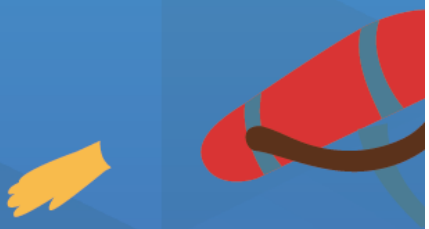
EUBS 2019

Hyperbaric Medicine & The Brain

& Conference Program Book of Abstracts

SEPTEMBER 09-12.2019

Tel Aviv, Israel



EUBS European Underwater and Baromedical Society

WELCOME LETTER

Dear EUBS members, the organizing committee of the EUBS 2019 scientific meeting, sponsors and corporate members!

On behalf of the EUBS executive committee and the local organizers of this year's scientific EUBS meeting in Tel-Aviv, Israel we present to you the yearly conference- and abstract book of the meeting. It is with great pleasure and appreciation that we now for the 3rd time welcome the EUBS members to a scientific EUBS conference organized by our honored Israeli colleagues.

In 1989 dr. Yehuda Melamed and his team organized the 15th EUBS conference in Eilat. Then in 1999 dr. Naomi Bitterman organized the 25th EUBS conference in Haifa- and Eilat and now in 2019 the 45th EUBS conference is organized by professor dr. Shai Efrati and his team.

The scientific program presented in this abstract book represents all fields of interest within the EUBS scientific world including diving- and hyperbaric medicine, environmental medicine as well as new exploratory and emerging fields of hyperbaric medicine. This year focus particularly on the effects- and application of hyperbaric oxygen therapy in brain disorders. Although controversies exist, recent years of research into this field has driven an increasingly intense discussion on the immune- and metabolic pathways modelling effects of hyperbaric oxygenation in the brain. We hope this meeting will provide all participants with inspiration and constructive discussions to promote more research and future work to stimulate even more high-quality clinical evidence. Overall, the organizing committee must be congratulated on a very interesting scientific program for the EUBS 2019 scientific meeting.

This year the meeting is also sponsored by several well-known companies and institutions. The EUBS wish to express our great appreciation to all sponsors whose contributions are essential for a successful meeting. Looking forward to seeing you all in Tel-Aviv September 2019 I wish you all a good conference!

Yours Sincerely,
Ole Hyldegaard
EUBS president

EUBS2019-HBOT & THE BRAIN

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Mr. Yonatan Zemel (MBA/RN)
Dr. Zemer Wang

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Wang Zemer, Israel
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Yanir Yoav, Israel

***In Alphabetical Order**

GENERAL INFORMATION

Conference Venue
David InterContinental Hotel
12 Kaufman Street Tel Aviv 61501, Israel
Tel: +972 3 795 1111
Fax: +972 3 795 1112
Website: www.intercontinental.com/telaviv

City / Country
Tel-Aviv, Israel

Currency
The New Israeli Shekel (NIS) is the official national currency. All major credit cards are accepted in Israel.

Conference Official Language
The official language of the conference is English. No translation service will be available.

Registration Desk
Location: Foyer, Convention Center, 1st floor.
Opening hours:
Monday September 9th 08:00-20:00
Tuesday September 10th 08:00-14:00
Wednesday September 11th 08:00-14:00

Full Registration Package
- All Scientific Sessions
- Exhibition Entrance
- Daily lunch, Coffee Breaks and Light Snacks
- Poolside Welcome Cocktail
- Gala Dinner Celebration
- Conference Printed Material

Single Day Package
- All Scientific Sessions
- Exhibition Entrance
- Daily lunch, Coffee Breaks and Light Snacks
  Conference Printed Material

Delegate Participation Badge
- The Badge can be Obtained @ the Registration Desk
- The Badge Is Mandatory for Admission
- Please Wear Your Badge During All Social Events and all Scientific Sessions

Speakers Preview Room
Speaker Preview Room is located at the foyer on the 1st floor.
All presentations should be submitted to the technical person, at least 90 minutes prior to the session, **the earlier the better**. The first morning session speakers are asked to submit their presentation the day before.

GENERAL INFORMATION

Communication

- The Organizing Committee is available via WhatsApp:
  +972-52-729-4123 (08:00-19:00)
- Wi-Fi is available at the conference floor for all EUBS2019 participants.

General Assembly

Annual EUBS General Assembly
September 12th, 08:30 AM
Location: Plenary Hall

Certificate of Attendance

Participating delegates will receive a Certificate of Attendance post conference via email.

Transportation from Tel Aviv

The International airport is 20-30 drive minutes from the city of Tel Aviv. There is a scheduled train service while private taxis are available 24 hours a day. Approximate cost is 150-200 NIS (40-60 USD) per taxi from the city center.

Personal Insurance

We recommend that all participants will acquire personal travel and health insurance during their stay in Israel.

Sponsor Liability & Insurance

The conference secretariat and organizers cannot accept liability for personal loss of or damage to private property of participants, either during or indirectly arising from the EUBS 2019 Conference.

Exhibition / Support

Mrs. Orna Sahahrabany
Sponsorship & Exhibition Specialist
Email: orna@kenes-events.com

Conference Organizer

Kenes Israel
Kenes Group Building
2 Hayarden St. Airport City, Ben Gurion Airport
70100, Israel
Email: EUBS2019@kenes-events.com

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SOCIAL EVENTS

~ Welcome Cocktail ~

Date - 9.9.19 | **Time -** 19:30

Venue: David InterContinental Hotel, **Poolside**

Dress Code: Causal

Come welcome your peers and celebrate with a taste of Israeli cuisine, Local wine&beer, Live latino tunes, while enjoying a Mediterranean sea breeze.

~ GALA Party Night ~

Date - 11.9.19 | **Time -** 20:00

Venue: David InterContinental Hotel, Convention Center, Grand Ballroom

Dress Code: Smart

The EUBS 2019 Grand Finale!

Join us for a celebration of friends, peers, and colleagues enjoying local market style cuisine and a Live Concert, followed by a night of dancing. It will also be a chance to thank you all for your participation and contributions.

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GENERAL PROGRAM OVERVIEW

SUNDAY 8/9/2019	MONDAY 9/9/2019	TUSEDAY 10/9/2019	WEDNESDAY 11/9/2019	THURSDAY 12/9/2019	FRIDAY 13/9/2019	SATURDAY 14/9/2019
	08:00-09:00 Gathering & Registration	**08:00-08:30** Gathering & Registration	**08:00-08:30** Gathering & Registration	**08:00-08:30** Gathering & Registration	Eilat Diving Workshop	Eilat Diving Workshop
06:00-16:00 Casserea pre-diving workshop	**09:00-09:30** Opening Ceremony			**08:30-09:30** Annual EUBS General Meeting		
	09:30-19:00 Scientific Sessions	**08:30-18:30** Scientific Sessions	**08:30-18:30** Scientific Sessions	**09:30-13:30** Scientific Sessions		
17:00-19:00 EUBS ExComm Meeting				**13:30-14:00** Closing Ceremony		
19:30-22:00 EUBS ExComm Dinner	**19:30 -23:00** Poolside Welcome Cocktail	Free Night	**20:00** GALA Party Night	**14:30** Bus departure to Eilat and Jeruslem.		

& Scientific Sessions Conference Schedule

Monday, September 9th, 2019

08:00-09:00	**Gathering & Registration**
09:00-09:30	**Opening Ceremony and Conference Perspective**

Session 1: Regeneration & Age- Related Functional Decline **Chairpersons:** Elliot Sussman, Zemer Wang	09:30 -13:00
09:30-09:55	How to Die Young at A Very Old Age **Nir Barzilai**
09:55-10:20	The Rational for Use of HBOT as an Intervention for Age Related Functional Decline **Shai Efrati**
10:20-10:45	The Effect of Hyperbaric Oxygen Therapy in Healthy Aging – Prospective Controlled Study **Amir Hadanny**
10:45-11:15	**Coffee Break, Professional Exhibition & Poster Viewing**
11:15-11:30	Hyperbaric Oxygen Therapy for Treatment of Stroke Reveals Transfer of Healthy Mitochondria **Cesar V. Borlongan**
11:30-11:45	Hyperbaric Oxygen Preconditioning Can Reduce Post-Abdominoplasty Complications: a Retrospective Cohort Study **Tali Friedman**
11:45-12:00	The Effect of Intermittent Hyperoxia on Stem Cell Mobilization and Cytokine Expression **KJ MacLaughlin**
12:00-12:15	Osteogenic Differentiation in Human Adipose-Derived Stem Cells Effects Modulated by Hyperbaric Oxygenation **Gerardo Bosco**
12:15-12:30	The Effect of Hyperbaric Oxygenation Therapy on The Heart **Marina Leitman**

12:30-13:00	Open Discussion
13:00-14:00	**Lunch**

Session 2: Traumatic Brain Injuries & Post-concussion **Chairpersons:** John S Peters, Pasquale Longobardi	14:00-16:30
14:00-14:25	Exploring the Invisible Wound: Interface Astroglial Scarring, a Pattern of Brain Damage Unique to Blast Exposed Service Members with Prominent Persistent Behavioral/Neurologic Symptomatology **Daniel Perl**
14:25-14:45	The Use of HBOT for Post-Concussion Syndrome- Current Status and Future Perspective **Amir Hadanny**
14:45-15:05	All You Need to Know About Youth Concussion **Gillian Hotz**
15:05-15:15	Hyperbaric Oxygen Therapy with a Pediatric TBI Population - Challenges of Assessment and Preliminary Neurocognitive and Neuroimaging Effects **Lynn Rothstein**
15:15-15:30	Tinnitus after Traumatic Brain Injury **Michael Hoffer**
15:30-15:45	HBOT for TBI induced Tinnitus **Nathan Shlamkovitch**
15:45-16:00	Hyperbaric Oxygen Enhances Neuroprotection and Stimulates Neurogenesis after Brain Injury in Rats: An Intermediary Role of Interleukin-10 **Predrag Brkic**
16:00-16:15	Adjunctive Hyperbaric Oxygen Therapy (Hbot) With Intensive Neurorehabilitation in Persons with Disorders of Consciouness (Doc) With Subacute Brain Oinjury **Srivastava Abhishek**
16:15-16:30	Open Discussion
16:30-17:00	**Coffee Break, Professional Exhibition & Poster Viewing**

Session 3: Post Traumatic Stress Disorder (PTSD) & Fibromyalgia **Chairpersons:** Rachel Lev-Wiesel, Jeff Lowenkron		17:00-19:00
17:00-17:30	The Nature of PTSD: Past Present and Future Perspectives **Ilan Kutz**	
17:30-17:55	Brain Circuitry in PTSD: A Combined Behavioral, Computational, and Physiological Approach **Roni Paz**	
17:55-18:20	Hyperbaric Oxygen Therapy for Combat Associated PTSD - First Presentation of the Results of the Prospective Randomized Controlled Trial on Israeli Veterans **Keren Doenyas-Barak**	
18:20-18:45	Fibromyalgia 2019: Central Sensitization, Neuromodulation and HBOT - Putting It All Together **Jacob Ablin**	
18:45-19:00	Open Discussion	
Social Event	**Poolside Welcome Cocktail**	19:30-23:00

Tuesday, September 10th, 2019

08:00-08:30	**Gathering & Registration**
Session 4: Diving: Decompression & Gases Physiology **Chairpersons**: Ben Aviner, Jean-Eric Blatteau	08:30-10:30
08:30-08:55	Perspective and Vision for Future Naval S&T - Funded Research for Diving Medicine **Sandra Chapman**
08:55-09:20	The Challenge of Engineering Decompression Procedures **Jean-Pierre Imbert**
09:20-09:35	Is Metabolism Responsible for Pre-Existing Small Gas Pockets Inducing Vascualr Gas Emboli During Divers` Decompressions? **Jean-Pierre Imbert**
09:35-09:50	A New Innovative Approach to Decompression Sickness Modeling **Michael Strauss**
09:50-10:05	Bubbles in Vascular Plexus of The Skin in Cutaneous Decompression Sickness. Report of Cases **Eduardo Garcia**
10:05-10:20	Proteomics Applied to Decompression Sickness **Jacky Lautridou**
10:20-10:30	Open Discussion
10:30-11:00	**Coffee Break, Professional Exhibition & Poster Viewing**
Session 5: Diving: Diving: Rescue Operations & Accidents **Chairpersons:** Yehuda Melamed, Ingrid Eftedal	11:00-13:00
11:00-11:30	The Challenging Cave Rescue Mission of 13 Young Boys in Thailand, 2018 **Natthasak Woracharoensri**

11:30-12:00	A Disabled Submarine Escape and Rescue **Yinnon Matsliah**
12:00-12:20	Israel Diving Community – A Case Study on DCI **Adam Konstantinovsky**
12:20-12:35	Decompression Disorders of Central Origin: Pathogenetic Hypothesis **Alberto Fiorito**
12:35-12:50	Newly Designed Albumin-Derived Perfluorocarbon - Based Nanocapsules Improve Clinical Symptoms, Mortality and Histological Findings in Decompression Illness **Dirk Mayer**
12:50-13:00	Open Discussion
13:00-14:00	**Lunch**

Session 6: Neuroplasticity and Cognitive Functions — 14:00-16:00

Chairpersons: Carla Vandeweerd, Shai Efrati

Session sponsored by SIEMENS Healthineers

14:00-14:25	How to Evaluate Brain Performance? **Gil Suzin**
14:25-14:40	The Cognitive Profile Before and After HBOT – What Can We Learn from The First 1000 Patients? **Rahav Boussi-Gross**
14:40-15:05	The Potential Biological Effect of Hyperbaric Oxygen Therapy on Alzheimer Disease- What Can Be Learned from Animal Models **Uri Asheri**
15:05-15:20	The Effect of Hyperbaric Oxygen on the Human Brain Microstructure: New Insights from MRI-DTI Studies **Merav Catalogna**
15:20-15:35	Perfusion MRI- Basics and Its Clinical Use for Evaluating the Effect of Hyperbaric Oxygen Therapy on Brain Perfusion **Efrat Sasson**
15:35-15:50	Beneficial Effects of Hyperbaric Oxygenation in Autism Spectrum Disorders **Necip Cem Kinaci**

15:50-16:00	Open Discussion
16:00-16:30	**Coffee Break, Professional Exhibition &Poster Viewing**

Session 7: Sport Physiology and Performance Enhancement **Chairpersons:** Costantino Balestra, Dror Ofir	16:30-18:30
16:30-16:55	Mitochondrial Physiology from Hypobaric to Hyperoxic Conditions **Erich Gnaiger**
16:55-17:15	The Physiology of Aerobic Exercise **Eldad Yaacobi**
17:15-17:35	The Effect of Hyperbaric Oxygen Therapy on Elite Athletes– Randomized Controlled Trial – Interim Analysis **Amir Hadanny**
17:35-17:50	The Effect of Hyperbaric Oxygen Therapy on Mitochondrial Function **Yafit Hachmo**
17:50-18:05	Influence of Elevated Oxygen Fraction On Breathing Gas Consumption During Physical Workload In Shallow-Water Submersion **Jochen Schipke**
18:05-18:20	Hyperoxia and the Cardiovascular System: Data from the Pressure Chamber **Jochen Schipke**
18:20-18:30	Open Discussion

Wednesday, September 11th, 2019

08:00-08:30	**Gathering & Registration**	
Session 8: Pulmonary and CNS Oxygen Toxicity (I) **Chairpersons:** Ran Arieli, Robert van Hulst		08:30-10:30
08:30-08:55	Introduction to Pulmonary and CNS Oxygen Toxicity **Yehuda Arieli**	
08:55-09:20	Available Oxygen Toxicity Models vs. Available Oxygen Toxicity Data **Barbara Shykoff**	
09:20-09:45	The Power Equations for Pulmonary and CNS Oxygen Toxicity **Ran Arieli**	
09:45-10:00	Detecting Pulmonary Oxygen Toxicity; One Breath Is Still the Future **Pieter Van Ooij**	
10:00-10:15	Imaging the effect of Hyperbaric Oxygen on Single Neuron and Overall Behavior in A Novel, live Animal Model **Ricarina Rabinovitz**	
10:15-10:30	Alterations of The Blood-Brain Barrier with Hyperbaric Oxygen and Ramifications for Traumatic Brain Injuries **Michael Strauss**	
10:30-11:00	**Coffee Break, Professional Exhibition & Poster Viewing**	
Session 9: Pulmonary and CNS Oxygen Toxicity (II) **Chairpersons:** Yehuda Arieli, Ole Hyldegaard		11:00-13:00
11:00-11:20	Oxygen Toxicity on Cellular Level **Andreas Koch**	
11:20-11:40	Oxygen Toxicity in Closed Circuit Human Diving **Yoav Yanir**	

11:40-12:00	CNS Oxygen Toxicity in HBO Treatments (HBOT) **Wang Zemer**
12:00-12:15	Blood Glucose Levels and Hyperbaric Pressure in Sod2 Enzyme Knockdown Mice **Mirit Eynan**
12:15-12:30	Impulse Oscillometery and Spirometry as Indices of Pulmonary Oxygen Toxicity Severity **David Fothergill**
12:30-13:00	Panel Discussion: "Redefining the Limits of Oxygen Exposure" **Balestra Costantino** **Jacek Kot** **Pieter Van Ooij** **Andreas Koch** **Ran Arieli** **Barbara Shykoffo**
13:00-14:00	**Lunch**
Session 10: Diving: HPNS and Pressure Physiology **Chairpersons:** Yoram Grossman, Jean-Claude Rostain	14:00-16:00
14:00-14:30	HPNS: Symptoms, Signs, Risk Factors and Short- And Long-Term Mechanisms **Jean-Claude Rostain**
14:30-14:50	Hyperbaric Pressure Modulation of Neurotransmitters' Receptors **Alice Bliznyuk**
14:50-15:10	Hyperbaric Pressure Modulation of Ionic Channels **Ben Aviner**
15:10-15:25	Effects of Inert Gas Narcosis on Attention and Memory Impairment **Jacek Siewiera**
15:25-15:40	Noble Gases Simulations at High Pressure **Alice Bliznyuk**
15:40-16:00	Open Discussion: "HPNS vs. Inert Gas Narcosis"

16:00-16:30	**Coffee Break, Professional Exhibition &Poster Viewing**	
Session 11: Diving: Physiology **Chairpersons:** Yinnon Matsliah,Aleksey Sobakin		16:30-18:30
16:30-16:45	Breath-Hold Deep Diving and the Lung **Jochen Schipke**	
16:45-17:00	Arterial Blood Gases in Breath-Hold Divers at the Breaking Point **Gerardo Bosco**	
17:00-17:15	Mental Stress May Cause High Gas Consumption and Heart Rate in Rapid Descending Scuba Divers **Nico Schellart**	
17:15-17:30	Scuba Diving and Congenital Heart Disease in Children and Adolescents **Christian Beyer**	
17:30-17:45	Scuba Divers Pulmonary Edema. Review of 10 Years Experience **Jose M. Inoriza**	
17:45-18:00	Latency to CNS-Oxygen Toxicity and Eeg Patterns Are Affected by Blood Magnesium Levels in Rats **Ben Aviner**	
18:00-18:15	Hyperbaric Oxygen Dose-Response of NMDA Receptor Currents **Alice Bliznyuk**	
18:15-18:30	Open Discussion	
Social Event	**Gala Dinner & Party**	20:00

Thursday, September 12th, 2019

08:00-08:30	**Gathering & Registration**
08:30-09:30	**Annual EUBS General Assembly**

Session 12: Wound Care I **Chairpersons:** Jacek Kot, Yair Bechor	09:30-11:30
09:30-09:45	Hyperbaric Oxygen Therapy for the Ischemic Diabetic Foot: A Systematic Review and Meta-Analysis **Robin Brouwer**
09:45-10:00	Algorithm for Evaluation and Management of Diabetic Foot Ulcers **Michael Strauss**
10:00-10:15	Uses and Abuses of Negative Pressure Wound Therapy and Biologics to Aid in Wound Healing **Michael Strauss**
10:15-10:30	Economic Evaluation on Hbot In Tissue Repair **Pasquale Longobardi**
10:30-10:45	The Effect of Hyperbaric Oxygen Therapy on Healing of Chronic Leg Ulcers **Abhishek Srivastava**
10:45-11:00	Hyperbaric Oxygen Therapy for Hemorrhagic Cystitis **Shachar Finci**
11:00-11:15	The Effects of Hyperbaric Oxygen Preconditioning in an Experimental Model of Acute **Kidney Injury** **Predrag Brkic**
11:15-11:30	Open Discussion
11:30-12:00	**Coffee Break, Professional Exhibition & Poster Viewing**

Session 13: CO Intoxication **Chairpersons:** Yoav Yanir, Claus M. Muth	12:00-13:30
12:00-12:15	Case Series of Carbon Monoxide Poisoning in Singapore **Ng Peng**
12:15-12:30	Treatment of Carbon Monoxide Intoxication/Encephalopathy with Hyperbaric Oxygen Therapy - 26 Years' experience 1986 - 2013 - Retrospective Review of an Alternative Treatment Protocol **Jeffrey Cooper**
12:30-12:45	Hyperbaric Oxygen Treatment beyond Usual Therapeutic Window Enhances Neurological Recovery after Carbon Monoxide Poisoning **Jeffrey Cooper**
12:45-13:00	Carbon Monoxide Stress Test **Jeffrey Cooper**
13:00-13:15	Treatment of Co Intoxication Due to Shisha Consumption: A Trend and Cause Analysis **Sven Dreyer**
13:15-13:30	Open Discussion
13:30-14:00	**Closing Ceremony**
14:00	**Light Lunch**

Abstracts

Session 1: Regeneration & Age- Related Functional Decline

HOW TO DIE YOUNG AT A VERY OLD AGE

Nir Barzilai,
Institute for Aging Research at Albert Einstein College of Medicine

Aging has a biology and this biology can be targeted as shown in many pre-clinical models. Targeting the biology of aging extends health span as well as may extend life span. We studied familied of centenarians and have discovered functional variant that allowed them to live healthier for longer. Some of those discoveries have already been developed into medications.

As we start developing drugs to target aging, it is important to realize that regulatory bodies across the world, such as the FDA, do not recognize aging as a target. That means that health care providers will not pay for a drug and that pharmaceuticals will not develop drugs that do not have business horizon. Our approach as scientists is demonstrated by creating the TAME trial (Targeting Aging with MEtformin), how a single drug can delays many aspects of aging. In agreement with FDA on the outcomes of the study and depending on positive results, the study is being launched and we are convinced that next-generation drugs and combinations will extend health span and lifespan.

THE RATIONAL FOR USE OF HBOT AS AN INTERVENTION FOR AGE RELATED FUNCTIONAL DECLINE

Shai Efrati
The Sagol Center for Hyperbaric Medicine and Research, Assaf-Harofeh Medical Center, Israel.
Sackler School of Medicine and Sagol School of Neuroscience, Tel-Aviv University, Israel.

Beginning at birth, developmental programs results in age-related changes in gene expression, growth and organs physiological function. With ageing, from a certain stage, organ functions start to deteriorate. There are 4 major denominators common to most age related physiological deterioration:

1. **Atherosclerosis**- Marked narrowing in the blood vessels which are responsible for oxygen supply can cause relative hypoxia in the supplied tissues. As the tissues suffer from hypoxia, mitochondria function, which is directly dependent on the partial pressure of oxygen, decreases significantly.
2. **Mitochondria dysfunction-** During aging, mitochondria number and function decline. Mitochondrial dysfunction accelerates atherosclerosis, which by itself increases mitochondrial damage. This vicious cycle seen in many of the age related diseases .
3. **Inflammation**- Inflammation is a natural and highly regulated response that provides protection when infection occurs. However, if left unregulated, these same processes can cause tissue injury and damage. Inflammatory processes, particularly those resulting in chronic inflammation, have been implicated in most age related diseases.
4. **Stem cells**- Age related depletion and reduce functionality of stem cells contributes to decrease regenerative capacity along the aging process shifting the net balance from regeneration to deterioration.

In recent years, there is growing evidence on the regenerative effects of hyperbaric oxygen therapy (HBOT). Even though many of the beneficial effects of HBOT can be explained by improvement of tissue oxygenation, it is now realized that intermittent increase of oxygen concentration can induce many of the mediators and cellular mechanism that are usually induced during hypoxia but without the hazardous hypoxia - termed Hyperoxic-hypoxic paradoxes. The intermittent hyperoxic exposure during HBOT can affect HIF-1 levels, MMP activity, VEGF, induce stem cells proliferation, augmented circulating levels of endothelial progenitor cells (EPCs) and angiogenesis factors. HBOT can decrease the inflammatory response in endothelial and thus promote vascular recovery. The fluctuations in the dissolved oxygen, generated by HBOT, directly sensed and affect the mitochondria and several animal and human studies have demonstrated the beneficial effects of HBOT on the mitochondrial level.

In this lecture we will review and discus the rational for the use of HBOT as an intervention that can target important aspects of age related functional decline.

THE EFFECT OF HYPERBARIC OXYGEN THERAPY IN HEALTHY AGING – PROSPECTIVE CONTROLLED STUDY

Amir Hadanny
The Sagol Center for Hyperbaric Medicine and Research, Assaf-Harofeh Medical Center, Israel.
Sackler School of Medicine and Sagol School of Neuroscience, Tel-Aviv University, Israel.

The understanding of the aging process and its related functional decline has gained fundamental advances in the last decade. From the thermodynamic perspective, aging can be considered as an energy deficit condition tightly related to tissue ischemia and mitochondria dysfunction.
Hyperbaric oxygen therapy (HBOT) has three major pharmacodynamic effects: First, it can eliminate the relative tissue ischemia induced by atherosclerosis. Second, because mitochondria function is directly influenced by the partial pressure of dissolved oxygen, HBOT can amend mitochondrial dysfunction. Third, HBOT induces proliferation and mobilization of stem cells needed for tissues regeneration. Accordingly, from the pathophysiologic perspective, HBOT has the potential to revert the negative regeneration/ degeneration balance associated with aging.
However, the effects of HBOT have never been evaluated on the healthy aging population. For the first time, a comprehensive study was developed to evaluate the therapeutic effects of HBOT on healthy aging.
A prospective controlled trial was conducted in Assaf Harofeh Medical Center, from 2016 to 2019, on 60 healthy volunteers, 65 years old and higher. The volunteers were divided to 2 groups: the treatment group – was evaluated at baseline, and after 3 months of HBOT daily sessions (60 daily session, 90 min of 100% oxygen at 2 ATA with 5 minutes air breaks every 20 minutes). The control group was evaluated 3 times – at baseline, after 3 months of follow up/control without intervention, and after a crossover of 3 months of HBOT protocol. Comprehensive evaluations included cognitive assessments, high resolution MRI scans of the brain heart abdomen and pelvis, cardiopulmonary exercise test (CPET) , blood samples (including stem cells and immune system cells differentiation), skin and muscle biopsies.
In this lecture, for the first time, the study results will be presented and will include the HBOT effects on:

- Cognitive Functions
- Brain perfusion (MRI)
- Brain microstructure (MRI-DTI)
- Cardiac Perfusion (MRI)
- Cardiopulmonary functional capacity – CPET
- Stem cells
- Mitochondrial function analysis
- Immune system cells differentiation

HYPERBARIC OXYGEN THERAPY FOR TREATMENT OF STROKE REVEALS TRANSFER OF HEALTHY MITOCHONDRIA

Eleonora Napoli[1], Trenton Lippert[2], and **Cesar V. Borlongan[2]**

[1]Department of Molecular Biosciences, University of California Davis, Davis, CA 95616
[2]Department of Neurosurgery and Brain Repair, University of South Florida Morsani College of Medicine, Tampa, FL 33612 USA

Background and Aim: Accumulating evidence has implicated dysfunctional oxygen consumption and metabolic activity in stroke. We now recognize a close interaction between abnormal function of the brain cells' mitochondria, which represent key cell organelles responsible for cellular energy production, and the onset and progression of secondary cell death in stroke. Many stroke symptoms may be the result of abnormal mitochondrial function, reduced oxygen availability and consequent energy production deficits. Accordingly, treating stroke with mitochondria-based treatments may present as an appealing approach in resolving this oxygen/energy abnormality that can lead not only to normalization of mitochondrial function, but also to improved quality of life of patients. That stroke is accompanied by mitochondrial dysfunction forms the basis of our overarching hypothesis that finding a treatment, such as hyperbaric oxygen therapy (HBOT), that can restore mitochondrial structure and function may exert therapeutic effects against stroke. To this end, we tested the hypothesis that HBOT preconditioning mitigates cell death in primary rat neuronal cells (PRNCs) through the transfer of mitochondria from astrocytes.
Methods: Primary rat neuronal cells were subjected to a 90-minute HBOT treatment at 2.5 absolute atmospheres prior to either tumor necrosis factor-alpha (TNF-alpha) or lipopolysaccharide (LPS) injury to simulate the inflammation-plagued secondary cell death associated with stroke and traumatic brain injury (TBI). After incubation with TNF-alpha or LPS, the cell viability of each group was examined.
Results: There was a significant increase of cell viability accompanied by mitochondrial transfer in the injury groups that received HBOT preconditioning compared to the injury alone groups (44 ± 5.2 vs 68 ± 4.48, $n = 20$, $P < 0.05$). The transfer of mitochondria directly after HBOT treatment was visualized by capturing images in 5-minute intervals, which revealed that the robust transfer of mitochondria commenced soon after HBOT and persisted throughout the treatment.
Conclusion: This study shows that HBOT preconditioning stands as a robust prophylactic treatment for sequestration of inflammation inherent in stroke and TBI, possibly facilitating the transfer of resilient mitochondria from astrocytes to inflammation-susceptible neuronal cells in mitigating cell death. Our long-term goal is the therapeutic application of HBOT and the use of mitochondrial outcomes as a robust biomarker in stroke patients. The demonstration that HBOT is therapeutic for stroke may similarly benefit many diseases characterized by mitochondrial deficits.

