

UNDERSEA & HYPERBARIC MEDICAL SOCIETY



# ANNUAL SCIENTIFIC MEETING

# PROGRAM & ABSTRACTS



**HOTEL BONAVENTURE  
MONTREAL, CANADA**

**JUNE 18-20, 2015**



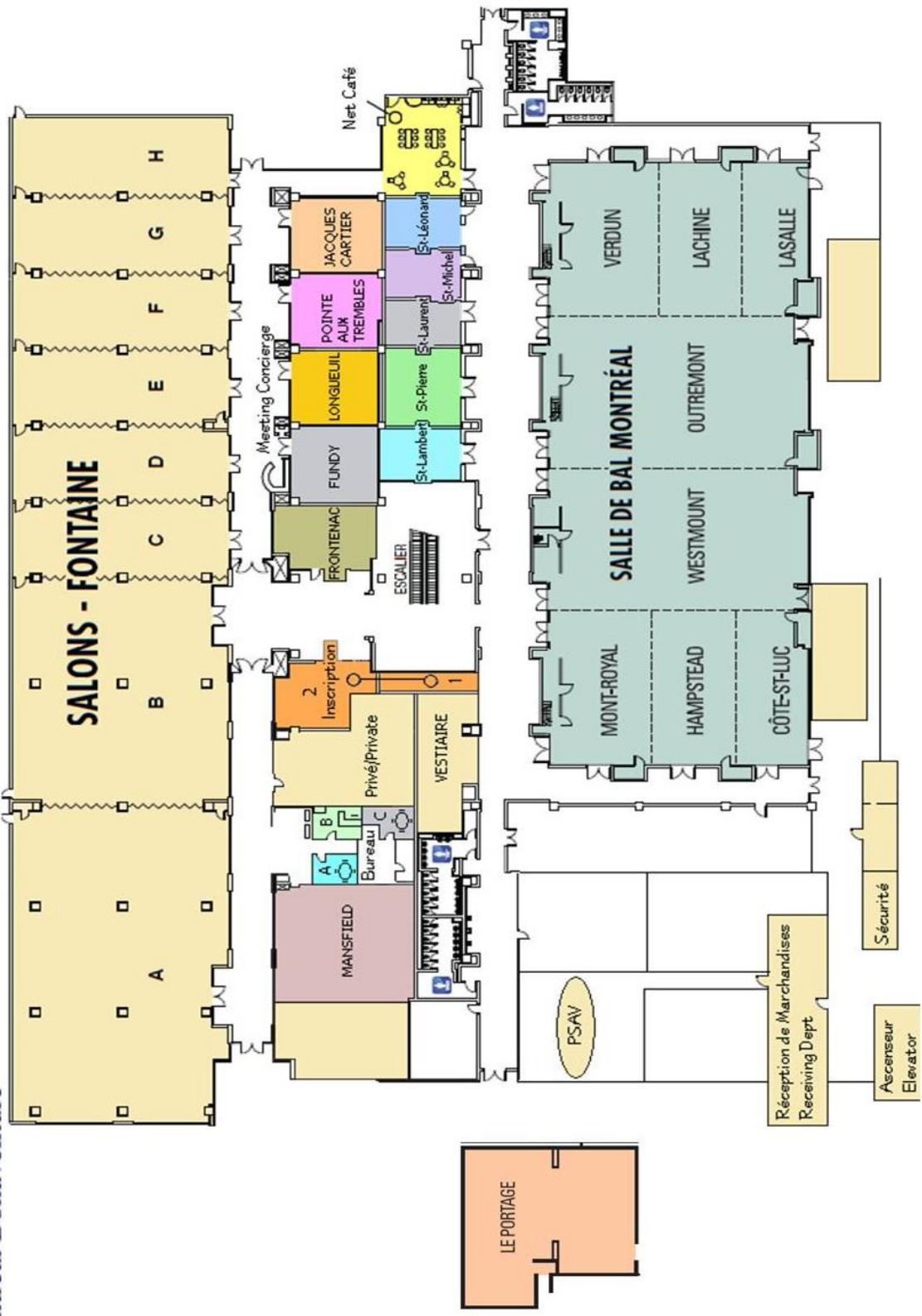


**2015**  
**UHMS Scientific Meeting**

**June 18-20**

**Montreal, Canada**

# Montréal Bonaventure



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Aktas S: D65; Allen BW: B23; Allinson P: C117; Alvarez Villela M: A7, A14, A15, C38, C50, D74, D75; Annis H: F102; Anselem L: F98; Arican N: D60; Arieli Y: A6, B29; Arnell P: E94, F95; Arslan A: D64; Balestra C: D58; Banham N: D58; Bao XC: A11, A12, A18; Barak O: D58; Baynosa R: C33; Beadnell M: C40; Bell JE: E77, E78; Bennett M: D58; Bennett PB: A7, A14, A15; Berry K: C34; Bhopale VB: A1, A2, B25, D58; Biram A: B29; Blake DF: D58; Blatteau JE: A4; Blogg SL: D57, D75; Boschuett T: F102; Bouak F: A4; Buckley J: F104; Buzzacott P: D73; Cable RA: E83, C35; Canela J: C98; Castillo E: D68, E81; Chan E: D55; Chang M: F101; Chang SC: D62; Chang SY: D62; Chen H: A12; Chen S: F110; Chng J: D63; Chopra A: B22; Churchill S: C37, E82, E83; Cialoni D: D58; Clarke NW: D56; Como N: C40; Cooper JS: C40C C117; Cormier JE: E93; Craig AB: F100; Crawford CC: C34; Daniller A: C49; Davis J: C37; Dayya D: D69; Demchenko IT: B23; Denoble PJ: D73; Derrick BJ: A7, A14, A15; Deru K: C35, C37, C38, C39, E77, E78, E82, E83; Desola J: F98; Dhar R: D74; Dituri J: F102; Dodson WW: B31, B32; Duchnik J: E81; Dujic Z: D58; Edney JJE: A13; Eftedal I: D76; Enomoto M: B21, B28; Erelel M: D61; Eriksson K: B24; Evangelista JS: E92; Fang S: D54, D66, D72; Fang XH: C33; Fang YQ: A11, A12, A18; Fowler K: C117; Freiburger JJ: A7, A14, A15, D75; Gasier HG: B23; Gavis M: B29; Gempp E: A4; Gendron-Fitzpatrick AP: A3; Gennser M: D57; Giardina C: B22, B26, B30; Gomez M: F98; Gorenstein S: C44, E88; Grinblat JY: F110; Grönlund MA: E94; Grover I: D68, E86; Hada EA: A19, A20; Hangan D: C44; Hangan D: E88; Hansen M: F102; Harch P: E91, F113; Hardy KR: D58; Hardy S: E91, F113; Hare MA: E92; Harlan NP: A7, A14, A15, C46, D75; Hendriksen S: E87, F109; Heravi MK: D59; Heyboer M: C43, D69, E79, E80, F106, F107, F108; High A: C40; Hightower LE: B22, B26, B30; Ho AM: C44; Horie M: B21, B28; Hostler D: D51, D52; Howle LE: A19, A20; Huang C: D55; Huang E: F101; Huang G: B27; Huang KL: A10, D62; Huchim O: D54, D55, D66, D72, E86; Hugon J: A4; Hussain SM: B31; Isbister C: C34; Jacoby I: D67; Jennings MS: E80, F106, F107, F108; Jennings S: C43; Jensen AJ: E85; Johnson G: F112; Joo E: D54; Jun Ma J: A11; Jurd KM: D57; Katz AE: C44; Kawashima M: F99, F105; Kawashima M: F99, F105; Keim LW: C 40, C117; Kernagis DN: D56; Kharlamova A: C36, D59; Kimmel E: A6; Kirsten D: F102; Koca E: D60; Kochunov P: A1, A114, A115; Koumandakis G: E77, 378; La SS: C48; Lambert D: C41, D58; Larsson AC: B24; Le J: D58; Le PNJ: A17, C49, D71, F100, F100; Lee CH: F97; Lee MS: B32; LeGros T: E91, F113; Lehner CE: A3; Lewandowski A: C36; Lim M: D63; Lindholm P: B24; Logue C: D58, E87, F109; Loveman GAM: A13; Lycke H: F95; Ma J: A12, A18; Ma MZ: B25; Mackey MN: D56; Madden D: D58; Manfred C: F104; Mao L: B25; Marroni A: D58; Martina SD: A7, A14, A15, D53, D56; Masters T: E87, F109; Matos P: F98; McBride L: F112; McGuire S: A1, A114, A115; McIntosh J: C39; Medak AJ: E92; Menajem D: A6; Merrow M: E88; Michetti Y: C41; Miller RS: E82; Miller SS: A16, A17, C48, C49, D71, E90, F100, F111; Millman MP: C47; Mirasoglu B: D64; Miyata K: F99, F105; Møllerlökken A: D57, D76; Moon H: C48, F100, F111; Moon RE: A7, A14, A15, D53, D75; Morgan A: D68; Morgan M: C43, E80, F106, F107, F108; Mueller B: D75; Mullokanov M: B29; Murphy FG: A19, A20; Murphy-Lavoie H: E91, F113; Natoli MJ: A7, A14, A15; Ng LJ: D64; Nihlén C: B24; Ninokawa S: D55; Nishi R: A4; Ofir D: A6; O'Hara RB: B31; O'Neill OJ: D69; Orrison WW: C39; Oscarsson N: F95; Otteni CE: D53; Owen E: D68; Oyaizu T: B21, B28; Oyama L: D67; Ozcelik C: D61; Papoutsidakis E: F98; Parikh M: A7, A14, A15, C38, C50, D75; Peacher DF: D53; Pearson M: E91, F113; Pendergast DR: D51, D512; Peng CK: A10, D62; Penny CEL: D70; Perdrizet G: B22, B26, B30; Piantadosi CA: B23; Pieri M: D58; Pollock NW: D56, D58; Ponce JP: F100; Popa D: D66, D72, E86; Potter JF: D53; Proano J: F101; Ptak J: F104; Pullis M: E84; Qingle L: E89; Raizada H: C36; Ranjbar NA: D59; Reetz S: F104; Regan K: C44, E88; Reiger R: C40; Risberg J: D76; Roberts AB: A7, A14, A15, C45, D75; Robinson T: E81; Robinson TD: F103; Rosén A: D58, F95; Rosenthal R: C116; Rosielle K: C42; Sabbisetti V: B22; Sadler C: D67, D68; Salehi HA: D59; Sam B: D60; Santiago W: C43, E79, E80, F106, F107, F108; Savaser D: D67, E81; Scahlberg P: C117; Schellart NA: A8; Schiffer T: B24; Schinazi EA: A7, A14, A15; Searing E: C36; Sethuraman K: C116; Sharma D: C43; Shelley DA: D64; Shen CH: D62; Sherman P: A1, A114, A115; Silver A: C33; Simon O: F101; Sippel J: C117; Sivrikaya H: D61; Smerdon GR: D70; Smith G: E79; Smith R: C43; Smyth JA: B22; Sobakin AS: A3; Sprau S: D54, D72; St Leger Dowse M: D70; Stephenson L: C33; Stomberg MW: E94; Strauss MB: A16, A17, C48, C49, D71, E90, F100, F110, F111; Swaby JA: D69,

D80, F106, F107, F108; Sward D: C116, D58; Takao K: F99, F105; Tamura H: F99, F105; Tan AM: F111; Tang KC: D63; Tec J: D72, E86; Teo L: C34; Tettelbach WH: C50; Thom SR: A1, A2, A115, B25, D58; Tian H: C33; Toklu AS: D60, D61, D64; Travis P: C40; Trout BM: D73; Usera PC: C33; Uusijärvi J: B24; Van Meter K: E91, F113; Verma R: B22; Virgilio G: D68; Wakefield M: F112; Walker A: A7, A14, A15; Walter J: E87, F96, F109; Wang FF: A11, A12, A18; Wang HT: A18; Wang WZ: C33; Wannholt R: E94; Weaver LK: C35, C36, C37, C38, C39, C45, C46, D74, E77, E78, E82, E83; Weber R: C33; Weigang Xu: B27; Weitzberg E: B24; Wester TE: D53; Westgard B: E87, F96, F109; Whelan H: F102; Wiley JM: D56; Williams S: C33; Wilson G: E83; Wilson MA: A3; Winn D: C117; Witucki P: D68, E81; Wojcik S: C43, E79; Wolf EG: B32; Won L: D72; Wright BA: B31; Wu CP: A10; Wudtke R: E84; Wyatt H: E91, F113; Xu W: A5; Yagishita K: B21, B28; Yamaguchi T: F99, F105; Yang M: B25; Yang E: C34; Yang M: A1, A2, D58; Yang W: C33; Yeast C: F112; Yontz D: E91, F113; You P: A11, A12, A18; Yuan HR: A11, A12, A18; Yuan J: A9; Zamboni WA: C33; Zetterberg M: E94; Zhang F: C33; Zhang K: A5, B27; Zhang S: A11, A18

**The following presenters have the following to disclose:**

Name of Individual	Individuals Role in Activity	Name of Commercial Interest (If Applicable)	Nature of Relationship
Chin W	D54, D55, D58, D66, D72, E86, F101	OxyHeal Health Group	Employer
Perdrizet G	B22, B26, B30	OxyHeal Health Group	Provided experimental hyperbaric chamber and funding for reagents

**Commercial Support:** The following have provided commercial support to this activity:

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<input checked="" type="checkbox"/>	No relevant relationship(s) to resolve		Provided talking points/outline
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Date: 6/9/2015

THERE WERE NO RELEVANT RELATIONSHIP(S) TO RESOLVE



UHMS CME Coordinator

**Overall Goal of the UHMS Annual Scientific Meeting**

The primary goal of the Undersea and Hyperbaric Medical Society ASM is to provide a forum for professional scientific growth and development to the participants. The meeting provides a basis for exchange of ideas, both scientific and practical, among physicians, researchers, and other health professionals. It affords an opportunity for participants to meet and interact with past and present leaders of the Society, and to become active in societal affairs.

# 2015 UHMS ANNUAL SCIENTIFIC MEETING

## SCHEDULE (O: Oral Presentation; P: Poster Presentation)

### WEDNESDAY, JUNE 17

HOURS

6:00 AM - 5:00 PM	Registration		
7:00 AM - 3:00 PM	Exhibit Company Set-up	Fontaine Salons A-B	
7:00 AM - 3:00 PM	Exhibit Company Poster Set-up	Fontaine Salons C-H	
3:00 PM - 6:00 PM	Exhibitors Move-in	Fontaine Salons A-B	
3:00 PM - 6:00 PM	Poster Move-in	Fontaine Salons C-H	
<b>PRE-COURSES</b>			
7:00 AM - 8:00 AM	Continental Breakfast for Pre-Course Attendees		
8:00 AM - 5:30 PM	<b>PRE-COURSE: PATENT FORAMEN OVALE AND FITNESS TO DIVING CONSENSUS WORKSHOP</b>	Verdun/Lachine Room	7
8:00 AM - 6:00 PM	<b>PRE-COURSE: HYPERBARIC OXYGEN SAFETY: CLINICAL AND TECHNICAL ISSUES</b>	Mt. Royal/Hampstead Room	7
8:00 AM - 5:00 PM	<b>PRE-COURSE: HOW TO PREPARE FOR ACCREDITATION</b>	Cote St. Luc Room	7
10:00 AM - 10:30 AM	AM Break for Pre-Course Attendees		
3:30 PM - 4:00 PM	PM Break for Pre-Course Attendees		
7:00 PM - 10:00 PM	Welcome Reception to Montreal & the UHMS Annual Scientific Meeting	Castillon & Terrace	

### THURSDAY, JUNE 18

7.5

6:00 AM - 5:00 PM	Registration		
7:00 AM - 8:00 AM	Continental Breakfast / Exhibits	Fontaine Salons A-B	
8:30 AM - 5:30 PM	<b>ASSOCIATE BREAKOUT</b>	Verdun Room	
12:00 PM - 1:15 PM	Associate Business Luncheon (50)		
8:00 AM - 5:45 PM	<b>GENERAL SESSION</b>	Salle De Bal Montreal	
8:00 AM - 8:30 AM	<b>PRESIDENT'S ADDRESS</b>	James Holm, MD	0.5
8:30 AM - 9:30 AM	<b>PLENARY SESSION: "MANAGING DECOMPRESSION STRESS: BEYOND THE ALGORITHM"</b>	Neal Pollock, PhD	1
9:30 AM - 10:00 AM	AM Break	Fontaine Salons A-B	
10 AM - 11:30 AM	<b>SESSION A: DIVING/DECOMPRESSION ILLNESS: THEORY &amp; MECHANISMS</b> <b>MODERATORS: Folke Lind, MD &amp; Neal Pollock, PhD</b>		1.5
O: 1000 - 1012 P: 1130 - 1200	A1	Microparticle, Neutrophil And Platelet Changes Associated With Hypobaric And Hypoxic Exposures	Thom SR, Yang M, Bhopale VB, Sherman P, Kochunov P, Mcguire S
O: 1012 - 1024 P: 1130 - 1200	A2	Ascorbic Acid Abrogates Microparticle Generation And Vascular Injuries Associated With High Pressure Exposure	Yang M, Bhopale VB, Thom SR
O: 1024 - 1036 P: 1130 - 1200	A3	Prompt Recompression Treatment For Limb Bends Limits The Induction Of Dysbaric Osteonecrosis In The Uw Sheep Model	Sobakin AS, Wilson MA, Gendron-Fitzpatrick AP, Lehner CE
O: 1036 - 1048 P: 1130 - 1200	A4	A Stress Index To Enhance DCS Risk Assessment For Both Air And Mixed Gas Exposures	Hugon J, Nishi R, Bouakc F, Blatteaud JE, Gemppe E
O: 1048 - 1100 P: 1130 - 1200	A5	Bubbles Cause Endothelial Damage In A Positive Correlation Manner	Zhang K, Xu W
O: 1100 - 1112 P: 1130 - 1200	A6	An Electron Microscope Study Of The Effects Of Decompression On The Spinal Cord And Hippocampus In The Rat: Preliminary Results.	Ofir D, Kimmel E, Menajem D, Arieli Y
O: 1112 - 1124 P: 1130 - 1200	A7	Hypercapnea: Cognitive Effects And Monitoring - Assessment Of Compressed Gas Narcosis Using Nasa's Matb-Ii Flight Simulator	Freiberger JJ, Derrick BJ, Natoli JJ, Schinazi EA, Walker A, Martina SD, Parikh M, Harlan NP, Alvarez MA, Roberts AB, Moon RE, Bennett PB
11:30 AM - 12:00 PM	<b>SESSION A: POSTERS</b>	Fontaine Salons C-H	0.5
O: P: 1130 - 1200	A8	Do Lipids Or Proteins In Plasma Reduce Bubble Surface Tension? Interrelationships Between Plasma Chemicals, Surface Tension And Post-Dive Venous Gas Embolism.	Schellart NA



O: P: 1130 - 1200	A9	The Etiology, Target Organ And Pathogenesis Of Decompression Disease	Yuan J, Lei C, Ji Z
O: P: 1130 - 1200	A10	Brief Hypercapnic Breathing Attenuates Lung Injury Induced By Rapid Decompression	Huang KL, Peng CK, Wu CP
O: P: 1130 - 1200	A11	The Therapeutic Effect Of Relatively Low Pressure Hyperbaric Oxygen For Decompression Sickness Induced By Fast Buoyancy Ascent Escape	Fang YQ, You P, Ma J, Bao XC, Yuan RH, Zhang S, Wang FF
O: P: 1130 - 1200	A12	Clopidogrel Reduces The Inflammatory Response Of Lung In A Rat Model Of Decompression Sickness	Bao XC, Chen H, Fang YQ, Yuan HR, You P, Ma J, Wang FF
O: P: 1130 - 1200	A13	Development Of A Quasi-Physiological Model For The Prediction Of Signs/Symptoms Of Decompression Sickness Following Submarine Tower Escape	Edney JJE, Loveman GAM
O: P: 1130 - 1200	A14	Cognitive Impairment Associated With An Inspired Partial Pressure Of Oxygen Of 1.2 Ata (124kpa) Versus 0.21ata (21kpa) At A Constant Nitrogen Partial Pressure Of 4.5 Ata, (456 Kpa)	Freiberger JJ, Derrick BJ, Natoli MJ, Schinazi EA, Walker A, Martina SD, Parikh M, Harlan NP, Alvarez MA, Roberts AB, Moon RE, Bennett PB
O: P: 1130 - 1200	A15	The Enhancement Of Cognitive Performance Associated With An Inspired Partial Pressure Of Oxygen Of 0.925 To 1.0ata Ata (93.7kpa To 101.3kpa) Versus 0.21ata (21kpa) During Normobaric Trials	Freiberger JJ, Derrick BJ, Natoli MJ, Schinazi EA, Walker A, Martina SD, Parikh M, Harlan NP, Alvarez MA, Roberts AB, Moon RE, Bennett PB
O: P: 1130 - 1200	A16	Reconciliation Of The Wienke Rgbm & The Strauss Gp Models For DCS	Strauss MB, Miller SS
O: P: 1130 - 1200	A17	Disordered Decompression And Undeserved Decompression Sickness	Strauss MB, Miller SS, Le PNJ
O: P: 1130 - 1200	A18	Pdte Ameliorates The Decompression Induced-Lung Injury Caused By Fast Buoyancy Ascent Escape Via Inhibition Of Nf-Kb Pathway	Fang YQ, Wang HT, You P, Zhang S, Ma J, Bao XC, Wang FF, Yuan HR
O: P: 1130 - 1200	A19	The Case For Mixed Pharmacokinetic Models As A Descriptor Of Decompression Sickness	Murphy FG, Hada EA, Howie LE
O: P: 1130 - 1200	A20	Delay Differential Equations As An Explicit Method Of Aligning DCS Model Prediction With Dive Trial Outcome	Hada EA, Murphy FG, Howie LE
O: P: 1130 - 1200	A114	Structural Magnetic Resonance Imaging Change Associated With Repetitive Hypobaric Non-Hypoxic Exposure	Mcguire S, Sherman P, Kochunov P
O: P: 1130 - 1200	A115	Transient Magnetic Resonance Imaging Change Associated With A Single Episode Of Hypobaric Non-Hypoxic Exposure – Preliminary Results	Mcguire S, Sherman P, Thom S, Kochunov P
12:00 PM - 1:15 PM	LUNCH - ON OWN		
1:15 PM - 2:15 PM	<b>LAMBERTSEN LECTURE: "LUNG INJURY WITH DIVING: BEYOND BOYLES LAW"</b>		Alfred Bove, MD 1
2:15 PM - 3:45 PM	<b>SESSION B: HBO<sub>2</sub> THERAPY MECHANISMS</b> <b>MODERATORS: Mike Bennett, MD &amp; John Feldmeier, DO</b>		1.5
O: 1415 - 1427 P: 1615 - 1645	B21	Effects Of Hyperbaric Oxygen Treatment On Muscle Disuse Atrophy.	Horie M, Enomoto M, Oyaizu T, Yagishita K
O: 1427 - 1439 P: 1615 - 1645	B22	HBOT Suppresses Renal Injury In A Rodent Model Of Diabetes Mellitus	Perdrizet G, Verma R, Chopra A, Giardina C, Sabbisetti V, Smyth JA, Hightower LE
O: 1439 - 1451 P: 1615 - 1645	B23	Critical Events In CNS O <sub>2</sub> Toxicity And Novel Approaches For Delaying Oxygen Seizures	Demchenko IT, Gasier HG, Allen BW, Piantadosi CA
O: 1451 - 1503 P: 1615 - 1645	B24	Effects Of Hyperbaric Oxygen On Nitric Oxide Generation In Humans	Uusjäärvi J, Eriksson K, Larsson AC, Nihlén C, Schiffer T, Lindholm P, Weitzberg E
O: 1503 - 1515 P: 1615 - 1645	B25	Inert Gas Chemotherapy	Thom SR, Ma MZ, Bhopale V, Yang M, Mao L
O: 1515 - 1527 P: 1615 - 1645	B26	Pre-Operative Stress Conditioning: Is Oxygen The Drug Of Choice?	Perdrizet G, Giradina C, Hightower L
O: 1527 - 1539 P: 1615 - 1645	B27	Signalling Pathways In Hyperbaric Oxygen-Induced Hsp32 Expression In Primary Cultured Rat Spinal Neurons	Huang G, Zhang K, Xu W
3:45 PM - 4:15 PM	PM Break		<b>Fontaine Salons A-B</b>
4:15 PM - 4:45 PM	<b>SESSION B: POSTERS</b>		<b>Fontaine Salons C-H</b> 0.5
O: P: 1615 - 1645	B28	Acceleration Of Muscle Volume Reduction And Recovery From Hypoxia Of Injured Skeletal Muscle By Hyperbaric Oxygen.	Oyaizu T, Enomoto M, Horie M, Yagishita K
O: P: 1615 - 1645	B29	Mitochondrial Function Following HBO Preconditioning	Mullokanov M, Biram A, Gavis M, Arieli Y
O: P: 1615 - 1645	B30	HBOT Protects Skin From Uv-A Damage In A Hairless Mouse Model	Perdrizet G, Giradina C, Hightower L
O: P: 1615 - 1645	B31	Research Collaboration: India - United States	Dodson WW, Hussain SM, O'hara RB, Wright BA
O: P: 1615 - 1645	B32	Hyperbaric Oxygen Treatment For Acute Traumatic Brain Injury – Research Potential	Lee MS, Dodson WW, Wolf EG
4:45 PM - 5:45 PM	<b>PLENARY SESSION: "ANGIOGENESIS AND HYPERBARIC MEDICINE"</b>		William Li, MD 1
5:45 PM - 6:45 PM	<b>Meet the Exhibitors "Wine &amp; Cheese Reception"</b>		<b>Fontaine Salons A-B</b>

6:00 AM - 5:00 PM	Registration		
7:00 AM - 8:00 AM	Continental Breakfast		Fontaine Salons A-B
8:00 AM - 6:00 PM	GENERAL SESSION		Salle De Bal Montreal
8:00 AM - 9:00 AM	PLENARY SESSION: "HYPERBARIC OXYGEN AND THE CANCER PATIENT: ARE YOU CONCERNED?"		John Feldmeier, DO 1
9:00 AM - 10:30 AM	SESSION C: CLINICAL HBO <sub>2</sub> THERAPY (#1) MODERATORS: Brett Hart, MD & Kristie Coleman		1.5
O: 0912 - 0924 P: 1100 - 1130	C34	Is Hyperbaric Oxygen Therapy Effective For Traumatic Brain Injury? A Rapid Evidence Assessment Of The Literature And Recommendations For The Field	Crawford CC, Teo L, Yang E, Isbister C, Berry K
O: 0924 - 0936 P: 1100 - 1130	C35	A Hospital Network-Wide Analysis Of Emergency Department Referrals For Carbon Monoxide Poisoning To Hyperbaric Medicine Services	Cable RA, Weaver LK, Deru K
O: 0936 - 0948 P: 1100 - 1130	C36	Baseline Vestibular And Audiology Findings In The Brain Injury And Mechanisms Of Action Of Hyperbaric Oxygen (HBO <sub>2</sub> ) For Persistent Post-Concussive Symptoms After Mild Traumatic Brain Injury (Mtb) Study (Bima)	Kharlamova A, Searing E, Weaver LK, Raizada H, Lewandowski A
O: 0948 - 1000 P: 1100 - 1130	C37	Brain Injury Symptoms (Sci-90-R) In A Prospective Cohort Up To 1 Year Following Carbon Monoxide Poisoning	Weaver LK, Churchill S, Deru K, Davis J
O: 1000 - 1012 P: 1100 - 1130	C38	Cardiac MRI Findings In Patients With Carbon Monoxide Poisoning	Alvarez Villela M, Parikh M, Weaver LK, Deru K
O: 1012 - 1024 P: 1100 - 1130	C39	Brain Imaging Abnormalities In Carbon Monoxide-Poisoned Patients With Ongoing Symptoms At Least 6 Months After Poisoning	Weaver LK, Orrison WW, Deru K, Mcintosh J
O: 1024 - 1036 P: 1100 - 1130	F98	Hyperbaric Oxygenation In The Treatment Of Acute Central Retinal Artery Occlusions. An Analysis Of 214 Cases Following A Prospective Protocol.	Desola J, Papoutsidakis E, Matos P, Gomez M, Anselem L, Canela J
10:30 AM - 11:00 AM	AM Break		Fontaine Salons A-B
11:00 AM - 11:30 AM	SESSION C: POSTERS		Fontaine Salons C-H 0.5
O: P: 1100 - 1130	C40	Treatment Of Carbon Monoxide Intoxication/Encephalopathy With Hyperbaric Oxygen Therapy - 26 Years' Experience 1986-2013 - Retrospective Review Of An Alternative Treatment Protocol	Keim LW, Cooper J, High A, Travis P, Beadnell M, Como N, Reiger R
O: P: 1100 - 1130	C41	Waterpipes: A Misconception	Michetti Y, Lambert D
O: P: 1100 - 1130	C42	Long-Term Patient Reported Outcome Of Hyperbaric Oxygen Therapy For Haemorrhagic Radiation Cystitis	Rosielle K
O: P: 1100 - 1130	C43	Carbon Monoxide Exposure And Timely Hyperbaric Oxygen Therapy	Sharma D, Wojcik S, Smith R, Morgan M, Santiago W, Jennings S, Heyboer M
O: P: 1100 - 1130	C44	Patient Outcomes Of Hyperbaric Oxygen Therapy For Radiation Cystitis	Ho AM, Regan K, Hangan D, Gorenstein S, Katz AE
O: P: 1100 - 1130	C45	Carbon Monoxide (Co) Poisoning Precipitation Of Nstemi - A Case Report	Roberts AB, Weaver LK
O: P: 1100 - 1130	C46	Inaccurate Pulse Oximetry Of Carboxyhemoglobin Due To Digital Clubbing	Harlan NP, Weaver LK
O: P: 1100 - 1130	C47	Clostridial Myonecrosis: An Urgent Indication For Hyperbaric Oxygen Therapy	Millman MP
O: P: 1100 - 1130	C48	The Validity Of Juxta-Wound Tcoms In Predicting Healing Of Diabetic Foot Ulcers	Moon H, Strauss MB, La SS, Miller SS
O: P: 1100 - 1130	C49	Experiences With Gentian Violet As A Wound Dressing Agent	Strauss MB, Miller SS, Le PNJ, Daniller A
O: P: 1100 - 1130	C50	Case Studies: Hyperbaric Oxygen Therapy After Accidental Ingestion Of 35% Hydrogen Peroxide	Parikh M, Alvarez Villela M, Tettelbach WH
O: P: 1100 - 1130	C116	Rapid Hyperbaric Oxygen Therapy To Reverse Post-Operative Posterior Ischemic Optic Neuropathy.	Sward D, Sethuraman K, Rosenthal R
O: P: 1100 - 1130	C117	Continuous Bladder Irrigation In The Monoplace Hyperbaric Chamber	Cooper JS, Allinson P, Winn D, Keim LW, Sippel J, Scahlberg P, Fowler K
11:30 AM - 12:30 PM	KINDWALL LECTURE: "HYPERBARIC SAFETY: A HALF-CENTURY COMMITMENT"		Paul Sheffield, PhD 1
12:30 PM - 1:30 PM	LUNCH – ON OWN		
12:30 PM - 1:30 PM	OPTIONAL LECTURE: "HYPERBARIC MEDICINE IN A VALUE BASED PAYMENT SYSTEM: WHAT IT WILL TAKE TO SURVIVE AND THRIVE"		Caroline Fife, MD; Laurie Gesell, MD; Helen Gelly, MD 1
1:30 PM - 3:00 PM	SESSION D: DIVING AND DECOMPRESSION ILLNESS MODERATORS: Tracy LeGros, MD & Matt Schweyer, CHT		1.5
O: 1330 - 1342 P: 1530 - 1600	D51	Factors Influencing The Energy Cost Of Free Fin Swimming	Pendergast DR, Hostler D
O: 1342 - 1354 P: 1530 - 1600	D52	Effect Of Pressure On Heating And Cooling Requirements For Thermal Protection Of Wet-Suited Divers	Pendergast DR, Hostler D

O: 1354 - 1406 P: 1530 - 1600	D53	Swimming-Induced Pulmonary Edema (Sipe) In Triathletes: Effect Of Age	Moon RE, Martina SD, Peacher DF, Otteni CE, Wester TE, Potter JF
O: 1406 - 1418 P: 1530 - 1600	D54	Measuring The Accuracy Of Artisanal Fishermen's Underwater Depth Perception	Chin W, Huchim O, Joo E, Fang S, Sprau S
O: 1406 - 1418 P: 1530 - 1600	D55	Diving Behavior And Decompression Stress Among Artisanal Fishermen From The Yucatan Peninsula, Mexico.	Chin W, Huchim O, Ninokawa S, Chan E, Huang C
O: 1418 - 1430 P: 1530 - 1600	D56	Field Dive Monitoring: Bubble Presentation In Recreational-Technical Closed-Circuit Rebreather Trimix Diving	Pollock NW, Wiley JM, Kernagis DN, Clarke NW, Mackey MN, Martina SD
O: 1430 - 1442 P: 1530 - 1600	D57	Observed Incidence Of Decompression Sickness And Venous Gas Bubbles Following 18 M Dives On Rn Table 11 / Norwegian Air Diving Table.	Blogg SL, Gennser M, Jurd KM, Mollerlokken A
O: 1442 - 1454 P: 1530 - 1600	D58	The Association Of Blood-Borne Microparticles And Neutrophil Activation With Decompression Sickness.	Thom SR, Bennett M, Banham N, Chin W, Blake DF, Rosen A, Pollock NW, Madden D, Barak O, Marroni A, Balestra C, Pieri M, Cialoni D, Le J, Logue C, Lambert D, Hardy KR, Sward D, Yang M, Bhopale VB, Dujic Z
3:00 PM - 3:30 PM	PM Break	<b>Fontaine Salons A-B</b>	
3:30 PM - 4:00 PM	<b>SESSION D: POSTERS</b>	<b>Fontaine Salons C-H</b>	0.5
O: P: 1530 - 1600	D59	Headache In Military Divers	Heravi MK, Ranjbar NA, Salehi HA, Khoshvaghti A
O: P: 1530 - 1600	D60	Evaluation Of The Autopsy Findings In Fatal Diving Accidents In Turkey	Koca E, Sam B, Arican N, Toklu AS
O: P: 1530 - 1600	D61	Spontaneous Pneumothorax In A Professional Divers Candidate; Case Report	Toklu AS, Sivrikaya H, Ozcelik C, Erelel M
O: P: 1530 - 1600	D62	Following The Surviving Sepsis Campaign Guidelines To Treat A Case Of Severe Decompression Sickness With Profound Shock	Huang KL, Peng CK, Shen CH, Chang SY, Chang SC
O: P: 1530 - 1600	D63	Facial Nerve Palsy Secondary To Oxygen Toxicity In Closed-Circuit Diving: A Case Report	Lim M, Chng J, Tang KC
O: P: 1530 - 1600	D64	Modeling Human Performance Limitations In The Submerged Environment	Shelley DA, Ng LJ
O: P: 1530 - 1600	D65	An Interim Report On Euroasia Tunnel Project	Toklu AS, Mirasoglu B, Arslan A, Aktas S
O: P: 1530 - 1600	D66	Educational Intervention Among Artisanal Fishermen Of The Yucatán Peninsula, Separating Engine Exhaust Gases From Compressor Intake.	Chin W, Huchim O, Popa D, Fang S
O: P: 1530 - 1600	D67	Reverse Takotsubo Cardiomyopathy In A Scuba Diver	Sadler C, Oyama L, Savaser D, Jacoby I
O: P: 1530 - 1600	D68	Outcomes Of Decompression Sickness Treated Using Ucsd Modified Treatment Table 6	Sadler C, Virgilio G, Owen E, Morgan A, Castillo E, Witucki P, Grover I
O: P: 1530 - 1600	D69	External Auditory Canal Diving Related Barotrauma – A Case Study	Swaby Ja, Dayya D, O'neill OJ, Heyboer M
O: P: 1530 - 1600	D70	Bleeding Gums And A Pain In The Face: The Dental Health Of Uk Divers	St Leger Dowse M, Penny CEL, Smerdon GR
O: P: 1530 - 1600	D71	Human Accommodations To Diving In Cold Water	Strauss MB, Miller SS, Le PNJ
O: P: 1530 - 1600	D72	Predictive Model Of Severe Decompression Illness In Artisanal Fishing Divers Of The Yucatan Peninsula	Chin W, Huchim O, Tec J, Wong L, Fang S, Sprau S, Popa D
O: P: 1530 - 1600	D73	Recreational Diving Fatalities: Harvesters Versus Non-Harvesters	Trout BM, Buzzacott P, Denoble PJ
O: P: 1530 - 1600	D74	Arterial Gas Embolism During A Dive	Alvarez Villela M, Weaver LK, Dhar R
O: P: 1530 - 1600	D75	Can My Patient Dive After A First Episode Of Primary Spontaneous Pneumothorax?	Alvarez Villela M, Parikh M, Harlan N, Roberts A, Moon RE, Freiburger JJ
O: P: 1530 - 1600	D76	Decompression Sickness (DCS) After Chamber Dive To 18 Msw For 100 Min	Møllerlækken A, Blogg SL; Mueller B, Risberg J, Eftedal I
4:00 PM - 5:00 PM	<b>PLENARY SESSION: "NEW PEARLS OF WISDOM IN THE DIVING AND HYPERBARIC MEDICINE LITERATURE"</b>	UHM Fellows: Gerald Godfrey, MD and Charlotte Sadler, MD	1
5:00 PM - 6:00 PM	UHMS Annual Business Meeting	<b>Salle De Bal Montreal</b>	

