WSACS | The Abdominal Compartment Society

Abdominal compartment syndrome

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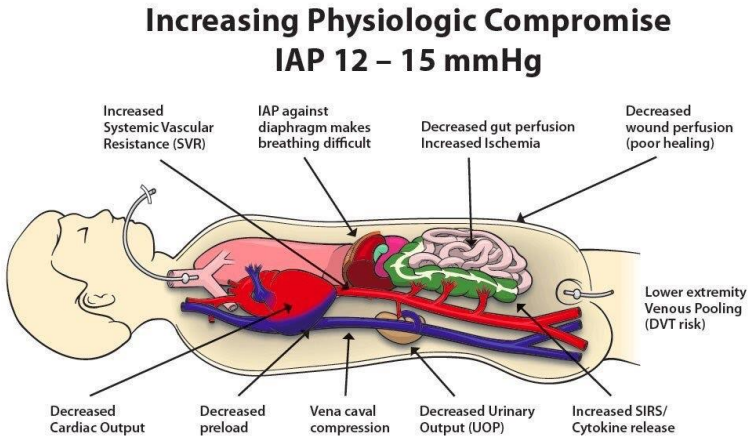
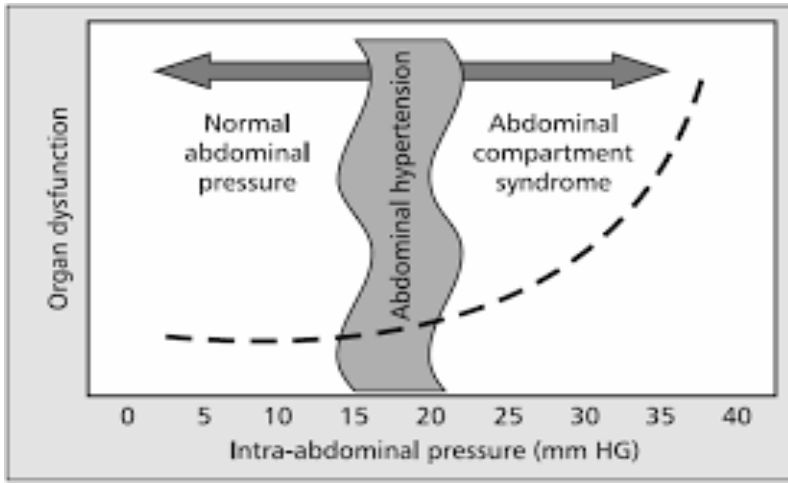
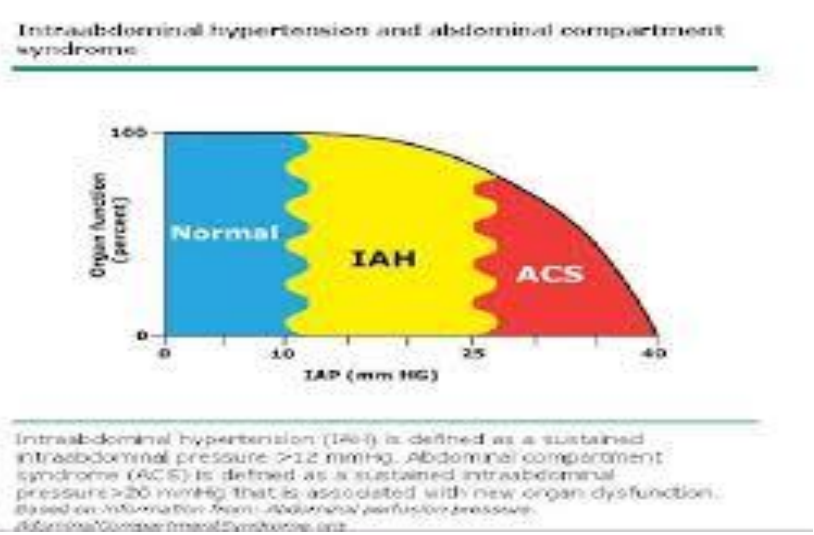


Practice Essentials

- *Compartment syndrome occurs when a fixed compartment, defined by myofascial elements or bone, becomes subject to increased pressure, leading to ischemia and organ dysfunction*

- *The exact clinical conditions that define abdominal compartment syndrome (**ACS**) are controversial; however, organ dysfunction caused by intra-abdominal hypertension (**IAH**) is considered to be abdominal compartment syndrome.*





Practice Essentials

- *The dysfunction may be respiratory insufficiency secondary to compromised tidal volumes, decreased urine output caused by falling renal perfusion, or any organ dysfunction caused by increased abdominal compartment pressure*

- *However, prevention and early treatment of the potential cause may prevent progression of IAH to ACS*

- recognized clinically in the 19th century when Marey and Burt observed its association with declines in respiratory function.

- In the early 20th century, Emerson's animal experiments demonstrated mortality associated with abdominal compartment syndrome.

- Initially, cardiorespiratory compromise was thought to be the cause; however, renal failure was hypothesized by Wendt and was later studied by Thorington and Schmidt.

- More recently, Kron and Iberti developed a simple method of accurately measuring intra-abdominal pressure. This has led to a better understanding of the relationship between IAH and abdominal compartment syndrome.

The World Society of the Abdominal Compartment Syndrome has published the following definitions and recommendations

- ***IAP*** is approximately 5-7 mm Hg in critically ill adults.
- Intra-abdominal ***hypertension*** is defined by a sustained or repeated pathological elevation in IAP of ≥12 mm Hg.
- ***ACS*** is defined as a sustained IAP >20 mm Hg associated with organ dysfunction/failure.

IAH is graded as follows:

Grade I: IAP 12-15 mm Hg;
Grade II: IAP 16-20 mm Hg;
Grade III: IAP 21-25 mm Hg;
Grade IV: IAP >25 mm Hg.

- Recommend measuring intra-abdominal pressure when any known risk factor for IAH/ACS is present in critically ill or injured patients.
- Recommend that studies of IAH or ACS adopt the trans-bladder technique as a standard IAP measurement technique.
- Recommend decompressive laparotomy to decrease IAP in cases of overt ACS.

Three categories

ACS occur as a consequence of a variety of primary clinical events.