HYPERBARIC OXYGEN PRECONDITIONING CAN REDUCE POST-ABDOMINOPLASTY COMPLICATIONS: A RETROSPECTIVE COHORT STUDY

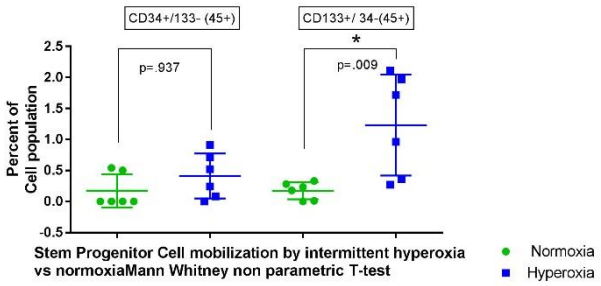
Tali Friedman, Shaked Menashe, Geva Landau, Yaakov Seligman, Moni Friedman, Amir Hadanny, Matan Sherf, Itay Wiser, Shay Efrati, Lior Heller
Plastic Surgery, Assaf Harofeh Medical Center, Israel

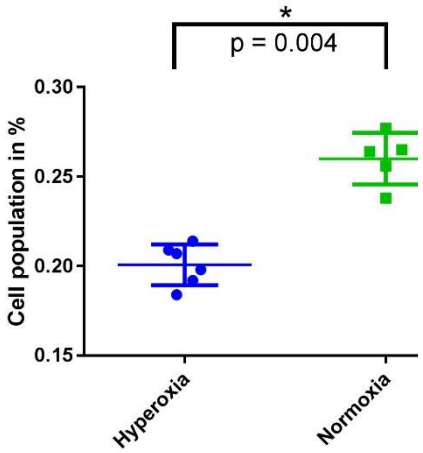
Background: hyperbaric oxygen therapy (hbot) can improve wound healing and was found to have positive preconditioning effects in animal models. Among esthetic surgical procedures, abdominoplasty poses the highest rate of postoperative complications. The aim of this study was to evaluate the effect of preoperative hbot as a preconditioning treatment on the expected post-surgical complications.
Methods: we conducted a retrospective cohort study among patients who underwent abdominoplasty at our institute and private practice during january 2012–november 2017. Patients who received perioperative hbot were compared with patients who did not receive hbot. Surgery complication data as well as demographic and perioperative data from patient records were collected.
Results: the study included 356 patients. Of them, 83 underwent hbot preoperatively. Using preoperative hbot, postoperative complications were significantly reduced from 32.6% (89 patients) to 8.4% (7 patients), p 0.001. Moreover, 17 (6.2%) patients in the comparison group and none in the hbot group experienced necrosis ($p = 0.016$).
In the multivariate analysis, preoperative hbot was an independent protective factor against postoperative complications (odds ratio, 0.188; 95% confidence interval: 0.082–0.432; p 0.001). After propensity score matching, the study results remained the same.
Conclusions: preoperative hbot can reduce postoperative complication rate in abdominoplasty patients. Nevertheless, further prospective studies are warranted to validate the findings and characterize patients who benefit the most from this treatment.

THE EFFECT OF INTERMITTENT HYPEROXIA ON STEM CELL MOBILIZATION AND CYTOKINE EXPRESSION

KJ MacLaughlin[1,2], Greg Barton[1,2], Rudolf Braun[1,2], Marlowe Eldridge[1,2]
[1]Department of Pediatrics, University of Wisconsin, USA
[2]John Rankin Laboratory of Pulmonary Medicine, University of Wisconsin, USA

Background: Mechanisms of HBOT include inducing transduction cascades modulating cytokine expression and proangiogenic stem/progenitor/cells(SPC). Accepted clinical HBOT PO_2 range minimally from 1520 mmhg, however, little is known about oxygen therapy below 760mmhg. Central dogma in oxygen therapy research asserts low values of hyperoxia are benign. We ask, are low values of PO_2 physiologically inert when used as a sham in research? Here we measure cytokine expression and SPC at PO_2 320mmhg.
Methods: Twelve, rats were divided into two-groups. The treatment group exposed to 319mmhg and the control group to room air. Treatments were 5days/week, 2hours/day/total/20hrs. Monocytes/cells harvested from venous blood were prepared for flow cytometry using antibodies for CD45+/CD34+/CD133+. Flow cytometry using BDLSRII/DIVA analyzed with flowjo software. Statistics with Mann-Whitney(p0.05)
Results: Treated animals showed increase mobilized CD133+/34-SPC's (p=0.009) compared to controls. Tnfα significantly decreased in treated animals(p=0.004).
Conclusions: To our knowledge, this is the first study to demonstrate physiological activity at PO_2 320mmhg. Data demonstrates a small oxygen dose ($PO_2$320mmHg), similar to that used as a placebo/sham in HBOT research, also mobilizes spcs and reduces tnfα plasma levels. Together these findings support the likelihood of biologic activity, consubstantial with HBOT, being activated at much lower dose of hyperoxia than previously postulated. These data further suggest that the interpretation of HBOT research using low levels of increased PO_2 as a sham/placebo/control should be re-evaluated. Future research examining oxygen/dose relationship will further elucidate the physiological effect of various doses of intermittent hyperoxia. This research will also establish basal active levels.





OSTEOGENIC DIFFERENTIATION IN HUMAN ADIPOSE-DERIVED STEM CELLS EFFECTS MODULATED BY HYPERBARIC OXYGENATION

Barbara Zavan[1], Chiara Gardin[2], Gloria Bellin[2], Enrico Camporesi[3], Alex Rizzato[4], Lisa Ricci[5], **Gerardo Bosco**[4]
[1]Department of Medical Sciences, University of Ferrara, Italy
[2]Maria Cecilia Hospital, GVM Care & Research, Translational Research Center (CRT), Italy
[3]Tampa General Hospital, TeamHealth Research, USA
[4]Department of Biomedical Sciences, University of Padova, Italy
[5]Domus Medica, Hyperbaric Medical Centre, San Marino

Background. This is the first work evaluating the influence of HBO on the proliferation and osteogenic differentiation of human Adipose-Derived Stem Cells (hadscs).
Methods. Hadscs were exposed daily for 60 minutes, and up to 21 days, at 2,4 ATA and 100% O_2. The effects of elevated pressure (hyperbarism, HB) or elevated oxygen (hyperoxia, HO) alone on stem cells proliferation and differentiation were contextually analyzed. Hadscs were either used as undifferentiated cells (1) or as osteogenic-committed cells (2). 1. Cells were exposed to osteogenic stimuli for 7 days before starting treatments. 2. The undifferentiated cells were cultured with osteogenic differentiation factors, alone or in the presence of the pro-inflammatory cytokines. Cell proliferation was evaluated by means of the MTT assay at 7, 14, and 21 days of culture, whereas osteogenic differentiation was assessed by gene expression analysis of osteogenic markers and quantification of extracellular calcium deposition after Alizarin Red S staining.
Results. HBO did not affect cell proliferation and osteogenic differentiation when hadscs were exposed to osteogenic stimuli for 7 days before treatment. Similarly, proliferation and osteogenic properties of hadscs did not differ between HBO, HB, and HO conditions when treatments started contextually to the osteogenic differentiation of the cells.
HBO significantly decreased proliferation when hadscs were cultured in osteogenic differentiation medium supplemented with the pro-inflammatory cocktail. Remarkably, the reduction in cell proliferation was accompanied by an increase in osteogenic differentiation, as demonstrated by elevated calcium deposition and up-regulation of osteogenic markers.
Conclusions. Our data seem to indicate that the exposure of hadscs to HBO under in vitro simulated inflammatory conditions enhances differentiation towards the osteogenic phenotype, providing evidence of the potential application of HBO in all those processes requiring bone regeneration.

THE EFFECT OF HYPERBARIC OXYGENATION THERAPY ON THE HEART

Marina Leitman[1,3], Shai Efrati[2,3,4], Shmuel Fuchs[1,3], Amir Hadanny[2,3,4], Zvi Vered[1,3]
[1]Cardiology, Shamir Medical Center
[2]Sagol Center for Hyperbaric Medicine and Research, Shamir Medical Center
[3]Sackler School of Medicine, Tel Aviv University
[4]Sagol School of Neuroscience, Tel Aviv University

Background: Hyperbaric oxygenation therapy is successfully implemented for the treatment of several disorders. There is not enough evidence to support hyperbaric oxygenation therapy in cardiovascular diseases. Data on the effect of hyperbaric oxygenation on echocardiographic parameters in asymptomatic patients is limited. The current study sought to evaluate the effect of hyperbaric oxygenation therapy on echocardiographic parameters in asymptomatic patients.
Methods: 31 consecutive patients underwent a 60-day course of hyperbaric oxygenation therapy in an attempt to improve cognitive impairment. In all subjects, echocardiography examination was performed before and after a course of hyperbaric oxygenation therapy. Conventional and speckle tracking imaging parameters were calculated and analyzed.
Results: The mean age was 70±9.5years, 28 [90%] were males. History of coronary artery disease was present in 12 [39%]. 94% suffered from hypertension, 42% had diabetes mellitus. Baseline wall motion abnormalities were found in 8 patients, however, global ejection fraction was within normal limits. During the study, ejection fraction [EF], increased from 60.7±6.0 to 62.3±5.2%, p=0.02. Left ventricular end systolic volume [LVESV], decreased from 38.1±13.3 to 35.4±13.3ml, p=0.01. Myocardial performance index [mpi] improved, from 0.29±0.1 to 0.26±0.1, p=0.03. Left ventricular [LV] global longitudinal strain increased from -19.3±3.2% to -20.1±3.3%, p=0.036. Twist increased from 18.3±6.7° to 23.1±6.3° p=0.01, due to improvement in the apical rotation, from 11.8±4.4° to 16.1±5.6°, p=0.004. Regional strain increased significantly in the apical and the anteroseptal segments.
Conclusions: Hyperbaric oxygen therapy appears to improve left ventricular function, especially in the apical segments, and is associated with better cardiac performance.

Session 2: Traumatic Brain Injuries & Post-concussion

EXPLORING THE INVISIBLE WOUND: INTERFACE ASTROGLIAL SCARRING, A PATTERN OF BRAIN DAMAGE UNIQUE TO BLAST EXPOSED SERVICE MEMBERS WITH PROMINENT PERSISTENT BEHAVIORAL/NEUROLOGIC SYMPTOMATOLOGY

Daniel P. Perl. [1,2]
1 Department of Pathology and 2Neurology, Uniformed Services University of the Health Sciences, F. Edward Hébert School of Medicine, Bethesda, MD, 20814
2 Center for Neuroscience and Regenerative Medicine, Brain Tissue Registry and Neuropathology Core, Bethesda, MD, 20814

Since 2001 approximately 2.6 million U.S. service members have been deployed to the Middle East in the war on terror. Almost daily, allied forces encountered attacks with high explosives that often resulted in mild traumatic brain injuries (mild TBIs, concussions). For current military conflicts, these blast TBIs have been called the "invisible wound" since numerous service members suffer from debilitating persistent neurologic and behavioral symptoms in the absence of detectable abnormalities on routine neuroimaging and lack of medical knowledge about underlying pathophysiology. Despite innumerable deaths from high explosives, especially in the military since the early 20th century, the medical literature offers few studies characterizing acute or chronic neuropathologic sequelae in the human brain after blast exposure.

At the Uniformed Service University (USU), we have developed the Center for Neuroscience and Regenerative Medicine (CNRM) Brain Tissue Repository, the only such facility in the world specifically dedicated to the study of military TBI. Within this facility, we have identified a distinct and previously undetected pattern of damage to the human brain in blast-exposed cases (see *Lancet Neurol.* 2016 15:944-953). We found astroglial scarring, indicative of neuroanatomical areas with damage, in a distinctive pattern occurring at the interfaces of tissues with differing densities, for example, between cerebrospinal fluid and brain parenchyma (subpial) and between the gray and white matter within brain parenchyma. This neuroanatomical lesion distribution at tissue interfaces complements known biophysics of the blast wave impinging on the human body. We have not found interface astroglial scarring (IAS) in postmortem brain tissues of patients with histories of impact TBIs (in the absence of blast exposure). Furthermore, most cases identified with IAS following blast TBI failed to show evidence of significant pathologic *tau* accumulation, indicative of chronic traumatic encephalopathy (CTE), a disorder mostly encountered following repeated impact TBI. These data suggest that the clinical phenotype of persistent neurologic/behavioral symptoms, particularly as seen after blast exposure, may be due to specific neuropathophysiology, and significantly differs from what is seen in non-blast forms of impact TBI.

In this talk, these findings will be illustrated and their pathophysiology and implications for diagnosis, prevention and treatment will be discussed.

The opinions expressed here are those of the author and are not necessarily representative of those of the Uniformed Services University, the United States Department of Defense or the United States Army, Navy or Air Force.

TRAUMATIC BRAIN INJURY – FROM PATHOGENESIS TO TREATMENT

Amir Hadanny
the Sagol Center for Hyperbaric Medicine and Research, Assaf-Harofeh Medical Center, Israel.
Sackler School of Medicine and Sagol School of Neuroscience, Tel-Aviv University, Israel.

Traumatic brain injury (TBI) has become a major public health concern worldwide for both civilian and military populations. Each year, an estimated 10 million cases of TBI arise globally, 1.7-3.8 million occurring in the United States alone.
Although most of these injuries are considered mild, they may initiate a chain of metabolic reactions including inflammation, toxic neurotransmitters release, calcium mediated injury, mitochondria dysfunction as well as vascular damage. This cascade leads to the reduction of energy level in the form of ATP molecules and the ability to restore hemostais. Under certain circumstances, this can propagate brain injury and eventually lead to persistent brain injury/post-concussion syndrome (PCS). Some of Persistent PCS patients develop chronic traumatic encephalopathy (CTE) which is a progressive neurodegenerative syndrome.
Today, there is no agreed-upon effective standard of care treatment/intervention for TBI. Experts agree that novel neurotherapeutic methods to repair brain damage are needed more than ever before.
The use of HBOT has been suggested by many basic science studies evaluating the different pathophysiological effects of HBOT on TBI-induced damage. In recent years, prospective clinical trials have been performed and demonstrated the significant value of HBOT in these patients at both the acute and chronic stages.
In this lecture, we will discuss the pathophysiology of TBI, the current available treatments and the evidence behind the treatment using HBOT.

ALL YOU NEED TO KNOW ABOUT YOUTH CONCUSSION

Gillian A. Hotz

Department of Neurological Surgery, KiDZ Neuroscience Center, WalkSafe™, BikeSafe™ & SkateSafe ™ Programs

Background: There are millions of children globally a year that sustain concussions from a variety of mechanisms. In the United States about 800,000 cases of children under 17 years present to Emergency Rooms and diagnosed with concussion with thousands that go undiagnosed. The identification and management of these children needs to be improved and sports related concussions (SRC) need to be better managed with protocols and surveillance systems. This presentation will present a review of post concussion syndrome (PCS) and a model to be utilized to develop and implement a comprehensive, concussion care program for youth that sustain SRC.

Methods: A comprehensive concussion care model was developed to improve management of SRC.

A 6 Steps to Play Safe model (pre season education, baseline testing, sideline testing, clinic visit, gradual return to play and concussion injury surveillance) was developed, implemented, evaluated and has been sustained by a clinical and research team at UHealth Sports Medicine.

Results: A total of about 200 high school athletes from 35 public high schools, per year sustain a SRC. Football related injuries account for about 56%, 12% soccer, 10% basketball and 7% wrestling and 15% other. The mean age is 16 years and males to females injured 74% vs 26%. The most common complaint being headaches and fatigue with the overall average days to return to play from date of injury about 26 days for both males and females.

Conclusion: With annual training and education concussion injury reporting has increased over the years. With the ability to track and monitor reported SRCs on a yearly basis has improved school reporting and proper management as well as implementation of prevention programs. Future research is needed to continue to follow these athletes long term to monitor any neurological, psychological and behavioral issues. This model can be implemented in other school districts and across all school age levels to assist with managing concussions and in improving education and awareness of this issue.

HYPERBARIC OXYGEN THERAPY WITH A PEDIATRIC TBI POPULATION- CHALLENGES OF ASSESSMENT AND PRELIMINARY NEUROCOGNITIVE AND NEUROIMAGING EFFECTS

Lynn M. Rothstein[1], Amir Hadanny[1], Merav Catalogna[1], Gil Suzin[1] and Shai Efrati[12]
[1]Sagol Center for Hyperbaric Medicine and Research, Shamir Medical Center (Assaf- Harofeh), Zerifin, Israel, [2]Sackler Faculty of Medicine & Sagol School of Neuroscience, Tel Aviv University, Israel.

In recent years there has been an increase in cumulative data from prospective clinical studies on the neuro-therapeutic effects of hyperbaric oxygen therapy (HBOT) on an adult population suffering from chronic neurocognitive impairments due to traumatic brain injury (TBI) years after the acute insult. Less research has been conducted on the therapeutic effects of HBOT on a pediatric population who have suffered a brain insult.

Pediatric TBI studies pose many challenges with regards to the neurocognitive and neuro-imaging evaluations. In general, children who have suffered a TBI show significantly more signs of impulsivity, fatigue, anxiety and attention difficulties resulting in the need for more emotional support, encouragement, time and adaptations during the evaluation process. Consideration must be taken in terms of the setting, tools used and analysis methods both for the neurocognitive and neuroimaging phases of evaluation.

In the current pilot, several studies of children who have suffered from prolonged-post concussion syndrome due to TBI and who have received HBOT at the Sagol Center for Hyperbaric Medicine and Research will be presented with an emphasis placed on improvement in cognitive functioning and cerebral blood flow and blood volume in the brain.

The lecture will review the challenges in performing neuro-cognitive evaluations in a pediatric population, review findings in several pilot cases already treated with HBOT and briefly review the ongoing prospective randomized clinical trial that is currently ongoing in Israel in a pediatric population suffering from post-concussion syndrome due to TBI who are being treated with HBOT.

TINNITUS AFTER TBI

Hoffer Michael
Otolaryngology & Neurological Surgery, University of Miami Hospitals and Jackson Memorial Hospital, USA

Traumatic brain injury is an increasingly important and increasingly common public health concern. Studies have shown that a majority of the symptoms that affect individuals are neurosensory in nature and over 50% of these individuals report problems with their ears including tinnitus. Unlike many of the other symptoms seen in TBI, post-traumatic tinnitus does not resolve spontaneously and can persist for years after the injury. In this presentation we examine the newest theories of tinnitus generation and how these theories apply to post-traumatic tinnitus. These theories include new data on tinnitus changes at the brain and at the cellular level. With this background, we then examine diagnostic modalities and a host of novel treatment approaches that can be used in this challenging group of patients. We follow this didactic talk with case presentations that highlight how we apply these approaches in patients with post-traumatic tinnitus.

THE EFFECT OF HYPERBARIC OXYGEN TREATMENT (HBOT) ON POST TRAUMATIC CENTRAL-TYPE CHRONIC DISABLING TINNITUS

Nathan Shlamkovitch MD*; S. Efrati MD**; H. Golan MD***; G. Fishlev MD**; H. Gavriel MD*

*- Otolayngology head & Neck surgery; **-Hyperbaric Unit; ***Nuclear medicine
Shamir (Assaf Harofeh) Med. Center, Zerifin, Israel

Objective: To define the effect of Hyperbaric Oxygen Treatment (HBOT) on post traumatic central-type chronic disabling tinnitus

Study Design and Methods: Twenty-one patients suffering from post traumatic chronic disabling tinnitus of the central type were included in this study. Seven of the patients suffered from prior whiplash injury, 13 from blunt head injury and one from direct blunt injury to the ear. Tinnitus was typed using Tinnitus disabling score questionnaire, a full oto-neurological physical examination, a complete hearing evaluation (including audiometry up to 20,000 Hz, and ABR), a complete Tinnitus matching score and brain SPECT scan with ECD was done in order to evaluate brain metabolism/perfusion. Half of the group were used as a control group, and after a 3-4 months of follow up and re-evaluation were crossed to receive HBOT. Post HBOT evaluation was done after mean of 60 HBOT daily sessions, 5 days per week, of 90 minutes of 100% oxygen at 2 ATA with 5 minutes' air breaks every 20 minutes. HBOT was initiated 1-5 years after the acute trauma. Information about dizziness, cognitive state and quality of life was taken as well as performance of brain SPECT scan, and compared pre and after treatment. A seven years follow up has been taken place after these patients

Results: Fourteen out of 21 patients (66%) had a major improvement of their tinnitus. Six out of these 14 patients (29%) reported that the tinnitus ceased completely. The average score of the Tinnitus Handicap Inventory (THI) score decreased from 87 to 42. All of the patients (100%) reported a significant improvement in their quality of life. Eighteen out of the 21 patients also complained of post traumatic dizziness, 13 of which (72%) reported an improvement in their dizziness after treated by HBOT. Nineteen out of the 21 patients also described a major cognitive depravation (memory, concentration abilities etc.). Fifteen of these patients (79%) reported a significant improvement of their cognitive abilities. An objective improvement in brain perfusion was demonstrated in brain SPECT scan in 18 out of the 21 patients (86%). Eleven of the 14 patients that benefited from the treatment report that the improvement of tinnitus lasts for 7 years period of followup. Two patients reported a regration throughout time but responded well for 20 adjuvant HBOT treatments. One patiebt did not benefit the HBOT adjuvant treatment though reporting that the reccurent tinnitus is less intense than the tinnitus before starting treatment.

Conclusions: HBOT can significantly improve post traumatic central-type chronic disabling tinnitus even years after the acute insult. More studies are needed in order to specify who are the patients that can benefit the most from this treatment.

HYPERBARIC OXYGEN ENHANCES NEUROPROTECTION AND STIMULATES NEUROGENESIS AFTER BRAIN INJURY IN RATS: AN INTERMEDIARY ROLE OF INTERLEUKIN-10

Predrag Brkic[1], Jeremic Rada[1], Marina Djelic[1], Sanja Dacic[2], Sanja Pekovic[3]
[1]Institute of Medical Physiology "Richard Burian", School of Medicine, University of Belgrade, Serbia
[2]Institute of Physiology and Biochemistry, Faculty of Biology, University of Belgrade, Serbia
[3]Department of Neurobiology, Institute for Biological Research "Sinisa Stankovic", University of Belgrade, Serbia

Background: Interleukin-10 (IL-10) is an important anti-inflammatory cytokine expressed in response to brain injury, where it facilitates the resolution of inflammatory cascade, which if prolonged causes secondary brain damage. The aim of present study was to elucidate the role of IL-10 in the effects of treatment with HBO (HBOT) on neuroprotection and neurogenesis after traumatic brain injury (TBI) in rats.
Methods: The TBI in male Wistar albino rats (10 weeks old) was induced by sensorimotor cortex ablation (SCA). Animals were organized into following groups (n = 10 per group): Control (C) intact animals, Control + HBO (CHBO) intact animals subjected to HBOT, Sham control (S) animals that underwent surgical procedure without damaging the brain tissue, Sham control + HBO (SHBO), Lesion group (L) – the right sensorimotor cortex was removed by SCA and Lesion + HBO (LHBO). HBO protocol: pressure applied 2.5 absolute atmospheres (ATA), for 60 minutes, once daily for 10 days.
Effects of HBOT were monitored using immunohistochemistry and double immunofluorescence labeling.

Results: Data analysis revealed that HBOT applied after SCA enhances expression of nestin, a marker of neural stem cells, in the subventricular zone (SVZ) and in the cortex around the lesion site. After HBOT, migratory pathway, consisted of $IL10^{+}$/$nestin^{+}$ cells, extends from SVZ up to the lesion site. Additionally, astrocytes around the lesion site expressed IL-10 indicating that IL-10 secretion from astrocytes may contribute to anti-apoptotic effects of HBOT.
Conclusion: Our findings indicate that IL-10 plays an important role in HBOT stimulation of neurogenesis and neuroprotection after TBI.
Keywords: brain injury, neuroprotection, neurogenesis, interleukin-10

ADJUNCTIVE HYPERBARIC OXYGEN THERAPY (HBOT) WITH INTENSIVE NEUROREHABILITATION IN PERSONS WITH DISORDERS OF CONSCIOUNESS (DOC) WITH SUBACUTE BRAIN OINJURY

Abhishek Srivastava, Faiz Mohammed, Tushar Sonawane, Navita Purohit
Physical Medicine & Rehabilitation, Kokilaben Dhirubhai Ambani Hospital, India

Objective: To assess the effect of adjunctive HBOT in improving neurological outcome in people with DOC with subacute brain injury.
Material and Methods: Retrospective study of persons with traumatic/non traumatic brain injury, underwent rehabilitation and adjunctive HBOT from April 2017-March 2019. HBOT was given in Perry ™ Monoplace Chamber 1.5 ATA for 60 minutes each session when neurologically and medically stable. Neurological status was assessed by Glasgow Coma Scale (GCS) and Glasgow Outcome Scale (GOS) before and after the intervention.
Results: 99 persons (M:F:82:17) with sub acute brain injury (traumatic brain injury (TBI) : Intracerebal haemorrhage (ICH) : ischemic stroke (IS): 49 : 35 : 15), age 8 – 86 years (48.8) were included. 57 persons underwent decompression surgery (24 TBI; 27 ICH; 6 IS). HBOT was started 10 days–240 days after the initial brain insult (mean 61 days). Number of HBOT sessions varied from 6 – 68 (mean 18).
GCS score pre-intervention improved from E1M1VT – E4M4V2 (m 5.94) to E4M3VT – E4M6V5 (m 9.91). The maximum improvement in GCS was in the component of spontaneous eye opening. GOS score pre-intervention were Grade II-84, III–12 and IV–3 persons. The post HBOT, GOS was gradei-3, II-44, III - 34, IV-6 and V–12 persons.
Out of 84 persons in GOSII pre intervention: 6 improved to gradev, 2 to IV, 29 to III, 44 in II and 3 in gradei. Of 12 persons in GOS III: 5 improved to gradev, 2 to IV and 5 in Grade III. Of 3 patients in Grade IV GOS: 1 improved to Grade V and 2 in gradeiv post intervention.
Conclusion: Adjunctive HBOT is safe and tolerated well by persons with subacute brain injury and can contribute in improving neurological and functional outcome of persons with DOC. Large scale randomized studies are required.

Session 3: Post Traumatic Stress Disorder (PTSD) & Fibromyalgia

THE NATURE OF PTSD: PAST PRESENT AND FUTURE PERSPECTIVES

Ilan Kutz
The Sagol Center for Hyperbaric Medicine and Research, Assaf Harofeh Medical Center, Israel.

This presentation reviews the history of Post-Traumatic Stress Disorder (PTSD), the variety of its clinical manifestations, the trajectory of this disorder(s) throughout human lifespan, and the treatment modalities that have been developed to date.
Though PTSD is regarded as a modern-age affliction, ancient texts reveal that it is as old as humanity itself. In the 20th century, with the advancement of the sciences of psychology and neurobiology, the concepts of trauma and its sequelae have undergone repeated re-conceptualizations, with both psychological and biological mechanisms implicated in its origin. However, despite impressive strides in the scientific exploration of this disorder, there is still no effective solution for this debilitating condition. PTSD has proven to be rather resistant to existing pharmacological treatment. Likewise, psychological interventions have met, at best, with limited success.
Yet, the accumulated recent findings of PTSD-related neurobiological research have laid down the theoretical groundwork for several innovative approaches. The HBOT-PTSD study, presented in this conference, is one of these potential intervention modalities, unique in its presumed mechanism of structural rehabilitation of brain tissues damaged by former trauma.

BRAIN CIRCUITRY IN PTSD: A COMBINED BEHAVIORAL, COMPUTATIONAL, AND PHYSIOLOGICAL APPROACH

Roni Paz
Department of Neurobiology, Weizmann Institute of Science, Rehovot, Israel

I will describe few behavioral models of learning and memory in emotional situations and how they might lead to pathological behaviors that underlie mood and anxiety disorders as well as PTSD. I will then show findings about the neural representation of these processes, both in humans using imaging (fMRI), and in animal models using electrophysiological recordings from single-neurons. The evidence suggests that mild imbalance in neural circuits that connect the amygdala and the prefrontal-cortex play a major role in emotional learning and memory formation, and in the transition from adaptive normal function to psychiatric conditions

HYPERBARIC OXYGEN THERAPY FOR COMBAT ASSOCIATED PTSD - FIRST PRESENTATION OF THE RESULTS OF THE PROSPECTIVE RANDOMIZED CONTROLLED TRIAL ON ISRAELI VETERANS

Doenyas-Barak Keren, Kutz Ilan, Catalogna Merav, Daphna-Tekoha Shir, Efrati Shai
The Sagol Center for Hyperbaric Medicine and Research at Assaf Harofeh Medical Center. Sackler School of Medicine and Sagol School of Neuroscience, Tel-Aviv University, Israel.

Post-traumatic stress disorder (PTSD) is a brain's long-term imprint of a traumatic event. In recent years there is growing knowledge about the biological consequences responsible for the unremitting nature of the disease. Using new and advanced brain imaging technologies demonstrates stunned, hypoperfused, inactive brain regions. Clinical studies published in recent years, present evidences that hyperbaric oxygen therapy (HBOT) can induce neuroplasticity in metabolic dysfunction brain regions even years after the acute insult. The new understanding regarding the biological long term damage caused by PTSD and knowledge gained on the neuroplasticity effect of HBOT initiated to an ongoing research program aiming to evaluate the effect of HBOT on patients suffering from long standing unremitting PTSD. The first clinical study done on patients suffering from fibromyalgia syndrome (FMS) due to childhood sexual abuse (CSA) was recently published. It demonstrated promising effects on trauma symptoms as well as brain microstructure and functionality.
In this lecture we will present, for the first time, the results from a prospective, randomized cross-over trial done in our instituted aimed to evaluate the effect of HBOT on veterans suffering from resistant combat associated PTSD. The preliminary results indicates that HBOT induce significant clinical improvement that can be explained by increase in brain perfusion/metabolism most dominant in the frontal cortex and the hippocampi. Changes in brain activation, as demonstrated in fMRI, were mainly seen in the left frontal lobe and left insula. The brain imaging indicates a trend towards normalization after HBOT.
Gathering the clinical data with advanced brain imaging enables an objective characterization of the damage brain tissue, objective monitoring of the biological response to treatment and in the future better selection of patients who can benefit the most from HBOT.

