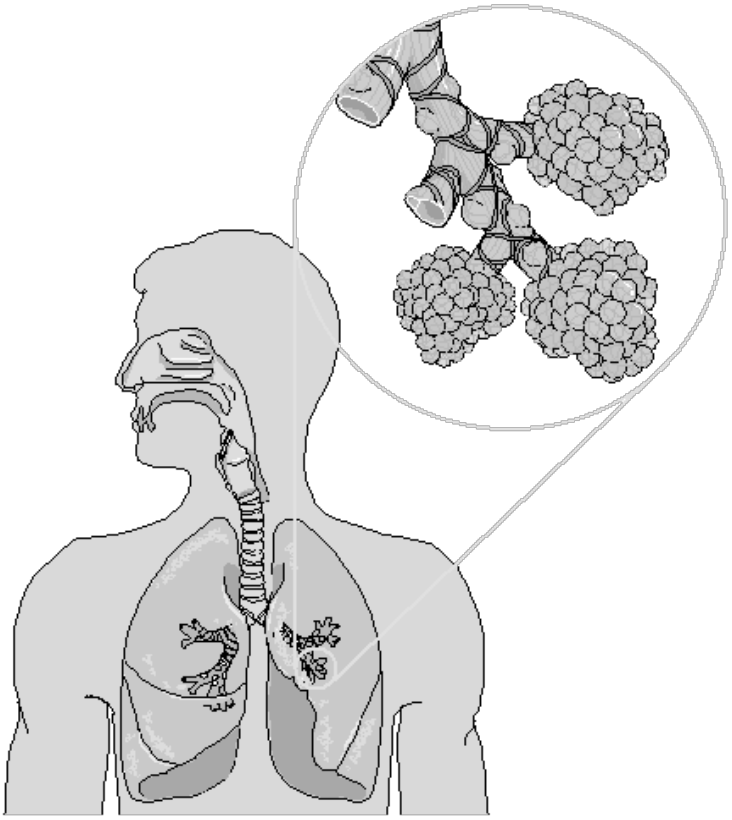
SDCED/DAN WORKSHOP

BAROTRAUMA AND SIPE IN FREEDIVING

San Diego, California USA - 27-28 October 2023

PROCEEDINGS



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The San Diego Center of Excellence in Diving at UC San Diego and the Divers Alert Network convened the Barotrauma and SIPE in Freediving workshop on 27-28 October 2023 in San Diego, California to provide a synthetic overview of the current knowledge of barotrauma and swimming-induced immersion pulmonary edema (SIPE). This workshop was an international, interdisciplinary effort to examine the prevalent emerging issues with freediving and different types of swimming-, immersion-, and altitude-related pulmonary edema. This volume presents 21 papers and panel discussions with particular focus on the definition, symptomatology, mechanics, prevention and treatment of SIPE and squeeze, a colloquial term amongst freedivers for lung barotrauma of descent. Immersion pulmonary edema is considered cardiogenic edema from increased transcapillary pressure. Fluid generated in the pulmonary interstitium or alveoli is dependent on inflow (arterial pressure); outflow (venous pressure); and, lymphatic drainage. Barotrauma of descent is coupled to a relative negative pressure in the airways and/or alveoli triggering anatomical distortion of lung parenchyma that presents as tears to the structures with bleeding. New data suggests that infection may be present providing permeability and inflammation as a contributing component. Mechanisms for individual sensitivity, influence of gender, and genetic predisposition are poorly known but hypertension has been identified as a factor. Workshop presentations spanned lung squeeze and DCI in freediving education, review of emergent freediving cases, current medical procedures at competitions, pathophysiology of SIPE, pulmonary capillary stress failure, infection as a risk factor, biomechanics of SIPE, sex and age difference prevalence, mechanisms of SIPE, pathophysiology and clinical course of HAPE (high-altitude pulmonary edema), patent foramen ovale in freediving, hemoptysis, diagnosis of SIPE in recreational swimmers, lung damage and long-term consequences of barotrauma. Finally, terminology, treatment, and return to freediving or swimming were addressed in a panel discussion format. A consensus was reached to introduce a new term to describe the multiple pulmonary injury types connected with freediving. Freediving-induced pulmonary syndrome (FIPS) was suggested, acknowledging that the classification of severity and recognizable symptoms of each subcategory of FIPS is not currently possible due to a lack of reliable data. Further discussion and collection of data is warranted from retrospective as well as prospective applied research. Workshop Proceedings can be downloaded at www.dan.org.

This diving safety workshop and the publication of this document was jointly sponsored by the UC San Diego Center of Excellence in Diving and Divers Alert Network.

Cover design by Michael A. Lang

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San Diego Center of Excellence in Diving
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The San Diego Center of Excellence in Diving (SDCED) is a unique interdisciplinary international research center administratively housed in the UC San Diego Department of Emergency Medicine. A partnership among UC San Diego Health Sciences, UC San Diego Scripps Institution of Oceanography, Divers Alert Network, Diving Unlimited International and Scubapro, SDCED is a unique research-focused, education-oriented, collaborative partnership promoting *"Healthy Divers in Healthy Oceans." Its* mission is to support and enhance excellence in diving through collaborative research, education, and training of divers and physicians worldwide, creating a nexus between diving medicine and marine science. SDCED accomplishes its mission through education, community partnerships, interdisciplinary collaborative research, and the translation of research into practical applications.

Human health and oceans are of increasing global importance. SDCED's research interests include: **Breathhold Diving Physiology** (diving adaptations of marine animals and their application to human medicine); **Diver Protection** (thermal technologies and life support breathing equipment for work in extreme and polar environments); **Human Health** (mechanisms and mitigation of severe DCS manifestations); and, **Public Safety Diving Health Initiative** (epidemiology of acute and chronic health effects of contaminated water diving). SDCED was founded in 2014 by Dr. Michael A. Lang and Dr. Karen B. Van Hoesen with founding partnership support provided by UC San Diego, Divers Alert Network, Scubapro, and Diving Unlimited International. Current SDCED executive management is provided by Dr. Michael Lang and Dr. Peter Lindholm as Co-Directors, Dr. Karen Van Hoesen as Associate Director, Dr. Frauke Tillmans, Dr. Ian Grover, and Dr. Paul Ponganis.

Divers Alert Network
www.dan.org

Mission

DAN helps divers in need of medical emergency assistance and promotes dive safety through research, education, products and services. Divers Alert Network (DAN), a nonprofit organization, exists to provide expert medical information for the benefit of the diving public. DAN's historic and primary function is to provide timely information and assistance for underwater diving injuries, to work to prevent injuries, and to promote dive safety. Second, DAN promotes and supports underwater dive research and education, particularly as it relates to the improvement of dive safety, medical treatment and first aid. Third, DAN strives to provide the most accurate, up-to-date and unbiased information on issues of common concern to the diving public, primarily, but not exclusively, for dive safety.

Vision

Striving to make every dive accident- and injury-free. DAN's vision is to be the most recognized and trusted organization worldwide in the fields of diver safety and emergency services, health, research and education by its members, instructors, supporters and the recreational diving community at large.

ACKNOWLEDGMENTS

We wish to acknowledge and extend sincere appreciation to the Barotrauma and SIPE in Freediving Workshop co-sponsors: San Diego Center of Excellence in Diving and Divers Alert Network. This scientific workshop would not have been possible without their combined support and that of our home institutions for supporting our time and all the workshop participants.

Many thanks to all the workshop speakers who helped immensely by cooperating and submitting their manuscripts in a timely fashion. The short turn-around time for the production of proceedings for a workshop of this size is remarkable. We take satisfaction in having assembled this expert cast of professionals who shared their expertise on this topic with the workshop. The international, interdisciplinary nature of this project is evidenced by the participation of colleagues from the medical, recreational, technical, military, and scientific diving communities and the papers they presented. Participant expertise from Sweden, Belgium, Canada, Croatia, Peru, Argentina, Brazil, Switzerland, Russia, Germany was present in collaboration with colleagues from across the United States.

DAN staff were helpful during various phases of this workshop's organization and participant travel. Kimberley Farkas provided expert assistance in the transcription of workshop discussions. Dr. Van Hoesen was instrumental in organizing the local logistics with hotel management, and workshop dinner. We thank Bill Ziefle, DAN CEO, for authorization of DAN resources in support of this effort.

Finally, we enjoyed our collaboration as Workshop Co-Chairs and, from all indicators, have the sense that we succeeded in accomplishing the workshop objectives.

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INTRODUCTION, QUESTIONS, AND GOALS

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Currently, the pulmonary symptoms that are experienced after breathhold diving fall under the term 'squeeze', which I consider a colloquial term for barotrauma of descent, but they can also be termed immersion pulmonary edema (IPE), or swimming-induced pulmonary edema (SIPE). A recent Pubmed search found 169 references for IPE and 102 for SIPE. Searching for barotrauma or squeeze produced lots of references that included ear and sinus symptoms, while breathhold diving edema identified 46 references.

Questions on this topic from active divers (or swimmers) and the health-care professionals that care for them include what to do, how to prevent and/or provide treatment? In addition, when can individuals return to swimming and/or diving?

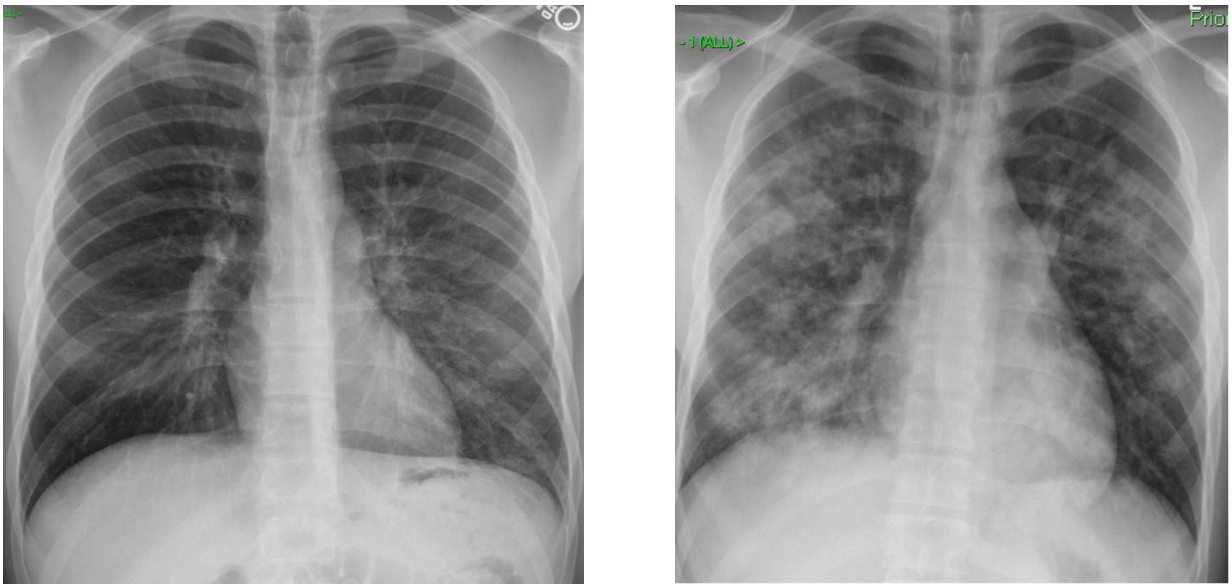
Generally, IPE is considered cardiogenic edema from increased transcapillary pressure, although there are multiple mechanisms involved with various levels of evidence. As such, generation of fluid in the pulmonary interstitium or alveoli could depend on three components: inflow /pressure on the arterial side; outflow (venous pressure, left ventricular diastolic [dys]function); and lymphatic drainage.

In addition, the barotrauma component must be considered with a relative negative pressure in the airways and/or alveoli (although alveoli can be assumed to collapse in atelectasis). There is also the external hydrostatic effect of position, as swimming on one side can add pressure to the dependent lung. In barotrauma of descent an anatomical distortion of the lung parenchyma is possible causing some form of tear to the structures with resulting bleeding. Volk et al. (2020) documented interstitial and alveolar types of edema (Fig. 1).

Adding to the classic cardiogenic edema there is new data suggesting infection could be involved, so permeability (and inflammation) could also be a contributing component.

Drugs such as NSAIDs have been associated with IPE in case reports and Sildenafil has been shown to be protective in surface swimming triathletes.

Mechanisms for individual sensitivity and genetic predisposition are largely unknown except an increased prevalence in women versus men. Hypertension has also been associated with IPE/SIPE.

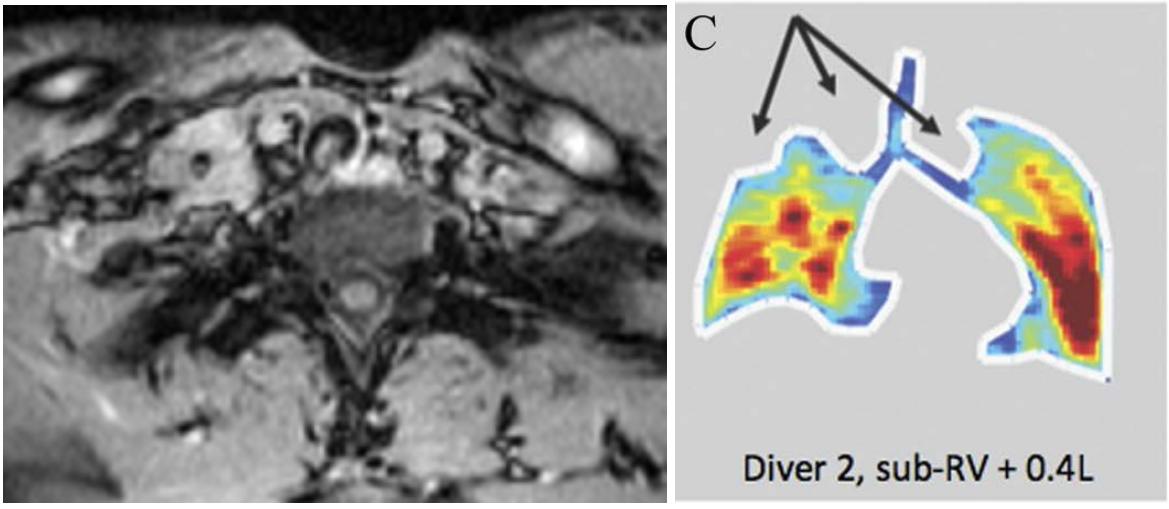


A. **B.**

Figure 1. Chest x-ray images from two types of edema in SIPE. Panel A represents classic Kerley B-lines as an interstitial edema, while panel B represents a typical image of predominantly patchy alveolar edema (Volk et al. 2020).

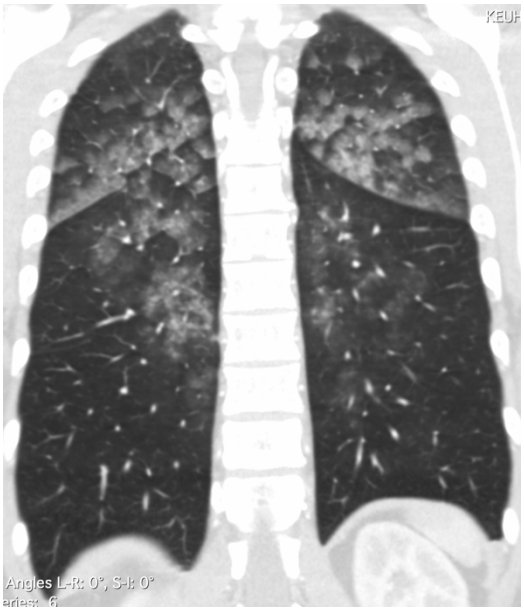
Barotrauma of descent, can there be negative (relative negative) pressure?

Atelectasis and collapse of alveoli and small airways is most likely the result of a reduced volume by compression, but the larger airways have cartilage reinforcements. Suggestions from the literature are shown in Figure 2.



A. **B.**

Figure 2. A: Tracheal squeeze from prolapse of the posterior tracheal membrane during glossopharyngeal exsufflation below residual volume in an MRI (Lindholm and Nyren 2005). B: Atelectasis with reopening on inspiration from RV+GE (Muradyan et al. 2010).



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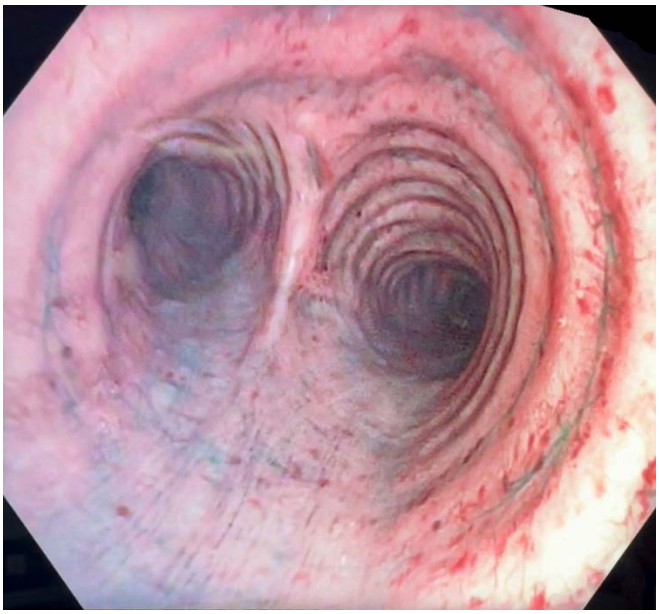

D.

Figure 2. C. Patchy alveolar edema or bleeding after extended hypoxia from dry breathholding at RV. (Valdivia et al. 2021). D. Mucosal petechiae and hemorrhage in pulmonary barotrauma of descent in breathhold diving: direct observation of a suspected pathophysiologic mechanism (Submitted for publication; Silva F, Gouin E, Lindholm P. 2023).

Questions

What is SIPE & squeeze?

- Pulmonary edema?
- Immersion pulmonary edema (submersion pulmonary edema?)
- Swimming induced pulmonary edema?
- Negative pressure edema?
- Barotrauma? Suction, shear stress? Contusions?
- Volume/tissue stretching involved?
- Fluid shift?
- Blood shift?
- Tracheal squeeze?
- Bronchial squeeze?
- Airway edema?
- Interstitial and alveolar edema?

What are risk factors for SIPE & squeeze?

- Exercise
- Swimming on the side
- Contractions
- Hypertension
- Cold water
- Stretching when compressed decreasing airway pressure or tissue strain?
- NSAID /aspirin/ ibuprofen
- Infection?
- Inflammation? Asthma?

- Divers have reported decompression illness after single dives. Is it traditional decompression sickness or arterial gas embolism? Is it connected to barotrauma or shunt? Can SIPE and squeeze cause 'air trapping' on ascent, or shunt venous gas emboli across the lungs like a patent foramen ovale?

What to do when it happens?

- Treatment?
- Fitness to return to dive?
- Are some at risk for recurrence?
- Long term effects?
- Level of preparation if you oversee/organize an event?

Goals

A current overview of the field by participants with invited scientists, physicians, competitive divers and freediving instructors. We will have group discussions aiming for 'new' or more granular terminology, and possibly develop recommendations on treatment and return to dive/swim?

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LUNG SQUEEZE AND DCI IN FREEDIVING EDUCATION

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Squeeze is a condition that can affect freedivers, yet it is not adequately covered in the four recreational freediving education systems taught worldwide. To avoid a squeeze divers are advised to physically relax before and during their dive, known as "the relaxation phase," and to avoid jerky or wide-reaching movements when diving to depths where the lungs reach residual volume. However, if a freediver does experience squeeze symptoms the only advice given is to seek medical attention, which may provide minimal help as freediving-related injuries are rare and doctors are often unable to help.

Many world-class freedivers experience squeeze symptoms, but they cannot disclose it as freedive operators and insurers follow the regulations of agencies and block freedivers with squeeze symptoms from further training or competition. This is known as the "Squeeze Omertà," where squeezes are not spoken of.

To tackle these issues a comprehensive approach is needed, especially with the introduction of competition-oriented courses that go beyond recreational depths. This approach should include definition, classification, and return-to-dive advice. A model for such a tool can be found in the FIFA Concussion Protocol, which offers a guideline for medical personnel to make a go/no-go call in three minutes and how to proceed from there.

Valdivia, Christen, and Maričić (unpublished) propose a framework for a similar tool in freediving. The framework includes pre-existing risks that may vary in every freediver and through a list of comorbidities and symptoms "trauma points" are added up to determine the status of a freediver after an incident, from mild to moderate to severe. The classification defines the return-to-dive prognosis and path. Further, evaluation frequency and evaluation tools are suggested.

Discussion

I Barković: Do you suggest that divers not dive deeper than residual volume? How do divers know their residual volume? Or do you suggest all competitors or all freedivers should confirm max volume?

O Christen: That's one of the very good questions. Nothing of what I introduced in that proposal has been tested. Can we use that or do we have to replace that wording or that practice with something easier to make a call? We are really at the beginning of a very long journey.