6:00 AM - 5:00 PM		Registration	
7:00 AM - 8:00 PM		Continental Breakfast	
8:00 AM - 5:00 PM		<b>GENERAL SESSION</b>	
8:00 AM - 9:00 AM		<b>PLENARY SESSION: "INTERNATIONAL PERSPECTIVES: UPDATE ON CLINICAL RESEARCH PROJECTS IN HYPERBARIC OXYGEN THERAPY"</b>	Folke Lind, MD; Nicklas Oscarsson, PhD; Ian Millar, MBBS
9:00 AM - 10:30 AM		<b>SESSION E: HBO<sub>2</sub> THERAPY, CHAMBERS, AND EQUIPMENT</b> <b>MODERATORS: Lin Weaver, MD &amp; Gus Gustavson, ACHRN</b>	1
O: 0900 - 0912 P: 1100 - 1130	E77	Performance Of The Zyno Medical Z-800f, Cme Body Guard 323 Color Vision™ And Baxter Flo-Gard® 6201 Infusion Pumps For Monoplace Chamber Use.	Bell JE, Deru, K, Koumandakis G, Weaver LK
O: 0912 - 0924 P: 1100 - 1130	E78	Preliminary Evaluation Of The Zyno Z-800f, Cme 323 Color Vision And Alaris Med System Iii Infusion Pumps For Use In The Multiplace Chamber	Bell JE, Deru K, Koumandakis G, Weaver LK
O: 0924 - 0936 P: 1100 - 1130	E79	Hyperbaric Oxygen Therapy Effects On Blood Pressure	Heyboer M, Smith G, Santiago W, Wojcik S
O: 0936 - 0948 P: 1100 - 1130	E80	Effects Of Hyperbaric Oxygen Therapy On Diabetic Serum Glucose Levels: An Extended Study	Swaby JA, Morgan M, Jennings MS, Santiago W, Heyboer M
O: 0948 - 1000 P: 1100 - 1130	E81	Sham Hyperbaric Analysis Model - Part 1: Standard Pressure (Pilot) Study	Robinson T, Castillo E, Duchnik J, Witucki P, Savaser D
O: 1000 - 1012 P: 1100 - 1130	E82	Adverse Events In A Blinded, Randomized Trial Of Hyperbaric Oxygen For Post-Concussive Symptoms	Churchill S, Miller RS, Deru K, Weaver LK
O: 1012 - 1024 P: 1100 - 1130	E83	Rates Of Myopia In Patients Receiving Hyperbaric Oxygen In Monoplace Or Multiplace Chambers	Wilson G, Cable R, Churchill S, Deru K, Weaver LK
10:30 AM - 11:00 AM		AM Break	
11:00 AM - 11:30 AM		<b>SESSION E: POSTERS</b>	<b>Fontaine Salons C-H</b>
O: P: 1100 - 1130	E84	Preparation And Implementation Of A 53.5 Hour Saturation Dive; Tech Perspective	Wudtke R, Pullis M
O: P: 1100 - 1130	E85	The Use Of Simulation Scenario Training In A Hyperbaric Multi-Place Chamber	Jensen AJ
O: P: 1100 - 1130	E86	A Case Study Of Severe Decompression Sickness In An Artisanal Fisherman In Yucatan, Mexico	Popa D, Chin W, Huchim O, Tec J, Grover I
O: P: 1100 - 1130	E87	Safety And Efficacy Of Needle Myringotomy Performed By Hyperbaric Medicine Physicians	Masters T, Westgard B, Logue C, Hendriksen S, Walter J
O: P: 1100 - 1130	E88	Incident Study Of Myopia In Patients Undergoing Hyperbaric Treatment	Gorenstein S, Hangan D, Regan K, Merrow M
O: P: 1100 - 1130	E89	The Chinese Standard - Clinical Application Technique Specification Of Hyperbaric Oxygen	Qingle L
O: P: 1100 - 1130	E90	An Extraordinary Case Of Type 2 DCS	Strauss MB, Miller SS
O: P: 1100 - 1130	E91	The Use Of Trend Analysis Of Blood Sugars In Diabetic Patients Undergoing Hyperbaric Oxygen Therapy To Predict Wound Failure	Pearson M, Harch P, Hardy S, Murphy-Lavoie H, Legros T, Wyatt H, Yontz D, Van Meter K
O: P: 1100 - 1130	E92	Case Report Of A Tension Pneumothorax During Hyperbaric Oxygen Therapy	Evangelista JS, Hare MA, Medak AJ
O: P: 1100 - 1130	E93	Thinking Outside The Box About Safety – Not Just The Monthly Formal Exercise	Cormier JE
O: P: 1100 - 1130	E94	Impacts On Daily Activities Following Changes In Visual Function During And After Hyperbaric Oxygen Treatment	Wannholt R, Arnell P, Zetterberg M, Stomberg MW, Grönlund MA
11:30 AM - 12:30 PM		<b>PLENARY SESSION: "MY SECRETS FOR EFFECTIVE PRACTICE-BASED CLINICAL RESEARCH"</b>	Neil Hampson, MD
12:30 PM - 1:30 PM		LUNCH – ON OWN	
1:30 PM - 3:00 PM		<b>SESSION F: CLINICAL HBO<sub>2</sub> THERAPY (#2)</b> <b>MODERATORS: Heather Murphy-Lavoie, MD &amp; Laura Josefsen, ACHRN</b>	1.5
O: 1330 - 1342 P: 1530 - 1600	F95	Necrotizing soft tissue infection	Rosén A, Arnell P, Lycke H, Oscarsson N
O: 1342 - 1354 P: 1530 - 1600	F96	Adjuvant Hyperbaric Oxygen Therapy for Necrotizing Soft Tissue Infections: Preliminary Analysis from a Study of Two Urban Centers	Walter J, Westgard B
O: 1354 - 1406 P: 1530 - 1600	F97	Globalization and international collaboration in publications of Undersea and Hyperbaric Medicine, 1974 to 2014	Lee CH
O: 1406 - 1418 P: 1530 - 1600	C33	Hyperbaric Oxygen Therapy For Compromised Flaps: Evaluation Of The Optimal Treatment Protocol	Weber R, Silver A, Williams S, Stephenson L, Usera PC, Zhang F, Tian H, Yang W, Wang WZ, Fang XH, Zamboni WA, Baynosa R
O: 1418 - 1430 P: 1530 - 1600	F99	Hyperbaric Oxygen Therapy For Pyogenic Spondylitis	Kawashima M, Kawashima M Tamura H, Takao K, Yamaguchi T, Miyata K

O: 1430 - 1442 P: 1530 - 1600	F100	The Incidence Of Confounding Factors In Diabetes Mellitus Patients Hospitalized For Lower Extremity Wounds	Strauss MB, Moon H, Craig AB, Ponce JP, Miller SS, Le PNJ
O: 1442 - 1454 P: 1530 - 1600	F101	Differences In Clinician Reimbursement Based On Treatment Of Hyperbaric Emergency Indications	Chin W, Chang M, Simon O, Proano J, Huang E
3:00 PM - 3:30 PM	PM Break		<b>Fontaine Salons A-B</b>
3:30 PM - 4:00 PM	<b>SESSION F: POSTERS</b>		<b>Fontaine Salons C-H</b> 0.5
O: P: 1530 - 1600	F102	Recruiting Divers To Study The Ketogenic Diet And Central Nervous System Oxygen Toxicity Symptoms	Annis H, Dituri J, Boschuetz T, Kirsten D, Hansen M, Whelan H
O: P: 1530 - 1600	F103	Recurrence Of Neurological Deficits In An F/A-18d Pilot Following Loss Of Cabin Pressure At Altitude	Robinson TD
O: P: 1530 - 1600	F104	Hyperbaric Oxygen For Exceptional Blood Loss Anemia: A Case Report	Ptak J, Reetz S, Manfred C, Buckey J
O: P: 1530 - 1600	F105	Medical Check-Ups And Treatment Of Dysbaric Osteonecrosis	Yamaguchi T, Kawashima M, Kawashima M, Tamura H, Takao K, Miyata K
O: P: 1530 - 1600	F106	Calcific Uremic Arteriopathy Successfully Treated With Hyperbaric Oxygen Therapy - A Case Study	Swaby JA, Morgan M, Jennings MS, Santiago W, Heyboer M
O: P: 1530 - 1600	F107	Hyperbaric Oxygen Therapy For Cutaneous Calciphylaxis - A Case Study	Swaby JA, Morgan M, Jennings MS, Santiago W, Heyboer M
O: P: 1530 - 1600	F108	Acute Idiopathic Sudden Sensorineural Hearing Loss Successfully Treated With Hyperbaric Oxygen Therapy - A Case Study	Swaby JA, Morgan M, Jennings MS, Santiago W, Heyboer M
O: P: 1530 - 1600	F109	Hyperbaric Oxygen Therapy For Central Retinal Artery Occlusion	Masters T, Westgard B, Logue C, Hendriksen S, Walter J
O: P: 1530 - 1600	F110	Using Objective Criteria to Determine Level of Pain	Strauss MB, Chen S, Grinblat JY, Le PNJ
O: P: 1530 - 1600	F111	Quantifying Compliance In Patients With Diabetic Foot Ulcers	Strauss MB, Tan AM, Moon H, Miller SS
O: P: 1530 - 1600	F112	Hyperbaric Oxygen Salvation Of Prolonged Male Genitalia Strangulation	Johnson G, McBride L, Wakefield M, Yeast C
O: P: 1530 - 1600	F113	Understanding Vasculopathy As A "Acute Peripheral Arterial Insufficiency" Amenable To Hyperbaric Oxygen Therapy	Pearson M, Harch P, Hardy S, Murphy-Lavoie H, Legros T, Wyatt H, Yontz D, Van Meter K
4:00 PM - 5:00 PM	<b>PLENARY SESSION: "TROUBLE WITH BUBBLES: LESSONS IN ALTITUDE DECOMPRESSION SICKNESS"</b>		Brett Hart, MD 1
7:00 PM - 7:30 PM	Annual Awards Banquet Reception		<b>Verdun Room Foyer</b>
7:30 PM - 10:00 PM	Annual Awards Banquet		<b>Verdun Room</b>
10:00 PM -	Annual Awards Banquet After Party		<b>Castillon &amp; Terrace</b>
<b>TOTAL CONTINUING EDUCATION CREDITS: 3 DAYS</b>			<b>21.5</b>

**Accreditation Statement:**

The Undersea and Hyperbaric Medical Society is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

**Designation Statements:**

The Undersea and Hyperbaric Medical Society designates this live activity for a maximum of **22.5** AMA PRA Category 1 Credit(s)<sup>™</sup>. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

**Nursing CEU** is approved by the Florida Board of Registered Nursing Provider #50-10881. ASM Credit hours **22.5**.

Licenses Types Approved:

- Advanced Registered Nurse Practitioner
- Clinical Nurse Specialist
- Licensed Practical Nurse
- Registered Nurse
- Certified Nursing Assistant
- Respiratory Care Practitioner Critical Care
- Respiratory Care Practitioner Non-Critical Care
- Registered Respiratory Therapist
- Certified Respiratory Therapist

**NBDHMT:** This live activity is approved for **23** Category A credit hours by National Board of Diving and Hyperbaric Medical Technology, 9 Medical Park, Suite 330, Columbia, South Carolina 29203.

**Full Disclosure Statement:** All faculty members and planners participating in continuing medical education activities sponsored by Undersea and Hyperbaric Medical Society are expected to disclose to the participants any relevant financial relationships with commercial interests. Full disclosure of faculty and planner relevant financial relationships will be made at the activity.

**Disclaimer:** The information provided at this CME activity is for Continuing Medical Education purposes only. The lecture content, statements or opinions expressed however, do not necessarily represent those of the Undersea and Hyperbaric Medical Society.

# ASSOCIATES' BREAKOUT SESSION

THURSDAY, JUNE 18

8:45am – 5:00pm

## MORNING SESSION

8:45	9:15	Reimbursement Update	Valerie Short
9:15	9:30	The Health of Diver's Project	Marguerite St Leger Dowse
9:30	10:00	EXHIBITRS / BREAK	<b>Fontaine Salons A-B</b>
10:00	10:10	Medicare Public Use Files and Clinicians Billing Medicare and those that treat Emergency Indications	Maxxine Chang
10:10	10:20	Measuring the Accuracy of Artisanal Fishermen's Underwater Depth Perception	Sophia Fang
10:20	10:30	Decompression profiles on 13 fishermen from the Yucatan Peninsula	Jacob Proano
10:30	11:00	NFPA Update	Robert Sheffield
11:00	11:10	A hospital network-wide analysis of emergency department referrals for carbon monoxide poisoning to hyperbaric medicine services.	Rebecca Cable
11:10	11:20	Rates of myopia in patients receiving hyperbaric oxygen in monoplace or multiplace chambers.	Gail Wilson
11:20	11:35	Performance of the Zyno Medical Z-800F, CME Body Guard 323 Color Vision™ and Baxter Flo-Gard® 6201 infusion pumps for hyperbaric conditions	Jim Bell
11:35	11:50	Adverse events in a blinded, randomized trial of hyperbaric oxygen for post-concussive symptoms	Sue Churchill
11:50	12:00	Incident Study of Myopia in Patients Undergoing Hyperbaric Treatment	
12:00	12:15	MTAC Update	Richard Barry
12:15	1:15	Associates Business Luncheon	Castillon
1:15	2:15	Lambertsen Keynote Lecture	<b>Salle De Bal Montreal</b>

## AFTERNOON SESSION

2:15	2:45	Fluoroscopy	Timothy Mayhugh
2:45	3:30	Leadership	Richard Gustavson
3:45	4:15	EXHIBITS / BREAK	<b>Fontaine Salons A-B</b>
4:15	4:45	NBDHMT Update	Valerie Short
4:45	5:00	Session Wrap Up	Kip Posey

# EVALUATION

In an effort to “GO GREEN” and improve the efficiency in evaluating our CME Program, we now offer the evaluation form online. A hard copy evaluation form can be provided upon request. Thank you for supporting our efforts to help reduce our carbon footprint.

**EVALUATION LINK: THIS WILL ALSO BE EMAILED TO YOU**

<https://www.surveymonkey.com/s/Mont15>

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# MAINTENANCE OF CERTIFICATION (MOC)

**ONLINE LINK TO THE MOC QUESTIONS:**

<https://www.surveymonkey.com/s/MOCASM15>

## ***ABPM Requirements for Maintenance of Certification (MOC) for 2015 Certification***

Becoming board certified is the first step towards a career in preventive medicine. Once board certified, physicians are expected to maintain lifelong learning and competencies, which are assessed through participation in the ABPM Maintenance of Certification (MOC) program. The American Board of Medical Specialties (ABMS) sets the MOC requirements, which are organized into 4 parts as identified below. The ABPM MOC program continues to evolve in concert with ABMS requirements; therefore it is anticipated that the actual requirements will evolve over time. Please ensure that your contact information remains up to date so that you may be included in notifications of these modifications. Also please make sure to check the ABPM website for updates as you will be responsible for successfully participating. The ABPM MOC program, in compliance with ABMS requirements as currently defined, includes the following four components:

### **Part 1: Professional Standing**

Diplomates must hold an active, valid and unrestricted medical license in all States, US territories, or Canadian Provinces in which the diplomate is licensed to practice medicine.

### **Part 2: Lifelong Learning and Self-assessment (LLSA)**

A total of 250 hours of Continuing Medical Education (CME) over the 10-year span of certification is required. A minimum of 100 hours of the CME must be ABPM-approved LLSA activities. A complete list of approved LLSA activities is available on the ABPM website at <https://www.theabpm.org/moc/modules.cfm>. A patient safety module must be completed in the first two years of the certification cycle as part of this requirement. Further information will be posted on our website as soon as the module is available. For those diplomates maintaining certification with another ABMS specialty board, 150 hours of CME can be satisfied by completing the MOC Part 2 requirements of the other ABMS specialty board, so that only the 100 LLSA/MOC credits need to be completed. More information is available on the ABPM website at [https://www.theabpm.org/moc/ABPM\\_MOC\\_Alternate\\_Credit.pdf](https://www.theabpm.org/moc/ABPM_MOC_Alternate_Credit.pdf).

### **Part 3: Assessment of Cognitive Expertise**

The cognitive exam may be taken starting seven (7) years after receiving certification, and may be repeated if necessary to pass prior to the expiration of the certificate.

### **Part 4: Assessment of Practice Performance**

NEW: Diplomates are required to complete two practice performance assessments during their 10 year certification cycle. One assessment is to be completed in the first 5 years of the cycle and a second in the last 5 years. Diplomates may complete the Assessment of Practice Performance through any of the three Preventive Medicine specialty societies. Please contact the appropriate specialty society for more detailed information.

ACOEM: [www.acoem.org/moc.aspx](http://www.acoem.org/moc.aspx)

ACPM: [www.acpm.org/?page=MOC\\_description](http://www.acpm.org/?page=MOC_description)

AsMA: [www.asams.org/MOC.htm](http://www.asams.org/MOC.htm)

Check the ABPM website periodically for future changes to Part 4 as the program evolves in concert with ABMS requirements. For those diplomates maintaining certification with another ABMS specialty board, a Practice Performance Assessment requirement may also be satisfied by completing the MOC Part 4 requirements of that ABMS specialty board. Diplomates eligible for this pathway can find further information at [www.theabpm.org/moc/ABPM\\_MOC\\_Alternate\\_Credit.pdf](http://www.theabpm.org/moc/ABPM_MOC_Alternate_Credit.pdf).

More information concerning the MOC program, fees and approved LLSA/MOC activities may be found on the ABPM website

[https://www.theabpm.org/moc/index\\_moc.cfm](https://www.theabpm.org/moc/index_moc.cfm). Please contact the ABPM if you have additional questions. We request that you maintain a current file with the ABPM including address, phone number, and e-mail address. Please check your information by signing in at [www.theabpm.org](http://www.theabpm.org). The ABPM uses email to send diplomates periodic updates about the MOC program.

# COMMITTEE MEETING SCHEDULE

<b>Tuesday, June 16</b>			
4:00pm	11:00pm	Board Meeting	Frontenac
<b>Wednesday, June 17</b>			
10:00am	11:00am	MOC Committee	St. Pierre
1:00pm	4:00pm	ABPM EXAM	St. Pierre
4:45pm	7:45pm	HBO Therapy Committee Meeting	Lougueuil
<b>Thursday, June 18</b>			
7:00am	8:00am	Editorial Board Meeting	Fundy
7:00am	8:00am	Safety Committee Meeting	St. Pierre
12:00pm	1:00pm	GME Committee	Fundy
1:00pm	6:00pm	CHT/CHRN Study Hall (1-3) / EXAM (4-6)	St. Pierre
3:00pm	5:00pm	Education Committee	Lougueuil
6:00pm	8:00pm	Accreditation Council Meeting	Lougueuil
6:30pm		BNA Board Meeting	Fundy
<b>Friday, June 19</b>			
7:00am	8:00am	Past-President's Breakfast	Longueuil
8:00am	11:00am	NBDHMT Board Meeting	Fundy
1:00pm	5:00pm	Safety Subcommittee Work Group (LeGros/Alleman)	Pointe-aux-Trembles
3:00pm	4:00pm	BNA General Meeting	Fundy
3:30pm	4:30pm	Publication Committee	Longueuil
5:00pm	6:00pm	UHMS Annual Business Meeting	General Session Ballroom
5:00pm	7:00pm	Specialty Council Meeting (Claus)	Longueuil
<b>Saturday, June 20</b>			
12:30pm	1:30pm	Membership/Chapter President's Committee	Fundy



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### SATCHEL SPONSOR



## Exhibitors

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**American Board of Preventive Medicine, Chicago, IL: TABLE # 2:** The American Board of Preventive Medicine, Incorporated (ABPM) is a member board of the American Board of Medical Specialities. ABPM originated from recommendations of a joint committee comprised of representatives from the Section of Preventive and Industrial Medicine and Public Health of the American Medical Association and the Committee on Professional Education of American Public Health Association. [www.theabpm.org](http://www.theabpm.org)

**Baromedical Nurses Association, Crystal River, FL: TABLE # 7:** The BNA provides nurses with a professional organization in order to maintain and promote the status and standards of practice in hyperbaric nursing. <http://www.hyperbaricnurses.org/>

**Best Publishing Company, Palm Beach Gardens, FL: BOOTH # 1:** Best Publishing Company was founded in 1966 and has become the largest and one of the most respected publishers of educational books on diving, wound care, and hyperbaric medicine. We produce educational books along with professional periodicals such as the *Wound Care & Hyperbaric Medicine Magazine*, a peer reviewed quarterly publication that covers all aspects of wound care, diving medicine, and hyperbaric oxygen therapy (HBOT). We also produce the *Wound Care & Hyperbaric Medicine Calendar* that promotes diving, wound care, and hyperbaric education courses worldwide. [www.bestpub.com](http://www.bestpub.com)

**Divers Alert Network, Durham, NC: Booth # 15:** The largest association of recreational scuba divers in the world, DAN is supported by membership dues and donations. DAN's mission is to help divers in need of medical emergency assistance and to promote dive safety through research, education, products and diving services. The benefits of DAN membership include emergency medical evacuation assistance through DAN *Travel Assist*, a subscription to *Alert Diver* magazine and access to DAN's insurance services. [www.dan.org](http://www.dan.org)

**Environmental Tectonics Corporation, Southampton, PA: BOOTH # 10:** ETC is a premier designer and manufacturer of clinical hyperbaric chambers serving a worldwide client base. The ETC BARA-MED monoplace has many unique features including fully redundant automatic and manual control systems, user-friendly SMOOTH•RIDE? compression protocol that significantly reduces complications due to middle ear squeeze without adding to compression time, and hard-copy record of the actual treatment profile. For more information on our products and product support services visit us at [www.etcBioMedical.com](http://www.etcBioMedical.com)

**Fink Engineering, PTY, LTD, Warana, Australia: TABLE # 10:** Fink Engineering Pty Ltd (a subsidiary of Fink International) was established in Victoria, Australia in 1987 to provide engineering design and consultancy to the offshore oil and diving industries. We have developed a world class set of Rectangular Hyperbaric Chamber Systems that are just beginning to be appreciated overseas as evidenced by systems shipped to New Zealand, Singapore, Canada and our recently completed projects in the USA. [www.fink.com.au](http://www.fink.com.au)

**Groupe Medical Gaumond Inc, Quebec, Canada: Booth # 2:** GMG offers innovative hyperbaric devices to perform medical treatments of Hyperbaric Oxygen Therapy (HBOT). GMG is a passionate company, specialized in biomedical engineering that took up an important challenge: design a truly transportable hyperbaric chamber. GMG innovates in developing its flagship product, the HematoCare™ a monoplace, flexible hyperbaric chamber working up to 3 ATA. The HematoCare™ distinguishes itself from competition by an unrivalled versatility and aims to make hyperbaric oxygen therapy more accessible. Our mission is to provide safe and reliable devices which meeting the real user's needs. GMG also offers service related to the certification and design of medical devices. <http://www.groupemedicalgaumond.com/en/>

**Hydrospace Group Inc., Claremont, CA: Booth # 13:** Hydrospace Group is dedicated to providing cost effective, innovative, high precision and reliable solutions for components, systems and vehicles designed to work underwater, from 20 to 20,000 feet. The dedication is rooted in the realization that the future will continue to demand new tools and methods to increase subsea productivity. These solutions will inherently combine a wide range of manned and unmanned technologies that need to work in concert. The harsh nature of this environment across the globe's oceans demands the highest levels of performance to achieve safety and reliability that will ensure commercial viability. <http://hydrospacegroup.com/>

**Intellicure, The Woodlands, TX : BOOTH # 3:** , Intellicure was developed by clinicians for clinicians. Our expertise and understanding empowered us to create a wound care software system that is built around your work flow needs. Intellicure provides clinicians with the tools they need to deliver evidence based care, ease concerns regarding billing and coding, facilitate clinical research, and make life easier. Intellicure is also at the forefront of research and development in the Wound Care and Hyperbaric community, working alongside healthcare professionals and vendors alike. By providing enormous amounts of quality data, Intellicure is a valued resource in its industry. <http://www.intellicure.com/>

**International ATMO, Inc., San Antonio, TX: TABLE # 4: Associate Luncheon Sponsor:** International ATMO, Inc. is one of the oldest continuous providers of hyperbaric medicine education services including hyperbaric consulting, hyperbaric safety training, hyperbaric oxygen treatment, wound center consulting, wound care education and wound center management. International ATMO's continuing education courses in hyperbaric medicine, wound center management, wound care center education and safety training attract an international attendance of physicians, nurses, and technicians annually. The Hyperbaric Medicine Team Training Course is the original UHMS-Designated Introductory Course in Hyperbaric Medicine that meets the requirements of all Medicare Intermediaries. We also offer various hyperbaric education books, wound care center books as well as books from NFPA and UHMS. [www.hyperbaricmedicine.com](http://www.hyperbaricmedicine.com)

**King Faisal Specialist Hospital & Research Center, Riyadh, Saudi Arabia: Booth # 4:** King Faisal Specialist Hospital & Research Centre provides the highest level of specialized healthcare in an integrated educational and research setting.

**Masimo, Irvine, CA: TABLE # 8:** Masimo (NASDAQ: MASI) is a global medical technology company that develops and manufactures innovative noninvasive patient monitoring technologies, including medical devices and a wide array of sensors. A key medical technology innovator, Masimo is responsible for the invention of award-winning noninvasive technologies that are revolutionizing patient monitoring, including Masimo SET™ pulse oximetry, Masimo Rainbow Pulse CO-Oximetry and new Masimo noninvasive and continuous total hemoglobin (SpHb™) monitoring technology. The recent introduction of the first-and-only FDA-cleared technology that noninvasively and continuously measures total hemoglobin without a painful needle stick and invasive blood draw is expected to open new markets and significantly expand the company's growth opportunities. [www.masimo.com](http://www.masimo.com)

**Novadaq Technologies, Mississauga, ON, Canada: BOOTH # 7** Enabling surgeons with clinically-relevant, innovative fluorescence imaging solutions to enhance the lives of patients and their surgeons, while reducing health care costs, is Novadaq's global mission. SPY fluorescence imaging technology provides surgeons with real-time visualization, leading to improved outcomes and reduced costs without exposing the patient to radiation. More than 65 peer-reviewed publications demonstrate that the use of SPY during complex surgery, leads to fewer post-operative complications and lower hospital costs. [www.novadaq.com](http://www.novadaq.com)

**OSnovation Systems, Santa Clara, CA: Booth #11:** OSNovative Systems, Inc. is a privately held company, owned and managed by a team of dedicated professionals. The company develops, manufactures and sells proprietary devices for advanced wound management, combat/emergency medicine and cosmetic skin care. <http://www.osnovation.com/>

**OxyHeal Health Group, National City, CA: BOOTH #: 14:** OxyHeal Health Group is made up of three corporations that have collectively been in business for over 35 years, specializing in the delivery of "high dose oxygen," or Hyperbaric Medicine, and the treatment of complex wounds. OxyHeal's member companies encompass both medical device manufacturing and the clinical operation of Hyperbaric Systems and Wound Healing Centers in Hospital and University programs worldwide. In addition, one of OxyHeal's companies operates didactic and on-line training programs for physicians, nurses and technicians. OxyHeal Health Group's predominant product is the company's unparalleled knowledge, training and experience in the delivery of Hyperbaric Oxygen Therapy and advanced wound care to patients both in clinical and emergency settings. [www.oxyheal.com](http://www.oxyheal.com)

**Perimed, Inc., North Royalton, OH: TABLE # 6:** With over 25 years of development, Perimed AB manufactures and markets state-of-the-art Laser Doppler Instruments for the measurement of microvascular perfusion. As a leader in the Wound Care and Hyperbaric Medicine departments, Perimed, Inc., offers the only combined Laser Doppler and Transcutaneous multi-channel monitor, the PeriFlux 5000 system. This system allows the user friendly flexibility of multiple site monitoring. PeriFlux instruments represent a commitment that begins with quality and performance, and continues with technical and applications support. Please visit our website at [www.perimed-instruments.com](http://www.perimed-instruments.com)

**Perry Baromedical, Riviera Beach, FL: TABLE # 3:** Perry Baromedical is the only company in the world which designs, manufactures, installs and services monoplace, dualplace and multiplace hyperbaric chambers. We provide the highest quality product, and are focused on assisting hospitals with a comprehensive Hyperbaric Oxygen Therapy department. For further information visit our website at [www.perrybaromedical.com](http://www.perrybaromedical.com) or call us at 561-840-0395. [www.perrybaromedical.com](http://www.perrybaromedical.com)

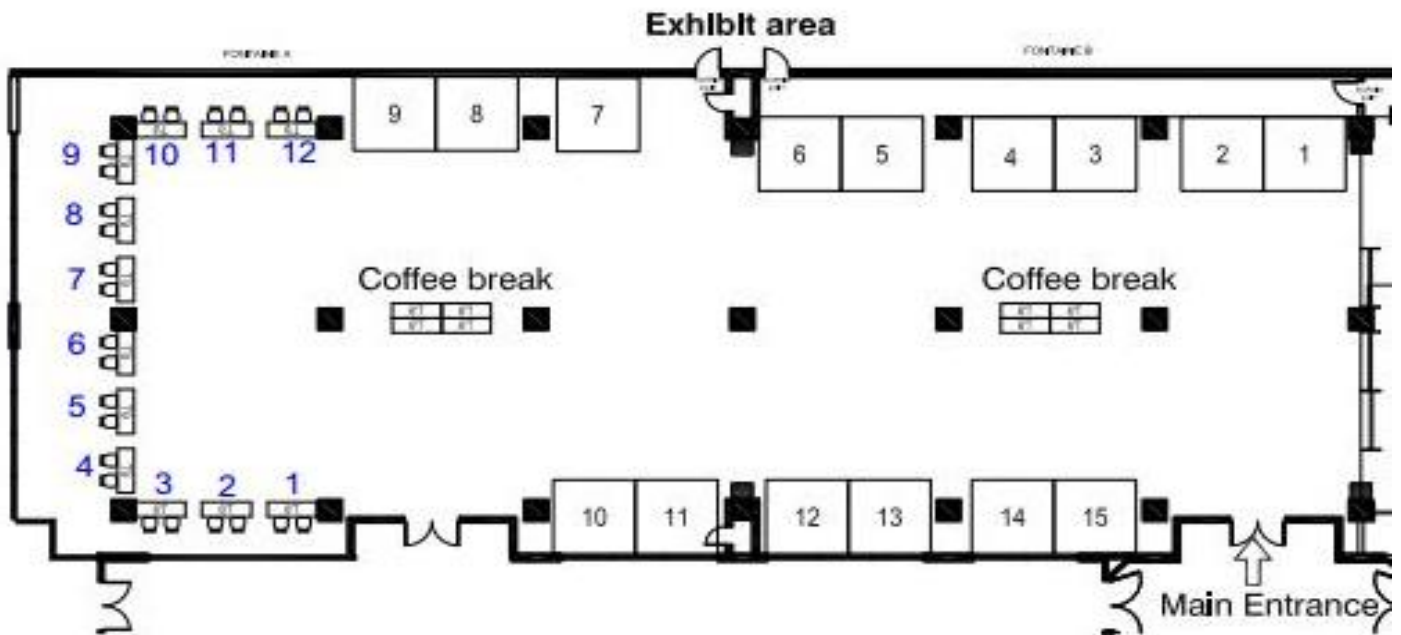
**Reimers Systems Division of PCCI, Inc., Alexandria, VA: BOOTH # 8:** With decades of experience, we offer hyperbaric chambers, research chambers, altitude chambers, oxygen service solutions, manifolds and other accessories like hood drivers, gas selection panels and utility penetrators, site development and engineering services, chamber installation and maintenance. Our sister company, Hyperbaric Clearinghouse, offers quality pre-owned chambers and equipment. [www.reimersystems.com](http://www.reimersystems.com)

**Sechrist Industries, Anaheim, CA: BOOTH # 5:** For over 30 years, Sechrist Industries, Inc., continues to be a leading worldwide manufacturer of hyperbaric chamber systems, neonatal, infant and pediatric intensive care ventilators, and air/oxygen mixers along with other ancillary accessories. All products are manufactured in accordance with FDA and GMP regulations. [www.sechristusa.com](http://www.sechristusa.com)

**West Care Medial Ltd., Coquitlam, BC Canada: TABLE # 1:** West Care Medical was established in 1996 and employs 29 full time staff members. We offer a range of services for our customers in both the health care industry, as well as individual homecare recipients. Our sales and service team brings a combined 170 years of medical experience to our health care customers in British Columbia, Alberta, Saskatchewan and Manitoba. <http://westcaremedical.com/Pages/home.php>

## EXHIBITOR FLOOR PLAN

- B1 Best Publishing
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- T7 Baromedical Nurses Association "BNA"
- T8 Masimo
- T10 Fink
- T12 UHM Fellowship



**SESSIONS  
&  
ABSTRACTS**

**THURSDAY, JUNE 18**

# “PRESIDENT’S ADDRESS”

**James Holm, MD**

**8:00AM – 8:30AM**



Dr. James R. Holm is currently the Medical Director of the Center for Hyperbaric Medicine at Virginia Mason Medical Center in Seattle, Washington. Dr. Holm received his medical degree from Georgetown University School of Medicine in 1985. He completed a combined residency and served as chief resident in Emergency Medicine and Internal Medicine at Northwestern University in 1989. He is triple board-certified in Emergency Medicine, Internal Medicine as well as Undersea and Hyperbaric Medicine.

Dr. Holm has been a member of the UHMS since 1996 and is currently a Member at Large on the UHMS Board of Directors. He has served on the UHMS Annual Scientific Meeting Program Committee for over six years and remains active with many society educational/scientific programs. He has been active in local and regional hyperbaric activities and was President of the Pacific Chapter of the UHMS in 2005. He has been faculty and program co-chair for the Winter Symposium in Hyperbaric Medicine and Wound Management as well as a frequent faculty member for both the UHMS and DAN diving and hyperbaric medicine courses since 2003. He is a faculty member for the NOAA/UHMS Physician Diving Medicine Course in Seattle for the last 3 years and is a graduate of that program in 1996.

Dr. Holm has been working in Hyperbaric Medicine since 1997 and has extensive experience in routine and emergency hyperbaric practice. This includes both multiplace and monoplace chamber systems. He has also been involved in clinical wound care practice and education. His clinical activity and research interests include decompression illness, carbon monoxide poisoning, and treatment of late effect of radiation tissue injury.

Dr. Holm grew up in Southern California and became a certified diver 1969 at the age of 13. He became a NAUI and PADI Instructor in 1978 and taught SCUBA while attending the University of California at Santa Barbara. He later worked as a divemaster and instructor on Grand Cayman Island from 1980 to 1981 before going to medical school. He is still an active diver and enjoys underwater photography and videography.

**ABOUT THE LECTURE:** The President's Address is intended to welcome meeting participants, provide an overview of the UHMS Annual Scientific Meeting's organization and planned presentations, and discuss topics of contemporary interest to the UHMS membership. These topics will primarily include identification of ongoing challenges to the Undersea and Hyperbaric Medicine profession and updates regarding current UHMS strategic initiatives.

**PLENARY:**  
**“MANAGING DECOMPRESSION STRESS:  
BEYOND THE ALGORITHM”**

**Neal Pollock, PhD**

**8:30AM – 9:30AM**



**ABOUT THE LECTURE:** The dive profile is most important, but there are a multitude of factors that can alter decompression stress. This presentation will consider both key and contributing factors that can change the risk. Practical strategies to optimize decompression safety will then be discussed.



**SESSION A**  
**DIVING & DECOMPRESSION ILLNESS:**  
**THEORY & MECHANISMS**

**Moderators: Folke Lind, MD & Neal Pollock, PhD**

**THURSDAY, JUNE 18**  
**10:00AM – 12:00PM**

# A 1

ORAL PRESENTATION TIME: 1000 - 1012

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **Microparticle, Neutrophil And Platelet Changes Associated With Hypobaric And Hypoxic Exposures**

**Thom SR<sup>1</sup>, YANG M<sup>1</sup>, Bhopale VB<sup>1</sup>, Sherman P<sup>2</sup>, Kochunov P<sup>3</sup>, McGuire S<sup>2,4</sup>**

<sup>1</sup>Department of Emergency Medicine, University of Maryland School of Medicine, Baltimore, MD.

<sup>2</sup>U.S. Air Force School of Aerospace Medicine, Aerospace Medicine Consultation Division, Wright-Patterson AFB, OH; <sup>3</sup>Department of Psychiatry, University of Maryland School of Medicine, Baltimore;

<sup>4</sup>Department of Neurology, Lackland AFB, TX

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**Introduction:** Research on altitude-induced decompression sickness (DCS) has been prompted because of an increased incidence of DCS in U-2 pilots. This study investigated whether changes occur in circulating microparticles (MPs), 0.1 – 1.0 nm vesicles, and other blood elements in human volunteers exposed to simulated 25,000 feet altitude in a hypobaric chamber.

**Materials and Methods:** With IRB approval, blood samples in commercial fixative were obtained from human subjects ½ hour before, immediately following, 24 and 72 hours after simulated altitude exposure. Subjects breathed oxygen through a face-mask while transiting to altitude. At altitude some subjects removed the mask to incur a hypoxic stress; others kept wearing the mask and so incurred decompression stress without hypoxia. Blood samples were analyzed for MPs characteristics, neutrophil and platelet activation following published techniques. No individuals sustained DCS.

**Results:** Hypobaric hypoxia, but not hypobaric stress alone resulted in significant alterations in MPs and neutrophils following exposures. Interestingly, the greatest MPs change occurred at 72 hours post-exposure. Subtle evidence of platelet-neutrophil interactions were found 24 hours post-exposure.

**Summary:** These are preliminary findings from an on-going investigation and thus, results must be interpreted with caution. It appears there are mild alterations in MPs and blood elements due to hypobaric hypoxia. Further work is required to validate changes, compare results against normal controls and to individuals subjected only to altitude.

## A 2

ORAL PRESENTATION TIME: 1012 - 1024

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

### **Ascorbic Acid Abrogates Microparticle Generation And Vascular Injuries Associated With High Pressure Exposure**

Yang M, Bhopale VB, Thom SR

Department of Emergency Medicine, University of Maryland School of Medicine

Baltimore MD

MYang@smail.umaryland.edu

**Introduction:** Circulating microparticles (MPs), 0.1 – 1.0 nm vesicles, are elevated in animals and humans after simulated or *bona fide* underwater diving. Studies with isolated neutrophils have shown that by inhibiting inert gas-mediated oxidative stress, ascorbic acid can inhibit MPs production. This study investigated whether pathological changes associated with elevations of MPs following high pressure exposures can be abrogated by ascorbic acid in a murine model.

**Materials and Methods:** Mice were exposed for 2 hours to 790 kPa air and euthanized at 2 or 13 hours post-decompression to study blood-borne and vascular changes.

**Results:** Pressure/decompression cause over 3-fold elevations in circulating MPs as well as sub-groups bearing surface proteins for neutrophils, platelets and erythrocytes. There was evidence of significant neutrophil activation, platelet-neutrophil interactions and vascular injury to brain, omentum, psoas and skeletal muscles assessed as leakage of high molecular weight dextran. Prophylactic ascorbic acid (500 mg/kg IP) administration prevented all post-decompression neutrophil changes and vascular injuries. Ascorbic acid administration immediately after decompression abrogated most changes, but evidence of vascular leakage in brain and skeletal muscle at 13 hours post-decompression persisted. No significant elevations in these parameters occurred after injection of ascorbic acid alone.

**Summary:** The findings support the idea that MPs production that occurs with exposures to elevated gas pressure is an oxidative stress response and that anti-oxidants may offer protection from pathological effects associated with decompression.

# A 3

ORAL PRESENTATION TIME: 1024 - 1036

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## Prompt Recompression Treatment For Limb Bends Limits The Induction Of Dysbaric Osteonecrosis In The Uw Sheep Model

Sobakin AS<sup>1</sup>, Wilson MA<sup>2</sup>, Gendron-Fitzpatrick AP<sup>3</sup>, Lehner CE

<sup>1</sup>Department of Pediatrics, <sup>2</sup>Dept. of Radiology UW Hospital and Clinics, <sup>3</sup> Comparative Pathology Lab, Research Animal Resources Center, University of Wisconsin-Madison, Madison, WI, 53590 USA

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**Background:** Dysbaric osteonecrosis (DON) is an active bone injury complication of the high pressure environment that occurs when caisson workers and scuba divers are exposed to compressed air and inadequate decompression. We investigated the induction of DON using the sheep model of the diver by prolonged compressed air exposures and provocative “dropout” decompressions. Dysbaric osteonecrosis may lead to the joint collapse in the long bones of disabling secondary osteoarthritis.

**Materials and Methods:** Twenty-seven adult female sheep (90.5 ± 15.5 SD kg) underwent dry-air chamber exposures at 2.27 atm abs (43 fsw, 12.8 msw) for 24 hours, then rapid or “dropout” decompression at 30 feet/min (0.9 atm/min) to surface, and all sheep developed limb bends. Air recompression treatment (Modified USN Table 1A) followed with latencies of 4, 8, 10, or 14 hours. One month after decompression, sheep were injected with <sup>99m</sup>Tc-methylene diphosphonate (MDP) for bone scans of radii and tibiae to identify “hot spots” signifying long-bone DON lesions. Alizarin complexone fluorochrome was injected IV to visualize DON. Necropsies were performed to confirm DON lesions.

**Results:** Of 27 sheep that underwent recompression in the 4 groups, 12 sheep sustained DON lesions with active remodeling. Logistic regression showed that DON occurrence was significantly associated with hours of delayed recompression (Wald p = 0.015), with the odds of developing DON about twice as large for each additional hour of recompression delay (odds ratio = 1.99; 95% CI [1.15, 3.45]). Based on the logistic model, predicted incidence of DON rose from 4% at 4 hours to 98% at 14 hours, with DON incidence predicted to be 50% at 8.47 hours of delay (95% CI [5.30, 10.52]).

**Conclusion:** Delaying recompression treatment of limb bends can markedly elevate the incidence of DON and the likelihood of potentially disabling osteoarthritis in the affected diver or the rescued submariners.

# A 4

ORAL PRESENTATION TIME: 1036 - 1048

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## A Stress Index To Enhance Dcs Risk Assessment For Both Air And Mixed Gas Exposures

Hugon J<sup>1</sup>, R. Nishi<sup>2</sup>, Bouak F<sup>3</sup>, Blatteau JE<sup>4</sup>, Gempp E<sup>5</sup>

<sup>1</sup>BF Systemes – Technopole de la Mer, 229 chemin de la Farlède, 83500 La Seyne sur Mer, France

<sup>2</sup>Defence R&D Canada – Toronto (retired), Toronto, ON, Canada; <sup>3</sup>Defence Research and Development

Canada, Toronto Research Centre, 1133 Sheppard Avenue West, Toronto, Canada ; <sup>4</sup>Institut de

Recherche Biomédicale des Armées, Équipe de Recherche Subaquatique Opérationnelle, BP 600 Toulon

Cedex 9, France; <sup>5</sup>French Navy diving school, BP 311 83800 Toulon cedex 9, France

julien.hugon@bf-systemes.fr

**Introduction:** There are two common ways to assess decompression-induced physiological stress. The first requires statistical predictive tools calibrated with a large *diving profile/DCS* database<sup>1</sup>. The second approach, used extensively by DRDC Toronto Research Centre<sup>2</sup>, relies on the detection of bubbles using Doppler ultrasound. We have determined a new composite decompression stress index that can lead to improved DCS risk assessment and predictability.