FIBROMYALGIA 2019: CENTRAL SENSITIZATION, NEUROMODULATION AND HBOT - PUTTING IT ALL TOGETHER

Jacob N. Ablin
Rheumatology and Internal Medicine Tel Aviv Sourasky medical center,
Tel Aviv University, Israel

Fibromyalgia syndrome, clinically characterized by widespread pain, fatigue, tenderness and associated symptoms, is currently considered a prototype of a chronic pain condition in which sensitization of the central nervous system plays a prominent pathogenetic role. This paradigm has undergone various developments and modifications since the neurophysiological phenomenon of "central sensitization" was first described by Woolf, over three decades ago. Neurophysiological mechanisms of central sensitization, such as a decrease in descending inhibitory control of pain (decreased conditioned pain modulation, or CPM) have been identified both in fibromyalgia as well as in overlapping functional disorders; aberrations in the CNS levels of various neurotransmitters involved in pain modulation have been identified and altered patterns of connectivity have been identified through the implementation of advanced functional neuro-imaging techniques such as fMRI. In addition, genetic and epigenetic markers involved in increasing susceptibility to chronic pain conditions have been described and constitute an active field of research. Moreover, the subtle role of inflammatory and immune mediated components such as glial cell activation in another piece of the puzzle regarding centralized pain, which recently has been given the taxonomic title of "nociplastic pain" – differentiating it from more classic forms of pain such as nociceptive and neuropathic pain. In light of all these exciting areas of research, it becomes increasingly apparent that fibromyalgia is all about a change in the way the central nervous systems carries out the processing of pain; thus, the concept of neuromodulation, i.e. attempting to re-wire the way in which pain is processed and transmitted, becomes particularly appealing.

Many forms of neuromodulation have been developed over the years, with ancient techniques such as movement meditative treatment (yoga, tai – chi etc.) also appearing to work towards the goal of altered brain function, even if not classically thought of in this context. More recently external physical stimulation has been developed, including trans cranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) have been applied to the treatment of chronic pain. Neurofeedback is another a non-invasive technique aimed at changing brain function.

Hyperbaric oxygen therapy (HBOT) stands out as a unique method towards modulation central nervous system function. It has recently been demonstrate to achieve positive clinical results in the treatment of fibromyalgia and a number of recent studies have described aspects of the specific effects of HBOT on fibromyalgia patients including favorable changes in brain perfusion, alterations of immune function etc. Thus, as our understanding of the intricate paradigm of central sensitization and nociplastic pain continues to broaden, HBOT is likely to find its place in our toolkit for achieving neuromodulation and as a therapeutic option for treating fibromyalgia and related conditions.

Session 4: Diving: Decompression & Gases Physiology

PERSPECTIVE AND VISION FOR FUTURE NAVAL S&T-FUNDED RESEARCH FOR DIVING MEDICINE

Sandra Chapman
Warfighter Protection & Applications Division Office of Naval Research

With the goal of providing improved manned undersea capabilities, the Office of Naval Research has funded Undersea Medicine (UMed) research from the early 1950s until the present day. The medical risks for Navy Divers include issues common to all divers such as nitrogen narcosis, decompression sickness (DCS), overexpansion injuries, hyperoxia/hypoxia, hypothermia, hypercapnia, etc. Furthermore, Navy divers are often placed in heightened risks and encounter hazardous exposures as they perform salvage, underwater construction and rescue operations as well as the complex and strenuous missions, anytime and anywhere performed by special warfare units.
The UMed program is producing fundamental biomedical discoveries to both mitigate health risks and augment warfighter capabilities in this extreme environment. Research investments include the discovery of selective gas channels, microparticle-based theory of DCS etiology, and delayed seizures in an animal model of oxygen toxicity with ketone ester supplementation. Recently, the UMed program has branched out to invest in new technologies that can offer innovative approaches to traditional diving gear such as a helmet-integrated heads-up display, Diver Augmented Visual Display (DAVD) and new approaches to underwater breathing apparatus that pulls oxygen directly from ocean water, Gill-Inspired Life Support (GILS). Additional areas of interest include a workshop next winter to discuss Contamination Risk to Underwater Divers (CRUD), new materials for a flexible, lightweight Atmospheric Dive Suit (SCALES), and new approaches to monitoring brain health/activity.

THE CHALLENGE OF ENGINEERING DECOMPRESSION PROCEDURES

Jp Imbert
Divetech, Divetech, France

Between operations and research, there is always a person in charge of designing decompression procedures for the immediate needs of worksites. This job is about managing ignorance because there is never enough knowledge nor data to do it right. Dr Bill Hamilton had his way to express it:" what works, works". It is the domain of trials and you have to accept the responsibility for the errors. But there are recipes: more oxygen and slower ascents will always solve the problem and the diving industry has empirically developed reasonable procedures.

This situation is the result of the shortage of funds for navies and laboratories. The industry cannot rely any longer on authorities to provide diving procedures and needs to commit itself. And the industry is entitled to do so because it works with management of changes systems. Small changes will produce small effects and, provided the monitoring is efficient, will allow improving procedures. Monitoring is the key to diving procedures evolution. In the good all times, DCS were plenty and it was easy to work statistics with monitoring databases. Nowadays, DCS have almost disappeared and the measurement of performances relies on more subtle physiological changes and therefore on more advanced analysis.

Designing tables requires more than intuition and algorithms are needed for a systematic approach. Current saturation procedures used in the offshore commercial diving are based on simplistic safe ascent criteria bound to basic exponential gas models. But there are variations that are better than the others. Reverse engineering of these differences has allowed identifying patterns that can be linked to the latest scientific advances: pre-existing small gas pockets, pulmonary filter transfer function, oxidative stress, arterial bubbles, etc. These bricks of understanding permit building better procedures, in the sense the always demanding industry wants to go: safe but also highly flexible operations.

IS METABOLISM RESPONSIBLE FOR PRE-EXISTING SMALL GAS POCKETS INDUCING VASCUALR GAS EMBOLI DURING DIVERS` DECOMPRESSIONS?

Jp Imbert[4], Sali Murat Egi[1], Peter Germonpré[2], Costantino Balestra[3]
[1]Computer Engineering Department, Galatasaray University, Turkey
[2]Centre for Hyperbaric Oxygen Therapy, Military Hospital Brussels, Belgium
[3]Environmental & Occupational (Integrative) Physiology Laboratory, Haute École Bruxelles-Brabant HE2B, Belgium
[4]Divetech, Divetech, France

Background: The risk of decompression sickness has been linked to the presence of vascular gas emboli (VGE) after surfacing from the dive.
VGE can be quantified by ultrasound Doppler and precordial echography. However, for an identical dive, VGE monitoring of divers shows a) a large variation related to individual susceptibility, and b), for a same diver, variations related to pre-dive preconditioning.
We hypothesize that metabolism acts through the oxygen window and sustain a population small pre-existing gas pockets that are the precursors of circulating VGE.
Method: We derive a coherent system of assumptions to describe these static gas pockets, located on the endothelium at hydrophobic sites, that are activated during decompression and become the source of VGE. We first refer to the tissue inherent unsaturation and show that it can generate and stabilize static gas phases in the diver prior to the dive. We then use Non-Extensive thermodynamics to calculate interfacial energy from volume rather than curvature. The final equation links gas pocket volume directly to the metabolism.
Results: The resulting equation describes both individual variations and preconditioning experiments from a metabolic point of view. It explains the variability in VGE counts based on age, fitness, type and frequency of physical activities. It predicts the results of vibrations and oxygen breathing preconditioning sequences.
Conclusion: Characterization of the pre-dive gas pocket population opens new possibilities for decompression modelling integrating diver's individual susceptibility and recent history (life style, exercise) to predict his level of VGE during and after decompression.

A NEW INNOVATIVE APPROACH TO DECOMPRESSION SICKNESS MODELING

Michael Strauss[1], Lientra Lu[2], Stuart Miller[1]
[1]Hyperbaric Medicine Program, Memorial Care, Long Beach Medical Center, USA
[2]Research, Tibor Rubin Veterans Affair Medical Center, USA

Background: Decompression sickness (DCS), being a syndrome, generates a multitude of theories why it occurs. These are often expressed as parallel, serial, and/or combination models for inert on/offgassing. We offer a new innovative model based on three tissue types and their on/offgassing characteristics.
Methods: In seeking an explanation (in over 500 patients we have treated for DCS) why and where the symptoms of DCS occur, especially in those divers where the presentation is unexplained, we integrate circulatory system physiology with contemporary on/offgassing knowledge.
Results: A five-liter blood volume must be distributed to a vascular system with over a 20-liter capacity and greater than 95,000 km of end-to-end blood vessels. Consequently, the flow must be precisely regulated by the sympathetic nervous system and chemical mediators. With these considerations, three tissue compartments are apparent. FIRST are the ultrafast tissues with almost instantaneous and/or very rapid on/offgassing. These have continuous, uninterrupted perfusion such as lungs, bloodstream, heart, and brain. SECOND are the tissues such as gut, skin, muscle, and subcutaneous tissues that have a highly regulated blood supply going from almost nil perfusion to 40-fold increases to meet their activity demands. THIRD are the slow tissues such as ligaments, tendons, joint capsules, and adventitial tissues in which on/offgassing are by diffusion of inert gas from their surrounding fluids. When diffusion gradients are exceeded leading to autochthonous bubble formation (a.k.a. Haldanian-type modeling) and/or diffusion/ perfusion are overwhelmed by the gradients, the offgassing, transport, and offloading of inert gas are overwhelmed. These two mechanisms are the causes of DCS in accordance with our Gradient-Perfusion Model.
Conclusions: Whereas other DCS models are designed to prevent DCS, our Gradient-Perfusion Model explains why it occurs and in what locations. This facilitates the management of DCS and provides guidance for return to diving when a DCS “hit” occurs.

BUBBLES IN VASCULAR PLEXUS OF THE SKIN IN CUTANEOUS DECOMPRESSION SICKNESS. REPORT OF CASES.

Eduardo Garcia, Eduardo Garcia-Magaña
Diving Medicine, SSS Recompression Chamber Network, Mexico

Decompression sickness (DCS) is the clinical expression of an inflammatory problem caused by inert gas bubbles that form in various tissues of the body, is essentially an ischemic event caused by gas embolism. The symptoms depend on the anatomical area where the bubbles block circulation and the degree to which the tissue reacts to this ischemia and inflammation. The Cutaneous decompression sickness (CDS) in one of the most frequent presentations of DCS, often taken as a mild problem, because it can occur in isolation and resolve spontaneously; CDS is also observed in divers with severe DCS and is associated with a high frequency of Right to left shunt (RLS). The cause of cutaneous decompression sickness (CDS) has several hypotheses, none of these tested. The precise pathophysiology of CDS remains unverified

Bubbles were observed in the circulation of the superficial and deep vascular plexuses of the skin, as well as in interconnected vessels of the plexuses. The four divers were treated with recompression, after the DCS, the four divers were evaluated with transthoracic ultrasound and bubble test with saline solution, all had positive PFO.

We conclude that CDS pathophysiological mechanism, is related to reactive vascular changes by arterialization of gas bubbles that is usually associated with a RLS with peripheral amplification when the emboli of the bubbles invade supersaturated nitrogen tissues.

PROTEOMICS APPLIED TO DECOMPRESSION SICKNESS

Jacky Lautridou[1], Emmanuel Dugrenot[4], Vianney Pichereau[2], Sébastien Artigaud[2], Luba Matic[3], François Guerrero[4], Ingrid Eftedal[1]
[1]Department of Circulation and Medical Imaging, Norwegian University of Science and Technology, Norway
[2]LEMAR UMR 6539, Ifremer, Université de Bretagne Occidentale, France
[3]Faculty of Medicine and Surgery, University of Malta, Malta
[4]Laboratoire Orphy, Université de Bretagne Occidentale, France

Background: Numerous physiological mechanisms have been highlighted during DCS onset, but none of them seem to be dominant. A broader angle of approach would allow for a better understanding of the interplay between these mechanisms. Three proteomics studies are conducted since 2015. Their aim is to pinpoint variations in the plasma proteome of both rats and divers in relation with both decompression stress and DCS, to identify biomarkers of DCS early development and gain new fundamental knowledge regarding DCS onset. The first two studies showed that proteomics could discriminate between decompression stress and DCS. We found insights of inflammatory processes during DCS. We also found a hypothetical biomarker of DCS development among rats: Transthyretin. The last study will investigate the impact of DCS among divers.
Methods: 18 divers will be separated into 3 groups: Control, Medullar DCS and Vestibular DCS. Their plasma proteome will be investigated by bi-dimensional electrophoresis. Statistical analysis will be performed with a moderated t-test (p0.05, FDR0.1).
Results: No results are yet to describe. The final results of the statistical analysis should be available at the end of July, and thus being presented at the EUBS.
Conclusion: These proteomic studies already gave interesting results, both among rats and humans. This last study will allow us to check if proteomics discriminates enough between decompression stress and DCS to identify biomarkers of DCS early development in humans. It will also allow us to check whether if transthyretin, the biomarker we identified in rats, can also be used for humans with DCS.
Keywords: Decompression sickness, Proteomics, Rats, Humans, Precision Medicine.

Session 5: Diving: Rescue Operations & Accidents

SEDATIVE DIVING CAVE RESCUE IN THAILAND

Natthasak Woracharoensri
Pulmonary and Critical Care Medicine
Underwater Medical Officer
Hyperbaric Medical Center, Somdechpranangchao Sirikit Hospital
Naval Medical Department, Royal Thai Navy.

During 23 June – 12 July 2018, 13 football players trapped deep inside Tham Luang cave in Khun Nam Nang Non Forest Park, Chiang Rai province. This circumstance has never happened anywhere else in the world. Rescue teams were faced tough situation. Due to the difficulties of landscape, awful weather, and time limit, the only possible way to rescue all football players was diving out through the cave. Furthermore, no one in the football team had diving skills and the routes in the cave were extremely dangerous. After preliminary consideration, although no one was used the drug during diving before but this way seemed to be the only possible way to evacuate and rescue all football players from the cave.
The medical teams from many parties worked together. There were only 48 hours of preparation before the operation. The medical teams had to be careful to evacuate all football players with the highest safety process. The teams started from diving plan for all football players and rescuers, using sedative during the evacuation, evacuation process, first aid plan, rescue process within the cave during the evacuation, the preliminary medical care at the field hospital nearby the cave, and the process of transferring the patients to the hospital. It was pleasure all operations were well supported by Public Health officers from both local and international parties. The special operations and collaborations can overcome all difficulties and ultimately successful.
The initial illnesses of football players after the rescue were Hypothermia, Pneumonia, Otitis Media, Acute Malnutrition, Constipation, and Contact Dermatitis and all football players were well taken care by the provincial hospital.
Finally, all 13 football players had returned their homes and communities safely on 18th July 2018 which was the day of the proudest and the most valuable reward for all rescue teams.

A DISABLED SUBMARINE ESCAPE AND RESCUE

Yinnon Matsliah,
Israel Naval Medical Institute

Submarines are considered one of the most sophisticated sea-going vessels produced. Due to their strategic importance, great effort is put in to developing and installing fail-safe and defence mechanisms to prevent a major event. However, the challenges of the sea and the complex nature of submarine operations still do result in serious accidents, with an average of 1-2 incidents every year worldwide. Due to rigorous safety measures and the resourcefulness of submarine crews, the vessel often withstands the initial accident, and at least part of the crew survives within the disabled submarine. The rescue of disabled submarine (DISSUB) survivors from the bottom of the sea is considered an extremely difficult task, and even powerful navies are often unable to cope with all aspects of the operation. After the disaster which befell the Russian submarine K-141 Kursk, NATO established the ISMERLO organisation (International Submarine Escape and Rescue Liaison Office), which coordinates submarine search and rescue operations

Escape from a DISSUB is defined as self-extrication of crew members from the DISSUB to the surface, using special escape suits which allow them to perform the procedure from as deep as 600 feet (183 meters). Rescue of a DISSUB crew requires the use of a submarine rescue vehicle, which connects directly to the submarine's hatch and transfers the rescued crew to the surface. The latter is by far the preferred option.

Survivors of escape or rescue operations may present with complex injuries. They may have suffered trauma from the initial violent accident that caused the submarine to sink, but may also have inhaled toxic gases. Decompression illness may result from the ascent to the surface, or because of an extended wait for rescue in the DISSUB with build-up of the internal pressure. Escape survivors are at a major risk for barotrauma and related arterial gas emboli because of the rapid ascent to the surface, and hypothermia while waiting for final rescue by a surface vessel. A further complication may be radiation exposure, which is relevant in the case of survivors from a nuclear DISSUB.

This lecture will review current methods and problems, and DISSUB exercises conducted in recent years.

ISRAEL DIVING COMMUNITY – A CASE STUDY ON DCI

Adam Konstantinovsky
the Israeli Diving Authority (IDA), Ministry of Culture and Sport, Israel

Background: The Israeli Diving Authority (IDA) has been regulating and monitoring the Local diving industry since the Israeli Diving Law introduced in 1979. In addition to the international diving agencies` standards and procedures, that are been implemented in Israel - all the diving centers and instructors are locally licensed and committed to operate under an additional local set of safety regulations. The Ministry of Health reports all diving accidents to the IDA and each case is documented in our database. It is rare that such extensive data is available within diving and hyperbaric community. In the past 5 years, we collected data on all diving accidents in the country.
Results: The number of all accidents resulting in hospitalization and HBO treatment including DCI cases for 2014-2018 is 58/year, with DCI incidence averaged 50-52/year, or 1 DCI case/10,000 dives, and
In comparison to annual incident reports publications, our performances is at the far low end of the scale. We speculate that the international analysis is based on partial reports and does not represent the full data per country, therefore this puts Israeli community at 2.5 -5 times less accidents, representing a "safer" diving community.
Discussion: Our aim is to challenge the diving and hyperbaric communities to improve diving accident data collection. We need evidence-based knowledge in order to understand the contributing factors that leads to accidents, and subsequently to implement appropriate educational and training tools for divers.
We suggest, that the Israeli dive industry may be viewed as a "Dive Lab", with majority of the accidents are human factors related (non-technical skills), and a centralized data repository exists. Our date analysis can be used as a community case study, regarding the human factors aspect in risk management for divers, and can shed light on the common contributing factors, which leads divers to DCI and diving related injuries.

DECOMPRESSION DISORDERS OF CENTRAL ORIGIN: PATHOGENETIC HYPOTHESIS

Alberto Fiorito, Valentina Spallanzani
Hyperbaric Medicine, CE.M.I.S. Massa (IT), Italy

Background: A fair number of neurological forms of PDD of central origin do not appear to have a suitable pathogenetic explanation. The hypothesis of the presence of a right-left shunt is the most probable cause even if it is not always possible to identify it anatomically. In an attempt to give a rational explanation, the concurrent causes are called into question, i.e. Predisposing conditions not directly connected to the functions exposed to damage. These include dehydration, stress, lack of rest or excess of lipid tissue.
Hypothesis: The recent discovery of a brain lymphatic system opens up new horizons for different pathogenetic hypotheses. The Glimphatic system, so called by Maiken Nedergaard that identified it first, consists of a system of perivascular lymphatic vessels, both arterial and venous, which has the function of draining impurities, toxins and privileging information passage. This draining system is closely related to the circadian production of norepinephrine. During the nocturnal phase the decrease of the production of this hormone allows the expansion of the glimphatic vessels and their functional activation. The hypothesis is that a high level of stress or a reduced amount of sleep can maintain elevated levels of norepinephrine and reduce the drainage capacity of the glimphatic. This would imply a reduction in the rate of discharge of the supersaturated gas and could lead to bubbles production. Furthermore, other concurrent causes could also be justified. An alteration of the oxidative balance or a chronic silent inflammation, linked to obesity, could result in the loss of efficacy of the blood-brain barrier with consequent accumulation of cerebral toxins, which, in turn, would make the glimphatic system less fluid.
Conclusions: Alongside the pathogenetic hypothesis, we suggest that the treatment of these forms of PDD must include the activation of drainage systems and the correction of the inflammatory processes.

NEWLY DESIGNED ALBUMIN-DERIVED PERFLUOROCARBON-BASED NANOCAPSULES IMPROVE CLINICAL SYMPTOMS, MORTALITY AND HISTOLOGICAL FINDINGS IN DECOMPRESSION ILLNESS

Dirk Mayer[1], Francois Guerrero[2], Christelle Goanvec[2], Anton Kreczy[3], Ljubkovic Marko[4], Christian Mayer[5], Michael Kirsch[6], Katja Bettina Ferenz[7]
[1]Department of Gastroenterology, REGIOMED Klinikum Coburg, Germany
[2]Laboratoire ORPHY EA 4324, Université de Bretagne Occidentale, France
[3]Department of Pathology, REGIOMED Klinikum Coburg, Germany
[4]Department of Physiology, University of Split, Croatia
[5]Institute of Physical Chemistry, CENIDE, University Duisburg-Essen, Germany
[6]Institute of Physiological Chemistry, CENIDE, University Duisburg-Essen, University Hospital Essen, Germany
[7]Institute of Physiology, CENIDE, University Duisburg-Essen, University Hospital Essen, Germany

Background: Perfluorocarbons (PFC) offer ideal prerequisites to prevent / treat decompression illness (DCI), but the basic necessity of adding emulsifiers beset with side effects obviated their clinical application. Albumin-derived perfluorocarbon-based nanocapsules (A-aocs) guarantee the gas transport potential of PFC thereby avoiding long organ retention times. A-aocs transport surplus nitrogen to the lung and improve oxygen supply in small vessels. The aim of this study was to assess in a rat model whether A-aocs could protect against DCI.
Methods: Before a simulated air dive in a hyperbaric chamber (Compression: 100 kpa/min up to 1,000 kpa, maximum for 35 min, decompression: 100 kpa/min pausing 5 min at 200 kpa and 160 kpa, 10 min at 130 kpa) forty-five Wistar rats were injected with A-aocs, albumin nanocapsules filled with neutral oil (AON) or albumin solution (AS, each group n=15). Fifteen rats injected with A-aocs stayed at normal pressure. Animals were observed for clinical symptoms of DCI over 45 min after surfacing. Paraffin-embedded sections of liver, spleen and kidney were stained using hematoxylin-eosin for histological evaluation according to a 4 grade rating scale (0=normal to 3=high grade). We sought for accumulation of macrophages (spleen), blood congestion (spleen, kidney, liver) and vacuolization, cell damage and circulatory disorder (liver). Statistical analysis: Chi square test, one way ANOVA with Tukeys´post hoc analysis (5% significance level).
Results: na-aocs prevented significantly DCI-occurrence ($\chi 2 = 10.17628$, dl=4, p=0.006). Histological evaluation of A-aocs-animals compared with AS-ones showed significant differences: in the spleen less blood congestion but a high accumulation of macrophages; in the liver less circulatory disturbances, vacuolisation and cell damage; in the kidney a tendency of a reduced blood influx in the cortico-medullary area.
Conclusion: A-aocs as a preventive application reduced severity and mortality of DCI - associated with less blood congestion in the spleen and less liver cell damage

Session 6: Neuroplasticity and Cognitive Functions

HOW TO EVALUATE BRAIN PERFORMANCE?

Gil Suzin
Sagol Center for Hyperbaric Medicine and Research, Assaf-Harofeh Medical Center, Israel

The human brain has a significant influence on almost any organ in our body with direct effect on our endocrinal, autonomic, motor and cognitive functions. Cognitive functions- which allow us to receive, select, store, transform, develop, and retrieve information- are the most challenging to evaluate. Over the years, many different methods were developed, investigated and implemented in order to evaluate such capacities. In this lecture we review the basic principles of cognitive evaluation and the pros and cons of different methods that are currently available for clinical use and clinical studies.

THE COGNITIVE PROFILE BEFORE AND AFTER HBOT – WHAT CAN WE LEARN FROM THE FIRST 1000 PATIENTS?

Rahav Boussi-Gross; Gil Suzin; Kobi Dagan; Ido Mey-Raz; Shiran Mugrabi; Doron Arbiv; Liron Cohen; Shai Efrati
Sagol Center for Hyperbaric Medicine and Research at Assaf Harofeh Medical Center. Sackler School of Medicine and Sagol School of Neuroscience, Tel-Aviv University, Israel

In recent years there is growing evidence regarding the neuroplasticity effect of Hyperbaric Oxygen treatment (HBOT) and its potential use for cognitive rehabilitation and enhancement. The use of computerized cognitive evaluation enables data gathering and objective analysis and comparison of different patients' populations. The neurocognitive data gathered from the first 1,000 patients who went through computerized cognitive assessments before and after HBOT enables us to do cross-populations comparisons. The data includes different cognitive domains (memory, information processing speed, attention, executive function, etc.), all objectively evaluated and analyzed by NeuroTrax testing battery. In this large group of patients, HBOT induced significant improvements ($p<0.005$) in the evaluated cognitive domains. The cognitive modules who benefitted the most were of memory, attention, and information processing speed. In addition, a distinct and unique cognitive profile for different patients' populations, according to diagnosis, was recognized. These findings, together with the brain imaging data, gathered in this large scale population, resemble the significant improvement in brain functionality after HBOT. The use of objective computerized cognitive testing battery enables us to gain better understanding of the cognitive profile of different patients' populations and characterize the patients who can benefit most from HBOT.

MULTIFACETED MITIGATING EFFECTS OF HYPERBARIC OXYGEN THERAPY IN ALZHEIMER'S DISEASE MOUSE MODELS

Ashery Uri
School of Neurobiology, Biochemistry and Biophysics, the George S. Wise Faculty of Life sciences, and Sagol School of Neuroscience, Tel Aviv University, Israel.

Alzheimer's disease (AD) is the most common form of dementia in the elderly. In addition to its main pathological signatures like accumulation of extracellular amyloid plaques and intracellular neurofibrillary tangles, AD is associated with hypoxia, neuroinflammation and diminished blood flow. Hyperbaric Oxygen Therapy (HBOT), the medical administration of 100% oxygen at environmental pressures greater than 1 atmosphere absolute (ATA), has been used successfully in the treatment of other neurological conditions. Here, we investigated the effects of HBOT on two different mouse models of AD pathology, the 3xTg-AD and 5xFAD mouse models.
HBOT attenuated neuroinflammatory processes by reducing astrogliosis, microgliosis, and the secretion of proinflammatory cytokines (IL-1β and TNFα) and increasing expression of scavenger receptor A, arginase1, and anti inflammatory cytokines (IL-4 and IL-10). Moreover, HBOT reduced hypoxia, amyloid burden, and tau phosphorylation in 3xTg and 5XFAD mice and ameliorated their behavioral deficits. In addition, HBOT enhanced neurogenesis and survival of newborn neurons in 5XFAD mice. Two-photon imaging of mice before and after HBOT or normobaric conditions revealed that HBOT reduced the volume of existing plaques and reduced the appearance of newly formed plaques. Moreover, 2-photon analysis of blood vessels diameter and flow uncovered that HBOT alleviated the reduction in blood vessels diameter in 5XFAD mice and therefore contributed to increment in blood flux.
Taken together, these findings suggest HBOT has multifaceted effects that reduce AD pathologies. Given that HBOT is used in the clinic to treat various indications, including neurological conditions, and is considered a safe and tolerable treatment, these results suggest HBOT as a novel therapeutic intervention for AD.

THE EFFECT OF HYPERBARIC OXYGEN ON THE HUMAN BRAIN MICROSTRUCTURE: NEW INSIGHTS FROM MRI-DTI STUDIES

Merav Catalogna, PhD[1], Efrat Sasson,PhD[1], Amir Hadanny MD[1], Sigal Tal, MD[2], and Efrati Shai, MD[1]
[1]Sagol Center for Hyperbaric Medicine and Research, Assaf Harofeh Medical Center, Zerifin, Israel
[2]Radiology Department, Assaf Harofeh Medical Center, Zerifin, Israel

Diffusion tensor imaging (DTI) is an advanced MRI imaging technique for characterizing microstructural changes by examining water diffusion in different tissues. In particular, DTI can evaluate the integrity and directionality of white matter tracts, and abnormalities in DTI measures were found in patients with cognitive impairments but also in normal aging. Animal studies suggest that hyperbaric oxygen therapy (HBOT) is associated with increased cerebral blood flow, improved mitochondrial and cellular metabolism, and with stem cells recruitment and mobilization. Therefore, HBOT induced brain microstructure recovery depicted by the DTI imaging markers may demonstrate improved axonal coherence, myelination and neuroplasticity. This lecture reviews the principles and basic concepts of DTI imaging, and highlight new insights into the effect of HBOT on brain microstructure in patients suffering from chronic brain damage, fibromyalgia patients with a history of brain injury or emotional trauma, and aging cognitive decline.