I Barković: Theoretically, it would be below 40 m, 1/5 of total lung capacity, but not taking into account that you are not getting total lung capacity and shifting air into your sinuses.

O Christen: I make a distinction between active residual volume and passive residual volume. How much can I pass and how deep can I go? Is that residual volume, passive residual volume? That would mean that really deep freedivers do not have residual volume anymore. Alexey Molchanov could now dive to 90 m right here in San Diego Bay and his lungs would not mind. For him the recommendation would be 90 m. We do not know.

B Smith: Regarding the recommendations for pure O_2 on recovery, are these people poorly saturated? You would not do that on ventilation. My big spiel is that we should promote positive pressure.

O Christen: I'll forward that question over to Bizo Silva.

F Silva: Perfect timing because you are the expert in this area. My concern is also the same, why have a starting level of oxygen when you can do positive pressure and have the same beneficial effect. I think that's where you're going with your question.

B Smith: Are people desaturated and having poor oxygen saturation?

F Silva: If you give oxygen, it is because saturation is not good. If saturation is acceptable, then oxygen is not the logical thing to give.

S Hopkins: How is the pure oxygen administered, by mask? They would only be getting 30% at most, not 100% oxygen.

F Silva: There are some systems that actually can deliver 100% oxygen.

S Hopkins: That would seem to be very counterproductive because from 10 minutes on 100% oxygen, you are going to get alveolar collapse and absorption atelectasis. I can provide citations for that work showing that. High O_2 concentrations would be concerning because parts of your lung that have low ventilation are just going to collapse and you are going to get atelectasis.

O Christen: In response to the likelihood that pure oxygen produces alveolar atelectasis and collapse is made worse, seeing older scuba divers come up with immersion pulmonary edema you might get an event based on the hypoxia. On that point I would agree that given a short course of high-level oxygen would be beneficial to actually save their lives and prevent myocardial infarction.

S Hopkins: I agree with you, something must be done.

J Valdivia: Usually once they are out of the water, the edema resolves pretty quickly.

O Christen: The good news is we do not need to find the answer right now.

J Valdivia: We provide 100% oxygen in tanks, and we use them with regulators because oxygen masks do not deliver high enough inspired fraction of oxygen. After 100% oxygen post-squeeze, the results are much better. Recovery is amazing through me and another 98% of freedivers. Without that five to 10 mins, it can be on normal pressure or hyperbaric (underwater). It is so evident that it helps so much that I now have it as standard protocol for all divers above 80 m and mandatory for 100 m and immediately at 110 m plus. I would like to add, as you mentioned, when we teach we don't allow squeeze. I know cases where people have been banned for 2-3 months from participating in their course because they had a light squeeze. This is a more or less standard practice scenario. We also have people who just dive the next day. There is a huge discrepancy with these two things and neither is based on anything other than who gets their hands on you. Of course, in this environment without objectivity, a diver is not going to tell you they squeeze. It is a huge problem.

K Krack: To add to the different protocols that are out there, PFI has for over 15 years had a 2, 4 and 6-week protocol. When you come up the first thing we need to do is figure out is whether it is lung or sinus. Are you going to do a snort test, spit, or are you going to do a cough test. We figure out that it's lungs, and that's just a tough thing. Then what we're going to do is say, if you spit and you have wisps of blood, that's going to be two weeks. If you have blood with a little spit, that's going to be four weeks. And then if you re-squeeze in a time or there are any signs of cyanosis where further care is needed, that will be six weeks out of the water. After you've done either 2, 4, or 6 weeks, then you're back in at half your depth, i.e., if you squeeze at 40 m, your first day of diving is at 20 m. Then on a day to day basis you are making 5 m jumps, working on what probably caused the issue in the first place, which can be head position, and the warm-ups for example. If you then have another light squeeze, little wisps, then you are automatically up to six weeks out of the water. I have seen more than enough people who have these light squeezes that are barely healing and they open it up and rip it again to the point where they squeeze themselves out of the sport or they take themselves to the point where just simply doing a residual volume, they could induce a squeeze, as seen through imaging.

A REVIEW OF 147 FREEDIVING CASES ON THE DAN HOTLINE 2020-23

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Objective

To provide a snapshot of the types of calls regarding freediving incidents received by the DAN America hotline.

Methods

We identified and reviewed 147 DAN hotline cases (records) received between January 2020 and August 2023 involving inquiries about freediving. The records were analyzed for Reason for Calling (RC), the chief complaint or primary problem of the injured diver; and Final Diagnosis category (FDx), and a categorization of the most likely causation of the manifestations after reviewing all case notes including the final diagnosis by the evaluating physician where available. Additionally, we collected basic demographic data and injury location.

Results

In terms of demographics, approximately 60% of callers were male, and the mean age of all callers was 31 years old, ranging from 14 to 76. The majority of calls (55%) originated from the United States, with Florida and Hawaii being the top states, followed by California, Washington, and North Carolina.

The key findings of this study indicate that ear and sinus issues were the most common chief complaints, accounting for 55% of the cases, and were also the most frequent final diagnoses, at 66%. It is noteworthy that this prevalence is slightly higher than what is typically observed in the general diving population, which stands at around 45%. Additionally, most of these cases were self-diagnosed by the individuals involved.

Decompression illness (DCI) was a concern in 9.5% of the calls but was confirmed as the final diagnosis in only 2% of all cases. Respiratory issues were the chief complaint in 21% of calls, with a final diagnosis in 10% of cases, including cases of tracheal squeeze (5%), lung squeeze (3%), and pulmonary barotrauma (1%). Manifestations consistent with immersion pulmonary edema (IPE) were observed in 8% of cases. There were reports of loss of consciousness (LOC) or blackouts during freediving in 5% of calls, but only 2% of these narratives strongly suggested a hypoxic LOC or blackout. Notably, there was one fatality in a minor who combined freediving with surface-supplied diving beyond decompression limits, where cardiopulmonary decompression sickness (DCS) was suspected as the cause of death. One case involved severe trauma as a diver was run over by a boat, and another case involved myocardial infarction.

Conclusions

The findings underscore the high occurrence of ear and sinus issues in incidents related to freediving, as reported to the DAN America hotline. Additionally, we note that the prevalence of respiratory issues, accounting for approximately 10% of all calls within this sample, appears to be higher than that typically

observed in the general diving population. Moreover, the specific pulmonary diagnoses encountered in this study seem to be rather unique to this diving modality.

However, it is essential to acknowledge certain limitations in this study. These limitations pertain not only to the sample size but also to the potential presence of selection bias due to the absence of a universally agreed-upon definition for "freediving", and the authors' subjective impression that dedicated freedivers may not always resort to the DAN Hotline for assistance.

In addition to more reporting efforts from community leaders and divers, a more precise definition of "freediving" might help DAN collect more precise data for this population.

Discussion

J Valdivia: Did you stratify data with level of training?

M Nochetto: No. Unfortunately, we hardly ever ask that question. It would be interesting. We don't even have good data on that question in the general population. If there is an interest in this particular group of diving, that's something we can ask.

J Valdivia: That would be a very good way to stratify. Are we talking freediving or are we talking snorkeling? Perhaps talk about insurance for agencies or for individuals, what are the numbers? A good way would be to ask "Are you certified for diving? Are you certified from an accredited agency?"

M Nochetto: That is something we can put in our protocols.

B Smith: Do you have any way to estimate an incidence rate, like incidence per number of dives?

M Nochetto: No, we don't have that denominator. In competitions it would be easier because you know how many dives are registered. We don't ask what type of divers they are. Even if we would, chances are they would have certifications for both.

CURRENT MEDICAL PROCEDURES AND POLICIES AT COMPETITIONS

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Squeeze is a commonly encountered condition at freediving competitions, especially depth events where records are repeatedly broken. The two main governing organizations that oversee events are the International Association for the Development of Apnea (AIDA) and the Confédération Mondiale des Activités Subaquatiques (CMAS). CMAS was founded in 1959 by Jacques Cousteau and was the first organization to host freediving competitions. However, a series of accidents led them to suspend further freediving activities. This void in freediving competition organization led to the establishment of AIDA in 1992. A few years later, CMAS resumed their ratification of freediving events and now both organizations oversee freediving events worldwide.

Both AIDA and CMAS offer a freediving certification program, along with a multitude of other organizations. Some, such as Molchanovs, are purely focused on freediving education and training whereas others, such as Scuba Schools International (SSI), have developed freediving courses adjacent to their scuba background. It is nearly impossible to compare the different schools due to varied training levels and requirements, some of which are proprietary and only available to dues paying members. However, most institutions will recognize the training of another or offer a "crossover" session.

With regards to competitive freediving, CMAS and AIDA have developed safety protocols and procedures for handling medical incidents during competition. The differences are outlined in the table below.

	AIDA	CMAS
Medical Team	1 Emergency Medical Technician (EMT) 1 CPR/first aid/DAN oxygen provider	1 Doctor Ambulance with doctor on board
Local Resources	Hyperbaric chamber	Hospital accessible by ambulance
Airway Equipment	Nasal cannula Nasopharyngeal airway (NPA) Oropharyngeal airway (OPA) Pocket mask Bag valve mask (BVM) Nonrebreather mask (NRB) Suction device Oxygen tanks	Bag valve mask (BVM) Oral pharyngeal airway (OPA) Advanced airway kit Manual suction Oxygen tanks
Other Equipment	Sphygmomanometer Pulse oximeter Stethoscope Thermometer Medical stapler Spine board Scissors Light	Automatic External Defibrillator (AED)

	pen/marker Automated External Defibrillator (AED) Adhesive tape Antiseptic solution Bandages/sterile dressings Gloves Syringes	
Evacuation	Emergency cellphone Evacuation vehicle	Boat with 2 operators
Protocols	Squeeze	Surface blackout Subsurface blackout Packing blackout

The AIDA squeeze protocol sets the current standard for responding to a pulmonary syndrome that is poorly understood, and likely represents a combination of both interstitial edema from immersion-related fluid shifts and alveolar hemorrhage from mechanical stress on lung parenchyma. In the evaluation of a freediver with a squeeze, they recommend basic vital signs, a focused pulmonary exam, and consideration of lung ultrasound in the field. This exam must be performed immediately after the incident, as well as at follow-up intervals of 15 minutes, 30 minutes, 1 hour, 12 hours, and each subsequent morning of competition.

A mild squeeze is defined as some blood-tinged sputum (hemoptysis), with hypoxemia of 10 minutes max, with symptoms completely resolving within 1 hour. A mild squeeze is expected to self-resolve. A moderate squeeze is defined as a mild squeeze with the addition of tachycardia, dyspnea, interstitial breath sounds, and/or lung pain that resolves within 24 hours. Oxygen therapy is mandated in these cases. A severe squeeze is defined as massive hemoptysis, prolonged hypoxemia, fatigue, and/or cyanosis with symptoms persisting beyond 24 hours. Oxygen therapy with positive pressure (if available) and transportation to a medical facility is required.

I had the opportunity to witness the medical care provided at both AIDA- and CMAS-run competitions this past summer. At the AIDA event, I noticed that squeezes (and blackouts with resulting hypoxemia) were managed primarily with continuous positive airway pressure (CPAP), as oxygen was a rare and expensive resource at the location of the event. Additionally, there was no hyperbaric chamber available for the event, with the closest one requiring an airplane trip. At the CMAS event, I noted that some advanced airway supplies and suction were unavailable, with the event running out of oxygen partway through the competition.

My evaluation of the medical policies for freediving events, combined with the real-life experience watching an event unfold, brought up some concerns about the expectations set forth in these policies. The timing of reassessment of a squeeze puts a huge strain on limited medical personnel at events. Additionally, the screening test of pulse oximetry may miss freedivers who have lung injury without hypoxemia. Those that have hypoxemia are primarily treated with oxygen, which can be a rare and expensive resource in some locations.

Therefore, I propose the following questions to the freediving medical community:

- What is the best way to define a squeeze and its severity?
- Is there a relationship between squeeze and blackout?
- Should divers be screened for squeeze? If so, how?
- Is oxygen therapy or positive pressure ventilation (CPAP) more beneficial for squeeze treatment and/or recovery?
- Is hyperbaric oxygen therapy beneficial for squeeze?
- When can you clear an athlete to dive again after a squeeze?

Discussion

K Van Hoesen: For the definitive airway, do they make recommendations toward an airway device such as an i-gel or laryngeal mask airway (LMA) or having a laryngoscope and ET tube? Did they go so far as to say you need to have this on site. Because once you start talking about laryngoscope and ET tube and paralytics and drugs, you are going down a rabbit hole.

E Yu: Not in the text of CMAS. It just said "definitive airway." What I encountered on the boat was multiple ET tubes without stylets, one mac 3 blade single use, no paralytics.

J Valdivia: The first time I was a medic in 2014, they told me that medics were there for show. Because all the medical issues get solved on the platform. It is extremely rare that somebody gets evacuated, because you have to stop the competition and delay everything. Therefore, it is extremely rare that somebody gets intubated. Most of these competitions are held in remote areas where there is no hospital or chamber. This is why I was involved in the guidelines development adapting to those conditions. When somebody is trying to wake up from blackout/trauma, you want to help them awake.

K Krack: Should the organizations actually sanction a competition where there is no access to a hospital, or at least reasonable access to a hyperbaric chamber? The other question is what would we want? Would I want a physician? Would I want an EMT? Or would I rather have a paramedic who has the infield experience of dealing at a high level who can do advanced cardiac life support, push drugs, versus a physician who is used to a room with all the people, nurses and the equipment around? Preferably at my competitions I have always had paramedical capability with that level of experience to get to advanced trauma care in a hospital.

SWIMMING-INDUCED PULMONARY EDEMA: WHAT DO WE KNOW ABOUT PATHOPHYSIOLOGY, PREVENTION AND TREATMENT?

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Immersion pulmonary edema (IPE, also known as swimming-induced pulmonary edema, SIPE) is a condition in which cough, hemoptysis, dyspnea and hypoxemia develop after surface swimming or scuba diving. [1-11] Case series from military populations consisted predominantly of males, usually trainees during strenuous training swims, [6,7,10,12,13] while more recent studies have reported female predominance. [14-17]

Identified risk factors for SIPE include swimming or diving in cold water, [1,3,11], negative static lung load (SLL), [18] heavy exertion, [3,4,6-8,10-12] fluid loading, [12] low vital capacity, [7] left ventricular hypertrophy (LVH), immersion-induced mitral regurgitation, [19] Takotsubo cardiomyopathy [20-22] and other cardiac abnormalities, [23] including myocardial edema. [24] Recent evidence implicates antecedent pulmonary infection or presence of respiratory pathogens. [25] SIPE also tends to recur. [1,3,7,11,13,23,26] The most common risk factor is hypertension. Indeed, appropriate treatment of hypertension with a sufficient period to allow the heart muscle to remodel will reduce the risk. The time course varies and can be up to 1-2 years. [27]

Approximately 30-40 cases per year have been reported during Basic Underwater Demolition/SEAL (BUD/S) Training at the US Naval Special Warfare Training Center. [8,10,28] Cases of SIPE are also seen at the Naval Diving and Salvage Training Center. Its prevalence in 2.4-3.6 km open sea swimming trials has been reported to be from 1.8-60%, depending upon the severity. [7,13] Prevalence at BUD/S is 1-3% of trainees during spring, summer and fall, and 5% in the winter. [29] In a Swedish open-water swimming event with distances of 1,000-3,000 m in cold water, the incidence of SIPE has been reported as 0.44% (0.75% in women, 0.09% in men). [15]

Pulmonary edema in a normal lung occurs when pulmonary artery (PA) and PA wedge pressure (PAWP) acutely exceeds a critical value of 18-25 mmHg. [30-32] Both PA and PA wedge pressures increase during immersion, primarily due to central redistribution of blood from the extremities. [33,34], which engorges the central veins, heart and pulmonary vessels. [33,35-37] This peripheral to central redistribution of blood is augmented by cold water immersion. [37,38] and attenuated by a concurrent reduction in venous tone. [35] Studies from our lab have demonstrated significant pulmonary arterial (PA) and venous hypertension (and systemic hypertension) during submersion, particularly in cold water. [37] We have demonstrated that in SIPE-susceptible volunteers the peripheral-to-central blood redistribution leads to excessively high PA and PA wedge pressures during submerged exercise at levels sufficient to precipitate pulmonary edema. [39]

In some cases, such as cardiac valve disease or cardiomyopathy, a plausible reason for high right sided pressures is self-evident. However, the reason may be less obvious, particularly when systolic and valve function are normal. We have investigated diastolic function using echocardiography during head-out immersed exercise in cold water and observed evidence of stiffer left ventricles during diastole in SIPE-susceptible volunteers. LV diastolic filling in the presence of reduced LV compliance requires greater LV end-diastolic pressure (LVEDP), which is further increased during immersion due to blood redistribution. Treatment of hypertension can improve left ventricular diastolic function.

During submersed exercise, a 50 mg oral dose of sildenafil was sufficient to reduce both PA and PA wedge pressures in volunteers with a history of SIPE, suggesting its efficacy as a prophylaxis against

SIPE, supported by a case report. [40] In SIPE-susceptible individuals pre-loaded with oral fluid who exercised head-out in 20°C water, a randomized, double-blinded study of 50 mg oral sildenafil vs. placebo did not support the utility of the drug, possibly due to negative SLL during the exposure. [18] It has been reported anecdotally that oral nifedipine 5 mg taken before a dive will reduce the risk of SIPE. [41] However, nifedipine may have a negative inotropic effect [42] and thus may not be ideal for situations involving heavy exercise, such as a triathlon or naval special warfare training.

Conventional wisdom is that SIPE spontaneously resolves within 24 hours or with β_2-adrenergic agonist or diuretic therapy. However, new evidence indicates that symptoms can be persistent, [26] and indeed can be fatal. [5, 43]

Some unanswered questions include reasons for the male-female difference, whether Takotsubo cardiomyopathy is a cause or effect and best preventive strategies for susceptible individuals.

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Discussion

A Luks: Regarding the use of sildenafil in this situation, the PD5 inhibitors like the calcium channel blockers are thought to be effective for prophylaxis of high-altitude pulmonary edema cessation, but in that situation it is all about arterial vasoconstriction, upstream of the capillary bed, left ventricular diastolic pressure is normal. Whereas, what you describe in SIPE it is more of a problem of elevated left ventricular diastolic pressure. If anything, you are going to vasodilate in the pulmonary capillary bed and send more forward towards the left side of the heart. We don't use PD5 inhibitors, for example, in people who just have heart failure with preserved ejection fraction at sea level. It occurs after load reduction for those people. It will be interesting to see a better powered study with more subjects to see if this flushes out. If it does not, that may account for why you are not seeing a positive effect of the sildenafil in this case. The second question would be have you looked at using other things like angiotensin receptor blockers or ACE inhibitors, which would focus more on after load reduction that might bring down the left ventricular diastolic pressure?

R Moon: Both sildenafil and nifedipine have been used as prophylaxis for SIPE. Sildenafil reduces pulmonary vascular resistance, which might suggest that it could increase blood flow to the lung and enhance the risk of SIPE. However, it is also a venodilator and hence reduces preload and both left atrial and pulmonary capillary pressures. The reason it works is due to inhibition or attenuation of central redistribution of blood. I have not tried ACE inhibitors or anything else to look at this, but clearly, they may work. We chose sildenafil because in the Navy, obviously, they are not interested in any drug that might impair ventricular function. Nifedipine reduces exercise capacity, where sildenafil does not.

C Seiler: The first thing that came out of your sildenafil study was too hard regarding no athlete drinks 2L before starting. Maybe you were over-hydrating in addition to this sitting position. Perhaps you should do it again without fluid loading?

R Moon: You are absolutely right. We did that because of our earlier work showing that it was a reasonable model. But a lot of triathletes are told to load fluid. Of course, that is actually counterproductive. They should wait until they get on the bicycle before they fluid-load.

C Seiler: In theory, the sildenafil should work.

J Valdivia: Do you think that decreasing left ventricular compliance would be a risk factor for pulmonary edema in breathhold diving? Would you agree with that statement?

R Moon: Well, breathhold diving is a little different in the sense that the primary mechanism is due to a high transcapillary pressure during descent. In other words, you can decrease your lung volume down to a residual volume, but then once you reach residual volume, your chest is relatively rigid. Any further descent beyond that point has to be the volume, or the pressure change has to be accompanied by something. What happens is blood redistributes into the pulmonary vascular bed to try and maintain low pressure or try and fill up the volume, if you like. Therefore, it is a different mechanism. I would not bet on sildenafil helping in that situation.

J Valdivia: I agree. Thank you.

