heat stroke patients.Med Sci Sports Exerc. 1990;22(1):6–14.
70. Schwellnus MP, Derman EW, Noakes TD. Aetiology of skeletal muscle ‘cramps’ during exercise: a novel hypoth- esis.J Sports Sci. 1997;15(3):277–85.
71. Lugo-Amador NM, RothenhausT, Moyer P. Heat-related illness.Emerg Med Clin North Am. 2004;22(2):315–27. viii.
72. Canpolat U, Bayazit Y, Aytemir K. Brugada syndrome unmasked by heat exhaustion.Ann Noninvasive Electro- cardiol. 2017;22(1).
73. Molina Borao I, Calvo Galiano N, Urmeneta Ulloa J, Rivero Fernandez E, Oloriz Sanjuan T, Asso Abadia A. Heat wave: a trigger of electrical storm in a patient with Brugada syndrome. Rev Esp Cardiol (Engl Ed.). 2016;69(12): 1228–30.
74. Binkley HM, Beckett J, Casa DJ, Kleiner DM, Plummer PE. National Athletic Trainers’ Association position statement: exertional heat illnesses.JAthlTrain. 2002;37(3):329–43.
75. Casa DJ, Becker SM, Ganio MS, Brown CM, Yeargin SW, Roti MW, et al. Validity of devices that assess body temperature during outdoor exercise in

the heat.J Athl Train. 2007;42(3):333–42.
76. Gonzalez-Alonso J, Calbet JA, Nielsen B. Metabolic and thermodynamic responses to dehydration-induced reduc- tions in muscle blood flow in exercising humans.J Phys- iol. 1999;520(Pt 2):577–89.
77. Casa DJ, Armstrong LE, Hillman SK, Montain SJ, Reiff RV, Rich BS, et al. National Athletic Trainers' Association position statement: fluid replacement for athletes.J Athl Train. 2000;35(2):212–24.
78. Lopez RM, Casa DJ, Jensen KA, DeMartini JK, Pagnotta KD, Ruiz RC, et al. Examining the influence of hydration status on physiological responses and running speed dur- ing trail running in the heat with controlled exercise intensity.J Strength Cond Res. 2011;25(11):2944–54.
79. Sawka MN. Physiological consequences of hypohydra- tion: exercise performance and thermoregulation.Med Sci Sports Exerc. 1992;24(6):657–70.
80. Castellani JW, Maresh CM, Armstrong LE, Kenefick RW, Riebe D, Echegaray M, et al. Intravenous vs. oral rehydration: effects on subsequent exercise-heat stress.J Appl Physiol (1985). 1997;82(3):799–806.
81. Epstein Y, Shani Y, Moran DS, Shapiro Y. Exertional heat strokethe prevention of a medical emergency. J Basic Clin Physiol Pharmacol. 2000;11(4):395–401.
82. Hostler D, Reis SE, Bednez JC, Kerin S, Suyama J. Com- parison of active cooling devices with passive cooling for rehabilitation of firefighters performing exercise in ther- mal protective clothing: a report from the Fireground Rehab Evaluation (FIRE) trial.Prehosp Emerg Care. 2010;14(3):300–9.
83. Hew-Butler T, Rosner MH, Fowkes-Godek S, Dugas JP, Hoffman MD, Lewis DP, et al. Statement of the Third International Exercise-Associated Hyponatremia Consen- sus Development Conference, Carlsbad, California, 2015. Clin J Sport Med. 2015;25(4):303–20.
84. McArdle WD, Magel JR, Lesmes GR, Pechar GS. Meta- bolic and cardiovascular adjustment to work in air and water at 18, 25, and 33 degrees C.J Appl Physiol. 1976;40(1):85–90.
85. Noakes TD. Body cooling as a method for reducing hyperthermia.S Afr Med J. 1986;70(6):373–4.
86. Proulx CI, Ducharme MB, Kenny GP. Effect of water temperature on cooling efficiency during hyperthermia in humans.J Appl Physiol (1985). 2003;94(4):1317–23.
87. Cooper KE. Physiology of hypothermia.Br J Anaesth. 1959;31(3):96–105.
88. Proulx CI, Ducharme MB, Kenny GP. Safe cooling limits from exercise-induced hyperthermia.Eur J Appl Physiol. 2006;96(4):434–45.
89. Wyndham CH, Strydom NB, Cooke HM, Maritz JS, Morrison JF, Fleming PW, et al. Methods of cooling sub- jects with hyperpyrexia.J Appl Physiol. 1959;14:771–6.
90. Ferris EB, Blankenhorn MA, Robinson HW, Cullen GE. Heat stroke: clinical and chemical observations on 44 cases.J Clin Invest. 1938;17(3):249–62.
91. Casa DJ, McDermott BP, Lee EC, Yeargin SW, Arm- strong LE, Maresh CM. Cold water immersion: the gold standard for exertional heatstroke treatment.Exerc Sport Sci Rev. 2007;35(3):141–9.

92. O'Connor JP. Simple and effective method to lower body core temperatures of hyperthermic patients.Am J Emerg Med. 2017;35(6):881–4.
93. Costrini A. Emergency treatment of exertional heatstroke and comparison of whole body cooling techniques.Med Sci Sports Exerc. 1990;22(1):15–8.
94. Khogali M, Weiner JS. Heat stroke: report on 18 cases. Lancet. 1980;2(8189):276–8.
95. Khogali M, al Khawashi M. Heat stroke during the Mak- kah pilgrimage.Saudi Med J. 1981;2:85–93.
96. Al-Aska AK, Abu-Aisha H, Yaqub B, Al-Harthi SS, Sal- lam A. Simplified cooling bed for heatstroke.Lancet. 1987;1(8529):381.
97. Hee-Nee P, Rupeng M, Lee VJ, Chua WC, Seet B. Treat- ment of exertional heat injuries with portable body cool- ing unit in a mass endurance event.Am J Emerg Med. 2010;28(2):246–8.
98. Platt M, Vicario S.Rosen's Emergency Medicine. 7th ed. Philadelphia, PA: Mosby; 2009.
99. Kielblock AJ, Van Rensburg JP, Franz RM. Body cooling as a method for reducing hyperthermia. An evaluation of techniques.S Afr Med J. 1986;69(6):378–80.
100. Richards D, Richards R, Schofield PJ, Ross V, Sutton JR. Management of heat exhaustion in Sydney's the Sun City-to-Surf run runners. Med J Aust. 1979; (9):457–61.
101. Phan S, Lissoway J, Lipman GS. Chemical cold packs may provide insufficient enthalpy change for treatment of hyperthermia.Wilderness Environ Med. 2013;24(1):37–41.
102. Lissoway JB, Lipman GS, Grahn DA, Cao VH, Shaheen M, Phan S, et al. Novel application of chemical cold packs for treatment of exercise-induced hyperthermia: a randomized controlled trial.Wilderness Environ Med. 2015;26(2):173–9.
103. Saper CB, Breder CD. Endogenous pyrogens in the CNS: role in the febrile response.Prog Brain Res. 1992;93:419–28. discussion 4289.
104. Downey JA, Darling RC. Effect of salicylates on eleva- tion of body temperature during exercise.J Appl Physiol. 1962;17:323–5.
105. Johnson SC, Ruhling RO. Aspirin in exercise-induced hyperthermia. Evidence for and against its role.Sports Med. 1985;2(1):1–7.
106. Beller GA, Boyd 3rd AE. Heat stroke: a report of 13 conse- cutive cases without mortality despite severe hyperpyrexia and neurologic dysfunction.Mil Med. 1975;140(7):464–7.
107. Demartini JK, Casa DJ, Stearns R, Belval L, Crago A, Davis R, et al. Effectiveness of cold water immersion in the treatment of exertional heat stroke at the Falmouth Road Race.Med Sci Sports Exerc. 2015;47(2):240–5.
108. O'Donnell Jr TF. Acute heat stroke. Epidemiologic, bio- chemical, renal, and coagulation studies.JAMA. 1975; 234(8):824–8.
109. Bouchama A, Dehbi M, Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical rec- ommendations.Crit Care. 2007;11(3):R54.
110. Hart GR, Anderson RJ, Crumpler CP, Shulkin A, Reed G, Knochel JP. Epidemic classical heat stroke: clinical char- acteristics and course of 28 patients.Medicine (Balti- more). 1982;61(3):189–97.

111. Gagnon D, Lemire BB, Casa DJ, Kenny GP. Cold-water immersion and the treatmentof hyperthermia: using 38.6℃ as a safe rectal temperature cooling limit.JAthlTrain. 2010;45(5):439–44.
112. Newsham KR, Saunders JE, Nordin ES. Comparison of rectal and tympanic thermometry during exercise.South Med J. 2002;95(8):804–10.
113. Smith JE. Cooling methods used in the treatment of exer- tional heat illness.Br J Sports Med. 2005;39(8):503–7. discussion 507.
114. Horowitz BZ. The golden hour in heat stroke: use of iced peritoneal lavage.Am J Emerg Med. 1989;7(6):616–9.
115. Yokobori S, Koido Y, Shishido H, Hifumi T, Kawakita K, Okazaki T, et al. Feasibility and safety of intravascular temperature management for severe heat stroke: a pro- spective multicenter pilot study.Crit Care Med. 2018; (7):e670–6.
116. Bouchama A, Cafege A, DevolEB, Labdi O, el-Assil K, Seraj M. Ineffectiveness of dantrolene sodium in the treatment of heatstroke. Crit Care Med. 1991; (2):176–80.

Wilderness Medical Society Clinical Practice Guidelines for the Treatment and Prevention of Drowning: 2019 Update

Andrew C.Schmidt DO, MPH [1] Justin R.SempsrottMD[2] Seth C.HawkinsMD[3] Ali S.ArastuMD[4] Tracy A.CushingMD, MPH[5] Paul S.AuerbachMD, MS[6]

The Wilderness Medical Society convened a panel to review available evidence supporting practices for acute management and treatment of drowning in out-of-hospital and emergency medical care settings. Literature about definitions and terminology, epidemiology, rescue, resuscitation, acute clinical management, disposition, and drowning prevention was reviewed. The panel graded available evidence supporting practices according to the American College of Chest Physicians criteria and then made recommendations based on that evidence. Recommendations were based on the panel's collective clinical experience and judgment when published evidence was lacking. This is the first update to the original practice guidelines published in 2016.

Keywords

submersion
immersion
cold water submersion
hypothermia

Introduction

Approximately 360,000 deaths globally are attributed to drowning every year.[1] Drowning often affects young victims and can have dire personal, emotional, and financial consequences for patients, families, and society. The goal of these practice guidelines is to reduce the burden of drowning through improvements in treatment and prevention. We present accepted drowning terminology as part of a review and evaluation of literature regarding acute care for the drowning patient, in both out-of-hospital and emergency medical care settings, with particular focus on the wilderness context. The authors relied upon the experience and knowledge of a panel of wilderness and emergency medicine practitioners to make recommendations where little or unreliable evidence is available.[2] This is the first update of the original publication from 2016.[3]

Methods

The authors of this update reviewed each section of the original document to determine relevance and need for updating. Articles were identified through PUBMED, MEDLINE, and Google Scholar using a keyword search appropriate to each topic. Randomized controlled trials, observational studies, case series, and review articles were reviewed and evidence assessed. Abstracts for which the full article could not be obtained were excluded. If no relevant studies were identified, recommendations were based on the panel's clinical experience and judgment. Recommendations were graded using the American College of Chest Physicians classification scheme (online Supplemental Table 1), in accordance with prior versions of the Wilderness Medical Society Practice Guidelines.[4]

Epidemiology

The highest-risk age group for drowning worldwide is children ages 1 to 4 y, primarily owing to unintentional falls into water; the next highest-risk group is adolescents and young adults in natural bodies of water. In the United States, there were on average 3536 drowning deaths per year from 2005 to 2014, plus an additional 679 boating-related deaths, 75% of which were from drowning.[5, 6] More than 90% of the world's drowning deaths occur in low- and middle-income countries.[1] In the context of low- and middle-income countries, natural sources of water are often ubiquitous and used for transportation, cleaning, food, and hydration and lack barriers. Based on World Health Organization and Centers for Disease Control and Prevention systems for classifying drowning statistics, these numbers exclude deaths occurring during floods and other natural disasters. In 2010, there were 12,900 emergency department (ED) visits in the United States for drowning, with 20% of patients admitted to the hospital. Drowning deaths were 48% more likely to occur on weekends compared to weekdays. Fifty-three percent of all male and 26% of all female drowning deaths occurred in natural bodies of water.[5]

The burden of drowning is underreported because most studies address the issue of fatal drowning. In the United States, a conservative estimate is that for every fatal drowning, another 5 persons seek emergency care for nonfatal drowning.[5] Internationally, the burden of nonfatal drowning is more difficult to estimate because many patients may not present to an emergency medical system or hospital, where data collection typically occurs.[5, 7, 8] In Bangladesh, a large population-based study showed fatal and nonfatal drowning rates of 15.8 per 100,000 and 318.4 per 100,00 compared to 1.17 per 100,000 and 10 per 100,000 in the United States.[9, 10] Risk factors for nonfatal drowning are similar to those for fatal drowning.[9, 11, 12, 13, 14, 15]

Terminology

The standard definition for drowning, as defined by the World Congress on Drowning in 2002, is “the process of experiencing respiratory impairment due to submersion or immersion in liquid.” Inspired by the Utstein Style for reporting cardiac arrest data, the standard definition allows for only 3 outcomes after drowning: 1) morbidity; 2) no morbidity; and 3) mortality. This definition is based on the understanding that “respiratory impairment occurs as the person’s airway goes below the surface of the liquid (submersion) or water splashes over the face (immersion).”[16] However, the inclusion of both submersion and immersion in this definition may cause confusion with the large body of work on survival and rescue related specifically to cold water immersion, which focuses more on hypothermia than on drowning. For the purposes of these guidelines, which could include cold water conditions, a further distinction is necessary. “Immersion” refers to situations in which airways are above water, whereas “submersion” refers to situations in which airways are under water. Thus immersion (in cold water) may lead to hypothermia, and submersion at any water temperature may lead to drowning. The following modifiers should *not* be used in association with drowning: near, wet, dry, active, passive, saltwater, freshwater, or secondary. Sufficient data related to human drowning pathophysiology show that none of these modifiers is valid because the final common pathway is hypoxemia and eventual cardiopulmonary arrest.[2, 16, 17] By understanding and using the standard definition for drowning and abstaining from using incorrect terminology, communication among medical practitioners, data collection agencies, researchers, and policymakers has become more consistent. Accurate communication better reflects the true incidence, prevalence, and sequelae of drowning and should improve clinical dialogue and management.[18, 19, 20]

Rescue of the Drowning Patient

Reaching the Patient

Rescuer safety is paramount during rescue operations; in the aquatic environment, specific skills, training, and physical capabilities are required. The physical characteristics of aquatic environments vary widely, with a spectrum including pools, lakes, rivers, ocean, swift river water, and ice scenarios, each requiring different sets of equipment and training for technical rescue. Few studies objectively measure effectiveness of in-water rescue techniques. Much of the literature on this topic is based on experiences and policies of the writers or organizational authorities. There is a high prevalence of fatal and nonfatal drowning of untrained persons attempting to perform in-water rescues, with 1 study revealing 114 rescuer deaths during a 3-y period in Turkey alone.[21, 22, 23] Hazardous water conditions that led to the initial person drowning often persist and place the well-intentioned rescuer at risk for becoming an additional drowning patient.[24] Rescue by untrained persons should be attempted without entering hazardous conditions by reaching out to the drowning patient with a paddle or branch; throwing a rope, buoy, cooler, or any floating object; or rowing a boat, canoe, or paddleboard to the patient. Trained rescue personnel should operate according to their level of training, expertise, equipment, and comfort level. Entering the water to perform a rescue should be attempted only by persons with

specific training to operate in that dangerous environment. Few studies have been conducted on the effectiveness of different water safety devices (eg, rescue tubes, rescue cans, throw bags, life rings), but what has been demonstrated is that proper and effective use of these devices requires basic knowledge of their function combined with regular practice.[25]

Recommendation

Persons without formal water rescue training should attempt rescues from a safe location by reaching, throwing, or rowing to the drowning patient. Persons with formal water rescue training should perform in-water rescues according to their level of training and with personal protective and safety equipment. There is insufficient evidence to recommend specific rescue devices. If specialized rescue equipment is available, participants should be familiar with the location and purpose of this equipment, and designated rescue personnel with proper training should be tasked with its use in the event of a water rescue. **Recommendation Grade:** 1C[26, 27, 28]

PATIENTS IN SUBMERGED VEHICLES

Death from entrapment and drowning in submerged vehicles is often not classified as a drowning death, confounding attempts to accurately track the epidemiology of this type of drowning.[29] Studies suggest that 10% of drowning deaths may be due to entrapment in submerged vehicles and that in the case of inland flooding as much as 10% of motor vehicle crashes result in a drowning death.[30, 31, 32, 33] There is a small body of medical and rescue literature on the topic of vehicle submersions.[31, 34, 35, 36, 37, 38, 39] A formal review of educational and public service information identified “three probable significant contributors to [the] high fatality rate [of drowning in submerged vehicles]: 1) ‘authorities’ provide an inadequate description of vehicle sinking characteristics; 2) contradictory and inadequate advice is often provided; and 3) a poor public perception of how to escape.”[34] Several sources recommend questionable escape practices without supporting evidence for efficacy. These practices include allowing the passenger compartment to fill with water so that it will be easier to open doors, waiting until the vehicle sinks to the bottom of a body of water to maintain orientation, relying on kicking out the windshield or opening doors after the vehicle has fully sunk, and relying on breathing trapped air in the passenger compartment. In a formal survey, more than half of the general public identify an option that involves staying in a vehicle while it sinks to the bottom as being the safest option when trapped in a submerging vehicle; this advice often appears in the popular media.[38] Research data derived from 35 vehicle submersions conducted in diverse locations and seasons suggest that this advice is erroneous. Evidence suggests the best time to escape from a submerging vehicle is immediately during the initial floating phase, ideally during the initial 30 s to 2 min after water entry when most vehicles remain partially above the surface.[38] An algorithm, using the acronym SWOC, has been developed to advise those entrapped in water how to sequence escape actions. The SWOC algorithm recommends the following sequencing of actions: Seatbelts off, Window open, Out immediately, Children first.[39] In 2008, a US-based proprietary out-of-hospital

emergency medical dispatcher system added an addendum to its standardized protocols that instructed emergency medical dispatchers not to persist in getting a location for a caller in a submerging vehicle. Instead, it recommends that a caller exit the vehicle immediately if it is submerging, before using precious time to determine location, and using the SWOC protocol.[40, 41]

Recommendation

The safest time to escape from a submerging vehicle is immediately after it enters the water, during the initial floating phase. If the vehicle remains floating, persons should climb out and remain on top of the vehicle. If it is sinking, they should move away from the vehicle and toward safety after exiting. **Recommendation Grade:** 2C

IN-WATER RESUSCITATION

The primary physiologic insult in a drowning patient is cerebral hypoxia; its rapid reversal is the primary objective of drowning resuscitation. For the purpose of these guidelines, in-water resuscitation (IWR) is defined as an attempt to provide ventilations to a drowning patient who is still in the water. This does not apply to chest compressions. It is impossible to perform adequate chest compressions while the victim and rescuer are in the water, and so they should not be attempted.[42] Successful use of IWR was first described in 1976, with a manikin-based feasibility study reported in 1980; however, the first clinical study to show a positive patient outcome was not published until 2004.[43, 44, 45]

Available outcome data for IWR are based on a single retrospective analysis of lifeguard rescues in Brazil and show significant improvement in survival and neurologic outcome in persons receiving IWR. These rescues were performed by trained, professional lifeguards in the ocean environment. Lifeguards would frequently tow the patient beyond breaking waves and perform mouth-to-mouth ventilations while awaiting helicopter pickup.[45] Subsequent studies, primarily using manikins, evaluated ease of performing this task in controlled aquatic environments and found that IWR increases overall rescue time, subjective rescue difficulty, number of submersions, and water aspiration.[46, 47] A single study comparing lifeguards to lay rescuers when using IWR found that lifeguards showed improved rescue times and decreased estimated pulmonary aspiration.[48] Consensus statements from the International Lifesaving Federation, United States Lifesaving Association, American Red Cross, and the Young Men's Christian Association recommend IWR by trained rescuers when a patient is rescued in shallow water or in deep water when a flotation device is present.[49, 50]

Rescuer safety and prevention of communicable diseases are of utmost importance, so consideration should be given to the use of barrier devices during IWR. Food and Drug Administration-approved, IWR-specific devices are available that use a self-purging mechanical one-way valve instead of the paper valve on standard CPR masks.[51, 52]

Recommendation

IWR should only be considered by a rescuer with adequate training, ability, and equipment to safely and effectively perform the skill in the aquatic environment. The aquatic conditions must be sufficiently safe for the rescuer to perform IWR, and the point of extrication from the water must be sufficiently distant to warrant an attempt of this technically difficult task. If conditions are too hazardous to safely perform the task, rapid extrication is indicated without a delay for IWR. Chest compressions should not be attempted in the water; all drowning patients without a pulse should be extricated as quickly and safely as possible so that early, effective chest compressions and ventilations can be initiated.
Recommendation Grade: 1C

Initial Resuscitation

HYPOTHERMIA

Water is thermally neutral at approximately 33°C (91°F). Because most patients drown in water at a lower temperature than this, concomitant hypothermia is common.[30] The main physiologic problem with drowning is brain hypoxia. Current practice suggests that the brain can withstand longer periods of hypoxia if the body is cooler than the normal physiologic range. On one hand, leaving a patient moderately cool, or warming them to a moderately cool degree, could be beneficial or at least innocuous. On the other hand, moderate to severe hypothermia should be corrected, with the understanding that warming may be operationally difficult in some drowning situations. Beyond initiation of basic warming measures, the details of hypothermia treatment, including augmented advanced life support measures, are beyond the scope of these guidelines. Readers are encouraged to review the most current version of the Wilderness Medical Society Practice Guidelines for the Out-of-Hospital Evaluation and Treatment of Accidental Hypothermia.[53]

Recommendation

Suspect and treat hypothermia. **Recommendation Grade:** 1C

CARDIOPULMONARY RESUSCITATION AND PRIORITIZATION OF AIRWAY

Because of the central role of hypoxemia in the pathophysiology of drowning, initial resuscitation should focus on establishing and maintaining a patent airway and providing oxygen. Recent updates to cardiopulmonary resuscitation (CPR) algorithms, specifically for the lay rescuer, include recommendations for compression-only CPR and prioritization of compressions before airway maneuvers.[54, 55] Compression-only CPR is likely to be of little to no benefit in drowning resuscitation, and its use is limited to untrained bystanders. Bystander CPR for infants and children includes compressions and ventilations, regardless of which is started first. Professional rescuer CPR should emphasize prioritization of airway and breathing before initiation of chest compressions. If the airway is overlooked in initial resuscitation, ongoing hypoxemia leads to decreased survival

and worse neurologic outcomes. Incorrect application of rescue breaths can delay care and cause gastric insufflation and pulmonary aspiration. For lay responders or persons without current training in rescue breathing, compression-only CPR is still the preferred method of resuscitation. All persons who may respond to a drowning person (eg, parents, trip leaders, lifeguards) should take CPR classes that include training on proper use of chest compressions and rescue breathing.

Recommendation

Supplying oxygen to the brain is critical to successful resuscitation of the drowning patient. Establishing an airway and providing oxygen are priorities in initial resuscitation. For the patient in cardiac arrest, provide positive pressure ventilations in addition to chest compressions using the traditional Airway-Breathing-Circulation model of resuscitation. If an advanced airway is available and properly placed, provide breaths at specified time intervals (every 6 to 8 s) while continuous compressions are administered. For lay people without training in rescue breathing, compression-only CPR is a preferred alternative to no intervention. **Recommendation Grade:** 1C

OXYGENATION

Few large-scale studies have evaluated different airway adjuncts applied to drowning patients. Although ideal rescue breathing includes supplemental oxygen and a positive pressure delivery device, any amount of oxygen delivery (eg, mouth-to-mouth, bag-valve-mask [BVM] with ambient air) is better than none if supplemental oxygen is not available. Manikin studies of supraglottic airways have shown that lifeguards can successfully insert them, but there is concern that this does not replicate real world usage.[56], [57] Additional concern is that because of pulmonary edema from drowning, certain supraglottic airway devices may perform poorly for oxygenation based on leak pressures.[58], [59] If the supraglottic airway fails to achieve adequate chest rise, a BVM or other method to oxygenate and ventilate the patient should be used.

Recent resuscitation data have brought into question the benefit of providing high oxygen concentrations in the acute setting of out-of-hospital cardiac arrest and stroke, primarily based on data correlating hyperoxemia after return of spontaneous circulation (ROSC) with increased mortality. Most of these data focus on the period after ROSC in the intensive care unit setting; no studies focus specifically on cardiac arrest associated with drowning or other primary respiratory events. A single retrospective case-control study involving arterial blood analysis during CPR provides support for using high levels of supplemental oxygen. This study showed a significant increase in survival to hospital discharge with increasing levels of arterial oxygenation in all cardiac arrest patients, even at levels that would be considered hyperoxemic.[60]

Recommendation

When resuscitating a drowning patient, oxygen should initially be delivered at the highest concentration available. For the patient in respiratory distress or arrest, positive pressure is preferred over passive ventilation. If multiple modalities are available, the method that most effectively delivers the highest concentration of oxygen should be used. If a modality or device fails, BVM or mouth-to-mouth ventilation should be attempted. **Recommendation Grade:** 1C

AUTOMATED EXTERNAL DEFIBRILLATOR

Although cerebral hypoxia is the primary cause of morbidity in the drowning patient, hypoxic myocardial injury might also occur. Drowning patients initially typically experience sinus tachycardia, followed by bradycardia, pulseless electrical activity, and then asystole, owing to the hypoxic nature of the event.[61] In drowning patients, ventricular fibrillation (VF) is rare, occurring in less than 10% of patients; thus, reversal of hypoxemia with ventilations and compressions should not be delayed in an attempt to apply an automated external defibrillator (AED).[61, 62, 63, 64, 65, 66, 67] Once resuscitation is established, early application of an AED might be beneficial, given the possibility of VF as the cause or result of drowning. In the drowning patient, if global myocardial hypoxia persists, attempts at defibrillation may be unsuccessful without concomitant oxygenation and ventilation.