PERFUSION MRI- BASICS AND ITS CLINICAL USE FOR EVALUATING THE EFFECT OF HYPERBARIC OXYGEN THERAPY ON BRAIN PERFUSION

Efrat Sasson,PhD[1] Amir Hadanny MD[1], Merav Catalunia, PhD[1], Sigal Tal[2], MD, Efrati Shai[1] MD,
[1]Sagol Center for Hyperbaric Medicine and Research, Assaf Harofeh Medical Center, Zerifin, Israel
[2]Radiology Department, Assaf Harofeh Medical Center, Zerifin, Israel

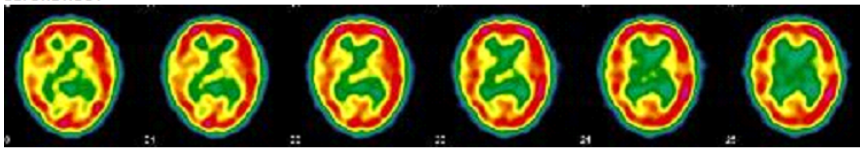
Dynamic contrast enhancement (DSC, perfusion) allows the evaluation of cerebral blood flow (CBF) and cerebral blood volume (CBV). Thus, perfusion MRI can serve as an important sensitive tool enabling us to get better understanding on the microcirculation and perfusion to specific brain regions. It is already well established that Hyperbaric Oxygen Therapy (HBOT) can induce angiogenesis and improve tissue perfusion to peripheral wounds, where the perfusion can be evaluated directly. In animal models, after brain resection, it was demonstrated that HBOT can induce angiogensis in the damage brain tissue. By using perfusion MRI, we can learn about the effect of HBOT on brain perfusion in human. The DSC MRI allows the evaluation of perfusion also in other organs such as heart and kidneys.
In this lecture, we will review the basics of perfusion MRI with special focus on the core aspects that the physicians need to know. We will also present and discuss the new knowledge gained about the effect of HBOT on brain perfusion in patients suffering from chronic brain damage due to TBI, stroke and age related cognitive decline.

OXYGENATION IN AUTISM SPECTRUM DISORDERS

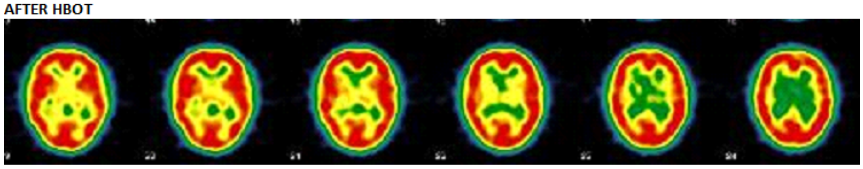
Necip Cem Kinaci
Nuclear Medicine, Autism Medical Institute, Turkey

Background: Impaired detoxification, depletion of antioxidants, oxidative stress, mineral deficiency, mitochondrial dysfunction, gastrointestinal dysfunction, chronic BBB dysfunction and immune system dysregulation are biochemical aftermath in Autism Spectrum Disorders (ASD). Many cases of ASD today are secondary to cases of gut-brain inflamation.
Methods: We performed a retrospective review in search of 127 children with ASD (99 males, 28 females) between the ages 3-12, who had done basal and control Brain Perfusion $Tc99^{m}$ HMPAO Single-Photon Emission Computed Tomography (SPECT) Scans, between the years 2004 - 2011. We also reviewed Magnetic Resonance Imaging (MRI) results of these children. They were all applied 50 sessions Hyperbaric Oxygenation Theraphy (HBOT) for each patient at 1.5 ATA for 60 minutes once a day. We compared the results of SPECT before and after HBOT.
Results/Discussion: Before HBOT, 125 of the patients were revealed focal areas of decreased perfusion in temporal, in 111 patients at frontal and in 77 patients at other areas of the brain. By contrast all patients had normal MRI findings. We determined that, after HBOT, 103 of 127 (81.10%) patient's SPECT findings were improved. **(Picture 1)**

BEFORE HBOT



AFTER HBOT



Perfusion changes were 76.80% (96 of 125) at temporal, 78.37% (87 of 111) at frontal and 72.72% (56 of 77) at the other areas and changes in total patients were 81.10% (103 of 127). **(Table 1)**

AREA	NUMBER OF PATIENTS	INCREASED PERFUSION AFTER HBOT	NOT CHANGED	% IMPROVEMENT SEEN IN TOTAL
TOTAL NUMBER OF PATIENTS WITH LESSIONS	127	103	24	81.10
NUMBER OF PATIENTS WITH LESSIONS AT TEMPORAL AREAS	125	96	19	76.80
NUMBER OF PATIENTS WITH LESSIONS AT FRONTAL AREAS	111	87	14	78.37
NUMBER OF PATIENTS WITH LESSIONS AT OTHER AREAS	77	56	16	72.72

Conclusions: SPECT scanning can identify between recoverable brain cells (referred to as sleeping cells, idling neurons, or the ischemic penumbra). HBOT which now being used for children with ASD is to address the neuroinflammatory component of the disorder. There is emerging evidence of chronic blood-brain barrier dysfunction in these children. The use of high dosage oxygen is based on the latest research into its role in the control of inflammation. After HBOT, extensive perfusion improvements involving the brain were found in this study.

Session 7: Sport Physiology and Performance Enhancement

MITOCHONDRIAL PHYSIOLOGY FROM HYPOBARIC TO HYPEROXIC CONDITIONS

Erich Gnaiger[1,2]
[1]Daniel Swarovski Research Laboratory, Department of Visceral, Transplant and Thoracic Surgery, Medical University of Innsbruck, Austria
[2]Oroboros Insbruments, Innsbruck, Austria – erich.gnaiger@oroboros.at

Background: Intracellular oxygen pressure, $p_{O2(ce)}$, is close to 2 kPa (15 mmHg; 10 % of air-level normoxia) in various tissues under normoxia, controlled by: (i) environmental or artificial oxygen supply, (ii) oxygen transport from lung to mitochondria, and (iii) metabolic oxygen demand and the kinetics of mitochondrial oxygen consumption. What can we learn from investigations of cellular and mitochondrial respiration and hydrogen peroxide production under the control of extracellular or mitochondrial oxygen pressure?
Methods: Isolated mitochondria are unintentionally exposed to effectively hyperoxic conditions in most *in vitro* studies. High-resolution respirometry (O2k, Oroboros Instruments) solved the technical difficulties of measuring accurately mitochondrial respiration as a hyperbolic function of p_{O2} in the physiological oxygen range. The O2k-FluoRespirometer allows simultaneous measurement of H_2O_2 production using Amplex Ultrared.
Results: The p_{50} is the p_{O2} at 50 % maximal oxygen flux, which varies in isolated mitochondria and small cells from 0.01 to 0.1 kPa as a function of (i) enzyme kinetic O_2 affinity of cytochrome *c* oxidase (CIV), (ii) apparent excess capacity of CIV in relation to electron transfer capacity of respiratory complexes and redox states upstream of CIV, (iii) activation of mitochondrial pathways with convergent electron transfer into the Coenzyme Q-junction, and (iv) coupling control of respiration from resting to active states of oxidative phosphorylation. Competitive inhibitors of CIV, particularly nitric oxide and hydrogen sulphide, increase the p_{50} to much higher values. In contrast to respiration, mitochondrial hydrogen peroxide production increases linearly with p_{O2} from hypoxia to hyperoxia.
Conclusions: Current models of the control of ergometric V_{O2max} appreciate the functional role of variable mitochondrial p_{50} in the face of oxygen limitation under normoxia. Feedback control between p_{50} and intracellular p_{O2} is a mechanism explaining the effect of hyperoxia on athletic performance. The oxygen conformance of hydrogen peroxide production provides a strong argument to avoid artificial hyperoxia in mitochondrial *in vitro* studies and points to a role of mitochondria in intracellular oxygen sensing.

THE PHYSIOLOGY OF AEROBIC EXERCISE

Eldad Yaakobi
Sagol Center for Hyperbaric Medicine and Research, Assaf-Harofeh Medical Center, Israel

Whole body movement and physical activity facilitate an immediate increase of energy consumption at the contracting muscles and induce a shift in the physiological hemostasis. Since Krogh's early discovery (1913) on crucial role of oxygen delivery to the contracting muscles for exercise performance, the different aspects oxygen delivery have become a target for many investigator's. The ability to maintain whole body movement for prolong period of time is limited mainly by three physiological factors; maximal oxygen uptake, the ability to maintain work at a high percentage of maximal oxygen uptake and the efficiency of the moving joints. Physiological adaptations to exercise such as increased cardiac output, increased blood oxygen carrying capacity, increased muscle capillarization and increased mitochondrial functionality have a key role of regulating those physiological limitations.

Cardiopulmonary Exercise Testing (CPX), non as "VO2 max test", allows a non-invasive monitoring of pulmonary ventilation, gas exchange between the lungs to the blood, cardiac output, and gas exchange between the capillary blood to the working muscles while exercising. CPX is the gold standard testing for aerobic capacity and physiological limitations among healthy and unhealthy subjects. Test results indicate the cardiac, cardiovascular, ventilatory, metabolic and muscular response to exercise. CPX standard measures include the subject's maximal aerobic capacity and ventilatory threshold, which represent the maximal oxygen uptake and the ability to maintain work at a high percentage of maximal oxygen uptake, respectively.

This lecture reviews aerobic exercise physiology through muscles energy transfer pathways, key physiological adaptations to aerobic exercise, particularly mitochondrial adaptation, and the guidelines for interpretation of CPX.

THE EFFECT OF HYPERBARIC OXYGEN THERAPY ON ELITE ATHLETES– RANDOMIZED CONTROLLED TRIAL – INTERIM ANALYSIS

Amir Hadanny
Sagol Center for Hyperbaric Medicine and Research, Assaf-Harofeh Medical Center, Israel.
Sackler School of Medicine and Sagol School of Neuroscience, Tel-Aviv University, Israel.

In recent years, several options for effective physical endurance enhancement were suggested including blood doping, erythropoietin. The common denominator is increasing the available blood oxygen content. However, due to their low safety margin, these interventions were banned.
Hyperbaric oxygen therapy (HBOT) may serve as an alternative option by increasing the plasma dissolved oxygen content. Thus, performing aerobic exercise while breathing oxygen in a hyperbaric environment can increase physical performance. Recent studies have shown contradictory results in endurance following a single HBOT session. Another recent trial showed mild positive effect of aerobic training during five HBOT sessions. However, the effects of a multi HBOT sessions have never been evaluated on the healthy athletes.
The aim of this study was to evaluate the physiologic effects of HBOT on athletes in a randomized prospective clinical trial.
The study was done in Assaf Harofeh Medical Center, from 2018 to 2019, on 60 healthy athelets,18-50 years old. The volunteers were randomized to 2 groups: the treatment group and the sham treatment group. Both groups were evaluated at baseline, and after 2 months of either HBOT daily sessions (40 daily sessions, 60 min of 100% oxygen at 2 ATA without air breaks) or sham daily sessions (40 daily sessions, 60 min of 21% oxygen at 1.01 ATA without air breaks). Comprehensive evaluations included cardiopulmonary exercise tests (CPET), mitochondria respiratory function, pulmonary function, cognitive assessments and brain MRI.
In this lecture, we will present the results of the study and discuss its implications.

THE EFFECT OF HYPERBARIC OXYGEN THERAPY ON MITOCHONDRIAL FUNCTION

Yafit Hachmo[1], Amir Hadanny[2], Ramzia Abu Hamad[1], Marina Sova[1], Rina Zaa'rura[1], Moshe Stark[1], Yuval Yegudayev[1], Nir Polak[2], Daniela Rozali[2], Erez Lang[2], Shachar Finci[2], Shai Efrati[1,2]
[1]Research and Development Unit,Nephrology Division, Assaf Harofeh Medical Center affiliated to Sackler Faculty of Medicine, Tel Aviv University, Israel
[2]Sagol Center for Hyperbaric Medicine and Research, Assaf Harofeh Medical Center affilated to Sackler School of Medicine, Tel-Aviv University., Israel

Background: Mitochondrial respiration plays a central role in cellular energy metabolism by coupling oxygen consumption to ATP production. Since mitochondria function is directly influenced by the partial pressure of dissolved oxygen, repeated fluctuation in oxygen generated by hyperbaric oxygen therapy (HBOT) may influence mitochondrial function. While previous pre-clinical studies showed that HBOT improves neuronal mitochondrial, there is no clinical data on the direct effect of HBOT on mitochondrial activity. The aim of the study was to evaluate for the first time in human the effects of HBOT on muscle mitochondrial function of athletes.
Methods: Prospective, randomize control trial including 40 healthy athletes randomly divided into HBOT and placebo group. The treatment group had 40 daily hyperbaric sessions, 5 days per week. Each HBOT session included 1-hour exposure to 100% oxygen at 2 ATA. The control group were exposed to 1-hour session of normal air, with increase pressure to 1.2 ATA at the first 5 minutes. Muscle biopsies were obtained from the Gluteus Maximus muscle at two time points: at baseline and following completion of HBOT protocol. Mitochondrial functions were assessed in small (1.5-3 mg) permeabilized muscle fibers by Oroboros O2k system for high-resolution respirometry for mitochondria and cell research with multiple substrate-uncoupler-inhibitor titration protocol.
Results: Compared to the placebo group there was a significant increase in the percentage fold change of complex I (CI)-linked respiration (p0.05) of the HBOT group which reflects the increase in mitochondrial mass. Furthermore, analysis of the qualitative changes of mitochondria revealed a significant increment in the percentage fold change of the coupling control ratio (CIP/CI+IIE p0.05) in the HBOT group.
Conclusions: HBOT induce both qualitative and quantitative mitochondrial changes by increasing the relative capacity for NADH oxidation and the mass-specific complex I-linked respiratory capacity.

INFLUENCE OF ELEVATED OXYGEN FRACTION ON BREATHING GAS CONSUMPTION DURING PHYSICAL WORKLOAD IN SHALLOW-WATER SUBMERSION

Jochen Schipke[1], Fabian Moeller[2], Elena Jacobi[2], Thomas Muth[3], Uwe Hoffman[2] [1]Research Group Experimental Surgery, University Hospital Düsseldorf, Germany [2]Department of Exercise Physiology, German Sport University Cologne, Germany [3]Institut für Arbeits-, Sozial- und Umweltmedizin, Heinrich Heine Universität, Germany

Background: Using oxygen enriched air (EAN) as breathing gas has become increasingly popular in recreational diving for the benefits of increased oxygen partial pressure (po_2) and lowered nitrogen partial pressure (pn_2). Whereas many beneficial and detrimental effects of altered gas fractions are known, little is known about the influence of po_2 on GC in underwater settings. Aim**:** As safety is highly important when acting or working underwater this study investigated a possible relationship between breathing gas O_2-fraction and gas consumption (GC) during physical exercise (PE).
Method: Eleven divers (age: 25.8±4.1; mean±SD; 6 females) participated in this prospective double-blind, cross-over study. Participants performed two dives each, swim-diving with increased speed until exhaustion while using either normal air (AIR) or EAN40 as breathing gas. Heart rate (HR) and GC were measured throughout the dive, lactate samples were taken once before and five times directly after the dive.
Results: Rest HR was 96±4 beats•min^{-1} and increased to 170±4 beats min^{-1} for 0.8 m•s^{-1} swim-diving speed. Anovas revealed a significantly lower GC with EAN40 (35±2 l•min^{-1}; 62±7 l•min^{-1}) compared to AIR (44±4 l•min^{-1} and 73±8 l•min^{-1}) only for the velocities 0.6 m•s^{-1} and 0.8 m•s^{-1} (both $p \leq 0.05$).
Discussion/Conclusion: These findings suggest that GC is influenced by the oxygen fraction in breathing gas when swim-diving in shallow-water with high PE (velocity ≥0.6 m•s^{-1}). The findings might become important for dives with limited gas supply and involuntary or planned high physical exercise.

HYPEROXIA AND THE CARDIOVASCULAR SYSTEM: DATA FROM THE PRESSURE CHAMBER

Jochen Schipke[1], Martin Hoffmanns[2], Thomas Muth[3], Sven Dreyer[4]
[1]Forschungsgruppe Experimentelle Chirurgie, Heinrich Heine Universität, Germany [2]Klinik f. Unfallchirurgie und Handchirurgie, Universitäts Klinikum Düsseldorf, Germany [3]Institut für Arbeits-, Sozial- und Umweltmedizin, Heinrich Heine Universität, Germany [4]Medical Process Organization, Heinrich Heine University, Germany

Background: Hyperoxia directly stimulates the parasympathetic nervous system inducing bradycardia. Alternatively does hyperoxia increase the vascular resistance thereby increasing blood pressure subsequently inducing bradycardia. Aim: Help resolving the controversy by investigating the temporal relation between oxygen (O_2) administration and heart rate (HR)
Methods: Transcutaneous partial O_2 pressure ($tcpo_2$; chest wall; oxymetry) and ecgs were recorded in 13 subjects without cardiovascular problems undergoing HBO treatment (HBOT). The problem wound scheme was used as experimental model. To account for respiratory effects, respiration rate during both air and O_2 intervals was assessed in a blinded manner.
Results: Transcutaneous po_2 increased from 101±46 to 1089±178mmhg (mean±SD) and heart rate decreased from 68±10 to 60±8/min. Calculation of half libe values gave a $tauo_2$ of 5.4±2.1mmhg/s and a tauhr of 0.45±0.19 /60s. Respiration rate was maintained while breathing air or 100% O_2.
Discussion/Conclusion: HBOT is supposed to improve the arterial O_2 concentration. The increase in our study was with 5.5mmhg ($tcpo_2$) per second rather fast. HR changes, on the other hand, were assessed from the ECG readings.. Other studies report on increases in the systemic vascular resistance that in turn induces increases in arterial blood pressure. With baroreceptors intact, HR will be decreased. Some studies supporting a direct vagus-stimulating effect of hyperbaric O_2 are based on analysing the heart rate variability although some of the HRV indices of vagal activity decrease in parallel with HR. Moreover, response times of the autonomic nervous system are usually considered as being short. We conclude that hyperoxia reduces endothelial derived vasodilators causing vasoconstriction, finally leading to a decreased HR. This explanation would be in line with a rather slow process as exhibited by a lengthy half live of 0.45/min.

Session 8: Pulmonary and CNS Oxygen Toxicity (I)

INTRODUCTION TO PULMONARY AND CNS OXYGEN TOXICITY

Yehuda Arieli
Israel Naval Medical Institute

Exposure to hyperbaric oxygen (HBO) is routine practice in hyperbaric medicine and when diving with closed-circuit apparatus. However, the oxygen without which most life forms are unable to survive may sometimes act as a double-edged sword, also resulting in severe damage and even death. High partial pressures of O_2 (ppO_2) may be toxic to most living creatures. We know of two variants of oxygen toxicity (OT) in mammals, pulmonary (P-OT) and that of the central nervous system (CNS-OT). These two forms of oxygen toxicity differ with regard to their symptoms, time constants, and underlying mechanisms.
Irregular breathing, pain, cough, shortness of breath, tracheobronchitis, and acute respiratory distress syndrome (ARDS) are some of the most common symptoms of P-OT. Symptoms can appear within 24 h of the initiation of hyperoxic exposure. Pulmonary "oxygen toxicity occurs when the partial pressure of alveolar O_2 (P_AO_2) exceeds that which is breathed under normal conditions" (Mach et al. 2011), leading to extensive production of reactive oxygen species (ROS). As a result, a disruptive cascade destroys the alveolar walls as well as the adjacent capillary walls, causing mild-to-severe lung injury.
CNS-OT symptoms, on the other hand, can appear within a few minutes of exposure to HBO. These include among others headache, tunnel vision, twitching of limbs and muscles, grand-mal convulsions, and loss of consciousness. When this occurs underwater, it may end in loss of life. As in P-OT, extensive ROS production is one of the underlying mechanisms. However, among further manifestations of CNS-OT we find blood-brain barrier (BBB) disruption, disturbance of mitochondrial function, and impaired performance of NMDA receptors. In contrast to our understanding of P-OT, the complete picture of the mechanisms underlying CNS-OT remains unclear and elusive.

AVAILABLE OXYGEN TOXICITY MODELS VS. AVAILABLE OXYGEN TOXICITY DATA

Barbara Shykoff
Naval Aerospace Medical Research Laboratory (NAMRL), Naval Medical Research Unit Dayton (NAMRU-D), Wright Patterson Air Force Base (WPAFB) Ohio , USA

Five separate categories of pulmonary oxygen toxicity models, all of which predict fractional changes in vital capacity (%ΔVC) during and after hyperbaric oxygen exposure, were examined in 2007. Four central nervous system (CNS) oxygen toxicity models, all of which estimate all-or-nothing risk of a toxic event, were found in the literature in 2013. All models in both categories were based on PO_2 and exposure time. The models were tested against their calibration sets plus all other diving data available. A striking finding was that models did not translate well to data from conditions different from their calibration set. CNS oxygen toxicity risk appeared to decrease after 1970. Models based on exposures to high PO_2 greatly overestimated risk at lower PO_2. Different Navies recorded different incidence of CNS oxygen toxicity from shallow oxygen dives. Pulmonary models ignored large individual variation. Perhaps basic research into oxygen toxicity explains the difficulty. Pulmonary oxygen toxicity appears to be a set of signs and symptoms with multiple causes, not a unique pathological process, and oxygen toxicity risk depends on additional factors, for example, nitrogen partial pressure and nitric oxide precursors, in addition to PO_2 and time. Current models are only a first level explanation

CALCULATED RISK OF PULMONARY AND CENTRAL NERVOUS SYSTEM OXYGEN TOXICITY: A TOXICITY INDEX DERIVED FROM THE POWER EQUATION

Ran Arieli
The Israel Naval Medical Institute, Haifa, Israel

Introduction

The risk of oxygen toxicity has become a prominent issue due to the increasingly widespread administration of hyperbaric oxygen (HBO) therapy, as well as the expansion of diving techniques to include oxygen-enriched gas mixtures and technical diving. However, current methods used to calculate the cumulative risk of oxygen toxicity during an HBO exposure, i.e., the unit pulmonary toxic dose (UPTD) concept, and the safe boundaries for central nervous system oxygen toxicity (CNS-OT), are based on a simple linear relationship with PO_2 and are not supported by recent data.

Oxygen Toxicity Index

The power equation: ***Toxicity Index*** = $t^2 \times PO_2^c$, where t represents time, was derived from the chemical reactions producing ROS or RNS. It was shown to have a good predictive capability, PO_2 with a power "c" of 6.8 for CNS-OT and 4.57 for pulmonary oxygen toxicity (POT). The ***POT index*** predicts reduced pulmonary functions, incidence of POT and POT in saturation diving. The ***CNS-OT index*** predicts CNS-OT and produce new exposure limits in between the various suggested limits. The ***POT index*** (PO_2 in atmospheres absolute [ATA], time in h) should not exceed 250. The ***CNS-OT index*** (PO_2 in ATA, time in min) should not exceed 26,108 for a 1% risk.

DETECTING PULMONARY OXYGEN TOXICTY; ONE BREATH IS STILL THE FUTURE.

Pieter-Jan van Ooij
Dive Medical Center Royal Netherlands Navy, Den Helder, the Netherlands

The decrease in vital capacity, and its derived marker the unit of pulmonary toxicity dosage (UPTD), are considered as the gold standard markers regarding the development of pulmonary oxygen toxicity (POT). At the time of its introduction, it was novel, based on a combination of available techniques and accuracy, speed and convenience. However, both the VC and the UTPD concept have some major limitations. As science advances, new techniques become available that could overcome these limitations and are more suitable in detecting the (early) development of POT.
In this presentation I will talk about the limitation of the VC and UPTD concept. Furthermore, I present some of the novel techniques as possible candidates for a new gold standard regarding the detection of POT. Finally, based on the results from our research department, I will demonstrate their usability.

IMAGING THE OF HYPERBARIC OXYGEN ON SINGLE NEURON AND OVERALL BEHAVIOUR IN A NOVEL, LIVE ANIMAL MODEL

Rabinovitz Ricarina[1,2], Arieli Yehuda.[2], Monsongeo Adir.[1], Lerer-Goldshtein Tali.[1], Appelbaum Lior.[1]
1The Faculty of Life Sciences and The Multidisciplinary Brain Research Center, Bar Ilan University
2Israel Naval Medical Institute, Haifa, Israel

Introduction: Breathing a high ppo_2 can lead to the development of Central Nervous System Oxygen Toxicity (CNS-OT), which may culminate in the onset of seizures similar to tonic-clonic epilepsy, a potentially fatal event underwater. The hyperbaric chamber presents a unique yet major limitation for researchers, who are unable to study the brain during hyperbaric exposures. Consequently, never in the past has the brain of an active, intact animal been directly observed during CNS-OT. In the present study, we set a precedent by devising an innovative toolset to solve this problem, and succeeded in visualising the active vertebrate brain directly and continuously during exposure inside the hyperbaric chamber.
Methods: To overcome the limitations of the mammal model, i.e., a highly complex brain sited within an opaque skull, we developed a unique, small pressure chamber that allows imaging of neuronal activity in the live transparent zebrafish.
Results: Changes were noted in the transcription levels of several genetic markers of brain activity (c-fos, phospho-ERK), oxidative stress (hif1α, hmox1), and seizure occurrence (mmp9, cox2). Using video-tracking, larvae were found to exhibit seizures, bouts of irregular mobility, and abnormal posture between 8 and 23 min into exposure to hyperbaric oxygen (HBO) at a pressure of at least 6 atmospheres absolute (ATA). Continuous imaging of genetically encoded calcium indicators (gcamp) in the brain of live transgenic zebrafish larvae during CNS-OT at 6 ATA disclosed seizure-like changes in overall brain activity, as well as in specific regions (cerebellum, hindbrain), that concur well with standard models of epilepsy. The above-mentioned changes were induced exclusively by HBO, and not by hyperoxia alone or by the rise in ambient pressure (normoxia at 6 ATA).

ALTERATIONS OF THE BLOOD-BRAIN BARRIER WITH HYPERBARIC OXYGEN AND RAMIFICATIONS FOR TRAUMATIC BRAIN INJURIES

Michael Strauss[1], Israel Chambi[2], Barry Ceverha[2]
[1]Hyperbaric Medicine Program, Memorial Care, Long Beach Medical Center, USA
[2]Surgery, Division of Neurosurgery, Memorial Care, Long Beach Medical Center, USA

Introduction: The blood-brain barrier (BBB) prevents noxious substances from entering the cerebral spinal fluid, but it also acts as a barrier for medicinals to enter the central nervous system (CNS) tissues. It also may impair waste products of metabolism and injury from exiting this compartment. A study in rabbits which we performed demonstrated that a single hyperbaric oxygen (HBO) exposure alters the blood-brain barrier. This may have important ramifications for the acute management of traumatic brain injuries.
Methods: We performed a prototype study with tryptophan blue injected into the carotid arteries of six rabbits and initiated single hyperbaric oxygen (HBO) exposures at 2 ATA for one hour. Six rabbits were used as controls and pressured in air at 1.03 ATA for one hour.
Results: Dense staining of brains was observed with the HBO study group as compared to the controls. The effects were observed whether the HBO exposures were given one hour before the injections of tryptophan blue or up to four hours after them.
Conclusions: A single HBO exposure after injecting tryptophan blue into the carotid arteries of rabbits visually altered their bbbs. This has ramifications for both getting agents into the CNS tissues such as antibiotics and chemotherapeutic agents as well as allowing exiting of waste products of metabolism from them. With anticipated increased metabolic waste products in the CNS after traumatic injuries, HBO provided in the "golden period" may significantly reduce their consequences. Further research of this potentially valuable use of HBO for CNS injuries is warranted based on this information.

Session 9: Pulmonary and CNS Oxygen Toxicity (II)

CELLULAR ADAPTATION TO RERPETITIVE HYPERBARIC HYPEROXIA IN CLOSED-CIRCUIT DIVING

A. Koch, S. Klapa, B. Grams, W. Kähler
Naval Insitute of Maritime Medicine - Departmental Research Institute and Christian-Albrechts-University Kiel, Germany

Introduction: Repetitive exposure of man to hyperbaric hyperoxia has shown not only cellular DNA-damaging effects, but also adaptive effects to oxygen toxicity in peripheral blood mononuclear cells (PBMCs), visualized by alkaline Comet Assay. It is open, if adaptation to hyperoxia is a permanent or transient process, and if, how long adaptation lasts after cessation of exposures.
Method: We investigated closed-circuit oxygen combat swimmers after a three months period of 85 challenging oxygen dives, lasting between one to three hours each. Blood of eight divers was collected directly after the last dive and then every seven days the following five weeks. The freshly isolated PBMCs from the divers were exposed ex-vivo to a hyperoxic stress-test at 400kPa pO_2 for 4 hours in an experimental pressure chamber. DNA double strand breaks were studied with the Comet Assay and analyzed by yes/no scoring and tail moment.
Results: Directly after the last oxygen dive, PBMCs of combat swimmers showed a significant less amount of PBMCs with DNA fragmentation compared to non-diving controls. This adaptive effect decreased over the next five weeks, and DNA-fragmentation after ex-vivo oxygen stress-test nearly reaching the level of the controls after this time.
Conclusions: The study shows that adaptation against oxidative stress is a non-permanent process, which completely regresses without another oxygen stimulus within five to six weeks.

CNS OXYGEN TOXICITY IN CLOSED CIRCUIT HUMAN DIVING

Yoav Yanir

Closed-circuit rebreathers with 100% oxygen are frequently used with combat diving. Exposure to high partial pressures of oxygen could cause intoxication to the central nervous system (CNS). Oxygen toxicity at dry environment has long been studied whereas studies regarding in-water oxygen toxicity are sparse. Exposure to a PO2 above 1.6 ATA can cause CNS toxicity, leading to a wide range of neurologic symptoms and given the wet environment may lead to drowning and death. Several co-factors has been described for attributing to oxygen toxicity: High level of carbon dioxide, intense exercise, low core temperature, personal susceptibility, sleep deprivation and even shifting the circadian cycle. Prediction models has been presented to prevent oxygen toxicity using algorithm with recovery time to neutralize oxygen stress. To-date CNS oxygen toxicity still presents a potentially fatal risk for Closed-circuit rebreather users.

OXYGEN TOXICITY DURING ROUTINE HBO TREATMENTS

Zemer Wang
Sagol Center for Hyperbaric Medicine and Research, Assaf-Harofeh Medical Center, Israel.