PULMONARY CAPILLARY STESS FAILURE: FROM THE THOROUGHBRED RACETRACK TO THE SUMMIT OF EVEREST

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The main function of the lung is as a gas exchanger, with a delicate barrier composed of alveolar endothelium, extracellular matrix and capillary endothelium separating blood from gas. Pulmonary gas exchange is accomplished by the process of *ventilation*, the delivery of fresh gas to the alveoli, accompanied by *perfusion* of the pulmonary capillaries with deoxygenated blood. Gas exchange then occurs with oxygen passively diffusing into and carbon dioxide out of the capillaries down their concentration gradients. Since the resistance to gas transport across the blood-gas barrier is proportional to its thickness, the barrier must be thin, and remarkably is <0.3μm over at least half of its surface area. At the same time the blood gas barrier must be strong (i.e., thick enough) to withstand mechanical forces. Thus, conflicting forces affect the regulation of the thickness and strength of the blood-gas barrier.

The exercising lung is particularly vulnerable and during high intensity exercise, the blood gas barrier is challenged both with a need for thinness for gas exchange and for thickness for mechanical integrity. Exercise increases the need for oxygen flux to supply working muscle and gas exchange efficiency decreases because of a deterioration in the matching of ventilation to perfusion and a variable amount of diffusion limitation for oxygen transport across the blood gas barrier [2]. A primary measure of the efficiency of the pulmonary gas exchange, the alveolar-arterial partial pressure difference for oxygen ($AaDO_2$), reflects the difference between the partial pressure of oxygen in alveolar gas and what subsequently ends up in the arterial blood. The magnitude of the increase in $AaDO_2$ is highly variable but in general it is higher in individuals with a higher maximal oxygen uptake and may reach over 50 Torr in some athletes [2] at near maximal exercise. While ventilation increases with increasing exercise intensity, when this is insufficient to overcome the increase in $AaDO_2$, hypoxemia results [2-4]. This condition is termed exercise-induced arterial hypoxemia and is largely a function of the amount of diffusion limitation that occurs [5]. Paradoxically, exercise induced arterial hypoxemia occurs in highly aerobic humans and animals. While exercise training can improve muscular and cardiac function and increase the demand placed on the pulmonary system, there is evidence that endurance training has a minimal effect on the lung itself [6]. Consequently, even despite its thinness, the blood gas barrier may fail to provide adequate diffusion of oxygen.

However, there is increasing recognition that the blood-gas barrier of the lung is also vulnerable to impairment in function induced by mechanical stress during exercise. During exercise cardiac output increases, resulting in exercise-induced increases in pulmonary arterial pressure, which recruit and distend capillaries [7-10]. Recruitment of capillaries is more likely at moderate increases in flow (<4 times resting), but thereafter, further rises in pulmonary blood flow result in capillary distention [11]. Mean left atrial/ pulmonary venous pressures increase with exercise [7-9] and result in further increases in pulmonary arterial pressure. Since recruitment and distension are not unlimited, and with the increase in pulmonary arterial and venous pressures, capillary pressure increases. Exercise also alters the pressure changes in the alveolus with active expiration, and increase lung inflation and stretch, which amplify the mechanical forces on the lung. Ultimately, pulmonary capillaries may rupture the blood-gas barrier [1,12]. The term "stress failure" has been used to describe mechanically induced breaks in the blood-gas barrier [1,13]. Thus, in addition to not being thin enough for unimpeded diffusion of oxygen the blood gas barrier may not be strong enough to withstand the increased mechanical forces imposed by heavy/maximal exercise.

Stress failure may also be important in lung injury in mechanical ventilation with high airway pressures [13], and in the development of high-altitude pulmonary edema [14], discussed below. The mechanical forces contributing to stress failure relate to circumferential tension (a function of capillary transmural pressure and the radius of curvature of the capillary), longitudinal tension in the alveolar wall elements associated with the inflation of the lung, and surface tension of the alveolar lining layer (Fig. 1). The vulnerability to pulmonary capillary stress failure is increased at high lung inflation and varies between species. In experimental animal models of increased transmural pressure [1], ultrastructural changes in the blood-gas barrier are observed consisting of discrete areas of structural damage to the blood-gas barrier with rupture of the capillary endothelium, basement membrane and the alveolar epithelium, interspersed with large areas of structurally intact blood-gas barrier. Mechanical stress failure results in increased permeability of the blood-gas barrier to protein and red blood cells but the blood-gas barrier retains sieving function for large molecular weight proteins. Leukotriene B_4 (LTB_4) is detected in the bronchoalveolar lavage fluid from these animals [15], and likely represent activation of neutrophils or other white cells by exposed basement membrane.

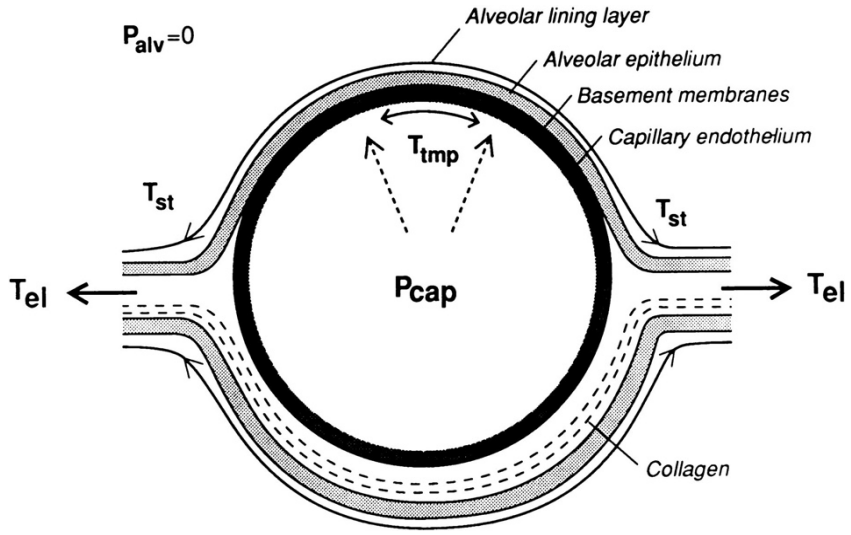

Figure 1. Three forces acting on blood-gas barrier. Circumferential tension (Ttmp) is given by capillary transmural pressure x radius of curvature (r). In this example, alveolar pressure (Palv) is atmospheric so that capillary transmural pressure is equal to capillary hydrostatic pressure (Pcap). Surface tension of alveolar lining layer (Tst) exerts an inward-acting force to support the capillary. Longitudinal tension in alveolar wall elements associated with inflation of lung (Tel) is presumably transmitted mainly by collagen fibers on the thick side of the capillary but may affect wall tension on the thin side, especially at high lung volumes. From West et al. (1991).

The poster child for the dilemma of the blood gas barrier is the thoroughbred racehorse, which has been selectively bred for centuries to do one thing - run extremely fast on the racetrack. Horses have an enormous $\dot{V}O_2max$ (~150 ml/kg/min) and cardiac output and estimated pulmonary capillary pressures may reach ~100 mmHg during exercise [16]. The animals experience profound diffusion limitation [17] during exercise. In addition, bleeding from the lungs is extremely common, and is termed exercise-induced pulmonary hemorrhage. Indeed, all racehorses in training have evidence of some bleeding in their lungs suggestive of stress failure with hemosiderin, a breakdown product from red cells observed in alveolar macrophages [18]. Electron micrographs of lung tissue after galloping in these animals show disruption of both the capillary endothelium and the alveolar epithelium with the result that red cells and protein enter the alveolar space [16], providing direct evidence of stress failure.

There is evidence that stress failure also occurs during exercise in some human athletes. Cardiac output increases as much as 10-fold in high performance aerobic athletes, and mean capillary pressures near the base of the human lung during heavy exercise may exceed 35 mm Hg [1]. There are increased concentrations of red blood cells, total protein and LTB_4 in bronchoalveolar lavage fluid from athletes who exercise at maximal levels for 6-8 minutes in a simulated race compared with sedentary controls. These findings are similar to the animal studies demonstrating stress failure [15]. However, these changes are not observed during lower intensity longer duration exercise suggesting that the human lung is regulated to meet all but the highest demands [12,19] but that the strength of the blood gas barrier can be exceeded at maximal exercise in some.

Another situation where stress failure has been implicated is in high altitude pulmonary edema (HAPE). HAPE is a non-cardiogenic high permeability edema, characterized by alveolar fluid with a high concentration of protein [1] that develops in otherwise healthy individuals following 24-72 hours exposure to altitudes above 2400 m (~8000 feet). Risk factors include rapid ascent, strenuous exercise, and a previous history of HAPE. Increased pulmonary artery pressure is a hallmark of HAPE and is secondary to hypoxic pulmonary vasoconstriction of the precapillary arterioles. HAPE-susceptible subjects have been shown to have increased pulmonary arterial pressures compared to controls prior to the development of HAPE [20,21]. Increased concentrations of red blood cells and protein in bronchoalveolar lavage fluid are observed in the early stages of HAPE before inflammatory cytokines are present [20] suggesting that stress failure may be important in the development of HAPE. Since hypoxic pulmonary vasoconstriction takes place in the pre-capillary arterioles for some time it was uncertain how capillaries might be exposed to high pressure but there is evidence from imaging studies [14,22] that hypoxic pulmonary vasoconstriction is uneven in HAPE thus exposing unconstricted lung segments to both high pressure and high flow.

As well as catastrophic failure of the blood gas barrier with exercise that occurs rarely in humans there is considerable evidence that a great many develop some extent of interstitial edema with exercise. Fluid efflux across the capillary wall is governed by Starling forces with capillary pressure determining transudation into the tissues and the osmotic pressure of blood determining absorption from the tissues [23]. With an increase in mean capillary pressure during exercise accumulation of interstitial edema fluid may distort the surrounding architecture of the alveoli and capillary network and affect distribution of air flow and blood flow in the lung [24] and ventilation-perfusion matching.

Most humans experience increased ventilation-perfusion mismatch with exercise [24-30]. Although this has not been conclusively established, the pattern of the changes is consistent with the development of interstitial edema. Those who increase ventilation-perfusion mismatch during exercise also have greater ventilation-perfusion mismatch in recovery which persists beyond the point at which ventilation and cardiac output normalize to pre-exercise levels [31]. Exercise in normobaric hypoxia, which increases pulmonary arterial pressure and thus mean capillary pressure, causes a greater increase in ventilation–perfusion mismatch than normoxic exercise, and this increase is relieved by breathing 100% oxygen [28]. Additionally, the extent of ventilation-perfusion mismatch increases with exercise duration even at relatively low exercise intensities [24]. Prolonged exercise increases the spatial heterogeneity of pulmonary blood flow [32], as would be expected from compression of small blood vessels, and the extent of this derangement is correlated with ventilation–perfusion mismatch. Finally, increases in ventilation-perfusion mismatch are spatially localized in the basal lung where capillary pressures are expected to be higher with upright exercise [33].

In summary, the lung is vulnerable during exercise from both a mechanical stress and gas exchange standpoint. There is considerable evidence that stress failure contributes to the development of exercise-induced pulmonary hemorrhage and high-altitude pulmonary edema. As well there is evidence that less intense exercise stress is associated with interstitial pulmonary edema.

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Discussion

Comment: Thank you very much for a very nice overview. Would you say that every case of high altitude or exercise-induced pulmonary edema with blood going into the alveoli is preceded with this interstitial edema? Is it sort of a continuum where we first start with interstitial edema and then if it goes further, you go to bleeding or red blood cells?

S Hopkins: We do not have a lot of data to support that one way or another, but that makes logical sense. If you experimentally increase pressure you first get interstitial edema, and then if you increase it more you will get catastrophic failure. We see a lot of people that have evidence for interstitial edema with exercise. We don't see a lot of people who get overt alveolar flooding unless something else happens, although I've seen exercised-induced pulmonary edema as well. Interestingly, the one person that I know that I saw had exercised-induced pulmonary edema developed SIPE sometime later. Sometime after that he ended up having some kind of ventricular arrhythmia. I don't know that

he's ever gone back in the water, at least not for long periods of time. It's certainly reasonable that it could be considered a continuum. We just don't have the data to support that.

A Luks: Eric Swenson makes the argument but it's not just no failure or stress failure. That this is a progression of events related to the degree of elevation in hydrostatic pressure. Early on there is an increased flux of fluid out of the vascular space that eventually overwhelms the ability of the lymphatics and other mechanisms to reabsorb the fluid. Only at the very high end of elevation in pressure would you see capillary stress failure.

G Boswell: I'll present a little bit of data, which kind of throws some of that into question because that's a question that I had as well. I'll show you an example of what seemed to go straight to pulmonary hemorrhage without prior interstitial edema.

S Hopkins: I'll be interested to see how you're documenting interstitial edema. For example, horses get pulmonary hemorrhage. Based on the gas exchange evidence they get some interstitial edema, but they almost never get alveolar flooding. They just bleed.

G Boswell: I'll show you some examples of pulmonary hemorrhage as opposed to that, percent who all of a sudden has asystole, goes into CPR, we give them a lot of fluids, and they have that batwing pattern of pulmonary edema. We're not seeing that in our Navy SEALS. We're seeing interstitial edema or some mix with some airspace, or just purely airspace without interstitial edema.

S Hopkins: So interstitial edema based on chest x-ray?

G Boswell: Chest x-ray.

S Hopkins: The type of interstitial edema that I'm talking about for most of this is not radiographic. It's so subtle that you can't see it on chest radiographs. Also, there's always the problem of what findings are due to increased intravascular volume versus extravascular water.

G Boswell: The peribronchial cuffing that you're talking about is in the old literature. Dr. Moon referenced some of that, which is very hard to see on chest x-ray. We are talking more about central lines.

S Hopkins: As an aside, I do MR research and I keep telling our pulse sequence programmers that the holy grail would be to be able to tell intravascular from extravascular blood and water and to be able to quantify that.

V Maričić: When you mentioned this higher risk of stress failure one would have overinflated lungs. Trying to make this for freediving specific, I question how critical it is to emphasize divers who pack and the role of blood shift.

S Hopkins: Lung inflation is complicated because it depends where the capillary is in relationship to the epithelium. Whether it is situated like a bubble on the top of the vessel and it's getting stretched or whether it's elongated and supported. High lung inflation can be protective in some circumstances and detrimental in others. It's not a simple answer. You probably know a lot more about it than I do.

V Maričić: We'll talk about that especially whether it is blood shift and vascular engorgement. The other placeholder would be are HAPE-susceptible individuals also squeeze-susceptible individuals? Can we use the tools that we use to identify HAPE-susceptible individuals to identify squeeze-susceptible individuals?

S Hopkins: I wonder if the HAPEs are just the SIPEs in a different environment.

A Patrician: Thank you for a really good talk. To touch on the V/Q mismatch during exercise you talk about how it resolves over time. Do you know the time course of that resolution?

S Hopkins: About 20 min.

A Patrician: And is it more severity dependent?

S Hopkins: Let me back up on that answer. The gas exchange studies show that it resolves within 20 min. But our imaging studies where we exercised people for a long time and then looked at their distribution of blood flow in ventilation show that it can persist for up to an hour. But those are people lying supine in an MR scanner. The amount of V/Q mismatch that you get based on MIGET (multiple inert gas elimination technique) is correlated with the spatial disruption in blood flow that we take could be evidence for edema. So we think so.

A Patrician: The amount of V/Q mismatch, would that be equivalent to some sort of clinical threshold, or is this all kind of subclinical?

S Hopkins: This is all subclinical in athletes. What they tend to develop is regions of high V/Q arising because fluid leaking out of the capillaries compresses the vessels and disrupts flow. They have regions where they're ventilated but not perfused, i.e., they're not getting hypoxemia that way.

P Lindholm: Combining the SIPE and the squeeze issue, in SIPE we're thinking of this exercising, high flow, probably high pulmonary artery pressure and probably high capillary pressure. In the squeeze condition we have probably a relative negative pressure inside the alveoli. That might be like breathing on a regulator or a snorkel, with -20, -30, maybe -40 cm of water in the alveoli. There is different distention in the lung. If the alveoli are almost collapsed, can it still be that the capillaries engorge so much because of that, and rupture and through stress failure or is that actually protective? Basically, if you collapse the lung is it the collapsed part that is protected, and the open parts suffer the edema or the squeeze? We don't really know that.

S Hopkins: That is an open question, certainly something maybe we can thrash out a bit and get some ideas that we can test.

P Lindholm: The negative pressure we get in the airways is probably in the same realm or more than when you develop this really high arterial pressure from the exercise.

S Hopkins: Then you are going to have high transmural pressure as well.

B Smith: The circulation in the airways is less delicate than in the alveolus. They are not designed for gas exchange, it is a thicker barrier between blood and gas there. On partial collapse you are still going to have high capillary pressures. You are also going to have a folding force applied to those capillaries. The more deflated the alveolus, the higher the risk of stress failure until you get to the point of complete collapse where the walls are closed, and no pressure differential from blood to air.

S Hopkins: I also wonder about the role of surfactant and what happens to it. Are you going to talk about surfactant?

B Smith: Yes.

S Hopkins: Good.

C Seiler: Susan, how do you equal the edema with a V/Q mismatch? Edema is diffusion difficulties and V/Q mismatch is V/Q mismatch. It's either too much ventilation or too low perfusion. But how is edema going to be V/Q mismatch?

S Hopkins: When you take an animal and you experimentally increase mean capillary pressure, the sequence of events that happens is first you get interstitial fluid on the thick side of the blood-gas barrier. Then you get progressive alveolar flooding. That is from Norman Stabbs' work from many years ago. Everybody likes to seize on the idea that the interstitial edema increases the diffusion distance and causes diffusion limitation of oxygen transport. We do not actually think that happens, because the site of edema is away from the thin side of the blood-gas barrier where gas exchange takes place. The idea then is that you get fluid extravasation into the interstitium around small airways and small blood vessels and it squashes them, and starts to disrupt the distribution of ventilation and perfusion. Some of the imaging studies that we've done suggest that it doesn't really happen around the airways that much but rather all around the blood vessels. That kind of makes sense because that's before the fluid is in the blood vessels. It leaks out around the blood vessels, and it squishes them, and it disrupts perfusion. Make sense?

C Seiler: Yes

INCIDENCE OF RESPIRATORY PATHOGENS IN NAVAL SPECIAL WARFARE SEA, AIR, AND LAND TEAM CANDIDATES WITH SWIMMING-INDUCED PULMONARY EDEMA

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Background

Swimming-induced pulmonary edema (SIPE) is a respiratory condition frequently seen among Naval Special Warfare (NSW) trainees. The incidence of positive respiratory panel (RP) findings in trainees with a diagnosis of SIPE is currently unknown.

Research Question

Does a significant difference exist in the incidence of respiratory pathogens in nasopharyngeal samples of NSW candidates with SIPE and a control group?

Study Design and Methods

Retrospective analysis of clinical information from NSW Sea, Air, and Land (SEAL) team candidates with a diagnosis of SIPE over a 12-month period. Candidates who demonstrated the common signs and symptoms of SIPE underwent a nasopharyngeal swab and RP test for common respiratory pathogens. SIPE diagnoses were supported by two-view chest radiography. RP tests were obtained for a selected control group of first-phase trainees without SIPE.

Results

Forty-five of 1,048 SEAL team candidates received a diagnosis of SIPE (4.3%). Five had superimposed pneumonia. Thirty-six of 45 showed positive results for at least one microorganism on the RP (80%). In the study group human rhinovirus/enterovirus (RV/EV) was the most frequently detected organism (37.8%), followed by coronavirus OC43 (17.8%), and parainfluenza virus type 3 (17.8%). Sixteen of 68 candidates from the control group showed positive RP (24%) findings. Patients with SIPE and positive RP results reported dyspnea (94%), pink frothy sputum (44%), and hemoptysis (36%) more frequently than the control participants with positive RP results. Those who reported respiratory infection symptoms in both the study and control groups showed higher incidences of positive RP results ($P = .046$).

Interpretation

We observed that 80% of trainees with a diagnosis of SIPE showed positive results on a point-of-care RP. This positivity rate was significantly higher than that of RP test results from the control cohort. These findings suggest an association between colonization with a respiratory pathogen and the development of SIPE in NSW candidates.

Hypotheses on Potential Pathophysiological Mechanisms

Inflammatory and oxidative lung injury play a significant pathophysiologic role in heart failure decompensation by further damaging the alveolar-capillary barrier and increasing its permeability. As a result the pulmonary capillary hydrostatic pressure threshold for pulmonary fluid accumulation decreases. Could this be the case with SIPE? (Pappas and Filippatos, 2011).

Cytokines from active infection and/or colonization with a pathogen may impair function of the paracellular alveolar permeability barrier and promote pulmonary edema (Mazzon and Cuzzocrea, 2007;

Hocking et al. 1990). Local inflammation causing fragmentation of the macromolecular structures in the interstitial space of the lung results in lung fluid imbalance (Beretta et al. 2021).

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Discussion

O Christen: Does the swimming position have either left side down or right side down, how do they swim?

B Sebreros: They do a stroke called a combat side stroke.

O Christen: With fins or without fins.

B Sebreros: With fins.

O Christen: We use a similar method in freediving. One arm stays up and you use your fins. This sort of directs you where you are going and the fins are working but the lower part of your body would be fully extended while the upper part is relaxed.