**Materials and Methods:** Several decompression stress indices based on the inert gas load,  $Q$ , in the body and the total ascent time,  $TAT$ , were investigated for single air dives and mixed gas exposures from the DRDC database (8700 exposures, 106 DCS). The receiver operating characteristic (ROC) curve method was used to assess the diagnostic relevance of a given index compared to the  $P\sqrt{t}$  index. The bubble database was also used to modulate the index according to the observed DCS risk ratio between bubble grades to obtain the best ROC curves for diagnosis.

**Results:** The best decompression stress index,  $I=(Q-Q^*)/TAT^\alpha$  where  $Q^*$  is a threshold value and  $\alpha$  a constant, gave an ROC curve for air dives with an area under curve (AUC) of  $0.74\pm 0.07$  (95% CI) versus  $0.65\pm 0.06$  for the  $P\sqrt{t}$  index. Taking into account the precordial peak bubble grade values, the AUC can reach  $0.82\pm 0.06$ . For mixed gas, this index gave an AUC of  $0.91\pm 0.04$ .

**Conclusions:** The combination of diving profile, the gas breathed and the bubble monitoring information significantly improves the predictability of DCS risk. Indeed, the AUC obtained are very adequate for diagnostic purposes, with the index qualifying as “good” to “excellent” according to statistic standards.

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<sup>1</sup> Weathersby PK, Homer LD, Flynn ET. On the likelihood of decompression sickness. *J Appl Physiol* 1984; 57: 815-825.

<sup>2</sup> Sawatzky KD. The relationship between intravascular Doppler-detected gas bubbles and decompression sickness after bounce diving in humans. MSc thesis, York University, Toronto; 1991.

# A 5

ORAL PRESENTATION TIME: 1048 - 1100

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION: YES

## **Bubbles Cause Endothelial Damage In A Positive Correlation Manner**

Zhang K, Xu W

Department of Diving Medicine, Faculty of Naval Medicine, Second Military Medical University,  
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wg\_hsu@163.com

**Introduction/Background:** Several studies have documented that intravascular bubbles lead to endothelial dysfunction which may play a pivotal role in the underlying process of decompression sickness. Bubbles and their adverse effects on the endothelium should be a useful model for assessing decompression injury. The aim of the current study was to investigate potential changes in biomarkers of endothelial damage (lung edema, ET-1, 6-keto-PGF1 $\alpha$ , ICAM-1, VCAM-1, MDA and NO) and evaluate the relationship between bubble score and the degree of endothelial injury.

**Materials and Methods:** A total of 39 rats were divided into three groups, one normal control group and two diving groups with different profiles. The amount of bubbles in the pulmonary artery was determined ultrasonically for 2 hours following surfacing. The lung wet-to-dry weight ratio and levels of ET-1, 6-keto-PGF1 $\alpha$ , ICAM-1, VCAM-1, MDA and NO in serum were measured.

**Results:** Bubbles were seen solely after rapid decompression. The amount of bubbles was scored according to a grading system. Rapid decompression induced a significant increase in lung wet-to-dry weight ratio, ET-1, 6-keto-PGF1 $\alpha$ , ICAM-1, VCAM-1 and MDA, while NO showed a significant decrease. No difference was found between the normal control group and standard decompression group. In the diving group with rapid decompression, the degree of the above changes correlated positively with the number of bubbles presented.

**Summary/Conclusions:** This study highlights some of the effects of intravascular bubbles on the endothelial cells after rapid decompression. The results suggest that the endothelial damage is mainly caused by bubbles in a positive correlation manner.

# A 6

ORAL PRESENTATION TIME: 1100 - 1112

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **An Electron Microscope Study Of The Effects Of Decompression On The Spinal Cord And Hippocampus In The Rat: Preliminary Results**

Ofir D<sup>1</sup>, Kimmel E<sup>2</sup>, Menajem D<sup>1</sup>, Arieli Y<sup>1</sup>

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**Introduction:** There are a number of reports on the vulnerability of the spinal cord to decompression stress, but less is known on other regions of the brain. The purpose of the present study was to compare the effect of decompression from 8 or 10 ATA (bottom time 30 min) on the cellular structure of the hippocampus and the spinal cord.

**Materials and methods:** Nine male Sprague-Dawley rats (2 control, 7 experimental) weighing about 300g were exposed to different dive protocols (no pressure, 8 ATA, 10 ATA, for 30 min). Following decompression animals were anesthetized. Perfusion fixation was performed through the heart with paraformaldehyde 4%. The hippocampus and spinal cord were removed for electron microscope evaluation. Slides were analyzed with a magnification of 5,000 – 10,000. Myelinated axons were defined as small ( $\leq 2 \mu\text{m}$ ) or large ( $> 2 \mu\text{m}$ ). Each axon was scored according to the level of conservation of the myelin architecture (normal, moderate, poor).

**Results:** 359 spinal cord axons (51 control, 308 experimental) and 263 hippocampal axons (92 control, 171 experimental) were analyzed. 79% of the spinal cord axons and 100% of the hippocampal axons were small. Poor conservation of the myelin structure was found in 33% of the large axons and 14% of the small axons in the spinal cord. In the hippocampus, only 3% of the axons were poorly conserved.

**Conclusions:** These preliminary results suggest that myelin, due to its unique structure, is a major target for decompression-induced destruction. The myelin structure was more vulnerable in axons having a diameter greater than  $2 \mu\text{m}$ . Larger axons, with a corresponding volume of myelin (80% fat), may attract nitrogen during pressure exposure. The hippocampus may be protected against major structural changes in myelinated neuron cells due to its having only small axons. These results may explain some of the neuromotor symptoms involved in decompression sickness.

# A 7

ORAL PRESENTATION TIME: 1112 - 1124

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **Hypercapnea: Cognitive Effects and Monitoring - Assessment of Compressed Gas Narcosis using NASA's MATB-II Flight Simulator**

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**Introduction:** This is a NAVSEA project aimed at developing an algorithm to predict equivalent narcotic depth based on breathing gas inspired partial pressures. NASA's MATB-II flight simulator was chosen as the cognitive testing platform. Preliminary data was used to assess the MATB-II's ability to detect cognitive impairment during the target tracking (TRACKING) and system monitoring (SYSMON) tasks, which involve reacting to RED and GREEN warning lights and gauges (SCALES).

**Methods:** For each of 28 subjects, baseline MATB-II scores (1 ATA air at rest) were compared to those recorded while breathing varying hyperbaric partial pressures of CO<sub>2</sub>, N<sub>2</sub> and O<sub>2</sub> at rest and exercise. The possible effects of MATB-II learning and breathing gas sequence were assessed by pre-to-post test correlations. Inspired partial pressures tested were: (a) P<sub>i</sub>CO<sub>2</sub>=0 and 0.075 ATA (7.5 kPa), (b) P<sub>i</sub>O<sub>2</sub>=0.21 ATA (21.3 kPa), 1.0 ATA (101.3 kPa) and 1.22 ATA (123 kPa), and (c) P<sub>i</sub>N<sub>2</sub>=0.79 ATA (80.0 kPa), 4.6 ATA (465 kPa) and 5.6 ATA (567.3 kPa). TRACKING scores were controlled for recent video game experience, exercise and pre-test hypercapnic ventilatory response (HCVR). Significance was set at p=.05.

**Results:** Pre-to-post tests were well correlated (TRACKING R<sub>sq</sub>= 0.63, SYSMON R<sub>sq</sub>= 0.45, p<.01 two-tailed, Pearson). Arterial CO<sub>2</sub>, P<sub>i</sub>O<sub>2</sub>, P<sub>i</sub>N<sub>2</sub>, HCVR, exercise and video experience were significant predictors for TRACKING task scores using linear regression. End-tidal CO<sub>2</sub>, O<sub>2</sub>, N<sub>2</sub>, their interactions and exercise were significant predictors for SYSMON task scores by linear regression and repeated measures ANOVA respectively. P<sub>i</sub>N<sub>2</sub> was not a significant independent predictor at the pressures tested using SYSMON scores.

**Conclusions:** Cognitive impairment from surface CO<sub>2</sub> and all hyperbaric gases was detectable using the MATB-II. The TRACKING and SCALES tasks were most sensitive. Interactions were not prominent.

**Acknowledgements:** Funding provided by NAVSEA Deep Submergence Biomedical Contract #N0463A-12-C-0001.



# A 8

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **Do Lipids Or Proteins In Plasma Reduce Bubble Surface Tension? Interrelationships Between Plasma Chemicals, Surface Tension And Post-Dive Venous Gas Embolism**

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**Introduction/Background:** Decompression sickness (DCS) of divers is mostly caused by inert venous gas bubbles; venous gas embolism (VGE). Bubbles with surfactant exist longer due to a reduced surface tension ( $\gamma$ ). Also proteins may play a role. Interrelations between albumin, total protein, triglycerides, total cholesterol and free fatty acids, FFAs (i.e. not bound in other lipids),  $\gamma$  and VGE, measured before and after a dry air-dive simulation (21msw/40min) will be studied.

**Materials and Methods:** Participating divers (52) either had a fat-rich or a fat-poor breakfast to manipulate lipid levels. Eleven subjects obtained both. VGE was examined 40, 80, 120 and 160 min after surfacing (precordial Doppler method). Scores were transformed to Kisman Integrated Severity Scores and  $\gamma$  was determined with the dynamic bubble method.

**Results:** Theoretically, it was calculated that albumin was enough to cover all bubbles  $10^7$  times. Molecular dissolved FFA (long-chain; nM range) could just cover all bubbles (irrespective bubble grade), but this process is (too) slow and, moreover, the critical micelle concentration of long-chain-FFAs is  $10^6$ x higher. With various statistical analyses, it could not be established whether  $\gamma$  (ca. 57 mN/m) as well as VGE scores are related to albumin, total protein, total cholesterol, triglycerides and FFAs, with the latter two varying substantially between subjects and between pre- and post-exposure. Correlation coefficients were small;  $< 0.27$  and on average 0.11. Moreover, a relation between  $\gamma$  and VGE was lacking. Similar findings hold for the paired differences between the two exposures of the 11 subjects.

**Summary/Conclusions:** From these findings and theoretical considerations it seems likely that proteins do lower  $\gamma$ ; lipids do not. However, proteins hardly stabilize bubbles. Since the findings are not in concordance with the classical surfactant hypothesis, this hypothesis possibly needs revision. Schellart, ASEM 2014;85:1086-91, Schellart et al., UHM, in press.

# A 9

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION: YES

## The Etiology, Target Organ And Pathogenesis Of Decompression Disease

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**Introduction:** It is believed that decompression sickness (DCS) results from arterial gas embolism or similar reasons. However, some subjects whose circulation systems were full of air bubbles didn't show manifestation of DCS, which suggested that air embolism be not the conclusive evidences. Our objective is to clarify the etiology, target organs and pathogenesis of DCS.

**Methods:** Dogs and guinea pigs were respectively exposed to 0.6Mpa or 0.8Mpa for 60min or 150-500min, and then decompressed to normal pressure at 0.08 or 0.04 MPa/min, respectively. Blood pressure measurement of femoral arteries was taken from anesthetized dogs and pathological examination of bulbar conjunctival was made with anesthetized guinea pigs.

**Results:** All dogs with blood pressure of 180-220mmHg suffered from severe DCS when they were conscious, however, the dogs with 130-140 mmHg had no manifestation of DCS. The microvessels of the guinea pigs presented spasm, dysfunction and injury with varying extent and different duration. The closed arteries due to serious spasm stopped the flow of blood and gas, and the air bubbles in peripheral blood vessels could flow easily through various vessels. The bubbles showed shape changes to blood vessels and were easily deformed and broken to have diverting flows. The severity of DCS was correlated to the extent and duration of vascular spasm and dysfunction. The signs and symptoms could disappear by recovery of vascular function. The great disparity of blood volume led animals to significant different tolerance to decompression; the different function state of circulation systems led animals to individual difference at onset of DCS.

**Conclusion:** When ambient pressure decreased, if the blood gas expansion power and the sizes (cause) increased enough to result in spasm, dysfunction and injury (pathogenesis) of blood vessels (target organ), and in very imbalance (pathogenesis) of blood distribution, there came signs and symptoms of DCS.

# A 10

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **Brief Hypercapnic Breathing Attenuates Lung Injury Induced By Rapid Decompression**

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Decompression sickness (DCS) remains the most important risk in both professional and recreational diving because standard decompression procedures sometimes fail to prevent bubble formation. Hypercapnic acidosis has been proved to have a protective effect on tissue injury in several animal models. We investigated the effect of therapeutic hypercapnia during decompression on the DCS-induced lung injury. Rats were pressurized with air to 6 atmospheres absolute (ATA) for 60 or 120 min and then decompressed rapidly to the surface within 5 min to induced DCS and DCS-related lung injury. Hypercapnia was induced by replacement of compressed air with 3% CO<sub>2</sub> in 97% of air. Our results showed that brief hypercapnia for 10 min before decompression significantly attenuated acute lung injury and reduced the rate of animal death, whereas a prolonged hypercapnia during the whole bottom time augmented the DCS-induced lung injury. To exclude the effect of hemodynamic alterations by hypercapnia, we further verified the protective effects on the gas bubble-induced tissue injury in an isolated rat lung model. Our results showed that hypercapnia before pulmonary artery air infusion attenuated gas embolism-induced lung injury. Hypercapnia significantly attenuated acute lung injury by inhibition of the inflammation cascade. In conclusion, our results suggested that brief hypercapnia before decompression may protect from DCS-induced lung injury via inflammation inhibition. However, the timing of administration and the dosage of CO<sub>2</sub> breathing remain to be stratified and the clinical application needs further investigation on human subjects.

# A 11

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## The Therapeutic Effect Of Relatively Low Pressure Hyperbaric Oxygen For Decompression Sickness Induced By Fast Buoyancy Ascent Escape

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**Objective:** To observe the therapeutic effect of relatively low pressure hyperbaric oxygen (HBO) for decompression sickness (DCS) induced by fast buoyancy ascent escape (FBAE). **Methods:** 96 SD rats were randomly divided into six groups: air exposure group (air control group), 100 kPa oxygen treatment group (100 O group), 220 kPa HBO treatment group (220 HBO group), 220 kPa hyperbaric air treatment group (220 air group), 280 kPa HBO treatment group (280 HBO group), and 400 kPa hyperbaric air treatment group (400 air group), each n=16. Rats of All groups firstly received the same compressing and decompressing protocol, which was used to simulate FBAE. After that, rats received different treatment. Researchers watched rats' breath and activity when decompression was accomplished, and recorded the number of deaths. The survival rats were anesthetized, and taken out of heart and lung for histopathological examination.

**Results:** The mortality of all groups were 62.5% (air control group), 31.25% (100 O group), 18.75% (220 HBO group, Pearson's Chi-squared test,  $p=0.0308$ ), 43.75% (220 air group), 18.75% (280 HBO group,  $p=0.0308$ ), and 12.5% (400 air group,  $p=0.0106$ ) respectively. The pathological appearance of the lung in the air control group showed marked eosinophilic staining in alveolar, and thicker alveolar septum, but the pathological changes in 220 HBO group, 280 HBO group or 400 air group were less than that. The myocardial fibers of the air control group showed obvious edema, degeneration and rupture but in 220 air group, 280 HBO group or 400 air group showed slight edema. **Conclusions:** 220 kPa HBO can down regulate incidence of DCS, and ameliorates DCS induced heart and lung injury. These effects are similar to 280 kPa HBO and 400 kPa hyperbaric air treatments. This in turn helps to provide fast and feasible rescue measures of DCS induced by FBAE.

# A 12

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **Clopidogrel Reduces The Inflammatory Response Of Lung In A Rat Model Of Decompression Sickness**

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Inflammation and platelet activation are critical phenomena in the setting of decompression sickness. Clopidogrel inhibits platelet activation and may also reduce inflammation. The goal of this study was to investigate if clopidogrel had a protective role in decompression sickness (DCS) through anti-inflammation way. Male Sprague-Dawley rats (n=110) were assigned to three groups: Control + vehicle group, DCS + vehicle, DCS + clopidogrel group. The experimental group received 50 mg/kg of clopidogrel or vehicle 3 days, then compressed to 1,600 kPa (150 msw) in 28 seconds, then maintained at 150 msw for 242 seconds and decompressed to surface at 3m/s. In a control experiment, rats were also treated with vehicle 3 days and maintained at atmospheric pressure for an equivalent period of time. Clinical assessment took place over a period of 30 min after surfacing. At the end, blood samples were collected for blood cells counts and cytokine detection. The pathology and the wet/dry ratio of lung tissues, immunohistochemical detection of lung tissue CD41 expression, the numbers of P-selectin positive platelets and platelet-leukocyte conjugates were also been tested. We found Clo significantly reduces the DCS mortality risk (mortality rate: 11/45 with Clo vs 28/46 in the untreated group,  $p < 0.01$ ). Clo reduced the lung injury, the wet/dry ratio of lung, the accumulation of platelet and leukocyte in lung, the fall in platelet count, the WBC count, the numbers of activated platelets and platelet-leukocyte complexes in peripheral blood. It is concluded that Clo can play a protective role in decompression sickness through reducing post-decompression platelet activation and inflammatory process.

# A 13

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## Development Of A Quasi-Physiological Model For The Prediction Of Signs/Symptoms Of Decompression Sickness Following Submarine Tower Escape

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**Introduction:** Predictions of survivability for submariners escaping a distressed submarine (DISSUB) via tower escape may be better informed through the knowledge of rates of different symptoms of decompression sickness (DCS). The objective of this task was to develop robust mathematical models which were capable of making predictions for the rates of four categories of DCS: neurological (CNS), limb pain, respiratory and skin.

**Methods:** These models were adaptations of an existing quasi-physiological model for predicting overall rates of DCS. A set of candidate models were calibrated against DCS data using the Levenberg-Marquardt algorithm from details of 3856 pressure exposures in man, goat, sheep and pigs. Four independent models were created and used to generate iso-risk curves for each of the DCS categories. The bootstrap method was used to generate 95% confidence limits on the models' predictions.

**Results:** The CNS model was the only one markedly affected by DISSUB depth. The CNS and respiratory models were affected by body mass by a greater amount than the limb pain and skin models. Comparison with logistic regression based models shows that the new models are better able to reflect increases in DCS rates that have been observed for small increase in saturation pressure.

**Conclusions:** Use of the models in extrapolation beyond the calibration data could lead to unreliable predictions. It would be inappropriate to use any of the models for designing decompression procedures. Despite showing lack of fit to the calibration data in some areas, these models are the best currently available for prediction of DCS symptoms following submarine tower escape. These models may be used to generate probability of survival estimations to inform advice for a DISSUB scenario.

# A 14

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **Cognitive Impairment Associated with an Inspired Partial Pressure of Oxygen of 1.2 ATA (124kPa) versus 0.21ATA (21kPa) at a Constant Nitrogen Partial Pressure of 4.5 ATA, (456 kPa)**

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**Introduction:** According to the Meyer-Overton theory of anesthetic potency, O<sub>2</sub> should cause cognitive impairment when breathed at hyperbaric pressures. Prior investigators have noted mixed effects; however, this may be because oxygen is a substrate for cellular respiration, a potent vasoconstrictor, and is CNS toxic at 1.6 ATA. Accordingly, a partial pressure below 1.6 ATA may be insufficient to detect a measurable narcotic effect. In this preliminary analysis we tested changes in the MATB-II TRACKING task, a test of cognitive function, at inspired O<sub>2</sub> partial pressures (PiO<sub>2</sub>) of 0.21 and 1.22 ATA (21.3 and 124 kPa) while holding PiN<sub>2</sub> constant at 4.5 ATA (456 kPa).

**Methods:** High PiO<sub>2</sub> stages were compared to “surface-normal” O<sub>2</sub> stages with and without exercise and added CO<sub>2</sub>. MATB-II TRACKING scores from 16 subjects were compared while breathing air (FiO<sub>2</sub>=.21, FiN<sub>2</sub>=.79) at 158 fsw (5.8 ATA, 588 kPa) or a specially-mixed gas (FiO<sub>2</sub>=.044, FiN<sub>2</sub>%=0.95) at 122 fsw (4.7 ATA, 476 kPa) with and without added CO<sub>2</sub> (0.075 ATA, 7.6 kPa) and exercise. Significance was set at p<.05.

**Results:** When PiCO<sub>2</sub> and PiN<sub>2</sub> were held constant the higher PiO<sub>2</sub> was associated with significantly greater mean TRACKING distances in 3 out of 4 categories. There was no difference between the non-exercising high PiCO<sub>2</sub> categories. Six of the 16 subjects experienced unusually severe cognitive impairment while breathing added CO<sub>2</sub> with elevated PiO<sub>2</sub> and PiN<sub>2</sub> at depth.

**Conclusions:** Additional subjects and gas partial pressures should be tested to confirm these preliminary findings. Incipient O<sub>2</sub> toxicity secondary to CO<sub>2</sub> mediated release of protective O<sub>2</sub> cerebral vasoconstriction will need to be distinguished from narcosis.

Acknowledgements: Funding provided by NAVSEA Deep Submergence Biomedical Contract #N0463A-12-C-0001.

# A 15

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **The Enhancement of Cognitive Performance Associated with an Inspired Partial Pressure of Oxygen of 0.925 to 1.0ATA (93.7kPa to 101.3kPa) versus 0.21ATA (21kPa) during Normobaric Trials**

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**Introduction:** In this preliminary analysis we compared CO<sub>2</sub> associated cognitive impairment at inspired O<sub>2</sub> partial pressures (PiO<sub>2</sub>) of 0.21 and 0.927 ATA (21.3 and 93.7 kPa) using the TRACKING task of the MATB-II at normobaric pressures with and without exercise.

**Methods:** MATB-II TRACKING scores from all available subjects (n=28) were compared while breathing air (PiO<sub>2</sub>=0.21ATA, 21.28kPa; PiN<sub>2</sub>=0.79ATA, 80.0kPa), 100% O<sub>2</sub> (PiO<sub>2</sub>=1ATA,101.325kPa), air with added CO<sub>2</sub> (PiO<sub>2</sub>=0.21ATA, 21.28kPa; PiN<sub>2</sub>=0.715ATA, 72.45kPa; PiCO<sub>2</sub>= 0.075ATA, 7.6kPa) or 100% O<sub>2</sub> with added CO<sub>2</sub> (PiO<sub>2</sub>=0.925ATA; PiCO<sub>2</sub>=0.075ATA, 7.6kPa) with and without exercise. Significance was set at p=.05.

**Results:** When compared to stages where subjects breathed either air or 100% O<sub>2</sub>, added CO<sub>2</sub> caused cognitive impairment both at rest and exercise (p<.001 ANOVA with Tukey's HSD post-hoc correction). However, post-hoc analysis showed significantly better performance during stages with higher PiO<sub>2</sub>s. When CO<sub>2</sub> was added a 17 to 31% performance decrement ensued. However, at the higher PiO<sub>2</sub> only a 7-12% decrement was observed.

**Conclusions:** These surface data confirm the findings of Gill and Vann where O<sub>2</sub> appears to counteract CO<sub>2</sub> associated cognitive impairment. Additional subjects and gas partial pressures should be tested to confirm these preliminary findings.

**Acknowledgements:** Funding provided by NAVSEA Deep Submergence Biomedical Contract #N0463A-12-C-0001.



# A 16

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## Reconciliation of the Wienke RGBM & the Strauss GP Models for DCS

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**Introduction:** In 2002, Wienke described his Reduced Gradient Bubble Model (RPBM) for preventing the occurrence of decompression sickness (DCS) and as explanation for the cause of bubble formation. His algorithms which were sophisticated computer upgrades of Haldane's tissue half times-compartment approach are used in many dive computers. Nonetheless about 50% of DCS cases occur without the divers' computers showing decompression violations. Because of this, we approach the bubbling phenomenon of DCS from a clinical perspective and postulate a Gradient-Perfusion explanation for why DCS occurs.

**Methods:** Accumulated knowledge about DCS has demonstrated that, during off-gassing, inert gas bubbles are invariably carried by the blood circulation to the lungs where they are harmlessly exhaled. For the majority of compressed gas diving, no problems arise, and they are rightfully labeled "silent" bubbles. From our experiences in evaluating events that interfered with off-gassing and led to DCS, i.e. "Disordered Decompression", the Gradient-Perfusion (GP) explanation has explained why the hit occurred.

**Results:** Perfusion is the limiting factor for off-gassing. Whereas, a human has approximately 5 liters of circulating blood, the capacity of the vascular tree is at least 20 times greater. This means that blood flow has to be carefully regulated for orderly off-gassing. When pressure gradients exceed the supersaturation limits of the tissues, autochthonous bubble formation occurs. Without adequate perfusion to deliver the bubbles to the lungs, they coalesce, enlarge and generate symptoms of DCS.

**Conclusions:** Whereas the RGPM model helps to prevent DCS, our GP explanation explains why DCS occurs and when to allow a diver to resume diving after a DCS event. The RPBM and GP theories therefore, complement each other.

# A 17

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **Disordered Decompression and Undeserved Decompression Sickness**

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**Introduction:** About 50% of decompression sickness (DCS) incidents occur without apparent dive computer or table violations. Analysis of the events associated with these apparently underserved cases of DCS reveals disordering events as the causes.

**Methods:** After nearly 500 cases of DCS treated at our facility, a cohort of patients who gets “bent” or have symptoms totally out of proportion to their inert gas loads have emerged. In each situation, an event that caused disordered perfusion for off-gassing could be identified.

**Results:** Our perfusion-gradient model for DCS accounted for the observed pathology and the clinical courses of the patients who had undeserved decompression events. Without precise sympathetic nervous system control of the five liter blood volume to the greater than 100 liter vascular system capacity, off-gassing of critical, fast tissues such as the brain, lungs, and heart results in “steal” syndromes. The associated perfusion component coupled with the gradient insult can cause neurological impairments, unexplained DCS presentations, and even deaths. The presentation presents five cases where disordered perfusion caused DCS presentations ranging from death to transient paralysis.

**Conclusions:** When DCS occurs with confusing presentations or in the absence of dive table and/or computer violations, disordering events should be considered, in order to optimize management and make decisions about return to diving.

# A 18

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **PDTC Ameliorates the Decompression Induced-Lung Injury Caused by Fast Buoyancy Ascent Escape Via Inhibition of NF- $\kappa$ B pathway**

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**Objective:** To study the roles of NF- $\kappa$ B and TNF- $\alpha$  (the critical proinflammatory factor) in the process of the rat lung injury of DCS caused by simulated fast buoyancy ascent escape unsafely.

**Methods:** Administration of NF- $\kappa$ B inhibitor, pyrrolidine dithiocarbamate (PDTC) and TNF- $\alpha$  antibody (Ab) before fast buoyancy ascent escape, and then researchers watched the rats' breath and activity (including fluctuation of the rat's ventrum, creeping and groveling) when decompression was accomplished, and recorded the number of deaths in each group. 0.5h later the survival rats were anesthetized, and taken out of lung for histopathological examination and detection of the rat lung tissue messenger ribonucleic acid (mRNA) and protein level variations of NF- $\kappa$ B, inhibitory  $\kappa$ B (I $\kappa$ B), TNF- $\alpha$ , IL-1 $\beta$ , IL-6, IL-10 and IL-13.

**Results:** PDTC could improve the survival rate of the rat of DCS caused by fast buoyancy ascent escape unsafely more effectively than TNF- $\alpha$  Ab, although the inhibition of TNF- $\alpha$  Ab on the nuclear translocated protein expression of NF- $\kappa$ B was more effective than PDTC. PDTC and TNF- $\alpha$  Ab could both abrogate the increment of the rat lung tissue mRNA levels of TNF- $\alpha$ , IL-1 $\beta$ , IL-6 and protein levels of NF- $\kappa$ B, TNF- $\alpha$ , IL-1 $\beta$  effectively and increase the rat lung tissue content of I $\kappa$ B significantly. **Conclusions:** TNF- $\alpha$ -mediated NF- $\kappa$ B signaling may be one of the critical signaling pathways in the pathogenesis of the rat lung injury of DCS caused by simulated fast buoyancy ascent escape unsafely. PDTC may ameliorate the rat lung injury of DCS caused by fast buoyancy ascent escape unsafely partly through inhibiting NF- $\kappa$ B pathway.

# A 19

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION: YES

## The Case for Mixed Pharmacokinetic Models as a Descriptor of Decompression Sickness

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**Introduction/Background:** We have previously shown that probabilistic models of decompression sickness (DCS), which typically rely upon a series of parallel perfusion-limited compartments, describing gas content within theoretical tissues do not always accurately predict the probability of DCS in divers [1]. Our conclusions were consistent with Doolette, Upton, and Grant's experimental pharmacokinetics work showing that a diffusion component was needed to accurately describe the gas uptake and washout in sheep [2,3]. We further found that a diffusion component did not unilaterally improve model agreement for human data, but did so for certain types of dive data (for example, air dives versus non-air dives).

**Materials and Methods:** Numerous pharmacokinetic gas content models, some previously proposed by Doolette et al. and some new, were programmed in C# and fitted to the NMRI98 dive data set [4] by the method of maximum likelihood. Fitting was carried using the Nelder-Mead Algorithm. In fitting the models, marginal DCS events were considered to be non-events [5].

**Results:** Using Akaike Information Criterion, we found that air dives were best described by a compartmental model containing a diffusion component. Repetitive dives conducted on breathing gases other than air were best described by a serial model structure without diffusion. Further, we found additional evidence that saturation dive data are likely not combinable with bounce dive data indicating that a mixture of models containing both diffusion and non-diffusion compartments might be a better model of diver physiology.

[1] Murphy, Hada, Doolette, Howle LE, UHM-ASM 2014; 2

[2] Doolette, Upton, Grant, Journal of Applied Physiology 2005; 563.2:529-539

[3] Doolette, Upton, Grant, Acta Physiologica Scandinavica 2005; 185:109-121

[4] Parker, Survanshi, Massell, Weathersby, Journal of Applied Physiology 1998; 84.3:1096-1102

[5] Howle, Weber, Vann, Campbell, Journal of Applied Physiology 2009; 107.3:1539-2547

Supported by ONR Grant #N00014-13-1-0063, NAVSEA Contract # N00024-13-C-4104.

# A 20

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## Delay Differential Equations As An Explicit Method Of Aligning DCS Model Prediction With Dive Trial Outcome

Hada EA<sup>1</sup>, Murphy FG<sup>1,3</sup>, Howle LE<sup>1,2</sup>

<sup>1</sup>Mechanical Engineering and Materials Science Department, Duke University, Durham, NC;

<sup>2</sup>BelleQuant Engineering, PLLC, Mebane, NC

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**Introduction:** The onset of symptoms associated with decompression sickness (DCS) are often delayed relative to the final surface time of the dive profile. This observation motivated the development of the Linear-Exponential (LE) probabilistic decompression model [1] and bubble dynamics models [2]. These models improve log-likelihood fit for time-windowed DCS events by implicitly shifting risk accumulation toward the event window. Existing pharmacokinetic models [3] can be augmented using delay differential equations (DDE) which explicitly adjust the temporal position of risk accumulation.

**Methods:** DDE models are programmed using C# and best fit parameters are obtained using a variety of algorithms including Nelder-Mead and Simulated Annealing. DCS onset time windowing is used in fitting the models to standard a Navy dive data set. Model performance and model selection is evaluated using classical statistics and information theory.

**Results:** Exploration of simple delay models has shown the ability to shift position of model-generated risk probability and improve the model's fit to the data. We report on model performance for a variety of pharmacokinetic delay differential equation models.

1. Thalmann, E.D., et al. *Improved probabilistic decompression model risk predictions using linear-exponential kinetics*. Undersea & Hyperbaric Medicine. **24**(4): p. 255-274.
2. Gerth, W.A. and R.D. Vann, *Probabilistic gas and bubble dynamics models of decompression sickness occurrence in air and nitrogen-oxygen diving*. Undersea & Hyperbaric Medicine, 1997. **24**(4): p. 275-292.
3. Murphy, F.G., E.A. Hada, D.J. Doolette, and L.E. Howle, *Perfusion-diffusion gas content compartmental models as a predictor of decompression sickness*. 2014 UHMS Annual Scientific Meeting.

Supported by NAVSEA Contract # N00024-13-C-4104 and ONR Grant #N00014-13-1-0063.

# A 114

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## Structural Magnetic Resonance Imaging Change Associated With Repetitive Hypobaric Non-Hypoxic Exposure

McGuire S<sup>1,3</sup>, Sherman P<sup>1</sup>, Kochunov P<sup>2</sup>

<sup>1</sup>U.S. Air Force School of Aerospace Medicine, Aerospace Medicine Consultation Division, Wright-Patterson AFB, OH; <sup>2</sup>Department of Psychiatry, University of Maryland School of Medicine, Baltimore; <sup>3</sup>Department of Neurology, Lackland AFB, TX  
dr.stephen.mcguire@gmail.com

**Introduction/Background:** We previously performed high resolution magnetic resonance imaging (MRI) on 106 U-2 pilots (U2P) and 162 doctorate controls (DOC), demonstrating increased subcortical white matter hyperintensity (WMH) burden and decreased neurocognitive test performance associated with normoxic hypobaric exposure ( $p < 0.001$ ). We postulated that subcortical white matter injury associated with repeated exposure to hypobaric non-hypoxic environments will be associated with other permanent MRI change.

**Materials and Methods:** Subjects underwent high resolution MRI with quantification of findings. We compared diffusion tensor imaging (fractional anisotropy) and cortical mapping to WMH burden and to exposure. Statistical analysis was performed utilizing the two-tailed parametric t-test.

**Results:** Average fractional anisotropy (FA) was significantly decreased in U2P ( $p = 0.004$ ) after correcting for MRI scanner differences. No significant difference was detected in mean cortical thickness ( $p = 0.341$ ). No significant correlation between WMH burden and FA was detected. No correlation between total hours of hypobaric exposure and FA change was detected.

**Summary/Conclusions:** Repetitive normoxic hypobaric exposure to 28-30k feet (4.73-4.37 psi) is associated with a reduction in average FA but not in mean cortical mantle thickness. We previously reported an increase in WMH burden with an associated decrease in neurocognitive test performance. The lack of correlation between WMH burden and FA suggests a more diffuse process of injury is occurring and that this may also be a factor in the neurocognitive test performance. The pathophysiology behind this cerebral white matter injury is unknown but does not appear to be a simple gaseous embolic phenomenon.

# A 115

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1130 - 1200

RESIDENT COMPETITION:

## **Transient magnetic resonance imaging change associated with a single episode of hypobaric non-hypoxic exposure – preliminary results**

McGuire S<sup>1,3</sup>, Sherman P<sup>1</sup>, Thom S<sup>2</sup>, Kochunov P<sup>2</sup>

<sup>1</sup>U.S. Air Force School of Aerospace Medicine, Aerospace Medicine Consultation Division, Wright-Patterson AFB, OH; <sup>2</sup>University of Maryland School of Medicine, Baltimore;

<sup>3</sup>Department of Neurology, Lackland AFB, TX

dr.stephen.mcguire@gmail.com

**Introduction/Background:** We previously reported increased subcortical white matter burden associated with repetitive occupational exposure to non-hypoxic hypobaric conditions. We hypothesized that a single hypobaric hypoxic occupational exposure would induce transient magnetic resonance imaging (MRI) changes that would revert to baseline after 72 hours.

**Materials and Methods:** Subjects underwent high resolution MRIs immediately prior to exposure, immediately after exposure, and 72 hours after exposure with quantification of findings. Each subject served as his own control. No subject experienced decompression sickness symptoms. To date 22 subjects have been evaluated during standard U.S. Air Force aircrew occupational training altitude chamber exposure to 25k feet (5.45 psi). Statistical analysis was performed utilizing parametric methodologies.

**Results:** Preliminary findings demonstrate that at 24 hours after exposure white matter cerebral blood flow increases by 5%, returning to baseline at 72 hours; a smaller increase in gray matter cerebral blood flow also occurs (3%). A corresponding increase in glutamate and glutamine activity also occurs at 24 hours. Additionally Q-space analyses suggest an increase in extra-axonal water content.

**Summary/Conclusions:** Preliminary results suggest transient cerebral injury associated with a routine occupational aircrew altitude chamber exposure is occurring with an increase in metabolic activity, increase in cerebral blood flow, and increase in extra-axonal water content with return to baseline at 72 hours. The mechanism behind this change is currently unknown but would suggest a diffuse process without corresponding clinical symptoms.

## CHRISTIAN LAMBERTSEN MEMORIAL LECTURE

THURSDAY, JUNE 18TH: 1:15pm to 2:15pm

Guest Speaker: Alfred Bove, MD

### "Lung Injury with Diving: Beyond Boyle's Law"



Although recent clinical experience has brought the problem of pulmonary edema related to diving and swimming to the fore, the reports of lung injury with immersion go back at least 50 years. The descriptions of Immersion Pulmonary Edema, Swimming Induced pulmonary edema, Exercise Induced Pulmonary Edema and Negative Pressure Pulmonary Edema raise questions about a common mechanism that results in exudation of fluid and blood from the pulmonary capillaries into the alveoli. On further analysis, one finds common pathophysiologic mechanisms for all of these clinical entities, and suggestions for amelioration of the process can be based on these mechanisms. While

Boyle's law does not contribute to the process of pulmonary edema in these clinical syndromes, a similar clinical syndrome can be found in competitive deep breath hold divers where Boyle's law does contribute to the development of pulmonary edema.

#### **About Dr. Bove:**

Dr. Alfred Bove is the former Chief of Cardiology at Temple University Medical Center and Emeritus Professor of Medicine at Temple University Medical School. He was president of the American College of Cardiology in 2009 and served on their Board of Trustees for 11 years. He was president of the Pennsylvania Chapter American College of Cardiology from 1989 to 1991, and President of the Undersea Medical Society in 1983. Dr. Bove was an established Investigator of the American Heart Association, and has published over 300 original research papers on coronary disease, valvular heart disease, cardiac hypertrophy, exercise medicine and physiology, coronary prevention, environmental medicine, and computers in medicine. He has authored texts on coronary disease, exercise medicine and diving medicine. He has conducted research in basic cardiac physiology, coronary disease, exercise physiology, environmental medicine and computer information systems in medicine. His current research involves population-based management of heart failure and cardiovascular risk factors using an Internet communication system.

Dr. Bove served as Editor in Chief of the American College of Cardiology educational web site: [www.Cardiosource.com](http://www.Cardiosource.com) from 2002-2007 and is present editor in chief of ACCEL, the ACC audio journal, and the ACC news journal Cardiosource World News. He is an expert in web site design and medical database development. Dr. Bove directs the Temple University Underwater Medicine program, which has provided postgraduate training in environmental medicine to over 400 physicians over the 41year tenure of the program. He was a team physician for the Philadelphia 76ers from 1987 to 2008, and is one of the founding members of the American College of Cardiology Council on Sport and Exercise. He is a member of the exam committee of the American Board of Preventive Medicine for Undersea and Hyperbaric Medicine, and the Information Technology task force of the American College of Cardiology. He is the Program Director for the Cardiology Fellowship at Temple University Hospital.