Experimental animal models have shown that as long as AED pads are placed firmly on a patient's chest and a rescuer is not in direct contact with that patient, use of an AED in a wet environment does not pose increased risk to the patient or rescuers.[68, 69, 70] AEDs have been tested and noted to correctly detect simulated arrhythmias and deliver shocks on moving boats.[71]

Recommendation

VF is rare in drowning, so incorporation of an AED in the initial minutes of drowning resuscitation should not interfere with oxygenation and ventilation. If available, an AED should be used during resuscitation of a drowning patient; its use is not contraindicated in a wet environment. **Recommendation Grade:** 1A

HEIMLICH MANEUVER

Drowning involves water obstructing the airway and causing cerebral hypoxia; in some cases, small amounts of water are aspirated into the lungs. This can cause atelectasis, direct cellular injury, and pulmonary edema. Even after unconsciousness, reflex swallowing of water from the hypopharynx into the stomach may occur. Dr Henry Heimlich advocated use of abdominal thrusts in initial treatment of the drowning patient, claiming that aspirated water must first be cleared from the airway to allow proper ventilations.[72, 73, 74] In the 30 y since his original report, concern has been raised about this recommendation, resulting in an Institute of Medicine report and a systematic literature review by the American Red Cross.[75, 76] All of these investigations failed to identify quality

data to support use of the Heimlich maneuver before providing ventilations. Its use during initial resuscitation delays delivery of ventilations and prolongs hypoxemia.[75]

Recommendation

Owing to the possibility of delaying ventilations, the Heimlich maneuver is not recommended for resuscitation of the drowning patient. **Recommendation Grade:** 1B

CERVICAL SPINE PRECAUTIONS

Recent discussions and research in the field of out-of-hospital medicine have brought in to question the utility, safety, and clinical benefit of what has been called routine spine immobilization. The most current published review of this topic specific to austere environments is the Wilderness Medical Society Clinical Practice Guidelines for Spinal Cord Protection: 2019 Update.[77] We recommend reviewing the updated guidelines for current evidence on the utility of this procedure.

Retrospective studies of drowning patients found the incidence of cervical spine injuries was low (0.5 to 5%) and that most injuries were related to diving from a height. In patients without obvious signs of trauma or a known fall or diving event, the risk of spine injury is low.[78, 79] In these patients, treatment maneuvers focused on restricting spine motion may distract rescuers from the critical role of oxygenation and ventilation.

Recommendation

The most current Wilderness Medical Society Practice Guidelines concerning the field treatment of possible spinal injuries should be reviewed when developing or reviewing agency protocols. Drowning patients who display evidence of spine injury, such as focal neurologic deficit, have a history of high-risk activity, or exhibit altered mental status are considered to be at a higher risk for spine injury. This does not include patients with altered mental status who were witnessed to have no trauma as an inciting event. Treatment considerations for this population should be carried out in accordance with the most current version of Wilderness Medical Society Clinical Practice Guidelines for Spinal Cord Protection. **Recommendation Grade:** 1C

Postresuscitation Management

OXYGENATION/VENTILATION

Mechanical ventilation

No literature is available comparing out-of-hospital or in-hospital mechanical ventilation strategies for the drowning patient. Current practice recommends a lung protective ventilation strategy similar to that used for patients with acute

respiratory distress syndrome (ARDS), on the premise that the lung injury pattern after drowning is similar.[16, 80, 81] This includes mechanical ventilation starting with a tidal volume (V_T) of 6 to 8 mL· kg^{-1}, augmentation of V_T and respiratory rate to maintain plateau pressure < 30 mm Hg, and augmentation of positive end expiratory pressure and fraction of inspired oxygen (F_IO_2) to maintain partial pressure of arterial oxygen (P_aO_2) at 55 to 80 mm Hg.[82]

Recommendation

Mechanical ventilation for the drowning patient should follow ARDS protocols.
Recommendation Grade: 1C

Noninvasive positive pressure ventilation

Noninvasive positive pressure ventilation (NIPPV) has been used successfully in the out-of-hospital setting. There are case reports describing its successful use in drowning.[83, 84, 85, 86] Similar to invasive ventilation, the addition of airway pressure to prevent atelectasis and support respiratory muscle use while preventing hypoxemia can be achieved with NIPPV. However, caution should be used with NIPPV in the drowning patient with altered mental status because there may be increased risk of vomiting and aspiration. Drowning patients who have mild to moderate hypoxemia and are being treated in out-of-hospital and emergency medical systems using NIPPV might benefit from this therapy. One small retrospective study showed similar neurologic outcomes and correction of hypoxemia and acidosis between patients treated with early endotracheal intubation versus NIPPV after drowning; in addition, patients receiving NIPPV had a lower incidence of infection and decreased hospital and intensive care unit length of stay.[87]

Recommendation

NIPPV may be usd in the alert drowning patient with mild to moderate respiratory symptoms. Caution should be taken with any patient displaying altered mental status and/or active emesis owing to the potential for aspiration.
Recommendation Grade: 2C

Diagnostics

RADIOLOGIC TESTING

Several retrospective ED studies of drowning patients found that the initial chest radiograph did not correlate with arterial blood gas levels, outcome, or disposition.[88, 89, 90] A study of admitted drowning patients showed that those who went on to develop acute lung injury or ARDS had abnormal chest radiograph findings within the first few hours, but not necessarily on arrival to the ED.[80] Head computed tomography (CT) has been studied in an attempt to

quantify anoxic brain injury in drowning patients. Retrospective studies have found that patients with abnormal initial CT all went on to develop severe brain injury or die, whereas initially normal head CT had no prognostic value.[91]

Recommendation

Initial chest radiograph findings do not correlate with arterial blood gas measurements or outcome; x-rays may be useful in tracking changes in patient condition, but not for determining prognosis if obtained at the time of presentation. A normal initial head CT does not have prognostic value in the drowning patient. Routine use of neuroimaging in the awake and alert drowning patient is not recommended unless dictated by a change in clinical status. **Recommendation Grade:** 1C

LABORATORY TESTING

Canine studies performed in the 1960s showed clinically significant hemodilution and red blood cell lysis associated with salt, chlorine, and freshwater drowning.[92, 93, 94] These studies were based on instilling up to 44 $mL \cdot kg^{-1}$ of fluid into the trachea of anesthetized dogs, far greater than the 1 to 3 $mL \cdot kg^{-1}$ typically aspirated by human drowning patients. Electrolyte abnormalities and hemodilution only occurred in dogs that had 11 $mL \cdot kg^{-1}$ or more instilled. No studies have identified clinically significant electrolyte or hematologic abnormalities in drowning patients that help guide initial therapy or provide prognostic information. In patients with altered mental status or decreased level of consciousness, laboratory evaluation for alternative causes that might have led to the drowning event, such as hypoglycemia or intoxication, can be helpful. Arterial blood gas analysis in symptomatic patients can be used to help guide respiratory resuscitation.

Recommendation

Routine use of complete blood count or electrolyte testing in the drowning patient is not recommended. Arterial blood gas testing in patients with evidence of hypoxemia or respiratory distress (eg, cyanosis, low oxygen saturation, tachypnea, persistent tachycardia) may be indicated to guide respiratory interventions. For patients whose mental status fails to respond to resuscitation or in whom the initial cause of submersion is unknown, laboratory testing for causes of altered mental status or any inciting event should be considered. **Recommendation Grade:** 1C

Other Treatments

ANTIBIOTICS

Although microorganisms present in aspirated water may eventually cause pneumonia, no study to date has shown benefit from empiric administration of antibiotics in drowning patients. This is in part because microorganisms found in

drowning-associated pneumonia are atypical bacteria or fungi and often are resistant to standard empiric treatments.[95, 96, 97] Aspiration of even small volumes of water can produce abnormalities on chest radiograph that can mimic pneumonia. The trauma of the drowning event and hypoxemia can cause leukocytosis from stress demargination as well as fever from inflammation and irritation caused by water in the airways, making it difficult to differentiate inflammatory from infectious pneumonitis.[98] The decision to administer antibiotics should be made after initial resuscitation and ideally be based on expectorated sputum or endotracheal aspirate bacterial culture, blood cultures, or urinary antigen tests.[95, 96, 97] Because these tests are not available in the wilderness setting, treatment should be initiated for symptoms consistent with pulmonary infection (eg, fever, increased sputum, abnormal lung auscultation) that continue after initial resuscitation and treatment phases.

Recommendation

There is no evidence to support empiric antibiotic therapy in the initial treatment of drowning patients. After initial resuscitation, if pneumonia is present, treatment should be guided by expectorated sputum or endotracheal aspirate bacterial culture, blood cultures, or urinary antigen tests. In the absence of these tests, decision to treat should be based on clinical examination focusing on physical evidence of pulmonary or systemic infection (eg, fever, increased sputum, abnormal lung auscultation). **Recommendation Grade:** 1A

CORTICOSTEROIDS

Corticosteroids were historically used in drowning patients to facilitate pulmonary recovery and surfactant production. However, there is not sufficient evidence to support empiric corticosteroid administration for drowning patients.[99]

Recommendation

Given limited data, corticosteroids should not be routinely administered specifically for treatment of drowning patients. **Recommendation Grade:** 1C

THERAPEUTIC HYPOTHERMIA

Mild therapeutic hypothermia (TH) has been shown to decrease cerebral oxygen utilization and improve neurologically intact survival in patients with witnessed VF cardiac arrest.[81] Current American Heart Association/International Liaison Committee on Resuscitation guidelines recommend targeted temperature management for adults after cardiac arrest, at a temperature between 32 and 34°C for at least 24 h.[100] Many institutions have extrapolated these data to include non-VF causes of cardiac arrest.

The 2002 World Congress on Drowning provided a consensus statement recommending TH of 32 to 34°C (90 to 93°F) for patients achieving ROSC after cardiac arrest due to drowning.[101] Our literature search yielded multiple case reports and retrospective reviews supporting neurologically intact survival in

hypothermic patients, but several older studies showed no benefit.[102], [103], [104], [105], [106], [107], [108], [109], [110], [111], [112], [113], [114] There is no prospective study comparing TH to normothermia after ROSC in drowning patients. There might be benefit to discontinuing rewarming interventions after a hypothermic drowning patient has reached TH temperature range, but this has been insufficiently studied to support an evidence-based recommendation.

Recommendation

Although current literature recommend targeted temperature management in postcardiac arrest care, there is insufficient evidence to either support or discourage induction or maintenance of TH in drowning patients.
Recommendation Grade: 2C

Disposition in the Wilderness

DECISION TO EVACUATE

If a patient survives a drowning event in the wilderness, objective physical examination findings may assist in the decision to evacuate the patient to advanced medical care. A single large retrospective study of nearly 42,000 ocean lifeguard rescues serves as the primary evidence for on-scene decision-making.[115] This study found that patients who experienced a drowning event but had no symptoms other than mild cough and who did not have abnormal lung sounds had 0% mortality. As symptoms worsened and abnormal lung sounds appeared, mortality increased. Hypotension (systolic blood pressure < 90 mm Hg or mean arterial pressure < 60 mm Hg) accounted for the next largest increase in mortality (Table 1). In a retrospective study of children who experienced nonfatal drowning, any clinical deterioration occurred within the first 4 h in patients presenting with mild symptoms and Glasgow Coma Scale score ≥13.[88] These findings are similar to those from another retrospective study of pediatric patients in which new symptom development after arrival to the hospital occurred within 4.5 h in all but 1 patient; the 1 outlier developed symptoms in 7 h and had a good outcome.[116] Additional recent emergency department studies are discussed in the *Disposition in Emergency Department* section of these guidelines. These studies revealed similar results in the fact that clinical decompensation, if present, occurred in the first few hours of observation.[26]

Table 1. Out-of-hospital management and classification of drowning patients

Grade	*Pulmonary exam*	*Cardiac exam*	*Mortality (%)*
0	Normal auscultation, without cough	Radial pulses	0
1	Normal auscultation, with cough	Radial pulses	0
2	Rales, small foam in airway	Radial pulses	0.6
3	Acute pulmonary edema	Radial pulses	5
4	Acute pulmonary edema	Hypotension	19
5	Respiratory arrest	Hypotension	44
6	Cardiopulmonary arrest		93

Adapted from Semprsott et al.[25]

Recommendation:

1.

Any patient with abnormal lung sounds, severe cough, frothy sputum, foamy material in the airway, depressed mentation, or hypotension warrants immediate evacuation to advanced medical care if risks of evacuation do not outweigh potential benefit.

2.

Any patient who is asymptomatic (other than a mild cough) and displays normal lung auscultation may be considered for release from the scene. Ideally, another individual should be with them for the next 4 to 6 h to monitor for symptom development or the patient should be advised to seek medical assistance if symptoms develop.

3.

If evacuation is difficult or may compromise the overall expedition, patients with mild symptoms and normal mentation should be observed for 4 to 6 h. Any evidence of decompensation warrants prompt evacuation if the risks of evacuation do not outweigh the potential benefit.

4.

If evacuation of a mildly symptomatic patient has begun and the patient becomes asymptomatic for 4 to 6 h, canceling further evacuation and continuing previous activity may be appropriate.

Recommendation Grade: 1C

CEASING WATER-BASED RESCUE AND RESUSCITATION EFFORTS

A wilderness search and rescue team can range from a small group of untrained participants with no equipment to a highly trained team with extensive resources. In the wilderness setting, available resources, risk to rescuers, and team safety

must be considered when deciding how long to search for a submerged patient. Although each drowning episode has unique patient and environmental factors, the most important predictor of outcome is duration of submersion.[67], [117], [118] Available evidence shows that prognosis is poor with submersion times greater than 30 min, regardless of water temperature.[119] There are also case reports of survival with good neurologic outcome despite prolonged submersion, predominantly in children aged ≤ 6 y in water < 6°C (43°F) and with use of advanced treatment modalities, such as extracorporeal membrane oxygenation.[120], [121], [122], [123], [124], [125] For the purpose of these guidelines, recommendations are based on available evidence relevant to a typical drowning patient and on the probability of neurologically intact survival in specific conditions. A literature review of 43 cases serves as the evidence for water-based rescue.[126] The report concludes that there is minimal chance of neurologically intact survival with submersion time > 30 min in water > 6°C (43°F) or > 90 min in water < 6°C (43°F). It is important to note that “submersion time” was defined as beginning upon arrival of emergency services personnel; total submersion time is often unknown.

If a drowning patient is removed from the water and resuscitation takes place, it might be necessary to decide when to cease resuscitation efforts if no signs of life return. Based primarily on retrospective studies, submersion times of >10 min appear to correlate with increased mortality or survival with severe neurologic dysfunction.[67], [118], [127] In addition, more than 25 min of resuscitation or prolonged time to advanced medical care also correlate with negative outcomes, but without the statistical significance of submersion time. In a Dutch retrospective review of 160 hypothermic drowning patients under the age of 16 y, 98 children received CPR for more than 30 min, with only 11 surviving to discharge, all of whom were neurologically devastated.[119], [127], [128], [129]

Recommendation:

1.

Based on resources, it might be reasonable to cease rescue and resuscitation efforts when there is a known submersion time of greater than 30 min in water > 6°C (43°F), or greater than 90 min in water < 6°C (43°F), or after 25 min of continuous cardiopulmonary resuscitation.

2.

If at any point during search and rescue efforts the safety of the rescue team becomes threatened, rescue efforts should be ceased.

3.

If resources are available and recovery team safety is maintained, body recovery efforts may continue beyond the search and rescue period with the understanding that resuscitation attempts will likely be futile.

Recommendation Grade: 1C

Disposition in the Emergency Department

Although many studies have addressed prognostic factors for neurologic survival at hospital discharge, only a few have addressed the question of which patients can be safely discharged from the ED. The first, a prospective study of primarily pediatric patients, included follow-up phone interviews with 33 patients who were either released on the scene or discharged from the ED within 1 to 6 h of arrival and found that none of these patients experienced delayed effects.[130] A retrospective review of 48 pediatric drowning patients who presented to a single ED with Glasgow Coma Scale score ≥13 studied whether factors predicting safe ED discharge could be identified.[88] Initial chest radiograph did not correlate with severity of disease, and all patients who deteriorated did so within 4 h of ED arrival. The authors concluded that patients could be safely discharged home if normalized and if there was no deterioration in respiratory function after 4 to 6 h of observation in the ED. A retrospective review of hospitalized pediatric patients found that in all patients who were initially asymptomatic, but who went on to develop symptoms during their stay, these symptoms developed within 4.5 h in all but 1 patient and did so within 7 h in the final patient.[116] In the 2 y preceding this current guideline update, 3 more pertinent retrospective studies investigating safe discharge of pediatrics patients were published.[90], [131], [132] The findings of these articles are in line with the aforementioned studies in that patients who initially presented as normal or with minimal symptoms, with normal mentation, and with no need for airway support generally could be safely discharged. Patients in this group who had a clinical decline did so within the first few hours and had subsequent safe discharge. One of the studies derived and validated a clinical score to assist in determining which patients may be safely discharged after 8 h of ED observation. The study found that the presence of 4 or more of the following factors predicted safe discharge: normal mentation, normal respiratory rate, absence of dyspnea, absence of need for airway support, and absence of hypotension.[132]

Recommendation

After an observation period of 4 to 6 h, it is reasonable to discharge a drowning patient with normal mental status in whom respiratory function is normalized and no further deterioration in respiratory function has been observed.
Recommendation Grade: 2C

Prevention

Prevention has the potential to save far more lives than rescue or treatment of a drowning person. A comprehensive prevention program includes participant screening for medical diseases that increase risk of drowning, swimming ability, use of safety devices, and use of safe practices when in and around water.

PARTICIPANT SCREENING

Retrospective studies have linked coronary artery disease, prolonged QT syndrome, autism, and seizure disorders with higher than normal rates of drowning and drowning deaths.[62], [133], [134], [135], [136], [137], [138], [139], [140]

Preparticipation screening should focus on uncovering any medical or physical condition that may potentially impair decision making, physical abilities, and thus swimming ability. These include a history of spontaneous syncope, exertional syncope, and family history of sudden cardiac death. There remains no reliable screening tool for evaluation of cardiac conduction disorders, but screening electrocardiogram and family history of sudden cardiac death can help clinicians differentiate which patients might benefit from further evaluation or genetic testing if indicated.

Recommendation

All patients with coronary artery disease, prolonged QT syndrome or other ion channel disorder, autism, seizure disorders, or other medical and physical impairments should be counseled about the increased risk of drowning and about steps to mitigate the risk, such as buddy swimming and rescue devices, should they choose to participate in water activities. Given the extremely high rate of drowning in patients with epilepsy, patients should be counseled to never swim without direct supervision. **Recommendation Grade:** 2C

SWIMMING ABILITY

Common sense dictates that an individual who is a competent swimmer and has the neurocognitive ability to make appropriate decisions about water safety has a decreased likelihood of drowning. However, the best ages to learn technique and specific swimming skills that reduce a person's chance of drowning are not well established. Most literature evaluates infant and pediatric populations for the effects of swimming and the effects of infant survival lessons on drowning and mortality.[26, 141] There is concern that by providing swim lessons to young children, parents may develop a false sense of security in their child's swimming ability, which might lead to increased drowning incidents.[27, 28, 142]

The American Academy of Pediatrics has always maintained that children should learn to swim at some point in their life. Previous recommendations were against formal swim lessons for all children age 4 y and under. The most recent review by the American Academy of Pediatrics acknowledges a lack of evidence surrounding pediatric swimming lessons and so does not formally recommend for or against lessons for children under age 4 y.[141]

There is considerable debate regarding the definition of "swimming" or "survival-swimming" and what constitutes the most protective approach to swim instruction. Although the ability to swim farther distances can be perceived as increased swim ability, for the purpose of swimming as a tool for drowning prevention, the distance of 25 m (82 ft) has been adopted by international lifesaving agencies and a large population-based study in Bangladesh.[143, 144]

Despite the lack of definitive evidence showing clear benefit to formal swim lessons, panel members agree that familiarity with and, more importantly, confidence in an aquatic environment would be beneficial in the event of

accidental immersion or submersion. In addition, unique aquatic environments, such as whitewater, should be approached only after focused instruction on swimming techniques specific to that environment.

Recommendation

All persons who participate in activities conducted in or around water should have, at a minimum, enough experience and physical capability to maintain their head above water, tread water, and make forward progress for a distance of 25 m (82 ft). **Recommendation Grade:** 2C

PERSONAL FLOTATION DEVICES

Within the category of personal flotation devices, devices such as lifejackets, manually or automated inflation systems, and neoprene wetsuits are available. Currently, lifejackets are the only devices with injury prevention data available and will, therefore, be used as the prototypical model for this category. In 2017, according to United States Coast Guard data, drowning was the cause of death in more than 76% of fatal boating accidents.[5] In addition, 85% of these fatalities were not wearing lifejackets. Three other retrospective studies have found an association between lifejacket use and decreased mortality in boating accidents.[145], [146], [147] One of these studies compared drowning deaths before and after increased lifejacket regulations, revealing improved survival rates after regulations went into effect. These data suggest that activities in and around water, especially while boating, should include lifejacket use.[145]

Recommendation

Properly fitted lifejackets that meet local regulatory specifications should be worn by participants when boating or engaging in any water sports for which lifejackets are recommended. **Recommendation Grade:** 1C

ALCOHOL USE

Alcohol is a known contributing factor to drowning deaths. Data obtained primarily from telephone studies likely underrepresents the true burden of alcohol in drowning causation. In 2017, alcohol was a leading factor in boating-related deaths.[5] A 2004 review found that 30 to 70% of drowning fatalities have a measurable blood alcohol level, with 10 to 30% of deaths being directly attributed to alcohol use.[148]

Recommendation

Alcohol and other intoxicating substances should be avoided before and during water activities. **Recommendation Grade:** 1C

LIFEGUARDS

There are no specific peer-reviewed studies on the utility of lifeguards on expeditions or wilderness trips.[149] A 2001 Centers for Disease Control and Prevention working group report recommends the presence of lifeguards for drowning prevention in open water settings. In 2017, the United States Lifesaving Association reported over 8 million preventative actions and over 75,000 water rescues covering a population of almost 386 million beachgoers. There were 17 reported drowning deaths at guarded beaches compared with 131 deaths at beaches without lifeguards.[150] Among nationally recognized lifeguard certifying agencies (Ellis & Associates, American Red Cross, Starfish Aquatics Institute, and National Aquatic Safety Company) there are no specific guidelines or recommendations for the number of lifeguards per number of participants in an event or at an aquatic facility.

Recommendation

Despite a lack of definitive evidence, all groups operating in or near aquatic environments, regardless of size, should consider water safety during planning and execution of excursions. This includes contingencies for prevention, rescue, and treatment of drowning persons. In high-risk environments or large groups, consider including personnel with technical rescue training and appropriate rescue equipment. **Recommendation Grade:** 1C

Special Situations

COLD WATER SURVIVAL

No single recommendation can address all possible scenarios in a water setting. An unintentional fall into a swift moving river, deep offshore ocean, inland waterways, backyard swimming pool, or through ice into static or moving water are all treated according to the skill level, preparation, and equipment available to patient and rescuer. Immediate attention must always be given to self-rescue and extricating oneself from a hazardous environment. After immersion in cold water, a person has a limited amount of time before fatigue and incapacitation render self-rescue impossible. Likelihood of survival is increased by having appropriate gear and training and by dressing for water temperature, not just air temperature, in the event of immersion.

Extensive controlled trials of cold-water survival are lacking, and the available literature is not generalizable to all scenarios. For example, presence of a lifejacket, sea state, weather, physical fitness, clothing, and mental preparedness all contribute to survivability in cold water. Whitewater is different from still water or the ocean in polar regions. A single large literature review serves as the source for recommendations about cold water survival under ideal conditions and must be interpreted according to the level of training, preparation, and situation presented to the patient.[151]

After immersion, the most important decisions a person must make are: 1) assessment of the presence of any potential immediate threats to life and 2) whether to swim to safety or await rescue. Should a person choose to await

rescue, preventing loss of body heat becomes paramount. By positioning the body to protect major areas of heat loss, a patient may lengthen immersion survival time. A position that has been proven in a laboratory setting to decrease heat loss is the heat escape lessening position. The goal of this position is to decrease heat loss from areas such as the arm pits, groin, and, to a lesser extent, neck. This position is achieved by pressing the arms against the sides of the chest and squeezing the legs together. If possible, additional protection may be obtained by flexing the hips and knees and shrugging the shoulders. In some cases, it may be possible to pull the knees to the chest with the hands. Some individuals will be unstable in this position; in this case the arms can simply be folded across the chest. In the event of group immersion, the huddle formation has been recommended to lessen heat loss, assist injured or weak persons, and improve group morale. Although this position has been shown to decrease cooling in participating individuals in a controlled environment, the effort needed to assist debilitated individuals in an actual emergency may result in increased heat loss (Figure 1, Figure 2).[152]

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Figure 1. Heat escape lessening position (used with permission from www.Boat-Ed.com).

1. Download : Download high-res image (481KB)
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Figure 2. Huddle formation (used with permission from www.Boat-Ed.com).

Swimming or treading water should be limited to minimize heat loss. Life jackets should be worn to aid insulation and flotation. If possible, the ideal location to await rescue is out of the water, even if only partially, to reduce heat loss and delay onset of hypothermia. Prolonged cold-water exposure eventually results in motor disabilities, which can appear within 10 min of immersion, making advanced maneuvers difficult. For this reason, it may be beneficial to affix one's body or clothing to a floating object using rope, or freezing clothing to the ice surface if exit is not possible. Prolonged immersion will also eventually lead to cognitive disabilities, rendering decision-making difficult.

Should a person decide to swim to safety, some important physiologic changes may occur. The initial cold shock, which lasts seconds to a few minutes, may prompt gasping and hyperventilation and can have a disorienting effect, making self-rescue attempts difficult. Upon immersion in cold water, if no immediate life threats are present, a person should focus on remaining calm and controlling breathing by taking slow, deep breaths. Once a person is able to obtain his or her bearings, he or she may have far less than 10 m of effective swimming, and up to 1 h of consciousness, before succumbing to hypothermia. All of these statements assume the person is wearing an appropriate lifejacket. Further detailed discussion of the science behind cold water immersion is available in chapter 8 of *Wilderness Medicine* (7th edition).[30]

Recommendations:

1.

Upon falling into cold water, distance oneself from any immediate life threats (eg, fire, sinking vehicle, whitewater, hazardous waves, rocks). Then, remain calm and focused and control breathing by taking slow deep breaths.

2.

Consider physical capabilities, location, resources, and chances of rescue to determine whether to swim to safety.

3.

If a decision is made to swim to safety, this should be done as soon as possible before physical capabilities deteriorate from the effects of cold stress.

4.

If a decision is made to await rescue, an attempt should be made to remove as much of the body from the water as possible. All clothing should remain on, unless it hampers buoyancy. Most clothing does not compromise buoyancy and will not pull one down, although the water within the garment may impede movement. If the person remains immersed and has a flotation garment on, the heat escape lessening position should be maintained if possible. In a group, the huddle position may be used.

5.

If prolonged rescue is expected, it might be beneficial to attach oneself to a buoyant object or to a surface out of the water to improve the chance for survival.

Recommendation Grade: 2C

Conclusions

Drowning is a process with outcomes ranging from no morbidity to severe morbidity to death. The most important aspect of treatment is to reverse cerebral hypoxia by providing oxygen to the brain. Drowning prevention can be effective and should be thoroughly deployed.

Author Contributions

All authors contributed to drafting, revision, and approval of final manuscript.