B Smith: Do you think the weight of the heart has anything to do with that?

B Sebreros: What do you think Dr. Moon?

C Seiler: We also see this in our studies. Our patients just swim prone but we also see the right side most. Usually cardiogenic pulmonary edema, when it comes unilaterally, comes on the right side. There are anatomic differences in the lung and the mitral valve. If the valve is insufficient edema generally shows on the right lung. Maybe this mechanism or the lung might have something to do with it. If it's on the left it's unusual and you have swimming on the left because otherwise it should be the right side.

Comment: Pulmonary edema has a predilection for the edema to start in the right middle lobe. That's usually where you're going to hear the crackles first.

Comment: I was going to address the level of detail that you just did. It has been described both in terms of physical exam and findings, but the mechanism has not been well established.

S Hopkins: The lymphatic drainage of the right middle lobe is not at the same level as the other lobes. First of all, in the HAPE literature it is very well accepted that viral illness is a predisposing factor. We don't dispute that but don’t understand the mechanism. If you have a viral upper respiratory infection, what does that do to your nasal nitric oxide and how much does your nasal NO production affect your pulmonary arterial pressures? The second placeholder is what does it do to the extent of sympathetic activation? Because in HAPE we think that being sympathetically activated is important in part, HAPE is more common in environments that are colder. There is a lot of HAPE in Colorado, but we have mountains that are very similar in elevation and people do equally stupid things, yet we don't see nearly as much HAPE in California. Sympathetic activation seems to be important in HAPE so could that be a link that could tie some of this together?

B Sebreros: We have to give guidance. Do you think switching sides would help decrease SIPE during the combat side show?

A Lussier: It might be interesting to know which side people breathe on, whether shoulders are turning more to just one side.

A Molchanov: From people who have had these symptoms, what type of ability do they have and different ranges of motion? When people are not very flexible, like reaching out for the stroke or swimming in different styles, this will create different types of tension on their lungs. Not trying to reach and do anything with a bigger range of motion at depth when they're compressed, nor wanting to pull on the rope or reach too far is the same with breaststroke. In some cases it is unavoidable. We pull our range of motion and try to work on our ability and the rib cage, diaphragm, shoulder blades. With better mobility we see in freediving, for example, there are less chances of getting squeezed. Those who are stiffer, less flexible, will be likely be injured.

Comment: If you are a right-hand dominated person versus a left-hand dominated person you'll try to do more on the right side and not have the overall flexibility right to left.

Comment: Everyone has one side worse than the other. When you try to do the back stroke, usually the right arm is the worst one because we write a lot with the right arm.

V Maričić: From the exercise you say they're always on one side?

B Sebreros: They'll go back to center and go like this.

V Maričić: Yes but they're always doing it on one side?

B Sebreros: Some people do. It's just preference. They'll be swimming with a swim buddy. They've got to swim down to a buoy, about a half mile, and then turn around and swim back. Some people only feel comfortable doing it on one side.

V Maričić: From a kinesiological perspective it's less efficient in the long term so it will create more stress. We have noticed that there's a buildup of lactic acid, maybe this is going to be an extreme case because they have some respiratory movement, while in freediving we do not. This builds up into acidosis, which we found is very connected with what we call lung squeeze. This is a much smaller model because they are breathing and it's probably not such a long duration exercise. Just in terms of efficiency and limiting that factor it would make sense to alternate the sides.

Comment: Does it make sense that there's a geographical component where you're located that they would favor one side because they can see the shore one way versus looking out into the open ocean the other way? The sun is in their eyes in one direction versus not in the other because they start in the morning and it ends in the afternoon all the time?

B Sebreros That's a thought. On the way there, they're against the shore and on the way back, they're towards it. It could depend on what time they're swimming.

P Lindholm: There still seems to be more going to the right side.

2023 LUNG SQUEEZE SURVEY

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Objective

The purpose of this study was to collect information from adult breathhold divers and their experience(s) with lung injuries, particularly events commonly referred to as squeeze(s). The goal was to find conditions under which the signs and symptoms compatible with squeeze may occur. Based on the findings of this survey, we hope to create best practice guidelines for the diagnosis and treatment of a squeeze and to provide recommendations on how to safely return to diving after experiencing a squeeze.

Methods

Participants for this study were adult freedivers, recruited through DAN's social media channels, website, and personal invitations. Participants completed an online survey. Divers who believed to have experienced a "squeeze" or Pulmonary Barotrauma of Descent took an online survey. The questionnaire contained questions about the divers' breathhold diving habits, medical history, squeeze symptoms, and subsequent treatment. The study was approved by the DAN Institutional Review Board.

Results

We received and de-identified 132 (71% male, 29% female) full submissions for analysis. The participants' ages ranged from 20-74, with a median of 35 (31; 42) years. Their experience ranged from under 1 to 45 years, with a median of 6.5 (3; 11) years. Main breathhold diving activities performed were recreational freediving (122), competitive freediving (89), and spearfishing (36), amongst others. The participants reported various training frequencies ranging from daily (24%) to weekly (45%) to monthly (6%) to seasonally (24%). 129 (98%) of the respondents were certified by one or more organizations. The survey further asked about certifying agencies, medical history, and experience with lung squeeze events. 103 participants filled out one or more squeeze reports detailing symptoms, target and achieved depths of squeeze dive, personal bests, equalization techniques performed on the inciting dive, and subjectively perceived potential contributing factors for such events. Symptoms ranged from cardiopulmonary manifestations like cough, hemoptysis, chest tightness, or dyspnea to otopharyngological with sputum production of varying qualities, congestion, or voice change to neurological symptoms like fatigue and lightheadedness. Four squeezes were associated with a blackout, less than 20% received treatment in form of oxygen administration. The questionnaire further inquired about medical follow-up and fitness to dive determination post lung squeeze and if recommendations were received about wait times before resuming training or competition. The time to return to the same post-squeeze depth ranged from the same day to 4 years with a median of 10 days. 158 freedivers responded over the course of 6 weeks. 130 of the participants (70% male and 30% female) reported they had experienced at least one lung squeeze in their lifetime with 24 reporting that they had 10 or more (range 1-200) squeeze events of various degrees of

severity. The data analysis is ongoing and first official results will be presented at the Barotrauma and SIPE in Freediving workshop.

Conclusion

A preliminary data analysis suggested that squeeze events are a common occurrence and severity and treatment of these lung injuries greatly varies between individuals. A relatively low incidence of long-term effects warrants further investigation of yet to be defined repair mechanisms pertaining to lung injury and review of current return to dive recommendations. It also seems advisable to carefully review current course content of training agencies and educate freedivers about post-squeeze medical follow-up.

Acknowledgements

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Discussion

K Krack: You talked about positioning or orientation. How many would identify that they were in a bad head position or would be looking up on their way down, which could extend the trachea with strong contractions?

J Valdivia: Do you think we can match discipline with severity, age with severity?

F Tillmans: Yes.

V Maričić: This survey should be available a bit more because we can get more numbers and much more statistical significance.

O Christen: I already talked to our contacts in China. As soon as we have the medical clearance, translation in Chinese, you will get thousands of reports. The picture will change then. With China you have a lot of not so well qualified freedivers that do extreme stuff that they're not ready for. Then the picture will change. It will get really interesting at that stage.

A Luks: You don't have a well-defined entity as to what exactly squeeze is. You're asking people to complete a survey saying that they've had a squeeze without providing a definition of it.

F Tillmans: That is correct and also why we are here today.

A Luks: I get that is the point of this workshop. My point is it makes it hard to interpret survey data if people are reporting different things.

F Tillmans: I am totally aware that we probably have people that taste blood but don't actually spit out blood and think, maybe I had a squeeze, and people who really cough up the thick stuff and define that as a squeeze. We do not know what the severities were.

S Hopkins: It seems to me that, at least based on this definition, squeeze is you do a freedive and then you have respiratory symptoms. Is that a correct statement? Is that how I'm interpreting this? I can see several subcategories, for example, if you're pouring out pink frothy sputum, you have pulmonary edema. One of the challenges is to sort out stuff that can be put neatly in a category. It seems right now squeeze is defined as you have respiratory symptoms after you did a dive.

P Lindholm: Yes. That is one of the reasons why we convened this workshop because we need to sort that out. The survey was short, and it was actually aimed to check how this is worded and reported among divers.

Comment: It is interesting to see what it is that people are perceiving and experiencing. That in and of itself is valuable. To really think about the data you're collecting, some things are definitely more objective. There is a pulse oximeter measurement, pink frothy sputum. Dyspnea itself is a really challenging symptom because it's highly subjective and there are tons of different inputs. You don't have to be hypoxemic, and people can have dyspnea.

J Valdivia: We are talking about a squeezing syndrome. It has multiple symptoms. They're not all the same. But it's causation, diving, symptoms, squeezing. I would consider it like that since we don't know exactly what it is.

S Hopkins: It feels like the terminology is unclear.

F Tillmans: I agree with that. We struggle with terminologies in diving medicine everywhere. Ask Dr. Nochetto how many times a decompression sickness is a decompression sickness or may be some other manifestation. It's so multi-factorial similar to a squeeze, it is a mess.

P Lindholm: There is a need for granularity. I know John Fitz-Clarke has divided it into a few subcategories. We can all take a swing at it if we don't like it. There is something missing here, and I hope that we can agree on a few more definitions over these two days and also figure out whether some of it is just exercise-induced or diving-induced interstitial edema and not actual pathology. Some of it is a physiological response to going in the water.

A Lussier: From the freedivers and from the SIPE researchers I've seen a couple presentations today that mentioned the lack of this presentation in older people. Is that a survey selection bias that older people don't freedive or they don't get SIPE from some sort of age-related lung structure and function?

R Moon: Older people do get SIPE. I've seen many people clinically who have been in their 60s and 70s. I do not know about squeeze, whether it's just older people don't freedive, but I have no information on that.

M Nochetto: With SIPE what we normally see at DAN is there seems to be a high prevalence of females in the 50-yr range.

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LUNG SQUEEZE: A KEY FACTOR IN DEEP DIVING

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Background

My background is in sport performance. As a professional athlete for quite some time in several sports I came into freediving and started making results and world records really fast. I started squeezing quite badly along the way and wanted to research its safety and whether I should stop diving. My experience is as an instructor and coach for 10 medals and 8 world records in the world championships in 2023. Most participants in my online and physical coaching platforms for two years attained 100 m in depth, affirming the methodology I brought in from other sports.

Symptoms

I have experienced more than 100 squeezes underwater but am much less sensitive to HAPE in the mountains. As a professional sports climber and expedition leader I only experienced HAPE once during a stressful, physically demanding rescue without oxygen and no medication but for blood thinners in the Andes between 6-7,000 m. I have not been able to make a correlation between squeeze and HAPE.

Other symptoms include many blackouts and edema on practically every deep dive. Of the hundreds of divers I have researched only two have had absolutely no edema, barotrauma or fluid in the chest. However, many are unaware of the symptoms but feel tired or other issues, but these are physiological disturbances. Anything out of the ordinary concerning the respiratory system after a dive, even mild, could be considered a squeeze, regardless of the diver's resistance.

Limitations

AIDA holds on average eight competitions yearly, allowing for the sampling of safety teams and victims. We personally treat the freedivers because of our interest in the symptomatology, treatment modalities and putting that knowledge to use with the athletes that I coach and train. I have determined that in very deep diving (100m + dives), there are currently three limitations: system acidosis, lung squeeze and narcosis.

Equalization is not a problem if properly trained. Hypoxic events occur in untrained divers or those who have some kind of lung squeeze that then creates a response from the body to use more oxygen or not being able to utilize the body's oxygen stores. These issues are all connected and emphasize each other.

System acidosis is interesting, a closed-circuit phenomenon at depth where the glycogen in the muscle is only used locally because of the restricted peripheral circulation. The lactates do not enter the blood flow so quickly. A diver will feel it only after surfacing, when it manifests extremely. We have measured 25mmol of lactates from 30 to 60 mins after a dive. It is unknown how the body is going to cope with it.

Narcosis susceptibility is highly individual. It is hard to understand at the beginning, especially with shallow divers. But realize that it is very prominent already on a 30-m dive. There is a high statistically significant difference between someone who is coherent or not at some cognitive task at 30-m dive, after a 30-m dynamic, and after just submerging or dry. The way it affects divers at deeper depths and the connectivity with blackout is interesting and deserves exploration on how to objectivize it. My own

extreme cases of narcotic blackout were extremely unpleasant. Luckily, I woke up when I heard that someone was about to cut w wetsuit and realized it was mine.

There could be a connection between this systemic acidity and the reaction of the brain producing cytokines and nitric oxide, i.e., a vicious triangle (Gasparini et el. 2022). The CO_2 is building up and not really getting back to the lungs. We have also seen a post-dive extreme immune response. There is possibly some gas bubble damage on ascent. Many unanswered questions remain on deep dives and residual volume (RV) dives. There appears to be a big difference in the mechanism of injury between the RV dives and the deep dives, which are much more aggressive but produce more significant serious damage.

Concerning gas bubble damage I figured out my squeeze happens closely related to time of immersion. I researched different types of stretching, nutrition, sleep habits, warming up versus not warming up, different temperatures and suits. Common thinking was to proceed gradually. I was already a 100-m diver, but I started the season with 10-m dives performing 300 dives up to 45 m depth. This gradual approach did not change a thing. I realized with long 4–6-minute hangs in water, regardless of the depth and the RV, I got squeezed. This surge that I feel and come up and notice there is this squeeze symptom and a bit of blood. It is subtle and I would not understand or recognize it if I did not see this. The squeeze is usually apparent on the ascent between 40 and 20 m, which leads me to believe it may have something to do with re-expansion of the lungs.

System acidosis is the critical part of the dive manifested as extreme muscle pain, and vomiting reflex similar to an 800-m runner doing a tolerance test. A diver in a half-conscious state after a quite short blackout understands where he is on a subconscious level. The way he is approached and communicated with impacts the way that he responds and comes back and the emotion that he will perceive after the dive. The important things are how the problem is addressed, how the diver is treated, his body position and whether there is fluid in the lungs (Barković et al. 2021). In most cases the best thing to mitigate acidity is respiration rate. For narcosis, prevention of DCS, and any kind of squeeze symptoms administration of oxygen preferably under water.

In an extreme lung squeeze the bleeding can go on for another day due to severe capillary rupture and followed by significant edema where the diver is half conscious and needs oxygen in order to walk. In one particular case the diver was fully recovered after five days and did a deeper competition dive and was perfectly fine. If you know how to recover, how to assess and you feel good after that, excluding the mental variant, then so far in our small sample size of experience everything is quite treatable and workable.

Adaptation

The warm-up is a contributing factor and one of the most important things. The old school warm-ups where you spend a lot of time in the water doing hangs can do two things. They can either do nothing so it is a waste of time, or they can create a mild pulmonary edema, start a lung squeeze, or create a small capillary leak, which may not be noticeable at first but will be on a bigger dive because the lungs are quite sensitive to fluid. It is difficult to connect the symptoms with the final outcome, which depends on how the diver feels the next day regardless of whether having experienced mild or severe symptoms.

Several events occur when we dive: immersion pulmonary edema happens anytime we jump in the water; some kind of barotrauma of the capillary network; and fluid in the lungs. Fluid can move through the membrane due to pressure. There could also be bubbles passing through the membrane causing micro damages. Divers who present with endemic symptoms can be distinguished from those with barotrauma. Oxygen rapidly aids recovery. Shallow dives, aggressive dives usually have short mechanics that create barotraumas. Deep dives and hangs usually have long mechanics that create more edema.

Most adaptation is not necessarily due to the time spent in the water but appears to be mental, the emotion connected to squeeze that creates fear. Rather than a physiological adaptation, managing the emotional response to narcosis results in relaxation and changes the symptoms experienced after the dive.

Some imaging shows a total bronchiolar collapse, which can happen in fully exhaled lungs under pressure. The air spaces that remain are the larger bronchi. The point of break is probably around that segment that cannot collapse because of its large size. The even larger segment seems to be quite flexible, and we have found no hard evidence of tracheal squeeze. The trachea appears to be quite flexible and can somehow change and collapse better than the bronchi, especially around the right side. In the majority of cases, the diver experiences pain either centrally or predominantly on the right side of the lungs. This may be closely related to equalization based on the measured volumes of the uncollapsed parts compared to volumes that used for equalization, particularly for divers who have a well-developed larynx movement. This can add a lot of volume and is good for the packing technique but can create much stress on the collapsed area.

Prevention

Preventative measures include several tactics: body position, head position, and how and when to equalize. A preferred treatment position for drainage of blood or fluid depends on the severity of the situation. Hips can be elevated even more, which is gentler because gravity helps resolve the fluid. As fluid is moved from the vasal region there is a larger surface area to reabsorb resulting in more oxygen saturation that allows the body to recover fast. Coughing may trigger more damage. It is better to redistribute blood and fluid versus have it stay in the vasal parts and coughing up drier old blood for days. One of the reasons we do decompression upside down is to provide positive pressure on the upside lung for a more effective, cleaner method to spit fluid out. This method is also efficient for recovery from decompression sickness symptoms, especially with oxygen.

Return to diving

Excluding subjects that are over emotional or exhibit fear is the thing to do. From our experience, the next day is usually good to go, but for extra safety and comfort a day or so off is good, especially before a big performance. There I not much correlation for a risk of recurrence unless the diver is mentally weak of overly emotional. Reassuring people helps a lot and changes squeezing patterns just by having a conversation that it is not going to kill them, and they do not need to hide it, and everything is going to be okay.

Assessment and Monitoring

Self-awareness is preferable to ultrasound, which produces strange results. Vital capacity is preferred. Oxygen saturation devices range from good to bad. How cold is your hand, how constricted are you? Sound of the lungs and dyspnea under load. RV dives and RV statics are going to be much better. In the long term there is the question of whether the diver will be damaged. In the last 16 years CT scans were done three months, one month, one week after the season to determine any changes after squeezing, and there were none. The more we squeeze, the more we improve and the deeper we can go without squeezing.

In 2019 an initial questionnaire on squeeze was provided by A. Molchanov for documentation and classification. Research needs to include divers that have a connectivity with science or engineering. Often the research that is being conducted is not in any way connected with freediving, rather done to be published. A distinction between sports and recreation needs to be made as O. Christen Drew mentioned. As the sport grows, performance grows, and we need to increase our knowledge base to provide a safe progression and performance increase for everyone. Subjective rules foster fear and non-reporting because of one incident that resulted in a tragic death were many things were done wrong. It is not so much about the injury as it is about the need for open communication about treatment as is the case in

other sports (e.g., swimming and triathlon. Competitive freediving is an extremely small sport, which grows an extremely strange community because people without any experience as athletes can come in and participate in a world championship as their first attempt to achieve a national record. There are many possible world records in freediving compared to other sports, which has a huge impact on their ego. Everyone is after some record.

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IS THERE AN INFLAMMATORY COMPONENT IN SIPE AND SQUEEZE?

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Objective

Immersion pulmonary edema has traditionally been considered a cardiogenic edema, but pulmonary edema is also highly dependent on permeability. The physiology of cardiogenic vs permeability edema will be discussed. We have an ongoing study on whether exhaled fraction of nitric oxide (FENO) would be correlated to SIPE or squeeze.

Methods

Freedivers are invited to be tested for exhaled FENO and correlated with SpO2 and symptoms of squeeze or sipe.

Results

Data from a limited number of measurements in recent competitions such as Vertical Blue 2023 and the CMAS WC 2023 was presented supporting the hypothesis and further data collection. 22 divers in vertical blue and 13 in CMAS competition in Honduras 2023 performed a total of 74 dives where post-dive FeNO was 17±11.4. 8 dives had FeNO>30, and of these, 7 endorsed symptoms of "FIPS" upon presentation.

Conclusions

The preliminary data suggests that airway inflammation could be a risk factor for squeeze symptoms and that its worth pursuing additional data collection for better statistical validation.

Reference

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THE BIOMECHANICS OF SIPE AND SQUEEZE: LESSONS LEARNED FROM ACUTE AND VENTILATOR-INDUCED LUNG INJURY

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Introduction

At first glance the forces of positive-pressure ventilation may appear profoundly different from those caused by hydrostatic pressure during surface swimming, snorkeling, freediving, and scuba. However, at a microscopic level these events share common mechanisms that could help clarify the biomechanical forces that cause swimming-induced pulmonary edema (SIPE) and the acute effects of breathhold diving (squeeze). There are three widely accepted causes of damage in ventilator-induced lung injury (VILI): overdistension (volutrauma), cyclic collapse and reopening (atelectrauma), and inflammation (biotrauma). These causative mechanisms are potentiated by the spatial heterogeneity of the lung tissue and stress distribution.