Dr. Bove maintains an active clinical practice in Cardiology, Sports Medicine and Diving Medicine. Dr. Bove received his bachelor's degree in Electrical Engineering from Drexel University in 1962, and received the MD and PhD (Physiology) degrees from Temple University Medical School in 1966 and 1970. After a medical internship and residency at Temple Hospital, and a post-doctoral fellowship at Temple and the Mayo Clinic, he served two active duty years in the U.S. Navy as an Undersea Medical



Officer, and then joined the staff in Cardiology at Temple in 1973. In 1981, he joined the staff of the Mayo Clinic in the Division of Cardiology, and returned to Temple as the section chief in Cardiology in 1986. He continued as a Naval Reserve officer, including active duty in Operation Desert Storm, and retired from the Navy in 1998 after 33 years. He has received awards and honors from the Undersea and Hyperbaric Medical Society, the Association of Military Surgeons of the United States, Temple University School of Medicine, Drexel University, The Southeastern Pennsylvania Affiliate of the American Heart Association, and West Philadelphia Catholic Preparatory High School. Dr. Bove is a member of the Institute of Electrical and Electronic Engineers, a Master of the American College of Cardiology, a Fellow of the American College of Physicians, a Fellow of the American Heart Association and a Fellow of the Undersea and Hyperbaric Medical Society. He has been named as one of America's Top Physicians from 2006 to 2014.

Dr. Bove is married to the former Sandra Seltzer. They have two living children, Jacqueline Graeber and Andrew Bove, and three grandchildren. Their third child, Christopher died in 2007. 2/26/15

### **CHRISTIAN J. LAMBERTSEN, MD, DSc (Hon)**



Dr. Christian J. Lambertsen received a B.S. Degree from Rutgers University in 1938 and a M.D. Degree from the University of Pennsylvania in 1943. During his medical school period, he invented and first used forms of the initial U.S. self-contained closed circuit oxygen rebreathing apparatus, for neutral buoyancy underwater swimming and diving. As a student, he aided the early Office of Strategic Services (O.S.S.) in establishing the first cadres of U.S. military operational combat swimmers. Dr. Lambertsen became a U.S. Army medical officer on graduation from medical school in early 1943, and immediately joined the O.S.S. Maritime Unit on active duty through its period of function in World War II. He joined the University of Pennsylvania Medical Faculty in 1946, and became Professor of Pharmacology in 1952. While a faculty member he combined diving research and further underwater rebreathing equipment developments for the Army and Navy. In 1967 he served as Founding President of the Undersea Medical Society (now Undersea and Hyperbaric Medical Society.) Dr. Lambertsen is recognized by the Naval Special Warfare community as "The Father of U.S. Combat Swimming." His hand has touched every aspect of military and commercial diving. Dr. Lambertsen's active contributions to diving began during WWII and became even more progressive in the post-war period through the evolutions of the U.S. Navy Deep Submergence and Naval Special Warfare developmental programs.

## **SESSION B**

# **HBO<sub>2</sub> THERAPY MECHANISMS**

**Moderators: Mike Bennett, MD & John Feldmeier, DO**

**THURSDAY, JUNE 18**

**2:15PM – 4:45PM**

## B 21

ORAL PRESENTATION TIME: 1415 - 1427

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### Effects Of Hyperbaric Oxygen Treatment On Muscle Disuse Atrophy

Horie M<sup>1,2</sup>, Enomoto M<sup>1,3</sup>, Oyaizu T<sup>1</sup>, Yagishita K<sup>1,3</sup>

<sup>1</sup>Hyperbaric Medical Center, University Hospital of Medicine, Tokyo Medical and Dental University; <sup>2</sup>Research Fellow of Japan Society for the Promotion of Science (University of Tsukuba); <sup>3</sup>Clinical Center for Sports Medicine and Sports Dentistry, Tokyo Medical and Dental University

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**Objective:** Hyperbaric oxygen (HBO) treatment is widely applied for sports-related soft tissue injuries for the purpose of rapid recovery. However, the effects of HBO treatments on muscle disuse atrophy (ex. Gibbs fixing and long-term bed rest) are unknown. Therefore, we tested whether HBO treatment would prevent muscle disuse atrophy of hind limbs in a rat experimental model.

**Methods:** In this study, we used 7-week-old Wistar rats. Muscle disuse atrophy was induced by tail suspension (TS) for 12 days. The animals were divided into 2 groups: TS+non-treatment (NT) group and TS+HBO group. For the HBO treatment, the rats were placed in an animal HBO chamber with 100% oxygen under 2.5 ATA for 2 h/day, 5 days/week. Over the TS treatment, we sampled soleus muscles and measured their weights in each rat. We performed histological analyses and quantitative RT-PCR for muscle atrophy factors.

**Results:** Muscle wet weight and cross-sectional area of muscle fiber were significantly decreased by TS in the NT and HBO groups. In the HBO group, the cross-sectional area of fast-twitch muscle fiber (type II muscle fiber) was significantly smaller than that in the NT group 12 days after TS. Next, we observed mRNA expressions of muscle atrophy factors (MuRF1, Atrogin1, FOXO, IGF-1). In both groups, MuRF1 mRNA expression was increased 12 days after TS. However, Atrogin1 mRNA expression was significantly decreased 12 days after TS in the HBO group. Other factors showed no differences of the mRNA expressions between the groups.

**Conclusion:** In this study, we showed that HBO treatment facilitated atrophy of type II muscle fiber and affected the Atrogin1 mRNA expression in soleus muscle. These results suggest that HBO treatment may exacerbate muscle disuse atrophy. Therefore, it is necessary to consider HBO application for muscle atrophy following immobilization after sports injury.

## B 22

ORAL PRESENTATION TIME: 1427 - 1439

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### **HBOT Suppresses Renal Injury in a Rodent Model of Diabetes Mellitus**

Perdrizet G<sup>1,2</sup>, Verma R<sup>1</sup>, Chopra A<sup>1</sup>, Giardina C<sup>1</sup>, Sabbisetti V<sup>1</sup>, Smyth JA<sup>1</sup>, Hightower LE<sup>1</sup>

<sup>1</sup>Dept. of Molecular and Cell Biology, University of Connecticut, Storrs, CT; <sup>2</sup>Emergency

Medicine and Hyperbaric Medicine, UCSD, San Diego, CA

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**Background** Diabetic kidney disease (DKD) is the leading cause of chronic kidney disease in developed nations. There are no therapies that can prevent DKD. Management relies on metabolic (blood sugar and lipid therapies) and blood pressure control. Despite these measures, DKD develops in 33% of individuals with diabetes mellitus. Systemic hyperglycemia drives the development of diabetic complications (neuropathy, retinopathy, nephropathy, myocardial and peripheral vascular disease). All diabetic complications are linked to damage of microvascular endothelium. New findings demonstrate involvement of acute inflammation and oxidative stress in early and progressive DKD. HBOT can reduce acute inflammation and enhance anti-oxidant defenses in a wide array of experimental models. Clinical studies repeatedly document HBOT's ability to restore microvascular health in the setting of advanced diabetic foot ulceration. We hypothesized that HBOT will attenuate renal damage in a rodent model of DKD.

**Methods:** Thirty db/db mice (males, 5 weeks of age) were distributed into three treatment groups: 1) 2.4 ATA, 2) 1.5 ATA, and 3) sham-HBOT controls. HBOT was administered for 1 hour per day, 4 days per week for 20 weeks. Blood, urine and renal tissue were collected for biomarker analysis (NGAL, clusterin, cystatin-C), renal function (creatinine, albuminuria), gene expression (Nrf-2, HSPA1A, Mt1, Hmox1) and renal histology (hemotoxylin and eosin and caspase-3 activity). One-way ANOVA with Fisher's LSD post hoc test was used to compare groups. Protocol was approved by institutional animal care and use committee at Univ. of Conn.

**Results:** HBOT significantly reduced urinary biomarkers of renal injury- NGAL and Cystatin-C, reduced expression of stress response genes in peripheral blood and caspase-3 activity within renal tissues. A significant reduction in albuminuria was seen for the 2.4 ATA group compared to sham controls.

**Summary:** HBOT reduces DKD in the db/db model of diabetes mellitus.

**References:** 1) García-García PM et al. Inflammation in diabetic kidney disease. *World J Diabetes*. 2014;5(4):431. 2) Shahzad K et al. Nlrp3-inflammasome activation in non-myeloid-derived cells aggravates diabetic nephropathy. *Kidney Int*. 2015;87(1):74. 3) Godman CA et al. Hyperbaric oxygen treatment induces antioxidant gene expression. *Ann N Y Acad Sci*. 2010;1197:178-83. 4) Verma R, et al. Hyperbaric oxygen therapy (HBOT) suppresses biomarkers of cell stress and kidney injury in diabetic mice. *Cell Stress & Chaperones*. 2015 Feb 4. [Epub ahead of print] PMID:25648080.

**Acknowledgments:** Funding source- OxyHeal Corp., San Diego, CA.

## B 23

ORAL PRESENTATION TIME: 1439 - 1451

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### Critical Events In CNS O<sub>2</sub> Toxicity And Novel Approaches For Delaying Oxygen Seizures

Demchenko IT, Gasier HG, Allen BW, Piantadosi CA

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**Background:** CNS O<sub>2</sub> toxicity develops in a progression of events: hyperemia supplants an initial protective cerebral vasoconstriction; antioxidant defenses are overwhelmed; and excessive ROS/RNS production, combined with impaired afferent restraint of CNS excitability, triggers abnormal neuronal firing. We are testing a novel approach to alter this progression using a rational selection of antiepileptic drugs (AEDs) and FDA-approved vasoactive agents.

**Methods:** Conscious or anesthetized S-D rats were exposed to O<sub>2</sub> at 5 ATA after IP administration of either the Na-channel blocker Carbamazepine (CBZ), the GABA transmission enhancer Vigabatrin (VGB), the beta-adrenergic blocker Propranolol (PPL), the prostaglandin inhibitor Indomethacin (INM), or the ROS scavenger Ascorbic acid (ASA). Protective effects were assessed by comparing the time to onset of EEG spikes or motor convulsions with that seen in saline treated control animals. Lung injury, hemodynamics, and ECG were measured. Sympatho-vagal imbalance, baroreceptor sensitivity, and indices of left ventricular function were computed.

**Results:**(i) In control rats, rising sympathetic drive peaks after 40-50 min of HBO<sub>2</sub>, leading to baroreflex impairment, increases in systemic BP, seizures, cardiac dysfunction and acute pulmonary hypertension. (ii) Each pharmacological agent used here delayed sympathetic activation and seizures in a different manner. PPL and INM were the most effective, increasing mean convulsion latency more than fivefold compared to untreated animals; and were associated with significant decreases in cerebral blood flow. CBZ and VGB provided a twofold increase in seizure latency without decreasing cerebral blood flow; whereas VPA and ASA were less effective.

**Conclusions:** (i) Some FDA-approved drugs can significantly delay seizures in HBO<sub>2</sub>; (ii) Based on known mechanisms of action, these drugs can be useful to investigate mechanisms of CNS O<sub>2</sub> toxicity; (iii) Drugs used clinically to treat epilepsy and other excitotoxic conditions may offer brief functional neuronal protection to those exposed to toxic hyperoxic environments.

*Supported by the Office of Naval Research.*

## B 24

ORAL PRESENTATION TIME: 1451 - 1503

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### Effects Of Hyperbaric Oxygen On Nitric Oxide Generation In Humans

Uusijärvi J <sup>1,2</sup>, Eriksson K <sup>1,2</sup>, Larsson AC <sup>1</sup>, Nihlén C<sup>1</sup>, Schiffer S<sup>1</sup>, Lindholm P<sup>1,3</sup>, Weitzberg E<sup>1,2</sup>

<sup>1</sup>Dept. of Physiology and Pharmacology, Karolinska Institutet, <sup>2</sup>Dept. of Anesthesia & Intensive Care, Karolinska University Hospital, <sup>3</sup>Dept. of Radiology, Karolinska University Hospital, Stockholm, Sweden

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**Background:** Hyperbaric oxygen (HBO<sub>2</sub>) has been suggested to affect nitric oxide (NO) generation in humans. NO is produced by NO synthases (NOSs) from L-arginine and molecular oxygen, and may also be formed by reduction of the inorganic anions nitrate and nitrite. Interestingly, oral facultative anaerobic bacteria are necessary for the first step to reduce nitrate to nitrite. The nitrate-nitrite-NO pathway is potentiated by hypoxia and low pH in contrast to classical NOS-dependent NO generation. We investigated the effects of HBO<sub>2</sub> on NO generation in healthy subjects including orally and nasally exhaled NO, plasma, salivary nitrate and nitrite as well as plasma cGMP and citrulline/arginine ratio. We also conducted in-vitro experiments to investigate the effects of hyperoxia on nitrate/nitrite metabolism and NO generation by oral bacteria.

**Methods:** Two HBO<sub>2</sub> experiments were performed. In a cross-over experiment (EXP1) subjects breathed air at 130 kPa (control) or oxygen at 250 kPa for 100 minutes and parameters were measured before and after exposure. In experiment 2 (EXP 2) measurements were performed also during HBO<sub>2</sub> at 250 kPa for 110 minutes.

**Results:** HBO<sub>2</sub> acutely reduced orally and nasally exhaled NO by 30 % and 16%, respectively. There was a decrease in salivary nitrite/nitrate ratio during and after HBO<sub>2</sub> indicating a reduced bacterial conversion of nitrate to nitrite and NO, also supported by in vitro experiments with oral bacteria showing that hyperoxia inhibited bacterial nitrate and nitrite reduction leading to a reduced NO generation. Plasma nitrate was unaffected by HBO<sub>2</sub> while plasma nitrite was reduced during HBO<sub>2</sub>. In contrast, plasma cGMP increased during HBO<sub>2</sub> as did citrulline/arginine ratio after treatment and control.

**Conclusion:** HBO<sub>2</sub>-exposure in humans affects NO generation in the airways and systemically differently. These data suggests that the individual NOSs as well as the nitrate-nitrite-NO pathway do not respond in a similar way to HBO<sub>2</sub>.

## B 25

ORAL PRESENTATION TIME: 1503 - 1515

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### **Inert Gas Chemotherapy**

Thom SR<sup>1</sup>, Ma MZ<sup>2</sup>, Bhopale V<sup>1</sup>, Yang M<sup>1</sup>, Mao L<sup>2</sup>

<sup>1</sup>University of Maryland School of Medicine Dept. of Emergency Medicine, <sup>2</sup>University of Maryland School of Dentistry Department of Oncology and Diagnostic Sciences Baltimore, MD  
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**Introduction:** Reactive oxygen species such as hydrogen peroxide at high concentrations are conventionally thought of as cytotoxic. Singlet O<sub>2</sub> is a potent anti-neoplastic agent but current technology limits its delivery because of the need for light penetration. However, collision complexes that occur between O<sub>2</sub> and inert gases within the membranes of cells under high gas pressure leads to singlet O<sub>2</sub> production (J Biol Chem 289: 18831, 2014). We hypothesized that normobaric high pressure nitrogen would inhibit tumor growth in a murine model.

**Materials and Methods:** Small islands of human non-small cell carcinoma or cultures A549 cells were placed subcutaneously in nude mice and allowed to grow into a palpable mass. Mice were then randomly divided into sham and experimental groups for exposure in a hyperbaric chamber at just ambient pressure (sham) or for a total of 50 minutes to 3.78 ATA of 5.6% O<sub>2</sub>/balance N<sub>2</sub> followed by staged decompression based on the 100 fsw US Navy air decompression table. Treatments were done daily 5 days/week for 3 weeks. Tumor size was monitored twice weekly and tissue fixed for histochemical analysis after mice were euthanized.

**Results:** Pressure exposures profoundly inhibited tumor growth, with inhibition greatest when initial tumor volume was 50 mm<sup>3</sup> or less. There were modest differences in gas sensitivity among three types of cancer cells – laboratory cultured A549 and two types of tumors harvested from human patients. Histological analysis documented extensive tumor cell death in the high pressure groups.

**Summary:** This pilot study suggests that intermittent exposures to normoxic high inert gas pressures will impede tumor growth and may have synergistic tumoricidal action in combination with some traditional chemotherapeutic agents.

## B 26

ORAL PRESENTATION TIME: 1515 - 1527

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### **Pre-operative Stress Conditioning: Is Oxygen the Drug of Choice?**

Perdrizet G<sup>1,2</sup>, Giradina C<sup>1</sup>, Hightower L<sup>1</sup>

<sup>1</sup>Dept. of Molecular and Cell Biology, University of Connecticut, Storrs, CT; <sup>2</sup>Emergency Medicine and Hyperbaric Medicine, UCSD, San Diego, CA  
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**Background:** Complications following major surgery are common and costly. Myocardial infarction, stroke, acute spinal ischemia and acute renal failure can follow in the wake of successful surgery. No clinical protocols exist to actively condition patients prior to high risk surgical interventions. Effective preconditioning has been repeatedly demonstrated in animal models for more than a quarter century, where brief exposure to hyperthermia (heat shock), ischemia, hypoxia or endotoxin confers transient protection against acute ischemia-reperfusion injury and is associated with new gene expression typical of a cell stress response (CSR). Development of a clinically safe and effective method of pre-stress conditioning (PSC) in humans is the ultimate goal of this work. We aim to use high dose oxygen to trigger a CSR and actively PSC human microvascular endothelial cells (HMEC-1) as proof of concept.

**Methods:** HMEC-1 cells exposed to 1.0 or 2.4 ATA oxygen for sixty minutes were recovered for one or 24 hours as a PSC algorithm. Cells were harvested and RNA extracted to perform a genome-wide microarray analysis. Gene expression was confirmed by quantitative PCR of selected genes. Cell survival following in vitro exposure to a strong oxidizing agent was also tested as a measure of cellular protection.

**Results:** Over 8,000 genes demonstrated significantly altered levels of expression. Pathway analysis identified Nrf-2 as a primary responder. Validation of several top performing genes was confirmed using quantitative PCR (e.g., Nrf2, HMOX1, HSPA1A). Exposed HMEC-1 cells develop significantly increased resistance to oxidant stress in vitro (t-butyl-hydroperoxide challenge, 0.05-0.24mM x 4hrs).

**Conclusion:** The molecular changes described here, together with our understanding of the CSR and PSC suggests high dose oxygen (2.4 ATA for 60 minutes) may be the drug of choice for clinical preconditioning protocols and should be systematically tested.

**Reference:** 1. Perdrizet, GA, et al. Preoperative Stress Conditioning Prevents Paralysis Following Experimental Aortic Surgery. *J.Thor.CV Surg.* 2002;124:162. 2. Godman CA, et al. Hyperbaric oxygen induces a cytoprotective and angiogenic response in human microvascular endothelial cells. *CS&C* 2010;15:431. 3. Godman CA, et al. Hyperbaric Oxygen Treatment Induces Antioxidant Gene Expression, *NY Acad Sci* 2010;1197:178.

**Acknowledgments:** Funding source- OxyHeal Corp., San Diego, CA.



## B 27

ORAL PRESENTATION TIME: 1527 - 1539

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### **Signalling Pathways In Hyperbaric Oxygen-Induced HSP32 Expression In Primary Cultured Rat Spinal Neurons**

Huang G, Zhang K, Weigang Xu

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**Introduction/Background:** Our previous study found that HBO preconditioning significantly protected the primary cultured rat spinal neurons via HSP32 against oxidative insult and oxygen glucose deprivation, which are two pivotal mechanisms involved in the physiopathology of decompression sickness spinal cord injury. In this study, the molecular mechanisms were further investigated.

**Materials and Methods:** After HBO exposure, the production or expression of ROS, NO and the related key signaling molecules were determined by fluorescent probe, protein microarray and western blotting in primary cultured spinal neurons. The participation and sequence of ROS, NO and activated signaling molecules in the signaling cascade from HBO to HSP32 were evaluated using specific inhibitors or gene knockdown.

**Results:** A single exposure of HBO (280 kPa, 60 min) significantly increased levels of intracellular ROS and NO and activated ERK1/2, p38MAPK, CREB and Nrf2. The expression of HSP32 by HBO was significantly reversed by pretreatment with ROS scavenger NAC, p38MAPK inhibitor SB203580 or Nrf2 knockdown, enhanced by ERK1/2 inhibitor U0126. Pretreatment with NAC significantly inhibited the activation of ERK1/2, p38 MAPK, and Nrf2. SB203580 inhibited the activation of Nrf2.

**Conclusion:** HBO induces HSP32 expression through the ROS/p38MAPK/Nrf2 pathway in primary cultured rat spinal neurons, and ERK1/2 pathway may contribute to a negative regulating mechanism. Further investigation is in progress.

## B 28

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### **Acceleration Of Muscle Volume Reduction And Recovery From Hypoxia Of Injured Skeletal Muscle By Hyperbaric Oxygen**

Oyaizu T, Enomoto M, Horie M, Yagishita K

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**Introduction:** Hyperbaric oxygen (HBO) treatment is used to promote rapid recovery of sports-related soft tissue injuries. We have reported that HBO accelerated myofiber maturation in a drug-induced skeletal muscle injury model. However, drug-induced injury does not replicate clinical contusion injury which often occurs in contact sports. In this study, we investigated whether HBO reduced calf muscle volume and elevated oxygen tension in injured muscle in rats using the drop mass method.

**Materials and Methods:** A muscle contusion was performed at the calf by the drop mass method. Ten-week-old male Wistar rats were divided into 2 groups: non-treatment (NT) and HBO group. The HBO protocol consisted of 2.5 atmospheres absolute pressure (2.53MPa), 100% oxygen, for 120 minutes in an experimental HBO chamber. Injured calf muscle volumes were measured sequentially by micro-computed tomography (CT) before injury and 6hours, 24hours, 3days, 5days, and 7days after the injury. HBO was performed once per day for 5 days. Oxygen tension at the injured calf was measured with a needle-type oxygen sensor before injury and 30mins, 3hours, 6hours, 24hours and 30hours following HBO treatment.

**Results:** In the NT group, calf volume significantly increased for 3days after injury, whereas calf volume increased for only 24hours following injury in the HBO group. At 24hours ( $p=0.002$ ) and 3days ( $p=0.04$ ), injured calf volumes were significantly lower in the HBO group compared to the NT group. Oxygen tension of the contused muscle decreased in both groups, from 45mmHg to 15mmHg. The NT group required 30 hours to recover to pre-contusion levels, whereas the HBO group required only 3hours for recovery and maintained for 30hours.

**Conclusions:** The current findings demonstrate that HBO treatment accelerates recovery of contused skeletal muscle in rats. Hyperbaric oxygen treatment could be highly useful in treating sports-related injuries.

## B 29

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### **Mitochondrial Function Following HBO Preconditioning**

Mullokanov M<sup>1</sup>, Biram A<sup>1</sup>, Gavis M<sup>2</sup>, Arieli Y<sup>1</sup>

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**Background:** In the present study, we examined the effect of HBO preconditioning on isolated mitochondrial function.

**Methods:** Male Sprague-Dawley rats were divided into four groups. Rats in the negative control group (Ctl<sup>-</sup>) were not exposed to any treatment. Rats in the control group (Ctl) were exposed to HBO at 6 atmospheres absolute (ATA) until the appearance of grand mal convulsions. The remaining two groups were exposed to 3 sessions of HBO at 2 ATA for 1 hour every other day as preconditioning (Pc). After this preconditioning procedure, one group was exposed to HBO at 6 ATA (Pc+6). Samples removed from the frontal cortex and the hippocampus were used to measure mitochondrial membrane potential with JC-1 dye, ATP production and levels of Grp75 under normal conditions and under oxidative stress.

**Results:** Both membrane potential and the driving force over the mitochondrial inner membrane in the hippocampus increased following exposure to HBO at 6 ATA, representing a hyperpolarization state of mitochondrial respiration. The same hyperpolarization was observed in the preconditioned group not exposed to HBO at 6 ATA (Pc). In addition, membrane potential in the Pc+6 group was slightly reduced compared with the Ctl group. ATP production was highest in the cortex of preconditioned rats. Following HBO exposure, ATP production decreased in the hippocampus of preconditioned rats but increased in the control rats. Similar findings were observed under oxidative stress. No significant changes in ATP production were found in the hippocampus in any of the groups. Measurement of the stress-induced chaperone Grp75 showed decreased levels in the hippocampus in the Pc and Pc+6 groups, and in the cortex in the Ctl and Pc+6 groups.

**Conclusions:** We found improved mitochondrial function following HBO preconditioning, expressed by an increase in cortical ATP production, elevated mitochondrial inner membrane potential in the hippocampus, and low levels of mitochondrial Grp75. We suggest that HBO preconditioning leads to uncoupled ATP production in the hippocampus.

## B 30

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### **HBOT Protects Skin from UV-A Damage in a Hairless Mouse Model**

Perdrizet G<sup>1,2</sup>, C. Giradina C<sup>1</sup>, Hightower L<sup>1</sup>

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**Background:** Photoaging of skin is a worldwide problem. Skin damage by sunlight ranges from mild erythema (sun burn) and premature photoaging (wrinkles) to skin cancer initiation (malignant melanoma). Skin damage by sun light is predominantly mediated by energy wavelengths in the ultra-violet range (290-400nm). UV light damages all dermal cell types, including microvascular endothelial cells. Damage is both direct and indirect, involving pathways of acute inflammation, oxidant stress and programmed cell death. Protection of the microvascular endothelium may enhance dermal repair mechanisms by preserving dermal perfusion. We have recently demonstrated that HBOT stimulates human microvascular endothelial cells to increase expression of cytoprotective and anti-oxidant defenses and thereby resist oxidant injury. We hypothesized that HBOT can attenuate UVA-induced photoaging in a hairless mouse model.

**Methods:** Thirty-seven SKH1-E mature, hairless mice (Charles River Lab) were distributed into groups: 1) HBOT x 2 sessions/wk, 2) HBOT x 4 sessions/wk, and 3) sham-HBOT control. HBOT was administered at 2.4 atmospheres for 60 min per session for a total of 22 weeks. All animals received UV-A irradiation (dose escalating 90-177 mJ/cm<sup>2</sup>) three times per week at one hour following HBOT session. Animals were euthanized and skin and liver tissues were recovered. Skin was assayed for micro-creasing (hematoxylin & eosin histology), cellular proliferation (PCNA immunostain), apoptosis (TUNEL stain), and functional elasticity (dermal ultrasound). Group data was compared using ANOVA with Tukey's post hoc or multiple comparison tests. The experimental protocol was approved by the animal care and use committee, Univ. of Conn.

**Results:** UV-A light increased apoptosis, cellular proliferation, skin creasing and decreased elasticity relative to non-irradiated control animals demonstrating dermal injury. All measures of dermal injury were significantly reduced by HBOT therapy. No episodes of oxygen toxicity were observed.

**Conclusion:** HBOT successfully protected skin from UV-A induced injury in a rodent model of photo-aging.

**References:** 1) Fuller AM et al. Hyperbaric oxygen preconditioning protects skin from UV-A damage. *Cell Stress Chaperones*. 2013;18(1):97-107. 2) Buras JA and Reenstra, WR. Endothelial-neutrophil interactions during ischemia and reperfusion injury: basic mechanisms of hyperbaric oxygen. *Neurol Res*. 2007;29(2):127-31. 3) Sklar LR et al. Effects of ultraviolet radiation, visible light, and infrared radiation on erythema and pigmentation: a review. *Photochem Photobiol Sci*. 2013;12(1):54-64.

**Acknowledgments:** Funding source- OxyHeal Corp., San Diego, CA.

# B 31

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

## **Research Collaboration: India - United States**

Dodson WW, Hussain SM, O'Hara RB, Wright BA

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### High Altitude (Ground Operations)

Five total projects have been proposed by the U.S. Army Research Institute of Environmental Medicine, Natick, MA, to India's Defense Institute of Physiology and Allied Sciences (DIPAS). DIPAS staff is reviewing the five proposed projects. The 711<sup>th</sup> Human Performance Wing (HPW) members are the potential U.S. collaborators for one of these projects currently titled "High-Altitude Study Using Dexamethasone to Maintain or Increase Physical Performance in Indian Military Personnel." If pursued, the project would be accomplished in India by Indian personnel. Initially, the Air Force Research Laboratory International Partnering Branch and the 711<sup>th</sup> HPW RH (Human Effectiveness Directorate) section were contacted by Secretary of the Air Force staff. Presently, 711<sup>th</sup> HPW RH and 711<sup>th</sup> HPW USAF School of Aerospace Medicine members are involved as consultants, not investigators.

The other four projects (not currently involving 711<sup>th</sup> HPW members) are 1) "Nutritional Intervention Strategies to Optimize Human Performance in High Altitude Environments," 2) "Cognitive Science Contributions to High Altitude Fatigue Management and Performance," 3) "Hypoxia Monitoring and Alerting System to Manage Fatigue and Optimize Human Performance in High Altitude Environments," and 4) "Supplemental Oxygen During Sleep to Manage Fatigue and Optimize Human Performance in High Altitude Environments."

In October 2014, a quad slide referring to the 711<sup>th</sup> HPW proposed project topic for collaboration cleared by the Foreign Disclosure Office was carried to India by a 711<sup>th</sup> HPW member. Ongoing discussions between all parties have been progressing. We plan to have this quad chart on our HP Summit poster. Currently, our collaborators in India are reviewing our input into the project and are preparing to present their suggestions for a final version of the project. Once that occurs, the project may actually commence in India.

## B 32

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1615 - 1645

RESIDENT COMPETITION:

### **Hyperbaric Oxygen Treatment for Acute Traumatic Brain Injury – Research Potential**

Lee MS<sup>1</sup>, Dodson WW<sup>2</sup>, Wolf EG<sup>3</sup>

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**Introduction:** Hyperbaric medicine has advanced significantly from treatment of low oxygen states, mechanical compression of gas emboli, and anemia. Now there are indications for conditions related to long-term remodeling and post-surgical reperfusion and remodeling therapy. However, the treatment of some acute neurologic incidents with hyperbaric oxygen therapy (HBOT) is relatively new. The Undersea and Hyperbaric Medical Society approved central retinal artery occlusions and idiopathic sudden sensorineural hearing loss as indications for HBOT over the past decade. In addition to effectively treating neurologic decompression sickness, HBOT studies in carbon monoxide poisonings have demonstrated neuroprotective benefits of HBOT in addition to increased carbon monoxide elimination. Recent focus on traumatic brain injuries (TBIs) in military combat operations and sports-related concussions has stimulated research regarding the potential benefit of HBOT related to TBIs. Although the total incidence of military members sustaining TBIs has recently decreased, TBIs continue to occur in the civilian population in significant numbers. Due to increased availability of HBOT treatment centers and improved accessibility to HBOT, treatment of TBI with HBOT within the first week (preferably 24 hours) of the inciting injury is a field of research with a tremendous potential to reduce disease burden and healthcare costs.

**Methods:** A literature review was done finding articles relevant to the use of Hyperbaric Oxygen in the realm of neurologic pathology focusing on acute brain injury. Online search for articles was done in collaboration with Franzello Aeromedical Library at the U.S. Air Force School of Aerospace Medicine, 711th Human Performance Wing, Wright-Patterson Air Force Base, Ohio.

**Results:** Reviewed are significant historic studies involving HBOT in neurologic conditions. Also contained is discussion related to the double-blind, randomized controlled studies involving HBOT for patients with TBI.

**Conclusion:** Recommendations are provided for the potential direction of future HBOT research for TBI.

**PLENARY:  
“ANGIOGENESIS AND HYPERBARIC  
MEDICINE”**

**William Li, MD**

**4:45PM – 5:45PM**



**FRIDAY, JUNE 19**



**PLENARY:  
“HYPERBARIC OXYGEN AND THE CANCER  
PATIENT: ARE YOU CONCERNED?”**

**John Feldmeier, DO**

**8:00AM – 9:00AM**



**SESSION C**  
**CLINICAL HBO<sub>2</sub> THERAPY (#1)**  
Moderators: Brett Hart, MD & Kristie Coleman

**FRIDAY, JUNE 19**  
**9:00AM – 11:30AM**

## C 33

ORAL PRESENTATION TIME: 1406 – 1418 (Saturday, June 20)

POSTER PRESENTATION TIME: 1530 – 1600 (Saturday, June 20)

RESIDENT COMPETITION: YES

### **Hyperbaric Oxygen Therapy for Compromised Flaps: Evaluation of the Optimal Treatment Protocol**

Weber R, Silver A, Williams S, Stephenson L, Usera PC, Zhang F, Tian H, Yang W, Wang WZ, Fang XH, Zamboni WA, Baynosa R

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**Introduction / Background:** The use of HBOT for compromised flaps/grafts is one of 14 indications approved by the UHMS. Failed flaps have significant clinical implications including flap loss, persistence of the original defect, and requirement of another donor site with associated morbidity, and psychosocial sequelae. While postsurgical etiologies may be multifactorial, these situations are common after trauma resulting in degloving and avulsion injuries. Although basic science and clinical evidence is significant, there is no consensus on the optimal treatment regimen. The purpose of this study is to examine whether twice daily (BID) treatments provide significant additional benefit compared to daily (QD) HBOT in a rat compromised flap model.

**Materials and Methods:** A dorsal rat random flap model was used with subjects divided into three groups: 1) Control group, 2) QD group treated with HBOT once a day, and 3) BID group treated with HBOT twice a day where HBOT was performed with 100% O<sub>2</sub> @ 2.4ATA for 90 minutes. On POD #10, area and percentage of flap necrosis were measured. Additionally, biopsies were taken from the middle and distal thirds of the flaps for histologic analysis. Statistical analysis was performed with T-test and P value <0.05 was considered significant.

**Results:** Initial data demonstrate that both treatment groups had significantly increased mean flap survival compared to controls ( $p < 0.05$ ). There was no significant difference in flap survival between the QD and BID groups ( $p = 0.25$ ).

**Summary / Conclusions:** The results of this study confirm the findings in the literature that both QD and BID HBOT protocols can significantly decrease flap necrosis. Preliminary data, however, suggests that there is no additional benefit gained with BID treatments. Additional clinical studies are warranted to confirm these findings and assist in utilization of resources and formalization of protocols for the use of HBOT in treating compromised flaps.

## C 34

ORAL PRESENTATION TIME: 0912 - 0924

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

### **Is Hyperbaric Oxygen Therapy Effective for Traumatic Brain Injury? A Rapid Evidence Assessment of the Literature and Recommendations for the Field**

Crawford CC, Teo L, Yang E, Isbister C, Berry K

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**Introduction/Background:** A systematic review was conducted to examine the efficacy of hyperbaric oxygen (HBO2) for Traumatic Brain Injury (TBI) in order to make recommendations based on the evidence for its application and for future research.

**Materials and Methods:** A comprehensive search was conducted through December 2014 to identify peer-reviewed, clinical research on HBO2 for TBI. Quality assessment was performed using Scottish Intercollegiate Guidelines Network and External Validity Assessment Tool. An analysis was conducted to explore potential placebo components. Synthesis and interpretation was performed with a diverse steering committee and subject matter experts.

**Results:** Twelve randomized controlled trials were included. Methodological quality of the four studies involving mild TBI populations was acceptable. There were no statistically significant differences between HBO2 and sham; statistically significant improvement occurred over time for specific outcomes within both groups. Of the moderate-severe TBI studies, four were acceptable and three low quality. The majority of these showed mostly positive results in favor of HBO2 when compared to “standard care”. All sham arms provided at least 1.2 ATA pressure. The placebo analysis was limited by the lack of details.

**Summary/Conclusions:** For mild TBI HBO2 is no better than the sham treatments used. In comparison to “standard care” there is not enough evidence to draw conclusions. Improvements in outcomes shown within groups for both HBO2 and sham cannot be ignored. Parsing effects from sham O2/pressure from placebo elements was impossible. For moderate-severe TBI, although the methodology appears flawed across some of the studies, because of the complexity and severity of brain injury in this population, HBO2 may be of value and could benefit these patients as a relatively safe adjunctive therapy if feasible. Further research outlined should be considered in order to resolve the controversy surrounding this field, but only if methodological flaws are avoided and bias minimized.

# C 35

ORAL PRESENTATION TIME: 0924 - 0936

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## A Hospital Network-Wide Analysis Of Emergency Department Referrals For Carbon Monoxide Poisoning To Hyperbaric Medicine Services

Cable R<sup>1,2</sup>, Weaver LK<sup>1,2,3</sup>, Deru K<sup>1,2</sup>

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**Introduction:** Despite availability of hyperbaric oxygen (HBO<sub>2</sub>) and clinical trials supporting its role in reducing the incidence of cognitive sequelae after carbon monoxide (CO) poisoning, emergency department (ED) providers often do not refer patients with CO poisoning for HBO<sub>2</sub>. We examined the ED referral pattern in a network of 20 hospitals, with ground and air transport capability, to four 24-hour network HBO<sub>2</sub> services over a two-year period.

**Methods:** We performed an electronic query across the hospital network to identify patients with CO poisoning (by diagnosis code or carboxyhemoglobin level  $\geq 10\%$ ) evaluated between January 1, 2013 and December 31, 2014, and then performed a manual review of each electronic chart. Data abstracted were demographics, ED location, the circumstances of poisoning, and referral for HBO<sub>2</sub>.

**Results:** Over the 2-year review interval, we identified 392 CO-poisoned patients. We excluded 70 individuals as not eligible for HBO<sub>2</sub> referral: 42 presented >24 hours after poisoning, 1 died in the ED soon after arrival, and 227 had incomplete chart data/notes. Of the remaining 322 patients, 166(52%) were not referred.

	Referred (n=156)	Non-Referred (n=166)
Age, years, mean $\pm$ SD (range)	35 $\pm$ 19 (0-86)	32 $\pm$ 20 (0-85)
Carboxyhemoglobin, %, mean $\pm$ SD (range)	17 $\pm$ 9.9 (0.0-44.5)	7.3 $\pm$ 6.4 (0.0-27.0)
Symptomatic at presentation, n (%)	149 (96%)	136 (82%)
Intentional poisoning, n (%)	27(17%)	38 (23%)
Rural ED, n (%)	32 (21%)	84 (51%)
Urban ED, n (%)	124 (79%)	82 (49%)

**Conclusions:** In this data set, a higher carboxyhemoglobin level and presentation to an urban ED appeared to increase the likelihood of a referral for HBO<sub>2</sub>. In our system, ground and air transport time to HBO<sub>2</sub> services does not exceed 3 hours, and ED location should not hinder referral for HBO<sub>2</sub>. Because carboxyhemoglobin levels do not correlate with long-term outcomes, we hope to educate ED providers on referral criteria.

## C 36

ORAL PRESENTATION TIME: 0936 - 0948

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

### **Baseline Vestibular And Audiology Findings In The Brain Injury And Mechanisms Of Action Of Hyperbaric Oxygen (HBO<sub>2</sub>) For Persistent Post-Concussive Symptoms After Mild Traumatic Brain Injury (mTBI) Study (BIMA)**

Kharlamova A<sup>1</sup>, Searing E<sup>1</sup>, Weaver LK<sup>2,3</sup>, Raizada H<sup>4</sup>, Lewandowski A<sup>4</sup>

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**Introduction/Background:** BIMA is a Department of Defense sponsored randomized, double-blind, sham-controlled trial of HBO<sub>2</sub> in a military population with persistent post-concussive symptoms three months or more following mTBI. A comprehensive assessment battery is administered to identify prevalence and magnitude of brain injury and determine the impact of intervention. Baseline vestibular and audiology results are presented.

**Materials/Methods:** BIMA enrolled eligible mTBI participants from September 2012 to May 2014. Exclusion criteria included perilymphatic fistula or deafness ( $\geq 90$  dB) in both ears. Both peripheral and central functions were tested. Tests performed in multiple (left and right) sides were summarized using performance in the worst side.

**Results:** Seventy-one participants were assessed. Most participants reported hearing loss (70%) and tinnitus (82%). However, only 2% had at least mild ( $>25$  dB) hearing loss found through the speech reception threshold assessment and 17% through pure tone average. Twenty-eight (39%) reported spinning, lightheadedness, or instability. Abnormalities were identified on middle ear pathology by tympanometry (17%). Auditory brainstem response stress testing and transient otoacoustic emission testing revealed abnormalities in 49% and 31% of patients, respectively. In auditory steady state response testing, 45% had abnormalities in at least one of four frequencies (0.5-4kHz). SCAN-3:A testing revealed abnormalities in 54%. Rates of abnormal Dix-Hallpike, pneumotoscopy, and nasal pinch findings were low ( $<10\%$ ). Vestibular assessments with higher rates of abnormalities included caloric testing (39%) and dynamic visual acuity loss ( $\log\text{MAR} \geq 0.2$ ) tested in the vertical (72%) and horizontal (67%) planes. Mean composite score on the Sensory Organization Test (SOT) was  $69.6 \pm 15.6$ .

**Summary/Conclusions:** In our study cohort of adults with a history of mTBI, most showed normal peripheral auditory function. However, SCAN abnormalities suggest more neurological deficits in the central auditory pathway. Vestibular testing revealed abnormalities in many of the tests completed, suggesting significant vestibular effects for this population.