Disclosures

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Supplementary materials

The following is the supplementary data related to this article.Download : Download Word document (13KB)

Table 1. American College of Chest Physicians (ACCP) classification scheme for grading evidence and recommendations in clinical guidelines

References

1

World Health Organization**Preventing drowning: an implementation guide**
Geneva (2017)
Google Scholar

2

E.F. van Beeck, C.M. Branche, D. Szpilman, J.H. Modell**Bierens JJ. A new definition of drowning: towards documentation and prevention of a global public health problem**
Bull World Health Organ, 83 (11) (2005), pp. 853-856
View Record in ScopusGoogle Scholar

3

A.C. Schmidt, J.R. Sempsrott, S.C. Hawkins, A.S. Arastu, T.A. Cushing, P.S. Auerbach**Wilderness Medical Society practice guidelines for the prevention and treatment of drowning**
Wilderness Environ Med., 27 (2) (2016), pp. 236-251
ArticleDownload PDFView Record in ScopusGoogle Scholar

4

G. Guyatt, D. Gutterman, M.H. Baumann, D. Addrizzo-Harris, E.M. Hylek, B. Phillips, *et al.***Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American college of chest physicians task force**
Chest., 129 (2006), pp. 174-181
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

5

Centers for Disease Control and Prevention (CDC). Unintentional drowning: get the facts. Available at: https://www.cdc.gov/homeandrecreationalsafety/water-safety/waterinjuries-factsheet.html. Accessed November 7, 2017.
Google Scholar

6

United States Coast Guard. 2017 recreational boating statistics. Available at: http://uscgboating.org/library/accident-statistics/Recreational-Boating-Statistics-2017.pdf. Accessed November 7, 2018.
Google Scholar

7

O. Alonge, P. Agrawal, A. Talab, Q.S. Rahman, A.F. Rahman, S.E. Arifeen, *et al.***Fatal and non-fatal injury outcomes: results from a purposively sampled census of seven rural subdistricts in Bangladesh**
Lancet Glob Health, 5 (8) (2017), pp. e818-e827
ArticleDownload PDFView Record in ScopusGoogle Scholar

8

S.A. Abdullah, M.S. Flora**Risk factors for nonfatal drowning in children in rural Bangladesh: a community-based case-control study**
WHO South East Asia J Public Health, 2 (2) (2013), pp. 88-95
View Record in ScopusGoogle Scholar

9

H. Felton, J. Myers, G. Liu, D.W. Davis**Unintentional, non-fatal drowning of children: US trends and racial/ethnic disparities**
BMJ Open, 5 (12) (2015), Article e008444
CrossRefGoogle Scholar

10

A. Rahman, O. Alonge, A.A. Bhuiyan, P. Agrawal, S.S. Salam, A. Talab, *et al.***Epidemiology of drowning in Bangladesh: an update**
Int J Environ Res Public Health, 14 (5) (2017), p. 488
CrossRefGoogle Scholar

11

G. Reijnen, M. van de Westeringh, M.C. Buster, P.J.E. Vos, U.L.J. Reijnders**Epidemiological aspects of drowning and non-fatal drowning in the waters of Amsterdam**
J Forensic Legal Med, 58 (2018), pp. 78-81
ArticleDownload PDFView Record in ScopusGoogle Scholar

12

R.W. Byard**Drowning and near drowning in rivers**
Forensic Sci Med Pathol, 13 (3) (2017), p. 396
CrossRefView Record in ScopusGoogle Scholar

13

B.L. Matthews, E. Andrew, R. Andronaco, S. Cox, K. Smith**Epidemiology of fatal and non-fatal drowning patients attended by paramedics in Victoria, Australia**
Int J Inj Control Saf Promot, 24 (3) (2017), pp. 303-310
CrossRefView Record in ScopusGoogle Scholar

14

C.C. Irwin, R.L. Irwin, T.D. Ryan, J. Drayer**The legacy of fear: is fear impacting fatal and non-fatal drowning of African American children?**
J Black Stud, 42 (4) (2011), pp. 561-576
CrossRefView Record in ScopusGoogle Scholar

15

W.J. Ma, S.P. Nie, H.F. Xu, Y.J. Xu, X.L. Song, Q.Z. Guo, *et al.***An analysis of risk factors of non-fatal drowning among children in rural areas of**

Guangdong province, China: a case-control study
BMC Public Health, 10 (2010), p. 156
View Record in ScopusGoogle Scholar

16
D. Szpilman, J.J. Bierens, A.J. Handley, J.P. Orlowski**Drowning**
N Engl J Med, 366 (2012), pp. 2102-2110
View Record in ScopusGoogle Scholar

17
A.H. Idris, R.A. Berg, J. Bierens, L. Bossaert, C.M. Branche, A. Gabrielli, *et al.***Recommended guidelines for uniform reporting of data from drowning: The "Utstein Style"**
Circulation., 108 (2003), pp. 2565-2574
View Record in ScopusGoogle Scholar

18
S. Hawkins, J. Semprott, A. Schmidt**'Drowning' in a sea of misinformation**
Emerg Med News., 39 (8) (2017), pp. 39-40
CrossRefView Record in ScopusGoogle Scholar

19
A. Schmidt, J. Semprott, S. Hawkins**Special report: the myth of dry drowning remains at large**
Emergency Medicine News, 40 (6) (2018), p. 22
View Record in ScopusGoogle Scholar

20
D. Szpilman, J. Sempsrott, J. Webber, S.C. Hawkins, R. Barcala-Furelos, A. Schmidt, *et al.***'Dry drowning' and other myths**
Cleve Clin J Med, 85 (7) (2018), pp. 529-535
View Record in ScopusGoogle Scholar

21
R.C. Franklin, J.H. Pearn**Drowning for love: the aquatic victim-instead-of-rescuer syndrome: drowning fatalities involving those attempting to rescue a child**
J Paediatr Child Health, 47 (1–2) (2011), pp. 44-47
CrossRefView Record in ScopusGoogle Scholar

22
A. Turgut**A study on multiple drowning syndromes**
Int J Inj Control Saf Promot, 19 (2012), pp. 63-67
CrossRefView Record in ScopusGoogle Scholar

23
A. Turgut, T. Turgut**A study on rescuer drowning and multiple drowning incidents**
J Saf Res, 43 (2) (2012), pp. 129-132
ArticleDownload PDFView Record in ScopusGoogle Scholar

24

K. Moran, T. Stanley**Bystander perceptions of their capacity to respond in a drowning emergency**
Int J Aquatic Res Educ, 7 (4) (2013), pp. 290-300
View Record in ScopusGoogle Scholar

25

J. Pearn, R. Franklin**"Flinging the squalor" lifeline rescues for drowning prevention**
Int J Aquatic Res Educ (2009), pp. 315-321
View Record in ScopusGoogle Scholar

26

R.A. Brenner, G.S. Taneja, D.L. Haynie, A.C. Trumble, C. Qian, R.M. Klinger, *et al.***Association between swimming lessons and drowning in childhood: a case-control study**
Arch Pediatr Adolesc Med, 163 (3) (2009), pp. 203-210
CrossRefView Record in ScopusGoogle Scholar

27

B.A. Morrongiello, M. Sandomierski, D.C. Schwebel, B. Hagel**Are parents just treading water? The impact of participation in swim lessons on parents' judgments of children's drowning risk, swimming ability, and supervision needs**
Accid Anal Prev, 50 (2013), pp. 1169-1175
ArticleDownload PDFView Record in ScopusGoogle Scholar

28

B.A. Morrongiello, M. Sandomierski, J.R. Spence**Changes over swim lessons in parents' perceptions of children's supervision needs in drowning risk situations: "his swimming has improved so now he can keep himself safe"**
Health Psychol, 33 (7) (2014), pp. 608-615
CrossRefView Record in ScopusGoogle Scholar

29

J. Semprsott, A. Schmidt, S. Hawkins, T. Cushing**Submersion injuries and drowning**
P. Auerbach (Ed.), Wilderness Medicine (7th ed.), Elsevier, Philadelphia (2017), pp. 1530-1549
View Record in ScopusGoogle Scholar

30

G. Giesbrecht, A. Steinman**Immersion into cold water**
P. Auerbach (Ed.), Wilderness Medicine (7th ed.), Elsevier, Philadelphia (2017), pp. 162-197
View Record in ScopusGoogle Scholar

31

G.J. Wintemute, J.F. Kraus, S.P. Teret, M.A. Wright**Death resulting from motor vehicle immersions: the nature of the injuries, personal and environmental contributing factors, and potential interventions**
Am J Public Health, 80 (1990), pp. 1068-1070

CrossRefView Record in ScopusGoogle Scholar

32

J.D. Yale, T.B. Cole, H.G. Garrison, C.W. Runyan, J.K. Ruback**Motor vehicle-related drowning deaths associated with inland flooding after Hurricane Floyd: a field investigation**

Traffic Inj Prev, 4 (2003), pp. 279-284

CrossRefView Record in ScopusGoogle Scholar

33

G.S. Smith, R.A. Brenner**The changing risks of drowning for adolescents in the US and effective control strategies**

Adolesc Med, 6 (2) (1995), pp. 153-170

View Record in ScopusGoogle Scholar

34

P. Lunetta, A. Penttila, A. Sajantila**Drowning in Finland: "external cause" and "injury" codes**

Inj Prev., 8 (4) (2002), pp. 342-344

View Record in ScopusGoogle Scholar

35

J. French, R. Ing, S. Von Allmen, R. Wood**Mortality from flash floods: a review of national weather service reports, 1969–81**

Public Health Rep, 98 (6) (1983), pp. 584-588

View Record in ScopusGoogle Scholar

36

M.M. Agocs, R.B. Trent, D.M. Russell**Activities associated with drownings in imperial county, CA, 1980–90: implications for prevention**

Public Health Rep, 109 (2) (1994), pp. 290-295

View Record in ScopusGoogle Scholar

37

A. Lobeto**Engine company operations: vehicle accidents in water**

Fire Eng, 156 (2003)

Google Scholar

38

G. McDonald, G. Giesbrecht**Vehicle submersion: a review of the problem, associated risks, and survival information**

Aviat Space Environ Med, 84 (5) (2013), pp. 498-510

CrossRefView Record in ScopusGoogle Scholar

39

G.G. Giesbrecht, G.K. McDonald**My car is sinking: automobile submersion, lessons in vehicle escape**

Aviat Space Environ Med, 81 (8) (2010), pp. 779-784

CrossRefView Record in ScopusGoogle Scholar

40

Priority Dispatch Corporation**Medical priority dispatch system ProQA, v5.1.1.23. Current v13.1; last**

update (January 12, 2018)
Google Scholar

41

G.G. Giesbrecht**The evidence base for a new "vehicle in floodwater" emergency dispatch protocol**
Ann Emerg Dispatch Response, 4 (2) (2016), pp. 7-11
View Record in ScopusGoogle Scholar

42

J.P. Orlowski, D. Szpilman**Drowning**
Rescue, resuscitation, and reanimation. *Pediatr Clin North Am*, 48 (3) (2001), pp. 627-646
ArticleDownload PDFView Record in ScopusGoogle Scholar

43

J.L. Ghaphery**In-water resuscitation**
JAMA., 245 (8) (1981), p. 821
CrossRefView Record in ScopusGoogle Scholar

44

N.F. March, R.C. Matthews**New techniques in external cardiac compressions**
Aquatic cardiopulmonary resuscitation *JAMA*, 244 (11) (1980), pp. 1229-1232
CrossRefView Record in ScopusGoogle Scholar

45

D. Szpilman, M. Soares**In-water resuscitation—is it worthwhile?**
Resuscitation., 63 (1) (2004), pp. 25-31
ArticleDownload PDFView Record in ScopusGoogle Scholar

46

G.D. Perkins**In-water resuscitation: a pilot evaluation**
Resuscitation., 65 (3) (2005), pp. 321-324
ArticleDownload PDFView Record in ScopusGoogle Scholar

47

B.E. Winkler, A.M. Eff, S. Eff, U. Ehrmann, A. Koch, W. Kähler, *et al.***Efficacy of ventilation and ventilation adjuncts during in-water-resuscitation—a randomized cross-over trial**
Resuscitation., 84 (2013), pp. 1137-1142
ArticleDownload PDFView Record in ScopusGoogle Scholar

48

B.E. Winkler, A.M. Eff, S. Eff, S. Eff, A. Koch, W. Kaehler, *et al.***Effectiveness and safety of in-water resuscitation performed by lifeguards and laypersons: a crossover manikin study**
Prehosp Emerg Care., 17 (2013), pp. 409-415
CrossRefView Record in ScopusGoogle Scholar

49

The United States Lifeguard Standards Coalition. United States lifeguard standards. http://www.lifeguardstandards.org/. Updated 2011. Accessed

November 10, 2018.
Google Scholar

50

International Life Saving Federation. Medical position statement: in water resuscitation. http://www.ilsf.org/about/position-statements. Updated 2001. Accessed November 15, 2018.
Google Scholar

51

Water Safety Products. BigEasy rescue breathing mask kit. http://www.watersafety.com/store/lifeguard-equipment/bigeasy-rescue-breathing-mask-kit.html. Updated 2015. Accessed November 20, 2018.
Google Scholar

52

The Lifeguard Store. Seal rite mask kit. https://thelifeguardstore.com/seal-rite-mask-kit-22961.html. Accessed November 10, 2018.
Google Scholar

53

K. Zafren, G.G. Giesbrecht, D.F. Danzl, H. Brugger, E.B. Sagalyn, B. Walpoth, *et al.***Wilderness Medical Society practice guidelines for the out-of-hospital evaluation and treatment of accidental hypothermia: 2014 update**
Wilderness Environ Med., 25 (4 Suppl) (2014), pp. S66-S85
ArticleDownload PDFView Record in ScopusGoogle Scholar

54

A. Truhlář, C.D. Deakin, J. Soar, G.E. Khalifa, A. Alfonzo, J.J. Bierens, *et al.***European Resuscitation Council guidelines for resuscitation 2015: section 4. cardiac arrest in special circumstances**
Resuscitation., 95 (2015), pp. 148-201
ArticleDownload PDFView Record in ScopusGoogle Scholar

55

E.J. Lavonas, I.R. Drennan, A. Gabrielli, A.C. Heffner, C.O. Hoyte, A.M. Orkin, *et al.***Part 10: special circumstances of resuscitation: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care**
Circulation., 132 (18 Suppl 2) (2015), pp. S501-S518
CrossRefView Record in ScopusGoogle Scholar

56

K. Adelborg, R.H. Al-Mashhadi, L.H. Nielsen, C. Dalgas, M.B. Mortensen, B. Lofgren**A randomised crossover comparison of manikin ventilation through Soft Seal, i-gel and AuraOnce supraglottic airway devices by surf lifeguards**
Anaesthesia., 69 (4) (2014), pp. 343-347
CrossRefView Record in ScopusGoogle Scholar

57

M. McKenna, M. Davies**Supraglottic airway use by lifeguards**
Anaesthesia., 69 (8) (2014), p. 928
CrossRefView Record in ScopusGoogle Scholar

58

P. Baker, J. Webber**Should supraglottic airway devices be used by lifeguards at all?**
Anaesthesia., 69 (8) (2014), pp. 928-929
CrossRefView Record in ScopusGoogle Scholar

59

P.A. Baker, J.B. Webber**Failure to ventilate with supraglottic airways after drowning**
Anaesth Intensive Care, 39 (2011), pp. 675-677
CrossRefView Record in ScopusGoogle Scholar

60

W. Spindelboeck, O. Schindler, A. Moser, F. Hausler, S. Wallner, C. Strasser, *et al.***Increasing arterial oxygen partial pressure during cardiopulmonary resuscitation is associated with improved rates of hospital admission**
Resuscitation., 84 (6) (2013), pp. 770-775
ArticleDownload PDFView Record in ScopusGoogle Scholar

61

C. Eich, A. Bräuer, A. Timmermann, S.K. Schwarz, S.G. Russo, K. Neubert, *et al.***Outcome of 12 drowned children with attempted resuscitation on cardiopulmonary bypass: an analysis of variables based on the "Utstein Style for drowning"**
Resuscitation., 75 (1) (2007), pp. 42-52
ArticleDownload PDFView Record in ScopusGoogle Scholar

62

S.A. Papadodima, E.I. Sakelliadis, P.S. Kotretsos, S.A. Athanaselis, C.A. Spiliopoulou**Cardiovascular disease and drowning: autopsy and laboratory findings**
Hell J Cardiol, 48 (4) (2007), pp. 198-205
View Record in ScopusGoogle Scholar

63

S. Grmec, M. Strnad, D. Podgorsek**Comparison of the characteristics and outcome among patients suffering from out-of-hospital primary cardiac arrest and drowning victims in cardiac arrest**
Int J Emerg Med, 2 (1) (2009), pp. 7-12
CrossRefView Record in ScopusGoogle Scholar

64

M.A. Ballesteros, M. Gutierrez-Cuadra, P. Munoz, E. Minambres**Prognostic factors and outcome after drowning in an adult population**
Acta Anaesthesiol Scand, 53 (7) (2009), pp. 935-940
CrossRefView Record in ScopusGoogle Scholar

65

M. Nitta, T. Kitamura, T. Iwami, V.M. Nadkarni, R.A. Berg, A.A. Topjian, *et al.***Out-of-hospital cardiac arrest due to drowning among children and adults from the Utstein Osaka project**
Resuscitation., 84 (11) (2013), pp. 1568-1573
ArticleDownload PDFView Record in ScopusGoogle Scholar

66
A. Claesson, J. Lindqvist, J. Herlitz**Cardiac arrest due to drowning—changes over time and factors of importance for survival**
Resuscitation., 85 (5) (2014), pp. 644-648
ArticleDownload PDFView Record in ScopusGoogle Scholar

67
P. Suominen, C. Baillie, R. Korpela, S. Rautanen, S. Ranta, K.T. Olkkola**Impact of age, submersion time and water temperature on outcome in near-drowning**
Resuscitation., 52 (3) (2002), pp. 247-254
ArticleDownload PDFView Record in ScopusGoogle Scholar

68
T. Lyster, D. Jorgenson, C. Morgan**The safe use of automated external defibrillators in a wet environment**
Prehosp Emerg Care, 7 (3) (2003), pp. 307-311
CrossRefView Record in ScopusGoogle Scholar

69
A. Schratter, W. Weihs, M. Holzer, A. Janata, W. Behringer, U.M. Losert, *et al.***External cardiac defibrillation during wet-surface cooling in pigs**
Am J Emerg Med, 25 (4) (2007), pp. 420-424
ArticleDownload PDFView Record in ScopusGoogle Scholar

70
J.C. Klock-Frezot, W.J. Ohley, R.B. Schock, M. Cote, L. Schofield**Successful defibrillation in water: a preliminary study**
Conf Proc IEEE Eng Med Biol Soc, 1 (2006), pp. 4028-4030
View Record in ScopusGoogle Scholar

71
W. de Vries, J.J. Bierens, M.W. Maas**Moderate sea states do not influence the application of an AED in rigid inflatable boats**
Resuscitation., 70 (2) (2006), pp. 247-253
ArticleDownload PDFView Record in ScopusGoogle Scholar

72
H.J. Heimlich, E.G. Spletzer**Drowning**
N Engl J Med, 329 (1) (1993), p. 65
View Record in ScopusGoogle Scholar

73
H.J. Heimlich, E.A. Patrick**Using the Heimlich maneuver to save near-drowning victims**
Postgrad Med, 84 (2) (1988), pp. 62-67

71
CrossRefView Record in ScopusGoogle Scholar

74
H.J. Heimlich**Subdiaphragmatic pressure to expel water from the lungs of drowning persons**
Ann Emerg Med, 10 (1981), pp. 476-480
ArticleDownload PDFView Record in ScopusGoogle Scholar

75
P. Rosen, M. Stoto, J. Harley**The use of the Heimlich maneuver in near drowning: Institute of Medicine report**
J Emerg Med, 13 (9) (1995), pp. 397-405
ArticleDownload PDFView Record in ScopusGoogle Scholar

76
P. Francesco, R. Fielding, P.G. Wernicki, D. Markenson**Sub-diaphragmatic thrusts and drowned persons**
Int J Aquatic Res Educ, 4 (2010), pp. 81-92
View Record in ScopusGoogle Scholar

77
S.C. Hawkins, J. Williams, B.L. Bennett, A. Islas, D.W. Kayser, R. Quinn**Wilderness Medical Society clinical practice guidelines for spinal cord protection: 2019 update**
Wilderness Environ Med, 30 (4S) (2019), pp. S87-S99
ArticleDownload PDFView Record in ScopusGoogle Scholar

78
R.S. Watson, P. Cummings, L. Quan, S. Bratton, N.S. Weiss**Cervical spine injuries among submersion victims**
J Trauma, 51 (4) (2001), pp. 658-662
View Record in ScopusGoogle Scholar

79
V. Hwang, F.S. Shofer, D.R. Durbin, J.M. Baren**Prevalence of traumatic injuries in drowning and near drowning in children and adolescents**
Arch Pediatr Adolesc Med, 157 (1) (2003), pp. 50-53
CrossRefView Record in ScopusGoogle Scholar

80
L. Gregorakos, N. Markou, V. Psalida, M. Kanakaki, A. Alexopoulou, E. Sotiriou, *et al.***Near-drowning: clinical course of lung injury in adults**
Lung., 187 (2) (2009), pp. 93-97
CrossRefView Record in ScopusGoogle Scholar

81
A.A. Topjian, R.A. Berg, J.J. Bierens, C.M. Branche, R.S. Clark, H. Friberg, *et al.***Brain resuscitation in the drowning victim**
Neurocrit Care, 17 (3) (2012), pp. 441-467
CrossRefView Record in ScopusGoogle Scholar

82

ARDS Clinical Network. Mechanical ventilation protocol summary. Available at: http://www.ardsnet.org/. Updated 2008. Accessed November 2, 2018.
Google Scholar

83

J. Thompson, D.A. Petrie, S. Ackroyd-Stolarz, D.J. Bardua**Out-of-hospital continuous positive airway pressure ventilation versus usual care in acute respiratory failure: a randomized controlled trial**
Ann Emerg Med, 52 (3) (2008), pp. 232-241
View Record in ScopusGoogle Scholar

84

M. Dottorini, A. Eslami, S. Baglioni, G. Fiorenzano, T. Todisco**Nasal-continuous positive airway pressure in the treatment of near-drowning in freshwater**
Chest., 110 (4) (1996), pp. 1122-1124
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

85

S. Nava, A. Schreiber, G. Domenighetti**Noninvasive ventilation for patients with acute lung injury or acute respiratory distress syndrome**
Respir Care, 56 (10) (2011), pp. 1583-1588
CrossRefView Record in ScopusGoogle Scholar

86

P. Ruggeri, S. Calcaterra, A. Bottari, G. Girbino, V. Fodale**Successful management of acute respiratory failure with noninvasive mechanical ventilation after drowning, in an epileptic-patient**
Respir Med Case Rep, 17 (2016), pp. 90-92
ArticleDownload PDFView Record in ScopusGoogle Scholar

87

P. Michelet, F. Bouzana, O. Charmensat, F. Tiger, J. Durand-Gasselin, S. Hraiech, *et al.***Acute respiratory failure after drowning: a retrospective multicenter survey**
Eur J Emerg Med, 24 (4) (2017), pp. 295-300
View Record in ScopusGoogle Scholar

88

A.L. Causey, J.A. Tilelli, M.E. Swanson**Predicting discharge in uncomplicated near-drowning**
Am J Emerg Med, 18 (1) (2000), pp. 9-11
ArticleDownload PDFView Record in ScopusGoogle Scholar

89

J.H. Modell, S.A. Graves, A. Ketover**Clinical course of 91 consecutive near-drowning victims**
Chest., 70 (2) (1976), pp. 231-238
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

90

C.E. Brennan, T.K.F. Hong, V.J. Wang**Predictors of safe discharge for pediatric drowning patients in the emergency department**
Am J Emerg Med, 36 (9) (2018), pp. 1619-1623
ArticleDownload PDFView Record in ScopusGoogle Scholar

91

K.T. Rafaat, R.M. Spear, C. Kuelbs, K. Parsapour, B. Peterson**Cranial computed tomographic findings in a large group of children with drowning: diagnostic, prognostic, and forensic implications**
Pediatr Crit Care Med, 9 (6) (2008), pp. 567-572
View Record in ScopusGoogle Scholar

92

J.H. Modell, J.H. Davis**Electrolyte changes in human drowning victims**
Anesthesiology., 30 (4) (1969), pp. 414-420
CrossRefView Record in ScopusGoogle Scholar

93

J.H. Modell, F. Moya**Effects of volume of aspirated fluid during chlorinated fresh water drowning**
Anesthesiology., 27 (5) (1966), pp. 662-672
CrossRefView Record in ScopusGoogle Scholar

94

J.H. Modell, F. Moya, E.J. Newby, B.C. Ruiz, A.V. Showers**The effects of fluid volume in seawater drowning**
Ann Intern Med, 67 (1) (1967), pp. 68-80
CrossRefView Record in ScopusGoogle Scholar

95

C. Wood**Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary. BET 1: prophylactic antibiotics in near-drowning**
Emerg Med J, 27 (5) (2010), pp. 393-394
View Record in ScopusGoogle Scholar

96

P.T. Ender, M.J. Dolan**Pneumonia associated with near-drowning**
Clin Infect Dis, 25 (4) (1997), pp. 896-907
View Record in ScopusGoogle Scholar

97

J.M. Tadié, N. Heming, E. Serve, N. Weiss, N. Day, A. Imbert, *et al.***Drowning associated pneumonia: a descriptive cohort**
Resuscitation., 83 (2012), pp. 399-401
ArticleDownload PDFView Record in ScopusGoogle Scholar

98

M. van Berkel, J.J. Bierens, R.L. Lie, T.P. de Rooy, L.J. Kool, E.A. van de Velde, *et al.***Pulmonary oedema, pneumonia and mortality in submersion victims; a retrospective study in 125 patients**
Intensive Care Med, 22 (2) (1996), pp. 101-107

View Record in ScopusGoogle Scholar

99

B.A. Foex, R. Boyd**Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary. Corticosteroids in the management of near-drowning**
Emerg Med J, 18 (6) (2001), pp. 465-466
View Record in ScopusGoogle Scholar

100

C.W. Callaway, M.W. Donnino, E.L. Fink, R.G. Geocadin, E. Golan, K.B. Kern, *et al.***Part 8: post-cardiac arrest care: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care**
Circulation., 132 (18 Suppl 2) (2015), pp. S465-S482
CrossRefView Record in ScopusGoogle Scholar

101

World Congress on Drowning. Recommendations of the World Congress on Drowning. Available at: http://www.ilsf.org/drowning-prevention/report. Updated 2002. Accessed November 10, 2018.
Google Scholar

102

R.K. Batra, J.J. Paddle**Therapeutic hypothermia in drowning induced hypoxic brain injury: a case report**
Cases J, 2 (2009), p. 9103
Google Scholar

103

J. Varon, P.E. Marik**Complete neurological recovery following delayed initiation of hypothermia in a victim of warm water near-drowning**
Resuscitation., 68 (3) (2006), pp. 421-423
ArticleDownload PDFView Record in ScopusGoogle Scholar