Volutrauma

Volutrauma is widely recognized as a driver of VILI and a reduction of volutrauma is the basis of the ARDSnet low tidal volume ventilation guidelines that have been prospectively shown to reduce mortality in critically ill patients. At the microscale the mechanism of volutrauma is relatively simple: the delicate alveolar septa are over-stretched leading to cellular injury, alveolocapillary barrier disruption, and the subsequent injury cascade. The idea of volutrauma occurring during subsurface swimming may seem highly counterintuitive since lung volumes are *reduced* by the external hydrostatic pressure. When considering a homogeneous lung this is indeed the case as the overall volume is reduced, and thus the *average* septal strain is also lowered. However, the changes in lung volume are not uniformly applied in either mechanically ventilated or diving subjects where, for example, the regions adjacent to the diaphragm may collapse first.

Regional collapse leads to stress concentrations at the boundary between aerated and atelectatic tissue. The reduced volume of the collapsed region exerts a traction force on the adjacent patent alveoli, which can induce deformations that are several times greater than normal inducing localized volutrauma that could contribute to SIPE and squeeze. The closure of conducting airways may also lead to localized overdistension on ascent. Here, a region of compressed gas is isolated from the rest of the lung by a collapsed segment of the conducting airways. As the diver returns to the surface and the external pressure is reduced the volume of that trapped gas will increase, resulting in localized overdistension, until the pressure in that isolated gas is sufficient to reopen the collapsed airway segment and restore an even distribution of pressure in the parenchyma.

Atelectrauma

Atelectrauma is damage caused by fluid-mechanical forces during the collapse and reopening of alveoli and small airways. This is a driver of injury in ventilated patients but may not be a primary factor in SIPE and squeeze. During normal respiration, the alveoli change size to accommodate changing lung volume through recruitment/derecruitment (i.e., opening and closing), the folding and unfolding of septal pleats (which is a form of recruitment/derecruitment), shape change, and stretching of the alveolar septa. In the healthy lung, with a functional pulmonary surfactant system, recruitment/derecruitment and folding/unfolding occur without injury. When surface tensions are elevated, which is often the case in the injured lung due to dysfunction of the surfactant system, these events cause greater stresses and strains that induce injury. This highlights one potential avenue for atelectrauma during swimming and diving: an

initially unhealthy diver with, for example, a respiratory infection may suffer injury during surfacing and lung re-expansion. However, this may not be a common occurrence since a lung injury that induced surfactant dysfunction could discourage participation in swimming and diving.

What is more intriguing is the potential for an escalation of injury in repeated dives. Here, on the initial dive, a degree of pulmonary edema may occur from other mechanisms such as increased pulmonary capillary pressure or regional overdistension. As is the case in acute lung injury and VILI, edema will carry plasma proteins that reduce surfactant function and cause an increase in surface tension. Subsequent dives, and the external pressure-induced atelectasis, may then yield atelectrauma as those regions reopen in the presence of higher surface tensions. The effects of extreme lung compression on the physical structure of the pulmonary surfactant monolayer that lines the alveolar epithelial cells may also be an important factor. Extreme compression of this air-liquid interface during mechanical ventilation has been shown to quickly degrade surfactant function leading to elevated surface tension which is associated with increased damage during recruitment (reopening) of alveoli and small airways. This may explain, by way of evolutionary pressure, the differential surfactant composition and function of diving mammals which is postulated to protect against surfactant dysfunction, and potential atelectrauma, during re-expansion from complete alveolar collapse.

Barotrauma is a somewhat contentious concept in the mechanically ventilated lung. While it seems obvious that applying high ventilation pressures will damage the delicate lung parenchyma, experimental evidence suggests that high pressures are not inherently damaging. In those studies, the expansion of the lungs was limited by bands around the thorax so that high pressures could be applied without a concurrent increase in lung volume. When distension was limited, the high inflation pressures could be applied without substantial damage. The same likely holds true in SIPE and squeeze where the application of high external pressure is unlikely to cause direct damage to the lung tissue. Instead, those high external pressures are likely to induce injury through other mechanisms. However, the effects of high external pressures are likely to have an effect in the pulmonary capillaries. Here, pressure-induced blood shift to the thorax, along with the diving reflex, causes an increase in capillary pressure. Concurrently, the pressure in the airspace may be effectively reduced at depth as the chest wall resists total collapse. These two factors yield a transmural pressure that pulls fluid into the airspace.

Biotrauma

Biotrauma is an important mechanism in VILI, particularly in the pre-injured lung. The effects of ventilation on pro-inflammatory gene expression can be observed after only a few minutes of mechanical ventilation. However, protein expression and the pro-inflammatory cascade take longer to manifest. As such, the effects of biotrauma may be a factor in people who dive many times per day for food collection or sport, and lead to a hyperinflammatory state. This inflammation does lead to an increase in alveolocapillary barrier permeability but is unlikely to be a contributor to acute SIPE and squeeze events.

Summary

The acts of surface swimming, snorkeling, freediving, and scuba induce mechanical forces on the pulmonary system that conspire to increase localized stress and strain that can lead to SIPE and squeeze. Prior studies of ventilator-induced lung injury suggest that the tethering forces induced by heterogeneous collapse are of particular importance and may cause injury on both descent and ascent.

Discussion

R Moon: Clinically, atelectrauma occurs in patients who are being ventilated many times a minute over hours and days. Whereas here we're talking about a single dive. During general anesthesia the incidence of atelectasis is close to 100%. Yet patients don't get lung injury or anything like that. Clinically, we give them big breaths every so often and the atelectasis resolves, and the saturation

comes up. You portray an interesting potential pathophysiological mechanism, but I'm not sure that it fits with what we see clinically.

B Smith: I tried to couch that one. I can maybe see it happening if you're doing 50 dives a day. We can kick some of that stuff off with a few minutes of ventilation, but if everything is well and good, you can collapse things, pop them back open, and the lung will be happy.

Comment: 50 dives in a day is easy for an instructor. That can happen in two hours. It's not just the deep performance dives.

P Lindholm: One of the triggers known to cause squeezes are contractions while deep diving. You're swimming around and your lungs are not full. My interpretation of that would be if that lung had some damage to it or collapsed, you're more likely to get trauma by stretching that. What if you have a lung that's partially collapsed, and you get these negative pressure swings from these diaphragmal contractions like “trying to breathe” in? If my lungs are completely nice, homogeneously open, all the alveoli can take that stretch and that pressure. But if you have only a part taking all of it, is that the volutrauma you showed there that could be the cause of those contractions?

B Smith: If you didn't have a contraction and managed to reopen the lung, it is probably not going to cause that much damage because it's like one reopening event. Where the risk is, in that overstretched region between open and collapse where the tethering of that smooshed down region is expanding out to the open region and you're exerting this traction force on the closed part with your exertion, and it's getting transmitted to that already overextended region between the open and collapsed.

P Lindholm: The edge or the border is where you get the tension?

B Smith: Yes, and even if it's evenly distributed in the rest of that open lung, that region has already been overstretched and it's going to be at higher risk for that tension. If you pull a little bit more, you've gone from okay to tragic.

P Lindholm: Would you not call that a contusion?

B Smith: It’s tensile.

P Lindholm: Tensile stretch?

B Smith: Yes. As opposed to the shear of, like, rubbing together.

P Lindholm: Shear is rubbing together. So tensile stress.

V Maričić: The reopening tension, which is bigger than closing, we could eliminate by exhaling on surfacing right? Another thing we considered many times and never actually found a good way is how to increase or develop surfactant faster. Do you have an idea on preventive measures that would create more surfactant? And is more surfactant better? Could this be done or something that's protective or a recovery measure after the dive?

B Smith: If you want to get weird, you can get exogenous surfactant and can squirt it into your own lungs. It's like there's a stash of this stuff in the lungs already that builds up just as a normal course of the cell’s existence, and it's responsive to stretch. Like a deep sigh stimulates those cells and they release the surfactant molecules. You could prime yourself, potentially, with a few deep breaths to get that surfactant released.

V Maričić:: We do some packing and releases before, and it seems to help to pack more comfortably and to dive more comfortably.

B Smith: In terms of recovery?

V Maričić: No, I mean I've read that steroids can affect surfactant.

B Smith: How to increase the intracellular supply, to have a bigger reserve? The most direct is the deep breaths. The packing, even if it's not necessary, to get that few extra meters worth of air in there, it's going to be helpful in terms of getting that surfactant released and getting ready to reopen more easily on ascent. In infants what they do on a ventilated unit, they disconnect the ventilator, squirt the surfactant in, they rock the baby around, put it back on the ventilator, and it's a life-saving intervention.

Comment: It was tried in adults and it did not work.

B Smith: No. But maybe because they didn't use enough.

Comment: Just one more on surfactant and geared towards how we currently treat squeeze. Does concentration of oxygen impair the recovery of surfactant? We have squeeze. We have blood in there, have less surfactant if we add tons of oxygen or are we delaying the recovery of the surfactant?

B Smith: I was really worried about the oxygen thing because I thought you guys were sucking 100% O_2 for four hours straight. I think in the acute exposure, 10 to 15 minutes, it's fine. I won't say that super confidently. I believe it's fine because it's such a short exposure. Recovery from that hypoxemia is much more important than what I would perceive to be a minor effect of the surfactant. I was more worried about oxygen toxicity.

SIPE IN RECREATIONAL SWIMMERS: POTENTIAL MECHANISMS FOR SEX AND AGE DIFFERENCE IN PREVALENCE

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Background and rationale

The alveolar-capillary membrane enables efficient gas-exchange and maintains lung fluid balance [1]. Heavy exercise and immersion in cold water challenges this two-cell-layer thick barrier between air and blood [2]. Exercise-induced pulmonary edema has been reported in racehorses and certain dog breeds, as well as occasionally in marathon runners [3]. Swimming-induced pulmonary edema (SIPE) and scuba divers pulmonary edema (SDPE), both referred to as immersion pulmonary edema (IPE), occasionally occur during surface swimming and diving [4,5]. A combination of factors contribute to elevated pulmonary capillary pressure and a subsequent hydrostatic pulmonary edema; central pooling of blood and peripheral vasoconstriction on immersion in cold water together with increased cardiac output during exertion. In addition, capillary stress failure due to increased vascular pressure and shear forces during heavy breathing can cause fluid leakage into the interstitial space [6].

Historically, case series of men have dominated the literature of SIPE and IPE including predominantly military trainees, triathletes or recreational divers [7-11]. However, sex-mixed cohorts have reported a higher incidence of both SIPE and IPE in women [12-15]. Based on our cohort of 47,000 recreational swimmers during Vansbrosimningen in Sweden, about 90% of the 211 SIPE cases were women despite an equal gender distribution among participants (Fig. 1) [13]. Advanced age was also associated with a higher incidence of SIPE. Altogether, the average incidence of SIPE during Vansbrosimningen was 0.44% but up to 1.1% in the oldest age group (>61 years) (Fig. 1). Increased risk of IPE with higher age, and cardiovascular risk factors, has also been suggested in divers [10,14,15].

The following discussion expose disparities in cardiopulmonary physiology based on sex and age. The intention is to link these differences to the pathophysiology of SIPE/IPE.

Let's talk about sex

Beginning in the embryonic phase and continuing throughout infancy, lung development differs between sexes [16]. As a result, the respiratory system differs in several aspects between adult men and women. Even after matching for height, women have smaller lung volumes than men and less total number of alveoli. Women also have smaller-diameter central airways (i.e., trachea to the third generation airways) than men (luminal areas about 15-30% smaller), after matching for lung volume and height [17 18]. Morphologically, men have more "pyramidal shaped" lungs with greater lower lung width and women more "prismatic shaped" lungs with more similar widths at upper and lower parts [19]. This fits with slightly different breathing movements with men using the large diaphragm muscle to a higher extent during inspiration compared to women [20,21]. Regarding elastic properties of lung parenchyma and chest wall, there are no major differences between the sexes [22]. Prevalence and outcome of all major respiratory diseases differs between men and women [23]. Asthma is more prevalent in adult women compared to men, and worsening of asthma symptoms before menstruation and during pregnancy suggest that sex hormones may play a role in the progression of the condition. Another respiratory phenomenon, more common among young women, is exercise-induced laryngeal obstruction (EILO) [24].

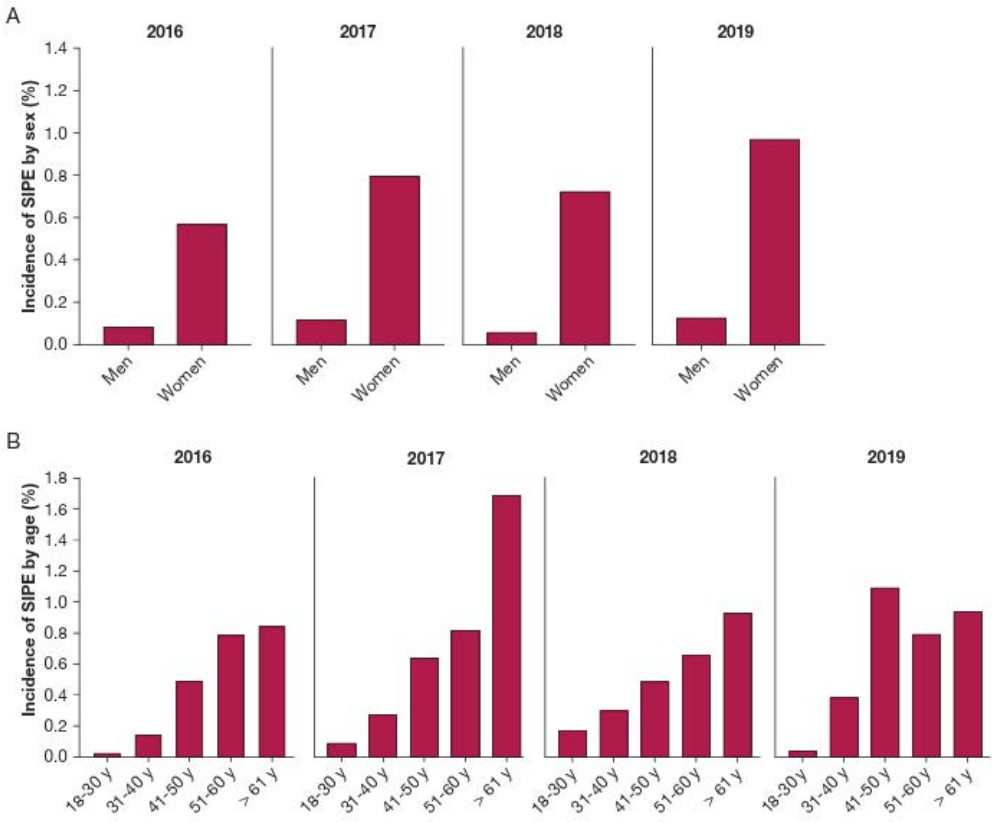

Figure 1. Age- and sex-dependent incidence of SIPE during Vansbrosimningen in 2016-2019. Cumulative incidence reported for men and women (A) and by age group (B) as follows: 18 to 30 years, 31 to 40 years, 41 to 50 years, 51 to 60 years, and 61 years or older. SIPE = swimming-induced pulmonary edema. From Hårdstedt et al., *Chest.* 2021;160(5):1789-98.

There are fundamental differences between men and women in cardiac and vascular anatomy and physiology [25]. Women have a smaller left ventricle (LV) and therefore lower stroke volumes. However, a higher resting heart rate maintains a similar cardiac output as men. The LV stiffness (elastance) is overall higher in women, both in systole and diastole, compared to men at a given age and this difference is accentuated with higher age [26]. Arterial stiffness increases with age, and more so in women after menopause [27]. Women have a narrower aortic arch, which might also contribute to an earlier pulse wave reflection and higher pulse pressure [26]. The concept of ventriculo-arterial (VA) coupling concludes that a higher arterial stiffness increases cardiac workload as well as LV wall thickness and stiffness, which will eventually affect the diastolic relaxation capacity [28,29]. Women and men are known to respond with different LV remodeling strategies. Women tend to develop a more concentric hypertrophy of the LV (normal LV mass index but thick walls) compared to eccentric hypertrophy of the LV in men (large LV mass index with comparable thinner walls) [30]. It is thought that these inherent disparities between sexes might explain why women are more likely than men to develop heart failure with preserved ejection fraction (HFpEF) [31]. A cardinal symptom of HFpEF is the reduced ability to cope with physical exertion. Men, on the other hand, have a well-known higher incidence of ischemic heart disease with a higher prevalence of heart failure with reduced ejection fraction (HFrEF). Interestingly, other aspects of cardiovascular morbidity also differ between the sexes, e.g., women having a higher prevalence of idiopathic pulmonary arterial hypertension and stress-induced cardiomyopathy compared to men [25]. Here, other mechanisms such as level of estrogen metabolites and sex differences in the immune system might play a role [32].

Implications of sex for SIPE and IPE

Immersion in water has an overall impact on respiratory physiology resulting in: (1) decreased expiratory reserve volume (ERV) and vital capacity (VC); (2) decreased overall lung compliance; (3) increased negative alveolar pressure; and, (4) increased work of breathing (WOB) [33,34]. These changes contribute directly or indirectly to an increased transmural capillary hydrostatic pressure difference within the alveoli. The large conducting airways (i.e., trachea to fifth generation bronchi) are responsible for the majority of airway resistance during breathing [22]. During heavy exertion requiring high airflows women have higher airway resistance creating turbulent airflows in the central airways. This results in a higher WOB, thus a higher relative oxygen demand of the respiratory muscles in women [22]. One could also speculate that narrower airways could predispose women for SIPE/IPE by increasing the negative alveolar pressure at immersion, especially during high respiratory constraints. In a case series by Shupak et al. small lung volumes and higher supposed obstruction in small and middle sized airways (measured as $FEV1_{25-75\%}$) were associated with SIPE in a group of military swimmers [8]. Interestingly, we could conclude in our cohort from Vansbrosimningen that comorbidity of asthma was associated with prolonged symptoms of SIPE, but also higher recurrence of SIPE over 30 months [35].

Postmenopausal women will, based on higher LV and arterial stiffness, be less able to withstand a high end-diastolic pressure and are more likely to develop pulmonary edema under increased volume load. Peripheral vasoconstriction in cold water, changes in buoyancy, and increased surrounding hydrostatic pressure perhaps in combination with a tight wetsuit result in central pooling of blood with up to 700 ml during immersion [36,37]. In combination with physical exercise while swimming in cold water, cardiac preload and afterload as well as central vein pressure increase. The diastolic relaxation of the LV will be crucial to maintain cardiac output during volume overload (the Frank-Starling mechanism) [38]. If the LV fails to keep up with the increasing demands, right and left ventricular mismatch will inevitably result in pulmonary edema [39].

How old is old? – Aging, cardiopulmonary function and SIPE

In our cohort study of SIPE incidence during Vansbrosimningen, middle-aged women (about 40 years and older) dominated. We know that aging comes with various changes of cardiopulmonary physiology. The lung matures by age 20 and lung function remains steady up to about 35 years, whereafter a progressive decline in lung function occurs [40]. Total lung capacity remains unchanged with age, but residual volume (RV) increases which results in a reduced vital capacity (VC). Aging results in decreased muscular strength and stiffening of the chest wall together with loss of elastic recoil of the lungs ("senile emphysema"). Overall lung compliance increases and chest wall compliance decreases, which results in a higher WOB at exercise [40].

Cardiovascular morbidity increases with age e.g., hypertension, diabetes, obesity and ischemic heart disease. Hypertension has been identified as a risk factor for SIPE and IPE [10,12,41]. Systemic hypertension typically leads to LV hypertrophy and abnormalities of LV systolic strain but spares the right ventricle [42]. Ischemic heart disease and LV hypertrophy in male recreational divers with IPE has been reported [10]. Both diabetes and obesity are associated with LV diastolic dysfunction, and more common with age in women. As discussed above, LV stiffness increases with age in both sexes leading to an age-dependent loss of LV diastolic function [26]. Elevated left end-diastolic pressure due to volume overload during exertion in cold water will lead to an elevated capillary hydrostatic pressure.

Conclusion

The balance of interstitial fluid in the air-bearing alveoli of the lungs has evolved over many hundred millions of years for a life on land. *Individual characteristics* (e.g., age, sex, comorbidity, fitness level, swimming skills) and *level of exposure* (e.g., water temperature, depth, tightness of wetsuit, degree of exertion, stress) will be decisive for development of pulmonary edema during immersion and exertion in

cold water. As presented, cardiopulmonary differences between sexes and with higher age can give us clues to differences in SIPE/IPE incidence. In reality, exposure is often associated with sex and age, which makes it hard to isolate the effect of each of these traits.

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Discussion

A Luks: Thank you for a really interesting talk. Regarding a large event like this, compared to the model that Dr. Moon has where they have a swimmer isolated in a tank in a very controlled environment, you have 10,000 people swimming in a river near each other with lots of turbulence in the water. Do

you think that some of the cases getting called SIPE are actually just aspiration from swallowing water from all that motion?