Oxygen seizures induced by Hyperoxia treatments is a rare but well-known side effect of Hyperbaric Oxygenation Treatment (HBOT). First evidence of oxygen toxicity and the occurrence of seizures during HBOT was published during the early 1970's (possibly even before). In the early 70's the incidence of HBOT induced seizures was as high as one case for every 500 treatments.

Along the years there was a growing awareness of precipitating factors that can increase the risk of CNS toxicity including the use of medications such as steroids, predisposing physical conditions such as elevated body temperature, acid base imbalance and more. Taking these factors into consideration, safety was improved and in the late 1970's the reported incidence of HBOT induced seizures declined to one case for 4690 treatments (Hart,Straus,Pierse, 1977).

In 2003, Hampson and Atic reviewed retrospectively approximately 10,000 treatments of routine non-emergency patients at their centre in Seattle and found and incidence of one HBOT associated seizure for every 3,388 treatments. All patients were using hoods (head tents) as means of oxygen breathing apparatus during HBOT. Is was suggested that the accumulation of CO2 in the hood could be an explanation for heightened oxygen sensitivity. Two other papers discussed a high rate of seizures, Welslau 1996 (1:6,704) and Plafki 2000 (1:2,844).

In recent years, all the hyperbaric centres in Israel and numerous centres globally, adopted 2ATA as their basic treatment depth for HBOT. Furthermore, more frequent air breaks have been set (5 minute air break every 20-30 minutes).

In 2016, Hadanny et al. published a retrospective analysis of 62,614 treatment sessions preformed at the Segol centre in Israel. An overall incidence of seven seizures were recorded (incidence of 1:8,945). Furthermore, only one of these patients had a clear oxygen toxicity induced seizure reducing the risk to 1:62,614.

In this talk, we will review the past, and present the published data concerning oxygen induce CNS toxicity. Additionally, a list of recommendations for patient selection and monitoring will be presented.

BLOOD GLUCOSE LEVELS AND HYPERBARIC PRESSURE IN SOD2 ENZYME KNOCKDOWN MICE

Mirit Eynan[1,2], Ricarina Rabinovitz[1,2]
[1]Research, Israel Naval Medical Institute, Israel
[2]Research, Israel Naval Medical Institute, Israel

Introduction: In our previous studies in rats, we found a linear correlation between the partial pressure of oxygen at 4–6 atmospheres absolute (ATA), blood glucose levels (BGL), and changes in the membrane potential of the mitochondria. In transgenic mice, knockdown of the antioxidant enzyme Mn-superoxide dismutase (SOD2) resulted in an increase in oxidative stress. We hypothesized that plasma glucose is influenced by oxidative stress, which in turn depends on the activity of the enzyme SOD2.
Methods: The study was conducted on 2 groups of mice: 1. Knockdown SOD2; 2. WT. Latency to CNS-OT was measured by preliminary exposure of animals to hyperbaric oxygen (HBO) at 5 ATA, and this was used to derive the time for subsequent exposure at the lower pressures. Mice were exposed to HBO from 2–5 ATA in increments of 1 ATA/wk for 60% of their latency to CNS-OT. BGL were measured before and immediately after each exposure. We evaluated the influence of hyperglycemia and hypoglycemia on latency to CNS-OT prior to HBO exposure.
Results: Glucose levels increased after HBO exposure at 3–5 ATA in the WT mice, whereas in the transgenic mice blood glucose levels increased after HBO exposure at 2–5 ATA. Latency to CNS-OT did not differ between the transgenic mice and the WT on exposure to 5 ATA. However, after the induction of hyperglycemia, latency in the WT mice was prolonged in comparison with the transgenic mice, and compared with the latency observed without hyperglycemia. Following the induction of hypoglycemia, latency in the transgenic mice was shorter than it had been without hypoglycemia.
Conclusion: The induction of hyperglycemic and hypoglycemic states showed that transgenic mice with knockdown of the antioxidant SOD2 are more sensitive to oxidative stress. This may be an indication that the mitochondria play a significant role in the development of CNS-OT.

OF PULMONARY OXYGEN TOXICITY SEVERITY

David Fothergill[1], Warren Ross[2]
[1]Command Suite, Naval Submarine Medical Research Laboratory, USA
[2]Submarine Escape Trainer, Naval Submarine School, USA

Introduction/Background: Assessment of the severity of pulmonary oxygen toxicity (PO_2tox) from hyperoxic exposures has traditionally relied upon measuring changes in lung function using spirometry and diffusion capacity for carbon monoxide (D_LCO). With the introduction of commercial impulse oscillometry (IOS) systems it may now be possible to detect changes in pulmonary function at an earlier stage in the oxygen toxicity process than is possible using the aforementioned traditional measures of pulmonary function. The objective of this study is to contrast IOS measures of pulmonary function with traditional spirometry following a provocative hyperbaric oxygen (HBO) chamber dive.
Methods: In a double-blind randomized crossover fashion 14 male US Navy divers conducted a nitrox (30.6% O_2 balance N_2) and a HBO (100% O_2) 390 min hyperbaric chamber dive at 2 ATA separated by 1 week: Basic spirometry, D_LCO, and IOS parameters were measured before and immediately after each dive.
Results: The relative changes (mean±SD) in pulmonary function following the nitrox and HBO dives are shown below.

* = p	Values are % change from pre-dive	
IOS Variable	Nitrox dive	HBO dive
Total airway resistance (R5)	+8.7±20.0	+24.2±26.7*
Proximal airway resistance (R20)	+7.3±14.8	+18.6±13.2*
Peripheral capacitive reactance (X5)	+8.7±53.7	+27.3±45.2*
Lung resonance frequency	+0.4±16.1	+23.4±47.5
Reactance area (AX)	+29.5±115	+116±243
Spirometry		
Forced Vital Capacity (FVC)	+0.1±3.1	-0.1±5.7
Force Expired Volume in 1 s (FEV1)	+0.8±3.3	-2.0±6.2
Forced Expiratory Flow at 25-75% of the pulmonary volume	+2.5±7.6	-5.9±11.9
Inspiratory Capacity (IC)	-1.3±8.9	-7.3±6.6*
D_LCO adjusted for Hb levels	-4.8±8.8	-2.5±9.8

Conclusions: In this study, early onset PO_2tox coincided with large increases in pulmonary impedance as evidenced by significant increases in the IOS parameters R5,R20 and X5 following the HBO dive. In contrast, the only traditional spirometry measure of lung function to show a significant decrease following the HBO dive was IC.

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Session 10: Diving: HPNS and Pressure Physiology

GAS PRESSURE EFFECTS ON NERVOUS SYSTEM: HPNS, NARCOSIS, SIGNS, RISK FACTORS AND MECHANISMS

Rostain Jean-Claude
Aix-Marseille University, C2VN, team V, Système Adénosinergique, Dysoxie, Inflammation. Faculté de Médecine nord, Bd Dramard, 130015 Marseille, France.

Compressed air or nitrogen-oxygen mixture produces nitrogen narcosis from 3 to 4 bars. Compression with breathing mixtures where nitrogen is replaced by helium which has a low narcotic potency induces from 10 bars, the high pressure nervous syndrome (HPNS). During several years, little was known about the origins and mechanisms of the narcosis. The traditional view was that anaesthesia or narcosis occurs when the volume of a hydrophobic site is caused to expand beyond a critical amount by the absorption of molecules of a narcotic gas. The observation of the pressure reversal effect on general anaesthesia has long time supported the lipid theory. However, recently, protein theories have known an increasing consideration since results have been interpreted as evidence for a direct anaesthetic-protein interaction. The question is to know whether inert gases that disrupt dopamine, GABA neurotransmissions and glutamatergic neurotransmission, act by binding processes on proteins of receptors to neurotransmitter.
The origins and mechanisms of the HPNS are related to neurochemical disturbances which include changes at the level of amino-acid and monoamine neurotransmissions. According to the neural structure and the neurotransmission concerned, helium pressure seems to act on synthesis, or on transmitter release or on receptor and on cellular interactions.
The use of narcotic gas added in helium-oxygen mixture such as nitrogen or hydrogen, reduced some symptoms of the HPNS but also had some effects which could be due to an additional effect of the narcotic potency of the gas. Recent researches at the level of basal ganglia of the rat brain could explain the effect of narcotic gas and pressure.

Key Words

Inert gas narcosis, HPNS, Neurotransmission, Nitrogen, Hydrogen, Helium, Basal Ganglia.

HYPERBARIC PRESSURE MODULATION OF NEUROTRANSMITTERS' RECEPTORS

Alice Bliznyuk
Israel Naval Medical Institute, Haifa, Israel

Exposure to high pressure (HP) induces abnormal activity of the CNS, known as the high pressure neurological syndrome (HPNS)**.** It is conceivable that HPNS arises from dysfunction of network synaptic activity. Different neurotransmitters` receptors are responsible for excitatory and inhibitory neurotransmissions; this is one of the fundamental blocks for the proper function of the mammalian brain. Recent studies revealed that the CNS hyperexcitability of HPNS is mostly induced by glutamatergic N-methyl-D-aspartate receptors (NMDARs). Other members of the iGluR receptor group contributed very little to CNS hyperexcitability under HP. AMPA receptors demonstrated no significant responses to HP, while kainate receptors were unaffected by pressure at all. Other amino acids ionotropic receptors such as GABA receptors are also insensitive to HP, whereas glycine receptor although maximal response was not changed, its IC_{50} was considerably increased under HP. Although HP affects various processes within the living cells, the specific modifications in neurotransmission path ways under these conditions is of the greatest importance for HPNS, and requires further extensive investigation on the molecular level to enable us to prevent it.

Key words
AMPA, GABA, high pressure, HPNS, kainate receptors, NMDA

HYPERBARIC PRESSURE MODULATION OF IONIC CHANNELS

Ben Aviner
Israel Naval Medical Institute, IDF Medical Corps, Haifa, Israel.

Pressure is one variable of the fundamental thermodynamic state. A change in pressure will therefore induce alterations in equilibrium or reaction rates. Hyperbaric pressure (HP) will depress a molecular reaction associated with a positive activation volume, whereas a negative activation volume reaction will be facilitated. Hence HP has many potential targets, affecting molecules such as membrane phospholipids, and proteins such as enzymes, transporters, ionic channels, etc. The consequent physiological effects are thus dependent on a multitude of pressure sensitive molecules in a variety of cells, and are extremely complex.

Exposure to HP may lead to development of the high pressure neurological syndrome (HPNS) in both humans and animals. HPNS signs and symptoms include malfunction of the nervous system, a reduction in cognitive function, decreased motor coordination, sleep disorders, and EEG changes. At extreme pressures, tremor, convulsions and seizures may occur.

Ion channels are transmembranal proteins that exhibit functional conformational changes. This, together with their major role in neuronal and synaptic transmission, singles them out as possible factors in the mechanism underlying HPNS. For example, voltage-dependent Na^+ and K^+ channels are responsible for generation and conduction of the action potential (AP) along neuronal axons and muscle fibers, with accumulating evidence that AP duration is lengthened under HP. Pressure was noted to slow the activation and inactivation of voltage-gated Na^+ channel currents, and to cause variable modulation of the AP current amplitude. An examination of the influence of HP on voltage-dependent K^+ channels in some studies showed enhancement of currents, whereas others demonstrated their depression. Similarly, recent investigations have indicated that HP selectively affects different types of voltage-dependent Ca^{2+} channels.

By considering these effects of HP on various ion channels, in combination with data regarding their density of expression in different sections of the nervous system, we may be able to develop a comprehensive model of the mechanism underlying HPNS and other pressure sensitive processes.

EFFECTS OF INERT GAS NARCOSIS ON ATTENTION AND MEMORY IMPAIRMENT

Tomasz Janus[1], Jacek Piechocki[2], Jacek Kot[3], **Jacek Siewiera**[4]
[1]Faculty of Psychology, SWPS University of Social Sciences and Humanities, Poland [2]Mazovian Hyperbaric Therapy Centre, Central Clinical Hospital of the MSWiA, Poland [3]National Center for Hyperbaric Medicine, Medical University of Gdansk, Poland [4]Department of Hyperbaric Medicine, Military Institute of Medicine, Poland

Introduction/Background: To date, several studies about impact of Inert Gas Narcosis (IGN) on long-term memory (LTM), as well as many papers evaluating IGN-related psychomotor decrements have been published. However, very little is known about attention impairment caused by IGN.
Methods/Materials: 7 experiments in 4 different environments (normobaric air, hyperbaric air (5 ATA, N=22, 18 men and 4 woman, 26-44 years old), heliox (5 ATA) and underwater conditions (air, 4 ATA)) were carried out.
During hyperbaric chamber testing (air and heliox), the evaluation of cognitive (LTM, attention) and psychomotor performance was made. LTM functioning were assessed in scheme: learning in normobaric and recall in hyperbaric conditions and learning in hyperbaric and recall in normobaric conditions. Attention functioning in hyperbaric conditions was measured using 2 tests assessing narrow and broad attention and 2 tests evaluating both psychomotor performance and visual search.
As a pilot study, during underwater experiment, the same 2 tests evaluating both psychomotor performance and visual search were carried out.
One-way analysis of variance (ANOVA) was used to assess for statistical differences.
Results: Comparing to control group, IGN disrupts encoding ($p=0.013$) and retrieval ($p=0.007$) from LTM. Neither narrow nor broad attention were affected (p0.05). No differences were found in the psychomotor performance during hyperbaric air exposure (p0.05).
Comparing to hyperbaric air, psychomotor decrements were found in underwater conditions ($p=0.000$, $p=0.041$). The effect of speed-accuracy trade off was present in underwater conditions, resulting in other kinds of errors during the visual search test ($p=0.027$).
Conclusions: IGN disrupts encoding and retrieval from LTM. Different components of attention are differently susceptible to the IGN-related impairment.
Extrapolation from the results of cognitive tests obtained in hyperbaric chamber to underwater performance is controversial. Psychomotor tests must not be extrapolated.

NOBLE GASES SIMULATIONS AT HIGH PRESSURE

Alice Bliznyuk, Yoram Grossman[2], Yevgeny Moskovitz[3]
[2]Physiology and Cell Biology, Ben-Gurion University of the Negev, Israel
[3]Institute of Natural Sciences and Mathematics, Ural Federal University, Russia

Introduction: Noble gases are thought to be chemically inert and therefore breathing them under high pressure (HP) conditions was always considered not harmful. Our recent experiments and simulations with helium showed that helium at HP may alter the transmembranal NMDA receptor (NMDAR) tertiary structure via protein-lipids interactions (contrary to common predisposition). This causes changes of the receptor physiology. The purpose of the present study was to systematically investigate the influence of various noble gases on NMDAR at HP.
Methods: GROMACS software [1] was used to perform Molecular Dynamics simulations of NMDAR, containing glun1-1a and glun2b subunits, embedded in the membrane under various pressure conditions in the presence and absence of the noble gases helium, neon, argon and xenon.
Results: The simulations explicitly showed that there is inverse ratio between the gas molecular weight and the substantial distortions inflicted there for in the membrane and the NMDAR. Root mean square deviation plots indicate that different pressurized gases induce a variety of protein conformational alterations. Furthermore, the alterations in transmembrane domain, containing the receptor's pore (responsible for ions movement across the membrane) are gas type dependent.
Discussion: Combining preliminary analysis of the simulations and previous experimental results, one can speculate that different noble gases at HP (and not just hydrostatic pressure per se) may cause diverse changes of the receptor physiology.

Session 11: Diving: Physiology

BREATH-HOLD DEEP DIVING AND THE LUNG

Jochen Schipke[1], Frederic Lemaitre[2], Clark Pepper[3], Kay Tetzlaff[4]
[1]Research Group Experimental Surgery, University Hospital Düsseldorf, Germany [2]UFR Sciences du Sport et Éducation Physique, Université de Rouen, France [3]Dept. of Neurology, Johanna Etienne Hospital, Germany
[4]Dept. of Sports Medicine, Eberhard-Karls-University Tübingen, Germany

Background: Breath-hold (BH) diving is a form of underwater diving that relies on divers` ability to hold their breath until resurfacing. BH diving has gained worldwide popularity. Some records are breath-taking: static apnea: 11:35min and no-limits: 214msw. While these achievements are remarkable in terms of physiology, acknowledgement is needed that these extreme exposures incur inherent risks.
Methods: Internet search with 'apnea', 'deep diving', 'lungs'.
Results/Discussion: This review-like presentation focuses on pulmonary injury in BH deep divers. When practicing their extreme leisure sport, they are exposed to increased pressure of pulmonary gas volumes, increased partial gas pressures and hypoxia. Increasing ambient pressures do present a serious problem to BH deep divers, because the semi-rigid thorax greatly prevents the deformation required by Boyle-Mariottes` law. As a result, a negative pressure barotrauma (=lung squeeze) with acute hemoptysis is not uncommon. Respiratory manoeuvres like glossopharyngeal insufflation (GI) and glosso-pharyngeal exsufflation (GE) are practiced in order to prevent lung squeeze and to permit equalizing of the paranasal sinuses and the middle ear. GI not only impairs venous return, thereby provoking hypotension and even fainting, but also produces intrathoracic pressure likely to induce pulmonary barotrauma that is speculated to induce long-term injury. GE, in turn, further increases the already negative intrapulmonary pressure, thereby favouring alveolar collapse (atelectasis).
Finally, hypoxia not only induces brain injury but seemingly initiates the opening of intrapulmonary shunts. These pathways are large enough to permit transpulmonary passage of venous N_2 bubbles, making stroke-like phenomena in deep BH divers possible.
Conclusions: BH deep divers have employed pathophysiological manoeuvres that have already led to pulmonary injury and serious accidents. The rule should be no hyperventilation, no glossopharyngeal insufflation or exsufflation and no exaggerated depths. We hope that BH deep divers will become better aware of the risks of their extreme leisure sport.

ARTERIAL BLOOD GASES IN BREATH-HOLD DIVERS AT THE BREAKING POINT

Gerardo Bosco[1], Alex Rizzato[1], Alex Rizzato, Gerardo Bosco, Luca Martani, Luca Martani[2], Matteo Paganini[1], Matteo Paganini, Giacomo Garetto, Giacomo Garetto[1,3], Enrico Camporesi[4], Enrico Camporesi, Richard Moon[5], Richard Moon
[1]Department of Biomedical Sciences, Mater in Diving and Hyperbaric Medicine, Italy [2]Fidenza Hospital, Parma, Anesthesiology, Critical Care and Hyperbaric Medicine Unit, Italy [3]Hyperbaric Medical Center, ATIP, Italy [4]Tampa General Hospital, TEAMHealth Research Institute, USA [5]Anesthesiology, Hyperbaric Medicine and Environmental Physiology, Duke University Medical Center, USA

Background: Previously we have measured the arterial blood gases (ABG) in breath-hold divers (BHD) at a depth of 40 m in a swimming pool (Bosco G, et al Front. Physiol. 2018; 9:1558). This project aimed to investigate human blood gases at BHD breaking point after a dive at 40 m.
Methods: After institutional approval and informed consent, six well trained, healthy breath-hold divers completed this study (41.67 ± 8.78 years, 72.83 ± 7.88 kg, 1.79 ± 0.08 m). Blood samples were obtained under the following conditions: Immediately before a dive (10 min prior to submersion, PRE); Moreover, samples were drawn immediately after three different diving condition (POST). In each post condition, blood sampling was performed at surface with the diver at the border of the pool with the face still submerged and before the breaking-point (Figure 1). In detail:

- After breath-hold dive at surface (POST SUR). Subjects performed a breath-holding of ~ 95 seconds while moving at surface using a sea-bob to mimic the sled-assistance without leg exercise.
- After breath-hold dive to 42 m (POST DP), accompanied by a professional instructor using a sled for descent and ascent.
- After breath-hold dive to 42 m (POST DP-EXE) with self-powered descent and ascent.

Results: ABG values are shown in the Table 1 (mean ± SD).
Conclusions: Significant hypoxemia occurs at the end of BHD, consistent with risk for loss of consciousness, with minimal changes in $paco_2$.

Condition	**PaO_2 (mmHg)**	**SaO_2 (%)**	**$PaCO_2$ (mmHg)**	**pH**
PRE	96.2 ± 7.0	97.67 ± 0.52	38.22 ± 2.96	7.43 ± 0.02
POST SUR	64.5 ± 4.7	92.00 ± 2.10	42.75 ± 6.03	7.40 ± 0,05
POST DP	39.8 ± 8.7	75.83 ± 11.41	31.38 ± 3.70	7.46 ± 0.04
POST DP-EXE	31.6 ± 17.0	55.60 ± 24.08	36.08 ± 5.25	7.43 ± 0.05
Condition	**HCO_3- (mmol/L)**	**tCO_2 (mmol/L)**	**BE, ecf**	**Lactate (mg/dL)**
PRE	25.22 ± 2.22	26.17 ± 2.14	0,83 ± 2,48	0.64 ± 0.38
POST SUR	26.35 ± 1.90	27.50 ± 1.87	1,50 ± 2,17	0.79 ± 0.17
POST DP	22.22 ± 1.62	23.33 ± 1.75	-1,67 ± 1,51	1.18 ± 0.17
POST DP-EXE	24.00 ± 3.02	25.20 ± 3.11	0.00 ± 3,39	2.09 ± 0.35

MENTAL STRESS MAY CAUSE HIGH GAS CONSUMPTION AND HEART RATE IN RAPID DESCENDING SCUBA DIVERS

Nico Schellart[1,3], Jean-Claude Le Péchon[2], Hans van Dam[3]
[1]Biomedical Engineering and Physics, Amsterdam UMC, University of Amsterdam, Netherlands [2]JCLP Hyperbarie, JCLP Hyperbarie, France
[3]Foundation for Dive Research (SDR), Foundation for Dive Research (SDR), Netherlands

Background: The descent is a critical part of a dive, both physically and mentally. Pulmonary ventilation, expressed as respiratory minute volume (RMV) and heart rate (HR) were recorded during fast and slow descents to 35 m in open sea while breathing compressed air, and during swimming horizontally at moderate velocity at an 11-m depth. Values of both types of descents were compared with reference values recorded at 11 m, the 'plateau' phase, halfway through the 35-m dives. It is hypothesized that the 'slow-descent' and 'plateau-phase' values will be less than the 'fast-descent' values.
Methods: Depth, cylinder pressure, water temperature and HR were recorded with dive computers yielding time-averaged means (mrmv and mhr) for descent and plateau. Of the 18 divers included, 16 performed the fast and 11 the slow descents.
Results: Fast descents (23 $m \cdot min^{-1}$ vertically), performed with 0-8 fin kicks, yielded $mrmv_{descent}$=28 ambient L (al)$\cdot min^{-1}$, which is 82% higher (P0.001) than $mrmv_{plateau}$ of 15 $al \cdot min^{-1}$. Further, $mhr_{descent}$ was121 beats$\cdot min^{-1}$ 23% higher (P0.001), than $mhr_{plateau}$ of100 bpm. Slow descents (2.4 $m \cdot min^{-1}$ vertically) yielded 17 $al \cdot min^{-1}$ with mhr=101 beats$\cdot min^{-1}$, values only slightly higher than at Plateau. The 11-m dive (swimming horizontally) yielded 24 $m \cdot min^{-1}$ with 32 fin kicks$\cdot min^{-1}$, mrmv=35 $al \cdot min^{-1}$ and mhr=115 beats$\cdot min^{-1}$.
Conclusions: Fast descents cause a higher RMV and HR that cannot be explained by physiology alone. Presumably mental stress is a main contributor. For dives deeper than 20 m, a descent velocity of 10 $m \cdot min^{-1}$ is recommended to reduce cardiac stress, especially for older divers.
Accepted by UHM.

SCUBA DIVING AND CONGENITAL HEART DISEASE IN CHILDREN AND ADOLESCENTS

Christian Beyer
Schwerpunktpraxis für Kinder-Jugendkardiologie / GUCH / Tauchmedizin, Dr. Christian Beyer, Germany

Most diving physicians who are asked to permit a child or adolescent with congenital heart disease to scuba are uncertain how to proceed. It can be dangerous. However, many patients with congenital heart disease can be granted a medical clearance to scuba dive.
Congenital heart disease is the most common malformation in childhood and the survival rate is now over 90%. Hence, the adult group (GUCH=Grown Ups with Congenital Heart disease) has increased dramatically.
Specific information is needed in order to adequately assess the patient's condition and fitness for scuba diving. This includes a deeper understanding of these malformations and the longtime results of surgical interventions.
In the presentation, proposals to manage the decision for scuba diving will be given for the most frequent congenital heart diseases with the topics:
- Information about symptoms of a congenital heart disease and how they may influence the necessary skills for scuba diving.
- Information about influences of specific medication and possible side-effects.
- The maximum recommended degree of exposure will be discussed in the light of current literature about sports with congenital heart disease and the physiology of immersion in cold water with respect to influences on specific heart disease.
The criteria outlined will aid a physician in judging whether a patient is capable of scuba diving.

SCUBA DIVERS PULMONARY EDEMA. REVIEW OF 10 YEARS EXPERIENCE

Jose M. Inoriza[1], Gemma Oliu[1,2], Pilar Garcia-Martin[1,2], Jose M. Villar-Marques[1], Maria J. Martí-Utset, Rodrigo Galan[1], Debora Gimenez-Babiloni
[1]Unitat de Medicina Hiperbàrica, Hospital de Palamós - Serveis de Salut Integrats Baix Empordà, Spain
[2]Servei d'Urgencies, Hospital de Palamós - Serveis de Salut Integrats Baix Empordà,, Spain

Aim: To review the experience of our Unit in the management of scuba divers pulmonary edema (SDPE)
Material and methods
The cases of SPDE diagnosed between 2009 - 2018 are reviewed. We analyzed data related to people (age, sex, medical history); the characteristics of the dives, the treatment and the results obtained.
Results: In the period studied, 9 cases were diagnosed (6 men, 3 women) between 49 and 68 years old.
The dives ranged between 18 and 52 meters of maximum depth, with duration of less than 30 min, except one of them.
The typical symptomatology was the sudden onset of dyspnea before the end of the dive (between 3 and 10 meters deep). In the physical examination, in all cases, basal crackles was present, only 2 out of 9 presented pulse oxymetry less than 90%. All cases the diagnosis was made by clinical examination and simple chest radiography.
In all cases, normobaric oxygen therapy and furosemide were administered, allowing the resolution of the symptoms in 24 hours usually.
In 8 out of 9 cases, the divers did not reside in our area of influence, so it was not possible to make a long-term follow and to know underlying cause.
Conclusions: The SDPE is a diagnosis relatively little known in the emergency services, which is expected to increase its incidence due to the increase in age of the diving population.

LATENCY TO CNS-OXYGEN TOXICITY AND EEG PATTERNS ARE AFFECTED BY BLOOD MAGNESIUM LEVELS IN RATS

Ben Aviner[1], Yehuda-Matan Danino[1], Mirit Eynan[1], Alice Bliznyuk[1], Liav Leiboviz[3], Valentina Borgil[3], Rivka Efrat[3], Adi Biram[1], Yoram Grossman[2]
[1]IDF Medical Corps, Israel Naval Medical Institute, Israel
[2]Department of Physiology and Cell Biology, Zlotowski Center for Neuroscience, Ben-Gurion University of the Negev, Israel
[3]Materials & Chemistry Department, Tel-Hashomer, Technology Division Test, Evaluation & QA Unit, Israel

Introduction: Combat divers, who use closed-circuit breathing apparatus, run the risk of developing central nervous system oxygen toxicity (CNS-OT). This may lead to seizures underwater, drowning and death. Previous studies have suggested that CNS-OT is related to the accumulation of reactive oxygen and nitrogen species, whose production is promoted by activation of N-Methyl-D-Aspartate receptors (NMDAR). Magnesium sulfate ($mgso_4$), an NMDAR blocker, has been reported to prolong latency to CNS-OT seizures in rats. The purpose of the present study was to determine the relationship between blood magnesium levels, latency to the onset of CNS-OT, and the patterns on the electroencephalogram (EEG) which precede the appearance of seizures.
Methods: Rats were divided into six groups: sham, control, high magnesium ($mgso_4$-treated), low magnesium (Mg^{2+}-deficient diet), memantine (an NMDAR antagonist) and low magnesium with memantine. Animals were implanted with a telemetric EEG transmitter and exposed to 6 atmospheres absolute breathing oxygen. Latency to seizures and EEG patterns were recorded.
Results: There was significant prolongation of the latency to CNS-OT in the $mgso_4$-treated group, and a significant reduction in the Mg^{2+}-deficient group. Memantine mimicked the effect of $mgso_4$, although to a lesser degree, and managed to overcome the effect of the Mg^{2+}-deficient diet. EEG pattern changes were identified during the minutes preceding seizure onset.
Conclusions: Our findings with regard to CNS-OT suggest that $mgso_4$ may be used as a protective agent, that nmdars are involved in the underlying mechanism, and that a predictive algorithm identifying modulations in the EEG may be developed to avoid its onset.

IMPULSE OSCILLOMETERY AND SPIROMETRY AS INDICES HYPERBARIC OXYGEN DOSE-RESPONSE OF NMDA RECEPTOR CURRENTS

Alice Bliznyuk[1], Michael Hollmann[2], Yoram Grossman[3]
[1]Israel Naval Medical Institute, Israel Defense Forces, Israel
[2]Department of Biochemistry I – Receptor Biochemistry, Ruhr University Bochum, Germany
[3]Department of Physiology and Cell Biology, Ben-Gurion University of the Negev, Israel

Introduction: Previous experiments using a rat model indicated that hyperbaric oxygen (HBO) at 5.5 atmospheres absolute (ATA) causes convulsions, a manifestation of central nervous system oxygen toxicity (CNS-OT). Other studies indicated that HBO may increases the N-methyl-D-aspartate-receptor (NMDAR) current response. This suggests that nmdars may be heavily involved in CNS-OT in combat divers. In addition, during HBO therapy (HBOT) patients are also exposed to 100% oxygen over a lower pressure range. The purpose of the present study was systematically to investigate the influence of HBO on NMDAR currents.
Methods: glun1-1a was co-expressed with glun2a in Xenopus laevis oocytes. Ionic currents were measured in Ba^{2+} solution with no added $[Mg^{2+}]_o$ or $[Ca^{2+}]_o$, using two-electrode voltage clamp, in response to bath application of the co-agonists glutamate (100 μm) and glycine (10 μm) in O_2 at 1, 2, 3, 4 and 5.5 ATA.
Results: An increase in the NMDAR current response was observed under elevated HBO pressure (Fig. 1). There was a good linear correlation between the increase in current response and pressure ($y=0.0668x+0.9004$, $R^2 = 0.9918$), although only the response at 5.5 ATA was statistically significant.