104

J.P. Williamson, R. Illing, P. Gertler, S. Braude**Near-drowning treated with therapeutic hypothermia**
Med J Aust, 181 (9) (2004), pp. 500-501
CrossRefView Record in ScopusGoogle Scholar

105

A.C. de Pont, C.P. de Jager, W.M. van den Bergh, M.J. Schultz**Recovery from near drowning and postanoxic status epilepticus with controlled hypothermia**
Neth J Med, 69 (4) (2011), pp. 196-197
View Record in ScopusGoogle Scholar

106

S.S. Rudolph, S. Barnung**Survival after drowning with cardiac arrest and mild hypothermia**
ISRN Cardiol, 2011 (2011), Article 895625

Google Scholar

107

S.P. Choi, C.S. Youn, K.N. Park, J.H. Wee, J.H. Park, S.H. Oh, *et al.***Therapeutic hypothermia in adult cardiac arrest because of drowning**
Acta Anaesthesiol Scand, 56 (1) (2012), pp. 116-123
CrossRefView Record in ScopusGoogle Scholar

108

R. Kawati, L. Covaciu, S. Rubertsson**Hypothermia after drowning in paediatric patients**
Resuscitation., 80 (11) (2009), pp. 1325-1326
ArticleDownload PDFView Record in ScopusGoogle Scholar

109

Mizobuchi M, Nakamura S, Muranishi H, Utsunomiya M, Funatsu A, Kobayashi T, et al. Hypothermia with extracorporeal membrane oxygenation for sudden cardiac death and submersion. *Am J Emerg Med*. 2010;28(1):115.e1–e4.
Google Scholar

110

S. Baldursdottir, K. Sigvaldason, S. Karason, F. Valsson, G.H. Sigurdsson**Induced hypothermia in comatose survivors of asphyxia: a case series of 14 consecutive cases**
Acta Anaesthesiol Scand, 54 (7) (2010), pp. 821-826
CrossRefView Record in ScopusGoogle Scholar

111

A. Oude Lansink-Hartgring, F. Ismael**Controlled hypothermia and recovery from postanoxic encephalopathy in near-drowning victim**
Neth J Med, 69 (7) (2011), p. 351
View Record in ScopusGoogle Scholar

112

U. Guenther, D. Varelmann, C. Putensen, H. Wrigge**Extended therapeutic hypothermia for several days during extracorporeal membrane-oxygenation after drowning and cardiac arrest two cases of survival with no neurological sequelae**
Resuscitation., 80 (3) (2009), pp. 379-381
ArticleDownload PDFView Record in ScopusGoogle Scholar

113

O.V. Hein, A. Triltsch, C. von Buch, W.J. Kox, C. Spies**Mild hypothermia after near drowning in twin toddlers**
Crit Care, 8 (5) (2004), pp. R353-R357
CrossRefGoogle Scholar

114

D.J. Bohn, W.D. Biggar, C.R. Smith, A.W. Conn, G.A. Barker**Influence of hypothermia, barbiturate therapy, and intracranial pressure monitoring on morbidity and mortality after near-drowning**

Crit Care Med, 14 (6) (1986), pp. 529-534
CrossRefView Record in ScopusGoogle Scholar

115
D. Szpilman**Near-drowning and drowning classification: a proposal to stratify mortality based on the analysis of 1,831 cases**
Chest., 112 (3) (1997)
66–5
Google Scholar

116
L. Noonan, R. Howrey, C.M. Ginsburg**Freshwater submersion injuries in children: a retrospective review of seventy-five hospitalized patients**
Pediatrics., 98 (3 Pt 1) (1996), pp. 368-371
View Record in ScopusGoogle Scholar

117
L. Quan, C.D. Mack, M.A. Schiff**Association of water temperature and submersion duration and drowning outcome**
Resuscitation., 85 (6) (2014), pp. 790-794
ArticleDownload PDFView Record in ScopusGoogle Scholar

118
P.K. Suominen, R. Vahatalo**Neurologic long term outcome after drowning in children**
Scand J Trauma Resusc Emerg Med, 20 (2012), p. 55
CrossRefGoogle Scholar

119
J.K. Kieboom, H.J. Verkade, J.G. Burgerhof, J.J. Bierens, P.F. Rheenen, M.C. Kneyber, *et al.***Outcome after resuscitation beyond 30 minutes in drowned children with cardiac arrest and hypothermia: Dutch nationwide retrospective cohort study**
BMJ., 350 (2015), p. h418
CrossRefGoogle Scholar

120
J.P. Orlowski**How much resuscitation is enough resuscitation?**
Pediatrics., 90 (6) (1992), pp. 997-998
View Record in ScopusGoogle Scholar

121
J.H. Modell, A.H. Idris, J.A. Pineda, J.H. Silverstein**Survival after prolonged submersion in freshwater in Florida**
Chest., 125 (5) (2004), pp. 1948-1951
ArticleDownload PDFView Record in ScopusGoogle Scholar

122
W.R. Hasibeder**Drowning**
Curr Opin Anaesthesiol, 16 (2) (2003), pp. 139-145
View Record in ScopusGoogle Scholar

123

T.G. Martin**Neardrowning and cold water immersion**
Ann Emerg Med, 13 (4) (1984), pp. 263-273
ArticleDownload PDFView Record in ScopusGoogle Scholar

124

M. Gilbert, R. Busund, A. Skagseth, P.A. Nilsen, J.P. Solbo**Resuscitation from accidental hypothermia of 13.7°C with circulatory arrest**
Lancet., 355 (9201) (2000), pp. 375-376
ArticleDownload PDFView Record in ScopusGoogle Scholar

125

M. Wanscher, L. Agersnap, J. Ravn, S. Yndgaard, J.F. Nielsen, E.R. Danielsen, *et al.***Outcome of accidental hypothermia with or without circulatory arrest: experience from the Danish Praesto fjord boating accident**
Resuscitation., 83 (9) (2012), pp. 1078-1084
ArticleDownload PDFView Record in ScopusGoogle Scholar

126

M.J. Tipton, F.S. Golden**A proposed decision-making guide for the search, rescue and resuscitation of submersion (head under) victims based on expert opinion**
Resuscitation., 82 (7) (2011), pp. 819-824
ArticleDownload PDFView Record in ScopusGoogle Scholar

127

L. Quan, D. Kinder**Pediatric submersions: prehospital predictors of outcome**
Pediatrics., 90 (6) (1992), pp. 909-913
View Record in ScopusGoogle Scholar

128

C.S. Youn, S.P. Choi, H.W. Yim, K.N. Park**Out-of-hospital cardiac arrest due to drowning: an Utstein Style report of 10 years of experience from St. Mary's hospital**
Resuscitation., 80 (7) (2009), pp. 778-783
ArticleDownload PDFView Record in ScopusGoogle Scholar

129

A. Claesson, L. Svensson, J. Silfverstolpe, J. Herlitz**Characteristics and outcome among patients suffering out-of-hospital cardiac arrest due to drowning**
Resuscitation., 76 (3) (2008), pp. 381-387
ArticleDownload PDFView Record in ScopusGoogle Scholar

130

F.D. Pratt, B.E. Haynes**Incidence of "secondary drowning" after saltwater submersion**
Ann Emerg Med, 15 (9) (1986), pp. 1084-1087
ArticleDownload PDFView Record in ScopusGoogle Scholar

131

R.M. Cantu, C.M. Pruitt, N. Samuy, C.L. Wu**Predictors of emergency department discharge following pediatric drowning**
Am J Emerg Med, 36 (3) (2018), pp. 446-449
ArticleDownload PDFView Record in ScopusGoogle Scholar

132

R.P. Shenoi, S. Allahabadi, D.M. Rubalcava, E.A. Camp**The pediatric submersion score predicts children at low risk for injury following submersions**
Acad Emerg Med, 24 (12) (2017), pp. 1491-1500
CrossRefView Record in ScopusGoogle Scholar

133

M.J. Ackerman, D.J. Tester, C.J. Porter, W.D. Edwards**Molecular diagnosis of the inherited long-QT syndrome in a woman who died after near-drowning**
N Engl J Med, 341 (15) (1999), pp. 1121-1125
View Record in ScopusGoogle Scholar

134

D.J. Tester, A. Medeiros-Domingo, M.L. Will, M.J. Ackerman**Unexplained drownings and the cardiac channelopathies: a molecular autopsy series**
Mayo Clin Proc, 86 (10) (2011), pp. 941-947
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

135

G.S. Bell, A. Gaitatzis, C.L. Bell, A.L. Johnson, J.W. Sander**Drowning in people with epilepsy: how great is the risk?**
Neurology., 71 (8) (2008), pp. 578-582
CrossRefView Record in ScopusGoogle Scholar

136

L. Albertella, J. Crawford, J.R. Skinner**Presentation and outcome of water-related events in children with long QT syndrome**
Arch Dis Child, 96 (2011), pp. 704-707
CrossRefView Record in ScopusGoogle Scholar

137

R. SoRelle**Genetic drowning trigger**
Circulation., 101 (3) (2000), Article E36
View Record in ScopusGoogle Scholar

138

G. Choi, L.J. Kopplin, D.J. Tester, M.L. Will, C.M. Haglund, M.J. Ackerman**Spectrum and frequency of cardiac channel defects in swimming-triggered arrhythmia syndromes**
Circulation., 110 (15) (2004), pp. 2119-2124
View Record in ScopusGoogle Scholar

139

P. Lunetta, A. Levo, P.J. Laitinen, H. Fodstad, K. Kontula, A. Sajantila**Molecular screening of selected long QT syndrome (LQTS)**

mutations in 165 consecutive bodies found in water
Int J Legal Med, 117 (2) (2003), pp. 115-117
CrossRefView Record in ScopusGoogle Scholar

140
D.J. Tester, L.J. Kopplin, W. Creighton, A.P. Burke, M.J. Ackerman**Pathogenesis of unexplained drowning: new insights from a molecular autopsy**
Mayo Clin Proc, 80 (5) (2005), pp. 596-600
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

141
J. Weiss**American Academy of Pediatrics Committee on Injury, Violence** Prevention of drowning *Pediatrics*, 126 (1) (2010), pp. e253-e262
View Record in ScopusGoogle Scholar

142
K. Moran, T. Stanley**Parental perceptions of toddler water safety, swimming ability and swimming lessons**
Int J Inj Control Saf Promot, 13 (3) (2006), pp. 139-143
CrossRefView Record in ScopusGoogle Scholar

143
T.S. Mecrow, M. Linnan, A. Rahman, J. Scarr, S.R. Mashreky, A. Talab, *et al.***Does teaching children to swim increase exposure to water or risk-taking when in the water? Emerging evidence from Bangladesh**
Inj Prev., 21 (3) (2015), pp. 185-188
CrossRefView Record in ScopusGoogle Scholar

144
Australian Council for the Teaching of Swimming and Water Safety. Audit of national water safety programs. Available at: http://www.watersafety.com.au/Reports.aspx. Updated 2013. Accessed November 20, 2014.
Google Scholar

145
L. Bugeja, E. Cassell, L.R. Brodie, S.J. Walter**Effectiveness of the 2005 compulsory personal flotation device (PFD) wearing regulations in reducing drowning deaths among recreational boaters in Victoria, Australia**
Inj Prev., 20 (6) (2014), pp. 387-392
CrossRefView Record in ScopusGoogle Scholar

146
P. Cummings, B.A. Mueller, L. Quan**Association between wearing a personal flotation device and death by drowning among recreational boaters: a matched cohort analysis of United States Coast Guard data**
Inj Prev., 17 (3) (2011), pp. 156-159
CrossRefView Record in ScopusGoogle Scholar

147

P.J. O'Connor, N. O'Connor**Causes and prevention of boating fatalities**
Accid Anal Prev, 37 (4) (2005), pp. 689-698
ArticleDownload PDFView Record in ScopusGoogle Scholar

148

T.R. Driscoll, J.A. Harrison, M. Steenkamp**Review of the role of alcohol in drowning associated with recreational aquatic activity**
Inj Prev, 10 (2) (2004), pp. 107-113
View Record in ScopusGoogle Scholar

149

C.M. Branche, S. Stewart**Lifeguard effectiveness: a report of the working group. Centers for Disease Control and Prevention, National Center for Injury**
Prev Control (2001)
Google Scholar

150

United States Lifesaving Association. 2017 national lifesaving statistics. Available at: http://arc.usla.org/Statistics/public.asp. Updated 2013. Accessed November 20, 2018.
Google Scholar

151

M.B. Ducharme, D.S. Lounsbury**Self-rescue swimming in cold water: the latest advice**
Appl Physiol Nutr Metab, 32 (4) (2007), pp. 799-807
View Record in ScopusGoogle Scholar

152

J.S. Hayward, J.D. Eckerson, M.L. Collis**Effect of behavioral variables on cooling rate of man in cold water**
J Appl Physiol, 38 (6) (1975), pp. 1073-1077
CrossRefView Record in ScopusGoogle Scholar

Wilderness Medical Society Clinical Practice Guidelines for Water Disinfection for Wilderness, International Travel, and Austere Situations

Howard D.Backer MD, MPH[1] Robert W.Derlet MD[2] Vincent R.Hill PhD[3]

To provide guidance to clinicians, the Wilderness Medical Society convened experts to develop evidence-based guidelines for water disinfection in situations where the potability of available water is not ensured, including wilderness and international travel, areas affected by disaster, and other areas without adequate sanitation. The guidelines present the available methods for reducing or eliminating microbiologic contamination of water for individuals, groups, or households; evaluation of their effectiveness; and practical considerations. The evidence evaluation includes both laboratory and clinical publications. The panel graded the recommendations based on the quality of supporting evidence and the balance between benefits and risks or burdens, according to the criteria published by the American College of Chest Physicians.

Keywords

drinking water
water purification
water microbiology
disaster planning
pasteurization
halogens

Introduction

Safe and efficient treatment of drinking water is among the major public health advances of the last century. Without treatment, waterborne diseases can spread rapidly, resulting in large-scale disease and death.1, 2 In industrialized nations, the population generally is protected from waterborne disease by sophisticated water supply systems that disinfect water and provide continuous monitoring. In contrast, travelers to wilderness and recreational areas anywhere in the world and to underdeveloped regions of some countries may be confronted with untreated or

contaminated water that poses a risk of acquiring enteric disease. In addition, disaster situations, such as the 2017 hurricanes that affected Houston, Texas, and Puerto Rico, may result in a breakdown of municipal water systems, exposing victims to nonpotable water. These situations necessitate knowledge of how to disinfect water at the point-of-use, prior to drinking.

Methods of water treatment that can be applied in the field include the use of heat, ultraviolet light, clarification, filtration, and chemical disinfection. The choices for the wilderness hiker or international traveler are increasing as new technology is applied to field applications. Different microorganisms have varying susceptibilities to these methods. The risk of waterborne illness depends on the number and type of organisms consumed, host factors, and the efficacy of the treatment system.

Methods

To develop these guidelines, specialists with expertise in wilderness medicine, travel medicine, public health, and microbiology were chosen on the basis of their clinical or research experience. Relevant articles were identified through the PubMed database using the following keywords or phrases: water disinfection, waterborne illness, wilderness water, water filtration, emergency or disaster drinking water treatment. This was supplemented by a hand search of articles from references in the initial PubMed search. Conclusions from review articles were cited in an effort to provide background information and to augment reference selection.

The evidence base for water disinfection has substantial differences from other clinical guidelines. Most of the literature concerning the effectiveness of specific disinfectants and methods against various waterborne microorganisms is laboratory based. Evidence on the benefits of disinfection is either population-based public health research of disease outbreaks or randomized household trials of water disinfection that are influenced by compliance and hygiene. Therefore, the evidence grade is a combination of laboratory, population, and household- or community-level studies.

The panel used a consensus approach to develop recommendations for the disinfection of water. Water treatment techniques and recommendations were not evaluated for the removal of chemicals or toxins. Evidence grades were assigned according to methodology stipulated by the American College of Chest Physicians for grading of evidence and recommendations[3] (online Supplementary Table 1). These recommendations are graded on the basis of the totality of supporting evidence and balance between the benefits and risks or burdens for each modality.

Etiology and Risk of Waterborne Infection

WILDERNESS SETTINGS

Millions of people enter wilderness areas each year and drink surface water. Even in developed countries with low rates of diarrheal illness, regular waterborne disease outbreaks indicate that the microbiologic quality of the water, especially surface water, is not ensured.[4, 5, 6, 7] Public health agencies regularly report outbreaks of disease associated with surface water from backcountry and parks as well as from campground water systems. The environment and activity upstream from the travelers' surface water source defines the risk. Side streams draining springs, snowmelt, and glaciers where there is no human or animal activity are lower risk. In contrast, upstream usage by humans, farm animals, or wildlife pose a major risk. Cattle excrete pathogenic strains of *Escherichia coli* and *Salmonella* and have been found in multiple studies to be the major animal species contributing to waterborne disease in North America.[8, 9] Giardiasis is a zoonotic infection with numerous host species, including farm animals, deer and other wild ungulates, beavers, and even household animals; however, the extent of transmission to humans is less defined.[10]

Nonalpine wilderness areas in the United States may have streams and rivers that are contaminated with animal waste, including farm animal runoff, or may be contaminated with incompletely treated sewage from towns and urban areas. In many countries, wilderness areas are co-occupied by local populations and domesticated animals that pollute water sources. Because it is very difficult to exclude animal and human activity in the watershed, the Centers for Disease Control recommend treating surface water before ingestion as a precaution to protect health.

International Travel

Substantial progress has been made in the past 20 years toward the goal of safe drinking water and sanitation worldwide, particularly in Asia and Latin America[11]; however, 780 million people (11% of world population) still lack a safe water source, and 2.5 billion people lack access to improved sanitation. Africa and Oceania are the regions with the greatest need for improvement. More than 890 million persons still practice open defecation, the largest number being in India and Africa.[11, 12, 13] Studies in underdeveloped regions around the world show high levels of microbes in the environment and water sources.[14, 15, 16, 17, 18] Contamination of tap water commonly occurs because of antiquated and inadequately monitored waste disposal, water treatment, and distribution systems.[19, 20]

In both developed and developing countries, after natural disasters such as hurricanes, tsunamis, and earthquakes, one of the most immediate public health problems is a lack of potable water. Wilderness visitors and international travelers have no reliable resources to evaluate local water system quality. Less information is available for remote surface water sources. Appearance, smell, and taste are not reliable indicators to estimate water safety.

Infectious agents with the potential for waterborne transmission include bacteria, viruses, protozoa, and nonprotozoan parasites. The list of microbial agents is similar to the list of microorganisms that can cause travelers' diarrhea, most of which can be waterborne as well as foodborne. Although the primary reason for

disinfecting drinking water is to destroy microorganisms from animal and human biologic wastes, water may also be contaminated with toxins and chemical pollutants from industrial sources or from the environment. *Escherichia coli* and *Vibrio cholerae* may be capable of surviving indefinitely in tropical water. Enteric bacterial and viral pathogens survive in temperate water generally only several days; however, some species such as *E coli* O157: H7 can survive 12 weeks at 25°C.[21] Most enteric organisms, including *Shigella* spp, *Salmonella enterica* serotype Typhi, hepatitis A, and *Cryptosporidium* spp, can retain viability for long periods in cold water and can even survive for weeks when frozen in water.

The risk of waterborne illness depends on the number of organisms consumed, which is in turn determined by the volume of water, concentration of organisms, and treatment system efficiency.[22, 23] Additional factors include virulence of the organism and defenses of the host. Microorganisms with a small infectious dose (eg, *Giardia, Cryptosporidium*, *Shigella* spp, hepatitis A, enterohemorrhagic *E coli,* and norovirus—the leading viral disease risk in water contaminated with human waste) may cause illness even from inadvertent drinking during water-based recreational activities.[10] Most diarrhea among travelers is probably foodborne; however, the capacity for waterborne transmission should not be underestimated. Because long-lasting immunity does not develop for most enteric pathogens, reinfection may occur.

The combined roles of safe water, hygiene, and adequate sanitation in reducing diarrhea and other diseases are clear and well documented. The World Health Organization (WHO) estimates that 94% of diarrheal cases globally are preventable through modifications to the environment, including access to safe water.[1] Recent studies of simple water interventions in households of developing countries clearly document improved microbiological quality of water, a 30 to 60% reduced incidence of diarrheal illness, enhanced childhood survival, and reduction of parasitic diseases, many of which are independent of other measures to improve sanitation.[24]

General recommendations for drinking water disinfection:

- Treat water when traveling in developing countries. **Evidence grade: 1A**
- Treat water in wilderness areas with nearby agricultural use, animal grazing, or upstream human activity. **Evidence grade: 1A**
- Treat water in wilderness settings without evidence of domestic animal and little to no wildlife or human activity. **Evidence grade: 2B**
- Treat water in disaster situations affecting municipal or private drinking water sources. **Evidence grade: 1A**

Water Treatment Methods

Multiple techniques for improving the microbiologic quality of water are available to individuals and small groups while hiking or traveling. Bottled water may be a convenient and popular solution but creates ecologic problems. Furthermore, in underdeveloped countries, the quality of bottled water may not meet the standards of developed countries and may contain pathogenic microbes.[25]

The term *disinfection*, the desired result of field water treatment, is used here to indicate the removal or destruction of harmful microorganisms, which reduces the risk of illness. This is sometimes used interchangeably with *purification*, but the latter term more accurately indicates the removal of organic or inorganic chemicals and particulate matter to improve color, taste, and odor. Unless specifically designed to remove chemical contaminants, disinfection techniques may not make water safe from chemical exposures. *Potable* implies drinkable water, but it technically means that a water source, on average, over a period of time, contains a minimal microbial hazard so that the statistical likelihood of illness is acceptably low. All standards, including water regulations in the United States, acknowledge the impracticality of trying to eliminate all microorganisms from drinking water. Generally, the goal is a 3 to 5 log reduction (99.9–99.999%), allowing a small risk of enteric infection. Newer standards from the US Environmental Protection Agency (US EPA) and the WHO set target goals to reduce some organisms to zero; however, all enforceable standards allow a small risk for enteric infection.[26]

Product Testing and Rating

Filters are rated by their ability to retain particles of a certain size, which is described by 2 terms. *Absolute* rating means that 100% of a certain size of particle is retained by the filter (ie, filtered-out). *Nominal* rating indicates that > 90% of a given particle size will be retained. Filter efficiency is generally determined with hard particles (beads of known diameter), but microorganisms are soft and compressible under pressure. The US EPA and NSF International are the primary agencies that set standards for disinfection products and protocols for testing to meet these standards.

The US EPA does not endorse, test, or approve mechanical filters; it merely assigns registration numbers that distinguish between 2 types of filters: those that use mechanical means only and those that use a chemical designated as a pesticide. Portable water treatment device claims for microbiologic reduction are based on consensus performance standards that serve as a guideline for testing.[27] Testing is done or contracted by the manufacturer; the US EPA neither tests nor specifies laboratories. Testing must be done with bacteria (*Klebsiella*), viruses (poliovirus and rotavirus), and protozoa (*Cryptosporidium* has replaced *Giardia*). A 3-log reduction (99.9%) is required for protozoan cysts, 4-log reduction (99.99%) for viruses, and 5- to 6-log reduction for bacteria. To be called a microbiologic water purifier, the unit must remove, kill, or inactivate all types of disease-causing microorganisms from the water, including bacteria, viruses, and protozoan cysts, so as to render the processed water safe for drinking. An

exception for limited claims may be allowed for units removing specific organisms to serve a definable environmental need, for example, removal of protozoan cysts.[27]

Clarification Techniques

Clarification refers to techniques that reduce the turbidity or cloudiness of water caused by natural organic and inorganic material. (Turbidity is measured in nephelometric turbidity units [NTU].) These techniques can markedly improve the appearance and taste of water. They may reduce the number of microorganisms, but not enough to ensure potable water; however, clarifying the water facilitates disinfection by filtration or chemical treatment. Cloudy water can rapidly clog filters designed to remove microorganisms. Moreover, cloudy water requires increased levels of chemical treatment, and the combined effects of the water contaminants plus chemical disinfectants results in unpleasant taste.

Adsorption

Granular activated carbon (GAC) is widely used in water treatment. When activated, charcoal's regular array of carbon bonds is disrupted, making it highly reactive for adsorbing dissolved chemicals.[28, 29] GAC is the best means to remove toxic organic and inorganic chemicals from water (including disinfection byproducts) and to improve odor and taste.[30, 31] Thus, it is widely used in municipal disinfection plants, in household under-sink devices, and in portable water filters. In field water treatment, GAC is best used after chemical disinfection to make water safer and more palatable by removing disinfection byproducts and pesticides, as well as many other organic chemicals and some heavy metals. It removes the taste of chemical disinfectants such as iodine and chlorine.

GAC does not kill microorganisms and is not designed for microbial removal; in fact, bacteria attach to charcoal, where they are resistant to chlorination because the chlorine is adsorbed by the GAC.[30, 31, 32]

Sedimentation

Sedimentation is the separation of suspended particles such as sand and silt that are large enough to settle rapidly by gravity. Most microorganisms, especially protozoan cysts, also settle eventually, but this takes much longer.[33] Simply allowing the water to sit undisturbed for about 1 h or until sediment has formed on the bottom of the container and then decanting or filtering the clear water from the top through a coffee filter or finely woven cloth will remove many larger particles from the water. A second method of disinfection must then be used to obtain potable water.

Coagulation–flocculation

Coagulation–flocculation (C-F) is a technique that has been in use since 2000 bc and remains a routine step in municipal water treatment.[34, 35] C-F can remove smaller suspended particles and chemical complexes too small to settle by gravity

(colloids). Coagulation is achieved with the addition of a chemical that causes particles to stick together by electrostatic and ionic forces. Flocculation is a physical process that promotes the formation of larger particles by gentle mixing. Alum (an aluminum salt), lime (alkaline chemicals principally containing calcium or magnesium with oxygen), or iron salts are commonly used coagulants. Alum is nontoxic and used in the food industry for pickling. It is readily available in most chemical supply stores and some grocery stores. C-F removes 60 to 98% of microorganisms, heavy metals, and some chemicals and minerals.[36], [37] The tendency of microorganisms to clump with small particles or clump together to form larger aggregates enhances their removal by C-F. C-F also has the benefit of reducing the amount of chemical disinfectant needed because turbidity increases demand for disinfectants such as hypochlorite.[37], [38], [39]

The amount of alum added in the field, approximately 1 large pinch (1 mL or 1/8 tsp) per 4 L (approximately 1 gal) of water, need not be precise. Stir or shake briskly for 1 min to mix, and then agitate gently and frequently for at least 5 min to assist flocculation. If the water is still cloudy, add more flocculent and repeat mixing. After at least 30 min for settling, pour the water through a fine-woven cloth or paper filter. Although most microorganisms are removed with the floc, a final process of microbiologic filtration or chemical disinfection (below) should be completed to ensure disinfection. Several products combine C-F with halogen disinfection, which allows a single-step process.[40], [41], [42], [43]

Improvisational techniques for clarification

Many inorganic and organic compounds can be used as a coagulant, including lime (calcium oxide) or potash (from wood ash).[44] In an emergency, bleaching powder, baking powder, or even the fine white ash from a campfire can be used.[45] Other C-F agents used traditionally by native peoples include seed extracts from the nirmali plant in southern India, moringa plants in Sudan, crushed almonds, dried and crushed beans, and rauwaq (a form of bentonite clay).[46]

Adsorbents such as charcoal, clay, and other types of organic matter have been used for water treatment since biblical times.[32] These substances are used as the filter media and also can act as coagulants.[47] Clays can decrease turbidity and microbes in water by about 90 to 95%, but adsorption is not the main action of ceramic or clay filters.