M Hårdstedt: Actually, that is why we recruited Claudia to the team. I heard about a doctor in the hospital that was really good with lung ultrasound. Since 2018 we do lung ultrasound on everyone that comes for respiratory problems in order to conclude whether they have pulmonary edema or not. We have collected data on the swimmers' experiences of aspiration during the swimming race, symptoms and lung ultrasound findings. In our data of four years now some swimmers with respiratory problems report aspiration and some do not. I believe that is more connected to problems during swimming, bumping into someone or losing your pace. We have not been able to associate aspiration as such with SIPE on lung ultrasound.

M Nochetto: In cases when we send divers for evaluation, they often come back with the result of water aspiration and blaming it on the regulator diaphragm being torn or malfunctioning. But the pattern will be different. I challenge anybody to be breathing water and not realize they are breathing water.

M Hårdstedt: We do not have the possibility to make x-rays or CT scans at the riverside. It is 80 km to the nearest hospital during the swimming event. Ultrasound was a way for us to be able to distinguish pulmonary edema from other respiratory problems, to actually prove that they had edema. We had been listening to the lungs of these patients, and during auscultation it sounded like pulmonary edema, but I couldn't publish in a scientific paper that "it sounds like pulmonary edema". The results from lung ultrasound have helped a lot.

O Christen: I find it so interesting when you say that women have a tendency to be more chest breathers and less diaphragmatic breathers. I work with a few really strong young guys like bodybuilder style, SEAL style who were also poor breathers. They were mostly chest breathers and did not use the diagram at all. Does it have something to do with chest breathing that when you were swimming on a high load, that you are breathing incorrectly or inefficiently because you use the lungs and not the diaphragm? Could there be a connection?

M Hårdstedt: I can't answer that question. One thing that matters is how you swim in the water. We can see that those who are experienced swimmers have a more horizontal position in the water. We seldom see those experienced swimmers in the SIPE tent or the CPAP container. It's usually those with slower breaststroke style. The lungs are deeper into the water.

C Seiler: Swimming with dry hair.

M Hårdstedt: I think it's really great that many different people participate in this swimming event. It's all ages and both sexes really. Participants are 10 years old to over 80. We have never had a patient with SIPE younger than 18 years old, none. It is definitely an effect of age.

Comment: On the age issue it is just more likely that the older participants will be less trained. It's not related to age as much as this group of people didn't train enough for years. It doesn't have anything to do with age. 60, 70-year-old people can be more trained than 30 or 40 year olds. If they don't train, they're in worse shape. We have good examples of our freedivers in very old age, performing well, without any problems and no squeezes. We have some freedivers who are 80 years old and holding their breaths.

M Hårdstedt: Our hypothesis when we started the study was that people were not exercising enough, at least not practicing open water swimming enough prior to the race. Now that we have studied SIPE over the last years, the patients are pretty well trained, but still mostly exercising on land. They are runners, bicyclists, skiers, but a majority do not swim regularly in open water.

Comment: It is just a more stressful environment?

M Hårdstedt: Yes.

Comment: The ones who have really good swim positions are good swimmers, so they don't present with SIPE.

M Hårdstedt: Exactly.

R Moon: Thank you for a wonderful outline of male/female differences that might impact on this topic. You compel me to go back and look at my data in terms of male/female.

M Hårdstedt: It's interesting, isn't it?

R Moon: Yes. Thank you.

A Lussier: I just had a quick thought about the Navy SEAL kind of experience and what you're presenting. If you have a set of twins who present with SIPE at the same time, possibly related to collagen or something within the vasculature walls or within the parenchyma of the lung. If you have someone who on oral contraceptives you can have an increase in collagen breakdown, which in women could maybe explain it.

M Hårdstedt: This topic has so much to talk about. That's one thing, estrogen is actually affecting the collagen balance.

B Smith: I think if you experimentally try to reproduce your curves, that would be great.

M Hårdstedt: I like that graph. I would have loved to have done it in a three-dimensional format if I was good enough.

P Lindholm: I remember a 60-70 year old diver who had done 11,638 scuba dives and suddenly he got pulmonary edema of immersion while swimming around in the Maldives. What happened there was probably the age-related development of one of the risk factors like hypertension.

SIPE: IS IT MORE THAN JUST EDEMA? PATTERNS AND POSSIBLE MECHANISMS

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Introduction

Our clinical experience in interpreting several hundred chest radiographs of active duty military undergoing training to become members of elite special operations units has demonstrated different patterns of swimming-associated lung injury.

Interstitial edema

The most common pattern is one of interstitial edema, which on occasion is associated with alveolar edema. These subjects report pink frothy sputum. Unique in this group is a positional preponderance associated with prone or predominantly side stroke swimming. In prone swimmers the interstitial edema is most notable anteriorly on the lateral chest radiograph. Rarely do these display a pleural effusion. These are typically associated with large heart shadows, typical of athletic heart with right and left heart prominence. In the acute setting these will also display mildly widened vascular pedicles, and larger azygos veins. Unique to this group is resolution within twenty four hours with resolution of anterior interstitial edema, fissural thickening, prominence of the vascular pedicle, and some decrease in the right heart size. This is the pattern that has classically been called 'swimming-induced pulmonary edema' (SIPE).

Consolidation

A separate pattern we have noted, although less frequent, is consolidation without a specific distribution. These subjects report a high rate of frank hemoptysis. They also demonstrate large heart shadows, consistent with athletic heart. This pattern resolves within 2-4 days, which is more consistent with pulmonary hemorrhage, differentiating this from infection, which resolves over a period of weeks. Unique to this pattern is absence of interstitial septal lines or effusions. Importantly, these radiographs are almost always obtained within 30 minutes following a swim evolution. It is unlikely that if these subjects had interstitial edema, it would have resolved in the 30 minutes prior to the radiographs. This is a pattern we term 'swimming-associated pulmonary hemorrhage.'

We also note a pattern that is a mixture of both the above findings. In some cases the consolidation may represent alveolar edema with interstitial edema. In others it is possible that both edema and pulmonary hemorrhage coexist.

Etiologies

The etiologies for these patterns have not clearly been delineated. A common feature in these are young extremely fit men with months of endurance and isometric training, often with athletic heart physiology. The nature of the training evolution over many days of continuous exercise results in anasarca with swelling of hands, feet, arms and legs. The swimming evolutions with cold water immersion leads to fluid shifts from the anasarca into the intravascular space, and maximizes lymph return. Cold temperature additionally leads to vasoconstriction. Physiologically then these subjects undergo increased preload and afterload effects in the heart. Importantly, this is occurring during peak exercise and maximum cardiac work. Proposed mechanisms for interstitial edema include: differences in right ventricle and left ventricle peak work capability, a left ventricle diastolic dysfunction at very high heart rates, transient left ventricle dysfunction due to high adrenaline states of continuous exercise over days, or some other unknown mechanism.

The cause for isolated pulmonary hemorrhage is unknown. We have noticed increased cases of "SIPE" of those testing positive for viral lung infections. Is it possible that some insult to the basement membranes within the alveolar capillary interface results in greater disruption at increased pulmonary artery pressures during exercise that results in hemorrhage? Could there be other factors that may weaken the basement membrane?

Summary

What has previously been called SIPE, should probably be categorized more broadly as swimming-associated lung injury with one subset being classic SIPE and another being swimming associated pulmonary hemorrhage. We should recognize that these patterns are in the setting of prolonged extreme exercise reaching the limits of physical capacity. Just as anasarca and cold water immersion are contributing factors, we should consider immune suppression, subclinical infection, and possible medication effects as factors that may be contributing to pulmonary hemorrhage.

Discussion

V Maričić: Thank you. Which type of imaging would you recommend as being all around best for the things we are discussing? Between type 1 and type 2, I have exactly the same experience. Type 2 is going to recover faster and is emotionally less. Basically, it's a leak from the nose. Type 1 is going to be quite disturbed leading to potentially quit diving, going home and a much harder recovery.

G Boswell: If I could do a CT on everybody, I would. All those x-rays I showed you are from an x-ray unit right on the beach. All those x-rays were done within a half hour of them coming out of the water. It is very nice data to have. If the pulse oximeter is below whatever level, 95, they're going in there, getting vitals, x-rays, and then observation. They want to make sure before they go back to training that they can be on a bike for however long and stay above 95%.

B Sebreros: Our functional test is on a stationary bike level 12, 90 RPMs, 3 minutes. They have to maintain oxygen saturation greater than 95%.

V Maričić: If you ever want to try freediving and you have that capacity at the beach, let me know.

F Silva: What are your thoughts on the role of ultrasound as an alternative modality? And then, how you chose to do the timing of your follow-up x-rays? I saw some were 12, 20, 32, and 40 hours. How did you select the timing?

G Boswell: Sometimes it depends on what time they come out of the water. If they come out at 8:00 o'clock at night, it will be the next morning. If they came out at 4:00 in the afternoon, the next one will be 8:00 in the morning.

B Sebreros: I can help with that question. During hell week, they have four hours they can be off training. Sometimes we'll repeat serial ones. Sometimes it's a Friday. We don't come in on the weekends to do repeat chest x-rays so you're going to see them 72 hours later, it just varies. Sometimes at the end of the day, then we do it the next morning on follow-up.

G Boswell: Usually we're not getting new cases of SIPE on a Friday morning because by that time they've finished their training. Wednesday is the busy day. Dr. Sebreros has a project like that.

Comment: He's the one who was supposed to do the cardiac ultrasounds.

G Boswell: As a radiologist, I would love to see that because there's a lot of literature out there. I would love to just let me read the x-ray blind and do the ultrasounds blind and determine how good the ultrasound is. Type 2 you may not see that on ultrasound because you're looking for B lines out of the periphery and thickened pleura. You may not pick up type 2 or maybe you won't pick up some of the mixed cases. You're not going to see a big effusion.

Comment: We're doing ultrasound right now. In many cases they're showing B lines, but the x-rays are negative.

Comment: You'll see more B lines on x-ray.

G Boswell: I just want to read the x-rays. Ideally, I would have a CT scan. But the thing that's hard, you can get a B line on an x-ray within half an hour of each other.

E Yu: Another thought on not being able to see that sort of type 2 with ultrasound and B lines, are reporting hemoptysis on doing a nasopharyngeal laryngoscope on the beach side? Not going beyond the vocal cords, but just looking?

G Boswell: For research purposes, I think that would be difficult because that is an invasive procedure.

R Moon: Could you please clarify. My understanding was that you said that the type 2, which has alveolar densities, is more likely to be hemorrhage and sometimes it takes days for them to resolve. Whereas the type 1 resolves fairly quickly, if I understood you correctly, and yet the type 1 are the ones who drop out?

G Boswell: That's right.

R Moon: So even though it takes a few days for the type 2s to get better?

G Boswell: I don't know what it is, this is purely conjecture. I still wonder why is it when I was in training or early in my career I did not see that much SIPE and now I'm seeing a lot of SIPE? We know we've had a problem with performance enhancing drugs in the community. Is there a relationship between those? Are people taking anabolic steroids to tolerate the pain better? I don't know. Now they're starting to screen for these routinely so going forward I want to see if we have less type 2.

HIGH ALTITUDE PULMONARY EDEMA: CLINICAL ASPECTS AND PATHOPHYSIOLOGY

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High altitude pulmonary edema (HAPE) afflicts unacclimatized lowlanders ascending to elevations above 2,500 m. The majority of cases develop within 1-5 days of ascent to a given elevation and present with a subacute time course, as opposed to the more sudden symptoms that might be seen with myocardial infarction, pulmonary embolism or pneumothorax. Some individuals – referred to as HAPE-susceptible – are at high risk for recurrence with repeated ascent to high altitude while other individuals experience sporadic cases in which the risk of recurrence is felt to be low. The symptoms and signs vary based on illness severity and rate of progression and range from dyspnea on exertion out of proportion to that expected for a particular elevation, dry cough and slight tachycardia and tachypnea in mild cases, to dyspnea at rest, cough productive of pink frothy sputum, cyanosis and altered mental status in more advanced cases marked by severe hypoxemia. If identified at a sufficiently early stage the majority of cases resolve with descent and/or supplemental oxygen with the rate of recovery varying from hours to days based on the severity of illness. Supplemental oxygen and pulmonary vasodilators, such as nifedipine, are often used in situations where descent is delayed or infeasible. [1] An episode of HAPE does not preclude future trips to high altitude, as slower ascent rates and/or pharmacologic prophylaxis with pulmonary vasodilating medications can reduce the risk of recurrence. [1] Cases have also been documented of individuals afflicted with HAPE successfully reascending to high altitude on the same expedition after a sufficient period of time and symptom resolution at lower elevation. [2,3]

Unlike pulmonary edema seen in patients with heart failure at low elevation HAPE does not develop as a result of left ventricular (LV) dysfunction as multiple studies have established that left atrial pressure and other markers of LV function are normal in such cases. [4,5] Instead, the primary factor causing accumulation of fluid in the interstitial and alveolar spaces is an increase in capillary hydrostatic pressure stemming from excessive hypoxic pulmonary vasoconstriction (HPV). This causal mechanism was initially recognized in early studies of HAPE conducted in the 1960s in which affected individuals underwent right heart catheterization during the acute and resolution phases of their illness [4,6] and subsequently validated in later studies that examined physiologic responses and outcomes in HAPE-susceptible and non-HAPE-susceptible individuals who ascended rapidly to 4559 m. Maggiorini et al, [7] for example, performed right heart monitoring following ascent and demonstrated that pulmonary artery (PA) and capillary pressures were higher among HAPE-susceptible individuals who developed HAPE on that expedition than in healthy controls and HAPE-susceptible individuals who did not develop edema. The link between increased PA pressure and edema formation was further established by Swenson et al. [8] who performed bronchoalveolar lavage in both HAPE-susceptible and non-HAPE susceptible individuals following ascent to 4559 m and demonstrated a clear relationship between the magnitude of the increase in PA pressure as estimated by echocardiography and the concentration of red blood cells and proteins in the bronchoalveolar lavage fluid. By performing bronchoalveolar lavage very early in the course of illness and finding no increase in the level of various inflammatory mediators this study also definitely put to rest an earlier notion that inflammatory responses have a causal role in HAPE in its initial stages.

Other bronchoalveolar lavage studies demonstrating the presence of high molecular weight proteins and increased cellularity in the alveolar spaces of patients with HAPE [9] as well as histopathologic analyses of animal models showing ultrastructural damage to the alveolar-capillary barrier [10,11] suggested that increased pulmonary artery and capillary pressure led to edema formation not just through alterations

in Starling forces but also through stress failure of the pulmonary capillaries. [12] More recently, however, Swenson [13] has argued that such overt structural failure may not be present in all cases. Instead, a spectrum of problems may occur in relation to the degree and duration of elevation in pulmonary artery and capillary pressure. Milder cases result from increased flux of fluid into the alveolar and interstitial spaces due to, for example, dynamic pressure-sensitive changes in transcellular vesicular transport or the size of fenestrations that exceeds the capacity for fluid removal through lymphatics or alveolar epithelial sodium channels and sodium potassium ATPases. Overt mechanical injury to the microvasculature is only seen in more severe cases. This hypothesis would fit with the observation that some patients with HAPE improve within just a few hours of descent while more severe cases require a longer duration for recovery.

Given that excessive increases in PA pressure are a key factor in HAPE the question arises as to why increased pulmonary vascular resistance (PVR) due to excessive HPV causes pulmonary edema when other classes of patients with high PVR, in particular patients with pulmonary arterial hypertension, do not experience this problem. In HAPE, the key factor appears to be that HPV is not uniform throughout the lung and, instead, occurs in a heterogeneous manner. As a result, while flow is reduced in lung regions in which vasoconstriction occurs, areas free of such vasoconstriction are exposed to increased flow which subsequently raises capillary pressure in the downstream vascular bed favoring the development of edema. [14] Uneven pulmonary venoconstriction may further contribute to the rise in pulmonary capillary pressure. Support for this notion of uneven vasoconstriction initially came from animal studies [15,16] in which flow was stopped in selected segments of the pulmonary vascular tree and edema formation was noted in perfused areas of the lung. More recently an MRI-based study of lung perfusion [17] found HAPE-susceptible individuals to have more heterogeneity of pulmonary perfusion when breathing a hypoxic gas mixture compared to non-HAPE-susceptible individuals and individuals with no prior exposure to high altitude.

While the role of excessive HPV and overly large increases in PA pressure has been well established as the primary causal factor in the development of HAPE the reasons for these exaggerated responses have not been fully elucidated. Much of the insights in this regard have come through studies that examine people deemed to be HAPE-susceptible based on a careful review of prior experiences at high altitude. Unlike people with sporadic cases who never experience recurrence with later ascents the cohort of HAPE-susceptible individuals are felt to have a 60% likelihood of recurrence with reascent to the same elevation at the same rate as during prior episodes. [18] One of the key features that distinguishes this cohort of individuals from people who remain healthy at high altitude is their exaggerated pulmonary vascular responsiveness, which has been nicely shown in multiple studies demonstrating significantly higher pulmonary artery pressures at rest in hypoxia as well as with exercise in normoxia and hypoxia when compared to the responses in non-susceptible individuals. [19,20] Importantly, although exaggerated HPV is likely necessary to cause HAPE, this alone may not be a sufficient condition as high altitude-naïve individuals found to have exaggerated HPV during low altitude screening develop HAPE at a far lower rate than seen among known HAPE-susceptible individuals ascending at the same rate to the same elevation. [21]

One potential mechanism for exaggerated HPV examined in this cohort of patients is reduced availability of nitric oxide as several studies have demonstrated that HAPE-susceptible individuals have reduced exhaled nitric oxide concentrations during exposure to hypoxia when compared to non-susceptible individuals. Climbers suffering from HAPE have lower nitrate and nitrite concentrations in bronchoalveolar lavage samples compared to healthy controls. [22,23] Further support for this concept comes from studies that have shown that administration of inhaled nitric oxide lowers pulmonary vascular resistance and PA pressure and improves gas exchange in individuals suffering from HAPE. [24,25] Differences in circulating catecholamines have also been proposed as having a causal role but the data remain unclear. Studies have shown that HAPE-susceptible individuals have increased sympathetic

activity in response to hypoxia compared to healthy controls [26] and that alpha-blockade reduces pulmonary vascular resistance and PA pressure at high altitude. [27] Other studies, however, have examined transpulmonary changes in plasma norepinephrine and epinephrine concentrations and suggest that pulmonary hypertension following ascent to high altitude is independent of sympathetic nervous system effects in the pulmonary circulation. [28] Endothelin-1 concentrations are also increased in hypoxia but given that these changes are only observed after 1-2 days at high altitude they are likely not playing a role in the acute pulmonary vascular responses following ascent. [29] Finally, altered alveolar fluid clearance has also been proposed as factor contributing to development of HAPE [30,31] as HAPE-susceptible individuals have been shown to have decreased nasal transepithelial potential differences - a surrogate marker of alveolar sodium transport - compared to non-HAPE susceptible controls. The relative weight of this issue compared to the other factors described above is not clear.

Beyond these studies examining features of known HAPE-susceptible individuals there are other reports in the literature, often involving more idiosyncratic cases with low risk of recurrence, suggestive of additional factors that occasionally contribute to development of HAPE. Luks et al., [32] for example, described the onset of HAPE in an ultracyclist at 2380 m during the Bicycle Race Across America which they attributed to extracellular volume expansion related to due to excessive salt intake during the first four days of the race. Durmowicz et al. [33] examined over 150 cases of HAPE diagnosed in children and adults at 2,800 m in Colorado and argued that preexisting inflammatory processes, such as viral respiratory tract infections, bronchitis and otitis media may have predisposed to HAPE particularly in the pediatric cases by increasing pulmonary capillary permeability in the days preceding high altitude travel. Given the nature of these reports it is difficult to draw firm conclusions regarding the precise role played by such factors.

In summary, HAPE is a non-cardiogenic form of pulmonary edema that develops in unacclimatized lowlanders within 1-5 days of ascent to elevations > 2,500 m and resolves with descent and/or supplemental oxygen over a varying time frame based on the severity of illness. The critical pathophysiologic mechanism is excessive, heterogeneous HPV that exposes certain regions of the pulmonary capillary bed to increased flow and hydrostatic pressure which, in turn, cause extravasation of fluid, red cells and protein from the vascular to the interstitial and alveolar spaces of the lungs. The reasons for such exaggerated HPV remain unclear but based on extensive studies comparing HAPE-susceptible to non-HAPE susceptible individuals may relate to differences in nitric oxide availability, sympathetic nervous system responses to hypoxia and alterations in alveolar sodium and fluid transport.

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Discussion

R Moon: SIPE and to a large extent squeeze in breathhold divers occurs during moderate to heavy exercise. Whereas the case report that you gave at the very beginning of your talk was something that occurred during sleep when cardiac output is probably quite low. Any thoughts on what's going on there?

A Luks: HAPE often involves climbers and may develop while they're actually engaged in physical activity. For her it started to manifest during sleep. But when people are climbing on Denali, they're actually doing a tremendous amount of physical work during the day. They're carrying very heavy loads and going up and down the mountain a lot. That may actually be one of the things that distinguished that expedition for her compared to her prior successful climbs where they're carrying much lighter loads at high elevation than she was doing. Even as her symptoms manifested a little bit during evening she was doing a fair amount of work during the preceding days of that expedition.