- ***Primary or acute*** abdominal compartment syndrome occurs when intra-abdominal pathology is directly and proximally responsible for the compartment syndrome

- ***Secondary*** abdominal compartment syndrome occurs when *no visible intra-abdominal injury* is present but injuries outside the abdomen cause fluid accumulation

- ***Chronic*** abdominal compartment syndrome occurs in the presence of cirrhosis and ascites or related disease states, often in the later stages of the disease

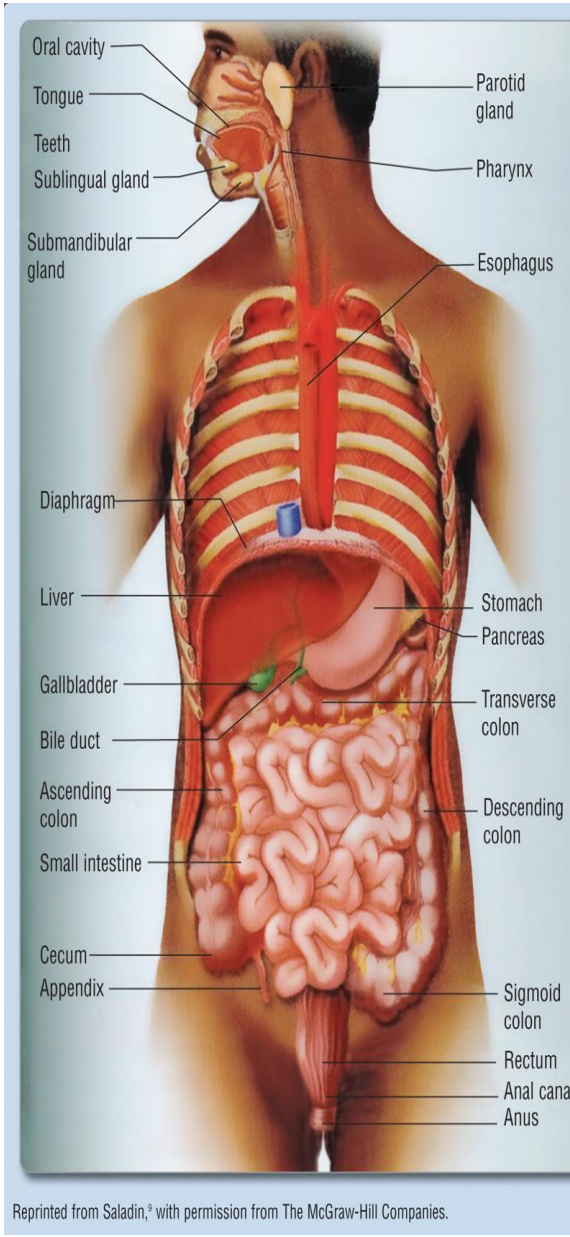
The Abdominal Compartment Society

WSACS

In the ED and ICU

- ACS is recognized with growing frequency as the cause of morbidity such as metabolic acidosis, decreased urine output, and decreased cardiac output. The cause of these events might easily be mistaken for other pathologic events such as ~~hypovolemia~~ if the clinician is not alert to the morbidity associated with abdominal compartment syndrome

- ~~Pharmacologic therapy~~ is less effective than mechanical drainage. Paracentesis may be a superior alternative to decompressive laparotomy



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Pathophysiology

- Organ dysfunction is a product of **the effects of IAH on multiple organ systems.**
- Acs follows a destructive pathway similar to compartment syndrome of the extremity.

- Problems begin at the organ level with direct compression; hollow systems such as the intestinal tract and portal-caval system **collapse under high pressure**.

- Immediate effects such as **thrombosis** or bowel wall **edema** are followed by translocation **of bacterial products**, leading to additional **fluid accumulation**, which further increases intra-abdominal pressure.

- At the cellular level, **oxygen delivery is impaired**, leading to ischemia and **anaerobic metabolism**. **Vasoactive** substances such as histamine and serotonin **increase endothelial permeability;** further **capillary leakage** impairs red cell transport; and ischemia worsens.

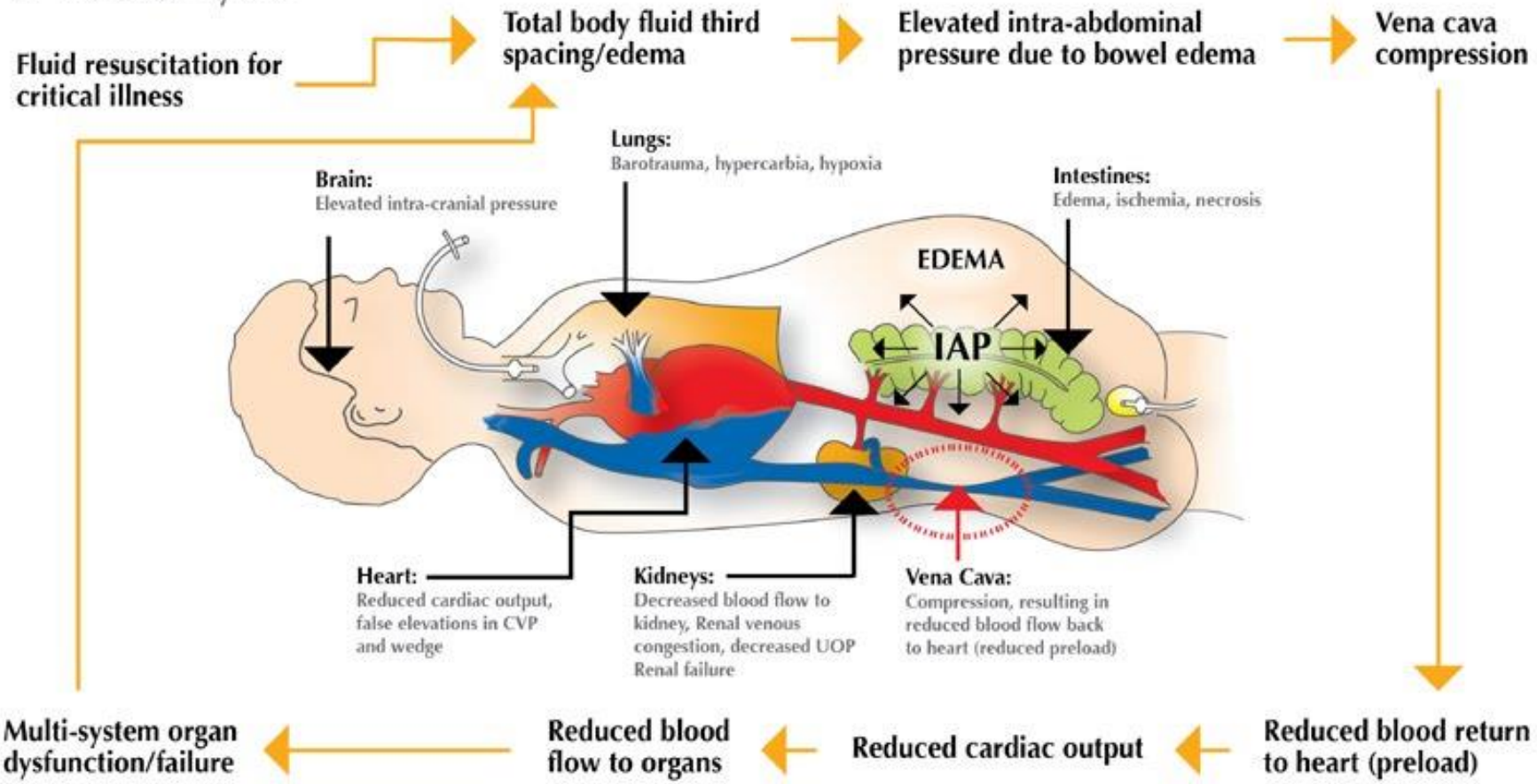
- Although the abdominal cavity (ie, the peritoneal and, to a lesser extent, retroperitoneal cavities) is much more distensible than an extremity, it reaches an endpoint at which the pressure rises dramatically. This is less apparent in chronic cases because the fascia and skin slowly stretch and thus tolerate greater fluid accumulation.