Assessment of supporting evidence:

- Clarification reduces cloudiness, particulate matter, and waterborne microorganisms; improves the taste and esthetics of water; and improves the effectiveness of chemical disinfectants, filtration, and ultraviolet disinfection. However, it does not reliably disinfect if used alone. **Evidence grade: 1A**
- GAC is highly effective at removing taste and odor compounds but is not adequate for microbial removal. **Evidence grade: 1A**
-

Sedimentation is effective for removing large particles such as sand and dirt but will not remove suspended or dissolved substances (see C-F). **Evidence grade: 2B**

- C-F removes most microorganisms, but it does not reliably disinfect if used alone. **Evidence grade: 1A**
- Traditional or improvisational C-F techniques (other than alum or those used in municipal disinfection plants) have empiric evidence but do not have robust scientific evidence or practical use guidance and should be used with caution to protect the health of consumers. **Evidence grade: 2C**

Disinfection Methods

HEAT

Heat is the oldest and most reliable means of water disinfection. Heat inactivation of microorganisms is a function of time and temperature (exponential function of first-order kinetics). Thus, the thermal death point is reached in a shorter time at higher temperatures, whereas lower temperatures are effective if applied for a longer time. Pasteurization uses this principle to kill food pathogens and spoiling organisms at temperatures well below boiling, generally between 60°C (140°F) and 70°C (158°F). Flash pasteurization occurs within 30 s at 70 to 72°C (158 to 162°F).[48, 49]

All common enteric pathogens are readily inactivated by heat at pasteurization temperatures, although microorganisms vary in heat sensitivity, with protozoan cysts being the most sensitive to heat, bacteria intermediate, and viruses less sensitive (Table 1[50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62]).[50, 51] Only bacterial spores are more resistant, but they are not generally enteric pathogens.[52]

Table 1. Heat inactivation of microorganisms

Organism	*Lethal temperature/Time*	*Reference*
Protozoan cysts, including *Giardia, Entamoeba histolytica*	50°C (122°F) for 10 min 55°C (131°F) for 5 min 100°C (212°F) immediately	53, 54, 55
Cryptosporidium oocysts	55°C (131°F) warmed over 20 min 64°C (148°F) within 2 min	50, 56
Parasitic eggs, larvae, and cercariae	50°C–55°C (122–131°F)	57
Common bacterial enteric pathogens (*E coli*, *Salmonella*, *Campylobacter*, *Shigella*)	55°C (131°F) for 30 min or 65°C (149°F) for less than 1 min (standard pasteurization temperatures)	48, 51
Viruses	56°C–60°C (133–140°F) in less than 20–40 min	52, 58, 59
Hepatitis A virus	98°C (208°F) for 1 min 75°C (167°F) for less than 0.5 min 85°C (185°F) for 1 min or less (in various food products)	60, 61, 62

As enteric pathogens are killed within seconds by boiling water rapidly at temperatures > 60°C (140°F), the traditional advice to boil water for 10 min to ensure potable water is excessive. The time required to heat water from 55°C (131°F) to a boil works toward disinfection; therefore, any water brought to a rapid boil should be adequately disinfected.[63] Boiling for 1 min is recommended by the US CDC to account for user variability in identifying boiling points and adds a margin of safety. The boiling point decreases with increasing altitude, but this is not significant compared with the time required for thermal death at these temperatures (Table 2).

Table 2. Boiling temperatures at various altitudes

Altitude (ft)	*Altitude (m)*	*Boiling point*
5000	1524	95°C (203°F)
10,000	3048	90°C (194°F)
14,000	4267	86°C (187°F)
19,000	5791	81°C (178°F)

Improvisational techniques

In wilderness or travel environments, the main limitation for using heat is availability of fuel. Although attaining boiling temperature is not necessary to kill microorganisms, boiling is the only easily recognizable endpoint without use of a thermometer. Based on microbiologic testing, hot tap water has been proposed as a means of heat disinfection.[64, 65]

Most water from hot water taps measured in countries outside the United States measured 55 to 60°C (131 to 140°F).[51] As a rule of thumb, water too hot to touch fell within the pasteurization range, but tolerance to touch is too variable to be reliable.[66]

If no reliable method of water treatment is available, tap water that has been kept hot in a tank for at least 30 min and is too hot to keep a finger immersed for 5 s (estimated 55 to 65°C; 131 to 149°F) is a reasonable alternative. However, this improvisational measure is less useful for hotels that use on-demand water heaters without a hot water tank. Travelers with access to electricity can boil water with either a small electric heating coil or a lightweight electric beverage warmer brought from home. In austere and desperate situations with hot, sunny climate, pasteurization temperature can be achieved with a solar oven or simple reflectors[67, 68] (see the Solar UV Disinfection [UV–SODIS] section).

Assessment of supporting evidence:

- Bringing water to boil (100°C/212°F) will kill pathogenic microorganisms. **Evidence grade: 1A**
- Bringing water at 5000 m (16,000 ft) elevation to boil (83°C/181°F) will kill pathogenic organisms. **Evidence grade: 1B**
- Tap water that has been tanked for 30 min or longer and is too hot to touch (60°C) has a significantly reduced number of pathogenic microorganisms, but this cannot be relied on as the sole means of disinfection. Such water may contain increased amounts of lead or other chemicals from the water heater and piping. **Evidence grade: 2B**
- Pasteurization temperatures can be achieved with a solar oven. **Evidence grade: 2B**

ULTRAVIOLET LIGHT

Ultraviolet (UV) radiation and UV lamp disinfection systems are widely used to disinfect drinking water at the community and household levels. At sufficient doses, all waterborne enteric pathogens are inactivated by UV radiation (UVR). UVC light in the range of 200 to 280 nm is the most effective. The germicidal effect of UV light is the result of action on the nucleic acids of microorganisms and depends on light intensity and exposure time. In sufficient doses of energy, all waterborne enteric pathogens are inactivated by UVR.[69] The UV waves must strike the organism, so the water must be free of particles that could act as a shield.[70] The UV waves do not alter the water, but they also do not provide any residual disinfecting power.[71] Bacteria and protozoan parasites generally require lower doses than do enteric viruses and bacterial spores. However, all viruses, including hepatitis A and norovirus, are susceptible, with relatively minor differences, and follow similar kinetics. The vegetative cells of bacteria are

significantly more susceptible to UVR than are bacterial spores or viruses. *Giardia* and *Cryptosporidium* are susceptible to practical doses of UVR and may be more sensitive because of their relatively large size.[72], [73], [74] Both large high-volume units and portable, lightweight battery-operated units are available for disinfection of small quantities of water.

Improvisational technique: UV-SODIS

UV irradiation by sunlight can substantially improve the microbiologic quality of water and reduce diarrheal illness in developing countries.[75], [76], [77], [78], [79], [80], [81], [82], [83], [84], [85] The optimal procedure for the SODIS technique is to use transparent bottles (eg, clear plastic beverage bottles), preferably lying on a dark surface and exposed to sunlight for a minimum of 4 h with intermittent agitation.[86] UV and thermal inactivation are strongly synergistic for the solar disinfection of drinking water.[67], [87], [88]

Assessment of supporting evidence:

- UV light is an effective means of water disinfection. **Evidence grade: 1A**
- Full sunlight exposure of clear water in a clear plastic bottle for at least 4 h significantly reduces and possibly eliminates microorganism contamination (**Evidence grade: 1B**); however, studies evaluating this technique for reduction of childhood diarrhea show mixed results. **Evidence grade: 2B**

FILTRATION

Filters are appealing because of their simplicity and suitability for commercial production. Portable water treatment products are the third highest intended purchase of outdoor equipment, after backpacks and tents.[89] Filtration is a standard step in municipal water treatment and widely used in the food and beverage industry and in many other industrial processes. Many different types of media, from sand to vegetable products to fabric have been used for water filtration throughout history in various parts of the world.[90] Filters have the advantages of being simple and requiring no holding time. They do not add any unpleasant taste and may improve taste and appearance of water. All filters eventually clog from suspended particulate matter (present even in clear streams), requiring cleaning or replacement of the filter. As a filter clogs, it requires increasing pressure to drive the water through it, which can force microorganisms through the filter or damage the filter. A crack or eroded channel in a filter will allow passage of unfiltered water. Bacteria can grow on filter media and potentially result in some bacteria in filtered water, but pathogenic bacteria and illness have not been demonstrated.[91] Silver is often incorporated into the filter media to prevent this growth, but it is not totally effective.

The primary determinant of a microorganism's susceptibility to filtration is its size (Table 3; Figure 1). Portable filters for water treatment can be divided into microfilters with pore sizes down to 0.1 µm, ultrafilters that can remove particles as small as 0.01 µm, nanofilters with pore sizes as small as 0.001 µm or less, and

reverse osmosis filters with pore sizes of 0.0001 µm or less.[69] All filters require pressure to drive the water through the filter element. The smaller the pore size, the more pressure required. Waterborne pathogens often adhere to larger particles or clump together, making them easier to remove by physical processes. Therefore, observed reductions are often greater than expected based on their individual sizes.

Table 3. Microorganism susceptibility to filtration

Organism	***Approximate size (µm)***	***Recommended filter rating (µm)***
Viruses[a]	0.03	Ultrafilter, nanofilter, reverse osmosis
Escherichia coli	0.5 by 3–8	0.2–0.4 (microfilter)
Campylobacter	0.2–0.4 by 1.5–3.5	
V cholerae	0.5 by 1.5–3.0	
Cryptosporidium oocyst	2–6	1 (microfilter)
Giardia cyst	6–10 by 8–15	3–5 (microfilter)
Entamoeba histolytica cyst	5–30 (average 10)	
Nematode eggs	30–40 by 50–80	20 (microfilter)
Schistosome cercariae	50 by 100	Coffee filter or fine cloth, or double thickness closely woven cloth
Dracunculus larvae	20 by 500	

a

Microfilters (includes most filters with pore size of 0.1–0.2 µm) can filter bacteria and protozoan cysts, but are not effective for virus removal unless designed to rely on electrostatic trapping of viruses. Hollow fiber filters with 0.02 µm pores and reverse osmosis filters are capable of filtering viruses.

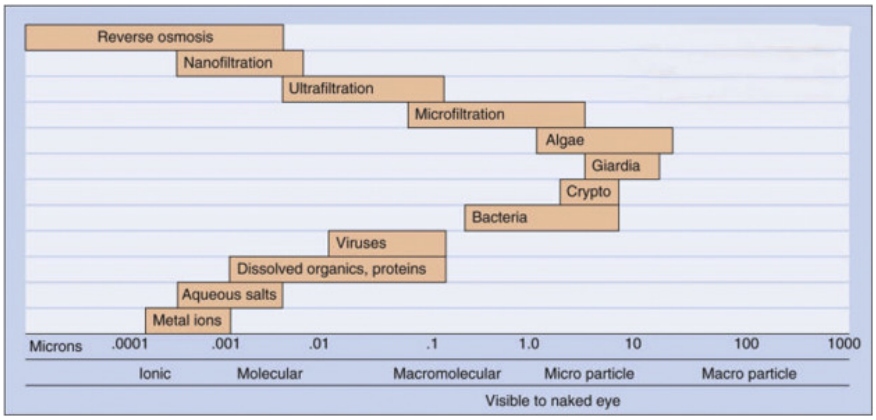

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Figure 1. **Levels of filtration and susceptibility of common microbial pathogens and other contaminants.** Adapted from Backer H. Water disinfection for international travelers. In: Keystone JS, Kozarsky PE, Connor BA, eds. *Travel Medicine*. 4th ed. Philadelphia, PA: Elsevier; 2019:31–41. Copyright 2019, reprinted with permission from Elsevier.

Most portable filters are microfilters that can readily remove protozoan cysts and bacteria but may not remove all viruses, which are much smaller than the pore size of most field filters.[92, 93] Viruses often clump together and to other larger particles or organisms, resulting in an aggregate large enough to be trapped by the filter; in addition, electrochemical attraction may cause viruses to adhere to the filter surface.[47, 94, 95] Through these mechanisms, mechanical filters using ceramic elements with a pore size of 0.2 µm can reduce viral loads by 2 to 3 logs (99–99.9%), but they are not adequate for complete removal of viruses.[96] Ultrafiltration membranes are required for complete microbial removal, including viruses; they can also remove colloids and some dissolved solids.[97]

Recently, hollow-fiber technology has been adapted for field use; this technology uses bundles of tube fibers whose pore size can be engineered to achieve ultrafiltration with viral removal.[98] The large surface area allows these hollow-fiber filters to have relatively high flow rates at low pressure. Small group and individual gravity or hand pump filters are available through several vendors.

Some filters on the market combine the porous filter material with other substances to help the disinfection process. This may include activated charcoal, iodine, silver, and other substances. Iodine molecules can be bound in a resin engineered into field products, but the effectiveness of the resin is highly dependent on the product design and function. Most companies have abandoned iodine resin–containing portable hand-pump filters due to excess iodine or viral breakthrough in the effluent. Only one drink-through bottle remains on the US market, but other products may still be available outside the United States. (GAC was discussed earlier, and silver is addressed later.)

Several factors influence the decision of which filter to buy: 1) flow volume sufficient for the number of persons relying on the filter; 2) whether the filter functional claims matches the microbiologic demands that will be put on the filter; 3) the preferred means of operation (eg, hand pump or gravity); and 4) cost.

Improvisational filtration techniques

Filtration using simple, available products, such as rice hull ash filters, crushed charcoal, sponges, and various fabrics and paper, have all been used in developing countries and in emergency situations. Typically, bacteria and viruses can be reduced by as much as 50 to 85% and larger parasites by 99%, depending on the media. The effectiveness for decreasing turbidity may be used as an indicator that a filter material will reduce microbiologic contamination.[38, 99, 100]

Ceramic filters are a common component in portable water pump filters, but they are also a cost-effective means of household disinfection in developing countries. Ceramic clay is widely available and very inexpensive to locally manufacture in the shape of a sink or flower pot that is set into a larger container that collects the filtered water.[101, 102, 103, 104, 105, 106, 107]

Biosand filters use a technology that has been used over centuries and is still used widely in municipal plants and at the household and community level.[108, 109, 110, 111] Sand filters can be highly effective at removing turbidity (in 1 study, from 6.2 NTU to 0.9 NTU) and improving microbiologic quality (99% efficacy), depending on their design and operation.[112, 113] Sand filters are constructed by forming layers of aggregate increasing in size from the top to the bottom. The top layer is very fine sand and the bottom layer consists of large gravel. The container needs an exit port on the bottom. The top layer forms a biolayer that is important for the function of the filter. The optimum depth of a community or household sand filter is 2 m, with diameter determined by the volume of water needed. An emergency sand filter can be made in a 20 L (5.3 gal) bucket, composed of a 10 cm (3.9 in) layer of gravel beneath a 23 cm (9.1 in) layer of sand; a layer of cotton cloth, sandwiched between 2 layers of wire mesh, separates the sand and gravel layers.[38] A sand filter also can be improvised with stacked buckets of successive filter layers with holes in the bottom to allow water passage. Many websites provide design and assembly instructions, but there are no data for comparative function.

Assessments of supporting evidence:

- Filtration is effective as a primary or adjunctive means of water treatment. **Evidence grade: 1A**
- Standard commercially available microfilters with a pore size of 0.2 microns are effective in removing protozoa and bacteria. **Evidence grade: 1A**
- Ultrafiltration with pore size of less than 0.01 is needed to completely remove pathogenic viruses. **Evidence grade: 1A**
- Filters may clog, so users should know how to clean them or consider carrying a backup method of disinfection. **Evidence grade: 1C**
-

Biosand filters are a reasonable improvised technique for filtration. **Evidence grade: 1B**

CHEMICAL DISINFECTION: HALOGENS (IODINE AND CHLORINE)

Worldwide, disinfection with chemicals, chiefly chlorine, is the most commonly used method for improving and maintaining the microbiologic quality of drinking water and can be used by individuals and groups in the field.[114] The germicidal activity of chlorine and other halogens is well established and results from oxidation of essential cellular structures and enzymes.[115, 116] Disinfection effectiveness is determined by characteristics of the microorganism, the disinfectant, contact time, and environmental factors. Both chlorine and iodine are widely available worldwide in multiple formulations. The most commonly available form of chlorine is hypochlorite (household bleach [5 to 8%] or concentrated swimming pool granules or tablets [70%]).

Both chlorine and iodine have been used for water disinfection for more than a century. Hypochlorite, the major chlorine disinfectant, is currently the preferred means of municipal water disinfection worldwide. Both calcium hypochlorite ($Ca[OCl]_2$) and sodium hypochlorite (NaOCl) readily dissociate in water to form hypochlorite, the active disinfectant.

Iodine is also effective in low concentrations for killing bacteria, viruses, and some protozoan cysts; in higher concentrations, it is effective against fungi and even bacterial spores. However, it is a poor algaecide. Elemental iodine (I_2) and hypoiodous acid (HOI) are the major germicides in an aqueous solution. Iodine is the only halogen that is a solid at room temperature.

Given adequate concentrations and contact times, both iodine and chlorine are effective disinfectants with similar biocidal activity under most conditions.[117] Taste preference is individual. Of the halogens, iodine reacts least readily with organic compounds and is less affected by pH, indicating that low iodine residuals should be more stable and persistent than corresponding concentrations of chlorine. Despite these advantages, because of its physiologic activity, WHO recommends iodine only for short-term emergency use.

Chlorine is still advocated by the WHO and the CDC as a mainstay of large-scale community, individual household, and emergency use.[118, 119] There are extensive data on effectiveness of hypochlorite in remote settings.[69, 120, 121, 122] The CDC/WHO safe water system for household disinfection in developing countries provides a dosage of 1.875 or 3.75 $mg \cdot L^{-1}$ of sodium hypochlorite with a contact time of 30 min, which is sufficient to inactivate most bacteria, viruses, and some protozoa that cause waterborne diseases.[123] Another advantage of hypochlorite is the ease of adjusting the dose for large volumes of water.[45, 99]

Vegetative bacteria (nonspore forming) are very sensitive to halogens.[116, 124] Viruses, including hepatitis A, have intermediate sensitivity, requiring higher concentrations or longer contact times.[125, 126, 127, 128, 129, 130] Protozoan cysts are more resistant than enteric bacteria and enteric viruses but some cysts (eg, *Giardia*) can be inactivated by field doses of halogens.[131, 132, 133, 134,

135 *Cryptosporidium* oocysts, however, are much more resistant to halogens, and inactivation is not practical with common doses of iodine and chlorine used in field water disinfection.136, 137 Little is known about *Cyclospora*, but it is assumed to be similar to *Cryptosporidium*. Certain parasitic eggs, such as those of *Ascaris*, are also resistant, but these are not commonly spread by water. (All of these resistant cysts and eggs are susceptible to heat or filtration.) Bacterial spores, such as *Bacillus anthracis*, are relatively resistant to halogens. With chlorine, however, spores are not much more resistant than are *Giardia* cysts; furthermore, they do not normally cause waterborne enteric disease. Relative susceptibility between organisms is similar for iodine and chlorine (Table 4).

Table 4. Disinfection data for chlorine and iodine to achieve 99.9% kill or inactivation[a] of select microorganisms

Organism	*Concentration ($mg \cdot L^{-1}$)*	*Time (min)*	*pH*	*Temp*	*Disi co (
Chlorine					
Escherichia coli	0.1	0.16	6.0	5°C (41°F)	0.01
Campylobacter	0.3	0.5	6.0–8.0	25°C (77°F)	0.15
20 enteric virus	0.5	60	7.8	2°C (36°F)	30
6 enteric viruses	0.5	4.5	6.0–8.0	5°C (41°F)	2.5
Norovirus	1 5	10 20 sec	6.0	5°C	10 1.66
Hepatitis A virus	0.5	1	6.0	25°C (77°F)	0.5
Amebic cysts	3.5	10		25°C (77°F)	35
Giardia cysts	2.5	60	6.0–8.0	5°C (41°F)	150
Giardia lamblia cysts	0.85	90	8.0	2–3°C (36–37°F)	77
Giardia muris cysts	3.05	50	7.0	5°C (41°F)	153
Cryptosporidium (2 strains)	20 20	755 501	7.5 7.5	23°C 23°C	15,3 10,4
Iodine					
Escherichia coli	1.3	1	6.0–7.0	2–5°C (36–41°F)	1.3
Hepatitis A‡	8	.4	7.0	25°C	3
Coxsackie virus	0.5	30	7.0	5°C (41°F)	15
Amebic cysts	3.5	10		25°C (77°F)	35
Giardia cysts	4	15	5.0	30°C (86°F)	60[b]
Giardia cysts	4	45	5.0	15°C (59°F)	170[b]
Giardia cysts	4	120	5.0	5°C (41°F)	480[b]

a

99.9% is for comparison of disinfection potency and microorganism susceptibility. The standard for potable water is 99.99% kill for viruses and 99.999% for bacteria. This would be achieved in each example with a higher concentration of disinfectant or a longer contact time.

b

100% kill; viability tested only at 15, 30, 45, 60, and 120 min.

Understanding factors that influence the disinfection reaction allows flexibility with greater reassurance. The primary factors of the first-order chemical disinfection reaction are concentration and contact time.[133] To achieve microbial inactivation in aqueous solution with a chemical agent, a residual concentration must be present for a specified contact time. Lower concentrations can be used with longer contact times. In field disinfection, this can be used to minimize halogen dose and improve taste or, conversely, to minimize the required contact time.

Cold water slows chemical reactions; the reaction rate can be adjusted by longer contact times or higher concentration of disinfectant chemical. Another important factor in chemical disinfection is the presence of organic and inorganic contaminants, mainly nitrogen compounds from decomposition of organisms and their wastes, fecal matter, and urea. These contaminants react, especially with chlorine, to form compounds with little or no disinfecting ability, effectively decreasing the concentration of available halogen.[26, 115] Halogen demand is the amount of halogen reacting with impurities. Residual concentration is the amount of active disinfectant remaining after demand of the water is met. Halogen demand is associated with turbidity (cloudiness).[39] Typical recommendations for field treatment double the amount of chlorine or iodine in cloudy water; however, it is preferable to use clarification techniques prior to chemical disinfection in cloudy water to improve efficacy and taste.[144, 145]

Because of the difficulty of estimating halogen demand, it is prudent to use 3 to 4 $mg \cdot L^{-1}$ as a target halogen concentration range for clear surface water. Lower concentrations (eg, 2 $mg \cdot L^{-1}$) can be used for back-up treatment of questionable tap water or high-quality well water (Table 5, Table 6).

Table 5. Halogen disinfection products and recommended doses

	Add to 1 L or qt of water	
Iodination techniques[a]	***Amount to achieve 4 mg·L^{-1}***	***Amount to achieve 8 mg·L^{-1}***
Iodine tabs[b]	0.5 tab (or 1 tab in 2 L)	1 tab
Tetraglycine hydroperiodide		
Emergency drinking water germicidal tablet		
Potable aqua		
Globaline		
2% iodine solution (tincture)	0.2 mL	0.4 mL
	5 drops[c]	10 drops
10% povidone-iodine solution[d]	0.35 mL	0.70 mL
	8 drops	16 drops
Saturated solution: iodine crystals in water[e]	13 mL	26 mL
Chlorination techniques[f]	Amount to achieve 2 mg· L^{-1}	Amount to achieve 5 mg· L^{-1}
Sodium hypochlorite (household bleach 5%)	1 drop	0.1 mL 2 drops
Sodium hypochlorite (household bleach 8.25%)	1 drop (in 2 L)	1 drop
1% bleach (CDC-WHO Safe Water System)[g]	4–5 drops	8–10 drops
Calcium hypochlorite[h] (Redi Chlor [0.1-g tab])	Cannot use in small quantities for low concentrations	0.25 tab
Sodium dichloroisocyanurate (NaDCC)[i] (Aquatab, Kintab)	0.25 tab of 8.5 mg NaDCC (may be impractical)	0.5 tab (8.5 mg NaDCC)
Chlorine plus flocculating agent (C-F)	Not practical for small volumes	0.5 tablet per gal yields 5 mg· L^{-1}

a

World Health Organization recommends only for short-term emergency use.

b

Iodine tablets were developed by the military with the criteria that they will disinfect water, including for *Giardia*, with a short contact (holding) time of 10 min because troops in the field may not wait longer. This high concentration is not necessary for field disinfection of clear water; it is preferable to target 4

mg·L^{-1} and wait longer. Additionally, the recommendation to use 8 mg·L^{-1} for cloudy water will result in poor taste, so it is recommended to clarify the water first.

c

Measure of a drop varies from 16–24 gtt·mL^{-1}, standard 20 gtt·mL^{-1} is used here.

d

Povidone-iodine solutions release free iodine in levels adequate for disinfection, but scant data are available (see text).

e

A small amount of elemental iodine goes into solution (no significant iodide is present); the saturated solution is used to disinfect drinking water. Water can be added to the crystals hundreds of times before they are completely dissolved.

f

Can easily be adapted to large or small quantities of water. Simple field test kits or swimming pool test kits with color strips are widely available to ensure adequate residual chlorine. In usual situations, EPA recommends a target residual of 4 mg·L^{-1}. For household use, the CDC recommends < 2 mg·L^{-1}. Many of the recommended emergency doses exceed this threshold.[97] For treatment of large volumes, see formula to calculate in Lantagne (2008).[20]

g

Safe water system for long-term routine household point-of-use water disinfection recommends a hypochlorite dose of about 2 mg·L^{-1} in clear water and 4 mg·L^{-1} in slightly turbid water. This results in a low yet effective target residual concentration but requires testing in a particular water source to ensure sufficient residual.

h

Stable, concentrated (70%), dry source of hypochlorite that is used for chlorination of swimming pools. Multiple products available in various size tablets or granular form. Best formulation for large quantities of water.

i

Available in different strengths to treat different volumes of water. Check packaging to determine proper dose.

Table 6. Recommendations for contact time using halogen disinfection in the field

Concentration of halogen	***Contact time (min) at various water temperatures***		
	5°C (41°F)	***15°C (59°F)***	***30°C (86°F)***
2 ppm	240	180	60
4 ppm	180	60	45
8 ppm	60	30	15

Concentration and contact time are based on the most resistant organism, which is the *Giardia* cyst. These are well beyond the time needed to kill bacteria and viruses. These contact times have been extended from the usual recommendations in cold water to account for the extended inactivation time required in very cold water and for the uncertainty of residual concentration.