# C 37

ORAL PRESENTATION TIME: 0948 - 1000

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Brain Injury Symptoms (SCL-90-R) In A Prospective Cohort Up To 1 Year Following Carbon Monoxide Poisoning

Weaver LK<sup>1,2</sup>, Churchill S<sup>1</sup>, Deru K<sup>1</sup>, Davis J<sup>1</sup>

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**Introduction:** Carbon monoxide (CO) poisoning causes long-term cognitive, affective, and neurological problems. We report patient-rated brain injury symptoms in a prospective cohort with CO poisoning. **METHODS:** During a double-blind, randomized clinical trial of hyperbaric oxygen (HBO<sub>2</sub>) for CO poisoning and a companion follow-up study of individuals refusing or ineligible for the randomized trial, we administered the Symptom Checklist 90-Revised (SCL-90-R) to participants at 6 weeks, 6 months, and 12 months after poisoning. For this analysis, a participant's symptom intensity rating of 0 (not at all) or 1 (a little bit) over the last week on a five-point Likert scale was considered a negative response, while 2 (moderately), 3 (quite a bit), and 4 (extremely) were positive.

**Results:** Between 1995 and 2000, we collected SCL-90-R data on 168 individuals (53 HBO<sub>2</sub>, 50 normobaric oxygen, 65 long-term follow-up only). 56 (33%) were female, mean age was 36±14 years (range 16-86), and mean initial carboxyhemoglobin was 21.7±11.4% (range 0.6-46.7%). 43 (26%) had intentional poisoning. Frequently reported symptoms (%):

	6 weeks N=135	6 months N=150	12 months N=150
Worrying too much about things	33	26	22
Feeling low in energy or slowed down	32	29	20
Feeling easily annoyed or irritated	32	23	19
Headaches	29	27	23
Trouble falling asleep	28	20	16
Feeling tense or keyed up	26	17	17
Difficulty making decisions	25	16	13

Of these symptoms, HBO<sub>2</sub> appeared to confer benefit at 6 weeks for headache (20% HBO<sub>2</sub> vs. 33% non-HBO<sub>2</sub>) and memory complaints (18% vs. 35%), and at 12 months for memory complaints (23% vs. 35%).

**Conclusions:** Using a standardized questionnaire, symptoms after CO poisoning were similar to symptoms after other types of brain injury and appeared to decrease over time. Consistent with other analyses from this group, HBO<sub>2</sub> reduced cognitive but not affective problems.

# C 38

ORAL PRESENTATION TIME: 1000 - 1012

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## Cardiac MRI findings in Patients with Carbon Monoxide Poisoning

Alvarez Vilella M<sup>1,2,4</sup>, Parikh M<sup>1,2,4</sup>, Weaver LK<sup>1,2,3</sup>, Deru K<sup>1,2</sup>

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**Background:** Myocardial injury is caused by carbon monoxide (CO) poisoning. Long-term cardiovascular outcomes following CO poisoning remain incompletely characterized. Cardiac MRI (C-MRI) can help define these cardiac abnormalities.

**Materials/Methods:** After approval by the Intermountain Healthcare IRB, a retrospective cohort was created by reviewing the electronic medical records of all adult patients with stress perfusion C-MRIs performed for a primary diagnosis of CO poisoning between 2005 and 2014.

**Results:** Ninety-one CO poisoned patients underwent C-MRI studies. Of these, 77 were done for a diagnosis of acute CO exposure and 14 for chronic exposure.

**Table 1**

C-MRI in acute CO intoxication				
	No. (%)	Mean COHb	Time to Imaging (mos.)	Elevated Troponin
Abnormal	15 (21)	15.6%	20	4 (27%)*
Normal	61 (79)	15.5%	20	8 (13%)*
<b>Total</b>	<b>77 (100)</b>			<b>12</b>

\*Seven patients with abnormal C-MRI (44%) and 42 patients (69%) with normal C-MRI had unknown troponin values.

**Table 2**

Findings Abnormal C-MRI's Acute Intoxication	
Abnormality	number/total abnormal
LV Dilation	7/15; 47%
RV Dilation	4/15; 27%
LV + RV Dilation	4/15; 27%
LV syst dysfx	9/15; 60%
RV syst dysfx	7/15; 47%
Segmental LV hypokinesis	2/15; 13%
Myocardial Scar	1/15; 7%

**Table 3**

Combination of Chamber Dysfunction and Dilation			
	LV syst dysfx (n=9)	RV syst dysfx (n=7)	LV + RV syst dysfx (n=4)
LV Dilation (n=7)	6 (66%)	0	0
RV Dilation (n=4)	0	3 (42%)	0
BiV Dilation (n=4)	0	0	3 (75%)

Of the 14 patients with chronic exposure, 3 (22%) had abnormal findings and 11 (79%) showed no abnormalities.

**Conclusion:** CO poisoning appears to affect the heart equally in patients with acute and chronic exposure. Mild chamber dilation (LV>RV) and depression of systolic function (LV>RV) are the commonest abnormalities. Troponin values did not predict cardiac injury assessed by MRI in this series.



## C 39

ORAL PRESENTATION TIME: 1012 - 1024

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

### **Brain Imaging Abnormalities In Carbon Monoxide-Poisoned Patients With Ongoing Symptoms At Least 6 Months After Poisoning**

Weaver LK<sup>1,2</sup>, Orrison WW<sup>3,4,5,6,7</sup>, Deru K<sup>1</sup>, McIntosh J<sup>1</sup>

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**Introduction:** Carbon monoxide (CO) poisoning can cause brain injury symptoms that persist for years after poisoning. We report preliminary brain imaging results of a self-selected cohort of CO-poisoned patients who presented for evaluation of continuing problems.

**Methods:** We performed a retrospective chart review of patients who underwent neuroimaging after CO poisoning for ongoing symptoms at least 6 months after the poisoning event. Patients were scanned on a 3.0 Tesla MR. Some had diffusion tensor imaging (DTI), functional MRI, and MR spectroscopy, as well as CT angiography (whole brain by Toshiba 320-detector). Data is presented as mean±1SD (range).

**Results:** From 2004-2015, 161 patients had MRI: 79 (49%) female, age 34±15 (3-73) years, interval from CO poisoning to imaging 2.2±1.7 (0.6-12.6) years. 121 (75%) had a CO exposure duration <24 hours. 147 (91%) were poisoned by heating sources, while 14 (9%) were poisoned by engines. All had ongoing symptoms at the time of imaging. According to clinical interpretation, hippocampal atrophy was present in 118 (73%), 84 (52%) had dilated perivascular spaces, and 73 (45%) had white matter hyperintensities. Of 134 with DTI, 99 (74%) had decreased fiber tracks across the corpus callosum. Of 83 with multi-voxel MR spectroscopy, 30 (36%) were abnormal, and of 41 undergoing CT angiography, 22 (54%) had abnormal perfusion.

**Conclusions:** By high resolution brain MRI and CT angiography, most symptomatic patients had abnormalities consistent with CO poisoning. In this series, the most common findings were corpus callosum fiber track abnormalities and hippocampal atrophy.

# C 40

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## **Treatment of Carbon Monoxide Intoxication/Encephalopathy with Hyperbaric Oxygen Therapy - 26 Years' experience 1986-2013 - Retrospective Review of an Alternative Treatment Protocol**

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**Introduction/Background:** Hyperbaric oxygen (HBO) has been demonstrated to be beneficial in the treatment of acute Carbon Monoxide (CO) poisoning while decreasing the risk of delayed neurologic sequelae. Reports by Thom et al have confirmed the rationale for 2.8 - 3.0 ATA initially in the first dive, while studies by Weaver (2002) have demonstrated improved outcomes when three HBO treatments is provided in the first 24 hours, an approach now advocated by the Undersea and Hyperbaric Medical Society (UHMS). Few studies have examined whether more HBO treatments may be beneficial in the treatment of acutely CO poisoned patients who persist with neuropsychiatric dysfunction following their initial three. Our institution has utilized a protocol for decades of continuing to treat CO poisoned patients one treatment beyond return to their prior perceived baseline neuropsychiatric status (normal + 1) or reaching a plateau in their improvement.

**Materials And Methods:** With IRB approval we retrospectively analyzed our experience at our institution with CO patients treated by our "normal + one" protocol. Our goal was to identify and review the subset of patients requiring more than three hyperbaric treatments to resolve acute symptoms.

**Results:** Data was available for 427 treated by the normal + 1 protocol from the more than 600 total CO patients treated with HBO during a 26 year time period. 57/427 (13.3%) patients required more than 3 treatments. 33/57 (57.8%) of these patients had complete subjective resolution of symptoms. 22/57 (38.5%) patients demonstrated substantial improvement. 2 of the 57 patients died despite ongoing treatments.

**Summary/Conclusions:** Patients with continuing neuropsychiatric symptomology following three initial HBO treatments may benefit from additional HBO therapy. Further investigation is required with collaborating institutions to identify ideal treatment pressures and numbers of dives to treat residual but potentially reversible CO induced brain injury.

# C 41

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## Waterpipes: A Misconception

Michetti Y, Lambert D

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**Introduction/Background:** 25 year old female with a history of migraines and depression complaining of a severe headache, unsteadiness with ambulation. She reports smoking a hookah in her house with friends for three days.

Carboxyhemoglobin: 35.8%

She was transferred to the Hospital of the University of Pennsylvania for Hyperbaric Oxygen Therapy. On arrival, symptoms improved and her neurological exam was unremarkable. Tolerated the Weaver protocol for acute CO poisoning with no issues.

**Summary/Conclusions:** Waterpipes, also known as a hookah, are becoming more popular. A study from one college campus showed that 48% of surveyed first-year college students used a waterpipe at some time in the past.<sup>1</sup> The same study showed that students who used a waterpipe in the past 30 days were less likely to feel that it is as harmful as cigarette smoking compared to their peers who have never used a waterpipe.<sup>1</sup>

“Tobacco-free” products for the hookah are marketed as not having the harmful effects associated with tobacco. It has been shown that the CO and other toxic agents levels remain unchanged in these products.<sup>2</sup> It is felt that this is contributed to by the use of charcoal. The waterpipe uses heated charcoal to cause the tobacco product to burn, creating a combined source of smoke from the charcoal and tobacco. The average CO exposure during a waterpipe smoking session is 143mg compared to a single cigarette which ranges from 1 to 22mg.<sup>3</sup>

We need to continue having discussions with our patients about avoiding cigarette smoking and smoking cessation; however, we also need to recognize that there are other significant tobacco-related health risks, such as waterpipes. Second-hand smoke from waterpipes contain toxic compounds from the tobacco, as well as, the charcoal. We must educate patients on the associated risks with smoking from a waterpipe, including carbon monoxide poisoning.

### References:

1. Eissenberg T, et al. Waterpipe tobacco smoking on a U.S. college campus: prevalence and correlates. *J Adol Health* 2008;42:526-529
2. Shihaden A, et al. Does switching to a tobacco-free waterpipe product reduce toxicant intake? A crossover study comparing CO, NO, PAH, volatile aldehydes, tar and nicotine yields. *Food Chem Tox* 2012;50:1494-1498.
3. Shihaden A, Saleh R. Polycyclic aromatic hydrocarbons, carbon monoxide, “tar”, and nicotine in the mainstream smoke aerosol of the narghile water pipe. *Food Chem Tox* 2005;43:655-661.

# C 42

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## Long-term Patient Reported Outcome of Hyperbaric Oxygen Therapy for Haemorrhagic Radiation Cystitis

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**Introduction/Background:** Haemorrhagic radiation cystitis (HRC) is a debilitating complication in 6.5-8% of patients after pelvic radiotherapy. Treatment of HRC is mostly symptomatic and evidence-based guidelines are lacking. Hyperbaric oxygen therapy (HBOT) has a different approach as it targets the origin of symptoms, i.e. local hypoxia. The objective of this study is to determine the effectiveness of HBOT to lessen or cure haemorrhagic cystitis in patients after radiotherapy of the lower pelvis

**Materials/Methods:** All living patients who had HBOT for HRC were retrospectively analysed. Effectiveness of HBOT was evaluated by patient-reported outcomes via a structured telephone interview. HBOT was given at 2.5 atmospheres for 115 min per session.

**Results:** Included were 74 patients (59 male, 15 female; mean age 72 years). Patients were referred for HBOT (median) 3.5 (range 0-30) years after radiotherapy and received (median) 40 (range 15-135) HBOT sessions. Of all patients, 44 (59%) reported complete resolution of symptoms, 19 (26%) reported partial relief, and 11 (15%) had no improvement of symptoms. After initial successful resolution of symptoms, 18 patients (24%) experienced recurrent symptoms of HRC.

**Summary / Conclusion:** HBOT can relieve or dissolve symptoms of HRC after radiotherapy of the pelvic area, especially in man after prostate cancer treatment. HBOT should be considered in patients suffering from mild to moderate HRC.

# C 43

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## **Carbon Monoxide Exposure and Timely Hyperbaric Oxygen Therapy**

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**Introduction/Background:** Hyperbaric oxygen therapy (HBOT) is used to treat carbon monoxide (CO) poisoning, and evidence suggests maximum benefit when administered within 6 hours. The limited number of facilities providing HBOT for emergent conditions makes this difficult.

**Materials and Methods:** Data was collected on patients with CO poisoning transferred for HBOT from October 1, 2010 – January 15, 2014 . Data collected included transport distance, time of arrival at sending and receiving hospital, and time HBO was initiated.

**Results:** 43 patients were transferred a mean distance of 88 miles. There was a mean distance of 67 miles for those who received treatment <6 hours after initial ED arrival and 95 miles for those who received treatment >6 hours after ED arrival ( $p=0.12$ ). The mean distance of patients transported by ground vs. air was 82 miles vs. 116 miles ( $p=0.17$ ). Among patients transported by ground there was a mean distance of 58.5 miles for those treated <6 hours and 93 miles for those who received treatment >6 hours after initial ED arrival ( $p=0.11$ ). Among patients transported by air there was a mean distance of 91 miles for those treated <6 hours compared to 147 miles for those treated >6 hours after ED arrival ( $p<0.001$ ). It took an average of 2 hours from ED arrival to contact with hyperbaric medicine. The mean time from contact with hyperbaric medicine to treatment was 288 minutes for ground and 211 minutes for air ( $p=0.02$ ).

**Summary/Conclusions:** Our findings suggest the need for air transport for distances > 58.5 miles. Patients requiring transportation of >147 miles may not receive maximum therapeutic benefit despite air transportation. Finally, there should be education to EM providers to make initial contact with hyperbaric medicine more rapidly. The results are useful in weighing risk vs. benefit of transportation and when to recommend air transportation.

## C 44

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

### **Patient Outcomes of Hyperbaric Oxygen Therapy for Radiation Cystitis**

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**Introduction/Background:** A side effect of radiation therapy to treat genitourinary cancers is radiation-induced cystitis, which in its most severe form can be hemorrhagic cystitis (HC). Some studies have found the incidence of HC in radiation patients to range from 3% to 6.5% after radiation therapy and can develop anywhere from 6 months after radiation therapy to 10 years or more after the last treatment. The severity of symptoms, which includes hematuria, may drastically reduce quality of life. Recent studies have shown that hyperbaric oxygen therapy has improved symptoms of radiation cystitis in around 80% of patients.

**Materials and Methods:** Sixty patients who received hyperbaric oxygen therapy (HBOT) for radiation-induced cystitis since 2010 at Winthrop University Hospital were identified. IRB approval was obtained for contacting these patients to gather outcome data. Patient reported outcomes were evaluated using a questionnaire that assessed the type and duration of radiation treatment, treatment other than HBOT for HC symptoms, surgical intervention, and patient perceived outcomes of HBOT.

**Results:** 36 patients were consented. The average age of the patient was 71 with a range from 29-91. The majority of the patients 78% had prostate cancer with the Colon, Uterine, Bladder and Testicular cancer also included. The average time from completion of radiation to onset of symptoms was 3 years. The mean number of treatments was 35 with a range of 18-120 treatments. All treatments were for 90 minutes at 2.4 ATA. The majority of the patients (24) had external beam radiation and 12 patients had a urological procedure after completion of their therapy. 78 % of the patients reports either moderate improvement or complete resolution of symptoms.

**Conclusion:** This case series seems to demonstrate that Hyperbaric Oxygen Therapy appears to be an effective treatment for radiation cystitis, however additional studies are necessary.

# C 45

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## Carbon Monoxide (CO) Poisoning Precipitation of NSTEMI - A Case Report

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**Introduction/Background:** We report a patient who presented 50 hours after acute CO exposure with persistent signs and symptoms of acute coronary syndrome, starting at the time of CO exposure.

**Materials/Methods:** In 2015, a 41-year-old truck driver with a remote twenty pack-year smoking history presented to the Emergency Department with complaints of nausea, weakness, headaches and intermittent blurred vision two days after a known CO exposure of six hours duration. Exhaust was found to communicate with the cab of his truck. He had taken a two-hour nap in the cab of his truck, which he kept running for heat, in between two two-hour drives. His carboxyhemoglobin was 3.5% (within allowable error limits for normal CO at our lab) and troponin I level was 17 ng/dL with EKG changes demonstrating inferior infarction. Transthoracic echocardiogram demonstrated LVEF of 57% and severe inferior / inferoseptal hypokinesis and akinesis. He was admitted to the Cardiac Care Unit for workup.

**Results:** Troponin I peaked at 19 ng/dL on day four after CO exposure. The LAD and RCA were found to be 100% occluded proximally (stented in the cardiac catheterization lab), with decreased flow through the circumflex coronary artery. PET scan day five post-exposure demonstrated moderate inferior & anterior wall hypokinesis, preserved LVEF and large inferoseptal / inferolateral ischemia with mid and apical segments recovering. The patient continued to experience chest pain and lightheadedness through day five post-CO exposure, and at six-week follow-up complained of persisting occasional lightheadedness as well as short-term memory problems.

**Summary/Conclusions:** This case emphasizes the potential for CO poisoning to precipitate myocardial infarction in patients with predisposing factors. Symptom persistence at six-week follow-up may be due to CO poisoning in addition to coronary artery disease.

# C 46

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YESC 45

## **Inaccurate Pulse Oximetry of Carboxyhemoglobin Due to Digital Clubbing**

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**Introduction/Background:** The RAD-57 (Masimo Corporation) pulse oximeter provides non-invasive rapid measures of oxyhemoglobin, carboxyhemoglobin (SpCO), and methemoglobin. Digital clubbing can cause inaccuracy of pulse oximeters. We present the first case of inaccurate SpCO measurement by pulse oximetry due to digital clubbing.

**Case Presentation:** The patient is an 18 year old man with cystic fibrosis who presented after a suicide attempt by inhalation of exhaust. His initial carboxyhemoglobin (COHb) level was 34%. He was intubated, provided 100% oxygen, and transferred to our facility. Upon arrival, we placed four different RAD-57 oximeters on different fingers and toes. The SpCO measured by these meters ranged from 9-11%. A venous blood gas drawn at the same time showed a carboxyhemoglobin level of 2.3%. Thirty minutes later, on an arterial blood gas, COHb was 0.9% and the SpCO by RAD-57 was 10%.

**Discussion:** This case supports that the RAD-57 non-invasive measurements of carboxyhemoglobin was erroneously elevated, likely due to digital clubbing.



# C 47

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## **Clostridial Myonecrosis: An Urgent Indication For Hyperbaric Oxygen Therapy**

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**Introduction:** Clostridial myonecrosis is a profoundly acute, progressive soft tissue infection. Clostridium produces several toxins including alpha-toxin which plays a primary role in tissue necrosis and hemolysis thus causing the classic gas bubbles evident on radiographs and profound soft tissue destruction. Alpha-toxin inactivates local host defense mechanisms. However, alpha-toxin production halts at tissue O<sub>2</sub> tensions of 250 Hg. Hyperbaric oxygen (HBO<sub>2</sub>) therapy at 3.0 ATA achieves tissue O<sub>2</sub> tensions above 300 Hg thus stopping alpha-toxin production.

**Background:** We present a non-diabetic patient who developed rapidly progressing right hand and arm erythema and swelling over 2 days without prior injury. He had pain out of proportion to his exam. X-ray and MRI showed subcutaneous air bubbles. Lab test results included C-reactive protein elevation, 109 mg/L, but normal sedimentation rate and white blood cell count.

**Materials and Methods:** He underwent emergent then serial surgical debridement on days 0, 2, and surgical evaluation day 4. On initial surgical debridement, the surgeon identified areas with abundant purulence, other areas with dishwasher-like drainage, and muscle necrosis. Surgical wound cultures were positive for Clostridium perfringens, among other bacteria. He was treated with multiple antimicrobials modified to ertapenem and metronidazole for 3 weeks based on culture and sensitivity results. He was treated with HBO<sub>2</sub> therapy for 3 sessions at 3.0 ATA within 24 hours, followed by 2 sessions at 2.0 ATA over the next 3 days.

**Results:** The patient had no additional tissue necrosis at the 3<sup>rd</sup> surgical wound evaluation. No amputation was required. His wounds healed in 4 weeks. He had no functional disability after 6 months.

**Summary/Conclusions:** HBO<sub>2</sub> therapy is bacteriostatic against clostridium, does not inactivate free-circulating or tissue bound alpha-toxin, but stops alpha-toxin production when delivered at 3.0 ATA. HBO<sub>2</sub> therapy in conjunction with surgical debridement has been shown to reduce amputation rates to 18% from 55%.

## C 48

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

### **The Validity of Juxta-Wound TCOMs in Predicting Healing of Diabetic Foot Ulcers**

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**Introduction:** In 2002 two papers (Fife, et al. *Wound Rep Reg*,10:198-207) and Strauss, et al., *Foot Ankle Intl*, 23:933-937) were published that studied the predictability of transcutaneous oxygen measurements (TCOMs) for healing wounds. The statistical methods used to draw conclusions from the two papers are subject to scrutiny. This presentation reviews the data presented in each paper and comprehensively re-analyzes it for the validity of TCOMs in predicting healing of diabetic foot ulcers.

**Methods:** A biostatistical review of the Fife and Strauss papers was done by the first author. The numbers presented in the papers were subjected to statistical analyses to compare like by like data as well as test for “p” values and odds ratios.

**Results** In the Strauss paper 143 subjects with TCOMs < 30 mmHg in room air were studied in a progressive series. In those TCOMs which exceeded 200 mmHg with HBO, the healing rate was 87.6% even if the room air TCOM was less than 30 mmHg. Although the Fife paper included 1144 subjects studied retrospectively from 6 facilities, TCOM studies had multiple permutations and combinations. Of the subset of 221 (19.3%) who had TCOMs with HBO exposure, failure rates decreased progressively from 35.7% to 14.3% with TCOMs in 100 mmHg increments from over 200 to 699 mmHg. This resulted in absence of statistical significance for any 100 mmHg range over 200 mmHg with HBO although the authors state a “cutoff” TCOM with HBO is 74% predictable of healing

**Conclusions:** Although substantial differences exist between the models used for the Fife and Strauss TCOM studies, portions of each complement the other. With the Strauss study TCOMs over 200 mmHg demonstrated healing in almost 90% of the study group regardless of the room air TCOM. The Fife data predicted failure in 75% of the patients.

# C 49

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Experiences with Gentian Violet as a Wound Dressing Agent

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**Introduction:** Over 2000 products are available for wound dressings. Prices range from a few cents to several thousand dollars per application, with no correlation between price and effectiveness. After treating many thousands of patients, we have found that Gentian Violet (GV) is an effective, inexpensive alternative when other wound dressing agents have been ineffective.

**Methods:** After mitigating deformities, deep infections (bursa, scar and bone), and ischemia-hypoxia, a small percentage of lower extremity wounds failed to improve with customary interventions such as negative pressure wound therapy, bioengineered dressings, moist gauze dressings, silver impregnated ointments, silicon dressings and/or dressing films. As a last resort, GV was used as the wound dressing agent. GV is an aniline dye that has drying and antimicrobial properties. A small jar (about 60 cc) costs around \$3.00.

**Results:** When other wound dressing agents have failed, especially where maceration secondary to fluid leakage through the wound base from fluid retention occurs, daily coating of the wound base with GV has resulted in diminution of the wound size, wound contraction, and elimination of marginal skin maceration (coupled with zinc oxide). All of the wounds refractory to other wound dressing agents have either healed or improved enough to become chronic stable wounds, and restoration of functional lower extremity activities restored, when GV was utilized.

**Conclusions:** Gentian Violet as a "last resort" intervention in refractory wounds has shown effectiveness. The clinical improvements observed using GV offer an additional agent that can be used on superficial and mildly cavitory wounds at earlier stages in a patient's wound course. The ease of application and the economies of GV have made this agent an increasingly important intervention in the armamentarium of wound care products.

# C 50

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## Case Studies: Hyperbaric Oxygen Therapy After Accidental Ingestion of 35% Hydrogen Peroxide

Parikh M<sup>1, 2, 3</sup>, Alvarez Villela M<sup>1, 2, 3</sup>, Tettelbach WH<sup>1, 2, 3</sup>, Weaver LK<sup>1, 2, 3, 4</sup>

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**Introduction:** Ingestion of hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) can cause portal venous and/or arterial gas emboli. One mL of 35% H<sub>2</sub>O<sub>2</sub> produces 100 mL of oxygen. Hyperbaric oxygen therapy (HBO<sub>2</sub>) for H<sub>2</sub>O<sub>2</sub> ingestions may decrease morbidity, mortality, and expedite recovery. We report two cases of accidental 35% H<sub>2</sub>O<sub>2</sub> ingestion treated with HBO<sub>2</sub> with full resolution of symptoms within 24 hours.

**Case 1:** 16-year old healthy male accidentally ingested 2 gulps of 35% H<sub>2</sub>O<sub>2</sub>, mistaking it for V8 juice. Immediate symptoms: lightheaded, dizzy, nausea, hematemesis, and oral foaming, without neurological deficits. Abdominal radiographs revealed portal venous air. He received one HBO<sub>2</sub> treatment (3.0/2.0 ATA), with symptom and radiological resolution by the next morning.

**Case 2:** 80-year old male ingests 200mL of 35% H<sub>2</sub>O<sub>2</sub> mistaking it for glucosamine chondroitin medication. Immediate symptoms: emesis, dizziness, weakness and fell without a loss of consciousness, confusion, disorientation, and slurred speech. Supplemental oxygen resolved the disorientation and slurred speech. CT head was unremarkable. CT abdomen and pelvis showed extensive gas emboli within the liver suspicious for portal vein origin with pneumatosis in the gastric antrum and duodenum with possible emboli in the mesenteric vessels. Patient was treated with USN Table 6 with full symptom and CT resolution.

**Conclusion:** HBO<sub>2</sub> expedited the recovery from H<sub>2</sub>O<sub>2</sub> poisoning and should be considered for all symptomatic cases.

# C 116

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## **Rapid Hyperbaric Oxygen Therapy to Reverse Post-Operative Posterior Ischemic Optic Neuropathy**

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**Introduction:** Posterior ischemic optic neuropathy (PION) is uncommon, a vexing ophthalmologic disease with few treatment options. PION results from retro-bulbar optic nerve infarction from a variety of causes: peri-operative, arteritic, and non-arteritic. Peri-operative causes include: hypotension, blood loss, prolonged surgery, and prone positioning. Chronic vascular diseases likely contribute to the risk of PION. Typical presentation is acute, painless vision loss, often bilateral when peri-operative. The optic disc typically appears normal. There is no standard treatment for PION although corticosteroids are considered especially in arteritic PION. Prevention is the primary consideration. Visual acuity does not usually improve.

**Materials and Methods:** An 82 year old female with history of hypertension, lupus, PVD, and subclavian stenosis. She developed profound bilateral vision loss after an unremarkable angiogram. There was no intra-operative blood loss; however, intravenous Hydralazine was given with subsequent relative hypotension (systolic blood pressure 170 to 100).

**Results:** The patient had profound bilateral painless vision loss when she recovered in the PACU (Hand Motion OU). She had no other focal findings but was mildly confused. Retinal exam was unremarkable. CTA and MRA of the brain were normal. The patient was transferred and treated within 5 hours of initial injury. Although the mechanism of injury and rationale for HBOT for PION was recognized to differ from Central Retinal Artery Occlusion (CRAO), we elected to follow our institutional protocol for CRAO. Compression was with a Navy Treatment Table 5. After the initial HBOT, intravenous corticosteroids were started. Subsequent treatments were 45 FSW for 120 minutes, 33 FSW for 120 minutes, and finally 45 FSW for 120 minutes.

**Summary/Conclusions:** Several hours after the initial compression, visual acuity improved to 20/40 OU which approximated the patient's baseline. We conjecture that the combination of rapid initiation of HBOT and intravenous corticosteroids resulted in dramatic improvement of visual acuity.

# C 117

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## **Continuous Bladder Irrigation in the Monoplace Hyperbaric Chamber**

Cooper JS, Allinson P, Winn D, Keim LW, Sippel J Scahlberg P, Fowler K

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**Introduction/Background:** Radiation induced hemorrhagic cystitis can be a serious side effect of radiation therapy. Transfusion requiring anemia can ensue. Treatment methods include bladder irrigation, fulguration, instillation of various agents, and Hyperbaric Oxygen Therapy (HBO). Failure of treatment leads to cystectomy associated with a high risk of severe complications (42%) and mortality (16%). Clot removal and urinary drainage are the initial treatment so as to prevent obstruction. Continuous bladder irrigation (CBI) is then used to prevent further clot formation.

HBO supports the healing process of radiation induced cystitis. In patients requiring CBI, the time in HBO can cause clot accumulation and urethral obstruction. In a multiplace chamber, CBI is continued without change of the CBI set-up. We describe a method of providing CBI in a monoplace hyperbaric chamber.

**Materials And Methods:** An IV to catheter tubing adapter is utilized to allow an IV pump to control the CBI flow into the chamber. The drainage from the irrigating urethral catheter is collected in an extra-large urinary collection bag. The flow rate is set to achieve the previously set output goal while ensuring that the volume does not exceed the collection bag's capacity by the end of the HBO treatment. The collection bag must be placed in a manner that precludes spilling and allows visualization to see if the drainage stops flowing from the tube.

**Results:** CBI was successfully maintained with this method. Brief case reports are presented and issues with this method are discussed.

**Summary/Conclusions:** CBI is easily maintained in a monoplace hyperbaric chamber with equipment readily available in a hospital setting. This allows for uninterrupted CBI of hemorrhagic cystitis with HBO mitigating the potential side effects of other bladder interventions in a previously irradiated area.

## ERIC P. KINDWALL MEMORIAL LECTURE

FRIDAY, JUNE 19TH: 11:30am to 12:30pm

Guest Speaker: Paul Sheffield, PhD, CAsP, CHT, FASMA, FUHM

### "Hyperbaric Safety: A Half-Century Commitment"



This presentation will be a review of lessons learned from mishap information collected during the presenter's experience as Medical Squadron Commander (1960s), Hospital Fire Marshall (1960s), Flying Safety Officer (1970s), Safety Monitor for USAF Hyperbaric Medicine Program (1970s-80s), Director of U-2/Sr-71 Pressure Suit Depot (1980s), Chief Aerospace Physiologist for the Air Force (1990s), and International ATMO, Inc educator (1990s-2015). The focus will be on hyperbaric chamber fire safety.

#### **About Dr. Sheffield:**

Paul J. Sheffield, PhD, CAsP, CHT is President, International ATMO, Inc. of San Antonio, Texas, which provides wound care and hyperbaric medicine management, consulting, and education services. His degrees include BS in Chemistry (Univ of Florida, 1962), MS and PhD in Physiology (Univ of Southern California, 1971, 1972). He is a Certified Aerospace Physiologist (CAsP); Certified Hyperbaric Technologist (CHT); Fellow, Aerospace Medical Association; Past Pres, Aerospace Physiology Society; Past Pres, Undersea & Hyperbaric Medical Society; Fellow, Undersea & Hyperbaric Medical Society; Chaired UHMS Education (CME) Comm (1994-2012); and International ATMO CME Program Director.

Dr. Sheffield began his career as a US Air Force Aerospace Physiologist, with primary responsibilities in aircrew training, research, and hyperbaric medicine. He received his initial training in Hyperbaric Medicine in 1965. He has served as Hospital Fire Marshall (1960s), Flying Safety Officer (1970s), Safety Monitor for USAF Hyperbaric Medicine Program (1970s-80s), Director of U-2/Sr-71 Pressure Suit Depot (1980s), Chief Aerospace Physiologist for the Air Force (1990s), and International ATMO, Inc educator (1990s-2015).

He wrote and produced the Air Force training film: "Plasma Bubbles and Decompression Sickness." He was on the original team, with Dr. Jefferson C. Davis, that established the USAF Hyperbaric Center at Brooks Air Force Base, Texas in 1974. He was one of the originators of the use of tissue oximetry for wound assessment and patient selection for hyperbaric oxygen therapy. He was Air Force Chief of Aerospace Physiology in Washington DC prior to his retirement in the grade of Colonel after 30 years of military service. He is one of the founders of International ATMO in 1977, became Director of Research and Education in 1992, and became President in 2000.

Dr Sheffield became Course Director and faculty member in US Air Force hyperbaric medicine courses in 1971. He took up recreational diving in 1977 when he began instructing in Medical Seminars' Medicine of Diving Courses. He also has about 1,000 hours of exposure in altitude chambers and hyperbaric chambers. Altitude exposures were above 70,000 ft altitude (pressure suit) and hyperbaric exposures were as deep as 200 fsw (air) and 225 fsw (heliox).

To date, he has been educating physicians, nurses, and technologists in hyperbaric medicine for 50 years. In 1998 his ATMO Hyperbaric Medicine Team Training course became the first UHMS Designated Introductory Course in Hyperbaric Medicine and the curriculum became the model for all others approved by UHMS. He is a frequent international lecturer in hyperbaric physiology, safety, and diving medicine, with invited lectures in 20 countries.

He has authored 140+ scientific publications in aerospace medicine and hyperbaric medicine and has chapters in 11 books. He has served as editor of two training manuals [USAF Physiological Training

Manual (1972); USAF Compression Therapy Manual (1976)] and coeditor of two books [Wound Care Practice (Best Publ., 2004, 2007); Wound Care Certification Study Guide (Best Publ., 2011, 2015)].

## ERIC P. KINDWALL, MD



Dr. Kindwall is known by so many as the "Father of Hyperbaric Medicine." Whether you knew him personally or simply by reputation, we have all benefited from his efforts, passion, wisdom, knowledge, energy and vision. Dr. Kindwall has played a great role in growing and shaping the specialty of Undersea and Hyperbaric Medicine. He has likewise been instrumental in molding the UHMS into what it is today. Dr. Kindwall began diving in 1950. He cultivated his interest in the field and during the Vietnam War served as the Assistant Director of the U.S. Navy School of Submarine Medicine. He also was the Senior Officer responsible for the Diving Medicine Program. In 1969, after leaving the Navy, Dr. Kindwall became Chief of the Department of Hyperbaric Medicine at St. Luke's Medical Center, Milwaukee, Wis. Shortly after the Undersea Medical Society was created in the mid-1960s, Dr. Kindwall identified the need for standardized education in the field. He created the UMS Education and Standards Committee to help elevate course content and ensure instructor competence. This committee later became our Education Committee. When the AMA initiated its Continuing Medical Education program, Dr. Kindwall persuaded the organization to recognize the UMS as a grantor of CME credits. In 1972, Dr. Kindwall felt that the Society's members would benefit from improved communication. He created our first newsletter and was named editor. Dr. Kindwall chose the name Pressure because clinical hyperbaric medicine was rapidly developing. Even though the UHMS had not yet incorporated "Hyperbaric" into the Society's name, he wanted a title for the newsletter that would encompass all who worked with increased atmospheric pressure. He stated: "The Society's goal then, as it is now, is to serve all who deal with the effects of increased barometric pressure." That same year, Dr. Kindwall recognized the need to have a relationship with Medicare to help provide insight on reputable clinical management. The UMS followed this lead, and a Medicare Panel was created. The recommendations were presented to the U.S. Public Health Service. The challenge was that no reliable hyperbaric medicine clinical guidelines were available that addressed appropriate applications of Hyperbaric Medicine. To remedy this deficit, the UMS Executive Committee created an Ad Hoc Committee on hyperbaric oxygen therapy. Dr. Kindwall was named Chair. The committee created the first Hyperbaric Oxygen Therapy Committee Report. Again, this text was published 10 years before the UHMS incorporated "Hyperbaric" into its name. The report was sent to HCFA and the Blues and became their source document for reimbursement. Dr. Kindwall updated the text two more times and thus was the Editor and Chair of the Committee and text for three of its 12 editions. Dr. Kindwall later worked to expand the available information on the specialty by creating one of the first complete texts on the field. He created Hyperbaric Medicine Practice in 1994 and later updated and revised his text two more times. The Society's first journal, Hyperbaric Oxygen Review, has also been influenced by Dr. Kindwall. His love for research and education was clear: He became the initial editor, creating a journal that at first consisted of review articles and one original contribution. Over the years, it has grown to one full of original research. Dr. Kindwall's presence is felt in so many of the UHMS' activities and initiatives. Much of what we all take for granted – what is just "there" and "available" – has his touch and influence. Some of us have been blessed to have had a closer impact by Dr. Kindwall's life, but I think that I can easily say that each of us has been influenced in some way.



**OPTIONAL PLENARY:  
“HYPERBARIC MEDICINE IN A VALUE  
BASED PAYMENT SYSTEM: WHAT IT WILL  
TAKE TO SURVIVE AND THRIVE”**

**Caroline Fife, MD**

**Laurie Gesell, MD**

**Helen Gelly, MD**

**12:30PM – 1:30PM**

**Objectives:**

1. At the conclusion of this talk the listener will be able to list 3 major changes to U.S. hyperbaric reimbursement affecting either the facility or the physician which will occur within the next 2 years
2. At the conclusion of this talk the listener will be able to identify 9 quality measures that can satisfy the requirements of the Physician Quality Reporting System in order to avoid substantial financial penalties

**SESSION D**  
**DIVING AND DECOMPRESSION ILLNESS**

**Moderators: Tracy LeGros, MD & Matt Schweyer, CHT**

**FRIDAY, JUNE 19**

**1:30PM – 4:00PM**

# D 51

ORAL PRESENTATION TIME: 1330 - 1342

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Factors Influencing The Energy Cost Of Free Fin Swimming

Pendergast DR, Hostler D

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**Introduction/Background.** Energy expended ( $E_{tot}$ ) during underwater fin swimming influences ventilation,  $CO_2$  retention and exercise intolerance.

**Materials/Methods.** Diver skill, equipment (fins, tanks, protective suits), and conditions (water temperature, swim time) were studied to determine their  $E_{tot}$  at 4fsw.

**Results.**  $E_{tot}$  increases as a 2<sup>nd</sup> order polynomial with velocity ( $V$ ) with good skill being  $-0.15 + 2.26 V + 1.49 V^2$  and poor  $0.045 + 1.65 V + 1.66 (2) V^2$ .  $E_{tot}$  increased with all fins ( $0.045 + 1.65 V + 1.66 V^2$  in best fin,  $0.25 + 1.03B V + 1.86 (2) V^2$  in worst fin).  $E_{tot}$  for one and two tanks increased from  $1.42 \pm 0.42 - 2.01 \pm 0.65$  L/min at 30.8 - 40.6 m/min, after which it increased to  $2.24 \pm 0.38$  L/min at 50.4 m/min (one tank) and 46.5 m/min at  $2.54 \pm 0.39$  L/min (two tanks).  $E_{tot}$  of fin swimming increased  $2.59 + -0.14V + 0.003 V^2$  for no suit and wetsuit and significantly more ( $-0.56 + 0.08 V + 0.00008 V^2$ ) with a dry suit. In thermal neutral conditions (TNC),  $T_c$  increased  $0.4^\circ C$ , while in cold conditions (CC) it decreased  $0.6^\circ C$ . In TNC water,  $\dot{V}_{O_2}$  was 1.05 L/min and did not change as a function to time, however in CC it increased to 1.44 L/min and swimming time decreased ( $60 \pm 23$  min). Swim time was reduced in CC compared to TNC, likely due to local respiratory and leg muscle fatigue secondary to exercise time and body cooling.