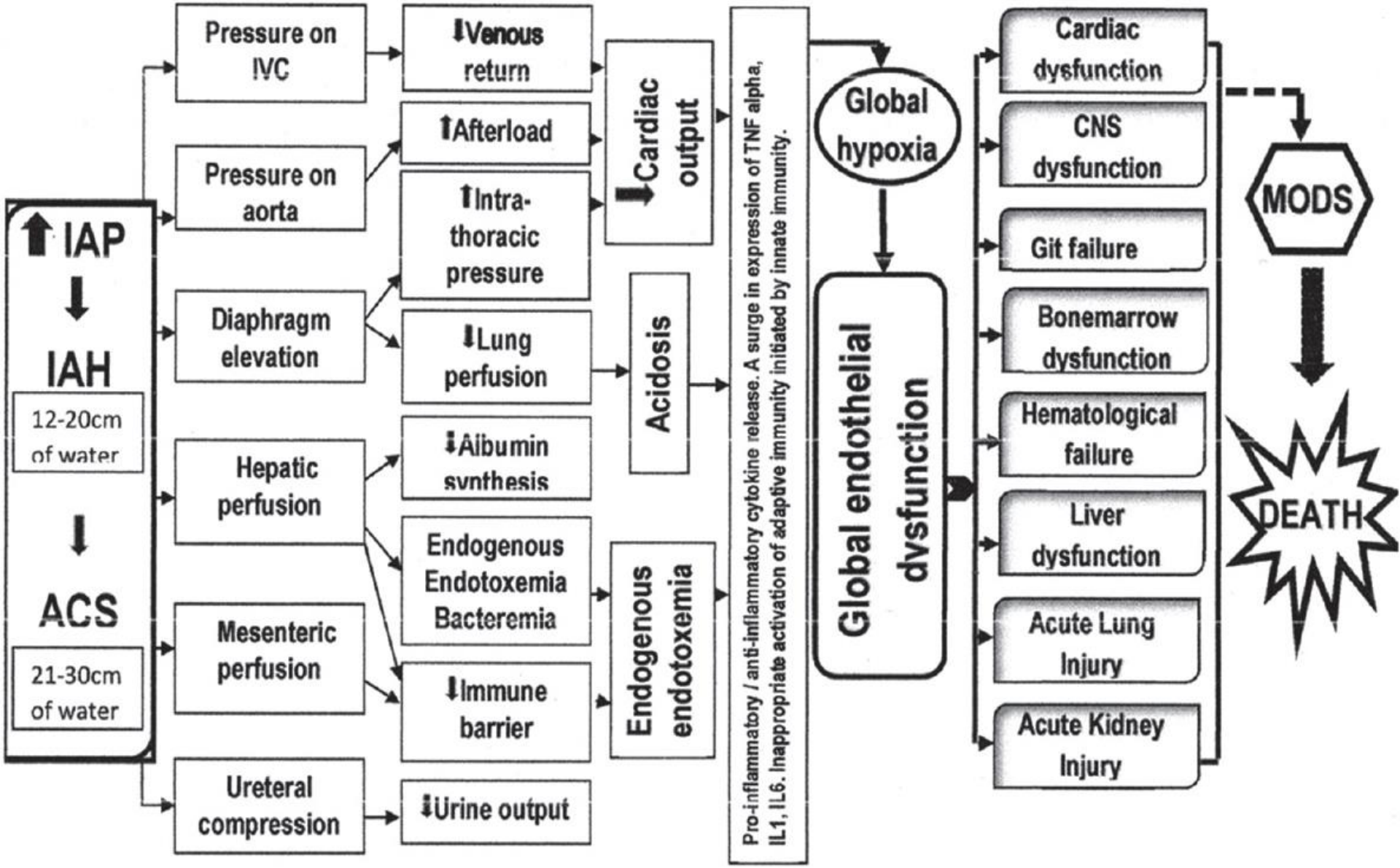
- As pressure rises, abdominal compartment syndrome impairs **not only** visceral organs but also the cardiovascular and the pulmonary systems; it may also cause a decrease in cerebral perfusion pressure.

- Therefore, abdominal compartment syndrome should be recognized as a possible cause of decompensation in any critically injured patient.

What Happens to the Body's Organs?