Halogen toxicity

Chlorine has no known toxicity at the concentrations used for water disinfection. Sodium hypochlorite is not carcinogenic; however, reactions of chlorine with certain organic contaminants yield chlorinated hydrocarbons, chloroform, and other trihalomethanes, which are considered to have carcinogenic potential in animal models. Nevertheless, the risk of severe illness or even death from infectious diseases if disinfection is not used far exceeds any risk from byproducts of chlorine disinfection.[146]

Despite several advantages over chlorine disinfection, iodine has not gained general acceptance because of concern for its physiologic activity. Some older data indicate that iodination of water with a low residual concentration of ≤1 to 2 $mg \cdot L^{-1}$ appears safe, even for long periods of time, in people with normal thyroid function.[147, 148] This is not the current recommendation of major agencies. Recently, the European Union stopped the sale of iodine products used for water disinfection. The WHO did not set a guideline value for iodine in drinking water because of a paucity of data and because it is not recommended for long-term disinfection. If the typical wilderness or international traveler disinfected 3 L of water a day using 2 to 4 $mg \cdot L^{-1}$ of iodine, the ingested amount of iodine would be 6 to 12 $mg \cdot d^{-1}$, well above US Institute of Medicine recommended dietary allowance levels. Levels produced by the recommended doses of iodine tablets are even higher (16 to 32 $mg \cdot d^{-1}$). Therefore, the use of iodine for water disinfection should be limited to short periods of ≤ 1 mo. Individuals planning to use iodine for prolonged periods should have their thyroid examined and thyroid function tests done to ensure they are initially euthyroid. Certain groups should not use iodine for water treatment: pregnant women (because of concerns of neonatal goiter); those with known hypersensitivity to iodine; persons with a history of thyroid disease, even if controlled on medication; persons with a strong family history of thyroid disease (thyroiditis); and persons from countries with chronic iodine deficiency.[149]

Improving halogen taste

Objectionable taste and smell limit the acceptance of halogens, but taste can be improved by several means. One method is to use the minimum necessary dose with a longer contact time, as in the CDC safe water system. Another method is to use higher doses and remove the taste through chemical reduction of chlorine to chloride and iodine to iodide; these have no color or taste. The best and most readily available agent is ascorbic acid (vitamin C), available in crystalline or powder form. A small pinch in a liter, mixed after the required contact time, will usually suffice. Ascorbic acid is a common ingredient of flavored drink mixes, accounting for their effectiveness in removing the taste of halogens. GAC (see

above) adsorbs organic and inorganic chemicals, including iodine and chlorine byproducts, thereby improving odor and taste—the reason for its common inclusion in field filters.

Improvisational techniques

There is no comparable substitute for proven chemical disinfectants, but there are many common substances that contain halogens. Household bleach is available in most parts of the world. The active disinfectant is sodium hypochlorite. Products for disinfection of swimming pools and spas generally contain calcium hypochlorite that provides much higher concentrations than bleach. Hypochlorite is readily released from different products formulated in liquid, powder, granules, and tablets. Iodine is also available in liquid or tablets; a common household source is tincture of iodine or similar topical disinfectants with an iodine concentration of 2 to 8%. These products also contain iodide, which has no disinfecting power but does contribute to iodine toxicity. Colorless iodine solution contains only iodide and should not be used. Povidone-iodine, a topical disinfectant commonly used in medical settings, contains active iodine bound to a neutral polymer of high molecular weight that gives the iodine greater solubility and stability. In dilute aqueous solution, povidone-iodine provides a sustained-release reservoir, releasing free iodine in a concentration of 2 to 10 $mg \cdot L^{-1}$.[150]

MIXED SPECIES DISINFECTANT (ELECTROLYSIS)

Passing a current through a simple brine salt solution generates free available chlorine and other mixed species disinfectants that have been shown to be effective against bacteria, viruses, *Cryptosporidium*, and bacterial spores.[151, 152] The process is well described and can be used on both large and small scales. The main disinfectant effect is probably attributable to a combination of chlorine dioxide, ozone, superoxides, and hypochlorous acid, giving the resulting solution greater disinfectant ability than a simple solution of sodium hypochlorite. Small units are now available commercially that use salt, water, and a 12-volt direct current (automobile) battery to create 60 mL of a 0.75% chlorine solution over a 5-min operation cycle that will treat up to 200 L of water.

Other common substances, including hydrogen peroxide and citrus juice that have some disinfectant activity, are discussed later.

Assessments of supporting evidence:

- Halogens chlorine and iodine are an effective means of disinfecting water of bacteria, viruses, and *Giardia* in the field or household when using appropriate contact time and halogen concentration. **Evidence grade: 1A**
- Usual field concentrations of iodine and chlorine are not effective for other protozoa including *Cryptosporidium* and *Cyclospora*. **Evidence grade: 2A**
-

Extended use of iodine should be weighed against risks of iodine toxicity. **Evidence grade: 1B**

- Simple techniques for improving taste of halogenated water are available for field use. **Evidence grade: 1B**
- Mixed species electrolytic disinfection techniques are effective for water disinfection of microbes that are susceptible to halogens. **Evidence grade: 1B**

MISCELLANEOUS DISINFECTANTS

Chlorine dioxide

Chlorine dioxide (ClO_2), a potent biocide, has been used for many years to disinfect municipal water and in numerous other large-scale applications. Until recently, the benefits of chlorine dioxide have been limited to large-scale applications because standard formulations must be made on-site and are associated with a risk for producing volatile gas. Newer methods enable cost-effective and portable ClO_2 generation and distribution for use in an ever-widening array of small-scale applications. ClO_2-production tablets contain 6.4% sodium chlorite as the active ingredient. After a tablet is added to water, a series of complex chemical reactions occurs, generating chlorine dioxide. Some of the intermediary chemical compounds may also have antimicrobial activity.

ClO_2 has no taste or odor in water. It is capable of inactivating most waterborne pathogens, including *Cryptosporidium parvum* oocysts.[153], [154], [155] It is at least as effective a bactericide as chlorine and far superior for virus and parasite inactivation. Several commercial point-of-use applications use ClO_2 in liquid or tablet form, but relatively few data are available on product testing these products.[137] A major disadvantage for field use of tablets is the long reaction or contact time required, with upward of 2 to 4 h needed to achieve dependable disinfection. ClO_2 does not produce a lasting residual, and water undergoing chlorine dioxide disinfection must be protected from sunlight.

Assessment of supporting evidence:

- Chlorine dioxide is a widely used and potent water disinfectant, including efficacy against the protozoan parasites *Cryptosporidium*. **Evidence grade: 1A**
- Individual use products have limited data demonstrating effective concentration and contact time. **Evidence grade: 2B**

Silver

Silver ion has bactericidal effects in low doses and some attractive features, including absence of color, taste, and odor. Scant data for disinfection of viruses and protozoan cysts indicate limited effect, even at high doses. Moreover, the

concentrations are strongly affected by adsorption onto the surface of any container. Silver is physiologically active but not likely to cause a problem in concentrations found in drinking water. The EPA has not approved silver for primary water disinfection in the United States, but silver is approved as a water preservative to prevent bacterial growth in previously treated and stored water. In Europe, silver tablets are sold for field water disinfection. One rational combination product combines silver with hypochlorite for both disinfection and preservation. There is some promise in steady release products and incorporation into nanoparticles.[156]

Assessment of supporting evidence:

- Use of silver in wilderness settings should be limited to water preservation and not as a primary disinfectant. **Evidence grade: 1B**

Hydrogen peroxide

Hydrogen peroxide is a strong oxidizing agent that is widely used as a preservative in food, as a sterilant for medical and food equipment, and in many other applications. Although hydrogen peroxide can sterilize water, it is not widely used as a field water disinfectant, perhaps because high concentrations known to be effective are very caustic, and there is a lack of data for protozoal cysts and quantitative data for dilute solutions. It can be used to remove the taste of hypochlorite and in combination with other processes.[157]

Assessment of supporting evidence:

- Hydrogen peroxide in typical concentration of 3% cannot be used as a primary drinking water disinfectant, and effective concentrations are not practical for field use. **Evidence grade: 1B**

Citrus and potassium permanganate

Both citrus juice and potassium permanganate have some demonstrated antibacterial effects in an aqueous solution.[158] However, data are few and not available for effect on cysts. In municipal water disinfection, potassium permanganate is used primarily for reducing contaminants to improve taste and odor.[159] Either substance could be used in an emergency to reduce bacterial and viral contamination or as an adjunct in combination with another technique, but they cannot be recommended as a primary means of water disinfection.

Assessment of supporting evidence:

- Citrus juice and potassium permanganate have limited applications for drinking water disinfection. **Evidence grade: 1C**

Nanoparticles: solar photocatalytic disinfection

Several nanomaterials have been shown to have strong antimicrobial properties and are being evaluated for use in water disinfection and purification.[160, 161] The metals are of particular interest for water disinfection applications because they can be activated by UV light to produce potent oxidizers that are excellent disinfectants for microorganisms and can break down complex organic contaminants and even most heavy metals into nontoxic forms. Titanium dioxide (TiO_2) is the most effective photocatalytic substance identified to date. Recent work demonstrated inactivation of *Cryptosporidium* by titanium dioxide.[161, 162] These methods are widely used in industry, but few products have incorporated the technology into individual or small group point of use products.[163, 164]

Assessment of supporting evidence:

- New technology using nanoparticles and photocatalytic disinfection is highly promising for translation into point-of-use water disinfection. **Evidence grade: 2A**

PREFERRED TECHNIQUE

The optimal water treatment technique for an individual or group will depend on the number of persons to be served, space and weight accommodations, quality of source water, personal taste preferences, and fuel availability. Because halogens are not effective for killing *Cryptosporidium* at drinking water concentrations and common microfilters are not reliable for virus removal, optimal protection for all situations may require a 2-step process of 1) filtration or C-F, followed by 2) halogenation. Heat (boiling) is effective as a 1-step process in all situations but will not improve the esthetics of the water. Table 7 summarizes effects of major water disinfection methods on categories of microorganisms. Persons living or working in communities where sanitation and water treatment are lacking are at higher risk than the average international traveler. Sobsey et al reviewed data for point-of-use methods for household disinfection in developing countries[165] (Table 8).

Table 7. Summary of field water disinfection techniques

	Bacteria	***Viruses***	**Giardia/*Ameba***	**Cryptospo**
Heat	+	+	+	+
Filtration	+	+/–[a]	+	+
Halogens	+	+	+	–
Chlorine dioxide and photocatalytic	+	+	+	+

DNA, data not available.

a

Most filters make no claims for viruses. Ultrafiltration with hollow fiber technology and reverse osmosis is effective.

b

Eggs are not very susceptible to halogens but have very low risk of waterborne transmission. No data available for photocatalytic disinfection.

Table 8. Efficacy and effectiveness of point-of-use technologies for developing world households

Treatment process	*Pathogen*	*Optimal log reduction*[a]	*Expected log reduction*[b]	
Ceramic filters	Bacteria	6	2	
	Viruses	4	0.5	
	Protozoa	6	4	
Free chlorine	Bacteria	6	3	
	Viruses	6	3	
	Protozoa	5	3	
Coagulation/Chlorination	Bacteria	9	7	
	Viruses	6	2–4.5	
	Protozoa	5	3	
Biosand filtration	Bacteria	3	1	
	Viruses	3	0.5	
	Protozoa	4	2	
SODIS	Bacteria	5.5	3	
	Viruses	4	2	
	Protozoa	3	1	

SODIS, solar disinfection.

Data from multiple studies, analyzed and summarized by Sobsey et al (2008).[165]

Data also from references47, 166, 167, 168 and Table 7.8 in WHO (2011).[26]

a

Skilled operators using optimal conditions and practices (efficacy); log reduction: pretreatment minus posttreatment concentration of organisms (eg, 6 log = 99.999% removal).

b

Actual field practice by unskilled persons (effectiveness) depends on water quality, quality, and age of filter or materials, following proper procedure, and other factors.

c

Summary estimates from published data vary with consistency and correct use of technique, integrity of techniques (eg, cracked filter), and other household sanitation measures.

In disaster situations such as floods, hurricanes, and earthquakes, sanitation and water treatment facilities are frequently damaged or inundated, so household or point-of-use water disinfection is advised. Chlorine is the simplest method, similar to household water disinfection where there is no sanitation or improved water sources.[20, 99, 169] Cloudy water should first be clarified before using hypochlorite.

On long-distance ocean-going boats where water must be desalinated as well as disinfected during the voyage, only reverse osmosis membrane filters are adequate. Water storage also requires consideration. Iodine will work for short periods only (ie, weeks) because it is a poor algaecide. For prolonged storage, water should be chlorinated and kept in a tightly sealed container to reduce the risk of contamination. For daily use, narrow-mouthed jars or containers with water spigots prevent contamination from repeated contact with hands or utensils.[170]

Relatively few studies compare multiple techniques or devices.[28, 92, 96, 168, 171, 172, 173, 174, 175, 176, 177, 178, 179] For more detailed discussion of disinfection techniques and available devices, see Backer.[180] For reviews of water disinfection techniques and effectiveness and efficacy data, see the following additional references.[69, 168, 181, 182]

Sanitation

Sanitation and water treatment are inextricably linked. Studies in developing countries have demonstrated a clear benefit of safe drinking water, hygiene, and adequate sanitation in the reduction of diarrheal illness and other infections.[183, 184, 185, 186, 187, 188] The benefit is greater when all are applied together, especially with appropriate education.[24, 189] Personal hygiene, particularly handwashing, prevents spread of infection from food contamination during preparation of meals.[190, 191] Disinfection of dishes and utensils is accomplished by rinsing in water containing enough household bleach to achieve a distinct chlorine odor. Use of halogen solutions or potassium permanganate solutions to soak vegetables and fruits can reduce microbial contamination, especially if the surface is scrubbed to remove dirt or other particulates, but neither method reaches organisms that are embedded in surface crevices or protected by other particulate matter.[192] Travelers to remote villages, wilderness areas, and persons in disaster situations should ensure proper waste disposal to prevent additional contamination of water supplies. Human waste should be buried 20 to 30 cm (8 to 12 in) deep, at least 30 m (100 ft) from any water, and at a location from which water run-off is not likely to wash organisms into nearby water sources. Groups of 3 persons or more should dig a common latrine to avoid numerous individual potholes and inadequate disposal.

Conclusion

Wilderness and international travelers should carry an effective means of disinfecting water. It is important for disaster and medical relief workers to understand the common methods of water treatment and improvisational methods. It is not possible for travelers to judge the microbiologic quality of water, and it is prudent to assume that even tap water is nonpotable in many locations. Simple and effective field techniques to improve microbiologic water quality are available to travelers. It is important to understand the basic principles and limitations of heat, filtration, and UV and chemical disinfection and then to become familiar with at least one technique appropriate for the destination, water source, and needs of the travelers.

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Study concept and design (HDB, RWD, VRH); obtaining funding: N/A; acquisition of the data: N/A; analysis of the data: N/A; drafting of the manuscript (HDB, RWD, VRH); critical revision of the manuscript (HDB, RWD, VRH); approval of final manuscript (HDB, RWD, VRH).

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Supplementary materials

The following is the supplementary data related to this article.Download : Download Word document (15KB)

Supplementary Table 1. American College of Chest Physicians (ACCP) classification scheme for grading evidence and recommendations in clinical guidelines

References

1

World Health Organization**Combating waterborne disease at the household level. Geneva, Switzerland**
Available at:
https://www.who.int/water_sanitation_health/publications/combating-waterborne-disease/en/ (2007), Accessed 25th Feb 2019
Google Scholar

2

D. Schoenen**Role of disinfection in suppressing the spread of pathogens with drinking water: possibilities and limitations**
Water Res, 36 (15) (2002), pp. 3874-3888
ArticleDownload PDFView Record in ScopusGoogle Scholar

3

G. Guyatt, D. Gutterman, M.H. Baumann, D. Addrizzo-Harris, E.M. Hylek, B. Phillips, *et al.***Grading strength of recommendations and quality of evidence in clinical guidelines: report from an american college of chest physicians task force**
Chest, 129 (1) (2006), pp. 174-181
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

4

J. Yoder, V. Roberts, G.F. Craun, V. Hill, L.A. Hicks, N.T. Alexander, *et al.***Surveillance for waterborne disease and outbreaks associated with drinking water and water not intended for drinking—United States, 2005–2006**
MMWR Surveill Summ, 57 (9) (2008), pp. 39-62
View Record in ScopusGoogle Scholar

5

J.M. Colford Jr., J.F. Hilton, C.C. Wright, B.F. Arnold, S. Saha, T.J. Wade, *et al.***The Sonoma water evaluation trial: a randomized drinking water intervention trial to reduce gastrointestinal illness in older adults**
Am J Public Health, 99 (11) (2009), pp. 1988-1995
View Record in ScopusGoogle Scholar

6

K.A. Reynolds, K.D. Mena, C.P. Gerba**Risk of waterborne illness via drinking water in the United States**
Rev Environ Contam Toxicol, 192 (2008), pp. 117-158
CrossRefView Record in ScopusGoogle Scholar

7

R.P. McClung, D.M. Roth, M. Vigar, V.A. Roberts, A.M. Kahler, L.A. Cocley, *et al.***Waterborne disease outbreaks associated with environmental and undetermined exposures to water—United States, 2013–2014**
MMWR Morb Mortal Wkly Rep, 66 (44) (2017), pp. 1222-1225
CrossRefView Record in ScopusGoogle Scholar

8

R.W. Derlet, J.R. Carlson**An analysis of wilderness water in Kings Canyon, Sequoia, and Yosemite national parks for coliform and pathologic bacteria**
Wilderness Environ Med, 15 (4) (2004), pp. 238-244
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

9

R.W. Derlet, K.A. Ger, J.R. Richards, J.R. Carlson**Risk factors for coliform bacteria in backcountry lakes and streams in the Sierra Nevada mountains: a 5-year study**
Wilderness Environ Med, 19 (2) (2008), pp. 82-90
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

10

Y. Feng, L. Xiao**Zoonotic potential and molecular epidemiology of *Giardia* species and giardiasis**
Clin Microbiol Rev, 24 (1) (2011), pp. 110-140
CrossRefGoogle Scholar

11

WHO-UNICEF**Progress on drinking water, sanitation and hygiene: 2017 update and SDG baselines. Geneva: World Health Organization (WHO) and the United Nations Children's Fund (UNICEF)**
Available at:
https://www.unicef.org/publications/files/Progress_on_Drinking_Water_Sanitation_and_Hygiene_2017.pdf (2017), Accessed 25th Feb 2019
Google Scholar

12

N. Ngwenya, E.J. Ncube, J. Parsons**Recent advances in drinking water disinfection: successes and challenges**
Rev Environ Contam Toxicol, 222 (2013), pp. 111-170
CrossRefView Record in ScopusGoogle Scholar

13

WHO-UNICEF**Progress on Sanitation and Drinking Water–2015 update and MDG assessment**
World Health Organization (WHO) and the United Nations Children's Fund (UNICEF, Geneva (2015)
Google Scholar

14

T.F. Clasen, A. Bastable**Faecal contamination of drinking water during collection and household storage: the need to extend protection to the point of use**
J Water Health, 1 (3) (2003), pp. 109-115
View Record in ScopusGoogle Scholar

15

A.I. Gil, C.F. Lanata, S.M. Hartinger, D. Mausezahl, B. Padilla, T.J. Ochoa, *et al.***Fecal contamination of food, water, hands, and kitchen utensils at the**

household level in rural areas of Peru
J Environ Health, 76 (6) (2014), pp. 102-106
View Record in ScopusGoogle Scholar

16
J.D. Kravitz, M. Nyaphisi, R. Mandel, E. Petersen**Quantitative bacterial examination of domestic water supplies in the Lesotho Highlands: water quality, sanitation, and village health**
Bull World Health Organ, 77 (10) (1999), pp. 829-836
View Record in ScopusGoogle Scholar

17
S.K. Rai, K. Ono, J.I. Yanagida, S. Ishiyama-Imura, M. Kurokawa, C.K. Rai**A large-scale study of bacterial contamination of drinking water and its public health impact in Nepal**
Nepal Med Coll J, 14 (3) (2012), pp. 234-240
View Record in ScopusGoogle Scholar

18
E. Haramoto**Detection of waterborne protozoa, viruses, and bacteria in groundwater and other water samples in the Kathmandu Valley, Nepal. IOP Conference Series**
Earth Environ Sci, 120 (2018), pp. 1-7
Google Scholar

19
J. Wright, S. Gundry, R. Conroy**Household drinking water in developing countries: a systematic review of microbiological contamination between source and point-of-use**
Tropical Med Int Health, 9 (1) (2004), pp. 106-117
View Record in ScopusGoogle Scholar

20
D. Lantagne**Sodium hypochlorite dosage for household and emergency water treatment**
J Am Water Works Assoc, 100 (8) (2008), pp. 106-119
CrossRefGoogle Scholar

21
G. Wang, M. Doyle**Survival of enterohemorrhagic *Escherichia coli* O157:H7 in water**
J Food Prot, 61 (6) (1998), pp. 662-667
View Record in ScopusGoogle Scholar

22
T.E. Ford**Microbiological safety of drinking water: United States and global perspectives**
Environ Health Perspect, 107 (suppl 1) (1999), pp. 191-206
View Record in ScopusGoogle Scholar

23

C. Hurst, R. Clark, S. Regli**Estimating the risk of acquiring infectious disease from ingestion of water**
C. Hurst (Ed.), Modeling Disease Transmission and its Prevention by Disinfection, Cambridge University Press, Melbourne (1996), pp. 99-139
Google Scholar

24

M. Sobsey, T. Handzel, L. Venczel**Chlorination and safe storage of household drinking water in developing countries to reduce waterborne disease**
Water Sci Technol, 47 (3) (2003), pp. 221-228
View Record in ScopusGoogle Scholar

25

J.L. Murphy, A.M. Kahler, I. Nansubuga, E.M. Nanyunja, B. Kaplan, N. Jothikumar, *et al.***Environmental survey of drinking water sources in Kampala, Uganda, during a typhoid fever outbreak**
Appl Environ Microbiol, 83 (23) (2017), pp. 1-11
CrossRefView Record in ScopusGoogle Scholar

26

World Health Organization**Guidelines for drinking water quality**
WHO, Geneva (2011)
Google Scholar

27

US Environmental Protection Agency**Report to Task Force: Guide Standard and Protocol for Testing Microbiological Water Purifiers**
USEPA, Cincinnati (1987)
(Revision)
Google Scholar

28

E. Geldreich, D. Reasoner**Home treatment devices and water quality**
G. McFeters (Ed.), Drinking Water Microbiology, Springer-Verlag, New York (1990), pp. 147-168
CrossRefGoogle Scholar

29

A. Rosen, R. Booth**Taste and odor control**
American Water Works Association (Ed.), Water Quality and Treatment: A Handbook of Public Water Supplies, McGraw Hill, New York (1971)
Google Scholar

30

US Environmental Protection Agency Drinking Water Health Effects Task Force**Health Effects of Drinking Water Treatment Technologies**
Lewis Publisher, Michigan (1989)
Google Scholar

31

National Academy of Sciences Safe Drinking Water Committee**The disinfection of drinking water**
Drinking Water and Health, 2 (1980), pp. 5-139
Google Scholar

32

M. Le Chevallier, G. McFeters**Microbiology of activated carbon**
G. McFeters (Ed.), Drinking Water Microbiology, Springer-Verlag, New York (1990), pp. 104-120
CrossRefGoogle Scholar

33

G.J. Medema, F.M. Schets, P.F. Teunis, A.H. Havelaar**Sedimentation of free and attached *Cryptosporidium* oocysts and *Giardia* cysts in water**
Appl Environ Microbiol, 64 (11) (1998), pp. 4460-4466
CrossRefView Record in ScopusGoogle Scholar

34

AWWA Coagulation Committee**Committee report: coagulation as an integrated water treatment process**
J Am Water Works Assoc (1989), p. 74
Google Scholar

35

J. Cohen, S. Hannah**Water Quality and Treatment: A Handbook of Public Water Supplies**
McGraw-Hill, New York (1971)
Google Scholar

36

J.A. Crump, G.O. Okoth, L. Slutsker, D.O. Ogaja, B.H. Keswick, S.P. Luby**Effect of point-of-use disinfection, flocculation and combined flocculation-disinfection on drinking water quality in western Kenya**
J Appl Microbiol, 97 (1) (2004), pp. 225-231
View Record in ScopusGoogle Scholar

37

K. Preston, D. Lantagne, N. Kotlarz, K. Jellison**Turbidity and chlorine demand reduction using alum and moringa flocculation before household chlorination in developing countries**
J Water Health, 8 (1) (2010), pp. 60-70
CrossRefView Record in ScopusGoogle Scholar

38

N. Kotlarz, D. Lantagne, K. Preston, K. Jellison**Turbidity and chlorine demand reduction using locally available physical water clarification mechanisms before household chlorination in developing countries**
J Water Health, 7 (3) (2009), pp. 497-506
CrossRefView Record in ScopusGoogle Scholar

39

M.W. LeChevallier, T. Evans, R.J. Seidler**Effect of turbidity on chlorination efficiency and bacterial persistence in drinking water**
Appl Environ Microbiol, 42 (1) (1981), pp. 159-167
CrossRefView Record in ScopusGoogle Scholar

40

J.A. Crump, P.O. Otieno, L. Slutsker, B.H. Keswick, D.H. Rosen, R.O.M. Hoekstra, *et al.***Household based treatment of drinking water with flocculant-disinfectant for preventing diarrhoea in areas with turbid source water in rural western Kenya: cluster randomised controlled trial**
BMJ, 331 (7515) (2005), pp. 478-484
CrossRefView Record in ScopusGoogle Scholar

41

M.E. Reller, C.E. Mendoza, M.B. Lopez, M. Alvarez, R.M. Hoekstera, C.A. Olson, *et al.***A randomized controlled trial of household-based flocculant-disinfectant drinking water treatment for diarrhea prevention in rural Guatemala**
Am J Trop Med Hyg, 69 (4) (2003), pp. 411-419
View Record in ScopusGoogle Scholar

42

E. Powers**Efficacy of flocculating and other emergency water purification tablets**
Report Natick/TR-93/033, United States Army Natick Research, Development and Engineering Center, Natick, MA (1993)
Google Scholar

43

E. Powers, C. Boutros, B. Harper**Biocidal efficacy of a flocculating emergency water purification tablet**
Appl Environ Microbiol, 60 (1994), pp. 2316-2323
CrossRefView Record in ScopusGoogle Scholar

44

M.S. Islam, M. Ansaruzzaman, Z.H. Mahmud, M.A. Marin, A.K. Malik, S.B. Neogi, *et al.***A novel and simple mixture as point-of-use water treatment agent to produce safe drinking water**
Trans R Soc Trop Med Hyg, 108 (5) (2014), pp. 290-296
CrossRefView Record in ScopusGoogle Scholar