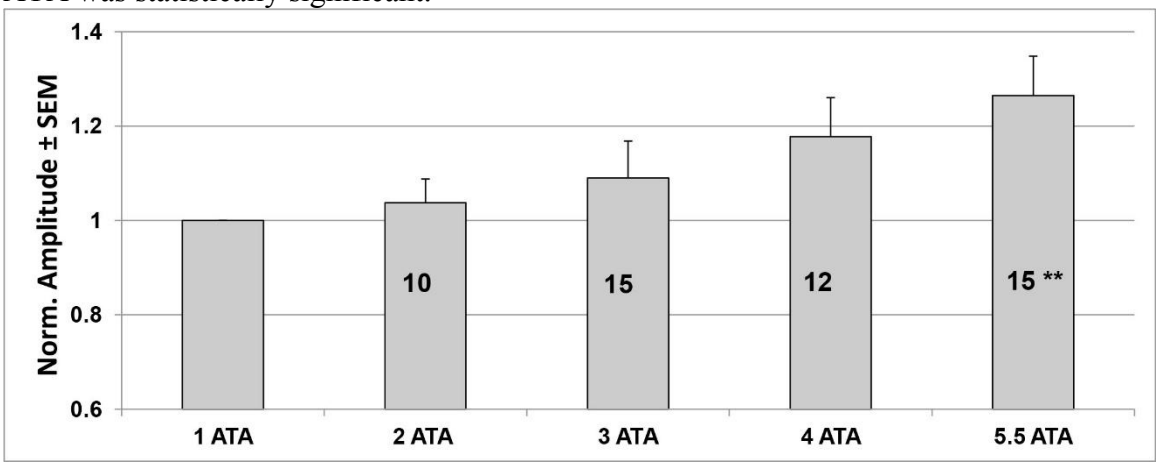


Figure 1. Current amplitudes of the GluN1-1+GluN2A subtype in HBO at different pressures. Numbers in the bars indicate number of oocytes tested. ** p 0.01
Discussion: Our results suggest that the response of NMDARs under HBO is involved in hyperexcitability of the CNS. However, we were unable to demonstrate a threshold for the effect. At 1-2 ATA the effect is less pronounced, suggesting little involvement of NMDARs in any effect of HBO at these pressures.

Session 12: Wound Care I

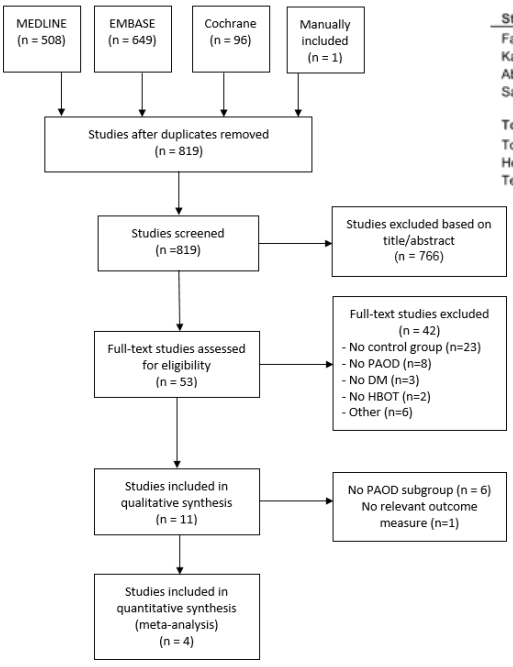
HYPERBARIC OXYGEN THERAPY FOR THE ISCHEMIC DIABETIC FOOT: A SYSTEMATIC REVIEW AND META-ANALYSIS

Robin Brouwer[1,5], Rutger Lalieu[2], Rigo Hoencamp[1,3,4], Rob Van Hulst[5], Dirk Ubbink[6]

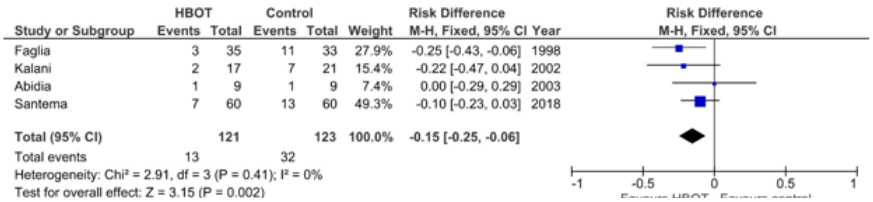
[1]Surgery, Alrijne Ziekenhuis, Netherlands
[2]Hyperbaric Oxygen Therapy, Hyperbaar Geneeskundig Centrum, Netherlands [3]Defense Healthcare Organisation, Ministry of Defence, Netherlands [4]Surgery, LUMC, Netherlands
[5]Anesthesiology, Amsterdam UMC, Netherlands
[6]Surgery, Amsterdam UMC, Netherlands

Background: Diabetic foot ulcers (DFU) are frequently associated with peripheral arterial occlusive disease (PAOD) and may ultimately lead to amputations of the lower extremity. Adjuvant hyperbaric oxygen treatment (HBOT) might foster better wound healing and lower amputation rates in patients with DFU and PAOD. A systematic review was conducted to assess the effects of HBOT as an adjunctive therapy to standard treatment for patients with dfus with PAOD.
Methods: Systematic review using the MEDLINE, EMBASE and Cochrane CENTRAL databases (from inception to October 2018). All original, comparative studies on the effect of HBOT on dfus with PAOD were eligible. The primary outcome measures were amputation rate, amputation-free survival, complete ulcer healing and mortality.
Results: Eleven studies, totaling 729 patients, were included for analysis, including seven randomized clinical trials, two controlled clinical trials, and two retrospective cohorts. Four were used for quantitative synthesis. Meta-analysis showed a significantly fewer major amputations in the HBOT group (10.7% vs. 26.0%; RD=-15%, 95%CI -25 to -6, P=0.002, NNT=7, 95%CI 4-20). No difference was found for minor amputations (RD=8%, -13 to 30, P=0.46). Three studies reporting on complete wound healing showed contrasting results. No significant difference was found for mortality or amputation-free survival.
Conclusion: Current evidence shows that adjuvant HBOT decreases major amputation rate, but not wound healing, in patients with dfus and PAOD. Given the wide range of patients included in the trials, a better patient selection may help define which patients with dfus and PAOD benefit most from HBOT as standard adjunctive treatment.

Figure 1. PRISMA flow chart for meta-analysis



Forest plot showing the effect of HBOT on major amputations; HBOT: hyperbaric oxygen therapy; CI: confidence interval; M-H: Mantel-Haenszel test.



ALGORITHM FOR EVALUATION AND MANAGEMENT OF DIABETIC FOOT ULCERS

Michael Strauss[1], Caroline Ko[2], Lientra Lu[3], Stuart Miller[1]
[1]Hyperbaric Medicine Program, Memorial Care, Long Beach Medical Center, USA
[2]Podiatric Medicine, Memorial Care, Long Beach Medical Center, USA
[3]Research, Tibor Rubin Veterans Affair Medical Center, USA

Background: Diabetic foot ulcers (dfus) have assumed increasing importance as a condition where hyperbaric oxygen (HBO) is used as an adjunct to surgical and medical management. Unfortunately, the current indications for using HBO are imprecise and lend themselves to misuse of this adjunctive modality. We have generated an algorithmic approach to the evaluation and management of dfus that integrates an innovative and validated wound score with the three predominant confounding factors that interfere with wound healing.
Methods: The Long Beach Wound Score (LBWS) summates five assessments (appearance of wound base, size, depth, infection, and perfusion); each grades on a 0 (worst)-to-2 (best)-point scale using objective criteria. Grades of 7.5 to 10 points categorize the wound as Healthy, 3.5 to 7 points as Problem, and 0 to 3 points as End-Stage. Each wound category dictates specific measures for wound management.
Results: Healthy wounds need only the simplest, least expensive dressing agents. If wound improvement is not observed after six to eight weeks, the biologic skin substitutes may be considered. Over 90 percent of Problem wounds have one of these confounding factors, namely deformity, deep infection, and ischemia/hypoxia. Each factor has specific measures for mitigation, for example, offloading and surgical correction for deformities, antibiotics and debridement for deep infection, and revascularization and/or HBO for ischemic/hypoxic wounds.
Conclusions: The LBWS provides a validated and quantifiable approach for the evaluation and management of dfus and shoes the specific niche where HBO is indicated. This approach facilitates comparative effectiveness research and the establishment of the minimally clinically important difference in wound management.

USES AND ABUSES OF NEGATIVE PRESSURE WOUND THERAPY AND BIOLOGICS TO AID IN WOUND HEALING

Michael Strauss[1], Ian Gordon[2], Lientra Lu[3], Stuart Miller[1]
[1]Hyperbaric Medicine Program, Memorial Care, Long Beach Medical Center, USA
[2]Vascular Surgery, Tibor Rubin Veterans Affair Medical Center, USA
[3]Research, Tibor Rubin Veterans Affair Medical Center, USA

Background: Wound healing has become a significant expense to the healthcare system. In the USA alone, it accounts for over 15 billion dollars expended each year. Two developments, negative pressure wound therapy (NPWT) and biologics, have been important contributors to the increasing costs of wound care.
Methods: In dealing with over 100 new lower extremity wound problems each year over a 20-year period, uses and abuses of NPWT and biologics have become well defined for us. The proponents of these interventions often use persuasive marketing techniques to convince wound care providers to use their products.
Results: Negative pressure wound therapy has unparalleled value in managing cavitary wounds without underlying deep infection such as pre-sacral, ischial, and trochanter pressure injuries. For superficial wounds and skin graft donor sites, we have observed that its value is limited and other less costly interventions such as ointments, membrane-type dressings, and skin grating can be more cost-effective.
The usefulness of biologics such as skin substitutes, platelet-rich plasma, and mesenchymal stem cells in wound healing has not been adequately clarified. The cost of using these agents is often borne by the consumers themselves. At best, we recommend using biologic skin substitutes when healthy-appearing, vascular-based wounds do not show signs of epithelialization after six to eight weeks of conventional, less expensive therapy.
Conclusions: A generation of new products for aiding wound healing has become available. Discriminative use of these products mandates that the providers be familiar with their specific uses and potential abuses.

ECONOMIC EVALUATION ON HBOT IN TISSUE REPAIR

Pasquale Longobardi
Hyperbaric Medicine, Centro Iperbarico SRL, Italy, P. Longobardi, K. Hoxha, Hyperbaric Center, via Augusto Torre 3. 48124 Ravenna (Italy)

To evaluate the value of the HBOT for the money spent the Number Needed to Treat (NNT) was used. The NNT is the estimate of the number of patients that need to be treated (in relation to the number of HBOT sessions for each patient) in order to have an impact on one. The NNT value is 4 up to 35 HBOT sessions and 3 beyond 35.
We surveyed the ECHM experts to know the cost of each HBOT session and amputation. In Belgium the cost of HBOT was estimated because the NHS reimbursement is restricted to first and second day of treatment. US data were included for comparison. (Table 1). Seeing as the weighted average cost of a leg amputation was not homogenous, it was decided to standardize for the UK (NHS) value of £12,000, plus the costs for prosthetic limbs, rehabilitation and wound care in the year after an amputation of £20,000 with a total cost for leg amputation and care in the first year of £32,000 per person (USD 41,352). Other costs such as home adaptations, community care, care homes and wheelchairs are likely to be borne by local authority social services departments and have not been estimated by the NICE.
In the majority of the Countries indicated, the HBO cost is from neutral to likely saving except of Norway and US where the cost pressure for the NHS of HBO is significantly high (due to the cost of each HBO session). The value for money spent remains the same when the number of the HBO sessions increases beyond 35, because the NNT improves from 4 to 3 (same NNT product for HBOT sessions: 120 HBOT sessions are required to save a leg amputation).

Nation	Cost HBOT session (USD)	Number HBOT sessions	NNT	Cost HBOT to save an amputation	Cost amputation (USD)	Value for money (USD)
Belgium	127	30	4	15,240	41,352	26,112
Czech	81	30	4	9,720	41,352	31,632
France	256	30	4	30,720	41,352	10,632
Italy	93	30	4	11,160	41,352	30,192
Malta	50	30	4	6,000	41,352	35,352
Norway	424	30	4	50,880	41,352	-9,528
Spain NHS	58	30	4	6,960	41,352	34,392
Spain private	173	30	4	20,760	41,352	20,592
Turkey NHS	22	30	4	2,640	41,352	38,712
UK	227	30	4	27,240	41,352	14,112
USA	500	30	4	60,000	41,352	-18,648

THE EFFECT OF HYPERBARIC OXYGEN THERAPY ON HEALING OF CHRONIC LEG ULCERS

Abhishek Srivastava[1], Praveen Krosuri[2], Navita Purohit[1], Raghuram Sekhar[2]
[1]physical Medicine & Rehabilitation, Kokilaben Dhirubhai Ambani Hospital, India
[2]general Surgery, Kokilaben Dhirubhai Ambani Hospital, India

Aims & Objectives: Chronic leg ulcers are a common cause of morbidity. The aim of present study was to assess the effect of hyperbaric oxygen therapy (HBOT) on healing of chronic leg ulcers as an adjuvant to standard wound care.
Material and Methods: Prospective observational comparative study and the selected patients were followed for 12 weeks. According to their choice, patients are divided into two Arms, Arm (1) consists of patients who received HBOT+ Standard wound care and Arm (2) consists of patient who received Standard wound care alone. Arm (1) receive 100% oxygen at 2 ATA for 90 min in a mono-place chamber (HBOT) 4 times a week for a period of 3 weeks along with standard wound care. In Arm 2, patients receive standard wound care alone. Wound care is standardized throughout the study to several different dressing types dependent on the type of the wound, on and off debridement, treatment of infection, off-loading and metabolic control. Patients are assessed at end of 3rd week at completion of HBOT and at 12th week for change in size of the Index ulcer and presence of healthy granulation tissue. Rate of healing is assessed by reduction in size of index ulcer i.e., ulcer area covered with epithelial regeneration.
Results: 35 patients were enrolled in each arm. Mean percentage regression in size of Index ulcer when compared to initial size was 39.9% (39.9% +/- 10.9%) in arm 1 and 20.0% (20.0% +/- 10.4%) in arm 2 at the end of 3rd week and 72.4% (72.4% +/- 17.5%) in arm 1 and 57.5% (57.5% +/- 21.2%) in arm 2 at 12th week.
Conclusions: Hyperbaric oxygen therapy has a definitive adjunctive role in the management of chronic non healing leg ulcers. Large scale randomized controlled studies are required.

HYPERBARIC OXYGEN THERAPY FOR HEMORRHAGIC CYSTITIS

Shachar Finci, Erez Lang, Gregory Fishlev, Nir Polac, Amir Hadany, Shay Efrati
Sagol Center for Hyperbaric medicine and Research, Assaf-Harofeh Medical Center, Israel

Background: Hemorrhagic cystitis (HC) is a significant and serious clinical problem occurring mostly after pelvic radiation therapy and/or chemotherapy, and is often refractory for conventional treatments.
Objectives: To evaluate the efficacy and safety of hyperbaric oxygen therapy (HBOT) for HC.
Methods: Retrospective analysis of patients suffering from HC treated with HBOT in the Sagol Center for Hyperbaric Medicine and Research from January 2014 TO December 2018.
The treatment protocol was based on daily session, 5 days per week, of 90 minutes 100% oxygen at 2 ATA with 5 minutes air breaks every 20 minutes.
Results: Sixty seven were included in the analysis. The mean age was 62.7 years (8-88 years). The mean time interval between radiation/ chemotherapy and the initiation of HBOT was 52.6 months (1-204 months). The patients received a mean of 31 HBO sessions (2-60). With HBOT, complete resolution of the hematuria was achieve in 73% of the patients, significant improvement was achieved in 25% of the patients and persisted hematuria unresponsive to HBOT in 2% of the patients. The mean number of HBOT sessions needed to achieve complete resolution was 18.
HBOT was found to be generally safe: 5 patients suffered from ear barotrauma, 3 of them had spontaneous resolution after few days of rest and 2 needed ear tube insertion in order to complete the HBOT protocol.
In one case HBOT was discontinued after 16 sessions due to pain and inability to seat in the chamber for 90 minutes.
Conclusions: HBOT is an effective and safe treatment for patients suffering from HC

THE EFFECTS OF HYPERBARIC OXYGEN PRECONDITIONING IN AN EXPERIMENTAL MODEL OF ACUTE KIDNEY INJURY

Predrag Brkic[1], Milan Ivanov[2], Rada Jeremic[1], Marina Djelic[1], Nevena Mihajlovic-Stanojevic[2], Djurdjica Jovovic[2], Una-Jovana Vajic[2], Danijela Karanovic[2], Zoran Miloradovic[2]
[1]Institute of Medical Physiology "Richard Burian", School of Medicine, University of Belgrade, Serbia
[2]Institute for medical research, National Institute of Republic of Serbia, University of Belgrade, Serbia

Background: The focus of our research was to investigate weather preconditioning with hyperbaric oxygen (HBO) may contribute to outcome of the experimental acute kidney injury (AKI).
Methods: Male 24 weeks old Wister rats (WR) and age matched spontaneously hypertensive rats (SHR) were organized in following groups: sham operated rats (SHAM), rats with induced postischemic AKI and group with HBO preconditioning. Each observed group was divided into two subgroups with different strains of rats- normotensive (WR) and hypertensive (SHR). Hyperbaric oxygen preconditioning protocol (HBOTP): pressure 2.0 absolute atmospheres (ATA), for 60 minutes, twice per day, at 12 hours interval, for 2 days. The haemodynamic parameters, kidney function markers, and oxidative status markers were observed.
Results: After AKI induction reduction of blood pressure and HR was recorded in both types of rats. HBOTP improved renal blood flow and vascular resistance in renal artery in WR and SHR rats with AKI and markedly decreased plasma creatinine in comparison to AKI groups. Thiobarbituric acid reactive substances plasma levels were significantly decreased in both WE and SHR by HBOTP in comparison to AKI groups.
Conclusion: Our study, for the first time, has shown that preconditioning with HBO can improve kidney function and decrease oxidative stress in experimental model of acute kidney injury. Our results demonstrate the therapeutic potential of HBO for the treatment of renal injury, but the underlying mechanism and appropriate clinical protocols are needed to be further investigated.
Keywords: hyperbaric oxygen therapy, acute kidney injury, oxidative stress
Poster presentation

Session 13: CO Intoxication

CASE SERIES OF CARBON MONOXIDE POISONING IN SINGAPORE

Ng Peng, Boon Hor Ho
Navy Medical Service, Republic of Singapore Navy, Singapore

Background: There is a low incidence of moderate to severe carbon monoxide poisoning cases seen in Singapore. The patients who survive CO poisoning may exhibit cardiac dysfunction and suffer from long-term neurocognitive sequelae. Recent practice recommendations by experts in the hyperbaric medicine field recommend hyperbaric oxygen therapy (HBOT) use as first line treatment for CO poisoning. However, the review of published trials found conflicting evidence regarding the usefulness of hyperbaric oxygen for the prevention of neurological injury.
Case Description: We present a case series of patients who suffered from acute moderate to severe CO poisoning. Five patients were rescued from carbon monoxide exposure and received HBOT in a tertiary institution in Singapore over a one year period from 2018 to 2019.
Of the five patients, one had accidental exposure while four had exposure from attempted suicide via charcoal burning in enclosed space. One patient was noted to be pregnant. Three patients were intubated due to metabolic acidosis and low GCS. Two patients were noted to have evidence of myocardial ischaemia. All patients received high flow oxygen therapy prior to initiation of HBOT and were treated with the same HBOT protocol: 1 x (3 ATA x 1 h; 2 ATA x 1 h) then 2 x (2 ATA) x 90 min.
All patients were followed up after completing HBOT to monitor for sequelae and concurrent psychiatric issues.
Conclusion: All the patients with CO poisoning responded well with this protocol of hyperbaric oxygen therapy. Our experience demonstrates that HBOT, when applied consistently for the management of CO poisoning, is effective and important as a first line treatment and can reduce the sequelae of CO Poisoning when treated early within 24 hours.

TREATMENT OF CARBON MONOXIDE INTOXICATION/ENCEPHALOPATHY WITH HYPERBARIC OXYGEN THERAPY - 26 YEARS' EXPERIENCE 1986-2013 - RETROSPECTIVE REVIEW OF AN ALTERNATIVE TREATMENT PROTOCOL

Jeffrey cooper, Lon Keim
Hyperbaric Medicine, Nebraska Medicine, USA

Background: Hyperbaric oxygen (HBO) is beneficial in the treatment of acute Carbon Monoxide (CO) poisoning while decreasing the risk of delayed neurologic sequelae. Reports have confirmed the rationale for 2.8 - 3.0 ATA initially in the first dive, while studies by Weaver have demonstrated improved outcomes when three HBO treatments are provided in the first 24 hours. 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 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.
Methods: We retrospectively analyzed our experience 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. 13.3% required more than 3 treatments. 57.8% of these had complete subjective resolution of symptoms. 38.5% demonstrated substantial improvement.
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.

HYPERBARIC OXYGEN TREATMENT BEYOND USUAL THERAPEUTIC WINDOW ENHANCES NEUROLOGICAL RECOVERY AFTER CARBON MONOXIDE POISONING

Jeffrey Cooper, Lon Keim, Dan Murman, Diego Torres-Russotto
Emergency Medicine and Neurology, University of Nebraska Medical Center, USA

Background: Carbon monoxide may be responsible for half of all fatal poisonings worldwide. Delayed neuropsychiatric sequelae, including apathy, gait disturbances, movement disorders, seizures, hallucinations, and dementia, have been reported in 15-45% of survivors after apparent recovery. One hundred percent oxygen administered immediately following initial rescue is the standard of care. Hyperbaric oxygen therapy (HBO2) within the first 24 hours of care is believed to offer additional benefits, and is recommended to those patients having lost consciousness or altered mental or neurologic state. HBO2 reduces the incidence of cognitive and neurological dysfunction by 46% at 6 weeks and 12 months from exposure. The therapeutic efficacy of providing HBO2 beyond the first 1-2 days after initial insult is unknown and generally unaccepted. However, some evidence exists for its potential benefit.
Methods: We report treating a patient with HBO2 14 months after carbon monoxide induced brain injury resulting in markedly improved neurologic status. He received a total of 100 treatments (90 minutes at 2.4ATA with 2 5 minute air breaks).
Results: The patient improved markedly from a disabled baseline of inability to cook, feed, ambulate, or dress, let alone work. The most relevant outcome is that the patient regained independence, the ability to drive, perform activities of daily living and became gainfully employed.
Conclusions: This may be the first report of beneficial HBO2 begun greater than a year following the initial exposure. Further studies evaluating the potential benefit of HBO2 for brain injury after the acute phase of carbon monoxide poisoning are warranted.

CARBON MONOXIDE STRESS TEST

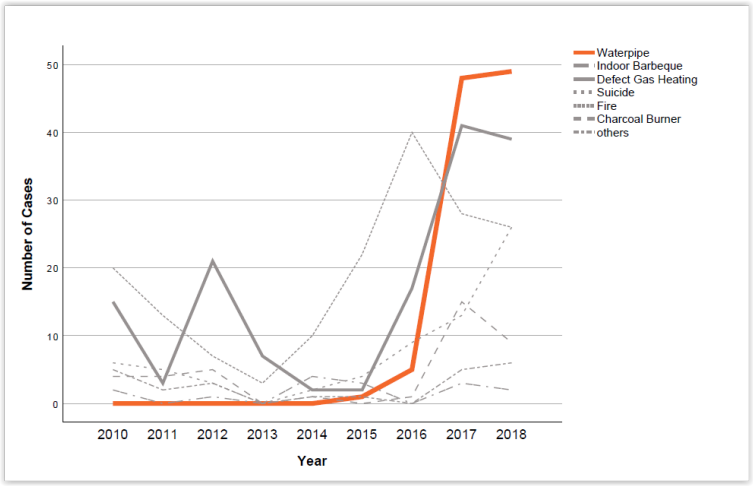
Jeffrey Cooper, Jinfeng Jiang
College of Medicine, University of Nebraska Medical Center, USA

Introduction: Carbon Monoxide (CO) poisoning presents with many different cardiac effects, but one important presentation is its effect as a CO stress test to reveal underlying coronary artery disease (CAD). There are a limited number of publications detailing this phenomenon, but it is important to suspect CAD in association with mild troponin leak or non-ST segment elevation myocardial infarction (NSTEMI) on electrocardiogram (EKG) after CO intoxication.
Case Reports: We recently treated three patients with CO poisoning with underlying CAD. These three cases highlight three different ways in which CO may cause or uncover cardiac disease. In the first case, a man presents to the ED with CO toxicity, a ST segment elevation myocardial infarction (STEMI) was found and emergent angioplasty discovered severe CAD. The second case saw a man present with CO poisoning with rising troponin levels. An angioplasty discovered a stable 90% occlusion. CO in this case acted as a carbon monoxide stress test. The third case was a patient with CO poisoning and a NSTEMI on EKG. Angioplasty only showed 30% occlusion, so the patient's presentation is likely due to direct CO cardiac toxicity.
Conclusions: These cases demonstrate the varied presentations that CO poisoning can have on patients with underlying heart disease. Clinicians must keep these effects in mind when treating patients presenting with CO poisoning.

TREATMENT OF CO INTOXICATION DUE TO SHISHA CONSUMPTION: A TREND AND CAUSE ANALYSIS

David Eichhorn, Johannes Schneppendahl, Martin Hoffmanns, Hartmut Strelow, Joachim Windolf, **Sven Dreyer**
Trauma and Hand Surgery, Hyperbaric Medicine, University Hospital Duesseldorf, Germany

Introduction: The proportion of cigarette-consuming adolescents (12-17yrs) in Germany has declined significantly in recent years (2001: 27.5% - 2015: 7.8%). This is in contrast to an increase in smoking waterpipe. One third of adolescents in Germany have used waterpipes. The health consequences are systematically underestimated. Consequence is an increasing number of young patients in German emergency rooms presenting a range of neurological symptoms, as a result of CO intoxication.
Method: To analyse the perceived trend of an increasing incidence of CO intoxication requiring treatment due to shisha consumption and to put it into context to other indications for emergency HBO therapy, all patients with carbon monoxide poisoning admitted to our emergency department from January 2010 to December 2018 were included in this study. The emergency admission protocols and medical reports of around 600 patients were evaluated regarding the sociodemographic data, intoxication causes and symptoms on the basis of a study protocol. Beginning 2018, contextual factors and symptoms were recorded systematically.
Results: Nearly one third of emergency HBO treatments for CO intoxication in 2017/2018 were caused by waterpipe (Fig. 1). CO Hb averaged 20%, and correlated positively (r = .381, sig .000) with age (average was 22 years, sex 50% m/f). Neurological symptoms were observed in 80.4% of patients, with no correlation to CO-Hb value. In the interviews conducted so far, the majority of responders smoked waterpipe more than thrice a week.
Conclusion: The significant increase in CO intoxication caused by waterpipe is worrying, especially in regards to the uncertain impact of HBO treatment on neurological sequelae. Also, context and risk factors are yet unknown and need further investigation. With regard to the rapidly increasing incidence, an increased awareness for and discussion of the risks and side effects of smoking waterpipe is desirable.


Waterpipe
Indoor Barbeque
Defect Gas Heating
Suicide
Fire
Charcoal Burner
others
Number of Cases
50
40
30
20
10
0
2010
2011
2012
2013
2014
2015
2016
2017
2018
Year

Posters

DECOMPRESSION ACCIDENT AND CLARKSON-LIKE SYNDROME: MEDICAL CARE AND REVIEW/PRE

Jean Morin[1], Kim Simon[2], Fabien Chadelaud[2], David Delarbre[3], Arnaud Druelle[1], Jean Eric Blatteau[1]
[1]hyperbaric Medicine, Hia Ste Anne, France [2]icu, Hia Ste Anne, France [3]medicine, Hia Ste Anne, France

Background: Capillary leak syndrome or Clarkson-like is a rare type of decompression sickness that may be responsible for hypovolemic shock with edema.
Clinical case: a young recreational diver had a labyrinthine DCS after a 96 ms air dive. He had severe hypovolemia and facial edema secondary to capillary leak syndrome.
Discussion: In DCS, the formation of bubbles alters the wall of blood vessels and activates complex biochemical mechanisms inducing extravascular protein leakage. This pathology is expressed from simple hemoconcentration to hypovolemic shock with multiorgan failure to death. The presence of high hematocrit with or without hypoalbuminemia in DCS requires enhanced clinical and laboratory monitoring. Prevention of vascular collapse involves early vascular filling with albumin infusion to improve the prognosis of his patients/pre

ENDOSCOPIC BUBBLE TROUBLE: HYPERBARIC OXYGEN THERAPY FOR CEREBRAL GAS EMBOLISM DURING UPPER ENDOSCOPY- CASE REPORTS AND LITERATURE REVIEW

Jeffrey Cooper
Emergency Medicine, University of Nebraska Medical Center, USA

Gas embolism is a rare but potentially devastating complication of endoscopic procedures. We describe three cases of gas embolism which were associated with endoscopic procedures (esophagogastroduodenoscopy (EGD) and endoscopic retrograde cholangiopancreatography (ERCP). We treated these at our hyperbaric medicine center with three different outcomes: complete resolution, death and disability. We review the literature regarding this unusual complication of endoscopy and discuss the need for prompt identification and referral for hyperbaric oxygen therapy (HBO). Prevention, mitigation, mechanisms, diagnosis and additional adjunctive therapies are also discussed.