A Vicious Cycle




⬆IAP
⬇
IAH
12-20cm of water
⬇
ACS
21-30cm of water
Pressure on IVC
Pressure on aorta
Diaphragm elevation
Hepatic perfusion
Mesenteric perfusion
Ureteral compression
⬇Venous return
⬆Afterload
⬆Intra-thoracic pressure
⬇Lung perfusion
⬇Albumin synthesis
Endogenous Endotoxemia Bacteremia
⬇Immune barrier
⬇Urine output
⬇Cardiac output
Acidosis
Endogenous endotoxemia
Pro-inflammatory / anti-inflammatory cytokine release. A surge in expression of TNF alpha, IL1, IL6. Inappropriate activation of adaptive immunity initiated by innate immunity.
Global hypoxia
Global endothelial dysfunction
Cardiac dysfunction
CNS dysfunction
Git failure
Bonemarrow dysfunction
Hematological failure
Liver dysfunction
Acute Lung Injury
Acute Kidney Injury
MODS
DEATH

Etiology sometimes overlapping causes.

Primary ACS

include the following:

- Penetrating trauma
- **Intraperitoneal hemorrhage**
- **Pancreatitis** [10]
- **External compressing forces, such as debris from a motor vehicle collision or after a large structure explosion**
- Pelvic fracture
- **Rupture of abdominal aortic aneurysm** [11]
- **Perforated peptic ulcer**

CrossMark

Abdominal compartment syndrome associated with endovascular and open repair of ruptured abdominal aortic aneurysms

Chen Rubenstein, MD,[a] Gabriel Bietz, MBChB,[b] Daniel L. Davenport, PhD,[c] Michael Winkler, MD,[c] and Eric D. Endean, MD,[d] *Jerusalem, Israel; San Antonio, Tex; and Lexington, Ky*

- In one review and meta-analysis of studies of patients who developed ACS after repair of ruptured abdominal aortic aneurysms, mortality was found to be 47%. Treatment included open decompression in 86 patients; percutaneous drainage in 18 (catheter only in 5; combined with tissue plasminogen activator infusion in 13); and conservative measures in 5.

Etiology sometimes overlapping causes.

Secondary ACS

occur in patients without an intra-abdominal injury, when fluid accumulates in volumes sufficient to cause IAH.

Causes include the following:

- Large-volume resuscitation: The literature shows significantly increased risk with infusions greater than 3 L
- Large areas of full-thickness burns

Hobson et al demonstrated abdominal compartment syndrome within 24 hours in burn patients who had received an average of **237 mL/kg over a 12-hour period**.

- Penetrating or blunt trauma without identifiable injury
- Postoperative
- Packing and primary fascial closure, which increases incidence
- Sepsis

- *A retrospective study reported on risk factors directly associated with mortality in patients with both intra-abdominal hypertension and ACS. Polytransfusion was a strong predictor of mortality, along with a reported history of diabetes and the total amount of blood products used.*

- Secondary ACS in patients with lower extremity vascular injuries from penetrating injury or blunt trauma was associated with a 60% mortality in one study.

Etiology sometimes overlapping causes.

Chronic

Causes of chronic abdominal compartment syndrome include the following:

- Peritoneal dialysis
- Morbid obesity
- Cirrhosis
- Meigs syndrome
- Intra-abdominal mass

Epidemiology

- the frequency of abdominal compartment syndrome in trauma ICU admissions is anywhere from 5-15% and 1% of general trauma admissions.
- in all age groups
- appears to be similar in the pediatric population.

Prognosis

- If left untreated, abdominal compartment syndrome is almost uniformly fatal.(a mortality of 68%)
- Furthermore, abdominal compartment syndrome is often a sequela to severe injuries that independently carry a high morbidity and high mortality

A 5-year retrospective study at 3 level 1 trauma centers in Chicago identified the following prognostic factors in trauma patients with ACS :

- The 30-day mortality was strongly associated with an **initial intra-abdominal** pressure >20 mm Hg and moderately associated with **blunt injury mechanism**.
- **Lactic acid level >5 mmol/L** on admission was moderately associated with increased blood transfusion requirements and with acute renal failure during hospitalization.
- **Developing ACS within 48 hr of admission** was moderately associated with increased length of stay in the ICU, more ventilator days, and longer hospital stay.
- **Initial operative intervention lasting more than 2 hr** was moderately associated with risk of developing multiorgan failure.
- **Hemoglobin level <10 g/dL** on admission, **ongoing mechanical ventilation**, and **ICU stay >7 days** were moderately associated with a disposition to long-term support facility.
-

History

- varies depending on the cause
- pain is commonly present. (precede the development of ACS and may be directly related to a precipitating event)
- Syncope or weakness may be a sign of hypovolemia.
- Difficulty breathing
- decreased urine output
- may be unable to communicate, because they are often intubated and critically ill.

- Increase in abdominal girth
- Difficulty breathing
- Decreased urine output
- Syncope
- Melena
- Nonsteroidal anti-inflammatory drug (NSAID) use
- Alcohol abuse
- Nausea and vomiting
- History of pancreatitis

Abdominal compartment syndrome may be **obscured** in patients with critical injuries.

Consider IAH and document intra-abdominal pressures in any of the following patients:

- **Intubated** patients who have high peak and plateau pressures and are difficult to ventilate
- Patients who have **GI bleeding or pancreatitis** and are not responding to intravenous (IV) fluids, blood products, and pressors
- Patients who have severe **burns or sepsis** with decreasing urine output and are not responding to IV fluids and pressors

Physical Examination

- increased abdominal girth.

(If this change is acute, the abdomen is tense and tender.)

(be easier to visualize with the patient standing or sitting upright.)

- other secondary effects of abdominal compartment syndrome are as follows:

- Wheezes, rales, increased respiratory rate
- Cyanosis

Complications

I. **Renal failure:** This is not prevented by intraureteral stents, which suggests direct compression of renal parenchyma and decreased renal perfusion as causes
II. **Respiratory distress and failure**: Initial signs of abdominal compartment syndrome include elevated peak airway pressures in intubated patients with decreased tidal volumes
III. **Bowel ischemia**
IV. Increased intracranial pressure **(ICP):** Decompressive laparotomy has been shown to reduce intractable elevated ICP in patients with IAH
V. **Failing cardiac output and refractory shock:** Abdominal compartment syndrome factitiously elevates central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) in patients who are hypovolemic or euvolemic

Diagnostic Considerations

-

A. Abdominal Trauma, Blunt
B. Appendicitis, Acute
C. Cholangitis
D. Congestive Heart Failure and Pulmonary Edema
E. Dissection, Aortic
F. Diverticular Disease
G. Foreign Bodies, Gastrointestinal
H. Mesenteric Ischemia
I. Pediatrics, Bacteremia and Sepsis
J. Urinary Obstruction

Laboratory studies and abdominal CT scan are part of the workup

Measure intra-abdominal pressure (IAP) if abdominal compartment syndrome is suspected.