45

US Army**Sanitary control and surveillance of field water supplies**
Dept. of Army Technical Bulletin (TB Med 577), Departments of the Army, Navy, and Air Force, Washington, DC (2005)
Available at:
https://armypubs.army.mil/epubs/DR_pubs/DR_a/pdf/web/tbmed577.pdf,
Accessed 25th Feb 2019
Google Scholar

46

M. Chaudhuri, S. Sattar**Domestic water treatment for developing countries**

G. McFeters (Ed.), Drinking Water Microbiology, Springer-Verlag, New York (1990)
Google Scholar

47
M. Sobsey**Managing water in the home: accelerated health gains from improved water supply**
WHO/SDE/WSH/02.07, Geneva (2002)
Available at:
https://www.who.int/water_sanitation_health/dwq/WSH02.07.pdf, Accessed 25th Feb 2019
Google Scholar

48
W. Frazier, D. Westhoff**Preservation by Use of High Temperatures**
McGraw-Hill, New York (1978)
Google Scholar

49
M.F. Islam, R.B. Johnston**Household pasteurization of drinking-water: the chulli water-treatment system**
J Health Popul Nutr, 24 (3) (2006), pp. 356-362
View Record in ScopusGoogle Scholar

50
R. Fayer**Effect of high temperature on infectivity of *Cryptosporidium parvum* oocysts in water**
Appl Environ Microbiol, 60 (8) (1994), pp. 273-275
Google Scholar

51
J. Bandres, J. Mathewson, H. DuPont**Heat susceptibility of bacterial enteropathogens**
Arch Intern Med, 148 (10) (1988), pp. 2261-2263
CrossRefView Record in ScopusGoogle Scholar

52
V. Alder, R. Simpson**Sterilization and disinfection by heat methods**
A. Russel, W. Hugo, G. Ayliffe (Eds.), Principles and Practice of Disinfection, Preservation, and Sterilization (2nd ed.), Blackwell Scientific, Oxford (1992), p. 483
Google Scholar

53
R. Aukerman, D. Monzingo**Water treatment to inactive *Giardia***
J Forestry, 87 (11) (1989), pp. 18-21
View Record in ScopusGoogle Scholar

54
J. Ongerth, R. Johnson, S. MacDonald, F. Frost, H. Stibbs**Backcountry water treatment to prevent giardiasis**
Am J Public Health, 79 (1989), pp. 1633-1637

View Record in ScopusGoogle Scholar

55

A. Bingham, E. Jarroll, M. EA**Physical factors of excystation in vitro and excystation vs eosin exclusion as determinants of viability**
Exp Parasitol, 47 (2) (1979), pp. 284-291
ArticleDownload PDFView Record in ScopusGoogle Scholar

56

B. Anderson**Moist heat inactivation of *Cryptosporidium***
Am J Public Health, 75 (1985), pp. 1433-1434
View Record in ScopusGoogle Scholar

57

M. Shephart**Helminthological aspects of sewage treatment**
R. Feachem, M. McGarry, D. Mara (Eds.), Water, Wastes, and Health in Hot Climates, John Wiley and Sons, New York (1977), pp. 299-310
Google Scholar

58

J. Perkins**Thermal destruction of microorganisms: Heat inactivation of viruses**
C. Thomas (Ed.), Principles and Methods of Sterilization in Health Sciences, Springfield (1969), pp. 63-94
Google Scholar

59

E. Tuladhar, M. Bouwknegt, M.H. Zwietering, M. Koopmans, E. Duizer**Thermal stability of structurally different viruses with proven or potential relevance to food safety**
J Appl Microbiol, 112 (5) (2012), pp. 1050-1057
CrossRefView Record in ScopusGoogle Scholar

60

L. Baert, J. Debevere, M. Uyttendaele**The efficacy of preservation methods to inactivate foodborne viruses**
Int J Food Microbiol, 131 (2–3) (2009), pp. 83-94
ArticleDownload PDFView Record in ScopusGoogle Scholar

61

S. Bidawid, J.M. Farber, S.A. Sattar, S. Hayward**Heat inactivation of hepatitis A virus in dairy foods**
J Food Prot, 63 (4) (2000), pp. 522-528
View Record in ScopusGoogle Scholar

62

S. Krugman, J. Giles, J. Hammond**Hepatitis virus: effect of heat on the infectivity and antigenicity of the MS-1 and MS-2 strains**
J Infect Dis, 122 (1970), pp. 432-436
CrossRefView Record in ScopusGoogle Scholar

63

World Health Organization**Boil water**

Available at:
http://www.who.int/water_sanitation_health/dwq/Boiling_water_01_15.pdf?ua=1 (2015), Accessed 25th Feb 2019
Google Scholar

64
H. Neumann**Alternatives to water chlorination (Correspondence)**
Rev Infect Dis, 3 (6) (1981), pp. 1255-1257
CrossRefView Record in ScopusGoogle Scholar

65
H. Neumann**Bacteriological safety of hot tapwater in developing countries**
Public Health Rep, 84 (9) (1969), pp. 812-814
CrossRefView Record in ScopusGoogle Scholar

66
C. Groh, D. MacPherson, D. Groves**Effect of heat on the sterilization of artifically contaminated water**
J Travel Med, 3 (1) (1996), pp. 11-13
CrossRefView Record in ScopusGoogle Scholar

67
K.G. McGuigan**Solar disinfection: use of sunlight to decontaminate drinking water in developing countries**
J Med Microbiol, 48 (9) (1999), pp. 785-787
CrossRefView Record in ScopusGoogle Scholar

68
D.A. Ciochetti, R.H. Metcalf**Pasteurization of naturally contaminated water with solar energy**
Appl Environ Microbiol, 47 (2) (1984), pp. 223-228
CrossRefView Record in ScopusGoogle Scholar

69
M. LeChevallier, A. Kwok-Keung**Water Treatment and Pathogen Control**
London: UK
(2004)
Google Scholar

70
S. Abd-Elmaksoud, J.E. Naranjo, C.P. Gerba**Assessment of a portable handheld UV light device for the disinfection of viruses and bacteria in water**
Food Environ Virol, 5 (2) (2013), pp. 87-90
CrossRefView Record in ScopusGoogle Scholar

71
F. Reygadas, J.S. Gruber, I. Ray, K.L. Nelson**Field efficacy evaluation and post-treatment contamination risk assessment of an ultraviolet disinfection and safe storage system**
Water Res, 85 (2015), pp. 74-84

ArticleDownload PDFView Record in ScopusGoogle Scholar

72

W.A. Hijnen, E.F. Beerendonk, G.J. Medema**Inactivation credit of UV radiation for viruses, bacteria and protozoan (oo)cysts in water: a review**
Water Res, 40 (1) (2006), pp. 3-22
ArticleDownload PDFView Record in ScopusGoogle Scholar

73

K.G. Linden, G.A. Shin, G. Faubert, W. Cairns, M.D. Sobsey**UV disinfection of *Giardia lamblia* cysts in water**
Environ Sci Technol, 36 (11) (2002), pp. 2519-2522
View Record in ScopusGoogle Scholar

74

J.L. Zimmer, R.M. Slawson, P.M. Huck**Inactivation and potential repair of *Cryptosporidium* parvum following low- and medium-pressure ultraviolet irradiation**
Water Res, 37 (14) (2003), pp. 3517-3523
ArticleDownload PDFView Record in ScopusGoogle Scholar

75

K.G. McGuigan, R.M. Conroy, H.J. Mosler, M. Preez du, E. Ubomba-Jaswa, P. Fernandez-Ibañez**Solar water disinfection (SODIS): a review from bench-top to roof-top**
J Hazard Mater, 235–236 (2012), pp. 29-46
ArticleDownload PDFView Record in ScopusGoogle Scholar

76

M. Berney, H.U. Weilenmann, A. Simonetti, T. Egli**Efficacy of solar disinfection of *Escherichia coli, Shigella flexneri, Salmonella typhimurium* and *Vibrio cholerae***
J Appl Microbiol, 101 (4) (2006), pp. 828-836
CrossRefView Record in ScopusGoogle Scholar

77

D.X.F. Cervantes**Feasibility of semi-continuous solar disinfection system for developing countries at the household level**
Massachusetts Institute of Technology (2003)
Available at
http://hdl.handle.net/1721.1/29551, Accessed 25th Feb 2019
Google Scholar

78

T. Joyce, K. McGuigan, M. Elmore-Meegan, R. Conroy**Inactivation of fecal bacteria in drinking water by solar heating**
Appl Environ Microbiol, 62 (2) (1996), pp. 399-402
CrossRefView Record in ScopusGoogle Scholar

79

R.M. Conroy, M.E. Meegan, T. Joyce, K. McGuigan, J. Barnes**Solar disinfection of drinking water protects against cholera in children under**

6 years of age
Arch Dis Child, 85 (4) (2001), pp. 293-295
View Record in ScopusGoogle Scholar

80
B.J. King, D. Hoefel, D.P. Daminato, S. Fanok, P.T. Monis**Solar UV reduces *Cryptosporidium parvum* oocyst infectivity in environmental waters**
J Appl Microbiol, 104 (5) (2008), pp. 1311-1323
CrossRefView Record in ScopusGoogle Scholar

81
C. Tedeschi, C. Barsi, S. Peterson, K. Carey**A pilot study of solar water disinfection in the wilderness setting**
J Wild Environ Med, 25 (3) (2014), pp. 340-345
ArticleDownload PDFView Record in ScopusGoogle Scholar

82
D. Mausezahl, A. Christen, G.D. Pacheco, F.A. Tellez, M. Inarte, M.E. Zapata, *et al*.**Solar drinking water disinfection (SODIS) to reduce childhood diarrhoea in rural Bolivia: a cluster-randomized, controlled trial**
PLoS Med, 6 (8) (2009), Article e1000125
CrossRefGoogle Scholar

83
M. Du Preez, K.G. McGuigan, R.M. Conroy**Solar disinfection of drinking water in the prevention of dysentery in South African children aged under 5 years: the role of participant motivation**
Environ Sci Technol, 44 (22) (2010), pp. 8744-8749
CrossRefView Record in ScopusGoogle Scholar

84
M. du Preez, R.M. Conroy, S. Ligondo, J. Hennessy, M. Elmore-Meegan, A. Soita, *et al*.**Randomized intervention study of solar disinfection of drinking water in the prevention of dysentery in Kenyan children aged under 5 years**
Environ Sci Technol, 45 (21) (2011), pp. 9315-9323
CrossRefView Record in ScopusGoogle Scholar

85
R.M. Conroy, M. Elmore-Meegan, T. Joyce, K.G. McGuigan, J. Barnes**Solar disinfection of drinking water and diarrhoea in Maasai children: a controlled field trial**
Lancet (Lond Engl), 348 (9043) (1996), pp. 1695-1697
ArticleDownload PDFView Record in ScopusGoogle Scholar

86
R. Meierhofer, M. Wegelin**SODIS Manual**
department of water and sanitation in developing countries, Swiss federal institute of envirnomental science and technology, Gallen (2002)
Available at https://www.sodis.ch/methode/anwendung/ausbildungsmaterial/dokumente_material/sodismanual_2016.pdf, Accessed 25th Feb 2019

Google Scholar

87

K. McGuigan, T. Joyce, R. Conroy, J. Gillespie, M. Elmore-Meegan**Solar disinfection of drinking water contained in transparent plastic bottles: characterizing the bacterial inactivation process**
J Appl Microbiol, 84 (6) (1998), pp. 1138-1148
View Record in ScopusGoogle Scholar

88

G.K. Rijal, R.S. Fujioka**Synergistic effect of solar radiation and solar heating to disinfect drinking water sources**
Water Sci Technol, 43 (12) (2001), pp. 155-162
View Record in ScopusGoogle Scholar

89

M. Jenkins**What's in the water?**
Backpacker (1996), p. 56
View Record in ScopusGoogle Scholar

90

K.L. Larson, C. Hansen, M. Ritz, D. Carreno**Acceptance and impact of point-of-use water filtration systems in rural Guatemala**
J Nurs Scholarsh, 49 (1) (2017), pp. 96-102
CrossRefView Record in ScopusGoogle Scholar

91

Y. Zhang, Q. Wang, W. Lou, Y. Wang, X. Zhu**Microbiological safety of household membrane water filter**
J Environ Biol, 34(2 Spec No) (2013), pp. 481-487
View Record in ScopusGoogle Scholar

92

Environmental Health Directorate Health Protection Branch**Assessing the Effectiveness of Small Filtration Systems for Point-of-Use Disinfection of Drinking Water Supplies**
Department of National Health and Welfare, Ottawa (1980)
80-EHD-54
Google Scholar

93

O. Schlosser, C. Robert, C. Bourderioux, M. Rey, M.R. de Roubin**Bacterial removal from inexpensive portable water treatment systems for travelers**
J Travel Med, 8 (1) (2001), pp. 12-18
View Record in ScopusGoogle Scholar

94

T. Clasen, J. Brown, O. Suntura, S. Collin**Safe household water treatment and storage using ceramic drip filters: a randomised controlled trial in Bolivia**
Water Sci Technol, 50 (1) (2004), pp. 111-115
View Record in ScopusGoogle Scholar

95

V. Rao, J.M. Symons, A. Ling, P. Wang, T.G. Metcalf, J.C. Hoff, *et al.***Removal of hepatitis A virus and rotavirus by drinking water treatment**
J Am Water Works Assoc, 80 (2) (1988), pp. 59-67
View Record in ScopusGoogle Scholar

96

A. Horman, R. Rimhanen-Finne, L. Maunula, C.H. von Bonsdorff, J. Rapala, K. Lahti, *et al.***Evaluation of the purification capacity of nine portable, small-scale water purification devices**
Water Sci Technol, 50 (1) (2004), pp. 179-183
View Record in ScopusGoogle Scholar

97

B. Mull, V.R. Hill**Recovery of diverse microbes in high turbidity surface water samples using dead-end ultrafiltration**
J Microbiol Methods, 91 (3) (2012), pp. 429-433
ArticleDownload PDFView Record in ScopusGoogle Scholar

98

E.D. Lindquist, C.M. George, J. Perin, K. Neiswender de Calari, W.R. Norman, T.P. Davis, *et al.***A cluster randomized controlled trial to reduce childhood diarrhea using hollow fiber water filter and/or hygiene-sanitation educational interventions**
Am J Trop Med Hyg, 91 (1) (2014), pp. 190-197
CrossRefView Record in ScopusGoogle Scholar

99

D. Lantagne, B. Person, N. Smith, A. Mayer, K. Preston, E. Blanton, *et al.***Emergency water treatment with bleach in the United States: the need to revise EPA recommendations**
Environ Sci Technol, 48 (9) (2014), pp. 5093-5100
CrossRefView Record in ScopusGoogle Scholar

100

A. Kozlicic, A. Hadzic, H. Bevanda**Improvised purification methods for obtaining individual drinking water supply under war and extreme shortage conditions**
Prehosp Disaster Med, 9 (2 Suppl) (1994), pp. S25-S28
View Record in ScopusGoogle Scholar

101

J.F. Morris, J. Murphy, K. Fagerli, C. Schneeberger, P. Jarohn, F. Moke, *et al.***A randomized controlled trial to assess the impact of ceramic water filters on prevention of diarrhea and Cryptosporidiosis in infants and young children-Western Kenya, 2013**
Am J Trop Med Hyg., 98 (5) (2018), pp. 1260-1268
CrossRefView Record in ScopusGoogle Scholar

102

R.C. Moropeng, P. Budeli, L. Mpenyana-Monyatsi, M.N.B. Momba**Dramatic reduction in diarrhoeal diseases through implementation of cost-**

effective household drinking water treatment systems in Makwane Village, Limpopo Province, South Africa
Int J Environ Res Public Health, 15 (3) (2018), p. 410
CrossRefGoogle Scholar

103
M.A. Kirby, C.L. Nagel, G. Rosa, M.M. Umupfasoni, L. Iyakeremye, E.A. Thomas, *et al.***Use, microbiological effectiveness and health impact of a household water filter intervention in rural Rwanda - a matched cohort study**
Int J Hyg Environ Health, 220 (6) (2017), pp. 1020-1029
ArticleDownload PDFView Record in ScopusGoogle Scholar

104
M. du Preez, R.M. Conroy, J.A. Wright, S. Moyo, N. Potgieter, S.W. Gundry**Use of ceramic water filtration in the prevention of diarrheal disease: a randomized controlled trial in rural South Africa and Zimbabwe**
Am J Trop Med Hyg, 79 (5) (2008), pp. 696-701
View Record in ScopusGoogle Scholar

105
T.F. Clasen, J. Brown, S. Collin, O. Suntura, S. Cairncross**Reducing diarrhea through the use of household-based ceramic water filters: a randomized, controlled trial in rural Bolivia**
Am J Trop Med Hyg, 70 (6) (2004), pp. 651-657
View Record in ScopusGoogle Scholar

106
T. Clasen, G. Garcia Parra, S. Boisson, S. Collin**Household-based ceramic water filters for the prevention of diarrhea: a randomized, controlled trial of a pilot program in Colombia**
Am J Trop Med Hyg, 73 (4) (2005), pp. 790-795
View Record in ScopusGoogle Scholar

107
J. Brown, M.D. Sobsey, D. Loomis**Local drinking water filters reduce diarrheal disease in Cambodia: a randomized, controlled trial of the ceramic water purifier**
Am J Trop Med Hyg, 79 (3) (2008), pp. 394-400
View Record in ScopusGoogle Scholar

108
S.S. Tiwari, W.P. Schmidt, J. Darby, Z.G. Kariuki, M.W. Jenkins**Intermittent slow sand filtration for preventing diarrhoea among children in Kenyan households using unimproved water sources: randomized controlled trial**
Tropical Med Int Health, 14 (11) (2009), pp. 1374-1382
CrossRefView Record in ScopusGoogle Scholar

109

C.E. Stauber, G.M. Ortiz, D.P. Loomis, M.D. Sobsey**A randomized controlled trial of the concrete biosand filter and its impact on diarrheal disease in Bonao, Dominican Republic**
Am J Trop Med Hyg, 80 (2) (2009), pp. 286-293
View Record in ScopusGoogle Scholar

110
C.E. Stauber, B. Kominek, K.R. Liang, M.K. Osman, M.D. Sobsey**Evaluation of the impact of the plastic BioSand filter on health and drinking water quality in rural Tamale, Ghana**
Int J Environ Res Public Health, 9 (11) (2012), pp. 3806-3833
CrossRefView Record in ScopusGoogle Scholar

111
A.M. Fabiszewski de Aceituno, C.E. Stauber, A.R. Walters, R.E. Meza Sanchez, M.D. Sobsey**A randomized controlled trial of the plastic-housing BioSand filter and its impact on diarrheal disease in Copan, Honduras**
Am J Trop Med Hyg, 86 (6) (2012), pp. 913-991
CrossRefView Record in ScopusGoogle Scholar

112
W.F. Duke, R.N. Nordin, D. Baker, A. Mazumder**The use and performance of BioSand filters in the Artibonite Valley of Haiti: a field study of 107 households**
Rural Remote Health, 6 (3) (2006), p. 570
View Record in ScopusGoogle Scholar

113
S. Regli**Regulations on filtration and disinfection. Conference on current research in drinking water treatment**
(1987)
Cincinnati, OH
Google Scholar

114
K. Fagerli, K.K. Trivedi, S.V. Sodha, E. Blanton, A. Ati, T. Nguyen, *et al.***Comparison of boiling and chlorination on the quality of stored drinking water and childhood diarrhoea in Indonesian households**
Epidemiol Infect, 145 (15) (2017), pp. 3294-3302
View Record in ScopusGoogle Scholar

115
World Health Organization**Water Treatment and Pathogen Control: Process Efficiency in Achieving Safe Drinking Water**
World Health Organization, London, UK (2004)
Google Scholar

116
G. White**Handbook of Chlorination**
(3rd ed.), Van Nostrand Reinhold, New York (1992)
Google Scholar

117

E. Jarroll, J. Hoff, E. Meyer**Resistance of cysts to disinfection agents**
S. Erlandsen, E. Meyer (Eds.), Giardia and Giardiasis: Biology, Pathogenesis and Epidemiology, Plenum Press, New York (1984), pp. 311-328
CrossRefView Record in ScopusGoogle Scholar

118

Centers for Disease Control and Prevention**Safe Water Systems for the Developing World: A Handbook for Implementing Household-Based Water Treatment and Safe Storage Projects**
Centers for Disease Control and Prevention, Atlanta, GA (2001)
Google Scholar

119

D.S. Lantagne**Viability of commercially available bleach for water treatment in developing countries**
Am J Public Health, 99 (11) (2009), pp. 1975-1978
View Record in ScopusGoogle Scholar

120

B.F. Arnold, J.M. Colford Jr.**Treating water with chlorine at point-of-use to improve water quality and reduce child diarrhea in developing countries: a systematic review and meta-analysis**
Am J Trop Med Hyg, 76 (2) (2007), pp. 354-364
View Record in ScopusGoogle Scholar

121

B. Pickard, S. Clarke, W. Bettin**Chlorine disinfection in the use of individual water purification devices**
US Army Center for Health Promotion and Preventive Medicine (USACHPPM) (2006)
Available at:
https://apps.dtic.mil/dtic/tr/fulltext/u2/a454058.pdf, Accessed 26th Feb 2019
Google Scholar

122

B. Mengistie, Y. Berhane, A. Worku**Household water chlorination reduces incidence of diarrhea among under-five children in rural Ethiopia: a cluster randomized controlled trial**
PLoS One, 8 (10) (2013), Article e77887
CrossRefGoogle Scholar

123

R.K. Shrestha, E. Marseille, J.G. Kahn, J.R. Lule, C. Pitter, J.M. Blandford, *et al.***Cost-effectiveness of home-based chlorination and safe water storage in reducing diarrhea among HIV-affected households in rural Uganda**
Am J Trop Med Hyg, 74 (5) (2006), pp. 884-890
View Record in ScopusGoogle Scholar

124

M. Blaser**Inactivation of *Campylobacter jejuni* by chlorine and monochlorine**

Appl Environ Microbiol, 51 (2) (1986), pp. 307-311
CrossRefView Record in ScopusGoogle Scholar

125

R. Engelbrecht, M.J. Weber, B.L. Salter, C.A. Schmidt**Comparative inactivation of viruses by chlorine**
Appl Environ Microbiol, 40 (1980), pp. 249-255
CrossRefView Record in ScopusGoogle Scholar

126

G.A. Shin, M.D. Sobsey**Inactivation of norovirus by chlorine disinfection of water**
Water Res, 42 (17) (2008), pp. 4562-4568
ArticleDownload PDFView Record in ScopusGoogle Scholar

127

W. Grabow, V. Gauss-Muller, O.W. Prozesky, F. Deinhardt**Inactivation of hepatitis A virus and indicator organisms in water by free chlorine residuals**
Appl Environ Microbiol, 46 (3) (1983), pp. 619-624
CrossRefView Record in ScopusGoogle Scholar

128

M. Sobsey, T. Fuji, R. Hall**Inactivation of cell-associated and dispersed hepatitis A virus in water**
J Am Water Works Assoc, 83 (11) (1991), pp. 64-67
View Record in ScopusGoogle Scholar

129

G. Craun, D. Swedlow, R. Tauxe, R. Clark, K. Fox, E. Geldreich, *et al.***Prevention of waterborne cholera in the United States**
J Am Water Works Assoc, 83 (11) (1991), pp. 40-45
View Record in ScopusGoogle Scholar

130

O. Thraenhart**Measures for disinfection and control of viral hepatitis**
S. Block (Ed.), Disinfection, Sterilization, and Preservation (4th ed.), Lea & Febiger, Philadelphia (1991), pp. 445-472
Google Scholar

131

L. Fraker, D. Gentile, D. Krivoy, M. Condon, H. Backer***Giardia* cyst inactivation by iodine**
J Wilderness Med, 3 (4) (1992), pp. 351-358
CrossRefView Record in ScopusGoogle Scholar

132

C. Hibler, C. Hancock, L. Perger, J. Wegrzyn, K. Swabby**Inactivation of Giardia Cysts with Chlorine at 0.5C to 5.0C**
Denver, AWWA Research Foundation (1987)
Google Scholar

133

J. Hoff**Inactivation of Microbial Agents by Chemical Disinfectants**
US Environmental Protection Agency, Cincinnati (1986)
EPA/600/2-86/067
Google Scholar

134

A. Rubin, D. Evers, C. Eyman, E. Jarroll**Inactivation of Gerbil-cultured *Giardia lamblia* cysts by free chlorine**
Appl Environ Microbiol, 55 (10) (1989), pp. 2592-2594
CrossRefView Record in ScopusGoogle Scholar

135

P. Wallis, B.R. Hammond**Removal and inactivation of *Giardia* cysts in a mobile water treatment plant under field condition: preliminary results**
P. Wallis, B. Hammond (Eds.), Advances in Giardia Research, University of Calgary Press, Calgary (1988), pp. 137-144
View Record in ScopusGoogle Scholar

136

C. Carpenter, R. Fayer, J. Trout, M.J. Beach**Chlorine disinfection of recreational water for *Cryptosporidium parvum***
Emerg Infect Dis, 5 (4) (1999), pp. 579-584
CrossRefView Record in ScopusGoogle Scholar

137

D.G. Korich, J.R. Mead, M.S. Madore, N.A. Sinclair, C.R. Sterling**Effects of ozone, chlorine dioxide, chlorine, and monochloramine on *Cryptosporidium parvum* oocyst viability**
Appl Environ Microbiol, 56 (5) (1990), pp. 1423-1428
CrossRefView Record in ScopusGoogle Scholar

138

G. Briton**Introduction to Environmental Virology**
Wiley, New York (1980)
Google Scholar

139

S. Chang**Modern concepts of disinfection: water treatment in the seventies**
Proceedings of National Specialty Conference on Disinfection (1970)
Google Scholar

140

E. Rice, J. Hoff, F. Schaefer**Inactivation of *Giardia* cysts by chlorine**
Appl Environ Microbiol, 43 (1982), pp. 250-251
CrossRefView Record in ScopusGoogle Scholar

141

J. Shields, V. Hill, M. Arrowood, M. Beach**Inactivation of *Cryptosporidium parvum* under chlorinated recreational water conditions**
J Water Health, 6 (4) (2018), pp. 513-520
Google Scholar

142

M. Sobsey, C. Oldham, D. McCall**Comparative inactivation of hepatitis A and other enteroviruses in water by iodine**
Water Sci Technol, 24 (2) (1991), pp. 331-337
View Record in ScopusGoogle Scholar

143

G. Berg, S. Chang, E. Harris**Devitalization of microorganisms by iodine**
Virology, 22 (1964), pp. 469-481
ArticleDownload PDFView Record in ScopusGoogle Scholar

144

C. Gerba, D. Johnson, M. Hasan**Efficacy of iodine water purification tablets against *Cryptosporidium* oocysts and *Giardia* cysts**
Wilderness Environ Med, 8 (2) (1997), pp. 96-100
ArticleDownload PDFCrossRefView Record in ScopusGoogle Scholar