S Hopkins: On the slide of Erik Swenson's category of HAPE severity I would suggest that we put in there where you have increased flux and lymphatic drainage a fourth one in between that is interstitial pulmonary edema. As a medical student Andy was a participant in an expedition to the Capanna Regina Margherita in the Italian Alps where they took a bunch of people up to high altitude and measured things like closing volume and found an incidence where 45%-50% of climbers who went up to this altitude developed evidence for interstitial edema. This is probably just from increasing the pressure to get fluid influx, no big deal. Only one of those subjects had any overt HAPE. Some of this interstitial edema parallels what you may be getting in the SIPE immersion edema, which would be those that recover quickly.

A Luks: A good point that Erik would argue is the interstitial edema in that film. That study with George Cremona and Peter Wagner was now 25 years ago. More recently people have been doing lung ultrasound studies at high elevation and showing that if you just bring people up to elevation and you perform lung ultrasound, even though they're asymptomatic, not hypoxic, and not manifesting any other signs of pulmonary edema, the number of B lines is increasing.

S Hopkins: Several years ago we did a study here at UCSD that is languishing in my pile of unpublished work. We volume overloaded people to give them interstitial pulmonary edema and we measured transthoracic fluid content. We found zero relationship between the number of B lines and how much fluid they had. I worry with these imaging techniques that are semi-quantitative where people start assigning a great deal of significance to this many B lines versus that many B lines. We also see it in other imaging techniques. B lines were validated initially as a marker of pulmonary edema in people who were very severely ill in the ICU with severe pulmonary edema and not this subtle kind of stuff.

Comment: Certainly, regarding the studies with ultrasound and B lines at high altitude no one has any idea of what the threshold should be of saying HAPE versus not HAPE.

P Lindholm: Thank you. As a radiologist by training I have looked at numerous accounts of pulmonary embolism. If heterogeneous perfusion can cause edema in high altitude, I've never seen it in pulmonary embolism, which is fascinating in this regard. If you would actually have that effect it must be something more than just heterogenous flow contributing. The B lines are very sensitive. It's also not published, but E Yu and F Silva did a study this summer submitted as an abstract where we showed that a lot of the freedivers had B lines that go away. In an hour most of them have disappeared. It's a very sensitive measure. The balance between what is a normal physiological fluid shift and type 1 interstitial edema, could that be relative ‘heart insufficiencies’ for the exercise Navy SEAL candidates were doing and that is why they failed? There could be something temporary there

but we don't know that. Then there are the ones who pull through but we also don't know the time factor. Could it be that the ones who have bleedings have had that interstitial edema, but that had cleared it somehow? There are a lot of questions. That issue with different phases seems like something we have talked about in our SIPE group as well.

Comment: It happened in ours where we were throwing in cardiac as a causal factor, as well, and you're excluding that.

A Luks: I removed some of this data in the interest of time. There was another paper from Japan where they performed right heart catheterization within a couple of hours of people coming down in the lower elevation group with clear cases of HAPE and normal cardiac index, normal pulmonary artery occlusion pressure, higher pulmonary vascular resistance, and high pulmonary artery pressure. It is a pretty consistent finding in the literature that the left heart is not contributing. The one thing I would say that they do not do, which often does suggest that someone might have diastolic dysfunction, is they're not volume loading these people to see what happens to pulmonary artery occlusion pressure and whether they have an overly large increase in response to volume loading. Otherwise, the studies have generally consistently shown that pulmonary artery occlusion pressures were low.

Comment: Echo studies at altitude have nothing showing tricuspid regurgitation like what Dr. Moon showed with his exercise.

A Luks: No mitral regurgitation. They're dependent on tricuspid regurgitation to estimate pulmonary artery systolic pressure, but they have not shown mitral regurgitation.

F Silva: Before There was a study in early 2000s describing B lines. I entirely agree that ultrasound for quantification concepts cannot be used because it's so sensitive, especially in freediving where so many other things can cause B lines. But I do think it's extremely useful. We can use it for some future research such as time for clearance. This is going to make a huge difference for time for clearance and to guide evolution it may be very useful.

S Hopkins: I completely agree.

F Silva: The thing that is kind of untapped territory there is the distribution. This is entirely informal. In squeeze it's those brighter, well-defined, and patchy not effused lines we see in specific areas that would make sense with a lung squeeze. Regarding the quantification everybody is going to have B lines. But we can still use ultrasound.

PATENT FORAMEN OVALE: A RISK FACTOR FOR BREATHHOLD DIVING PATHOLOGY

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Patent Foramen Ovale (PFO) is a widely prevalent intracardiac shunt in humans (approx. 25-30%), which is recognized as a risk factor for decompression sickness (DCS) after compressed-gas diving. The proposed mechanism is paradoxical embolization of decompression-induced inert gas bubbles, which may seed into the cerebral and general arterial circulation to cause symptoms. It is generally accepted that the risk for a diver with PFO to incur DCS is between 2 and 3 times greater than if no PFO is present. The risk of a PFO in breathhold (BH) diving has not been formally explored and described. Based on a review of the physiology of human circulation and diving/immersion, several possible issues can be described by which a breathhold diver with a PFO may have a higher risk for diving-induced pathology. Paradoxical embolization of decompression-induced bubbles may be a causal factor in decompression syndromes described in BH divers. However, it seems that except for the case of extreme depth BH diving or extreme-repetitive BH diving, the likelihood of decompression-induced inert gas bubbles is low. Hemodynamic changes, induced by immersion or, more likely, the technique of glossopharyngeal breathing (lung-packing) may however play a significant role in increasing the right-to-left shunting through a PFO. Although there appears to be no significant change in cardiac output or stroke volume in "normal" BH diving, it has been shown that lung-packing significantly reduces both parameters at the surface. The subsequent 'release' of pressure when descending (Boyle's Law) would most probably induce significant shunting of venous blood - possibly carrying VGE after repeated deep BH diving - into the arterial circulation. A PFO could possibly become more 'shunt-prone' during descent owing to spatial configuration changes caused by the decreasing volume of the lungs and the thoracic blood shift. This may contribute to the observation that some BH divers do not experience an increase of arterial pO_2 when descending contrary to what is expected based on the Laws of Physics. Future research on BH diving should, where appropriate, include a systematic detection of PFO in order to clarify the influence of this potentially important factor in the genesis of BH diving-induced pathology.

Introduction

Patient foramen ovale (PFO) is a developmental variant in mammals, resulting from the incomplete transition from placenta-based oxygenation of blood towards pulmonary-based oxygenation after birth. During the embryonic development of the (human) heart, a permanent shunting of blood between the right and the left atrium allows over 95% of the oxygenated blood coming from the inferior caval vein to enter the left-sided circulation without having to pass through the (non-oxygenating) pulmonary circulation. This interatrial connection is formed by the overlap of two septal fold originating respectively from the 'basal' end of the atria (the 'septum primum') and the 'upper' end (the 'septum secundum'), overlapping each other so that a slit-like opening remains from 'right-down' to 'left-up', which coincides with the blood flow from the inferior vena cava that is fully directed towards the 'fossa ovalis'. In those cases where both septa do not overlap, a permanent and bidirectional opening between both atria exists, which allows for a predominant left-to right shunt: Atrial Septal Defect (ASD).

After birth the pulmonary circulation sees a dramatic decrease in resistance (due to the expansion of the pulmonary alveoli) and all of the venous blood is 'aspirated' into the right ventricle and further into the pulmonary artery. The pressure in the right atrium drops below the pressure in the left atrium. This

causes the valve-like opening of the foramen ovale to close shut and within a few days or weeks the developing cardiac tissue ensures a permanent closure.

In a large number of persons, however, this fusion is not complete and a tunnel-like passage persists, which may at times when the blood pressure in the right atrium would exceed that of the left atrium allow passage of some venous blood toward the left. This condition is called 'PFO', Persistent or Patent Foramen Ovale. Anatomy studies have determined the exact prevalence of PFO [1]: it appears that on average, 25% of humans have this condition; younger people (<40 years old) have a higher prevalence – up to 40%) than older people (20%). However, the anatomical diameter in older people tends to be greater. This has led to the hypothesis that PFO's may continue to close over the first decades of life, unless, for reasons yet unknown, they become larger with age. This hypothesis has been verified in a repeat longitudinal study where up to one third of small ('grade 1') PFO's was found to have become larger ('grade 2') after an 8 year interval. [2]

A PFO is generally considered uneventful, remains fully asymptomatic, and most subjects with PFO may never know they have one. This is in contrast with ASD, which is both much less frequent (less than 1 per 1000) but much more symptomatic (causing volume loading of the right atrium which may lead to right atrial hypertrophy, exertional dyspnea, arrhythmias and cardiac insufficiency).

However, some diseases and pathological conditions are associated with PFO, related to the shunting of venous blood to the left atrium in conditions of increased venous return and/or increased pulmonary artery pressure (see Table 1). Some of those conditions may be present in divers, both freedivers and scuba divers. In this context 'scuba' refers to those divers having access to a quantity of breathing gas during the dive, either carried with them - true 'scuba', or supplied via a conduit - 'tethered' diving, which allows them to stay under water much longer than freedivers.

Table 1. Pathological conditions associated with PFO

Paradoxical embolization
- Stroke
- Decompression Sickness
Migraine (with aura)
Platypnea-orthodeoxia syndrome
Mountain Sickness -High Altitude Pulmonary Edema (HAPE)
Exertional arterial blood desaturation

In scuba divers there exists a risk for formation of inert gas bubbles in the venous blood during and after ascent to the surface. These gas bubbles could pass through a PFO (a so-called 'paradoxical embolism') and enter the arterial blood stream where they may cause blockage of small blood vessels and symptoms of 'decompression sickness'. This risk has been described in 1989 [3,4] and has since then been extensively studied. It is now accepted that having a PFO exposes a scuba diver to a statistical risk of DCS that is between 2 and 3 times higher than a diver who does not have a PFO [5-7]. The absolute risk and whether it is acceptable remains of course dependent on the type and intensity of the diving exposure. For recreational diving, the risk of DCS 'all divers included' varies considerably (see Table 2).

In order to reduce the risk of DCS, some advocate PFO closure by means of catheter-mounted 'umbrella' devices. Whereas these are in most cases effective in closing the intracardiac shunt - and may thus permit deeper and more saturated dives with a similar risk to 'non-PFO' divers [8,9] - the procedure carries in itself a certain risk. Therefore, for recreational divers it appears more logical to simply reduce the diving (inert gas) exposure. [10,11] It also seems that a number of cases of DCS, even after a PFO

closure, can be attributed to (non-permanent) intrapulmonary arterio-venous anastomoses (IPAVA) that could open in the case of extensive gas bubble embolization in the pulmonary artery. [12]

Table 2. Estimated and measured risk for decompression sickness of SCUBA diving (from: [13])

Population	Risk	Source	Accuracy
Military Divers (US)	1/76,000 dives	Arness 1997 [14]	Estimation
Recreational Divers (Australia)	1/15,000 dives	Gorman 1995 [15]	Estimation
Recreational Divers (USA)	1/2,900 dives	Bove [6]	Measured (fact)
Commercial Divers (USA)	1/280 dives	Bove [6]	Measured (fact)
Military Divers (USA)	1/3,770 dives	Bove [6]	Measured (fact)
Recreational Divers <-30m (Europe)	1/40,228 dives	DAN Europe [16]	Estimation
Recreational Divers (any depth) (Europe)	1/6,604 dives	DAN Europe [16]	Estimation
Sports Divers (UK)	1/10,500 dives	BSAC [17]	Estimation
Sports Divers (Cold Water Wrecks)	1/270 dives	DAN USA [18]	Measured (fact)
Dive Instructors (Liveaboard)	1/1,000 dives	DAN USA [18]	Measured (fact)
Sports Divers (Cold Water)	1/1,250 dives	Trevett et al. [19]	Measured (fact)

Methods

The risk of a PFO in freediving has not been formally explored and described. Based on a review of the physiology of human circulation and diving/immersion, several possible situations can be described by which a breathhold diver with a PFO may have a higher risk for diving-induced pathology.

Results

PFO and decompression sickness in freedivers

For decompression-induced gas bubbles to appear in the venous blood ('VGE': Venous/Vascular Gas Emboli), a certain level of inert gas saturation of the tissues is needed. While this amount is not quantifiable with certainty all currently employed decompression models and their ensuing algorithms define a 'no-decompression limit' (NDL) indicating a diving exposure from which a direct ascent to the surface is possible without a significant risk of DCS. Even if in such exposures some bubbles may be observed, which are eliminated by the execution of a so-called 'safety stop', keeping the dive exposure below this NDL is accepted as having a negligible risk for DCS.

Even if a single dive exposure may generate sufficient VGE to allow paradoxical embolization, often in conjunction with physical maneuvers temporarily increasing the right atrial pressure, in general PFO-mediated DCS is observed in those divers having done multiple days of diving close to or beyond the NDL. It seems inevitable that the dive computer algorithms cannot account for the individual variation in desaturation during the surface intervals and thus become less and less reliable in predicting a safe 'next dive' exposure. Thus, DCS attributable to PFO is often associated with repetitive or multiple-day diving, often after three or four days. Conditions that increase the release of VGE into the blood stream may be intense physical exercise, a hot shower or whole-body vibrations, e.g., a fast boat ride over rough seas, shortly after surfacing. Conditions that may increase shunting of venous blood through the PFO may be isometric sustained effort (a so-called 'straining maneuver') such as inflating a BCD by mouth, climbing a dive boat ladder with heavy gear on, or other physical efforts. Finally, a continuing stream of VGE transiting into the pulmonary circulation may cause a progressive increase in the pulmonary artery pressure and retrograde the right atrium pressure, which after 20-30 minutes may become for significant periods of time during the cardiac cycle higher than the left atrial pressure with subsequent shunting of VGE-laden blood into the left atrium. [20]

The resulting DCS symptoms are characterized by vestibular, cochlear, high-spinal, cerebral or cutaneous symptoms, the latter possibly caused by embolization of VGE into the area of thermoregulatory

neurons in the brainstem, an area devoid of collateral circulation and thus more vulnerable than the rest of the cerebral tissue to even a few embolizing VGE. [21]

There may be circumstances where VGE become arterialized through permanent or intermittent pulmonary shunts or even directly arise in the arterial circulation [22] but the predominant cause of arterial bubbles after diving seems to be from an intracardiac right-to-left shunt.

The diving exposure (time and depth) in freediving seems to be much less than the habitual NDLs in scuba diving. However, several cerebral and/or mental syndromes have been described after freediving, possibly related to decompression sickness.

'Taravana syndrome' is a complex of neurological and psychomental symptoms observed originally in Polynesian and Japanese harvest divers (Ama) [23] but has also been evoked in recreational 'extreme exposure' freediving activities such as spearfishing or other freediving activities with the help of underwater scooters (DPV: diver propulsion vehicles). [24] Cerebral decompression sickness symptoms have also been reported in 'no-limits' freediving exposures where mechanical aids allow the diver to reach extreme depths in a very short time with accordingly rapid decompressions. It is commonly assumed that freediving beyond a depth of 100 meters carries a higher risk of DCS. Even though decompression-induced VGE have been very rarely observed in freedivers, theoretically, the decompression-induced bubbles may arise from such short but very deep and repeated exposures. [25] Other pathophysiological mechanisms for such cerebral symptoms have however been evoked, such as arterial gas embolism from lung damage caused by glossopharyngeal breathing (lung packing), hypoxic brain infarcts, hemodynamic factors or microparticle-induced inflammatory damage. [26,27]

When considering PFO-related shunting after repeated freediving, another factor must be considered: The practice of 'lung packing' (glossopharyngeal insufflation, GI), which is very common before deep excursions, increases the pulmonary air content by actively 'pushing' air into the lungs. This may increase the available lung volume by up to 2 liters (normobaric equivalent). However, this is accompanied by a significant increase in transpulmonary pressure, effectively reducing the venous return and in some cases reducing the cardiac output to close to zero. [28] During the subsequent immersion, this transpulmonary pressure decreases very rapidly according to Boyle's Law and this is strikingly similar to the release of the pulmonary 'straining maneuver' utilized in echocardiography PFO detection to increase shunting by briskly increasing right atrial filling after a period of increased pulmonary pressure. Such a rapid increase of right atrial filling is not observed when freediving is performed without prior glossopharyngeal insufflation. [29] It is thus conceivable that performing GI after a prior series of deep freediving excursions might facilitate PFO-mediated shunting of any VGE present.

PFO and arterial desaturation in freedivers

A PFO normally has no significant influence on exercise capacity, in contrast with more constant intracardiac shunts (ASD, VSD) where syndromes may be present at rest (but that are not compatible with a freediving activity, so are not further considered here).

However, release of a sustained Valsalva maneuver may provoke a transient arterial blood desaturation by mixing of some venous blood through the PFO, and this has even been proposed as a diagnostic tool for PFO detection. [30] Whereas these desaturations are on the order of seconds a recent paper described significant arterial desaturations in up to 30% of PFO positive subjects after intense physical exercise. [31] This may be due to the increased pulmonary artery pressure (PAP) commonly observed during high-intensity exercise where the PFO may serve as a pressure 'safety valve' much like the opening of IPAVA during exercise. [32]

Other possible mechanisms causing increased right-to-left shunting may include left atrial hypovolemia once the lung volume drops below residual volume upon deep breathhold immersion[28], hypoxic pulmonary vasoconstriction [33], but also positional changes in the heart favoring vena cava inferior flow through a PFO, even with non-significantly changed left and right atrial pressures.

Platypnea-Orthodeoxia Syndrome (POS) has been first described in 1949 as a condition in patients with extensive pulmonary shunting, and in 1984 a similar syndrome was described in patients with intracardiac shunts. A little more than 200 cases have been described to date, of which 80% involved a PFO as the shunting pathway. [34] The defining symptom is dyspnea and hypoxemia with a hemoglobin desaturation of more than 5% from baseline in the upright sitting position, being instantly relieved when the patient assumed a recumbent/lying down position.

In cases with PFO, the mechanism by which an increased shunting occurs in the upright versus the supine position includes the presence of a prominent Eustachian valve, an ascending aortic dilatation or aneurysm, ascending aorta elongation or horizontalization, thoracic spine kyphosis, and hemi-diaphragm paralysis or elevation such as after pneumonectomy. All of these may change spatial configuration of the right atrium in the upright position changing the flow pattern in the right atrium by opening up the foramen ovale and directing the inferior cava vein blood flow straight to and through the fossa ovale, causing the blood to 'flow uphill'. [35]

Strikingly enough, when arterial blood gases have been measured in deep immersions of freedivers, most of the divers had, as is to be expected by Dalton's Law of physics, an increase in their arterial oxygen pressure when arriving at depth, except for 30% of them. In Bosco et al. (2018) [36], 4 of 6 subjects had an increased PaO_2 at 40 mfw (from 94 SD 6 mmHg to 263 SD 32 mmHg), but 2 of 6 failed to increase PaO_2 at depth. In Paganini et al. (2023) [37], 5 of 14 subjects likewise did not exhibit the expected increase in PaO_2 at 42 mfw of depth. These 'anomalies' were hypothesized to be due to a possible ventilation-perfusion mismatch and atelectasis caused by thoracic blood shift and alveolar collapse due to immersion, but could be just as well caused by a transient increase of right-to-left shunt by a spatial distortion of the cardiac cavities when the pulmonary volumes are compressed to an extreme level causing a reversible 'pressure-induced POS'. Combined with thoracic blood pooling this cardiac shape-change could cause arterial blood desaturation to a degree as to level out the expected PaO_2 increase. The presence of a PFO could thus be (partially) responsible for the maximum attainable depth in elite freedivers, by restricting the further uptake of oxygen from what is left of the alveolar space.

However, in order to confirm this obviously subjects for such future studies should be examined for PFO. When cardiac flow measurements with echocardiography would become possible at these depths this could obviously clarify the issue further.

PFO and the risk of Immersion Pulmonary Edema and pulmonary barotrauma in freedivers

There appears to be no reasonable physiological mechanism by which a PFO would increase the risk for pulmonary 'squeeze' or immersion-induced pulmonary edema following freediving or even surface immersion (swimming). If any, a PFO might even exert a protective effect by relieving excess PAP through the intracardiac shunt. An ASD might over time cause cardiac insufficiency and possibly pulmonary hypertension but the presence of this would exclude a freediving activity in the first place. However, there are no formal data on this topic.

Conclusions

Because of the possible implications of a PFO in breathhold diving-induced pathology, future research on BH diving should, where appropriate, include a systematic detection of PFO. For this, a transthoracic contrast echocardiography would suffice, which offers a relatively non-invasive detection method. [38]

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Discussion

J Valdivia: When you test for PFO flowing to the atria, you release this training maneuver, you see more shunting, correct? Would that also increase the pressure inside the pulmonary vascular system?