The following lab studies may be indicated:

- ✓ Comprehensive metabolic panel (CMP)
- ✓ Complete blood cell count (CBC)
- ✓ Amylase and lipase assessment
- ✓ Prothrombin time (PT), activated partial thromboplastin time (aPTT) if the patient is heparinized
- ✓ Cardiac marker assays
- ✓ Urinalysis and urine drug screen
- ✓ Measurement of serum lactate levels (at many institutions, the sample must be kept on ice)
- ✓ Arterial blood gas (ABG): This is a quick way to measure the pH, lactate, and base deficit

CT and Other Imaging Studies

Abdominal CT scanning can reveal many subtle findings.

- **Round-belly sign:**

Abdominal distention with an increased ratio of anteroposterior-to-transverse abdominal diameter (ratio >0.80)

- Collapse of the vena cava
- Bowel wall thickening with enhancement
- Bilateral inguinal herniation

Plain abdominal radiographic studies are often ~~useless~~ in identifying abdominal compartment syndrome, although they may show evidence of free air or bowel obstruction. Abdominal ultrasonography may reveal an aortic aneurysm, particularly with large aneurysms, but bowel gas or obesity makes performing the study difficult.

-

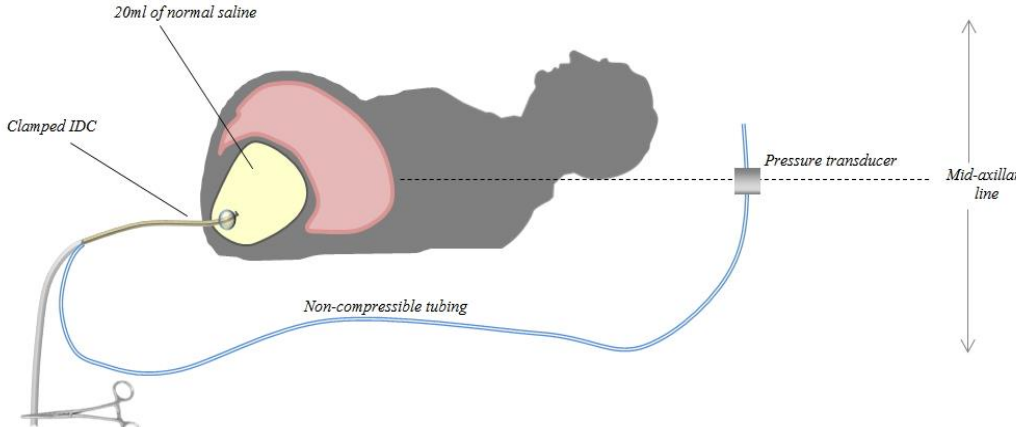
Intra-abdominal Pressure Measurement

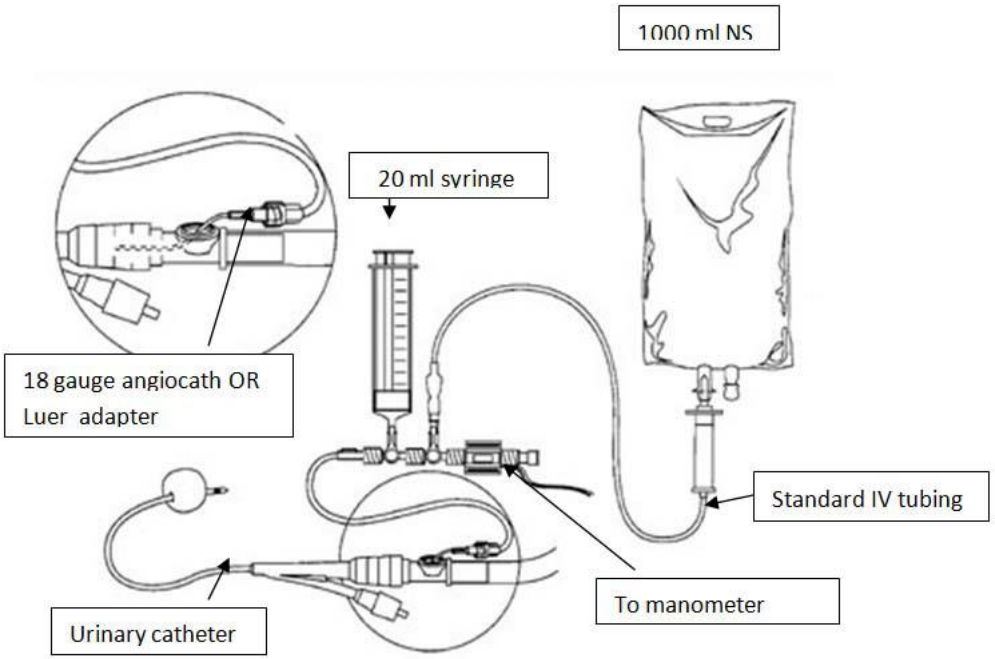
1. IAP can be easily monitored by measuring bladder pressure.

2. consists of instilling about 50 mL of saline into the urinary bladder through the Foley catheter.

3. The tubing of the collecting bag is clamped, and a needle is inserted into the specimen-collecting port of the tubing proximal to the clamp and is attached to a manometer.

4. Bladder pressure (measured in mm Hg) is the height at which the level of the saline column stabilizes with the symphysis pubis as the zero point.





Grading

In an excellent group of articles, Burch et al developed a grading system. [27] Patients with higher-grade abdominal compartment syndrome have end-organ damage, which is evidenced by splenic hypercarbia and elevated lactate levels, even if they appear clinically stable.

The following grading system has become accepted if IAH is present:

- ***Grade I: 10-15 cm H_2O***
- ***Grade II: 15-25 cm H_2O***
- ***Grade III: 25-35 cm H_2O***
- ***Grade IV, greater than 35 cm H_2O***

- ***End-organ damage*** has been observed with IAP as low as 10 cm H_2 O, and multiple studies have found damage at values ranging from 20-40 cm H_2 O. Disparity exists because abdominal compartment syndrome never occurs as an isolated event.