**Conclusion.** The  $E_{tot}$  of fin swimming at 4fsw can be minimized by improving skill, fin selection, and using a wet suit. Swimming for a prolong time in cold water or with a dry suit increases  $E_{tot}$ . These studies were funded by the Office of Naval Research and Naval Sea Systems Command.

## D 52

ORAL PRESENTATION TIME: 1342 - 1354

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

### **Effect of Pressure on Heating and Cooling Requirements for Thermal Protection of Wet-suited Divers**

Pendergast DR, Hostler D

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**Introduction/Background.** Divers often wear wet suits for thermal protection. Previous studies determined the energy requirement to prevent body cooling or heating at shallow depths. This study reports the effect of pressure on the energy requirement to protect divers in cold and warm water.

**Materials/Methods.** Male divers ( $177\pm 6$  cm in height,  $79.43\pm 7.72$  kg in weight,  $14.9\pm 3.5\%$  body fat) were studied. Subjects were studied at rest submersed in a hyperbaric chamber at 4, 55, and 120fsw. Divers wore a prototype 6-zone tubesuit (head, torso, arms, hands, legs and feet) under a 6.5mm foam neoprene wetsuit. The tubesuit was perfused with  $30^{\circ}\text{C}$  water at 0.5 L/min from a diver thermal protection system (DTPS) using thermoelectric modules (TEC) that heated and cooled the circulating water. The tubesuit inlet and return flow temperatures were measured and multiplied by the flow to get the power required to protect the diver (total body and zoned calorimeter).

**Results.** Mean torso skin temperature ( $T_s$ ) ( $29.9 \pm 0.07^{\circ}\text{C}$ ) and core temperature ( $T_c$ ) ( $36.5 \pm 0.04^{\circ}\text{C}$ ) were maintained in all conditions by the DTPS. The on-off cycle of the TEC was  $35\pm 14\%$  at 4fsw and increased to  $48\pm 18\%$  at 120fsw in cold water and in warm water it was  $85\pm 18\%$  and  $100\pm 0\%$ , respectively. Finger and foot  $T_s$  changed exponentially at 4fsw to a plateau of  $23.7\pm 0.3^{\circ}\text{C}$  and  $17.0\pm 0.2^{\circ}\text{C}$  in  $10^{\circ}\text{C}$ , increased to  $32\pm 0.3^{\circ}\text{C}$  at 4ft in  $40^{\circ}\text{C}$ , and  $34\pm 0.5^{\circ}\text{C}$  at 120ft in  $35^{\circ}\text{C}$  water. The heating/cooling to maintain thermal protection was  $-214\text{W}$  to  $242\text{W}$  of heating (-) and cooling (+) in  $10^{\circ}\text{C}$  and  $40^{\circ}\text{C}$  water, respectively.

**Summary/Conclusion.** The hydrostatic compression of the wet suit at depth reduced insulation capacity and thus increased DTPS duty cycle to provide more heat/cooling to the diver for thermal protection. These studies were funded by the Office of Naval Research and Naval Sea Systems Command.

# D 53

ORAL PRESENTATION TIME: 1354 - 1406

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Swimming-Induced Pulmonary Edema (SIPE) in Triathletes: Effect of Age

Moon RE, Martina SD, Peacher DF, Otteni CE, Wester TE, Potter JF

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**Introduction:** SIPE is a condition that occurs in divers and swimmers, and the evidence overwhelmingly supports high pulmonary capillary pressure as the cause. SIPE is particularly common among triathletes, with symptoms reported by 1.5% of 1400 responders to a survey (Miller et al. Am J Emerg Med 2010;28:941-6). In many cases the condition appears to be idiopathic, with no apparent underlying heart or lung disease. We hypothesized that the susceptibility to SIPE among triathletes is related to the normal age-related decline in LV compliance. We therefore tested the hypothesis that triathletes who experience SIPE are older than the general population of triathletes.

**Methods:** We compared the age distribution of published cases of SIPE among non-military swimmers and divers reported in the literature or responding to advertisements for an IRB-approved study (N=54) and compared this with the age distribution of triathletes obtained from a 2008 survey by USA Triathlon.

**Results:** The data are shown below.

Age	SIPE Cases	USA Triathlon
20-29	3	2,970
30-39	13	5,340
40-49	21	4,410
50-59	14	1,650
60-69	3	330
≥70	0	45
<b>TOTALS</b>	<b>54</b>	<b>14,745</b>

The age distribution of the SIPE cases is significantly different from USA Triathlon members (P=0.0028, Fisher's exact test). There were 64.8% females in the SIPE group vs. 39.5% among USA Triathlon members (P=0.00026).

**Conclusions:** We confirm that among triathletes increased age is a risk factor for SIPE. Females are at higher risk of SIPE, as has been previously observed.

**Acknowledgements:** Supported by NAVSEA Contract #N0463A-10-0005.

# D 54

ORAL PRESENTATION TIME: 1406 - 1418

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Measuring the Accuracy of Artisanal Fishermen's Underwater Depth Perception

Chin W, Huchim O, Joo E, Fang S, Sprau S

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**Introduction/Background:** Artisanal fishermen in the Yucatán Peninsula harvest marine protein for sustenance and profit. These divers use surfaced supplied air to dive for up to 5 hours per day but often dive without standard equipment, including depth gauges. Thus they estimate depth based on general sensory perceptions. We hypothesized that the fishermen would underestimate their depths because of underwater light refraction. Nitrogen narcosis could also contribute to the reduction of accuracy in their sense of perception.

**Materials and Methods:** Six fishermen belonging to the cooperatives of Río Lagartos were invited to participate. Sensus Ultra Dive™ recorders with an accuracy of  $\pm 1$  Foot of Seawater (FSW) were attached to their weight belts. The recorders activated at 3 FSW and recorded data every ten to thirty seconds. The fishermen were followed throughout each observed fishing day for two months. After each dive, they were asked to recall their estimated working depths in arm strokes (1 arm stroke = 1.86 meters).

### Results:

n = 120	Estimated Depth FSW	Recorded Depth FSW
Average	53.69	44.19
Standard Deviation	19.63	14.05
Median	54	43

Paired samples *t*-test showed a significant difference between the means of estimated and recorded depths,  $t(119)=7.3647$ ,  $p=0.000$  and  $r=0.70$ .

**Conclusion:** The survey results demonstrated that, on average, the fishermen significantly overestimated their diving depths. Experimenter bias may have caused the fishermen to report deeper depths because they may have been influenced to impress the researcher or provide values that they thought would have been more accurate. More importantly, even though the fishermen seemed to acknowledge the correlation between depth and bottom time and symptoms of bends, they dove long and reported diving deeper than they actually did. Such diving behavior suggests the fishermen's tendency to ignore potential risks to their health and safety in pursuit of a greater catch.

# D 55

ORAL PRESENTATION TIME: 1406 - 1418

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Diving Behavior and Decompression Stress among Artisanal Fishermen from the Yucatan Peninsula, Mexico

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**Introduction & Background:** Artisanal fishermen divers (AFD) dive for sustenance. The prevalence of decompression sickness (DCS) among this population is alarmingly high. The aim of this study is to understand the diving behavior in ten AFD.

**Materials & Methods:** Approved by the UCLA IRB two #13-000532, this study was conducted during fishing seasons 2012 through 2015. Consenting male fishermen, ages (41-49), were instructed to attach dive recorders to their waists during every fishing day throughout the study period. SENSUS ULTRA dive recorders (ReefNet Inc.), with an accuracy of  $\pm 1$  foot of seawater (FSW), were used to record parameters. Sampling interval was set to 10 seconds with an activation depth of 10 FSW. Data sets from recorders were downloaded onto desktop workstations and saved as comma separated values files. A subroutine in Microsoft Visual Basic© was created to extract the parameters of depth, bottom time (BT), dives, diving days (DD), and repetitive dives. Stata 13.1 was used for statistical analysis

Results:

Subjects	Dives	DD	Single Dives	Diving Behavior		BT Mean	Bounces
				Multiple Dives	FSW Mean		
SU-10527	404	141	138	266	50.04	94.69	1.83
SU-10779	116	66	60	56	34.37	195.29	3.44
SU-10782	441	153	62	379	42.58	117.98	4.88
SU-11156	46	36	35	11	34	198.77	2.02
SU-11160	339	106	9	330	42.4	110.88	2.73
SU-12511	260	90	9	251	38.41	117.92	3.40
SU-12512	186	105	186	0	29.57	183.36	3.36
SU-12514	366	88	366	0	51.77	73.73	2.32
SU-12515	353	118	353	0	40.63	106.1	3.28
SU-13241	49	15	49	0	37.78	105.67	1.62
<b>TOTAL</b>	<b>2,560</b>	<b>918</b>	<b>1267</b>	<b>1293</b>	<b>42.79</b>	<b>114.86</b>	<b>2.99</b>

**Discussion:** We recorded 2,560 dives. These exposures clarify the level of decompression stress these fishermen undergo. These dives, fishing yields, and diving behaviors will serve as input for a deterministic decompression table for these AFD.

# D 56

ORAL PRESENTATION TIME: 1418 - 1430

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Field Dive Monitoring: Bubble Presentation In Recreational-Technical Closed-Circuit Rebreather Trimix Diving

Pollock NW<sup>1,2</sup>, Wiley JM<sup>1</sup>, Kernagis DN<sup>2</sup>, Clarke NW<sup>1</sup>, Mackey MN<sup>1</sup>, Martina SD<sup>1</sup>

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**Introduction:** Recreational-technical diving has grown markedly over the past 20 years, particularly with closed-circuit rebreather (CCR) use. Divers increasingly rely on mathematical algorithms incorporated into dive computers to manage their decompression profiles, many of these promising to control bubble formation. There is often little or no human testing of such extended range exposures. We evaluated decompression stress in recreational-technical CCR dives conducted beyond 40 msw.

**Methods:** Observational studies were conducted on multi-day, deep recreational CCR trips. Divers controlled all of their own diving activity. Subject monitoring included high resolution two-dimensional echocardiographic imaging (GE Vivid q) at 20 min intervals for two hours post-dive. Bubble loads were scored on a semi-quantitative ordinal scale (0, I, II, IIIa, IIIb, IVa, IVb, IVc, V). Grades were recorded for both right and left heart, each in three conditions: rest; following three full engagement arm movements; and following three full engagement leg movements. Scores are reported as the highest of the three conditions for a given scan. Data presented as mean±SD with ranges, as appropriate.

**Results:** We summarize five research trips; capturing a total of 287 open water CCR dives conducted by 55 individuals (41 male, 14 female) (48±8 y, 27.2±4.0 kg·m<sup>-2</sup> body mass index). Maximum dive depth was 73±17 (40-157) msw (238±56 [131-515] fsw) and total run time 103±32 min. Right ventricular gas emboli (RVGE) were observed in all but one individual, with peak non-0 grades following 80% of dives and peak IIIa-V grades following 39%. Left VGE were observed in 33% of subjects and on 13% of all dives, peaking at grade IIIa-IIIb in five cases. Decompression sickness symptoms were noted in two cases.

**Conclusions:** Reflecting substantial decompression stress, the presentation of LVGE, high grade RVGE, and symptomatic DCS indicate that further evaluation of decompression procedures employed by recreational-technical divers is warranted.



# D 57

ORAL PRESENTATION TIME: 1430 - 1442

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Observed Incidence Of Decompression Sickness And Venous Gas Bubbles Following 18 M Dives on RN Table 11 / Norwegian Air Diving Table

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**Background:** When investigating the efficacy of interventions to reduce bubbling upon decompression, a dive profile that produces some bubbles in most subjects is desirable. An 18 m / 100 min dive profile, with stops at 6 m for 5 min and 3 m for 15 min (Royal Navy Table 11 / Norwegian Air Diving table) has been used, as it is known to produce a 'reasonable' amount of venous bubbles. This profile is more conservative than the exceptional exposures defined by the UK Military Diving Manual and the Health and Safety Executive's single dive duration limit.

**Materials and Methods:** Two-hundred and nineteen dives were made in hyperbaric chambers using this profile. Varying, controlled experimental interventions were tested, including nitric oxide, exercise, bed rest and multi-day diving regimens. Ultrasound Doppler or 2D imaging was carried out after all dives, with multiple measurements made up to two hours post dive on the Kisman Masurel (1) or Eftedal Brubakk (2) scales.

**Results:** Over 219 dives, only one case of DCS was noted (initially ankle pain), while there were two 'niggles' on surfacing; both manifested as shoulder discomfort that resolved without hyperbaric treatment. These results gave an incidence of DCS of 0.49% ( $\pm$  0.92 at 95% CI), or 1.4% ( $\pm$  1.53) if including niggles. Individual bubble grades ranged from zero to maximum, while the highest median maximum grade for different experimental series varied between 0 - 3 on the KM scale. According to the USN 93 probabilistic model (3) the pDCI of this profile is 3.7%.

**Summary:** Following the incidence of DCS reported here, there have been some worries that this profile is high risk. However, our data show the incidence of DCS is below predicted levels; it remains a useful profile for studies where bubbles are used as measure of DCS risk.

1. Kisman K, Masurel G, Guillerm R. Bubble evaluation code for Doppler ultrasonic decompression data. Undersea Biomed Res, 1978.
2. Eftedal O, Brubakk AO. Detecting intravascular gas bubbles in ultrasonic images. Med Biol Eng Comput. 31:627-33, 1993.
3. Thalmann, E.D., Undersea Hyperbaric Med 24, 255-274, 1997.

# D 58

ORAL PRESENTATION TIME: 1442 - 1454

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## The Association of Blood-Borne Microparticles and Neutrophil Activation with Decompression Sickness

Thom SR<sup>1</sup>, Bennett M<sup>2</sup>, Banham N<sup>3</sup>, Chin W<sup>4</sup>, Blake DF<sup>5</sup>, Rosen A<sup>6</sup>, Pollock NW<sup>7</sup>, Madden D<sup>8</sup>, Barak O<sup>8</sup>, Marroni A<sup>9</sup>, Balestra C<sup>9,10</sup>, Pieri M<sup>9</sup>, Cialoni D<sup>9</sup>, Le J<sup>11</sup>, Logue C<sup>11</sup>, Lambert D<sup>11</sup>, Hardy KR<sup>11</sup>, Sward D<sup>1</sup>, Yang M<sup>1</sup>, Bhopale VB<sup>1</sup>, Dujic Z<sup>8</sup>

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**Introduction:** Circulating microparticles (MPs), 0.1 – 1.0 µm vesicles, are elevated in animals and humans after simulated or *bona fide* underwater diving. Murine studies suggest that MPs play a role in high pressure gas pathophysiology and possibly with gas bubble nucleation. Maneuvers which decrease the incidence of decompression sickness (DCS) also diminish MPs production. However, data supporting a relationship between MPs and DCS in humans is lacking.

**Methods:** Blood was obtained from 254 SCUBA divers exposed to maximum depths from 7 to 64 meters; 159 were asymptomatic and 95 were diagnosed with DCS based on signs/symptoms occurring proximal to diving that improved with hyperbaric oxygen therapy. Blood was mailed and analyzed by published methods and results correlated with summary diving exposure data and DCS variables.

**Results:** Elevations of MPs and neutrophil activation occurred between 15 minutes and 6 hours post-diving in all divers, but resolved within 24 hours among those without DCS. The median time to clinical presentation and blood sample acquisition from divers with DCS was 25 hours, 82% had performed repetitive diving (median = 2). Logistic regression analysis documented significant associations ( $p < 0.001$ ) between DCS and MPs bearing surface markers for the following proteins: CD66b, CD41, CD31, CD142, CD235 and von Willebrand Factor; and for neutrophil activation assessed as membrane-associated myeloperoxidase. No significant associations were found between DCS and number of MPs without surface proteins listed above, use of alternative breathing gases, or with maximum dive depth.

**Summary:** MPs production and neutrophil activation exhibit strong associations with DCS. The temporal pattern of changes in neutrophils and MPs indicate that a persistent inflammatory process post-diving occurs in those with DCS.

# D 59

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## Headache in Military Divers

Heravi MK<sup>1</sup>, Ranjbar NA<sup>2</sup>, Salehi HA<sup>2</sup>, Khoshvaghti A<sup>2</sup>

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**Background:** Military Scuba divers work in stressing environments and have a high cerebrovascular risk, both conditions which are supposed to contribute to the genesis of cephalalgia. Headache occasionally occurs during or after scuba diving. Although its significance often is benign, headache may signal a serious neurological disorder in some circumstances.

**Methods:** Review research of medical electronic data bases from 2000 up to now.

**Results :** In addition to the usual causes of headache, the diagnostic evaluation should consider otic and Para nasal sinus barotrauma, arterial gas embolism, decompression sickness, carbon dioxide retention, carbon monoxide toxicity, hyperbaric-triggered migraine, cervical and temporomandibular joint strain, supraorbital neuralgia, carotid artery dissection, and exertional and cold stimulus headache syndromes.

**Discussion:** Correct diagnosis and appropriate treatment require a careful history and neurologic examination as well as an understanding of the unique physiologic stresses of the subaquatic environment. Focal neurologic symptoms, even in the migraineur, should not be ignored, but rather treated with 100% oxygen acutely and referred without delay to a facility with a hyperbaric chamber.

**Conclusion:** Even benign headaches, when moderate or severe in intensity, can distract the diver and pose a safety hazard underwater. The decision regarding fitness to dive should be made on an individualized basis, taking into account not only the headache history but also the overall health of the diver.

# D 60

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Evaluation of the Autopsy Findings in Fatal Diving Accidents in Turkey

Koca E<sup>1</sup>, Sam B<sup>2</sup>, Arican N<sup>3</sup>, Toklu AS<sup>1</sup>

<sup>1</sup>Department of Underwater and Hyperbaric Medicine, Istanbul Medical Faculty, Istanbul University, Istanbul, Turkey; <sup>2</sup>Autopsy Department, The Council of Forensic Medicine, Ministry of Justice, Istanbul, Turkey; <sup>3</sup>Department of Forensic Medicine, Istanbul Faculty of Medicine, Istanbul University, Istanbul, Turkey

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**Objective:** In this study it was aimed to reach the documents regarding fatal diving accidents at archives of the forensic medicine units of Turkish Ministry of Justice and Turkish Underwater Sports Federation, to evaluate the cases in terms of age, sex, reason of death, preexisting medical problem, types of diving, aim of diving, distribution according to years and months, and the techniques of autopsy.

**Material and Methods:** The records of fatal diving accidents were reached in the archives of last 7 years, by screening drowning cases at the forensic medicine units in Turkey, to review in accordance with aim of the study.

**Results:** A total of 52 autopsy reports regarding diving fatalities were reached. Out of the dives resulted with accidents, 28 dives were breath-holding, 20 dives were SCUBA and 2 dives were surface supplied. Most of the accidents were recorded between May and October. Majority of the cases (94%) were male and the average age was 38,6. The most frequent reported cause of death was drowning. The special autopsy techniques that should be performed in diving fatalities were done in 17% of the cases. Coronary arteriosclerosis detected in 38% of the cases and different alcohol levels in the bloods were found in 19% of the cases.

**Conclusion:** The documents we reached were not sufficient enough to be able evaluate the fatal diving accidents. The forensic physician who performs autopsy when there is a diving fatality, should be informed about the details of eventful diving, investigation of diving gears and diving accident scene. Establishing a standard archiving system and database about fatal diving accidents will enable to perform such studies in an easy way. The results of the evaluation of 52 cases were in accordance with previous studies.

# D 61

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Spontaneous Pneumothorax in a Professional Divers Candidate; Case Report

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**Introduction:** Spontaneous pneumothorax (SP) is considered as absolute contraindication for diving. The main concerns are the possibilities of the developing tension pneumothorax during ascent if a recurrence of SP occurs during a dive, and pulmonary barotrauma facilitated by sub pleural bullae or blebs. If there is some missing points in the medical history, intentionally or unintentionally given during the examinations for fitness to dive (FTD), it might not be possible to detect previous SP history by physical examinations and imaging.

**Case:** 19 year-old student enrolled in a two years program to be able to get professional diver license, after getting medical certification for FTD. In the middle of his first year, he had a sharp pain on his left side of thorax while resting and he applied to emergency department since the pain persisted. The chest x-ray revealed pneumothorax in the left side and he was admitted to the hospital. CAT scan of the chest confirmed the pneumothorax, but no bullae or blebs were detected. Videothoroscopic wedge resection and pleural abrasion were applied to the apex of the left lung, to prevent recurrences. During the operation no abnormality differing from normal lung tissue was detected visually, and pathological examination revealed emphysematous changes. The patient was discharged on 8<sup>th</sup> day after the operation. There was no air trapping lesion in control CAT scan. The patient was considered unfit to dive and he had to leave his program.

**Result:** Previous SP attack may not be detected if it is not reported in medical history. Chest x-ray, and even CAT scan may not detect air-trapping lesions such as bullae or blebs. Signature of the medical history given during FTD examination by the candidate might be legally protective for the physicians performing FTD examinations.

## D 62

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

### **Following the Surviving Sepsis Campaign Guidelines to Treat a Case Of Severe Decompression Sickness With Profound Shock**

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The US Navy Diving Manual concludes that corticosteroid is no longer recommended for the adjunctive treatment of decompression sickness (DCS) because of its ineffectiveness in reduction of neurological sequelae and its high risk in worsening the outcome of CNS injury. A growing amount of animal studies demonstrated that corticosteroids and chemicals inhibiting inflammation cascades may attenuate gas embolism-induced tissue injury. The Surviving Sepsis Campaign International Guidelines for Management of Severe Sepsis and Septic Shock suggests intravenous hydrocortisone at a lowest dose for patients of septic shock if adequate fluid resuscitation and vasopressor therapy are not able to restore hemodynamic stability. A severe DCS may share the pathophysiology of septic shock as a fulminant systemic inflammation, resulting in elevation of capillary permeability and profound shock refractory to initial recompression therapy, fluid resuscitation, and vasopressors. Currently, there is no feasible guideline for the short-term use of corticosteroid in the management of severe DCS with profound shock. We herein reported a case of severe DCS with rapid onset of multi-organ dysfunction syndrome (MODS) including conscious disturbance, acute respiratory failure, acute renal failure, and profound shock. After recompression therapy using US Navy Treatment Table 6A, his blood pressure was still barely maintained by high doses of norepinephrine and dopamine. The critical conditions were quickly reversed by continuously intravenous infusion of hydrocortisone at a dose of 8 mg/h. Patient recovered from MODS on the 3rd day and was discharged from hospital 2 weeks later without neurological sequela. We therefore suggest to follow the Surviving Sepsis Campaign International Guidelines for management of severe DCS with profound nonseptic shock.

# D 63

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## **Facial Nerve Palsy Secondary to Oxygen Toxicity in Closed-Circuit Diving: A Case Report**

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**Introduction:** Central nervous system oxygen toxicity is a known risk factor for divers participating in underwater diving operations who use closed-circuit oxygen rebreather systems. The general signs and symptoms are well documented in diving literature, but little is reported about atypical presentations of CNS oxygen toxicity.

**Case Presentation:** We report a case of a military diver who had an unusual presentation of CNS oxygen toxicity, and explore the differential diagnoses in this case. The diver did a dive on 100% oxygen, at a maximum depth of 5m and 85 min bottom time. On the surface, he had left sided facial asymmetry and weakness of the facial muscles. With his presentation of lower motor neuron facial nerve palsy, the initial differential diagnoses were oxygen toxicity, facial nerve baroparesis and Bell's palsy. The diver experienced significant improvement of symptoms within 4 hours of incident, and full resolution within 24 hours, thus confirming the diagnosis of CNS oxygen toxicity.

**Conclusion:** Oxygen toxicity affecting the central nervous system is common, but there has been no literature about oxygen toxicity affecting the peripheral nerve. This case highlights the signs and symptoms of oxygen toxicity may be more varied than previously described in the literature, and reminds clinicians to keep in mind both diving and non-diving pathologies when managing a diving incident.

# D 64

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Modeling Human Performance Limitations in the Submerged Environment

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**Introduction/Background:** Modeling respiratory and inert gas exchange in the submerged environment is beneficial in developing predictive tools to estimate human performance limitations, as well as tracking dynamic gas concentrations relevant to diving/decompression illness. Physical performance is a function of both locomotor and respiratory muscle fatigue, and the performance-limiting factor is not always obvious in the submerged environment where additional breathing resistance can increase the lung workload.

**Materials and Methods:** The Dynamic Physiology Model (DPM) was developed to predict real-time human physiologic response, physical performance and endurance based on environmental exposure and exercise demand. The model incorporates a dynamic ventilatory system, whole-body circulation, and muscle metabolism. The DPM was expanded to account for diver-specific conditions related to the use of rebreathers such as increased breathing resistance and inspired CO<sub>2</sub> fraction. The DPM is able to predict work of breathing and total energy of respiration, as well as locomotor muscle fatigue. Simulations were completed under conditions relevant to underwater rebreathing with exercise to compare with published experimental results.

**Results:** The model accurately predicts minute ventilation (VE) and fraction of expired CO<sub>2</sub> responses to exercise under increased breathing resistances and inspired CO<sub>2</sub> concentrations of 1% to 3%. Increasing the O<sub>2</sub> supply to 100% mitigated the reduction in endurance caused by increased resistance and CO<sub>2</sub>. A logistic regression analysis was performed with model-predicted work of breathing and published human endurance data which correlates respiratory fatigue and work of breathing in a dose-response dependent manner.

**Summary/Conclusions:** The modified DPM is able to predict human performance limitations for diver-specific conditions such as rebreather use. The model can be applied to any dynamic exercise task and predicts respiratory and inert gas exchange, locomotor muscle fatigue, and respiratory muscle fatigue.



# D 65

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## **An Interim Report on Eurosia Tunnel Project**

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Istanbul Strait Road Tube Crossing Project (Eurasia Tunnel Project) will connect the Asian and European sides via a highway tunnel going underneath the seabed of Bosphorus. A Tunnel Boring Machine (TBM) designed exclusively for this project is being used in the strait crossing tunnel. The tunnel construction was started on the Anatolian side of Bosphorus and about 2 km of the tunnel underneath the seabed was bored. Four interventions into the pressurized heading of the TBM through a man-lock were needed for inspection, maintenance and servicing the cutter head, since the TBM started to advance. The first intervention was performed by bounce diving on air to the pressure of 4,2-4,6 bar without using a diving helmet. Saturation dives were needed for the other three interventions and in each intervention three divers saturated on trimix that contains 2 % Oxygen, and 6 % Nitrogen to 10 bar. Interventions were done on bottom mix that contains 4 % Oxygen and 12 % Nitrogen balanced by Helium, by using surface supplied equipment. The longest saturation lasted 16 days including 97 hours decompression time. Medical support were given by four diving physicians and always a doctor was kept at job site, another another one on call. There was no significant health issue encountered during the saturation except some minor ear problems. The divers were examined before and after saturation. Bubble check were done by Doppler device on subclavian vein and pulmonary artery and no bubble detected within 20 minutes after the decompression. The minimum and maximum oxygen fractions were changed from 4,5 % to 21 % and from 5,1 % to 23 during the decompression from 10 bar to 0 bar. The Eurasia Tunnel Project was planned to be completed by the end of 2016.

# D 66

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## **Educational Intervention Among Artisanal Fishermen of the Yucatán Peninsula, Separating Engine Exhaust Gases from Compressor Intake.**

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**Introduction Background:** Artisanal fishermen use surface supplied compressed air to dive for sustenance and profit. Entrainment and wind patterns affecting gasoline engine exhaust (GEE) cause carbon monoxide (CO) to be compressed into volume tanks aboard the fishing boats<sup>3</sup>. Simple separation of GEE from compressor intake has been shown to drastically reduce CO in the volume tanks<sup>4</sup>. Focus groups and educational interventions in these artisanal fishermen helped establish a clear intervention model. We sought to determine the efficacy of our previous educational interventions after a one-year period in the fishing village of Río Lagartos.

**Materials Methods:** In 2014, focus groups of fishermen provided insight into concerns regarding impacts of the proposed intervention. Fishermen from one cooperative participated in a workshop with schematics, power point presentations, and models illustrating the importance of separating GEE and compressor intake. Posters explaining the intervention were hung in 6 fishing cooperatives. After 1 year, we visited Río Lagartos, counted boats using the intervention, and interviewed local fishermen.

**Results:** After 1 year, the number of boats using the gas separation intervention increased from 7 to 34 of 198 total boats. Supplies used for the intervention varied, especially with hose material. The lack of a particle filter and direct connection of the hose to the air intake were common modifications to the original intervention.

**Conclusion:** The simplicity of the intervention, its efficacy, and the collaboration of fishermen in the design helped with its adoption by other fishermen. Workshops in surrounding communities may help continue spreading this efficacious, simple intervention.

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<sup>3</sup>*CO and CO<sub>2</sub> Analysis in the Diving Gas of the Fishermen of the Yucatan Peninsula.* Undersea and Hyperbaric Medicine Journal 2014; 41(5): 480

<sup>4</sup>*Modeling Carbon Monoxide Reduction in a Single-Compressor Hookah Dive System.* Undersea and Hyperbaric Medicine Journal 2014; 41(5): 481

# D 67

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## **Reverse Takotsubo Cardiomyopathy in a Scuba Diver**

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**Introduction/Background:** A 70 yo female developed reverse Takotsubo cardiomyopathy with exertional swimming following a scuba dive. Takotsubo Cardiomyopathy, typically seen in acute illness or stress, is characterized by hypokinesis of the apex, described as “apical ballooning.” Rarely, patients present with reverse Takotsubo cardiomyopathy, which demonstrates preservation of apical function and hypokinesis of the basal and midventricular segments. To our knowledge, this is the first reported case of reverse Takotsubo cardiomyopathy following a scuba dive.

**Materials/Methods:** 70 yo female presented with chest pain after a scuba dive to 65 ft for 28 min on air. The dive was uneventful with controlled ascent. While swimming back to the boat, she developed substernal chest pressure and dyspnea. She was placed on oxygen and taken to the Emergency Department (ED).

**Results:** In the ED, EKGs showed borderline ST elevation and she had troponin elevation. Her exam was normal, except for missed “serial 7s” on mini-mental status exam. Chest radiograph was normal. Emergent cardiac catheterization showed no significant coronary artery disease, but did reveal hypokinesis of the left ventricle. Echocardiogram showed decreased ejection fraction (31%), preservation of systolic function at the apex and otherwise global hypokinesis. She was diagnosed with reverse Takotsubo cardiomyopathy, thought to be induced by extreme exertion while swimming after scuba diving. She remained stable and was discharged home.

**Summary/Conclusions:** The stress of exercise associated with diving and swimming can induce reverse Takotsubo cardiomyopathy, which may mimic coronary artery disease, pulmonary edema, decompression illness, or arterial gas embolism.

# D 68

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## Outcomes of Decompression Sickness Treated using UCSD Modified Treatment Table 6

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**Introduction/Background:** Decompression Sickness (DCS) is a well-recognized complication of scuba diving that has been described in the literature for years. Hyperbaric oxygen therapy is the standard treatment for DCS. However, outcome measurements have been difficult to assess, due to a lack of standardized diagnostic criteria and outcome measures. Additionally, treatment protocols vary among treatment facilities. At UCSD, we use a modified Navy Treatment Table 6 (will include image) and to our knowledge, we are the only center in the country using our protocol. The aim of our study was to review cases of DCS, using previously studied diagnostic criteria, and to assess the outcomes of patients after treatment at our facility. We reviewed the charts of every patient treated for DCS at UCSD from 1994-2014 in order to assess the outcomes of our treatment protocol. We hypothesized that our treatment protocols adequately treat DCS.

**Materials/Methods:** 325 charts reviewed. 87 patients met diagnostic criteria for DCS based on SANDHOG criteria of  $\geq 3$  points. We evaluated patients based on presenting symptoms and classified them as mild or moderate/severe. We then evaluated symptoms at follow up.

### Results:

Presenting Symptoms	At Follow up			
	Resolved	Mild	Moderate/Severe	Death
Mild (n=37)	28	9	0	0
Moderate/Severe (n=50)	23	13	12	2

Those with residual symptoms at follow-up were more likely to have presented with moderate/severe symptoms ( $p=0.005$ ). Overall, 58.6% of cases had complete resolution and this is commensurate with what has been seen with other protocols. At follow up, 93.3% of those presenting with mild symptoms and 90.9 % of those with moderate/severe symptoms had improved.

**Summary/Conclusions:** Similar to outcomes seen in other studies, the vast majority of our patients with DCS improved with hyperbaric treatment. We believe that using the UCSD Modified Treatment Table 6 is an effective treatment for DCS.

# D 69

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## **External Auditory Canal Diving Related Barotrauma – A Case Study**

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**Introduction/Background:** We report a case of asymptomatic barotrauma of the external auditory canal (EAC) involving a public safety diver following a non-provocative dive profile.

**Materials and Methods:** A 36 year old male presented to our dive clinic with left sided hemorrhage from the EAC following scuba diving. The patient works as a public safety diver and presented following a routine fresh water dive training exercise. Based on diver obtained history he had completed a total of 84 dives, the deepest being to a depth of 104 feet with no known barotrauma. On the day of injury, the patient completed only one dive to 67 feet of fresh water (FFW). The descent took 1 minute and 14 seconds. His total ascent time was 6 minutes and 17 seconds. His total dive time was 12 minutes. He was using a dive computer and reported no ascent rate warnings. He had no difficulty equalizing middle ear pressure on the day of injury. On arrival at surface he was asymptomatic. When leaving the dive site his fellow officers noticed bleeding from his left ear.

**Results:** On examination he was found to have bilateral O'Neill Grade 2 barotrauma with intact hearing. The bilateral bleeding subsided, however the source of bleeding could not be identified. He was referred to Neuro-Otolaryngology who diagnosed the patient with bilateral external auditory canal hematomas obscuring the TM's. These were evacuated after which the TM's were easily visualized and intact. Both the audiogram and tympanogram were within normal limits.

**Summary/Conclusions:** We present a case of asymptomatic external auditory canal hematomas from shearing forces after a non-provocative dive profile without hearing deficits. We have not found any significant literature on dive related barotrauma resulting in external auditory canal hematomas. Further studies are warranted.

# D 70

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## **Bleeding Gums and a Pain in the Face: The Dental Health of UK Divers**

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**Background:** Scuba divers should ensure a high standard of dental health and receive dental checks regularly. Studies have discussed the barodontalgia, and orofacial barotraumas of scuba divers, but few have attempted to observe the broad state of dental health in recreational divers. As part of an ongoing study of the health of UK recreational divers, we attempted to gain an insight into the general dental health of divers and other related problems experienced.

**Methods:** An anonymous on-line survey was publicised through diving exhibitions and social media. Measures included diver and diving demographics, frequency of dental checks, dentist's knowledge of diving, dental work, tooth extractions, bleeding gums, perceived maxillofacial pain experienced whilst diving, water temperature, and aborted dives due to pain.

**Results:** Data were provided by 416 respondents (females 33% males 67%), age range 16-70 (median 44). Dental checks every 6 or 12 months were reported by 78%, with 22% only attending when experiencing a problem or not attending at all. Overall 38% reported their dentist was unaware of their sport diving activities, with 10% unsure, and some respondents reporting dentists unacquainted with the implications of scuba on dental health. Maxillofacial pain had been experienced by 240/416 (57%) whilst diving; 78/416 (19%) had aborted a dive due to pain associated with ascent, descent and/or water temperature. Blood in the mask when surfacing was reported by 118/416 (28%) respondents, with 30/118 (25%) of this group experiencing sinus pain whilst diving with a cold. In total 42% of respondents reported bleeding gums when cleaning their teeth.

**Conclusions:** Not all divers in this study maintained a high standard of dental health. Divers should be encouraged to pay more attention to their dental health and inform their dentist of their diving activities. Sinus barotrauma was relatively common in this group.

# D 71

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Human Accommodations to Diving in Cold Water

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**Introduction:** At the 2006 UHMS meeting the lead author presented an overview of mammalian adaptations to diving and their counterparts in human divers. This presentation describes the accommodations humans make to meet the challenges of diving in cold water and compares and contrasts them with those found in diving mammals.

**Methods:** Although body mass effects and subcutaneous fat influence tolerance to cold water, other factors in our review of the literature were found to offer protection in humans and occur as adaptations in diving mammals. This information provides rationale for preventative and emergency measures to mitigate hypothermia in human divers.

**Results:** The primary measure the human diver utilizes to prevent immersion hypothermia is the exposure suit. This is, in effect, what the diving mammal achieves with its fat tissues. However, other means to prevent hypothermia include 1) Shunting of blood flow to the extremities, 2) Monitoring of respiration 3) Optimizing metabolic heat production and 4) Countercurrent heat exchange. These protective measures have variable degrees of expression in human divers. When effective, they complement cardiovascular, metabolic, and respiratory adaptations to diving.

**Conclusions:** Anatomical and physiological features make it possible for diving mammals to tolerate cold water and most have counterparts in humans. Of all mammals' adaptations to diving, the tolerances to cold water have the most counterparts in humans. Education in methods to conserve body heat in cold water have importance for preventing hypothermia in divers and victims of accidental immersion.

# D 72

ORAL PRESENTATION TIME:  
POSTER PRESENTATION TIME: 1530 - 1600  
RESIDENT COMPETITION:

## Predictive Model of Severe Decompression Illness in Artisanal Fishing Divers of the Yucatan Peninsula

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**Introduction/Background:** Artisanal fishermen use surface supplied air to dive for marine protein. Many of the fishermen experience DCS annually. We wanted to identify predictors of repetitive DCS in this population.

**Materials and Methods:** Retrospective review of 115 fishermen divers treated for DCS from 4 fishing villages during the first seven months of the 2014-2015 fishing season was conducted from the Hyperbaric Center in Tizimin, Yucatan, Mexico. Symptom Severity Scale (SSS) was formulated based on symptoms present upon admission. Symptoms were scored using a two-point scale (0-1) and assigned an importance factor from 25%-100%, where 0 means no symptoms and 100% means a critical symptom, e.g. paralysis. Univariate and bivariate analysis of variables were used to build a binary logistic regression model (BLRM), using length of fishing years (FY) and SSS. Break for the outcome was set at a prevalence of DCS of more than 5.

**Results:** DCS symptoms included upper and lower extremity pain (58%), thoraco-abdominal pain (49%), ecchymosis (13%), and inability to walk (13%). Minor symptoms included nausea, vomiting, vertigo, and pruritis.

Binary Outcome	≥ 5 DCS Incidents (n = 66)	< 5 DCS Incidents (n = 49)			
Predictors	Mean ± Std.Err	Mean ±Std.Err	Odds Ratios	p-value	95% C.I.
FY	20.72 ±.913	13.49 ±1.16	1.09	.004	1.02-1.16
SSS	17.85 ±.726	10.70 ±.723	1.28	.001	1.13-1.38
Constant	.0124	.011	.0021706	.001	.0021-.0719

N	115
LR chi2(2)	50.07
p-value	.0001
Log likelihood	-53.41

One-unit change in the log of the odds for FY predicted outcome.

**Summary/Conclusion:** Group comparisons separated by FL showed that prevalence of DCS becomes asymptotic after 55 years. Fishermen with repeated injuries were more symptomatic. One possible explanation is comfort of vocalizing symptoms after repetitive DCS treatment.



# D 73

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Recreational Diving Fatalities: Harvesters Versus Non-Harvesters

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**Introduction:** Marine life harvesting is popular in North America among mainly male recreational divers. Additional risks faced, compared with non-harvesters, remain unquantified.

**Methods:** Records for adult male divers (n=774) in North America from 2004-2014 were identified from the DAN Diving Fatality Database. Non-recreational divers were excluded. Divers engaged in harvesting (hunters, n=110) were compared with non-hunters (n=290).

**Results:** Of the 400 fatalities, 39 (10%) were in Canada and 361 (90%) the United States. Florida (n=51, 46%) and California (n=27, 25%) accounted for the majority of hunters (n=78, 71%). Surface supply was more common among hunters (n=4, 4% vs. n=3, 1%), as was basic/open water certification (n=13, 12% vs. n=30, 10%).

Anthropometric and dive history data are presented in Table 1.