INVESTIGATION OF ORAL MICROBIOME IN COMMERCIAL SATURATION DIVING

Roxane Monnoyer, Jacky Lautridou, Kjersti Haugum, Ingrid Eftedal
Department of circulation and medical imaging, Norwegian University of Sciences and Technology, Norway

Background: During commercial saturation diving operations, divers live and work in hyperbaric and hyperoxic conditions. Preserving health and physiological balances in such harsh environments requires successful acclimation; this applies equally to human cells and to the myriads of bacterial species that live in/on the human body. This study is the first to examine the activity and composition of bacteria residing in the oral cavity of commercial saturation divers, the so-called oral microbiome. With the emerging awareness of the associations between human health and the state of our microbiomes, we aim to add further knowledge of the impact of saturation diving on diver's physiology using omics techniques and without any a priori bias.
Methods: Information and saliva samples from thirty commercial saturation divers working offshore have been collected at four different times; at surface (baseline), twice during saturation (at 190-200 meters of sea water), and after decompression. The composition of the oral microbiome will be described by sequencing of highly conserved bacterial regions (16S ribosomal RNA), followed by similarity clustering. Bioinformatic analysis will be used to study dynamic gene expression profiles of the identified microbial species.
Results: No results are yet described.
Conclusion: Once a status for the composition and activity of the oral microbiome during and after saturation diving is established, these data can be used to further assess the interplay between health and fitness, and the bacteria residing on the diver's body. Consequently, the study of the oral microbiome in divers would potentially highlight hypothetical relations with the diver's physiology during hyperbaric and hyperoxic exposure.

INVESTIGATION OF PARACENTESIS FOR THE AVOIDANCE OF BAROTRAUMA DURING HYPERBARIC OXYGEN THERAPY

Katharina Schaumann[2], Lena Colter[2], **Sven Dreyer**[1], Hartmut Strelow[1], Joerg Schipper[2], Christian Plettenberg[2]
[1]Trauma and Hand Surgery, Hyperbaric Medicine, University Hospital Duesseldorf, Germany [2]Otorhinolaryngology, University Hospital Duesseldorf, Germany

Introduction: In 14-43% of hyperbaric therapy sessions a barotrauma is developed. Patients with artificial airway, I.e. Tracheotomy or tubal dysfunction have an increased risk of developing a barotrauma (BT). Prediction of barotrauma is difficult. There is no published data for the prophylactic use of therapeutic options, e.g. Paracentesis. For this reason, we asked ourselves, if the use of pc in tubal dysfunction or in patients undergoing therapy intubated is useful in the prevention of BT.
Method In a retrospective analysis all Patients undergoing emergency hbot in the period from January until December 2017 were recorded with regard to history, otoscopy, valsava maneuver, tympanogram before hbot and after the onset of BT.
Results: In total, 149 patients received HBOT during the stated period. 23 patients received a PC (15%), 19 of them prophylactically and 4 after the onset of bt. 10 of the 19 prophylactic PC patients were intubated. In 9 patients clinical examination led to the indication of pc; 9/9 had negative valsava, 3/9 additionally a flat tympanogram and 2/9 had conspicuous otoscopy findings. 4 patients had BT despite inconspicuous ENT examination (2,6%), 2 patients had a history of tubal dysfunction. 6 Patients tolerated HBOT despite negative valsava.
Conclusion: PC carried out prophylactically seems to be an effective way to reduce the incidence of BT in patients with missing pressure compensation. Non the less I appears reasonable to find further criteria eg. Scores (in order ?)To avoid bt or unnecessary surgery respectively.

HYPERBARIC OXYGEN THERAPY FOR THE TREATMENT OF PERIANAL FISTULAS IN CROHN`S DISEASE (HOT-TOPIC TRIAL): PRELIMINARY RESULTS

Krisztina B Gecse, **Nina Lansdorp**[1], Krisztina B Gecse[2], Christianne J Buskens[3], Mark Löwenberg[2], Jaap Stoker[4], Willem A Bemelman[3], Geert RAM D'Haens[2], Rob A van Hulst[1]

[1]Anesthesiology/Hyperbaric Medicine, Amsterdam UMC, University of Amsterdam, location AMC, Netherlands [2]Gastroenterology, Amsterdam UMC, University of Amsterdam, location AMC, Netherlands [3]Surgery, Amsterdam UMC, University of Amsterdam, location AMC, Netherlands [4]Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, location AMC, Netherlands

Background: Positive effects of hyperbaric oxygen (HBO) therapy for perianal fistulizing Crohn's disease (pcd) have been suggested in previous publications. The HOT-TOPIC study was designed to further investigate its feasibility and therapeutic effect in 20 patients. Here we present the preliminary results.

Methods: 9 patients with pcd refractory to standard-therapy 6 months were treated with 40 sessions of HBO therapy (243-253 kpa, 110 minutes). Seton drain(s) were removed after 30 treatments. Primary endpoints were changes in the perianal disease activity index (PDAI) and MRI-scores (modified van Assche score) 2 months after treatment. Secondary outcomes were fistula drainage assessment (FDA), laboratory findings and patient-reported outcomes.

Results: 4 male and 5 female patients (median age 34 years) were treated. Median PDAI scores decreased from 9 to 4 (p=0.012) and MRI scores from 9.4 to 7.3 (p = 0.027). Of the 24 external openings draining at baseline, 10 were closed after treatment (assessed by FDA). Two patients, both with one external opening at baseline, had no remaining openings after treatment. C-Reactive Protein (CRP) and fecal calprotectin were increased (5 ml/L and 50 mg/kg, respectively) in 5 and 8 patients before HBO, and 2 and 3 patients after HBO, respectively. Median scores of the inflammatory bowel disease questionnaire (IBDQ) increased from 169 to 185 (p=0.008) and VAS scores from the Euroqol-5-dimensions questionnaire increased from 71 to 75 (p=0.058), higher scores reflecting better qol. When asked on a validated decision regret scale if patients regretted their decision to undergo HBO, the mean score was 10 (0-100, higher scores indicating higher regret).

Six patients experienced trouble equalizing inner ear pressure, with three patients needing tympanostomy tubes. No other clinically relevant adverse events occurred.

Conclusions: Based on preliminary data, HBO treatment is associated with significant improvement in pcd, as measured by clinical and MRI endpoints.

SUCCESSFUL TREATMENT OF RADIATION-INDUCED COLITIS AND ENTERITIS WITH HYPERBARIC OXYGEN THERAPY

Jeffrey Cooper[2], Mark Ringle[1]
[1]College of Medicine, University of Nebraska, USA [2]Emergency Medicine, University of Nebraska, USA

Background: Most radiation induced enteritis and colitis treatment is directed at providing symptomatic rather than in treating the cause of the symptoms. One systematic literature review noted 66 of 74 publications reporting positive results in the treatment and prevention of an assortment of radiation induced injuries with hyperbaric oxygen therapy (HBO2). A series in 2007 had 67% of patients with chronic gastrointestinal radiation injury treated with HBO2 improving. We report on two cases highlighting the effectiveness of HBO2 for radiation enteritis and colitis.
Case Reports: A 70-year-old female treated for cervical cancer with hysterectomy with adjuvant radiation at age 32 developed radiation induced sigmoid stricture with episodes of diarrhea and incontinence. Colonoscopy showed severe narrowing (a pediatric scope could not pass through) of the sigmoid colon as well as granulomatous and erythematous mucosa. After 40 HBO2 sessions she noted marked improvement with fewer and firmer stools, no tenesmus or urgency. Endoscopy showed the stricture relieved to the extent that an adult scope was readily passed.
A 45-year-old female treated for endometrial cancer with hysterectomy, bilateral salpingectomy, chemotherapy and radiation. Nine months thereafter, she developed small bowel obstruction with dehydration and malnutrition. She required total parenteral nutrition (TPN), bowel rest and naso-gastric tube decompression. She underwent 31 HBO2 treatments without complications. Although still needing surgery for lysis of adhesions and ileocolic resection, she had good resolution of symptoms following surgery without need for colostomy.
Conclusions: These cases reaffirm the use of HBO2 in radiation induced enteritis and colitis allowing patients to return to an improved level of functioning, avoiding surgery, decreasing pain and other symptoms. It should be routinely considered in the treatment of radiation induced enteritis or colitis.

HIGH-PRESSURE RECOMPRESSION THERAPY FOR DECOMPRESSION ILLNESS

Johannes Schneppendahl, Stephan Tanner, Fabian Seiler, Hartmut Strelow, Joachim Windolf, Sven Dreyer
Trauma and Hand Surgery, Division of Hyperbaric Oxygen Treatment, University of Düsseldorf, Germany

Backgraound: Hyperbaric oxygen therapy for decompression illness (DCI) most commonly includes recompression at 2.8 ATA. However, remaining symptoms are seen frequently.
Methods: We introduced a treatment with initial recompression to 6 ATM, followed by a gradual decompression followed by a minimum of 2 following sessions at 2.8 ATM. Of all 64 patients treated from 2001 to 2010 charts were reviewed and a follow-up evaluation including a standardized questionnaire including a Short Form 36 Health Survey (SF-36) to rate the quality of life before and after the incident.
Results: Mean age of 21 female and 41 male patients was 37.7 (±9) years. The average diving depth was 24 (±11,5) meters and the duration 46.4 (±48.7) minutes. Presentation was later than 48h after the accident in 72,3% and none of the patients suffered any significant adverse effects during the treatment. Of the 27 patients returning the questionnaire 18 (66,7%) were free of symptoms whereas 9 (33,3%) reported of persisting symptoms, such as numbness, tingling, coordination deficit or hearing loss. Overall 85,2% where back to work within one month. According to the SF-36 overall no significant deterioration in the health status was observed, however patients with persisting symptoms showed a deterioration in physical and emotional health. Neither gender, duration between accident and HBO, nor severity of initial symptoms had an impact on persisting symptoms or result of the SF-36.
Conclusions: While little evidence for the use for higher compression than 2.8 ATM is available treatment tables for AGE consisting of recompression to an ambient pressure of 6 ATM, such as USN table 6a, have been introduced. In this study we display good clinical results and a low complication rate using an alternative treatment regimen consisting of high-pressure recompression therapy.

NARCOTIC N_2 EFFECTS AFTER A SIMULATED DEEP DIVE

Jochen Schipke[1], Thomas Muth[2], Sven Dreyer[3]
[1]Research Group Experimental Surgery, University Hospital Düsseldorf, Germany
[2]Institut für Arbeits-, Sozial- und Umweltmedizin, Heinrich Heine University, Germany
[3]Medical Process Organization, Heinrich Heine University, Germany

Background: Scuba divers breathing air are sooner or later confronted with the nitrogen (N_2) narcosis when diving deeper than 40m. Frequently, it is overseen that narcotic N_2 symptoms progressively worsen from depths 10m at the latest, i.e. Cognitive and fine motor skills start to be compromised. Conversely, narcotic symptoms have not disappeared after surfacing. Aim of this retrospective study: Display the effects of oxygen (O_2) breathing before surfacing after a simulated deep dive.
Volunteers/Methods: Volunteers performed a dive in the pressure chamber (50m; 5min bottom time). 58 divers (air-group) were decompressed according to the dive tables. 28 other divers (O_2-group) breathed 100% O_2 while decompressing. Before and after the dives, all participants performed the Romberg and the sharpened Romberg test (R; SR; sense of balance) and a tweezers test (fine motor skills; picking beads).
Results: SR resulted always in more positive participants than R (data not shown). In the air-group SR positives equaled to 53% before and to 67% after the dive equivalent to a 14% increase. The number of beads amounted to 42±10 (before) and 41±8 (after). In the O2-group SR positives equaled to 68% both before and after the dive. The number of beads amounted to 36±7 (before) and 42±9 (after) after the dives.
Discussion/Conclusion: As N_2 does not abruptly leave the tissue, narcotic symptoms will persist after surfacing. In the air-group, SR showed a slight adverse N_2 effect. In the O2-group, the number of SR positives did not differ between pre- and post-dive. To our surprise, the number of beads had increased after the dive possibly via the learning effect. Such improvement was absent in the air group presumably due to persisting N_2 effects. We confirm both post-dive persistence of narcotic N_2 effects that could be avoided by O_2 breathing during decompression.

A UNIQUE CLASSIFICATION SYSTEM OF MEDICAL PROBLEMS OF DIVING

Michael Strauss[1], Lientra Lu[2], Stuart Miller[1], Igor Aksenov[3]
[1]Hyperbaric Medicine Program, Memorial Care, Long Beach Medical Center, USA
[2]Research, Tibor Rubin Veterans Affair Medical Center, USA [3]Internal Medicine, Critical Care, Memorial Health University Medical Center, USA

Background: A cohesive classification of medical problems of diving (mpds) does not exist. To classify mpds in a logical fashion, we have generated a stress-stimulus/reaction-Response system.
Methods: Diving subjects the human body to stresses. Almost all are resolved spontaneously and often without conscious effort. In evaluating and managing mpds over a 60-year interval, we have generated a classification system that specifies four stress types that can account for almost every MPD.
Results: The four stress types and their related disorders include:

- PHYSICAL STRESSES such as ear barotrauma and arterial gas embolism explained by Boyle's law, as well as other physical challenges such as environmental (thermal, wave action, etc.) And injuries from marine animals.
- PHYSIOLOGICAL STRESSES where the body tissues interact with the stress causes. There are explained by Henry's and Dalton's laws and include decompression sickness, nitrogen narcosis, oxygen toxicity, carbon dioxide toxicity, and carbon monoxide poisoning.
- PSYCHOLOGICAL STRESSES_which are related to loss of control and include panic syndrome and disorientation challenge
- UNRECOGNIZED STRESSES where stimuli to breathe are obviated by alterations in the recognition of hypoxia by the body. This group includes breath-holding blackouts after hyperventilation, distractional and diffusional blackouts and dilutional (associated with closed-circuit rebreather diving) blackout causes of loss of consciousness in the water

Conclusions:
Our classification system based on stress recognition in diving provides a logical approach to teaching diving medicine and the prevention of mpds.

THE CONCEPT OF QUALITY MANAGEMENT IN HYPERBARIC AND DIVING UNITS

Angeliki Chandrinou[1], Konstantina Gaitanou[1], Konstantinos Exarchos[1], Maria Malliarou[2], George Fildisis[1]
[1]Hyperbaric Medicine Department, National and Kapodistrian University of Athens, Greece [2]General Department, University of Thessaly, Greece

Introduction: The concept of quality in all services is ambiguous, much more for the Hyperbaric and Diving Units where a variety of services such as the effective use of hyperbaric recompression chamber require proper coordination and organization. Quality management in Hyperbaric and Diving Units can be implemented by setting quality standards and monitoring their use. The aim is to underline the need for quality management and quality assurance in Hyperbaric and Diving Units and to declare what ebass has recognized as of much importance that all personnel working with HBOT must be well-trained and independently certified.
Material – method: It is a review that highlights the benefits of quality management in Hyperbaric and Diving Units.
Results: The benefits a Hyperbaric and Diving Unit derives from applying standards are immense, with some of them to be: a) optimization of the provided health services b) minimization of likelihood of occurrence of adverse events c) increase of satisfaction of patients, relatives d) increase of productivity and reduce of operating costs e) improvement of working conditions and easier integration of new staff. The documentation and quality assurance system of a Hyperbaric and Diving Unit includes Quality Manual documentation, Implementation of Standard Operating Procedures and Guidelines as foreseen and described in the ECHM 2004 European Code of Good Practice on Hyperbaric Oxygen Therapy and The ebass/ECHM Resources manual. The ebass/ECHM Resources manual is intended to be a reference document for European countries for guidelines, regulations and standards in hyperbaric medicine.
Conclusions: Baromedical personnel in all European Baromedical centers should follow standardization of education in order to support and a safe approach in daily practice.

VALIDATION OF A PLACEBO GROUP IN HYPERBARIC

Pierre Louge, Marie-Anne Magnan, Michel Pellegrini, Marc-Alain Panchard, Claude Lae, Rodrigue Pignel
Hyperbaric Center, Geneve University Hospital, Switzerland

Background: As part of a double-blind placebo controlled randomized controlled trial of hyperbaric oxygen therapy in the treatment of vaso-occlusive crisis in sickle cell patients over 8 years of age at Geneva University Hospitals, we decided to evaluate which profile of compression at shallow depth best simulated a therapeutic table.
Method: this study was conducted with healthy volunteers (medical and paramedical staff of Geneva University Hospitals). Each volunteer was randomized according to 3 types of air compression (1, 3 and 5 meters). After the compression, the volunteer had to answer if it seemed to him to have been compressed or not.
Results and discussion: 20 volunteers were included in each arm. The negative response rate was significantly higher in the 1 meter arm than in the other 2 arms (64% vs 36% and 26%, p=0.048, Fisher test). There was no difference between compressions at 3 or 5 meters.To improve the blindness we have, in a second time, decreased the compression time by 2 from 10 `to 5` to increase the sensation of pressure variation, with the addition of a forced ventilation and heating during the 10 `of fictional compression. During decompression forced ventilation and cooling of the enclosure was performed. 22 new volunteers were included with a negative response rate of 4.5%.
Conclusion: A compression at 3 meters associated with confounding elements such as forced ventilation and enclosure heating simulates a therapeutic recompression table and can easily be used as a hyperbaric placebo. It has the advantage of an extremely low risk and a pio2 of 0.27, which has little effect on the oxygen effect.

HYPERBARIC OXIGENATION IN THE TREATMENT OF LEGG-CALVÉ-PERTHES DISEASE

Nadia Hamzeh[1], Jordi Desola[1], Solerme Morales[2]
[1]CRIS-UTH, the Hyperbaric Therapy Unit of Barcelona. Moises Broggi Hospital, Spain
[2]Auxiliary Consultant Professor of Physiology, Hermanos Ameijeiras Hospital, Cuba

Background: Legg-Calvé-Perthes disease (PD) is an idiopathic childhood aseptic necrosis of the femoral epiphysis causing ankylosis and serious handicapping dysfunction, requiring agressive surgical intervention. Social impact is very high. The world`s largest experience comes from Cuba, where PD is an accepted indication for HBO.
Objective: To describe the experience of CRIS-UTH in the application of HBO in children suffering from PD.
Methods: A retrospective descriptive clinical analysis of patients diagnosed of PD that received HBO in CRIS-UTH during the period 2005-2012. Outcome validation in the short term was based on radiographic signs of bone regeneration and improvement of functional status. In the middle and long term, it was ascertained by patients, familial reports, and phone interviews.
Results: Five males and 1 female. Age: 8.6 ± 2.16 (6-12) y.o. Hbot regime : 62.3 ± 38.08 (20-116) sessions of 90 minutes at 2.3 ATA. Elapsed time between PD diagnose and hbot : 12.5 ± 6.50 (5-24) months. Bone regeneration was observed in 5 patients who did not need mobility aids at less than 2 years after beginning hbot. Surgical interventions were avoided in 4 patients. Two patients discharged crutches/wheelchairs during the 1st year. One of them experienced a complete reossification. A normal functional status was reached and maintained in the 5 patients in the 7-14 years term.
Conclusions: The highest final severity of the disease was related to the delay in application of hbot that dramatically improved the functional recovery in 5 of our 6 paediatric patients affected of Perthes disease.

ECTOPIC GAS DUE TO ACCIDENTAL HYDROGEN PEROXIDE INGESTION TREATED WITH HYPERBARIC OXYGEN THERAPY

Jayanth Adusumalli, Jesse Higgins, Nathan Brytowski, Gary Toups
Department of Preventive, Occupational, and Aerospace Medicine, Mayo Clinic, USA

Background: Hydrogen peroxide poisoning can be serious and possibly fatal depending the amount and strength ingested. We present a case where the effects of hydrogen peroxide ingestion with treated with hyperbaric oxygen therapy.
A 51 year old farmer accidentally ingested 2 cups of 35% hydrogen peroxide mistaking it for water. He kept it at home for cleaning farm equipment. He had acute onset of pain in his upper chest and epigastric region. This was followed by nausea, 1 episode of vomiting, generalized body aches and fatigue. He presented to an outside medical care facility where CT imaging revealed portal venous gas, gastric emphysema and pneumo-mediastinum. He was transferred to our facility to receive a higher level of care.
Methods: On evaluation, he did not exhibit any signs or symptoms of neurological or cardiac compromise. He also did not have any signs or symptoms suggestive of peritonitis. Vitals were stable.
Given the large amount of gas in the portal venous system, and the potential of an arterial gas embolism, it was decided to treat him with U.S. Navy treatment table 6.
He underwent treatment in a multiplace chamber for 4 hours, 58 minutes without any extensions. The inside attendant was an intensive care unit registered nurse with hyperbaric Medicine experience.
Results: Repeat imaging showed resolution of portal venous gas. There was improvement in the gastric emphysema and an improvement in the pneumomediastinum. However imaging also revealed worsened mural edema, consistent with severe gastritis.
A CT with contrast, IV proton pump inhibitor therapy, upper endoscopy, a swallow study and continued monitoring was planned. However the patient left against medical advice, and was lost to follow-up.
Conclusions: -hydrogen peroxide ingestion can cause venous gas embolism with the potential for arterial gas embolism
-hyperbaric oxygen therapy is recommended emergently for the same.

EUROPEAN CERTIFIED HYPERBARIC SAFETY MANAGER - ACCREDITATION OF TRAINING CENTERS

Angeliki Chandrinou[1], Peter Atkey[2], Konstantina Gaitanou[1], Miguel Parceiro[3], Manuel Preto[4], Damianos Tzavellas[1]
[1]EBAss, European Baromedical Association, Greece [2]EBAss, European Baromedical Association, UK [3]EBAss, European Baromedical Association, Switzerland [4]EBAss, European Baromedical Association, Portugal

Introduction/ Background: There is a need to develop a formal, standardized management team within European hyperbaric facilities and safety should be a major part of this team for any Hyperbaric Unit. The medical director should have a competent individual as part of this team who can be relied on to ensure the safety of the hyperbaric facility.
Methods/ Materials: This is a presentation of the certification module of training centres for European Certified Hyperbaric Safety Managers (ECHSM).
Results: European Baromedical Association (ebass) and the European Committee for Hyperbaric Medicine (ECHSM) are working together with the European College of Baromedicine (ECB) to ensure that centres wishing to conduct training for ECHSM at the required standards, can easily achieve accreditation for their courses. The lecturers should be of level 1 (Outline Knowledge) or Level 2 (Knowledge) depending on the topic (medical or technical) as well as recognized members of an HBO team (e.g. Chamber operator, nurse). For Level 3 (Detailed Knowledge) on medical topics they will be a hyperbaric physician or, under the responsibilities of the hyperbaric physician, a registered nurse specialized on HBO. For Level 3 on technical and safety topics, can be a safety manager or a medical director (ECHM level II or equivalent). The ECHSM examination will be in theory and practice. Training courses can be accepted by satisfying required criteria, ultimately gaining accreditation from the ECB.
Conclusions: Training centers can conduct training for ECHSM, following the ebass/ECHM/ECB scheme thus the candidate will gain in addition to the basic hyperbaric education (ECB Certification, ECHCO, ECHRN) a specific and recognized, advanced certification. The Safety Manager will be appointed by the Medical Director.

RIGHT-TO-LEFT SHUNT IN DECOMPRESSION ILLNESS: CONSECUTIVE SERIES OF 414 SCUBA DIVERS

Gian Paolo Anzola[1], Clara Bartolaminelli[2], Paolo Lega[3], Paolo Limoni[4] and Pasquale Longobardi[5]

[1]Consultant Neurologist Villa Gemma Hospital Gardone Riviera Italy, [2]Emergency Department Montichiari Hospital - Asst Degli Spedali Civili Di Brescia, Italy, [3]Consultant Anesthesiologist at Hyperbaric Centre Ravenna, Italy, [4]Consultant Neurosonologist, Neurologist at Hyperbaric Centre Ravenna, Italy [5]Chief Medical Director of Hyperbaric Centre Ravenna, President of SIMSI (Italian Diving and Hyperbaric Medical Society)

Introduction and Method: The presence of a Right-to-Left Shunt (RLS), most commonly produced by a patent foramen ovale, has been correlated to Decompression Illness (DI) in scuba divers and it is suspected to increase the risk of suffering diving accidents. In the present study, RLS was comparatively investigated in a consecutive series of 414 divers with (DI+) and without (DI-) DI and in 119 healthy control subjects. All cases were submitted to Transcranial Doppler (TCD) examination. The presence of RLS was confirmed by pulmonary scintigraphy and / or echocardiographic tests if the RLS as assessed with TCD was large.

Results: The frequency of DI was the same in recreational and professional, but higher in female than in male divers. DI+ divers were significantly older than DI- divers (46 ± 10 vs. 43 ± 11 year old respectively, $p = 0.047$). Large shunts were statistically more frequent in DI+ (68%) than in DI- (47%) divers, in DI+ than in controls (18%) and in DI- than controls. Small shunts were equally represented in DI+ (14%), DI- (17%) and controls (18%). The presence of a large shunt, female sex, number of dives per year and age turned independent predictors of DI.

REAL WORLD THORACIC ECHOCARDIOGRAPHY FOR THE ASSESSMENT OF RIGHT-TO-LEFT SHUNT: EXPERIENCE FROM SCUBA DIVERS

Gian Paolo Anzola[1], Clara Bartolaminelli[2], Paolo Lega[3], Paolo Limoni[4] and Pasquale Longobardi[5]

[1]Consultant Neurologist Villa Gemma Hospital Gardone Riviera Italy, [2]Emergency Department Montichiari Hospital - Asst Degli Spedali Civili Di Brescia, Italy, [3]Consultant Anesthesiologist at Hyperbaric Centre Ravenna, Italy, [4]Consultant Neurosonologist, Neurologist at Hyperbaric Centre Ravenna, Italy [5]Chief Medical Director of Hyperbaric Centre Ravenna, President of SIMSI (Italian Diving and Hyperbaric Medical Society)

Introduction and Method: The presence of a Right-to-Left Shunt (RLS), most commonly produced by a patient foramen ovale, has been correlated to Decompression Illness (DI) in scuba divers and is suspected to increase the risk of suffering diving accidents. It is therefore mandatory to ensure a proper detection of RLS in divers who have suffered a DI and also in asymptomatic subjects who are being assessed for the fitness to diving. Transthoracic Ecocardiography (TTE) is commonly used to assess RLS, but its sensitivity is probably less than optimal. Therefore we compared the performance of TTE for identifying RLS in consecutive 141 divers studied at the Hyperbaric Centre of Ravenna where the protocol for assessing the fitness to diving includes a standardized contrast enhanced transcranial Doppel (TCD).

Results: Overall, RLS was identified by TCD in 117 subjects, whereas TTE was positive for RLS in 70. In the 117 subject with RLS detected by TCD the shunt was large in 104 and small in 13. TTE missed 52 out of 117 shunts (5/13 small and 47/104 large), whereas TCD missed 5 of the 70 cases deemed positive by TTE.

Conclusion: Compared with standardized contrast enhanced TCD, the performance of real world TTE is insufficient to guarantee a reliably accurate identification of RLS in scuba divers. This will have to be taken into account when assessing the risk of diving in asymptomatic subjects as well as in survivors of DI.

HBOT IN TRAUMATIC BRAIN INJURY, A CASE STUDY

Andrea Galvani, Pasquale Longobardi
Centro Iperbarico / Centro Cura Ferite Difficili di Ravenna, Centro iperbarico di Ravenna, Italy

Introduction: In august 2016, at the Ravenna Centre, we visited a patient who had suffered a severe head injury occurred at home in 2013 with immediate subcortical hemorrhage and caused visual impairment, ataxia, balance disorder and dysarthria. The patient underwent emergency surgerty act and 2-year-long rehabilitation program with a slight clinical improvement. We treated with a first round of HBOT at lower p02 pressure (20 sessions at 193 kpa with mask Fi0290% - average – 80 minute-sessions, 5 session/week). A functional MRI was performed together with a physiatric assessment conducted before starting the HBOT cycle and one year after treatment. Furthermore, during treatment the patient attended regular rehabilitation sessions both physical (FKT) and specific for vision impairment (orthoptist).
Results: Both the functional MRI and the physiatric check-up performed after a little over 12 months from the end of the first HBOT cycle resulted in a significant improvement in the functional radiological frame and in the clinical picture. The patient's quality of life also improved significantly, being able to go back to University and take his exam autonomously.
Discussion: There is some evidence in literature regarding the benefits of HBOT in (carefully selected) patients affected by severe neurological damage derived from a head injury. More specifically, the case presented shows how an integrated approach (drug therapy + rehabilitation treatment + HBOT) could accelerate the results (measurable through functional MRI) and improve the clinical outcome at a positive risk-benefit balance.