- abdominal perfusion pressure (APP) to be a much better predictor of end-organ injury than lactate, pH, urine output, or base deficit. [25] The APP is equal to the mean arterial pressure minus the IAP.

Approach Considerations

- immediately transport the patient to the emergency department.
- Remove any constricting garments.
- Do not place anything on the patient's abdomen (eg, external defibrillators, bundles of blankets, oxygen tanks).
- Avoid overly aggressive fluid resuscitation, especially in extremity injuries.
- In the emergency department, the first priority of the ED physician is to consider the diagnosis in any patient

- Therapy should include fluid resuscitation and transfusion if needed, as well as surgical consultation.
- early use of an open abdomen has been shown to reduce mortality.

A group in Taiwan has used laparoscopic decompression successfully in blunt abdominal trauma patients who have an IAP of 25-35 cm H_2 O.

- WSACS has noted that that correct fluid therapy and perfusional support during resuscitation form the ***cornerstone*** of medical management in patients with abdominal hypertension.

- Pharmacologic therapy is less effective than mechanical drainage.

- Pressors have a role but may not be equally effective in treating abdominal compartment syndrome.

- Dobutamine was shown to be superior to dopamine in restoring intestinal mucosal perfusion in a porcine model.

- IAH may be an ongoing process in any patient with pathology producing intra-abdominal fluid loss. Repeat or continuous IAP measurement is indicated. The abdomen should be clear of any heavy objects.

Paracentesis

- Multiple reports document the efficacy of paracentesis in burn patients who develop ACS.

- it appears to be a superior alternative to decompressive laparotomy in this patient population.

(It may be performed quickly at bedside and avoids potential complications associated with larger incisions.)

- Paracentesis is also extremely useful in patients with chronic ACS from large-volume ascites.

Reperfusion Syndrome

- Secondary effects of abdominal compartment syndrome occur immediately after evacuation.
- Many cases of hypotension and even asystole have been observed.
- Theories to explain these effects include washout of products of anaerobic metabolism (eg, lactic acid), which may be directly tissue toxic, and suddenly decreased systemic vascular resistance (SVR).

- Volume resuscitation immediately before decompression has been shown to significantly decrease these events.
- Adding mannitol and sodium carbonate ($NaCO_3$) to the IV fluid bolus may decrease the toxicity of reperfusion syndromes.

Further Outpatient Care

- directed at the primary etiology of ACS.

- Chronic ACS requires lifelong medications and lifestyle changes, which may include the following.
  - Diuretics
  - Fluid restriction
  - Weight loss
  - Avoidance of alcohol

Prevention

- much more effective than treating it.

(postsurgical care regarding prevention of abdominal compartment syndrome.

Various types of surgical mesh are helpful to decrease the incidence of abdominal compartment syndrome)

- Prevention is also focused on earlier treatment of IAH. Many authors now recommend managing IAH before full abdominal compartment syndrome develops.
- This can only be accomplished by proactive IAP measurement and monitoring

- Controlled, randomized studies have highlighted the possibility of preventing ACS by avoiding pure crystalloid resuscitation in trauma and burn patients.

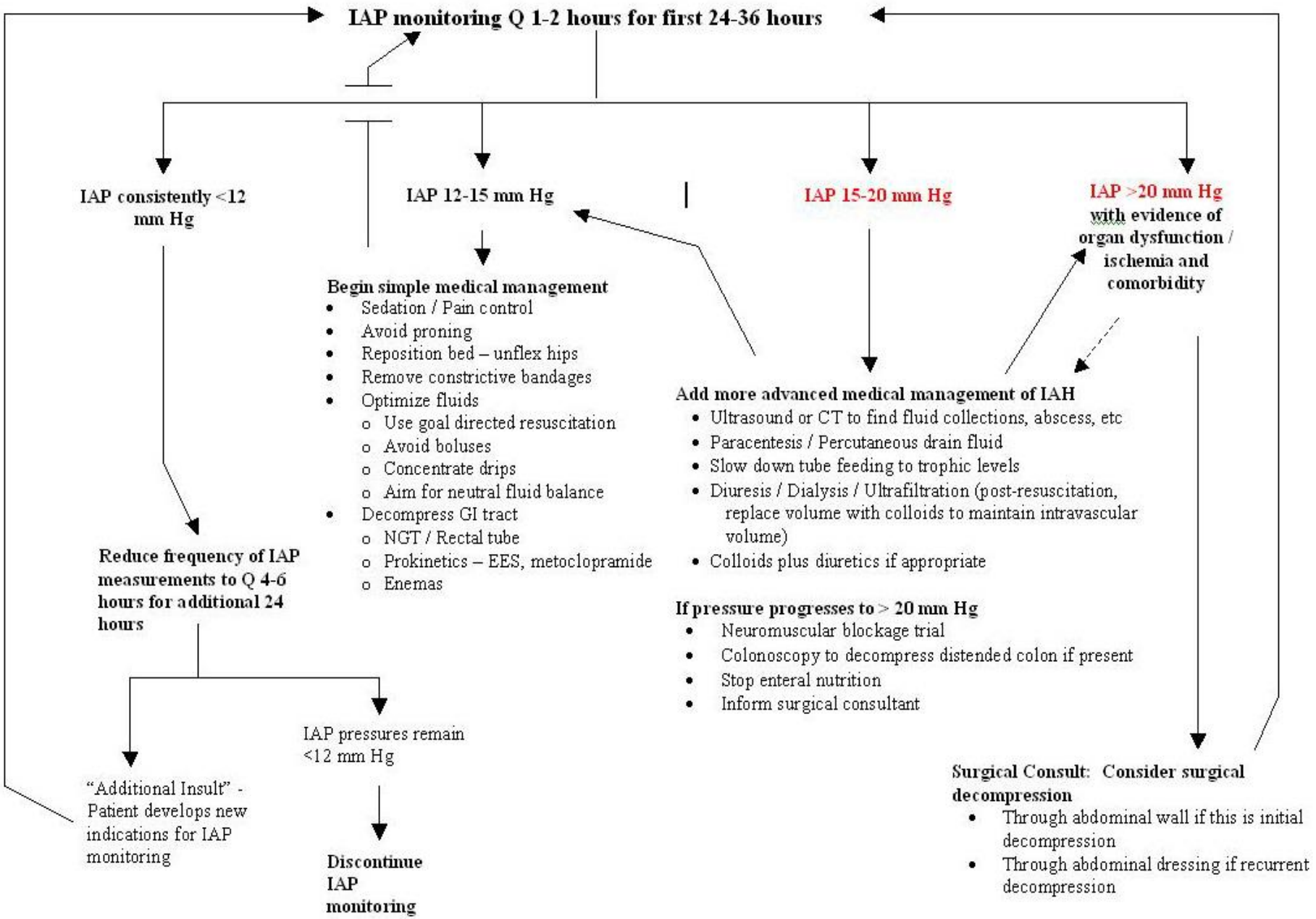
(O'Mara et al demonstrated a significantly lower IAP in burn patients resuscitated with a colloid combination of fresh frozen plasma and lactated Ringer solution versus lactated Ringer solution alone, given by the Parkland formula.)