Table 1. Recreational divers (n=400) anthropometry and dive history by hunting status

		Hunters (n=110)	Non-Hunters (n=290)	Overall (n=400)
<b>Anthropometric</b>				
Age (years)	$\bar{x}$ (SD)	46 (13)	49 (12)	48 (12)
BMI (kg·m <sup>-2</sup> )	$\bar{x}$ (SD)	30 (5)	29 (6)	30 (6)
Marital status	n (% single)	56 (51)	142 (49)	198 (50)
<b>Dive History</b>				
>61 dives experience	n (%)	36 (33)	76 (26)	112 (28)
Dives in last year	$\bar{x}$ (SD)	16 (15)	24 (31)	22 (27)
Max depth fatal dive	$\bar{x}$ fsw (SD)	67 (51)	80 (66)	76 (62)
	$\bar{x}$ msw (SD)	20 (16)	24 (20)	23 (19)

Compared with non-hunters, boat diving (n=92, 84% vs. n=167, 58%, p=0.0002), solo diving (n=28, 25% vs. n=36, 12%, p=0.008) and night diving (n=13, 12% vs. n=8, 3%, p=0.001) were more common among hunters. Of the divers who were low-on or out-of air, n=20 (18%) were hunters and n=30 (10%) non-hunters (OR=1.9, p=0.04).

**Conclusions:** Diving differences between hunters and non-hunters may influence cause of death. Most fatalities involving hunters occurred in Florida and California, therefore safety interventions should target those two states.

# D 74

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## Arterial Gas Embolism During a Dive

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**Introduction/Background:** A healthy SCUBA diver presented with a NSTEMI, pneumothorax and pneumo-mediastinum after diving.

**Materials/Methods:** A 34 year-old woman, experienced diver with no significant medical history, presented to the emergency department after an episode of loss of consciousness and seizure-like activity immediately after an uncontrolled ascent while diving in a fresh-water lake at 6,000 feet.

Her initial chest radiograph showed pneumothorax and pneumo-mediastinum, septal infarction by ECG, and elevated troponin. Echocardiogram revealed a mildly depressed LVEF with apical-septal akinesis and infero-septal hypokinesis. A cardiac catheterization showed normal coronaries.

Hyperbaric oxygen was not given since she was evaluated 28 hours after the event, had a pneumothorax and was cognitively without complaint and was neurologically normal. She was discharged with an ACE-I and opioids for chest pain.

A resting echocardiogram showed normalization of cardiac function 2 weeks later, but her continued complaints supported microvascular insufficiency treated with long-acting nitrates and dihydropyridine calcium channel blockers for 1 month. Also she developed pericarditis confirmed by C-MRI- requiring escalating treatment with NSAIDs, Colchicine and steroids.

Five months after her initial presentation, the patient had returned to her usual level of activity with resolution of symptoms.

**Summary/Conclusions:** Reports of myocardial injury caused by arterial gas embolism are rare. This case shows -coronary air embolism complicating pulmonary barotrauma. Moreover, this case shows how severe cardiac injury can occur after resolution of neurological involvement. This may be the first case of pericarditis complicating myocardial injury after an AGE.

# D 75

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## Can My Patient Dive After a First Episode of Primary Spontaneous Pneumothorax?

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**Background:** It is conventional wisdom that individuals with a history of primary spontaneous pneumothorax (PSP) should never dive. We performed this analysis to look for hard evidence to support or reject this recommendation.

**Methods:** We conducted a systematic literature review on the recurrence of PSP in the general population. Using pre-defined search criteria in PubMed, in addition to a hand search, 482 publications were evaluated. Only those based on original research were reviewed. After excluding reports that combined patients with primary and secondary pneumothorax or that lacked follow-up beyond one month, we included 38 articles in the final analysis.

### Results:

Table 1

Summary of Included Studies	
Number of studies	38
Mean follow-up time	36 months
Follow-up range	10.7-120 months
Mean time to first recurrence	10 months
Overall incidence of recurrence	17.3%
Recurrences in first 12 months	12.5%
Recurrence after VATS* for 1 <sup>st</sup> episode	0-2%

\*Includes combined populations of pleurodesis and bleb/bullae resection

Table 2

Studies with CT Scan and Survival-type analysis						
	Studies(N)	Mean f/u	Average Recurrence	RFS 1y.	RFS 2y.	RFS 3y.
Patients with normal CT	2 (231)*	44 months	10%	93%	87%	86%
Patients with blebs/bullae	2 (231)*	44 months	37%	71%	68%	58%
Patients with "high-grade" blebs	1 (176)*	58 months	57%	50%	30%	22%

RFS: Recurrence free survival; \*0% of these patients underwent VATS after their first PSP episode.

Table 3

Studies with CT evaluation after first PSP occurrence						
Author	Year Published	n	Mean f/u time	Overall Recurrence	Recurrence with blebs/bullae	Recurrence w/o blebs/bullae
Casali	2013	176	58 mo	57.0%	Ipsi-68.1%/contra-19%	Ipsi-6.1%/contra-0%
Martinez-Ramos	2007	55	30.7 mo	24.0%	26%	24%
Mitlehner	1992	35	31.7 mo	23.0%	N/R*	N/R*
Ouanes-Besbes	2006	80	34 mo	19.0%	11.2%	7.5%
Sihoe	2000	28	59 mo	18.0%	contra-26.7%	contra-0%
Huang	2007	231	92 mo	14.30%	Ipsi-1.5%/contra-26%	Ipsi-0%/contra-0%

\*Recurrence rates described as significantly higher in patients with blebs/bullae although not reported; CT: computed tomography; PSP: primary spontaneous pneumothorax; N/R: not reported; Ipsi-: Ipsilateral/Contra: contralateral to side of first PSP occurrence

**Conclusions:** Average recurrence of PSP in the general population is estimated at 17.3% over the first 3 years of follow-up. Recurrence after surgery, combining patients with pleurodesis and bullectomy, ranges 0-2%. Recurrence varies significantly according to the presence or absence of blebs/bullae on CT scan: 29.5% and 5.4% respectively, without surgical intervention after the first episode. Seventy-two percent of all recurrences occur within the first year, and follow-up beyond 10 years is nonexistent. Although exposure to increased gas density and high trans-pulmonary pressures while diving can increase the risk of pneumothorax recurrence, it seems that in the absence of thoracic imaging abnormalities, patients who have not experienced a recurrence after several years of follow-up are at low risk of recurrence and could be considered for clearance to engage in recreational diving.

# D 76

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Decompression Sickness (DCS) After Chamber Dive to 18 msw for 100 min

Møllerløkken A<sup>1</sup>, Blogg SL<sup>1</sup>, Mueller B<sup>2</sup>, Risberg J<sup>2</sup>, Eftedal I<sup>1</sup>

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**Background:** This case study details a reported DCS following an 18m chamber dive for 100 min.

**Methods:** Chamber dives were made studying multi-day repeated exposure on vascular function, bubble production and stress biomarkers. Study subjects (n = 11) underwent four dives each, with 72 h between each dive. The dive profile required 5 min decompression at 6 m, and 15 min at 3 m. Echsonography, flow mediated dilation measurements and blood-samples were made post-dive,

**Results:** Immediately after diving, the subject of this case did not report any problems. Echsonography was not carried out, as having missed the second dive, only blood samples were taken. The subject went home feeling well after 4.5 h. The next day the subject reported feeling cold in both feet with pain in his ankles. As DCS could not be ruled out, the diver was transported to the chamber facility. Just before start of treatment, symptoms and signs had increased. Initial treatment with US Navy Table 6 brought partial improvement. After three additional treatments (100% oxygen for 90 minutes daily at 2.4 ATA) the diver was symptom free. MRI of cerebrum and spinal cord did not reveal pathology.

**Summary:** The cause of this outcome is interesting for several reasons. The subject missed his second dive due to involvement in a car crash, after which he reported feeling fine. The next day he made the dive, then returned home and shoveled snow wearing ankle-exposing shoes, then reported being cold for the rest of the night. Coldness and physical activity may have triggered DCS. The car crash confused the diagnostic considerations, especially regarding the symptomatic pain. However, the additional symptoms and gradual therapeutic response point to a combination of joint and central nervous system DCS as the correct diagnosis. The subject is currently symptom free.

# **PLENARY: “NEW PEARLS OF WISDOM IN THE DIVING AND HYPERBARIC MEDICINE LITERATURE”**

**Gerald Godfrey, MD  
Charlotte Sadler, MD**

**4:00PM – 5:00PM**

**Gerald Godfrey, MD: "New Pearls of Wisdom in the Hyperbaric Medicine Literature" will review three articles in hyperbaric medicine published over the approximate last 12 months.**

Articles so included are:

1. CD 34+/CD45- dim stem cell mobilization by hyperbaric oxygen - changes with oxygen dosage. M. Heyboer et al. including S.Thom; February 28, 2014 - Elsevier-Science Direct.
2. Effect of Hyperbaric Oxygen Treatment on Irradiated Oral Mucosa: Microvessel Density. J. Svalestad et al.; International Journal of Oral Maxillofacial Surgery (2015)
3. Effects of Hyperbaric Oxygen on Symptoms and Quality of Life among Service Members with Persistent Post-concussion Symptoms: a Randomized Clinical Trial. R. Miller, L. Weaver et al.; JAMA Internal Medicine 2015;; 175 (1):43-52.

Goals of the lecture are to familiarize the audience with the conclusions and limitations of each study and to appreciate findings that may affect patient management in the future, or provide the basis for further investigation.

**Charlotte Sadler, MD: “New Pearls of Wisdom in the Diving and Hyperbaric Medicine Literature”**

The purpose of this lecture is to review the most relevant diving literature published within the past year. This is limited to primary research, both basic science and clinical, and does not include case reports, reviews, etc. Objectives of this lecture include:

1. Review the most relevant diving literature published within the past year
2. Briefly discuss emerging and future areas of research still needed

**SATURDAY, JUNE 20**

# PLENARY: “INTERNATIONAL PERSPECTIVES: UPDATES ON CLINICAL RESEARCH PROJECTS IN HYPERBARIC OXYGEN THERAPY”

**Folke Lind, MD**

**Nicklas Oscarsson, PhD**

**Ian Millar, MBBS**

**8:00AM – 9:00AM**



**Folke Lind**, Stockholm, Sweden:

Hyperbaric oxygen treatment of cerebral abscess and other neurosurgical infections



**Nicklas Oscarsson**, Gothenburg, Sweden:

The Scandinavian “Rich-art” randomized radiation cystitis trial design

**Ian Millar**, Melbourne, Australia: The international randomized Hyperbaric Oxygen in Lower Limb Trauma (HOLLT) clinical trial design and preliminary results

This plenary draws attention to the clinical use of hyperbaric oxygenation to combat 1) acute CNS infections including spontaneous cerebral abscesses and 2) refractory late radiation cystitis mainly in men having received curative radiation for prostate cancer and 3) the prophylactic use of HBO<sub>2</sub> in severe open tibia fractures/ crush injuries. Trial design and preliminary results, pending publication, will be presented and discussed.

## OBJECTIVES:

- To understand the use of HBO<sub>2</sub> to treat severe CNS infections
- To understand possible advantages of prophylactic HBO<sub>2</sub> in severe trauma/ischemia/reperfusion injuries
- To understand possibilities for creative randomized research design when double-blindin is not feasible.

**SESSION E**  
**HBO<sub>2</sub> Therapy, Chambers and Equipment**  
Moderators: Lin Weaver, MD & Gus Gustavson, ACHRN

**SATURDAY, JUNE 20**  
**9:00AM – 11:30AM**



# E 77

ORAL PRESENTATION TIME: 0900 - 0912

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Performance of the Zyno Medical Z-800F, CME Body Guard 323 Color Vision™ and Baxter Flo-Gard® 6201 Infusion Pumps For Monoplace Chamber Use

Bell JE<sup>1,2</sup>, Deru K<sup>1,2</sup>, Koumandakis G<sup>1,2</sup>, Weaver LK<sup>1,2,3</sup>

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**Introduction:** We evaluated accuracy of the Zyno Medical Z-800F, CME Body Guard 323 Color Vision™ and Baxter Flo-Gard® 6210 infusion pumps with monoplace chamber conditions.

**Methods:** Occlusion pressure was adjusted for all pumps, allowing infusion up to 3 ATA. The Baxter and Zyno pumps were connected to the chamber pass-through with rigid prenatal tubing. The CME infusion set would not allow this setup and was connected directly to the pass-through. We tested pump accuracy and performance with saline running to a collection manifold inside a monoplace chamber at flow rates from 1 to 100 ml/hr under pressures ranging from ambient (0.85 ATA) to 3.0 ATA. Each condition was repeated 3-5 times. Averaged results are presented.

**Results:** At ambient pressure, all pumps performed within ECRI Institute's tolerance of  $\pm 5\%$ . Using a carbon monoxide treatment protocol (3 ATA/2ATA) and measuring flow door-to-door, the Baxter, CME, and Zyno pumps performed within  $\pm 5\%$  at 10 ml/hr (95%, 103%, 95%, respectively); at 1 ml/hr, the pumps did not meet this standard (91%, 83%, 83%). During timed testing for accuracy (total volume recorded just before decompression), pump accuracy was within 10% at 100 ml/hr and 10 ml/hr, but tubing capacitance compromised performance at lower flow rates, magnified by increased pressure.

Flow Rate (ml/hr)	Pressure (ATA)	% of expected delivery		
		Baxter 6201	CME 323	Zyno Z-800F
100	2.0	96	106	100
	2.4	99	105	101
10	2.0	101	94	96
	2.4	93	96	95
	3.0	92	91	94
5	2.0	96	97	97
	2.4	91	97	90
	3.0	89	81	85
1	2.0	70	73	83
	2.4	40	57	80
	3.0	65	40	50

**CONCLUSIONS:** These infusion pumps have potential for monoplace hyperbaric use, though this application is not approved by the manufacturers or the FDA. At low fluid delivery rates, tubing compliance affects delivered volumes.

# E 78

ORAL PRESENTATION TIME: 0912 - 0924

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## **Preliminary Evaluation Of The Zyno Z-800f, Cme 323 Color Vision And Alaris Med System Iii Infusion Pumps For Use In The Multiplace Chamber**

Bell JE,<sup>1,2</sup> Deru, K,<sup>1,2</sup> Koumandakis G,<sup>1,2</sup> Weaver LK.<sup>1,2,3</sup>

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**Introduction/ Background:** Several infusion pumps have been evaluated for performance and safety inside the multiplace hyperbaric chamber. Although we use the Alaris MedSystem III pumps (pressurized to 3 ATA/44.1 psia regularly), we performed preliminary testing of the Zyno Medical Z-800F and the CME 323 Color Vision infusion pumps.

**Materials/Methods:** Both the Alaris and the two tested pumps use non-rechargeable lithium batteries for clock and memory function, and do not currently meet NFPA 99 chapter 14 requirements. Nevertheless, we have internally approved these batteries for use in our chamber as they are contained within the devices, do not recharge, are not exposed to heat, and were not damaged by pressure testing to 90 psig/200 fswg. While the Z-800F main battery (nickel metal hydride) met NFPA 99 chapter 14 requirements, we replaced the CME pump's main lithium polymer battery with an accessory 9V alkaline battery pack. To assess risk, we completed unmanned pressurization of the Z-800F and CME 323 in our test chamber (90 psig/200 fswg, nitrogen) and manned pressurization (2 ATA/29.4 psia, air) in our multiplace chamber.

**Results:** We completed three unmanned pressurizations to 90 psig for each pump in the test chamber and two manned pressurizations to 2.0 ATA in the multiplace chamber. The CME 323 Color Vision continued to perform within manufacturer tolerances for all test conditions. However, the Z-800F shut down before reaching 2 ATA and did not operate at pressure.

**Summary/Conclusions:** This application is not approved by the manufacturers or the FDA. We plan continued use of the Alaris MedSystem III, its main battery (NiCad) meets NFPA 99 chapter 14 requirements. Further study is warranted for the CME 323 Color Vision. However, the Z-800F was most likely affected by hyperbaric pressurization and will not work inside the multiplace chamber without design modification.

# E 79

ORAL PRESENTATION TIME: 0924 - 0936

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Hyperbaric Oxygen Therapy Effects on Blood Pressure

Heyboer M, Smith G, Santiago W, Wojcik S

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**Introduction/Background:** Previous studies suggest an increase in blood pressure with hyperbaric oxygen therapy (HBOT). We sought to further quantify blood pressure (BP) changes in patients undergoing HBO and identify risk factors.

**Materials and Methods:** Data was prospectively collected on pre and post HBOT BP changes in patients undergoing HBOT from March 2012 – December 2014. Additional data including specific anti-hypertensive therapy and HBOT treatment parameters was collected retrospectively

**Results:** Data is reported on 145 patients who underwent a total of 2952 treatments. There was a median increase in MAP of 5.3mmHg, systolic BP of 7mmHg, and diastolic BP of 4mmHg. There was no significant difference in BP change when comparing those with hypertension (57.5%) versus those without hypertension (42.6%). When comparing different anti-hypertensive medications among those with hypertension, there was a more pronounced increase in patients taking angiotensin receptor blockers (MAP 7mmHg vs. 5mmHg,  $p=0.04$ ) and beta-blockers (MAP 6.3mmHg vs. 4.3mmHg,  $p=0.02$ ). There was a decreased effect in patients taking ACE inhibitors (MAP 4.3mmHg vs. 6mmHg,  $p=0.03$ ). When comparing treatment pressure the median MAP change was 6.3mmHg (2 ATA), 4.7mmHg (2.5 ATA), and 3.5mmHg (2.8 ATA,  $p<0.0001$ ). Finally, there was an increase in MAP with longer treatment time which was not statistically significant (90 minute: 5mmHg vs. 120 minute: 6.3mmHg,  $p=0.68$ )

**Summary/Conclusions:** The results of this study demonstrate an overall increase in blood pressure in those undergoing HBOT regardless of the presence of hypertension. Among those with hypertension, angiotensin receptor blockers and beta blockers appear to extenuate this effect, while ACE inhibitors were protective. Finally, these results suggest less effect with higher pressure which seems counter intuitive and warrants further investigation.

# E 80

ORAL PRESENTATION TIME: 0936 - 0948

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## Effects of Hyperbaric Oxygen Therapy on Diabetic Serum Glucose Levels: An Extended Study

Swaby JA, Morgan M, Jennings MS, Santiago W, Heyboer M

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**Introduction/Background:** It is believed that serum glucose levels decrease in diabetic patients undergoing hyperbaric oxygen therapy (HBOT). General practice guidelines within the hyperbaric medicine community require a minimum glucose level (120 mg/dL) prior to treatment. The purpose of this study was to quantify the amount and direction of change in serum glucose levels of diabetic patients undergoing HBOT.

**Materials and Methods:** Pre and post HBOT serum glucose levels, type of diabetes, medications, and HBOT parameters were recorded for patients seen for HBOT between 2008 and 2014.

**Results:** There were 80 patients who underwent a total of 2125 treatments. Serum glucose decreased in 71% of all treatments. The median change for all treatments was a decrease in glucose of 22 mg/dL. In 1826 of the treatments the initial glucose was  $\geq 120$  mg/dL without any intervention. Only 1.2% of these treatments resulted in a post-treatment glucose  $< 90$ mg/dL (25mg/dL median decrease).

A greater proportion of type 1 diabetics (48.2%) had an increase in their blood glucose ( $p < 0.0001$ ) while a greater proportion of type 2 diabetics (77.5%) had a decrease in their blood glucose ( $p < 0.0001$ ) following HBOT.

A greater proportion of treatments for insulin only diabetics (27.5%) had an increase in blood glucose ( $p < 0.0001$ ) and a greater proportion of treatments for oral medication only (83.3%) had a decrease in their blood glucose following HBOT ( $p < 0.0001$ ).

**Summary/Conclusions:** Our findings demonstrate an overall decrease in serum glucose levels for diabetics undergoing HBOT. However, the number of treatments in which patients resulted in clinically relevant hypoglycemia post-treatment was low, and type 2 diabetics were more likely to have a decrease in serum glucose than type 1 diabetics. These findings suggest current minimum serum glucose levels may be too high and require adjustment for insulin dependent versus non-insulin dependent diabetics.

# E 81

ORAL PRESENTATION TIME: 0948 - 1000

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Sham Hyperbaric Analysis Model - Part 1: Standard Pressure (Pilot) Study

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**Background:** Randomized controlled trials compose the foundation of evidence based medicine. Hyperbaric medicine possesses a unique set of challenges to design and application of pressure control (sham/placebo) arms in clinical studies. To date, there is no standard sham protocol that has been established, though most studies perform shams at depths of 1.06 to 2.5 atmospheres absolute (ATA), breathing air or mixed gas with lower partial pressures of oxygen.

**Methods:** A pilot study (SHAM 1) of four participants underwent a blinded dive profile of pressure changes (ranging 3 to 20 fsw) in our multi-place hyperbaric chamber while breathing air and answered questions as to pressure effect sensed (pressurization, depressurization, or neutrality) and perceived depth (ranging from 0 to 60 feet of seawater [fsw] or 0 to 2.8 ATA) in a series of multiple 5 minute travel periods. No decompression obligations were incurred during the study.

**Results:** Of 34 observations for perception of pressure effect, 23 or 67.6% of them were discordant from actual pressure changes or points of neutrality, compared to a theoretical 66% possibility of discordance by chance alone. When comparing all data, the average depth difference (defined as absolute difference between perceived and actual depth) was recorded to be 12.0 fsw. Stratifying by periods of pressure change, stasis, or oscillation at neutral depth, the average depth differences were 12.7 fsw, 10.2 fsw, 13.7 fsw, respectively.

**Conclusions:** Based on our pilot study, we conclude that it is possible to create shallower dive profiles as a standardized sham for hyperbaric studies. Data suggests either an oscillating or pressure change profile offers greater placebo effect than remaining at a neutral depth. Absolute depth differences are also variable at shallower sham depths allowing for optimization of future potential standardized placebo dive profiles for use in clinical hyperbaric studies (SHAM 2 Study underway).

# E 82

ORAL PRESENTATION TIME: 1000 - 1012

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Adverse Events in a Blinded, Randomized Trial of Hyperbaric Oxygen for Post-Concussive Symptoms

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**Introduction:** We report chamber- and protocol-related adverse events (AEs) for participants enrolled in a randomized, double-blind clinical trial of hyperbaric oxygen (HBO<sub>2</sub>) for persistent post-concussive symptoms (HOPPS).

**Methods:** This study randomized 72 military service members at 4 sites to 13 weeks of local care (LC) or forty 60-minute sessions of HBO<sub>2</sub> (1.5 ATA, oxygen) or sham (1.2 ATA, air). AEs were recorded from study enrollment through 13-week outcomes. Injury severity and relationship to chamber sessions or study participation for AEs were determined by a physician.

**Results:** 28 participants (40%) reported no AEs during the study (14 LC, 7 HBO<sub>2</sub>, 7 sham). Another 10 (14%) reported only acute illness or injury with no/remote relationship to study participation (3 LC, 4 HBO<sub>2</sub>, 3 sham).

Other AEs	LC (n=23)		HBO <sub>2</sub> (n=24)		Sham (n=25)	
	Related	Unrelated	Related	Unrelated	Related	Unrelated
Barotrauma	1 otic	0	3 otic 1 tooth	1 otic	1 otic 3 sinus	1 otic
New onset or change in headaches	0	2	2	3	2	3
Other pain (back, flank, sternum)	0	1	0	3	0	3
Vision problems	0	1 presbyopia	1 myopia	1 dry eye	0	0
Affective problems	0	2	0	1 insomnia	1 anxiety	1 insomnia 1 fatigue
Other problems	0	1	1 nausea	1 numbness 1 multiple issues	0	0

Two sites reported these AEs at a higher frequency than the other two sites (12 events/14 participants, 23 events/28 participants vs. 1 event/8 participants, 6 events/22 participants). Two participants withdrew from chamber sessions due to adverse events (1 HBO<sub>2</sub>-change in headaches, 1 sham-claustrophobia/anxiety). Two participants experienced additional concussions during study participation. One participant (HBO<sub>2</sub>) became pregnant during the study and withdrew from further chamber sessions.

**Conclusions:** AEs in this study were rare, and chamber exposures were safe in this population. Site-to-site differences may have influenced AE reporting.

# E 83

ORAL PRESENTATION TIME: 1012 - 1024

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Rates of Myopia in Patients Receiving Hyperbaric Oxygen in Monoplace or Multiplace Chambers

Wilson G<sup>1</sup>, Cable R<sup>1</sup>, Churchill S<sup>1</sup>, Deru K<sup>1</sup>, Weaver LK<sup>1,2</sup>

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**Introductions:** Myopia is a known side-effect of hyperbaric oxygen (HBO<sub>2</sub>). We report vision changes by chamber type.

**Methods:** We queried our patient dataset for patients receiving ≥10 HBO<sub>2</sub> sessions with at least 2 weeks of vision checks by Snellen eye chart. Visual change was defined as myopia (Snellen test worsened to 20/40 or greater) or by loss of ≥2 lines. Patients were categorized by chamber type (multiplace, monoplace, or both, if the patient received >10% of sessions in both chambers). Data presented as mean±1SD (range).

**Results:** We identified 85 patients treated 1/2013-2/2015 meeting inclusion criteria. 53 (62%) were male. Patients were treated for radiation injury (n=29), osteomyelitis (n=24), compromised flap (n=15), diabetic wound (n=11), and other approved indications (n=6).

	All Patients	Monoplace	Multiplace	Both Chambers
Patients, N	85	34	34	17
Sessions, n	35±16 (10-79)	28±12 (10-52)	38±16 (10-70)	43±20 (11-79)
Age, years	57±15 (17-85)	55±15 (17-82)	61±14 (32-82)	54±18 (18-85)
Lines lost, number	1.2±1.6 (0-8)	1.1±1.3 (0-5)	1.3±1.9 (0-8)	1.2±1.9 (0-8)
2-Line Change, N(%)	24 (28)	10 (29)	11 (32)	8 (47)
20/40 or worse, N(%)	20 (23)	11 (32)	6 (18)	3 (18)
20/70 or worse, N(%)	8 (9)	4 (12)	2 (6)	2 (12)
No vision change, N(%)	34 (40)	14 (41)	14 (41)	6 (35)

19 patients (22%) reported vision complaints, and 10 (12%) received counseling to limit driving. The number of sessions received by those with new onset myopia (20/40) was 36±14 (11-60), and for those with at least a 2-line change was 36±15 (15-60).

**Conclusions:** In this retrospective review, about 25% of patients experienced visual change with HBO<sub>2</sub>, including 2 patients with fewer than 20 HBO<sub>2</sub> sessions. Though monoplace patients averaged a shorter HBO<sub>2</sub> course, vision outcomes appeared worse in this group. However, patient effort and variable lighting conditions could have influenced this result.

# E 84

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Preparation and Implementation of a 53.5 Hour Saturation Dive; Tech Perspective

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**Background:** 64 y/o adult male had a planned dive using USN Rev 6 table 220/20 on Air with a scheduled total ascent time of 87 minutes and 20 seconds. Diver forgot his weight belt, after roughly 18 minutes at 220 fsw, the diver lost buoyancy control and surfaced in roughly 3 minutes. Bilateral lower extremity paralysis developed and the diver was taken to a local monoplace chamber for a TT6. Treatment was aborted due to patient vomiting. The diver's symptoms persisted, a second TT6 was administered some 13 hours later but patient status continued to worsen. DAN was contacted and patient was flown to HCMC. Patient treatment began at HCMC multiplace chamber T32 hours after incident.

**Methods:** Fink TL20-171 (The Rat Pack) is a three lock chamber. Lock 1 (Frank) has a max treatment depth of 66 fsw whereas locks 2 (Sammy) and 3 (Dean) have a max treatment depth of 165 fsw. Medical gases include: Medical Air, Oxygen, Heliox 80/20, Nitrox 50/50, Nitrogen, and Special Mix capable. Pre-dive briefing to discuss tables, ascent rates, lock-in/outs, staffing changes, emergency procedures, and other subjects related to the longevity of this dive. On Sept, 20<sup>th</sup> 2014 at 0221, pressurization began to 165 fsw for TT6A, then was transitioned into TT4, and ending with a TT7 on Sept 22, at 0752. Total Time of Dive was 3211 minutes. The dive was overseen by an MD, operator, and tender, that were on site for the entire duration of the tx. Eight extra attendants were locked-in on 13 different occasions and three extra chamber operators filled in for the core operator during rests.

**Conclusion:** Saturation dives can be intimidating and complex, but with continuous training, a detailed plan and a committed staff, saturation dives can be executed with confidence.



## E 85

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

### **The Use of Simulation Scenario Training in a Hyperbaric Multi-place Chamber**

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**Introduction/Background:** Due to the unique and complex situations of treating patients with HBOT, safety and skill could mean the difference between life and death. Simulation training offers specialized scenarios specific to the hyperbaric environment, to increase staffs' skill and comfort level in taking care of their patients safely and competently. This is the first time simulation training has been used in the hyperbaric environment.

**Materials and Methods:** We created two complex scenarios that are unique to the Hyperbaric environment requiring coordination between chamber technicians, RNs and MDs. 1.) Flash pulmonary edema progressing to cardiac arrest during an elective HBOT. 2.) Urinary retention requiring Foley catheter placement and neurological O2 toxicity during a USN TT #6 in a spinal cord DCS patient.. Simulation staff, SIM man, the Chamber, and Props (crash cart, meds, lines, etc), were used to make the situation as true to life as possible.

**Results:** Successful training sessions led to open communication of all staff present, need for additional specific training were identified, and staff conveyed more confidence in their competency level.

**Summary/Conclusions:** Participants reported increased comfort and skill competence with rare emergent situations that are not frequently encountered. Suggestions were made to have additional scenarios per training sessions and to have more frequent sessions.

# E 86

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## A Case Study of Severe Decompression Sickness in an Artisanal Fisherman in Yucatan, Mexico

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**Introduction/Background:** Artisanal fishermen around the world dive for their livelihoods and often subject themselves to provocative dive profiles due to economic pressures. Consequently, rates of decompression sickness (DCS) are much greater than in recreational divers (~3.3% vs 0.03% respectively). Here we present the case of a surface supply diving fisherman in the Yucatan Peninsula of Mexico who suffered a significant episode of DCS.

**Materials and Methods:** Data was collected via a site visit to Tizimin, Mexico, review of medical records, and interviews with the treating physician and the patient. This 32 year old male had 10 prior episodes of decompression sickness and had been diving for over 10 years. He dove to a depth of 22 "arms lengths" (~33m, 4atm) for ~60min, ascended rapidly, and had intense vertigo, vomiting, dyspnea, bilateral leg numbness and weakness, generalized pain, and malaise. The patient was admitted to the hospital in Tizimin and underwent a prolonged course of hyperbaric oxygen (HBO) therapy. US Navy Treatment Tables 5 and 9 were used for a total of 17 treatments.

**Results:** The patient's initial symptoms worsened and required admission to the ICU for hypotension. Later, he developed more severe lower extremity weakness and temporary urinary retention. Crystalloid fluids and IV ketorolac were used as adjuncts. MRI results demonstrated a hypoxic infarct at T8-T10. Upon completion of HBO therapy, the patient required additional physical therapy and is now ambulatory but no longer diving.

**Summary/Conclusions:** The remote locations of artisanal diving fishermen, difficult to access HBO treatment, and lost economic opportunity can lead to delayed presentation of DCS in the Tizimin area. The case presented here highlights the provocative dive profiles of these diving fishermen and the potential for future intervention. Despite challenging conditions, this patient's therapy resulted in a good outcome.

# E 87

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## **Safety and Efficacy of Needle Myringotomy Performed by Hyperbaric Medicine Physicians**

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**Introduction/Background:** Otic barotrauma is a common complication of hyperbaric oxygen therapy (HBOT), occurring in 2-82% of patients. Though there are multiple etiologies, available treatments to date have been limited to (1) continuation of HBOT, with risk of further barotrauma, (2) painful needle myringotomy, (3) treatment deferral for medical therapy or tympanostomy tube placement, or (4) discontinuation of HBOT. The clinical availability of a simple topical phenol anesthetic applicator allows for myringotomy in awake patients, performed by hyperbaric physicians, with minimal discomfort, and with minimal delay or complications.

**Materials and Methods:** To guide the development of a future prospective study, we examined a case series from our existing treatment data to assess the indications, course, and outcomes of HBOT patients with difficult ear clearance who were treated with topical phenol anesthesia and awake myringotomy as performed by hyperbaric physicians. Patients were called back 6 weeks after their last HBOT.

**Results:** In our series, 39 of 161 (24.2%) non-ventilated patients treated over a 6-month period developed otic barotrauma requiring myringotomy. 23 (58.9%) required bilateral myringotomies. 30% of myringotomies were performed prior to treatment, 42.4% during an HBOT, and 27% after treatment. 48.7% of patients who required myringotomy sustained TEEDs 0 or 1 injury, and 51.3% sustained TEEDs 2 or 3 injury. Patients who required myringotomy completed an average of 17 HBOT, with 18 patients receiving more than 20 HBOT. Only 5 patients (12.8%) required a revision of the site prior to completing HBOT, and 3 (7.7%) required PE tubes or could not complete HBOT. 23 patients were reached for follow up, and only 1 patient had any concern about hearing.

**Summary/Conclusions:** With the availability of a simple and safe means of applying tympanic anesthesia, awake myringotomy in patients at risk of or experiencing otic barotrauma should constitute a new standard of care in Hyperbaric Medicine.

## E 88

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

### **Incident Study of Myopia in Patients Undergoing Hyperbaric Treatment**

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**Background:** Progressive myopia has been observed in patients undergoing prolonged periods of daily HBO<sub>2</sub> therapy. Although the exact mechanism remains obscure, it is apparently lenticular in origin and is usually reverses completely within a few days to several weeks after the last treatment. The number of hyperbaric treatments administered increases its occurrence and it has been previously reported that this is usually not seen when less than 20 treatments are given.

**Purpose:** To quantify the rate of myopia in hyperbaric patients

**Method:** Vision testing was done using the Snellen Eye Chart at 20 feet. Vision was tested prior to their initial HBO<sub>2</sub> treatment and again every 20 treatments. Testing was also performed regardless of treatment number if patient complained of any change in vision. Examination was performed with both eyes, obscured right eye, and obscured left eye with the patient wearing corrective lenses if they normally do so. If a change was noted the patient was given adjustable lenses and retested using the new eyewear to ensure proper adjustment.

#### **Results:**

- Data collection is still ongoing.
- To date, an initial examination has been conducted on 42 patients. (10 patients were discharged for various reasons prior to completing 20 tx)
- Patients include a range of ages from 28 to 80. Average age was 63 years old.
- 25 were identified as having diabetes.
- 20 wore corrective lenses pre-HBO.
- 8 had a vision decline by tx 20. Average age was 66 years old
- 6 of those with decline had diabetes

**Conclusion:** A known side effect of hyperbaric oxygen is worsening myopia. We have found that this affect is more common than previously thought and affects people with diabetes greater than those without diabetes. Using adjustable corrective lenses is effective in improving the vision in these patients.

## **E 89**

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

### **The Chinese standard - Clinical Application Technique Specification of Hyperbaric Oxygen**

Qingle L

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## **WITHDREW**

# E 90

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## An Extraordinary Case of Type 2 DCS

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**Introduction:** This case of decompression sickness deserves reporting for reasons of information sharing, optimizing management, legal ramifications and measures taken to prevent future events of this kind.

**Methods:** A healthy 73 year old female with arrested breast cancer was referred to our facility for emergency hyperbaric oxygen (HBO) recompression because of transient lower extremity paralysis after a homemade chamber dive to “perk” herself up. Analysis, management and outcome of the event generated an amazing narrative.

**Results:** The patient’s enterprising son fabricated a hyperbaric chamber initially constructed in Mexico and set-up in a central California coastal community in order to pressurize family and friends for invigoration purposes. Compressed air was used and pressure was monitored with a pounds per square inch pressure gauge. After an hour “dive” at an equivalent depth of 72, feet the patient decompressed without incident and “felt great.” About six hours later while at her home her lower extremities became numb and paralyzed. A computerized tomography scan at a local emergency department demonstrated gas in the pelvic veins and inferior vena cava. Symptoms remitted with O<sub>2</sub> breathing and after being referred to us, a four hour drive, the patient was almost asymptomatic. Two HBO recompression treatments over a 12 hour period resulted in full recovery. Legal counsel was obtained for advice regarding patient’s son.

**Conclusions:** Since the son broke no laws, the recommendation was made for taking an educational approach. He was advised to learn about chamber safety, chamber standards and indications for HBO therapy. References such as the NFPA-99 and UHMS safety manuals were cited. The patient’s amazingly healthy mother was advised not to undergo additional “perk-up” treatments. Shortly thereafter at a chapter UHMS meeting, the patient’s son was in attendance.

# E 91

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION: YES

## **The Use of Trend Analysis of Blood Sugars in Diabetic Patients Undergoing Hyperbaric Oxygen Therapy to Predict Wound Failure**

Pearson M, Harch P, Hardy S, Murphy-Lavoie H, LeGros T, Wyatt H, Yontz D and Van Meter K  
LSU Undersea and Hyperbaric Medicine Fellowship, New Orleans, Louisiana, Feb, 2015.  
tle Gros1@cox.net

**Introduction:** A number of investigations have focused on improving blood glucose control in diabetic patients undergoing hyperbaric oxygen therapy (HBO<sub>2</sub>). However, there is a paucity of data on the efficacy of trending blood sugar levels to identify patients at risk to fail therapy. We analyzed blood glucose levels of HBO<sub>2</sub> patients with infectious wounds or traumatic injuries to determine if glucose trends could portend outcomes in healing.

**Methods:** The blood glucose levels were collected from a convenience sampling of diabetic patients at two locations: (1) an urban HBO<sub>2</sub> referral center (in-patients); and (2) an urban hyperbaric clinic (out-patients). This data was then retrospectively analyzed for trends relating to the progression or regression of the patients' wound(s) or injuries.

**Results:** We found that in the out-patient setting, a consistent rising trend of > 100 mg/dL in blood glucose levels was predictive of imminent treatment regression or failure, whereas a continued downward trend correlated well with a successful therapeutic outcome regardless of the HBO<sub>2</sub> treatment indication. In the in-patient setting, where there is presumably tighter glucose control, the prognostic value was lost, perhaps due to aggressive interventions. However, exaggerated glucose fluctuations were noted immediately prior to surgical debridement or amputation in this group.

**Conclusions:** Multiple studies have shown that strict blood sugar and weight control are crucial to wound healing. This study evaluated blood sugar levels in signifying overall success or failure in diabetic HBO<sub>2</sub> patients. Although not specific, these analyses may help clinicians better predict when treatment failure is imminent. In the out-patient setting, where patients may not adjust their medication or diets, early and aggressive intervention may be required. Fortunately, plotted blood sugars are automatically tracked in most electronic medical records and provide immediate data for analyses.

# E 92

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Case Report of a Tension Pneumothorax During Hyperbaric Oxygen Therapy

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**Introduction:** Pulmonary barotrauma (PBT) is a rare but potentially serious complication of hyperbaric oxygen therapy (HBOT). Underlying lung pathology contributes to air trapping, increasing the risk of PBT. Risk factors include age, restrictive lung disease, recurrent lung infections, tuberculosis, smoking, obstructive airways disease, cysts, bulla, and low MEF 25.

**Methods:** We present the case of a 73 year old Filipino male with a history of diabetes, started on HBOT for a Wagner grade 4 foot ulcer. He had a 60 pack year history of smoking but no known lung disease. During decompression of the 13th HBOT, he developed sudden onset chest pain and dyspnea. He was placed on 100% oxygen and ascent was slowed. He became unresponsive, with absent breath sounds on the left chest. Urgent needle thoracostomy resulted in immediate return of consciousness. Chest x-ray (CXR) revealed pneumothorax (PTX). CT revealed bilateral apical fibrosis, but no blebs or emphysema.

**Results:** HBOT was discontinued. His gangrene worsened, requiring an amputation. He suffered another unprovoked PTX 54 days after the initial event. PBT during HBOT is extremely rare. A survey of 98 HBOT centers worldwide found 9 cases of PBT out of nearly 2 million HBOT sessions (0.00045% incidence). Of these centers, 66% treated patients with underlying lung blebs but used preventative measures during HBOT. Although not entirely sensitive, CXR was the most widely utilized screening tool. There are only 8 reported cases of PBT occurring during HBOT. Most of these patients had lung pathology seen only on CT.

**Summary:** Our patient likely developed PTX secondary to regional air trapping from occult lung disease. To date, there are no formal screening guidelines prior to the initiation of HBOT for this patient population. Further research is needed to predict which patients are at risk for PBT during HBOT.