CALCIFIC UREMIC ARTERIOLOPATHY (CUA) TREATED BY MULTIMODAL APPROACH INCLUDING HBOT: A CASE REPORT

Andrea Galvani, Pasquale Longobardi
Centro Iperbarico / Centro Cura Ferite Difficili di Ravenna, Centro iperbarico di Ravenna, Italy

Introduction: Calciphylaxis, or Calcific Uremic Arteriolopathy (CUA), refers to a clinical condition that starts with painful skin plaques due to the calcification of blood vessels of the skin involving in progressive, painful skin wounds. It is estimated an incidence rate between 1:1.000 and 1:5.000 cases per year within dialysis patients. The mortality is up to 80% mainly due to the severe sepsis.
Methods: In March 2019, Nephrology Unit of the Ferrara Hospital sent to the Hyperbaric Centre of Ravenna a patient (40 yrs) undergoing a triweekly hemodialysis therapy. She arrived with severe multifocal hard to heal, painful, CUA lesions (large abdominal lesion, lower limbs lesions, lumbar lesions). The blood exams showed altered CRP and PCT. The patient was treated by surgical debridement of the necrotic tissue, HBOT (30 sessions at 253 kpa, 90 minutes every other day she was not undergoing dialysis), dressings, Negative Pressure Wound Therapy, and drugs. The patient underwent weekly clinical checkups, medication and blood exams.
Results And Conclusion: There was a significant improvement of the severe CUA lesions. The blood chemistry indexes (CRP, PTC) constantly improved. The patient reported an improvement of her general conditions and a less painful state.
Therapeutic approaches in patients with calciphylaxis are limited. The available amount of data is limited as weel, and no prospective data is available. We suggest that a multimodal approach should be considered when CUA lesions are diagnosticate: surgical treatment, drug therapy, dressings and HBOT.
As stated in the literature, HBOT has an interesting therapeutic option (the recovery of normal values of tissue po2 improves and enhances the tissue repair). Furthermore it has well-known anti-inflammatory, neo-angiogenetic properties, the direct and indirect antibacterial action. Although, the biological basis and the clinical-therapeutic rationale should be further investigated through robust research and clinical studies, the clinal case presented is encouraging.

HYPERBARIC OXYGEN THERAPY IN IMPROVING HEARING LOSS IN IDIOPATHIC SUDDEN SENSORINEURAL HEARING LOSS

Abhishek Srivastava, Faiz Mohammed, Navita Purohit
Physical Medicine & Rehabilitation, Kokilaben Dhirubhai Ambani Hospital, India

Objective: To assess the role of adjunctive hyperbaric oxygen therapy (HBOT) in improving hearing in Idiopathic Sudden Sensorineural Hearing loss (ISSNHL)?
Material and Methods: Retrospective analysis of patients who have undergone HBOT for ISSNHL from January 18- March 19. The patients received HBOT according to the brain protocol of 1.5 ATA for 60mts each session, 6 days/week for 2 weeks for total 12 sessions. All patients received steroids and antiviral drugs as per the Otolaryngologist opinion. Assessment of improvement in hearing was done by Pure Tone Audiometry before and after HBOT. Pure tone average is taken at 500Hz, 1000Hz, 2000Hz and 4000Hz. Patients degree of recovery was classified into four groups according to Siegel's method into complete recovery, partial recovery, slight recovery and no recovery based on post treatment improvement in final hearing abilities as tested on audiometry.
Results: Total 18 patients (M:F:13:5), age 30-70 years (mean 45.6 years), post hearing loss duration 2-20 days (mean 8.6 days) were included in the study. 63 % of patients had improvements in their hearing abilities after treatment. 27% patients had complete recovery, 18% had had partial recovery, another 18% had slight recovery, and 36% had no recovery. Those who took treatment within the 1st few days had higher chances of improvement. Younger patients had better recovery.
Conclusion: HBOT can be used along with conventional treatment to improve hearing in SNHL especially in the younger population. It should be started as early as possible for maximum benefits.

A FULLY AUTOMATED METHOD FOR LATE VENTRICULAR DIASTOLE FRAME SELECTION IN POST-DIVE ECHOCARDIOGRAPHY WITHOUT ECG GATING

David Le[1], Eric Markley[1], Peter Germonpré[2], Costantino Balestra[3,4], Frauke Tillmans[5], Petar J Denoble[5], John J Freiberger[6], Richard E Moon[6], Paul A. Dayton[1], Virginie Papadopoulou[1]

[1]Joint Department of Biomedical Engineering, The University of North Carolina at Chapel Hill & North Carolina State University, USA [2]Center for Hyperbaric Oxygen Therapy, Military Hospital Brussels, Belgium [3]Environmental & Occupational Physiology Laboratory, Haute Ecole Paul Henri Spaak, Belgium [4]European Research Division, Divers Alert Network, Belgium [5]Research Division, Divers Alert Network, USA [6]Center for Hyperbaric Medicine and Environmental Physiology, Duke University, USA

Introduction: In each cardiac cycle of the recording, a frame during late ventricular diastole (LVD) must be selected in order to fully automate the counting of venous gas emboli in post-dive 2D echocardiograms (Germonpré et al., Diving and Hyperbaric Medicine 2014). We have previously shown that this is possible through the minimization of a manually selected region-of-interest's (ROI) intensity (Markley et al. UHMS conf. 2019). Here we assess a revised and fully automated method which does not necessitate any user input.

Methods: First, the frame with the highest average intensity is selected and by a factor of two along each axis. Next, a Gaussian blurring filter is applied followed by Sobel edge-detection. A modified circular Hough transform is then applied to the edge-detected frame to estimate the right ventricle center location and its radius. Finally, a square ROI centered at the predicted center with side length twice the predicted radius is drawn. The remainder of the processing steps are the same as in the previous work (Markley et al., UHMS conf. 2019).

Results: All 20 echocardiography videos from the 2014 publication were analyzed in matlab and the method's sensitivity, specificity and accuracy, together with their 95% confidence intervals, were computed by comparing the results to manual LVD frame selection. From the 7139 frames analyzed, sensitivity was found to be 0.92 [0.88-0.95], specificity 0.99 [0.99-0.99] and overall accuracy 0.99 [0.99-0.99], thus superseding our earlier semi-automated results.

Conclusions: Full automation of LVD frame selection has been established with excellent accuracy.

COMPARISON OF NONLINEAR ULTRASOUND IMAGING STRATEGIES FOR THE DETECTION OF DECOMPRESSION MICROBUBBLES

David Le[1], Maneesha Palakurthi[1], Sotiris Evgenidis[2], Konstantinos Zacharias[2], Ourania Oikonomou[2], Thodoris Karapantsios[2], Paul A. Dayton[1], Virginie Papadopoulou[1]

[1]Joint Department of Biomedical Engineering, University of North Carolina - Chapel Hill & North Carolina State University, USA [2]Department of Chemistry, Aristotle University of Thessaloniki, Greece

Introduction/Background: Venous gas emboli are theorized to grow from microbubble nuclei during decompression. Presence of microbubbles may serve as a biomarker for assessing decompression strategies and predicting decompression sickness. We hypothesize that nonlinear ultrasound imaging techniques used in clinical contrast-enhanced ultrasound can detect decompression microbubbles smaller than the limit of detection for traditional B-mode ultrasound. We investigate multiple imaging techniques to determine the optimal scheme for detecting decompression microbubbles.

Methods/Materials: Three nonlinear ultrasound imaging schemes were programmed on a Verasonics ultrasound scanner; pulse-inversion (PI), amplitude modulation (AM), and pulse-inversion/amplitude modulation (PIAM). Imaging was performed at 4.5 mhz and 220 kpa.

A tank filled with 2L water was pressurized to 3 atm with nitrogen and allowed to saturate. The saturated water was injected into a water bath through a 600 μm nozzle forming microbubbles. 300 images were acquired of the bubbles with a tissue-mimicking phantom in the imaging window.

The average bubble to tissue signal ratio was compared to the water to tissue signal ratio to determine each technique's sensitivity to microbubbles.

Results: PIAM showed the greatest increase in microbubble signal of the imaging techniques with an increase of 3.62 db whereas PI and AM had a signal change of 0.08 and -0.94 db respectively.

Conclusions: Decompression microbubbles were detectable with an imaging scheme modifying both amplitude and pulse polarity to enhance nonlinear signals from microbubbles while suppressing tissue. With further validation of microbubble detection using nonlinear imaging and optimization of acoustic parameters, we believe this can be a valuable tool in measuring decompression stress.

SUCCESSFUL TREATMENT OF DAPSONE-ASSOCIATED METHEMOGLONINEMIA WITH HYPERBARIC OXYGEN THERAPY RESISTANT TO CONVENTIONAL THERAPIES

Handan Ozturk, Selcen Yüsra Abaylı, İclal Karatop Cesur
Department of Underwater and Hyperbaric Medicine, Ankara Dışkapı Yıldırım Beyazıt Training and Research Hospital, Turkey

Background: Methemoglobinemia is a serious clinical condition with tissue hypoxia. Methemoglobinemia can be congenital, but most commonly results from exposure to an oxidizing chemical such as dapson. The conventional treatments of methemoglobinemia are high flow oxygen and methylene blue therapies.
Methods: We present an 18-year-old female who admitted to emergency room with weakness, dizziness, vision deterioration and palpitation after receiving fifty 50-mg dapsone tablets for her suicide attempt. The patient had diagnosed with methemoglobinemia with a methb level of 34.7mm/Hg. Patient`s symptoms and increased methb levels were resistant to activated charcoal, high flow oxygen, methylene blue and ascorbic acid therapies. So hyperbaric oxygen (HBO) therapy as an alternative treatment was initiated and the clinical and laboratory improvement occurred.
Results: In the patient who did not respond to active charcoal, high flow oxygen, ascorbic acid and recurrent methylene blue treatments during 24-hour follow-up, a significant clinical and laboratory improvement was observed after hyperbaric oxygen therapy.
Conclusion: In this case report, we aimed to discuss alternative treatment modalities in persistent methemoglobinemia patients who do not respond to standard therapies.

CUTANEOUS DECOMPRESSION SICKNESS

Eylem Koca[2], Elif Ebru Ozer[1]
[1]The Department of Underwater and Hyperbaric Medicine, Izmir Bozyaka Education and Research Hospital, Turkey [2]The Department of Underwater and Hyperbaric Medicine, Sakarya University Education and Research Hospital, Turkey

Introduction: Decompression sickness (DCS) is a major complication of diving and may present with various symptoms of varying severity and specificity. Mild symptoms either may remain stable or progress to severe manifestations. Recompression therapy is the main therapy for DCS.
Method: Evaluation of 5 commercial divers who had cutaneous DCS and treated at our department.
Cases: 5 male divers referred skin manifestations of DCS were identified. The average age was 35,2 with a range of 29-41. All 5 commercial divers had symptoms in the skin such as itching, swelling, rash, marbled pattern and had not had any neurological deficit. The findings regarding all divers demonstrated the cutaneous DCS. In 4 cases, the information of repetitive dives in the same day was obtained. Four of the cases were recompressed according to US Navy treatment table 5. After the first recompression treatment, US Navy treatment table 9 was applied for residual symptoms. The treatment was not needed in one case. In all cases, the complete resolution of all symptoms was provided after the recompression treatments. Patent foramen ovale (PFO) was investigated for each diver.
Conclusion: Skin manifestations of DCS, also known as cutaneous DCS, may be a limiting disease or on some occasion lead to more severe manifestations of DCS. In this study, our cases were evaluated as mild DCS with isolated skin findings and recompressed according to US Navy treatment table 5 and 9. If a diver faces any skin change after a dive, DCS should be considered. In mild or moderate DCS with no signs of serious neurological effects, at first, US Navy treatment table 5 is enough to resolve the symptoms.

THE ROLE OF HYPERBARIC OXYGEN THERAPY AS ADJUSTIVE TREATMENT OF INFECTED NEUROMODULATION DEVICES

Evangelos Papoutsidakis[1], Gerard Plans Ahicart[2], Jordi Desola Ala[1]
[1]Hyperbaric Therapy Unit - Moises Broggi Hospital, CRIS-UTH, Spain [2]Neurosurgery Department, University Hospital of Bellvitge, Spain

Background: Implantable deep brain stimulation devices are used to treat neurological and psychiatric diseases such as Parkinson's disease, dystonia, obsessive-compulsive disorder and depression). These devices can become infected requiring device removal and re-implantation for infection resolution, resulting in highest social and economical consequences. Hyperbaric oxygen therapy (HBOT) was previously suggested as an adjuvant valid therapy for these complicated infections.
Methods: We evaluated four patients (3 male, 1 female, aged 58-76 years-old) with infected neuromodulation devices who already had prior hardware removal. Indications for the devices included Parkinson´s disease tremors (n=3) and idiopathic tremor (n=1). The patients received 40 once-daily HBOT sessions in a multiplace Hyperbaric chamber, at 2.3 ATA, in combination with antibiotic treatment. A follow up assessment was performed 6 months after completion of HBOT.
Results: Three of the four patients had complete resolution of their infection, while the remainder required hardware removal 2.5 months after completion of HBOT, due to persistent infection. No adverse effects of HBOT were observed.
Conclusions: HBOT is safe and may be considered as adjustive treatment in patients of infected neuromodulation devices together with antibiotic therapy. Additional studies with a larger number of patients are needed to further evaluate this treatment approach.

HYPERBARIC OXYGEN PRECONDITIONING TO IMPROVE PHYSICAL PERFORMANCE IN AN ENCLOSED ATMOSPHERE - PRELIMINARY RESULTS.

Dror Ofir, Itamar Ish Shalom, Sofia Rozenberg, Mirit Eynan, Dmitry Kazantsev, Ronen Bar
Medical, Israel Naval Medical Institute, Israel

Background: During combat in a built-up area in general, and underground warfare in particular, one of the major concerns is that walls and roofs may collapse, leading to entrapment of warfighters in an enclosed space having limited ventilation. A decline in physical and cognitive function due to the reduction of O_2 levels and increase in CO_2 may be expected within a few hours. Repeated hyperbaric oxygen (HBO) exposure as preconditioning has been found to contribute to both brain and muscle tissue resistance to oxygen shortage due to reduced blood flow (ischemia). However, less is known about the benefits of HBO preconditioning on subsequent exposure to low O_2 in combination with elevated levels of CO_2.
Purpose: To determine whether HBO preconditioning can improve physical performance under hypoxic-hypercapnic conditions.
Methods: Twenty rats performed exercise tests in air and under hypoxic-hypercapnic conditions. In the second phase, rats performed a VO_2max under hypoxia (10%) and hypercapnia (8%) before and after exposure to HBO treatment as preconditioning (no treatment; 2 or 4 HBO sessions).
Results: Rats reached a speed of 20 ± 5.2 m/min on a 15% incline (n=6), whereas under hypoxia and hypercapnia they reached a speed of 17.9 ± 1.9 m/min (n=9; P = 0.28). In the endurance test, rats held out for an average of 24.2 ± 6.5 min in air compared with 7.4 ± 2.0 min in hypoxic-hypercapnic conditions (P 0.05). When we examined the effect of exposure to HBO on VO_2max, we found that 2 and 4 exposures to HBO had no significant effect on maximum performance.
Discussion and conclusions: 1. Exposure of rats to low O_2 and high CO_2 affects mainly sub-maximal physical performance. In the small group of rats evaluated in this investigation, 2–4 HBO preconditioning sessions failed to produce statistical improvement in VO_2max under hypoxic-hypercapnic conditions. A further study will examine the effect of 6 HBO preconditioning sessions on both maximal and sub-maximal physical capacity.

HYPERBARIC OXYGEN THERAPY FOR IDIOPATHIC SUDDEN HEARING LOSS: THE EXPERIENCE OF 401 CASES FROM PEDRO HISPANO HOSPITAL- PORTUGAL

Diogo Abreu Pereira[1], André Carção[1], Luís Guedes[2], Óscar Camacho[3], Tiago Fernandes[3], Abílio Leonardo[1], Delfim Duarte[1], Nuno Oliveira[1]
[1]Otolaryngology, Unidade Local de Saúde de Matosinhos, Portugal [2]Matosinhos Public Health Unit, Unidade Local de Saúde de Matosinhos, Portugal [3]Hyperbaric Medicine, Unidade Local de Saúde de Matosinhos, Portugal

Introduction: Idiopathic sudden deafness is a sensorineural hearing loss (SNHL) greater than 30 db in at least 3 contiguous frequencies in less than 72 hours of unknown cause. Recent evidence suggests hyperbaric oxygen therapy (HBOT) might be used as a treatment.
Objective: Describe our experience using HBOT for idiopathic SNHL.
Material and Methods: Retrospective, observational study. 401 patients diagnosed with SNHL from 2006 to 2017 and treated with HBOT were included in this study. A descriptive and inferential statistical analysis was performed.
Results: The average Pure Tone Average (PTA) was assessed on three occasions: after the diagnose, 81,36 db (SE = 1,26); before beginning HBOT, 77,62 db (SE = 1,28); and after HBOT, 59,38 db (SE = 1,42) . The mean differences observed were: between the initial and pre-HBOT audiogram +3,74 db (95% CI 2,61 - 4,87), between the initial and post-HBOT +21,99 db (95% CI 19,86 – 24,1) and between the pre- and post-HBOT 18,25 db (95% CI 16,25 – 20,24).
The improvements observed were statistically significant (p0,01).
Discussion: A significant hearing improvement after HBOT was observed. As far as we know, this is one of the largest sample sized used for assessing HBOT on idiopathic SNHL. Nonetheless, these results should be taken with caution, since a partial spontaneous recovery might be responsible for part of the improvements. On our sample, many patients were sent for HBOT after complete failure of conventional treatments and this might explain the small recovery before HBOT.
Even though the promising results, the unpredictability of the disease does not allow definite conclusions. Prospective studies are needed to clarify the real value of this treatment.
Conclusion: The results reinforce the idea that HBOT may play an important role in the treatment of patients with idiopathic SNHL.

HYPERBARIC OXYGEN THERAPY FOR CALCIFIC GLOMERULONEPHRITIC ARTERIOLOPATHY (CALCIPHYLAXIS). A CASE SERIES.

Evangelos Papoutsidakis, Olga Enciso, Angeles Zamora, Mar Giménez, Paola Casbas, Jordi Desola
Hyperbaric Therapy Unit of Barcelona.¨Moisses Broggi¨ Hospital, CRIS-UTH, Spain

Background: Glomerulonephritis with small blood vessels calcification (Calciphylaxis) is a rare disease characterized by progressive vascular calcification and cutaneous necrosis with a ~1-4% prevalence and mortality of ~60-80%, in relation to chronic renal failure, and skin superinfections, as the most common complications. Hyperbaric oxygen therapy (hbot) may be a valid adjunctive therapy for such a serious disease that has no other definitive therapy.
Methods: Descriptive, retrospective analysis of the 6 patients, with biopsy-proven calciphylaxis, that received hbot in CRIS-UTH in the period 2016-2018 because of typical wounds in their right legs (n=2), in left legs (n=1) and in both legs (n=3). All them were female and additionally received Sodium thiosulfate, antibiotics, and topical wound care. Patients age were 71.1±11.866 (53-85) [n=6] y.o. Hbot was applied in a multiplace hyperbaric chamber during 40 daily sessions of 90 minutes at 2.3 ATA. Four patients were diabetic, one suffered from chronic renal failure after shoulder surgery, and another one suffered from chronic obstructive arteriopathy. All patients underwent haemodialysis three times a week. Their calciphylaxis induced wounds were midway evaluated at reaching 20 hbot sessions and after the completion of the whole 40 hbot treatment.
Results: Four of the six patients experienced a complete resolution of their wounds after completion of hbot. The remaining two achieved a partial improvement. No hbot related adverse effect was detected. **Conclusions:** HBOT is a safe and promising adjunct therapy for patients suffering from cutaneous necrotic wounds induced by Calciphylaxis, mainly after an early diagnosis in order to prevent serious long-term complications.

SEVERE GAS EMBOLISM CAUSED BY A 15M DEEP DIVE

Marie-Anne Magnan, Pierre Louge, Marc-Alain Panchard, Michel Pellegrini, Claude Lae, Rodrigue Pignel
hyperbaric medicine, Geneva universitary hosiptal, Switzerland

Introduction: We will describe one case report who suffered from a severe DCS after a 15m dive without fast ascent.
Case report: The patient, a 30 year old man, was a diver confirmed diver.
One morning, he went diving in the Leman's lake: 14mn at 15m, 10mn for the ascent with 2 extra mn stop. At 2m deep, he suffered from severe vertigo, weakness sensation, right hemiparesis, vision disorder and dyspnea. At arrival at hospital, he had a real vestibular syndrom and a slight vision disorder. The neurological examination did not identify other neurological disorde. Pulmonary examination and lungs Xray was normal. The CT scann showed a localised pulmonary air bubble. It was present on the The CT scann done 3 years before. The most evident diagnosis was : gas embolism due to a gas tight pulmonary gas buble.
The patient had a USN5. At 18m, neurological disorder disappeared and vertigo decreased. No problem during the session until the decompression : at 1,3 ATA, he sufferd from violent pain in both shoulders, skin rash and new vision disorder. Those symptoms last 20mn and then disappered. Another CT scann showed non change in the pulmonary air bubble. The following day, the patient almost recover.
Conclusion: This patient did many dives without any problem and finally did a severe DCS after a 15m dive without fast ascent.This pulmonary air bubble is actually a contra-indication to dive. During the decompression, we suppose the patient did new gas embolism, but this time it was oxygen and it has been consummed localy by cells as all symptoms have disappeared within 20mn. Even if there was still slight vestibular syndrom after the first session, we have decided not to do another recompression as the pulmonary buble was tight and could cause new gas embolism.

MANAGEMENT OF MAJOR TRAUMA IN REMOTE LOCATION: CASE REPORT

Marco Zacchilli
Hyperbaric Medicine, Centro Iperbarico srl, Italy
Pasquale Longobardi M.D., Marco Zacchilli M.D.

Introduction: Severe trauma in remote locations is still an unexplored field and we believe this case is very important to better understand the needs and the lacks in this kind of scenario in order to provide a better first aid and stabilize the victim until the arrival to the nearest hospital or Advanced Trauma Center.
Methods: We present a case of a 56-year-old scuba diver woman who was crushed between the safari boat sunk in rough seas and the reef (Red Sea, 2018)
Discussion: After the trauma the victim had a severe lacerated wound with bulging of the anus, and tear in skin, sphincters and muscle of the anus, gluteus maximum tear with severe bleeding, complete laceration of the anus and right sciatic nerve injury (Figure 1). On primary survey haemostatic compression on the groin and drinking water were provided to the victim, in absence of advanced emergency equipment. After 8 hours of sailing, the victim was retrieved by the emergency team, who stabilized her while transporting her to the local hospital. After several blood transfusions and a surgical hemostasis, the patient was stable, awake and in good clinical conditions.
Conclusions: According to this case, we propose a Play and Run approach as a winning strategy. Besides the good epilogue, the presence of emergency trained non-medical staff and adequate first aid equipment, especially in remotely located diving center and safari boat, can guarantee a higher safety to potential victims and reduce mortality, providing Prehospital Trauma Life Support (PHTLS).
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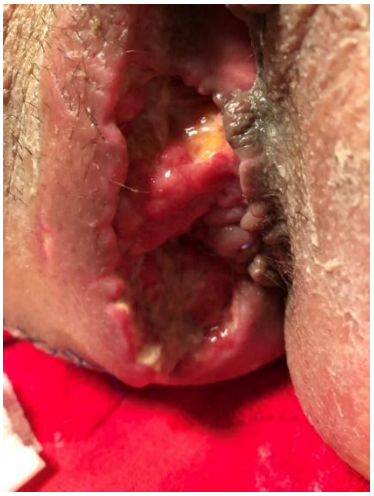


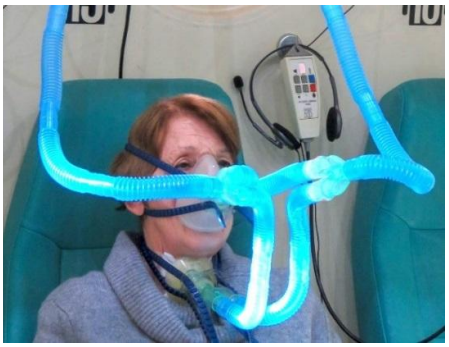
2BREATHE OR NOT TO BREATHE

William Portier, Nathalie Mouton, Yoerik Neirynck, Bart Van Molle, Igor Berrens, Danny Koeken, Peter Germonpré

Background: Hyperbaric Oxygen Therapy implies the respiration of 100% oxygen at pressures not lower than 2 ATA and for durations no less than 60 minutes (7th European Consensus Conference on hyperbaric medicine 2004). Administration of oxygen can be achieved by various devices: oronasal mask, head tent ("hood") or endotracheal tube connection. The efficacy of O_2 administration can be verified by measuring exhaled oxygen pressure: at least 1520 mmhg.
In some patients, neither of these administration devices is readily feasible, and technical adaptations must be made. We present a case where a patient with a laryngeal "stoma stent" (without cuff), needed to be treated for laryngeal radionecrosis..
Methods: For the first session we instructed the patient not to breathe through the mouth, and placed a nose clip in combination with a baby mask on the tracheostomy. The measured values of exhaled oxygen were very far below 1520 mmhg, mainly because the patient continued to breathe supportively through the mouth. Fixation of the baby mask with elastic bands and bandages did not improve the efficacy.
We then created a "double mask" construction, allowing the patient to breathe through both mouth and tracheostomy at the same time: the "2Breathe Mask"
Results: The mask system was well tolerated by our patient.
The patient was treated with the "2Breathe Mask" for 36 consecutive sessions . During 75% of the sessions, we measured values of exhaled oxygen above the intended 1520 mmhg. In the sessions where lower values were measured we could identify various causes: difficult placement of the baby mask due to skin irritation and higher sensitivity at the tracheal site or coughing, causing poorer and irregular breathing.
Conclusion: In contrast to the traditional options, with the "2Breathe Mask" we were able to offer a treatment that complies with the definition of Hyperbaric Oxygen Therapy.

Figure 1.

Figure 2.



HYPERBARIC OXYGEN PRECONDITIONING IMPROVES ANTIOXIDANT DEFENCE AND KIDNEY STRUCTURE IN EXPERIMENTAL MODEL OF ISCHEMIC ACUTE KIDNEY INJURY IN SPONTANEOUSLY HYPERTENSIVE RATS

Sanjin Kovacevic[1], Milan Ivanov[2], Zoran Miloradovic[2], Predrag Brkic[3], Una Jovana Vajic[2], Nevena Mihailovic Stanojevic[2], Djurdjica Jovovic[2], Danijela Karanovic[2], Maja Zivotic[4], Rada Jeremic[3], Jelena Nesovic Ostojic[1]
[1]Department of Pathophysiology, Medical Faculty, University of Belgrade, Serbia
[2]Department of Cardiovascular Physiology, Institute for Medical Research, University of Belgrade, Serbia [3]Department of Medical Physiology, Medical Faculty, University of Belgrade, Serbia [4]Department of Pathology, Medical Faculty, University of Belgrade, Serbia

Background: Acute kidney injury (AKI) is a serious post-surgery complication and hypertensive patients are at a particular risk of fatal outcome. Recent studies suggest a potential beneficial effects of hyperbaric oxygen (HBO) preconditioning in ischemic/reperfusion (I/R) injury, so the aim of our study was to examine those effects on oxidative stress and kidney structure in spontaneously hypertensive rats with postischemic AKI.
Methods: Animals were randomly selected in three experimental groups (n=8): sham-operated group (SHAM), AKI control group and AKI group with HBO preconditioning. HBO preconditioning was performed by exposing to pure oxygen (2.026 bar) twice a day for two consecutive days for 60 minutes and 24 hours before AKI induction. AKI was induced by removal of the right kidney and atraumatic clamp occlusion of the left renal artery for 45 minutes.
Results: In AKI group there was a significant increase (p0.01) in TBARS levels, compared to SHAM rats. Preconditioning with HBO significantly decreased lipid peroxidation in plasma (p0.01). Catalase activity dropped in AKI group (p0.001), while HBO preconditioning significantly elevated its activity (p0.05). In superoxide dismutase activity there was no significant difference among the groups. In AKI group glutathione reductase activity was significantly lower compared to SHAM (p0.05), while in comparison to AKI, enzyme activity increased in group with HBO preconditioning (p0.01). Considering renal morphology, significant morphological alterations present after AKI induction were significantly improved in HBO preconditioning group with reduced tubular dilatation, tubular necrosis and PAS positive cast formation. Histopathological score, as a sum of these changes was significantly lower in group with HBO preconditioning before AKI induction in comparison to AKI group (p0.05).
Conclusions: Our results suggest that HBO preconditioning improves antioxidant defense and kidney structure and its protective role in I/R kidney injury should be evaluated in further clinical and experimental trials.

SOS FROSTBITE RESEARCH PROGRAMME: BENEFIT OF EARLY HBOT ON SEVERE FROSTBITE INJURY OUTCOME.

Marie-Anne Magnan, Pierre Louge, Michel Pellegrini, Marc-Alain Panchard, Claude Lae, Rodrigue Pignel
Hyperbaric medicine center, Geneva university hospital, Switzerland

Background: Frostbite is a specific, infrequent pathology. When extensive, and without proper treatment, it can result in serious amputations.
Hyperbaric oxygen therapy, was until now not recommended due to insufficient data. SOS frostbite is the first trial that compares 2 groups of patients with severe frostbite: the first group with early iloprost and HBO treatment, the control group with iloprost only
Method: Three year study to evaluate hyperbaric oxygen therapy (HBOT) efficiency on severe frostbite outcome.
The SOS Frostbite group: inclusion criteria: stage 3 and 4 (DMTM classification) then early HBO treatment: 155mn per day for 14 days after the first iloprost infusion.
Patients treated with HBO are compared with a restrospective review of 39 patients from Mont-Blanc hospital who had iloprost after the rewarming from 2000 to 2012.
The main evaluation criteria on frostbite outcome is the amputation.
This study also provides a long term medical follow-up to evaluate early and late frostbite sequelae: clinical examination (1-6months, 1-2-3 years), Xray at 3months, 1 year, 3 years.
Results: The study will be finished by the end of May 2019. 30 patients have already been included from the beginning of the project in the SOS frostbite group. The statistic analysis will be done by the end of June 2019 and bo presented at the next EUBS congress. At first sight, HBOT has lower the amputation level and did improve the speed of healing. It has to be confirmed by the statistic analysis before any conclusion.
Conclusion: Studies have shown that iloprost infusion given late isn't very efficient. We still have interesting frostbite outcome when HBOT is given over 48h. There is no literature on long-term frostbite medical outcome. This review will be an interesting database about frostbite sequelea as arthrosis.
Key Words: Frostbite – outcome- Classification - Prognosis – HBOT

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