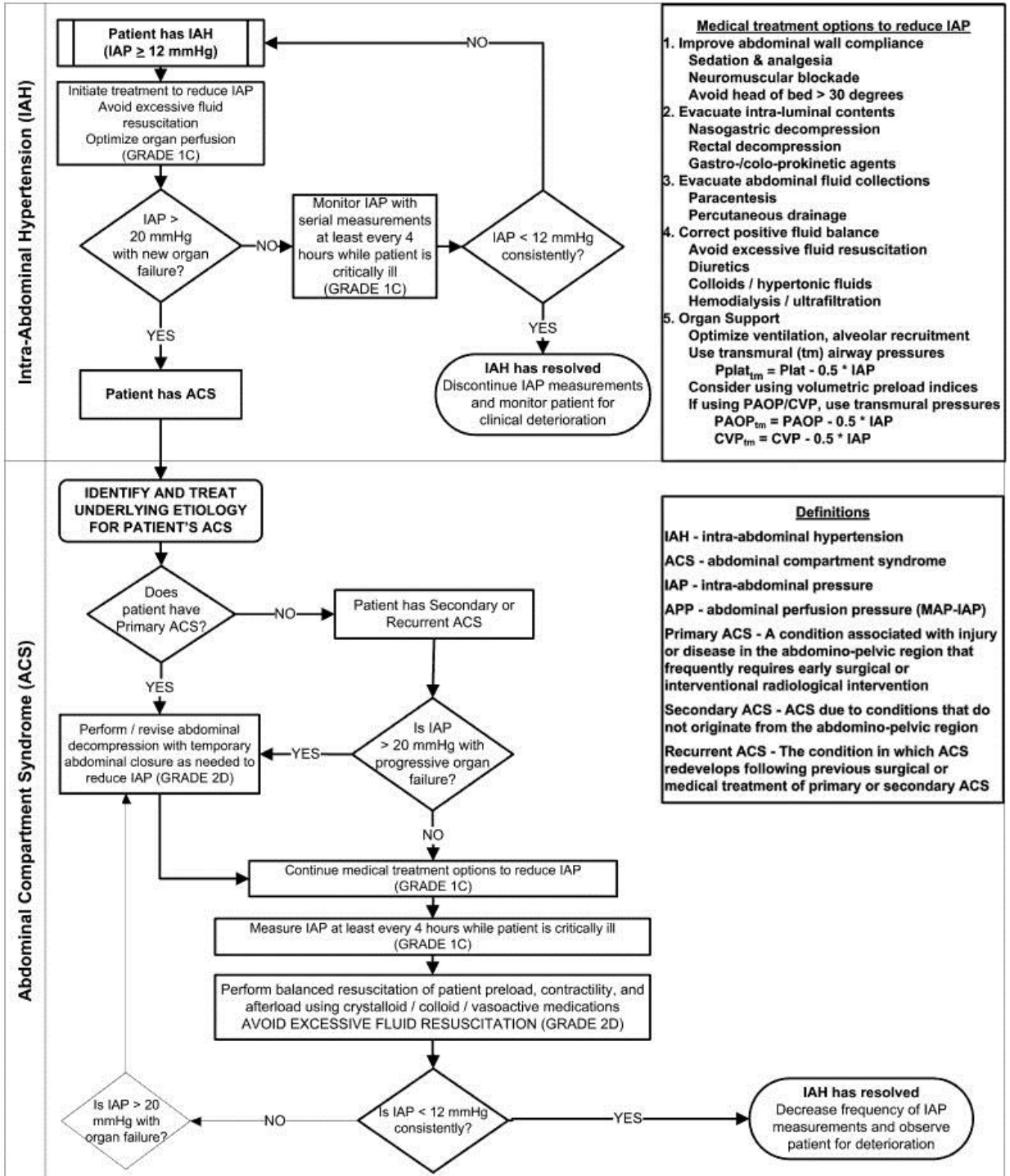
(At a large Japanese burn center, Oda et al demonstrated hypertonic lactated saline could be used in smaller volumes to maintain adequate urine output and significantly reduce the rate of abdominal compartment syndrome and associated morbidity.)