145

H. Mohamed, J. Brown, R.M. Njee, T. Clasen, H.M. Malebo, S. Mbuligwe**Point-of-use chlorination of turbid water: results from a field study in Tanzania**
J Water Health, 13 (2) (2015), pp. 544-552
CrossRefView Record in ScopusGoogle Scholar

146

D.S. Lantagne, F. Cardinali, B.C. Blount**Disinfection by-product formation and mitigation strategies in point-of-use chlorination with sodium dichloroisocyanurate in Tanzania**
Am J Trop Med Hyg, 83 (1) (2010), pp. 135-143
CrossRefView Record in ScopusGoogle Scholar

147

R. Kinman, A. Black, W. Thomas**Disinfection with iodine**
Water Treatment in the Seventies: Procedings of national specialty conference on disinfection (1970)
Google Scholar

148

W. Thomas, A. Black, G. Freund, R. Kinman**Iodine disinfection of water**
Arch Environ Health, 19 (1) (1969), pp. 124-132
CrossRefView Record in ScopusGoogle Scholar

149

H. Backer, J. Hollowell**Use of iodine for water disinfection: iodine toxicity and maximum recommended dose**
Environ Health Perspect, 108 (8) (2000), pp. 679-684
View Record in ScopusGoogle Scholar

150

W. Gottardi**Iodine and iodine compounds**
S. Block (Ed.), Disinfection, Sterilization, and Preservation (4th ed.), Lea & Febiger, Philadelphia (1991), pp. 152-167

View Record in ScopusGoogle Scholar

151

W.A. Rutala, D.J. Weber**New disinfection and sterilization methods**
Emerg Infect Dis, 7 (2) (2001), pp. 348-353
CrossRefView Record in ScopusGoogle Scholar

152

L. Venczel, M. Arrowood, M. Hurd, M. Sobsey**Inactivation of *Cryptosporidium parvum* oocysts and *Clostridium perfringens* spores by a mixed-oxidant disinfectant and by free chlorine**
Appl Environ Microbiol, 63 (4) (1997), pp. 1598-1601
CrossRefView Record in ScopusGoogle Scholar

153

R.M. Clark, M. Sivagnesan, E.W. Rice, J. Chen**Development of a Ct equation for the inactivation of *Cryptosporidium* occysts with chlorine dioxide**
Water Res, 37 (11) (2003), pp. 2773-2783
ArticleDownload PDFView Record in ScopusGoogle Scholar

154

J.L. Murphy, C.N. Haas, M.J. Arrowood, M.C. Hlavsa, M.J. Beach, V.R. Hill**Efficacy of chlorine dioxide tablets on inactivation of cryptosporidium oocysts**
Environ Sci Technol, 48 (10) (2014), pp. 5849-5856
CrossRefView Record in ScopusGoogle Scholar

155

J.T. Pereira, A.O. Costa, M.B. de Oliveira Silva, W. Schuchard, S.C. Osaki, E.A. de Castro, *et al.***Comparing the efficacy of chlorine, chlorine dioxide, and ozone in the inactivation of *Cryptosporidium parvum* in water from Parana State, Southern Brazil**
Appl Biochem Biotechnol, 151 (2–3) (2008), pp. 464-473
CrossRefView Record in ScopusGoogle Scholar

156

G. Marchin**Fina L Contact and demand-release disinfectants**
Crit Rev Environ Control, 19 (1989), pp. 227-290
View Record in ScopusGoogle Scholar

157

M.B. Fisher, C.R. Keenan, K.L. Nelson, B.M. Voelker**Speeding up solar disinfection (SODIS): effects of hydrogen peroxide, temperature, pH, and copper plus ascorbate on the photoinactivation of *E. coli***
J Water Health, 6 (1) (2008), pp. 35-51
CrossRefView Record in ScopusGoogle Scholar

158

M. D'Aquino, S.A. Teves**Lemon juice as a natural biocide for disinfecting drinking water**
Bull Pan Am Health Organ, 28 (4) (1994), pp. 324-330

View Record in ScopusGoogle Scholar

159

United States Environmental Protection Agency**Alternative Disinfectants and Oxidants Guidance Manual**
EPA 815-R-99-014, US EPA (1999)
Available at:
https://nepis.epa.gov, Accessed 26th Feb 2019
Google Scholar

160

M.N. Chong, B. Jin, C.W. Chow, C. Saint**Recent developments in photocatalytic water treatment technology: a review**
Water Res, 44 (10) (2010), pp. 2997-3027
ArticleDownload PDFView Record in ScopusGoogle Scholar

161

Q. Li, S. Mahendra, D.Y. Lyon, L. Brunet, M.V. Liga, D. Li, *et al.***Antimicrobial nanomaterials for water disinfection and microbial control: potential applications and implications**
Water Res, 42 (18) (2008), pp. 4591-4602
ArticleDownload PDFView Record in ScopusGoogle Scholar

162

O. Sunnotel, R. Verdoold, P.S. Dunlop, W.J. Snelling, C.J. Lowery, J.S. Dooley, *et al.***Photocatalytic inactivation of *Cryptosporidium parvum* on nanostructured titanium dioxide films**
J Water Health, 8 (1) (2010), pp. 83-91
CrossRefView Record in ScopusGoogle Scholar

163

J. Blanco-Galvez, P. Fernandez-Ibanez, S. Malato-Rodriguez**Solar photocatalytic detoxification and disinfection of water: recent overview**
J Solar Energy Eng, 129 (1) (2006), pp. 4-15
Google Scholar

164

D.M. Alrousan, P.S. Dunlop, T.A. McMurray, J.A. Byrne**Photocatalytic inactivation of *E. coli* in surface water using immobilised nanoparticle TiO2 films**
Water Res, 43 (1) (2009), pp. 47-54
ArticleDownload PDFView Record in ScopusGoogle Scholar

165

M.D. Sobsey, C.E. Stauber, L.M. Casanova, J.M. Brown, M.A. Elliott**Point of use household drinking water filtration: a practical, effective solution for providing sustained access to safe drinking water in the developing world**
Environ Sci Technol, 42 (12) (2008), pp. 4261-4267
CrossRefView Record in ScopusGoogle Scholar

166

A.R. Bielefeldt, K. Kowalski, R.S. Summers**Bacterial treatment effectiveness of point-of-use ceramic water filters**
Water Res, 43 (14) (2009), pp. 3559-3565
ArticleDownload PDFView Record in ScopusGoogle Scholar

167

J.K. Mwabi, B.B. Mamba, M.N. Momba**Removal of *Escherichia coli* and faecal coliforms from surface water and groundwater by household water treatment devices/systems: a sustainable solution for improving water quality in rural communities of the Southern African development community region**
Int J Environ Res Public Health, 9 (1) (2012), pp. 139-170
CrossRefView Record in ScopusGoogle Scholar

168

T. Clasen, S. Menon**Microbiological performance of common water treatment devices for household use in India**
Int J Environ Health Res, 17 (2) (2007), pp. 83-93
CrossRefView Record in ScopusGoogle Scholar

169

J.M. Brunkard, E. Ailes, V.A. Roberts, V. Hill, E.D. Hilborn, G.F. Craun, *et al.***Surveillance for waterborne disease outbreaks associated with drinking water—United States, 2007–2008**
MMWR Surveill Summ, 60 (12) (2011), pp. 38-68
View Record in ScopusGoogle Scholar

170

J. Sobel, B. Mahon, C. Mendoza, D. Passaro, F. Cano, K. Baier, *et al.***Reduction of fecal contamination of street-vended beverages in Guatemala by a simple system for water purification and storage, handwashing, and beverage storage**
Am J Trop Med Hyg, 59 (3) (1998), pp. 380-387
CrossRefView Record in ScopusGoogle Scholar

171

D. Cullimore, H. Jacobsen**The efficiency of point of use devices for the exclusion of *Giardia muris* cysts from a model water supply system**
P. Wallis, B. Hammond (Eds.), Advances in Giardia Research, University of Calgary Press, Calgary (1988), pp. 107-112
View Record in ScopusGoogle Scholar

172

P. Hutton, J. Ongerth**Performance evaluation for portable water treatment filters**
Department of Environmental Health, University of Washington, Seattle, WA (1995)
Google Scholar

173

P. Hutton, J. Ongerth**Performance evaluation of ten commercially available portable water filters**

Department of Water Engineering, University of New South Wales, Sydney, Austalia (1997)
Google Scholar

174
E. Jarroll, A. Bingham, E. Meyer***Giardia* cyst destruction: effectiveness of six small water disinfection methods**
Am J Trop Med Hyg, 29 (1) (1980), pp. 8-11
CrossRefView Record in ScopusGoogle Scholar

175
G. Logsdon, J. Symons, R. Hoye, M. Arozarena**Alternative filtration methods for removal of *Giardia* cysts and cyst models**
J Am Water Works Assoc, 73 (2) (1981), pp. 111-118
CrossRefView Record in ScopusGoogle Scholar

176
J. Naranjo, C. Gerba**Evaluation of portable water treatment devices by a condensed version of the guide of standard protocol for microbiological purifiers (USEPA, 1987)**
University of Arizona, Tucson, AZ (1995)
Google Scholar

177
E. Powers, C. Boyd, B. Harper, A. Rubin**Removal of biological and chemical challenge from water by commercial fresh and salt water purification devices**
Technical Report Natick/TR-91-042, United States Army Natick Research, Development and Engineering Center, Natick, MA (1991)
Google Scholar

178
R. Tobin**Performance of point-of-use water treatment devices**
Proceedings of the first conference on cold regions environmental engineering (1983)
Fairbanks, AK
Google Scholar

179
R. Tobin**Testing and evaluating point-of-use treatment devices in Canada**
J Am Water Works Assoc, 79 (10) (1987), pp. 42-45
View Record in ScopusGoogle Scholar

180
H. Backer**Field water disinfection**
P. Auerbach (Ed.), Wilderness Medicine (7th ed.), Elsevier, Philadelphia (2017)
Google Scholar

181
G.E. McDonnell**Antisepsis, Disinfection, and Sterilization**
ASM Press, Washington, DC (2007)

Google Scholar

182

A.R. Bielefeldt**Appropriate and sustainable water disinfection methods for developing communities**

K. Buchanan (Ed.), Water Disinfection, Nova Science Publishers, New York City, NY (2011), pp. 45-75

View Record in ScopusGoogle Scholar

183

T. Clasen, I. Roberts, T. Rabie, W. Schmidt, S. Cairncross**Interventions to improve water quality for preventing diarrhoea**

John Wiley & Sons, Cochrane Library (2006)

Google Scholar

184

M.C. Opryszko, S.W. Majeed, P.M. Hansen, J.A. Myers, D. Baba, R.E. Thompson, *et al.***Water and hygiene interventions to reduce diarrhoea in rural Afghanistan: a randomized controlled study**

J Water Health, 8 (4) (2010), pp. 687-702

CrossRefView Record in ScopusGoogle Scholar

185

S. Jung, Y.A. Doh, D.B. Bizuneh, H. Beyene, J. Seong, H. Kwon, *et al.***The effects of improved sanitation on diarrheal prevalence, incidence, and duration in children under five in the SNNPR State, Ethiopia: study protocol for a randomized controlled trial**

Trials, 17 (1) (2016), p. 204

View Record in ScopusGoogle Scholar

186

V. Garrett, P. Ogutu, P. Mabonga, S. Ombeki, A. Mwaki, G. Aluoch, *et al.***Diarrhoea prevention in a high-risk rural Kenyan population through point-of-use chlorination, safe water storage, sanitation, and rainwater harvesting**

Epidemiol Infect, 136 (11) (2008), pp. 1463-1471

View Record in ScopusGoogle Scholar

187

R. Dreibelbis, M.C. Freeman, L.E. Greene, S. Saboori, R. Rheingans**The impact of school water, sanitation, and hygiene interventions on the health of younger siblings of pupils: a cluster-randomized trial in Kenya**

Am J Public Health, 104 (1) (2014), pp. e91-e97

View Record in ScopusGoogle Scholar

188

S. Cha, J. Lee, D. Seo, B.M. Park, P. Mansiangi, K. Mernard, *et al.***Effects of improved sanitation on diarrheal reduction for children under five in Idiofa, DR Congo: a cluster randomized trial**

Infect Dis Poverty, 6 (1) (2017), pp. 137-149

CrossRefView Record in ScopusGoogle Scholar

189

R.E. Quick, A. Kimura, A. Thevos, M. Tembo, I. Shamputa, L. Hutwagner, *et al.***Diarrhea prevention through household-level water disinfection and safe storage in Zambia**
Am J Trop Med Hyg, 66 (5) (2002), pp. 584-589
CrossRefView Record in ScopusGoogle Scholar

190

S.P. Luby, M. Agboatwalla, J. Painter, A. Altaf, W.L. Billhimer, R.M. Hoekstra, *et al.***Effect of intensive handwashing promotion on childhood diarrhea in high-risk communities in Pakistan: a randomized controlled trial**
JAMA, 291 (21) (2004), pp. 2547-2554
View Record in ScopusGoogle Scholar

191

A. Hashi, A. Kumie, J. Gasana**Hand washing with soap and WASH educational intervention reduces under-five childhood diarrhoea incidence in Jigjiga District, Eastern Ethiopia: a community-based cluster randomized controlled trial**
Prev Med Rep, 6 (2017), pp. 361-368
ArticleDownload PDFView Record in ScopusGoogle Scholar

192

Y.R. Ortega, C.R. Roxas, R.H. Gilman, N.J. Miller, L. Cabrera, C. Taquiri, *et al.***Isolation of *Cryptosporidium parvum* and *Cyclospora cayetanensis* from vegetables collected in markets of an endemic region in Peru**
Am J Trop Med Hyg, 57 (6) (1997), pp. 683-686
CrossRefView Record in ScopusGoogle Scholar

GUIDELINE 9.5.1 EMERGENCY MANAGEMENT OF A VICTIM WHO HAS BEEN POISONED

INTRODUCTION

A poison is a substance (other than an infectious substance) that is harmful to human health if ingested, inhaled, injected, or absorbed through the skin. Substances that are benign or therapeutic at low levels (for example, pharmaceuticals and herbal remedies) may be poisonous at higher concentrations. Toxins are poisons that are produced by living organisms. Venoms are toxins that are injected by an organism.

RECOGNITION

- Poisons can cause harm by a wide range of mechanisms and can cause a wide range of symptoms including unconsciousness, nausea, vomiting, burning pain in the mouth or throat, headache, blurred vision, seizures, difficulty breathing, respiratory arrest, and cardiac arrest.
- Recognition of poisoning may be obvious from the circumstances of the incident, but this is not always true. A person may complain of physical symptoms without realising these are due to a poison. Alternatively, they may exhibit abnormal behaviour, which may be misinterpreted as alcoholic confusion or psychiatric disturbance.
- Most pharmaceuticals are poisonous in overdose. Some are relatively safe unless many times the recommended dose is taken, but many are lethal if less than a single pack is taken simultaneously.
- Poisons may have a rapid effect, but their effects may also be delayed. Speed of effect is determined by the nature of the poison, its concentration, and the time of exposure.
- It is important to seek medical assessment or advice after significant exposure to a poison, even if symptoms are initially mild or absent.

MANAGEMENT

The principles of managing a patient who has been poisoned are:

- Prevention of poisoning of the rescuer.
- Decontamination of the patient.

- Resuscitation and supportive care, using the Australian Resuscitation Council and New Zealand Resuscitation Council Basic Life Support Flowchart (Guideline 8).
- Specific management of particular poisons: antidotes, techniques to remove the poison from the body, and the treatment of complications of the poison. [Class A; LOE Expert Consensus Opinion]
- If the victim is unconscious or is not breathing normally, commence resuscitation if necessary, following the Australian Resuscitation Council and New Zealand Resuscitation Council Basic Life Support Flowchart (Guideline 8) - Ensure that an ambulance has been called

- Prevention of poisoning of the rescuer
    - During first aid and subsequent treatment, the suspected poison should be identified and safely handled to minimise further exposure. The victim may pose a danger if the poisonous substance can be transferred to the rescuer (for example, by contact with contaminated clothing).
    - If the poisoning occurs in an industrial, farm or laboratory setting suspect particularly dangerous agents and take precautions to avoid accidental injury.
    - If more than one person simultaneously appears affected by a poison, there is a high possibility of dangerous environmental contamination.
    - The rescuer may need to wear personal protective equipment (PPE) during decontamination and resuscitation. The need for PPE will be guided by knowledge of the likely poison. If equipment is not available to safely decontaminate and treat a victim, rescue may not be possible. [Class A; LOE Expert Consensus Opinion]
- Decontamination
    - Separate the victim from the poisonous substance. How this is done will depend on the type of the poison. Examples are listed below.

If the poison is **SWALLOWED**

- Give the person who has swallowed the poison a sip of water to wash out their mouth.
- Do NOT try to make them vomit. Do NOT use Ipecac Syrup. 1;2 [Class A; LOE IV]

If the poison is **INHALED**

- Immediately get the person to fresh air, without placing yourself at risk.
- Avoid breathing fumes. Special breathing apparatus may be required, for example, with cyanide or agricultural chemicals poisoning.
- If it is safe to do so, open doors and windows wide.

If the poison enters the **EYE**

- Flood the eye with saline or cold water from a running tap or a cup/jug.
- Continue to flush for 15 minutes, holding the eyelids open.

If the poison contacts the **SKIN**

- Remove contaminated clothing, taking care to avoid contact with the poison.

- Flood skin with running cold water.
- Wash gently with soap and water and rinse well.

- Resuscitation and supportive care
    - If the victim is unconscious or is not breathing normally, commence resuscitation if necessary, following the Australian Resuscitation Council and New Zealand Resuscitation Council Basic Life Support Flowchart (Guideline 8). [Class A; LOE Expert Consensus Opinion]
    - Before commencing resuscitation, quickly wipe obvious contamination from around the mouth.
    - Ensure that an ambulance has been called
    - A self-inflating bag-valve-mask apparatus is the safest way to provide ventilation for the BLS rescuer. If this equipment is not available, mouth-to-mask or mouth-to-mouth ventilation may be considered depending on the chemical ingested. Mouth-to-mouth ventilation should be avoided if cyanide or organophosphate poisoning is suspected. [Class A; LOE Expert Consensus Opinion]
    - Inhaled poisons are unlikely to pose a risk during mouth to mouth ventilation unless the victim is contaminated with the liquid phase of the inhaled poison.
- Specific Management of particular poisons
    - If possible, ascertain what poison or pharmaceutical has been taken, how much, and when. Then obtain medical advice promptly. The source of medical advice will depend on the situation. Options include:
        - Australian Poisons Information Centre on 13 11 26 anywhere in Australia 24 hours a day, 7 days a week.
        - New Zealand poisons centre 0800 764 766 (0800 POISON)
        - Occupational health facilities

Some poisons have specific antidotes, but (with some exceptions, such as cyanide) these are rarely used outside hospital. However, accurately identifying these poisons will help treatment so if there are packets or bottles they should go with the victim to hospital.

Poisons with antidotes include:

- Cyanide
- Organophosphates
- Iron
- Paracetamol
- Antifreeze
- Methanol
- some Antidepressants
- Digoxin
- Warfarin

If unable to get advice, or while waiting for help to arrive:

- Monitor the victim, especially the Airway, Breathing and Circulation, and manage according to the Australian Resuscitation Council and New Zealand

Resuscitation Council Basic Life Support Flowchart (Guideline 8). [Class A; LOE Expert Consensus Opinion]

SUBSTANCES COMMONLY CAUSING POISONING

Paracetamol is the most common pharmaceutical overdose leading to hospital admission, and is also responsible for the most calls to Australian Poisons Information Centres.^3 Paracetamol is involved in a large proportion of accidental poisoning in children. Without treatment, even small amounts of paracetamol are sufficient to cause an adult significant liver damage and even death and effects may be delayed. Any poisoning in excess of recommended doses requires immediate medical attention. The treatment for paracetamol poisoning is most effective if administered as early as possible.

Organic substances such as glues, hair spray, aerosol paints, lighter fluid, dry cleaning fluid, nail polish remover and petrol may be deliberately inhaled to produce altered sensation. Poisonous effects include:

- Hyperactivity, followed by drowsiness and unconsciousness
- Irregular heartbeat, followed by cardiac arrest
- Difficulty breathing

These dangers are increased by exercise, inhaling poison from bag, or inhalation in a confined space.

In Australia, household chemicals are the third most common cause of poisoning in children after pharmaceuticals and venoms.^4 Household chemicals may include caustic substances (e.g. dishwasher detergent) which have a risk of significant damage to the oesophagus and lung.^5 Do not induce vomiting to prevent further damage to the oesophagus and possible lung damage due to aspiration.

Fungi (mushrooms; toadstools) grow widely throughout Australia. Some are edible, but some are poisonous, causing hallucinations, vomiting, and diarrhea. Ingestion of even one Amanita phalloides mushroom can cause liver failure and death. Cooking does not neutralise the toxin. Most reported cases of mushroom poisoning are in children less than five eating mushrooms growing in their home gardens.^6 The risk can be reduced by regularly checking for and removing garden mushrooms.

Cyanide is not a common cause of poisoning but may occur from inhalation of fume during house or industrial fires or occupational exposure. As early treatment with an antidote can be lifesaving, workplaces with a risk of cyanide exposure should be adequately prepared. Depending on the risk, preparation should include:

- frequent inspection of work practices,
- plans for containment and decontamination of spills,
- access to a resuscitation device capable of delivering high oxygen concentrations with the ability to assist ventilation if necessary, and
- a cyanide antidote.

The contents of a workplace Cyanide Emergency Kit should be determined by a qualified occupational health assessor, taking into account the nature of the threat, first aider training, and the proximity of external assistance. Several cyanide antidotes are commercially available.

Unlike other strategies, intravenous hydroxycobalamin (vitamin B 12) has few adverse effects.7;8 There is more evidence for the efficacy of hydroxycobalamin (three fair quality studies9-12 and three poor quality studies13-15) than for the alternatives.

Adult patients with suspected severe cyanide poisoning (including those in cardiac arrest) should receive immediate intravenous hydroxycobalamin, 5mg over 15 minutes with repeat dosing up to 15mg. [Class A; LOE IV] Even if only BLS rescuers are likely to be immediately available, keeping hydroxycobalamin for use by pre-hospital ALS or hospital personnel may still be useful.

PREVENTION

- Many poisons are substances that also have a useful purpose. Poisoning is particularly common in children and vulnerable adults. Ensuring poisons are only accessible by people who need and know how to use them reduces their risk of harm.
- Make a survey of your home or workplace and identify all poisonous substances.
- Remove poisons or medicines that are unwanted. Dispose of chemicals safely using their accompanying directions. Pharmaceuticals can be returned to a pharmacy for safe disposal, which is safer and more environmentally friendly than disposal in domestic waste or flushing down a toilet. The Poisons Information Centre can also advise on methods of safe disposal.
- Store poisonous substances in their original containers in locked or child-resistant cupboards or containers out of reach of children. Do not store medicines in the refrigerator unless advised to do so by a pharmacist.
- Use non-poisonous alternatives to cleaning products, insecticides, etc. when possible.
- Keep the amount of poisonous substances stored in a home to a minimum.
- When possible, choose substances available in child-resistant packaging. However, do not rely on child-resistant packaging to prevent a child’s access to a poison.
- Read medicine labels and use according to the directions. Ensure the right:
  - medication
  - person
  - dose
  - route of administration
  - time and frequency of administration
- Wear the recommended personal protective equipment when using toxic or caustic chemicals, for example spraying, painting, or oven cleaning.
- Do not eat or drink near poisons.

FURTHER READING

ANZCOR Guideline 8: Cardiopulmonary Resuscitation

ARC Guidelines 9.4.1 – 9.4.8 Envenomations

For information on specific poisons, consult the Australian Poisons Information Centre on 13 11 26 or New Zealand poisons centre 0800 764 766 (0800 POISON)

REFERENCES

1. Heard K: The changing indications of gastrointestinal decontamination in poisonings. Clin.Lab Med. 2006; 26: 1-12, vii
2. Greene SL, Dargan PI, Jones AL: Acute poisoning: understanding 90% of cases in a nutshell. Postgrad.Med.J. 2005; 81: 204-
3. Daly FF, Fountain JS, Murray L, Graudins A, Buckley NA: Guidelines for the management of paracetamol poisoning in Australia and New Zealand--explanation and elaboration. A consensus statement from clinical toxicologists consulting to the Australasian poisons information centres. Med.J.Aust. 2008; 188: 296-
4. Lam LT: Childhood and adolescence poisoning in NSW, Australia: an analysis of age, sex, geographic, and poison types. Inj.Prev. 2003; 9: 338-
5. Turner A, Robinson P: Respiratory and gastrointestinal complications of caustic ingestion in children. Emerg.Med.J. 2005; 22: 359-
6. Fungi poisoning. 1999. Melbourne, State of Victoria. Ref Type: Pamphlet
7. Uhl W, Nolting A, Golor G, Rost KL, Kovar A: Safety of hydroxocobalamin in healthy volunteers in a randomized, placebo-controlled study. Clin.Toxicol. (Phila) 2006; 44 Suppl 1: 17-
8. Forsyth JC, Mueller PD, Becker CE, Osterloh J, Benowitz NL, Rumack BH et al.: Hydroxocobalamin as a cyanide antidote: safety, efficacy and pharmacokinetics in heavily smoking normal volunteers. J.Toxicol.Clin.Toxicol. 1993; 31: 277-
9. Borron SW, Baud FJ, Barriot P, Imbert M, Bismuth C: Prospective study of hydroxocobalamin for acute cyanide poisoning in smoke inhalation. Ann.Emerg.Med. 2007; 49: 794-801, 801
10. Houeto P, Hoffman JR, Imbert M, Levillain P, Baud FJ: Relation of blood cyanide to plasma cyanocobalamin concentration after a fixed dose of hydroxocobalamin in cyanide poisoning. Lancet 1995; 346: 605-
11. Espinoza OB, Perez M, Ramirez MS: Bitter cassava poisoning in eight children: a case report. Vet.Hum.Toxicol. 1992; 34: 65
12. Fortin JL, Desmettre T, Manzon C, Judic-Peureux V, Peugeot-Mortier C, Giocanti JP et al.: Cyanide poisoning and cardiac disorders: 161 cases. J.Emerg.Med. 2010; 38: 467-
13. Fortin JL, Giocanti JP, Ruttimann M, Kowalski JJ: Prehospital administration of hydroxocobalamin for smoke inhalation-associated cyanide poisoning: 8 years of experience in the Paris Fire Brigade. Clin.Toxicol.(Phila) 2006; 44 Suppl 1: 37-
14. Borron SW, Baud FJ, Megarbane B, Bismuth C: Hydroxocobalamin for severe acute cyanide poisoning by ingestion or inhalation. Am J.Emerg.Med. 2007; 25: 551-

15. Pontal PG, Bismuth C, Garnier R: Therapeutic attitude in cyanide poisoning: retrospective study of 24 non-lethal cases. Veterinary and Human Toxicology 1982; 24: 286