# E 93

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Thinking Outside the Box about Safety – Not Just the Monthly Formal Exercise

Cormier JE

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**Introduction:** NFPA standard 14.3.1.4.5 (2015 Edition) states “Emergency procedures and fire drills shall be conducted at least annually and documented by the safety director”.

**Setting:** An out-patient wound and hyperbaric clinic with 2 monoplace chambers that averages 5 HBOT and 30 - 32 wound care patients per day. Staffing includes 6 full time nurses (one is the safety director and a CHT), one full time office coordinator, 4 part time nurses, 1 part time CHT, the medical director and two other part time physicians. Two of the full time nurses are cross trained in HBO and function as chamber operators. All staff work 10 hour days except for the office coordinator, the four HBO trained staff rotate through chamber operation responsibilities; this results in a slightly different daily compliment of personnel. Some staff members are only on site one day per week.

**Problem:** Challenges presented by this rotating schedule include: ensuring all staff have the opportunity to participate in hyperbaric continuing education and safety drills without disrupting patient care, and maintaining compliance and conformity with daily safety measures and proper documentation (checklists, checking for prohibited substances, patient education). The nature of the healthcare environment further contributes to this problem due to last-minute patient add-ons and unexpected complications that can conflict with scheduled drills.

**Solution:** Regular rotations through hyperbarics for all HBO trained staff, education moments added to every staff meeting with minutes disseminated via email, random HBO quizzes for chamber operators with email discussions after completion, a prohibited item compliance activity with patient participation, staff participation in drills conducted elsewhere in the facility, physician chamber emergency operation training, safety drill pop quizzes, daily chart audits and role-sharing among staff.

# E 94

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1100 - 1130

RESIDENT COMPETITION:

## Impacts on Daily Activities Following Changes in Visual Function During and After Hyperbaric Oxygen Treatment

Wannholt R<sup>1</sup>, Arnell P<sup>1</sup>, Zetterberg M<sup>2</sup>, Stomberg MW<sup>3</sup>, Grönlund MA<sup>2</sup>

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**Background:** Transient myopic refractive changes have previously been described as a side-effect of multiple hyperbaric oxygen (HBO) treatments. The aim of this study was to measure visual acuity (VA) and refraction as well as to evaluate subjective visual difficulties during daily activities, before and after receiving HBO treatments.

**Materials and Methods:** Twenty-two patients (15 male, 7 female), with a mean age of 62 years, received 40 HBO treatments in a multiplace chamber at 2.4 ATA for 90 minutes, five days a week. Oxygen was administered by a Sea-Long series 7000 oronasal mask. Visual acuity and refraction were measured and perceived visual difficulties during a number of daily activities were evaluated by a questionnaire; before, at completion, and at 12-week follow-up after the last HBO treatment.

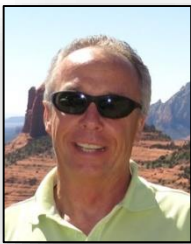
**Results:** There was no significant difference in mean VA change in better eye from baseline to completion of HBO treatments or to 12-week follow-up. Mean refractive change after receiving 40 HBO treatments was -0.8 spherical equivalent (SE) diopters (D) ( $p=0.001$ ). Eight patients had a myopic shift of  $\geq 1D$ . At follow-up mean refractive change didn't differ significantly from baseline. After receiving HBO treatments, nine patients experienced a deterioration of visual function and three patients indicated improvements. Impact on daily activities surveyed was generally low or moderate, with few exceptions. Activities such as reading, driving and workplace performance were affected. At follow-up, all except two patients indicated a subjective return of visual function compared to baseline. Eye exams on these two showed no significant changes.

**Conclusions:** Refractive changes and remission are in line with previous studies. In some individuals changes in vision may become pronounced and very bothersome, i.e. inability to drive. This stresses the importance of following patients' visual function in conjunction with HBO treatment and providing adequate information about possible adverse impacts on daily life.

**PLENARY:**  
**“MY SECRETS FOR EFFECTIVE PRACTICE-  
BASED CLINICAL RESEARCH”**

**Neil Hampson, MD**

**11:30AM – 12:30PM**



"Dr. Hampson has over 100 publications in his career, many of which have had significant impact on public health in the United States. In this presentation, he will discuss the impact that some of his publications have had, as well as his secrets for doing clinical research in a practice-based setting without protected time for academics. He will also give examples of how such research can be done without significant grant funding and emphasize the importance of a good idea and maintaining vigilance for research opportunities during day-to-day clinical practice."

**SESSION F**  
**CLINICAL HBO<sub>2</sub> THERAPY (#2)**

**Moderators: Heather Murphy-Lavoie, MD & Laura Josefsen, ACHRN**

**SATURDAY, JUNE 20**

**1:30PM – 4:00PM**

# F 95

ORAL PRESENTATION TIME: 1330 - 1342

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Necrotizing Soft Tissue Infection

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**Introduction/Background:** Prompt antibiotics and early surgery are the mainstay of treatment for necrotizing soft tissue infections (NSTI). Hyperbaric oxygen treatment (HBOT) is considered an adjuvant, but important resource. Centralizing the treatment of patients with NSTI has been shown to reduce mortality. SU/O has been regional NSTI-center since 2008. Data concerning all ICU-admitted patients are prospectively collected, which enables assessment of risk factors, treatment effects and mortality.

**Materials and Methods:** Data on all patients over 18 years, treated at the ICU for NSTI between 2008-2014 were analyzed using SPSS.

**Results:** 78 patients met the inclusion criteria. Mean age was 57 (s=15). There were 53 (67.9%) males. 13 (16.7%) were smokers, 8 (10.4%) had a reported substance abuse, 11 (14.1%) were diabetics, 3 (3.8%) immunocompromised and 11 (14.1%) had a malignancy. Mean time from hospital arrival until administration of antibiotics and primary surgery were 5.1 (s=5.8) and 21.5 (s=23.2) hours, respectively. HBOT (280kPa, 70-90 minutes) was given to 60 (76.9%) of the patients. Mean number of treatments was 4.4 (s=2.9). No correlation was found between mortality and delay of antibiotics or delay of surgery. The estimated mortality rate (EMR) for all included patients, based on APACHE-II and SAPS-III, was 43.0% (s=26.6). Actual mortality rate at one month was lower, 11.5% (p<0.001). EMR for the HBOT-group was 43.6% (s=25.2) and 40.6 (s=32.0) for the non-HBOT-group. Actual mortality at one month was 6.7% in the HBOT-group and 27.8% in the non-HBOT-group. The difference in mortality for the two groups was significant (p=0.01), but not when adjusted for EMR (p=0.11).

**Summary/Conclusions:** In this prospective material the overall mortality was significantly lower than predicted. This might be due to centralization and multimodal care. The role of HBOT remains unclear. Continued registration will provide more clinical useful information.

# F 96

ORAL PRESENTATION TIME: 1342 - 1354

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## **Adjuvant Hyperbaric Oxygen Therapy for Necrotizing Soft Tissue Infections: Preliminary Analysis from a Study of Two Urban Centers**

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**Introduction/Background:** Necrotizing soft tissue infections (NSTIs) are relatively rare and life-threatening, with mortality as high as 73%, but with an average reported mortality of 25-39%. Given rapid tissue destruction, frequent sepsis, and multisystem organ failure, morbidity and mortality associated with NSTIs are sensitive to timely interventions. Standard therapy consists of surgery, antibiotics, and intensive peri-operative care. Increasingly, adjuvant hyperbaric oxygen therapy (HBOT) has also been used to treat NSTI.

**Materials and Methods;** We conducted a retrospective study of NSTI patients in two urban trauma centers over a 10-year period. Both centers treated patients with surgery, antibiotics and supportive care while one also treated patients with adjuvant HBOT. To date, we have examined 7 years of data, comparing demographics, illness severity, debridement's, length of stay, and inpatient mortality in patients receiving standard therapy and those receiving standard therapy plus adjuvant HBOT.

**Results:** Demographic characteristics were similar in both HBOT and standard groups. The mean age for both groups was 53 years old, with 38% female patients. Across both hospitals, the mean hospital length of stay (LOS) was 17 days in both groups, the mean number of debridement's was 3 with HBOT and 2.32 without, and mortality was 5.4% with HBOT and 10.3% without. At the hospital with HBOT, the mean LOS was 17 days with HBOT and 21 without, with an average of 3 debridement's for both groups, and 13% mortality without HBOT. Odds ratios of 4.29 (0.87-21.1, HBOT hospital alone) and 3.43 (0.78-15.1, both hospitals) trend in favor of survival with adjuvant HBOT.

**Summary/Conclusions:** Mortality in our study was less than previously observed, suggesting advances in NSTI recognition and care. Following other meta-analyses, our preliminary data review suggests a trend toward improved survival and possibly diminished hospital length of stay with adjuvant HBOT. Our final analysis should achieve sufficient power to detect these differences.

## F 97

ORAL PRESENTATION TIME: 1354 - 1406

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

### **Globalization and International Collaboration in Publications Of Undersea and Hyperbaric Medicine, 1974 To 2014**

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**Background:** *Undersea and Hyperbaric Medicine* (UHM) is the leading hyperbaric medicine journal. The globalization and international collaboration of UHM publications reflect the global evolution on hyperbaric medicine researches.

**Materials and Methods:** All publications in UHM were retrieved from SciVerse Scopus database by the search term: "ISSN(1066-2936) OR ISSN(0093-5387) AND PUBYEAR < 2015". We collected data on publications including publication year, author's affiliated country, and cited times for further analyze. The publication's origination was classified according to the first author's nationality. The international collaboration publication was defined as the authors were affiliated with more than two countries. Linear regression was used for trend analysis. The slope ( $\beta$ ) of the linear regression was adopted as representative of trends. The cited times between groups were analyzed by Wilcoxon rank-sum test.

**Results:** A total of 1444 publications were enrolled and 74 (5.1%) were international collaboration publications. In 1974, the 22 publications were originated from 3 countries without international collaboration. In 2014, the 71 publications were originated from 17 countries and there were 8 (11.3%) international collaboration publications. The trends of publication originations and international collaboration publications were 0.279 and 0.185, respectively ( $p < 0.001$ ). In international collaboration publications, the United States (US) (18), Japan (7), Australia (5) are the leading first author's nationalities and the US (32), Canada (9), Australia (7) are the leading co-author's nationalities. The average cited times of single nation originated and international collaboration publications since 2001 were 6.34 and 4.29, respectively ( $p = 0.042$ ). There was no publication types difference between these two groups (Chi-square test,  $p = 0.20$ ).

**Conclusions:** The globalization and international collaboration on hyperbaric medicine researches are increasing. The international collaboration publications were more frequently cited than single nation originated publications. The US is the leading contributor of international collaboration publications.

## F 98

ORAL PRESENTATION TIME: 1024 – 1036 (Friday, June 19)

POSTER PRESENTATION TIME: 1100 – 1130 (Friday, June 19)

RESIDENT COMPETITION:

### **Hyperbaric Oxygenation In The Treatment Of Acute Central Retinal Artery Occlusions: An Analysis of 214 Cases Following a Prospective Protocol**

Desola J<sup>1</sup>, Papoutsidakis E<sup>1</sup>, Matos P<sup>1</sup>, Gomez M<sup>1</sup>, Anselem L<sup>2</sup>, Canela J<sup>3</sup>

<sup>1</sup>CRIS-UTH, the Hyperbaric Therapy Unit of Barcelona, Moises Broggi Hospital, Sant Joan Despí, Barcelona; <sup>2</sup>Department of Ophthalmology Moisés Broggi Hospital, Sant Joan Despí, Barcelona;

<sup>3</sup>Preventive Medicine and Public Health Department, Biostatistics, University of Barcelona  
jordi.desola@cris-uth.cat

**Introduction:** The Retina is the most critically oxygen-sensitive tissue of the human body. An Acute Central Retinal Artery Occlusion (ACRAO) produces a loss of visual function. No drug is effective and spontaneous recover never happens.

**Background:** The 10-15% of the retinal perfusion depends on Choroids' circulation. Some retinal cells can remain in hypoxic ischemic penumbra. HBOT enhances plasmatic oxygen delivery to hypoxically injured although not necrotic cells maintaining the retina in good conditions until aching spontaneous reperfusion, what is proved to be ever achieved in 15-21 days. A prospective protocol was designed in CRIS-UTH in 1986.

**Methods:** HBOT was applied during at least 15 days, 1-2 sessions daily at 2.3 ata, 7 days a week. Outcome was validated as Healing, Improvement, No change and Worsening. A statistical analysis was done with SPSS-v.21.

**Results:** Patients studied: 214; valid for study: 184. Men: 124 (67.4%), women: 60 (32.6%). Age: 63.3 ± 14.95 (15-96). Days of delay: 2.83 ± 6.35 (0-65). Number of HBOT sessions: 20.6 ± 3.62 (15-40). Number of HBOT days: 19.08 ± 6.36 (15-47). Right eye: 103 (56.0%); left: 81 (44.0%). Whole recovery: 6 (3.3%); improvement: 132 (71.7%); no change: 46 (25.0%). Better outcome when right eye affected ( $\chi^2=32.32-p=0.0001$ ). No significant relation between delay and outcome. Patient's age correlated with a better outcome ( $F=7.659 - p=0.006$ ). Logistic regression not applicable.

**Conclusions:** HBO was effective in the treatment of ACRAO even when applied up to 10 weeks after onset, conditioned to applying more than 15 HBOT sessions during minimum 15 days, 7 days a week. HBO is not a reperfusion enhancing therapy, so an improvement after the first HBO treatments should not be expected. Assuming that HBO can only be useful when applied in the first hours is not rational. The UHMS Guideline for ACRAO comes from an erroneous approached.



## F 99

ORAL PRESENTATION TIME: 1418 - 1430

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

### **Hyperbaric Oxygen Therapy for Pyogenic Spondylitis**

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**Introduction:** Pyogenic spondylitis is associated with significant rates of morbidity and mortality. Conservative therapy with antibiotics is generally the initial treatment of choice for pyogenic spondylitis, and surgical intervention is indicated in patients with symptomatic neural compression and/or spinal instability. Many patients with osteomyelitis have been treated using hyperbaric oxygen therapy (HBO) with antibiotics. The adjunctive use of HBO may have therapeutic potential in the treatment of pyogenic spondylitis. We report our experience with HBO in the treatment of primary pyogenic spondylitis

**Method:** We retrospectively analyzed 18 cases of primary pyogenic spondylitis treated in our hospital from March 2008 to February 2015. All cases had a treatment with both antibiotics and HBO. A typical HBO treatment protocol is conducted in a multiplace chamber for 1 hour at a pressure of 2.0 atmospheres absolute. The diagnosis of pyogenic spondylitis in these patients was made on the basis of clinical, laboratory, and radiological evaluations. Serological markers were monitored in the course of treatment.

**Result:** The patients included 9 men and 9 women with an age range of 54-88 years (mean 70.8 years). HBO did not cause any treatment-related complication. One patient was diagnosed as spinal tuberculosis. Seven cases had a prior history of diabetes mellitus. None of the patients had a prior history of spinal surgery. Two cases were treated with surgical debridement. The mean duration of healing was 41.7 days (range 7 to 123 days) except for 2 surgical patients and 1 case of tuberculosis. Two recurrences were observed during follow-up. All cases were successfully treated finally.

**Conclusion:** Diabetes mellitus was a risk factor for pyogenic spondylitis. 16 out of 18 cases did not need surgical treatment. HBO may be used as adjunctive and safe treatment to improve clinical outcomes in patients with pyogenic spondylitis.

# F 100

ORAL PRESENTATION TIME: 1430 - 1442

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## The Incidence of Confounding Factors in Diabetes Mellitus Patients Hospitalized for Lower Extremity Wounds

Strauss MB, Moon H, Craig AB, Ponce JP, Miller SS, Le PNJ

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**Introduction:** Three confounding factors: underlying deformity, deep infection, and ischemia-hypoxia, or combinations of these are highly associated with healing challenges of diabetic foot ulcers (DFUs). This paper reports the incidence of these confounding factors, which we label the “Troublesome Triad” (TT) in a prospective series of 62 diabetic patients hospitalized secondary to lower extremity wounds.

**Methods:** After IRB approval for a wound scoring project, prospective data was gathered in a series of patients hospitalized because of their lower extremity wounds. The diabetes mellitus (DM) cohort was analyzed for the incidence of each of the components of the TT. The severity of the wound was graded using our 0 to 10 Wound Score (with 10 being best) and compared with the patients who had components of the TT and those who did not.

**Findings:** One or more components of the TT were observed in 56 (91.9%) patients. As the number of confounders increased, the mean Wound Scores worsened from 5.2 for one confounder to 2.9 for three confounders ( $p = 0.023$ ). The percentage with one or two confounders was nearly equal (44.4% and 46% respectively), while only 3 (4.8%) patients had all three confounders. Unresolved infection was the major confounder present in 48 (76.2%) of the patients, with ischemia-hypoxia in 41.3% and deformity in 33.3% of the patients.

**Conclusions:** For those DM patients hospitalized because of LE wounds, confounders that require remedial interventions were present in over 90% of patients. The infection confounder required debridement and antibiotics. The deformity confounder, when off-loading is insufficient, required surgical correction. The ischemia-hypoxia confounder required revascularization and/or HBO treatments depending on the blood vessel pathology.

# F 101

ORAL PRESENTATION TIME: 1442 - 1454

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Differences in Clinician Reimbursement Based on Treatment of Hyperbaric Emergency Indications

Chin W, Chang M, Simon O, Proano J, Huang E

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**Introduction:** In the last decade there has been a proliferation of hyperbaric centers across the United States (US). In a study of centers across the US it was noted that only 16% of 345 centers surveyed regularly treat hyperbaric emergency indications (HEI)<sup>5</sup>. In 2014 the Centers for Medicare & Medicaid Services (CMS) released data from the National Claims History via Public Use Files (PUF). PUF data was used to compare providers that treat HEI with those that do not.

**Materials Methods:** Database was constructed and the following parameters were extracted from the PUF: demographics, number of treatments, payments, charges, and average payments received for the entire year of 2012. Survey results from a previous study were used to identify centers that treat HEI<sup>1</sup> and clinicians from the PUF data were matched with these centers based on address.

### Results:

	Mean		Std. Err.		95% CI	
	No HEI N= 92	Yes HEI N= 63	No HEI N= 92	Yes HEI N= 63	No HEI N= 92	Yes HEI N= 62
<b>Treatments</b>	241	226	28	24	185-296	179- 272
<b>Charges</b>	\$375	\$439.	\$13	\$27	\$348- \$401	\$386- \$492
<b>Payments</b>	\$124.14	\$119.02	\$2.42	\$0.91	\$119.36- \$128.92	\$117.22-\$120.83
<b>Payments /Year</b>	\$23,221.86	\$21,367.64	\$2,631.11	\$2,248.71	\$18,024.13- \$28,419.58	\$6,925.34- \$25,809.94

The only statistical difference observed was the number of treatments given per year by clinicians that worked in centers that treated HEI ( $U$  2228.5,  $z = -2.44$ , 2-tailed  $p = .015$ ).

**Discussion:** Clinicians that belong to centers that treat HEI treated fewer patients and were compensated less for their work than clinicians who did not treat HEI. Influx of new hyperbaric facilities that do not treat HEI may reflect recognition that outpatient hyperbaric therapy is reimbursed better than critical care inpatients, but this cannot be determined from this dataset.

<sup>5</sup> Centers Capable of Treating Hyperbaric Emergencies in the United States. Undersea and Hyperbaric Medicine Journal 2014; 41(5): 464

# F 102

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Recruiting Divers to Study the Ketogenic Diet and Central Nervous System Oxygen Toxicity Symptoms

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**Introduction:** A ketogenic diet has been proposed as a method to decrease oxygen toxicity on the central nervous system and reduce the frequency of seizure episodes during deep-sea dives with Closed Circuit Rebreathers (CCR). A study is underway requiring the participation of technical/CCR divers that will compare divers in ketosis to those under normal metabolic conditions in order to evaluate the neuroprotective benefits of ketosis on CCR diving. This study requires the recruitment of active technical divers willing to complete a survey after each completed dive.

**Methods:** A list of active divers from the International Association of Nitrox and Technical Divers (IANTD) was used as a database of contacts to solicit participation in the study. Active technical divers that showed interest were e-mailed the participation consent form as well as a copy of the background pilot study and encouraged to reach out if questions arose. Alternative contacts (of divers as well as dive shops) not listed on the IANTD database was obtained during phone calls of divers on the IANTD database as well as through research on Google.

**Results/Summary:** 106 phone calls were made directly to divers listed on the IANTD database. 9 contacts expressed interest in the study and further information was sent via e-mail. On 29 calls, a message was left on voicemail. 15 contacts no longer participated in technical diving or were not interested. On 53 calls, the person listed at the number was no longer available or the phone numbers were no longer serviceable. Therefore, 30 additional calls were made to dive shops and dive clubs not listed on the initial IANTD database. Three technical diving instructors were most effective personally provided several hundred diver contacts who agreed to participate. Data will be presented.

# F 103

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## Recurrence of Neurological Deficits in an F/A-18D Pilot Following Loss of Cabin Pressure at Altitude

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**Background:** Randomized controlled trials compose the foundation of evidence based medicine. Hyperbaric medicine possesses a unique set of challenges to design and application of pressure control (sham/placebo) arms in clinical studies. To date, there is no standard sham protocol that has been established, though most studies perform shams at depths of 1.06 to 2.5 atmospheres absolute (ATA), breathing air or mixed gas with lower partial pressures of oxygen.

**Methods:** A pilot study (SHAM 1) of four participants underwent a blinded dive profile of pressure changes (ranging 3 to 20 fsw) in our multi-place hyperbaric chamber while breathing air and answered questions as to pressure effect sensed (pressurization, depressurization, or neutrality) and perceived depth (ranging from 0 to 60 feet of seawater [fsw] or 0 to 2.8 ATA) in a series of multiple 5 minute travel periods. No decompression obligations were incurred during the study.

**Results:** Of 34 observations for perception of pressure effect, 23 or 67.6% of them were discordant from actual pressure changes or points of neutrality, compared to a theoretical 66% possibility of discordance by chance alone. When comparing all data, the average depth difference (defined as absolute difference between perceived and actual depth) was recorded to be 12.0 fsw. Stratifying by periods of pressure change, stasis, or oscillation at neutral depth, the average depth differences were 12.7 fsw, 10.2 fsw, 13.7 fsw, respectively.

**Conclusions:** Based on our pilot study, we conclude that it is possible to create shallower dive profiles as a standardized sham for hyperbaric studies. Data suggests either an oscillating or pressure change profile offers greater placebo effect than remaining at a neutral depth. Absolute depth differences are also variable at shallower sham depths allowing for optimization of future potential standardized placebo dive profiles for use in clinical hyperbaric studies (SHAM 2 Study underway).

# F 104

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Hyperbaric oxygen for exceptional blood loss anemia: A Case Report

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**Introduction/Background:** A 63 yo Jehovah's Witness was admitted to the ICU with a hemoglobin (Hgb) of 2.9 due to an acute on chronic GI bleed. She refused blood products. Due to evidence of multisystem organ failure she was referred for hyperbaric oxygen treatments (HBOT). Subsequent questioning of family members revealed a history of alcohol abuse.

**Materials & Methods:** HBOTs were administered 2 to 3 times a day for up to 90 minutes for 5 days in a monoplace chamber.

### Results:

HBO tx day	Pre	1	2	3	4	5	Post
#HBOT	0	2	3	2	3	3	0
ATA		2.0	2.0	2.0	2.0	2.0	
Minutes HBOT	0	136	254	163	251	223	0
Hgb	2.0	3.0		3.1	3.6		3.4
K	5.2	5.5		4.6	4.2		3.8
BUN	42	49		68	68		46
Cr	3.1	3.9		4.3	3.6		2.2
AST	343			1212	474		99
ALT	11			506	342		181
Ammonia					75		47

HBOTs improved renal and liver function markedly, despite her low Hgb. However, she became progressively more confused and agitated. By day 5 she could not tolerate HBOT. She was intubated to increase sedation and decrease the work of breathing. The patient recovered and was discharged.

**Summary/Conclusions:** This patient had multiple risks for hepatic encephalopathy (blood in gut, alcoholism, hypoxic liver failure, diminished kidney function). HBO treatments improved liver and kidney function, but not rapidly enough to prevent hepatic encephalopathy. Hepatic encephalopathy should be considered in cases of exceptional blood loss anemia due to gastrointestinal bleeding, particularly when the patient's mental status deteriorates despite a positive effect of HBOT. Pulmonary oxygen toxicity is also possible when HBO is combined with supplemental oxygen over several days. These cases are difficult to manage at centers without 24/7 hyperbaric capability, and raise ethical issues about assessing patient competency, and deploying limited resources.

# F 105

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Medical Check-Ups And Treatment Of Dysbaric Osteonecrosis

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**Introduction:** Japan is surrounded by sea, therefore fish diving or construction work are often required. Dysbaric osteonecrosis (DON) is one of the most serious problems for full-time divers who have been diving for a long time. We do medical check-ups specializing in DON for divers who request it.

**Method:** The diagnosis of DON is mainly judged by X-rays and MRIs. We take X-rays of the shoulders, hip joints and knees where there are high incidences of DON, additionally we take them of the elbows, if they tell us they are having any symptom in their elbows. We use the Ohta-Matsunaga criteria. It categorizes DON as type A (juxta-articular lesion) and type B (head, neck and shaft lesion). Moreover, type A is classified from A1 to A6, and type B is classed from B1 to B3. MRI is effective to know the range of DON.

**Result:** The number of divers who took medical check-ups in our hospital from 1981 to 2014 was 274 and the total number was 531. DON was found in 80 out of 274 divers (29.2%). It is less than our previous research which we checked 747 fish divers from 1966 to 1981. The incidence of DON was 56.4% ( $p < 0.0001$ ). All DON cases are not treated immediately. Type B DONs have no symptoms very often, and they are not so serious. But, type A DON may be an obstacle in their daily lives or develop into collapse of caput femoris/humeral head. Therefore, the progression of type A DON should be observed carefully or treated.

Surgical treatments for DON are varus osteotomy, anterior rotation osteotomy or prosthesis replacement. 22 cases of surgical treatments were performed for 21 divers. 20 cases were caput femoris and 2 cases were humeral heads. Varus osteotomy was performed in 5 cases, anterior rotation osteotomy was in 13 cases, replacement arthroplasty was in 2 cases and sterearthrolysis was to 2 cases. 4 cases of anterior rotation osteotomy/sterearthrolysis had to be re-operated on because the osteonecrosis progressed after the operation. Finally, 6 cases had prosthetic femoral head replacements.

**Conclusion:** One should choose to salvage the patient's bone heads as far as possible. In that case, regular check-ups and early treatments are important. We knew the incidence of DON relates to bends and the pathophysiology by our previous research. For the prevention of DON occurrence, treating the bends early is important. Even if the incidence of DON decreases, we have to enlighten the divers about DON.

# F 106

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## **Calcific Uremic Arteriopathy Successfully Treated with Hyperbaric Oxygen Therapy - A Case Study**

Swaby JA, Morgan M, Jennings MS, Santiago W, Heyboer M

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**Introduction / Background:** Calcific uremic arteriopathy (CUA) also known as calciphylaxis is a rare complication of end stage renal disease (ESRD). It is characterized by cutaneous ischemia and necrosis due to microvascular destruction from arteriole calcification. It is often fatal. A multidisciplinary management approach is vital including good wound care, management of serum calcium, and evaluation of parathyroid hormone. There are reports of the successful use of hyperbaric oxygen therapy (HBOT) and IV sodium thiosulfate. We report on the use of HBOT in conjunction with IV thiosulfate for a patient with CUA.

**Materials and Methods:** A 62-year-old male with ESRD on dialysis for 7 years was found to have cutaneous calciphylaxis lesions. HBOT and IV sodium thiosulfate were initiated in addition to standard wound care. The pt was treated at 2.0 ATA for 90 minutes for a total of 40 treatments.

**Results:** The patient had complete resolution of the cutaneous lesions and associated pain at the completion of treatment. The patient had no recurrence at 6-week follow-up.

**Summary/Conclusions:** We present a case of successful resolution of cutaneous CUA with adjunctive HBOT and sodium thiosulfate. HBOT promotes local tissue neogenesis and improves wound healing. HBOT for treatment of CUA warrants further study.



# F 107

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## Hyperbaric Oxygen Therapy for Cutaneous Calciphylaxis - A Case Study

Swaby JA, Morgan M, Jennings MS, Santiago W, Heyboer M

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**Introduction:** Calcific uremic arteriopathy (CUA), or calciphylaxis, is an often fatal complication of patient's with end stage renal disease (ESRD). It is characterized by cutaneous ischemia and necrosis as a result of calcification of arterioles.

Standard care involves management of serum calcium, evaluation for hyper-parathyroid, and good wound care. Hyperbaric oxygen therapy (HBOT) has been used as an effective adjunctive therapy. We report on a patient with CUA successfully treated with local wound care, HBOT, IV sodium thiosulfate, and parathyroidectomy.

**Materials and Methods:** A 55-year-old female with systemic lupus erythematosus and end stage renal disease (ESRD) who developed cutaneous CUA of her right medial thigh. The pt had local wound care, parathyroidectomy, serum calcium management, sodium thiosulfate, and HBOT (2.0 ATA for 90 minutes daily x 14 treatments).

**Results:** The patient's cutaneous CUA resolved upon completion of HBOT.

**Summary/Conclusion:** We present a case of cutaneous CUA treated successfully with adjunctive HBOT. Cutaneous CUA results from local destruction of the microvasculature, and HBOT has been shown to stimulate neogenesis and wound healing within damaged locally hypoxic tissues. Adjunctive HBOT appears to resolve cutaneous CUA and warrants further study.

# F 108

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## **Acute Idiopathic Sudden Sensorineural Hearing Loss Successfully Treated with Hyperbaric Oxygen Therapy - A Case Study**

Swaby JA, Morgan M, Jennings MS, Santiago W, Heyboer M

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**Introduction/Background:** Idiopathic sudden sensorineural hearing loss (ISSHL) is defined as hearing loss of at least 30 dB occurring within a 3-day period over at least three contiguous frequencies. ISSHL is one of the most recently added UHMS indications for hyperbaric oxygen therapy (HBOT). We present the case of a patient treated with HBOT for ISSHL.

**Materials and Methods:** A 54-year-old male presented with acute onset of left ear hearing loss and tinnitus. He was evaluated by ENT 3 days after onset. His audiogram revealed sensorineural hearing loss in his left ear and he was started on steroids without improvement. He presented to the UH ED 6 days after onset while awaiting outpatient consultation with an ENT specializing in ISSHL. MRI brain was unremarkable. ENT diagnosed ISSHL and recommended Hyperbaric Medicine consultation. The patient was treated with HBOT (2 ATA x 90 minutes) for 10 treatments.

**Results:** The patient demonstrated significant objective improvement in his hearing. A repeat audiogram showed significant improvement (MCL 80dB HL, UCL 100 dB HL pre-treatment to 0 dB HL post-treatment) on the left. He had complete resolution of hearing loss at one-month follow-up.

**Summary/Conclusions:** A number of publications have evaluated the use of HBOT in ISSHL, including 8 randomized control trials showing improvement in hearing deficits from moderate/severe to slight/no impairment. Such improvement in hearing deficits has the effect of markedly improving functional quality of life. In this case, the patient had complete resolution of his hearing loss following steroids and HBOT. This supports the use of HBOT for ISSHL.

# F 109

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## **Hyperbaric Oxygen Therapy for Central Retinal Artery Occlusion**

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**Introduction/Background:** Central retinal artery occlusion (CRAO) is a relatively rare eye emergency involving dramatic painless vision loss. None of the many possible treatments is widely accepted, and prognosis is generally poor. Up to 40% of patients may improve with conventional treatment. Hyperbaric oxygen (HBO) has also demonstrated efficacy in animals and humans, but only a small number of studies are available. Treatment of CRAO with hyperbaric oxygen is routine at HCMC, and we have recently seen an increasing number of CRAO patients due to local retinal specialists' awareness and understanding of HBO.

**Materials and Methods:** We conducted a retrospective case series of CRAO patients who received HBO therapy in 2014 to review the presentation, workup, and timing of HBO and other treatments for patients with CRAO. We measured: time from onset to presentation and HBO treatment, comorbidities, medical workup (echocardiography and carotid imaging), visual acuity and peripheral vision both at presentation and after HBO treatments, and measurements from optical coherence tomography, visual-field testing, and follow-up ophthalmology exams where available.

**Results:** Cardiovascular comorbidities were prevalent in CRAO patients, and 91% presented with no measurable visual acuity. Around 70% of patients initially presented to a healthcare facility less than six hours from onset, but only 16.7% received HBO within six hours from onset. Where available, 50% of patients had broad improvement in visual fields, while another 50% had only some improvement. Medical workup routinely included echocardiography but carotid imaging and other treatments varied. 66.7% of all patients and 75% of those who presented less than 6 hours from symptom onset had improvement in their vision.

**Summary/Conclusions:** Our results suggest that HBO therapy can improve vision over standard of care in the treatment of CRAO. As we design our own prospective trial, there is clearly room to further standardize care and improve time to HBO for patients with CRAO.

# F 110

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Using Objective Criteria to Determine Level of Pain

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**Introduction:** Pain is not only difficult to describe, but it is hard to quantify. The usual pain scoring systems (e.g. Visual Analogue Scales (VAS) and their permutations) are highly subjective. This makes the evaluation and management of pain imprecise. We have developed a pain evaluation system useful for wounds as well as other types of pain that is objective, simple to use, and helpful in determining what treatment interventions are indicated

**Methods:** Three domains to assess pain are used: 1) Current requirements for medications, 2) How pain restricts activities and 3) How pain interferes with thought processes. Specific criteria are used for grading each domain on a 0 to 10 scale. On the medication requirement domain, level 1 pain does not require medication, while level 5 can be managed with over-the-counter agents, and level 10 requires hospitalization for intravenous analgesics. Analogous objective criteria are used to determine the scores of the other two domains.

**Results:** Although patients with DFUs often are insensate around their wounds, disabling neurogenic pain often is experienced proximal to their feet and requires appropriate medical management. The activity and thought processes domains quantify how severe the neurogenic pain is. VAS and their permutations do not differentiate this very well. Narcotic requirements do not correlate well with how pain interferes with the patient's activities or thought processes. The system utilized at the Long Beach Memorial Medical Center Hyperbaric Program helps determine those patients who magnify their pain symptoms.

**Conclusions:** This approach for assessing analgesic use, activity level interference, and hindrance of thought processes objectifies the evaluation of pain and helps to determine optimal pain management, for wounds in general and DFUs in particular.

# F 111

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Quantifying Compliance in Patients with Diabetic Foot Ulcers

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**Introduction:** Patient compliance is essential for achieving successful, long duration outcomes in patients with diabetic foot ulcers (DFUs). However, compliance is difficult to quantify as evidenced by a dearth of literature on quantifying it. This presentation demonstrates our approach to quantifying compliance and provides correlations with wound severities and wound outcomes.

**Methods:** A review of the literature shows that there is a plethora of articles stating how important patient compliance is for achieving successful wound healing outcomes, but none attempts to quantify it. As part of our Goal Score tool, compliance is graded as full, some, or none. Compliance was further subdivided into five local and five systemic components and graded each on a 2 (full) to 0 (none) scale. Outcomes were then reviewed in 15 patients with "Healthy," "Problem" or "Futile" diabetic foot wound types. Our Wound Score was utilized to determine objectively each wound type. Then, Wound Scores were compared with the compliance assessment of the Goal Score, as well as with five local and five systemic components of compliance.

**Results:** The patients with the worse DFU wounds had the lowest compliance scores whereas the patients with the healthiest wounds had the highest scores. Local and systemic measures of compliance correlated closely with each other as well as with the simple to determine Goal Score assessment of compliance.

**Conclusions:** Our study demonstrates that compliance can be quantified in patients with DFUs, and the higher the compliance marks, the better the outcomes. This aids in making management decisions, especially in the clinic setting.

# F 112

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION:

## Hyperbaric Oxygen Salvation of Prolonged Male Genitalia Strangulation

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**Introduction/Background:** Penile strangulation injuries are a rare but well-documented entity in the medical literature. Prolonged ischemia and secondary edema have led to both parenchymal and neurovascular damage.

**Materials and Methods:** This case report is novel both in that this is the first report in the medical literature of both severe, prolonged penile and testicular strangulation (as opposed to penile only) , and in that this is the first report in the literature of treatment of penile strangulation by hyperbaric oxygen (HBOT).

**Results:** A patient who was greater than 72 hours after initial strangulation, with a largely numb, diffusely edematous and eccemotic external genitalia approximating in size and shape and that of a large eggplant, was begun on HBOT. Failure to respond to conservative therapy by that point was thought to be consistent to ongoing ischemia-reperfusion injury and poor outcome. He received five HBOT exposures over the next three days and returned home after a nine day hospital stay with much improved sensation and significantly improved edema and eccemosis and no further complications.

**Summary/Conclusion:** Post-strangulation penile treatment varies based on the grade of injury incurred. Typically, when severe necrosis or gangrene is present a partial or total penectomy is performed. We propose hyperbaric oxygen treatment as a novel, minimally invasive method of penile and/or testicular sparing in such injuries.

# F 113

ORAL PRESENTATION TIME:

POSTER PRESENTATION TIME: 1530 - 1600

RESIDENT COMPETITION: YES

## **Understanding Vasculopathy as a “Acute Peripheral Arterial Insufficiency” Amenable to Hyperbaric Oxygen Therapy**

Pearson M, Harch P, Hardy S, Murphy-Lavoie H, LeGros T, Wyatt H, Yontz D and Van Meter K.

LSU Undersea and Hyperbaric Medicine Fellowship, New Orleans, Louisiana, June 2015.

tlegros1@cox.net

**Introduction:** Vasculopathy is a rare and poorly recognized embolic disease affecting the lower extremities resulting in micro-infarctions of the medium arterioles. Vasculitis is an inflammation of the arterial and venous walls leading to decreased circulation. These micro-infarctions of the skin are painful and each have their own treatments. Both should be diagnosed early with biopsy and receive optimal wound care. Those with vasculopathy should also be treated with anticoagulants and hyperbaric oxygen therapy (HBO<sub>2</sub>) to restore microcirculation and obviate reperfusion injury. Patients with vasculitis should be treated with topical steroids.

**Methods:** We analyzed a convenience sample of patients that continually failed standard wound care management until vasculopathy was appropriately diagnosed. Data was collected regarding previous diagnoses, therapeutic management, progression and regression of healing, and the use of laboratory studies and biopsies in patient evaluations.

**Results:** These patients presented with complicated painful leg ulcerations that were ultimately diagnosed as vasculopathy. All received optimized wound care with minimal responses in pain reduction or healing. After biopsies showed livedoid vasculopathy, all were treated with aspirin or dipyridamole, and trental or peltal. Elevated homocysteine levels were found in 75% of our patients and they were treated with vitamin supplementation to reduce homocysteine levels. These interventions were successful in treating the micro-infarctions and pain in 75% of cases. One patient was treated with Lovenox and then Xarelto, but developed a GI bleed and anticoagulation was discontinued. These were fairly recalcitrant wounds, and required more than nine months to heal unless HBO<sub>2</sub> was utilized.

**Conclusions:** Vasculopathies encompass changes in the dermal microcirculation that disrupts tissue perfusion by developing focal thrombosis leading to skin infarction. Treatment with vitamin B supplementation, rheologic agents, anticoagulation, and HBO<sub>2</sub> promotes faster wound healing and pain reduction. HBO<sub>2</sub> should be considered for these arterial insufficiency wounds.

**PLENARY:**  
**“TROUBLE WITH BUBBLES: LESSONS IN  
ALTITUDE DECOMPRESSION SICKNESS”**

**Brett Hart, MD**

**4:00PM – 5:00PM**



**Objectives:**

1. Discuss the physiology and risk factors associated with development of altitude-induced decompression illness.
2. Discuss the increasing incidence of “rapid decompression” events in USN high performance aircraft and the potential for civilian hyperbaric facility support in providing needed recompression treatment.
3. Provide attendees with recommended strategies for initial diagnosis and management of altitude decompression illness, so as to reduce delays to recompression treatment and maximize chances for patient recovery.



# AUTHOR LISTING

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