Diuretics

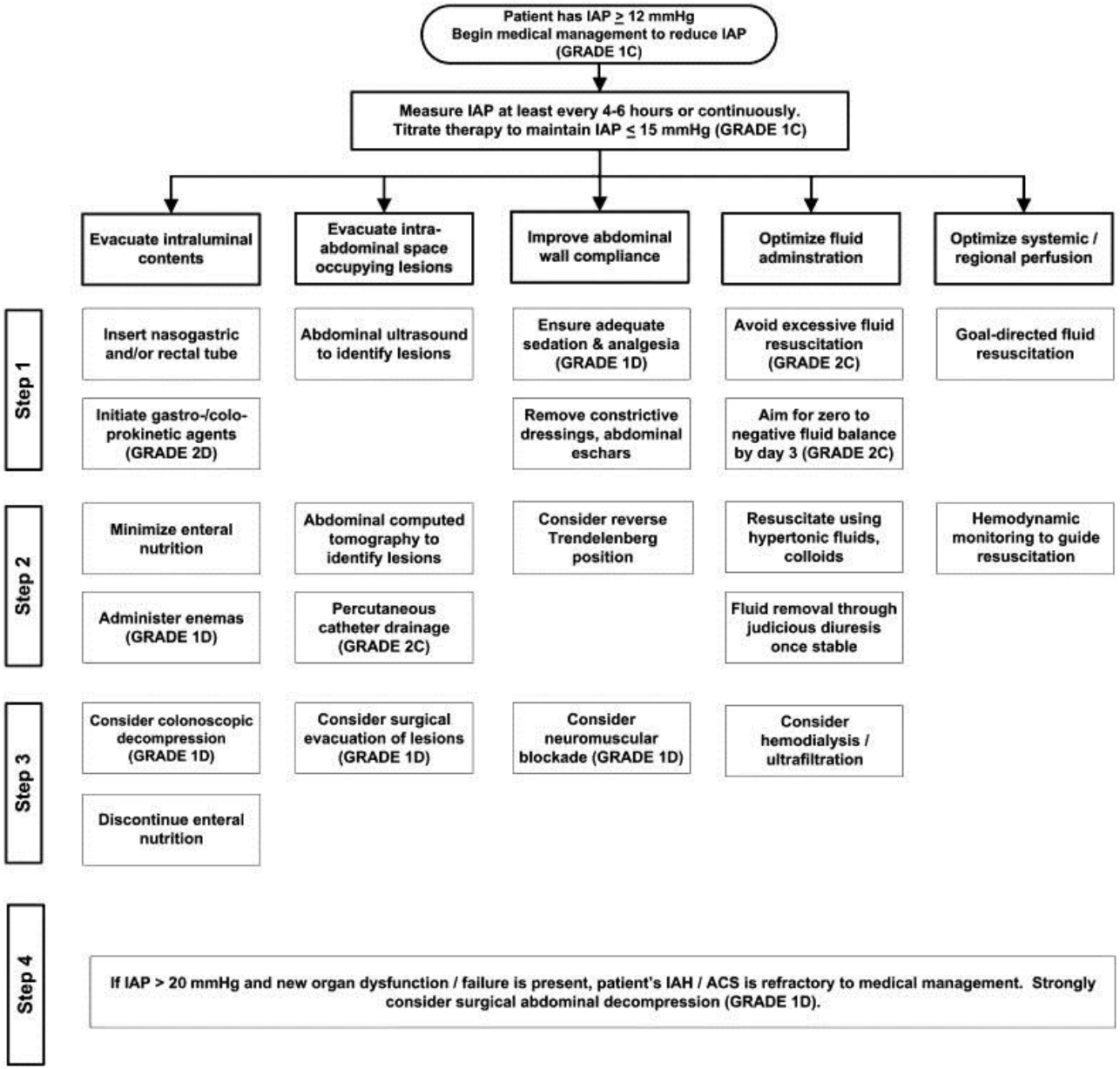
- **Furosemide (Lasix)**
- The dose must be individualized. Depending on response, administer at increments of 20-40 mg no sooner than 6-8 h after previous dose, until desired diuresis occurs.
- When treating infants, titrate with 1-mg/kg/dose increments until satisfactory effect is achieved.
- **Spironolactone (Aldactone)**
- **Amiloride**

Intra-Abdominal Pressure Monitoring Algorithm




Intra-Abdominal Hypertension (IAH)
Patient has IAH (IAP ≥ 12 mmHg)
NO
Initiate treatment to reduce IAP
Avoid excessive fluid resuscitation
Optimize organ perfusion
(GRADE 1C)
IAP > 20 mmHg with new organ failure?
NO
Monitor IAP with serial measurements at least every 4 hours while patient is critically ill (GRADE 1C)
IAP < 12 mmHg consistently?
YES
IAH has resolved
Discontinue IAP measurements and monitor patient for clinical deterioration
YES
Patient has ACS
Medical treatment options to reduce IAP
1. Improve abdominal wall compliance
Sedation & analgesia
Neuromuscular blockade
Avoid head of bed > 30 degrees
2. Evacuate intra-luminal contents
Nasogastric decompression
Rectal decompression
Gastro-/colo-prokinetic agents
3. Evacuate abdominal fluid collections
Paracentesis
Percutaneous drainage
4. Correct positive fluid balance
Avoid excessive fluid resuscitation
Diuretics
Colloids / hypertonic fluids
Hemodialysis / ultrafiltration
5. Organ Support
Optimize ventilation, alveolar recruitment
Use transmural (tm) airway pressures
Pplat$_{tm}$ = Plat - 0.5 * IAP
Consider using volumetric preload indices
If using PAOP/CVP, use transmural pressures
PAOP$_{tm}$ = PAOP - 0.5 * IAP
CVP$_{tm}$ = CVP - 0.5 * IAP
Abdominal Compartment Syndrome (ACS)
IDENTIFY AND TREAT UNDERLYING ETIOLOGY FOR PATIENT'S ACS
Does patient have Primary ACS?
NO
Patient has Secondary or Recurrent ACS
YES
Perform / revise abdominal decompression with temporary abdominal closure as needed to reduce IAP (GRADE 2D)
YES
Is IAP > 20 mmHg with progressive organ failure?
NO
Continue medical treatment options to reduce IAP (GRADE 1C)
Measure IAP at least every 4 hours while patient is critically ill (GRADE 1C)
Perform balanced resuscitation of patient preload, contractility, and afterload using crystalloid / colloid / vasoactive medications
AVOID EXCESSIVE FLUID RESUSCITATION (GRADE 2D)
Is IAP < 12 mmHg consistently?
YES
IAH has resolved
Decrease frequency of IAP measurements and observe patient for deterioration
NO
Is IAP > 20 mmHg with organ failure?
Definitions
IAH - intra-abdominal hypertension
ACS - abdominal compartment syndrome
IAP - intra-abdominal pressure
APP - abdominal perfusion pressure (MAP-IAP)
Primary ACS - A condition associated with injury or disease in the abdomino-pelvic region that frequently requires early surgical or interventional radiological intervention
Secondary ACS - ACS due to conditions that do not originate from the abdomino-pelvic region
Recurrent ACS - The condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS

- *The choice (and success) of the medical management strategies listed below is strongly related to both the etiology of the patient's IAH / ACS and the patient's clinical situation. The appropriateness of each intervention should always be considered prior to implementing these interventions in any individual patient.*
- *The interventions should be applied in a stepwise fashion until the patient's intra-abdominal pressure (IAP) decreases.*
- *If there is no response to a particular intervention, therapy should be escalated to the next step in the algorithm.*



Thank you