P Germonpré: Yes. I see where you're getting at, it is sort of a safety valve. If the pulmonary vascular pressure is high, the PFO will open. We see that if you have lots of bubbles or a pulmonary embolism then basically if the pressure in the pulmonary artery increases the PFO will open.

J Valdivia: The shunting will be only through the atria?

P Germonpré: Yes.

J Valdivia: I have heard from freedivers that if they have some symptoms of pulmonary edema when they come up from depth they would pack and go to 6 or 7 m for 10 seconds. When they come up the symptoms will be better right away. They believe it works.

P Germonpré: It's interesting, but I can't say anything about it. Those would be freedivers I would like to have on my ultrasound table though.

R Moon: Peter, that was a phenomenal outline of PFO. I know by personal communication with Bosco that he's done some PFO studies and there's no consistent relationship with hypoxia. The most likely cause of those, the relative hypoxic measurements at depth, was probably regional lung collapse and right to left shunt through the lung.

P Germonpré: The thing with PFO testing is that I made it look simple enough. Around 50% of the divers that come to see us with the typical history of PFO-related decompression sickness have been to a cardiologist who says I don't have a PFO. This is because of the straining maneuver. Normally if you inject your contrast medium in the upper arm and it comes from the superior caval vein and all the blood coming from the lower caval vein is actually pushing that contrast away from the septum. There's a very high proportion of folks with negative results in PFO testing. This is why, as long as I can, I have them come to me rather than rely on a cardiologist. Unless I know the cardiologist and know how he's doing it right or unless I can see the video. Cardiologists don't like to take videos of more than one heartbeat. They like these very short videos. They need to take 10 or 15 seconds of video. Yes, it's one of both, but measuring these pressures, measuring the flows at depth is difficult.

HEMOPTYSIS IN LUNG SQUEEZE - WHERE DOES IT COME FROM, WHY, AND WHEN?

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Introduction

Freediving is a human activity where a person dives to extreme depths without any breathing aids. As freedivers venture into the depths they engage in a profound interaction between physiology and environment and expose themselves to very high pressures and extended hypoxic conditions, which were once thought a human cannot survive. Today we know it is possible to go even beyond 100 meters of depth but these and even shallower dives may come with a cost as sometimes these athletes experience lung barotrauma on descent, which is more popularly referred to as lung squeeze. These athletes suffer from different symptoms such as chest pain, cough and shortness of breath. One relatively uncommon or maybe underreported symptom is hemoptysis. Hemoptysis is the expectoration of blood from the respiratory tract, not a condition in itself, but rather a symptom of an underlying medical issue or condition. Hemoptysis can vary in severity ranging from the coughing up of small streaks or flecks of blood to the expectoration of larger quantities of blood and can vary from minor and self-limiting to life-threatening. Hemoptysis can be caused by bleeding from anywhere within the respiratory system. Current literature is incomplete in evidence and hypothesis about hemoptysis in freedivers, especially as being a symptom of lung squeeze. The etiology and pathophysiology of remains unclear. There has been very little or no investigation into compression, collapse and re-expansion of human lungs during one dive on a single breath.

Methods

This discussion of the enigma of hemoptysis in freediving reviews current literature, experts' opinions, author's experience and common sense using recent and ongoing research.

Discussion

Reports have surfaced about lung barotrauma occurring in freedivers, where athletes exhibit hemoptysis. [1] The current explanation for this phenomenon involves a mixture of cardiovascular changes during immersion, increased hydrostatic pressure, alveolar closure and reopening and breathholding. Boyle Mariotte's law states that, during breathhold diving ambient pressure rises while the volume of air in the lungs decreases. When the lung capacity is reduced to residual volume further ambient pressure increases lead to negative pressure in the thorax causing blood to shift. Immersion and apnea trigger an autonomic response, inducing peripheral vasoconstriction and bradycardia. [2] The "buoyancy effect" in water also shifts blood to the thorax due to the loss of gravity's impact. Immersion, hydrostatic pressure, and apnea cause substantial thoracic blood shift and significant pulmonary vascular engorgement. If pulmonary capillary pressure surpasses oncotic pressure, transudation from capillaries may lead to pulmonary edema. Massive blood shift can increase transmural pulmonary capillary pressure potentially causing endothelial damage and stress-induced failure. [3] Other possible causes of squeeze can also be the physical impact of collapsing parts of lungs and "squeezed" lung interstitium and vasculature. Trauma can also be caused by alveoli popping open on ascent.

Hemoptysis in freediving is underreported and large epidemiological study needs to be done in order to quantify the problem. From my experience hemoptysis has individual variation and is very interesting

why some individuals almost never experience it even when diving to extreme depths. We can also state that tolerance to it can be trained and there are also risk factors involved that can increase the probability of hemoptysis such as cold temperature, poor visibility, underwater currents, psychological stress as well as physical exertion, body position and certain body movements.

Hemoptysis case reports involve bronchoscopy with bronchoalveolar lavage several days after hospital admission. Lindholm et al. confirmed hemoptysis in breathhold divers using laryngoscopy, locating bleeding below the vocal cords. [4] In our study [5] bronchoscopy directly visualized bleeding in both study participants with blood traces observed from all three segments of the right upper lobe bronchus following deep freedives. We attempted to explain why this specific site was the source of bleeding.

Unfortunately, there is scarce research on how human lungs compress, collapse, and re-expand under high pressure. Fitz-Clarke created a computational model on airway and alveolar collapse during deep dives. [6,7] However, we argue that lung mechanics under pressure cannot be solely determined by Boyle-Mariotte's law as lungs and airways differ in structure, compliance, perfusion, and surfactant across anatomical regions.

Muradyan et al. [7] found a lack of ventilation in apical lung regions during small-volume inhalation after below-residual volume exhalation suggesting that apical parts may collapse first. During breathhold diving airway pressure remains constant throughout the lungs but pleural pressure depends on body position. Diving head-first results in higher pleural pressure in apical lung parts causing faster shrinking of apical alveoli. When the diver ascends apical alveoli are under the lowest pleural pressure and tend to reopen first. Alveoli reopening does not happen uniformly or simultaneously. [4] Consideration must be given to the fact that mechanics during deep dives vary across different lung and airway regions. Some parts may be more susceptible to volume reduction, perfusion changes, and collapse, depending on body position, diaphragmatic contraction, and autonomous airway muscle stimulation.

Although we visualized bleeding proving histopathologically whether the source is alveoli or blood vessels is challenging underwater. Damage visible on land affects both and bronchoscopy cannot be conducted underwater. As seen in our usual study participants hemoptysis can even be provoked on land by "reverse packing" the lungs. The elite freedivers which we usually follow practice during their usual training process exhaling air after reaching residual volume thus mimicking freediving descent and causing "lung squeeze" on land which sometimes even causes hemoptysis. However, provoking hemoptysis and then doing bronchoscopy on these athletes and even taking biopsies may cause ethical concerns.

Conclusion

All conclusions that we can make can be based solely on assumptions, experience of authors and common sense because there is very little data supporting it. Hemoptysis in freediving quite possibly comes from significant pulmonary vascular engorgement, collapsing and reopening of parts of lungs or a combination of all. Hemoptysis is underreported, shows individual variability and tolerance to it can be trained. In this field it is difficult to perform research on animal models and even more difficult if not impossible on humans at depth because of ethical problems and risk for freediver safety. This type of research can be done only on a small number of elite athletes and why the precise mechanism of hemoptysis in freediving remains to be elucidated. Hemoptysis in the context of lung squeeze merits exploration due to its potential implications for freedivers' health and safety.

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Discussion

S Hopkins: Regarding stress failure, the guy who was drinking in the bar 3 hours after hemoptysis John West showed, that in experimentally-induced stress failure, that the breaks close very quickly. If they had sampled the lung 10 minutes after they reduced the pressure, they wouldn't see breaks, which supports what you said. You mentioned there may be a training effect on the blood gas barrier. Again, this is what John has cited. In mitral valve disease patients who have chronically high pulmonary capillary pressures have a thicker blood-gas barrier than other people. That also supports that there could potentially be a training effect. In terms of athletes that get exercise-induced pulmonary hemorrhage, anecdotally there seems to be a tendency to happen more often in the early season. Finally, squeeze implies a mechanism, as you said. Why not call it dive-induced pulmonary syndrome (DIPS) or something like that to divorce the idea of the mechanism from what right now is a constellation of symptoms. Athletes can call it whatever they want. But as a scientific organization and a collection of doctors and people that are interested in moving the field forward, I think that it might be helpful. Terminology becomes important and it would be consistent with SIPE.

V Maričić: We had a discussion in Florida, and we came up with pulmonary barotrauma of descent.

S Hopkins: But do you know it's barotrauma, which implies a mechanism? There could be several mechanisms within this broad constellation.

I Barković: It should be syndrome. In different divers one may have a symptom of hemoptysis, one physiology or one pathophysiology versus another. We have pulmonary patients with palliative care, with cancer who sometimes present with pleural effusion, which constricts the lungs so they completely collapse. You can see it through ultrasound, no air. When you draw some 2 liters out the lungs expand and they go home without any problems. That speaks to the fact that alveoli cannot collapse and then reopen again. It is totally different if that happens as in ventilator-induced lung injury or what we saw in patient-induced lung injury where you have closing and opening like this and breaking something inside. I would agree with a syndrome but would not say it's something with going deep because I had my squeeze even in shallow 10 meter water. While spearfishing I stayed for a long time, had a lot of contractions and squeezed.

M Nochetto: Diving is very vague. It doesn't say anything about the mechanism.

S Hopkins: But that's kind of the point. I know nothing about a squeeze except what I've learned this session, but it seems to me that what is referred to as squeeze can also include symptoms like nausea, vomiting and dizziness. It's not necessarily pulmonary. It could be stress failure, edema, or barotrauma. You can describe a syndrome without attributing it to a particular mechanism. As soon as you say it's barotrauma or a squeeze or this or that you send yourself down the path of that's what causes it. The real issue is we don't know what causes these things, and there could be multiple causes.

M Nochetto: I was thinking more like vacuum lung syndrome.

P Lindholm: It's not a vacuum. We will have this discussion later so everybody can contemplate this suggestion as an overall discussion.

K Krack: I find it interesting that the two people had it localized in the upper area of the lungs. In my experience with students, predominantly what I've seen or experienced is divers will be able to tell you whereabouts at some point being on the surface where they felt it more centralized. They'll be able to tell you at around a certain depth and specifically some things that they believe led to it, like head position or strong contractions. What would you generalize in what you've seen in students?

I Barković: Maybe you have some experience where you had someone who could tell left from right. In my experience I haven't seen anyone who would be able to determine this so specifically. It would be more in general or felt it more in the neck or more in the chest. There is a good chance that they would try to understand what happened during the dive, but it's not necessarily going to be very accurate because they might be fantasizing about it and trying to understand it. Head position is important, just like too much CO_2 produced because of poor technique in general, and then if contractions are too strong. In general, in freediving we have this ability to talk to the students. I feel like it's all about the awareness. To become a freediver you need to become aware of your body, your breathing, your lungs. We study our breathing cycles, know how to control them, breathe slower, do breathholds, become aware of the tensions in the breathing muscles. Advanced freedivers have more awareness. We have this tool so we can educate freedivers and ask them to analyze their sensations after the dive. They could become better at this if we give them specific instructions and specific descriptions of how it feels. If they have a list of potential outcomes that are described well through the education process at the different levels, we will have freedivers who can give us feedback. They're actually trying to understand what happened because they're really interested in solving this. We can have a lot of data from this education process.

B Smith: Regarding localization it would be great if one of you diving teams could get an electro-infused monitor machine. It's a belt of electrodes that passes electrical current through the chest and you could localize regions of collapse and it's something you could do on the boat. They're commercially available.

A Luks: Can they go in the water?

P Lindholm: No, the ones that I've seen do not go in the water easily.

B Smith: The ones I've seen are for critical care. You put the electrodes on someone and ventilate them. You can watch lung aeration.

DIAGNOSIS AND TREATMENT OF SIPE IN RECREATIONAL SWIMMERS: THE USE OF LUNG ULTRASOUND AND PREHOSPITAL CPAP

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Introduction

Individuals performing water sports occasionally present with acute respiratory distress due to pulmonary edema. In divers the condition is reported as "immersion pulmonary edema" (IPE) or "scuba diving-induced pulmonary edema" (SDPE), while "swimming-induced pulmonary edema" (SIPE) is used for surface swimmers. [1,2] Unfortunately, terminology is inconsistent. SIPE/IPE is characterized by acute onset of respiratory symptoms during or immediately after swimming or diving, predominantly in cold open water. Patients mostly present with cough and dyspnea, often combined with pink froth or bloody sputum. Chest tightness or chest pain have also been described. [3] Clinical findings are various degrees of hypoxia together with crackles or wheezing on pulmonary auscultation. [3] SIPE/IPE can be visualized on X-ray, computed tomography, or lung ultrasound (LUS). [4-9]

The incidence of SIPE and IPE varies widely. [5,10-14] Larger cohort studies have reported an incidence of SIPE of 0.44% in recreational swimmers and of 5% in Navy SEAL candidates. [14,15] The incidence of IPE was 1.1% in a survey of 1250 divers. [4] Large variations in incidence may depend on different populations and lack of consensus on diagnostic criteria for SIPE. Diagnostic criteria vary from a merely descriptive definition based on symptoms to well defined clinical parameters and radiological verification of pulmonary edema. [3,10,16] In 2020, Hårdstedt et al. [17] proposed a clinical algorithm based on findings of pulmonary edema on LUS.

Diagnostics of SIPE /IPE with lung ultrasound

LUS for diagnostics of pulmonary edema has been used for swimmers, scuba divers and breathhold divers. [7-9] Point-of-care LUS could be proposed as the first line diagnostic technique for SIPE/IPE. Handheld ultrasound devices are now easily accessible, which enables assessment of patients in prehospital settings. Furthermore, LUS has proven higher sensitivity and equal specificity compared to conventional radiography in diagnostics of pulmonary edema. [18] LUS findings in SIPE/IPE, or other forms of pulmonary edema, are characterized by bilateral disseminated B-lines. B-lines are defined as "discrete laser-like vertical hyperechoic reverberation artefacts that arise from the pleural line, extend to the bottom of the screen without fading, and move synchronously with lung sliding". [19] The presence of three or more B-lines in one lung region is considered pathological, and two or more pathologic regions bilaterally suggest a positive exam (Fig. 1). [19] The number of B-lines correlates with the amount of extravascular lung water. [20] Consequently, the course of B-line-count has been able to monitor regress of lung water in patients treated for cardiogenic pulmonary edema, during dialysis, as well as after treatment for SIPE. [21-23] However, semiquantitative methods to evaluate B-lines rather than the precise count of B-lines are recommended, due to a weak correlation between various transducers and ultrasound machines. [24]

Some limitations of LUS are important to keep in mind: B-lines are non-specific and may be associated with other conditions characterized by less aerated lung tissue such as pneumonia, pulmonary fibrosis, pulmonary hemorrhage or atelectasis. [19,25] Medical history and other clinical findings of the patient with dyspnea after swimming or diving are important for differential diagnosis of SIPE/IPE. Furthermore, LUS only penetrates superficial or non-aerated lung tissue. Conventional radiology is

recommended to detect pathology that is located deep in the lungs or covered by aerated tissue. [19] In addition, whether considered as limitation or strength, LUS has proven to be highly sensitive in detecting increased extravascular lung water. An increased amount of B-lines reflecting early stages of pulmonary edema without symptoms or changes in oxygen saturation has been shown in animals as well as in asymptomatic divers and triathlon athletes. [8,26,27] Patients with asymptomatic pulmonary edema after swimming were also found in our Swedish cohort (unpublished data). Similarly, remaining but clinically insignificant pulmonary edema on LUS has been reported in patients with improved oxygenation and symptoms after treatment for SIPE. [23] A proposed LUS-protocol for patients with suspected SIPE/IPE includes scanning of eight regions, four bilaterally (Fig. 2). [19] Hårdstedt et al. defined pulmonary edema in patients with respiratory distress after swimming as bilateral or unilateral presence of two or more positive regions (≥3 B-lines). [17] Unilateral pulmonary edema, mostly on the right lung, can be found in patients with SIPE as well as in patients with cardiogenic pulmonary edema. [14,17,28,29] As previously stated, other causes of unilateral B-lines must be ruled out.

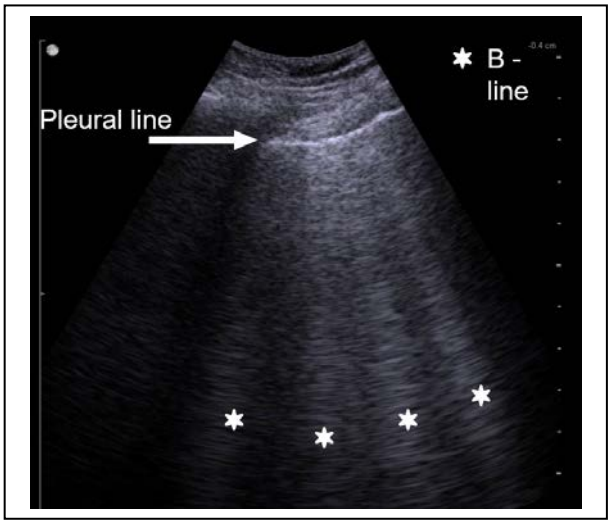

Figure 1. Pathologic lung ultrasound with three or more B-lines in one region.

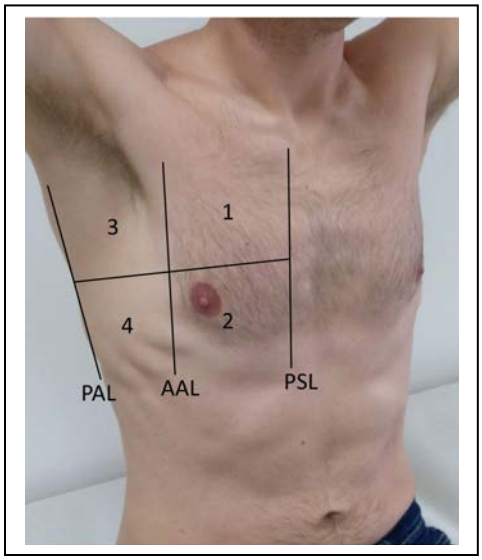

Figure 2. Lung ultrasound protocol including eight regions, four bilaterally, for evaluation of the interstitial syndrome. PAL=posterior axillary line, AAL=anterior axillary line, PSL=parasternal line.

Reflections on lung ultrasound in breathhold divers

Breathhold diving differs from surface swimming or scuba diving by rapid changes in intrapulmonary pressures related to the surrounding hydrostatic pressure. These pressure shifts may result in barotrauma of the lungs. [30] Speculated mechanisms of negative barotrauma (lung squeeze) during descent are increased pulmonary capillary pressures, atelectasis and high negative airway pressures that cause pulmonary edema, capillary rupture or airway mucosal bleeding. Positive barotrauma with overdistension of the lungs during ascent or after lung packing includes alveolar damage, pneumomediastinum, subcutaneous emphysema as well as serious conditions such as pneumothorax or arterial gas embolism. [30] Some of symptoms and findings of negative and positive barotrauma as well as SIPE/IPE may be similar. For instance, cough, dyspnea, hemoptysis or chest pain can be found in all these conditions. [3,30] Also, LUS findings in barotrauma and SIPE/IPE may overlap because pulmonary edema, pulmonary hemorrhage, and atelectasis can all present with B-lines. [19,31]

There are features of barotrauma characterized with findings other than B-lines on LUS. For diagnosis of pneumothorax, LUS is superior to chest radiography. [19] Sonographic signs of pneumothorax on LUS are absence of lung sliding, absence of lung pulse and B-lines, as well as possible presence of a lung point. [19] In patients with subcutaneous emphysema, air impairs ultrasound imaging of structures underneath the subcutis. Subcutaneous gas may produce so called E-lines on ultrasound that should not be confused with B-lines. E-lines do not arise from the pleural line and do not move synchronously with breathing. [32] Unfortunately, pneumomediastinum cannot be detected by LUS due to its central location in the chest. Altogether, pulmonary edema and lung injury due to barotrauma can present with overlapping symptoms and findings on LUS and might be hard to distinguish in breathhold divers with respiratory distress.

CPAP for treatment of SIPE

Treatment for SIPE/IPE described in literature consists of supportive oxygen treatment to reverse acute hypoxia, often combined with diuretics, sometimes also with beta-2-agonist inhalation or non-invasive positive pressure ventilation (NPPV), including continuous positive airway pressure (CPAP) or non-invasive ventilation (NIV). [3] Occasionally, use of other drugs such as corticosteroids, antibiotics or nitric oxide has been reported. [3] The desired effects of diuretics in cardiogenic pulmonary edema are vasodilatation and reverse of fluid overload. [33] However, divers and swimmers are considered dehydrated. [30] The rationale for beta-2 agonist inhalation can be increased alveolar fluid absorption of pulmonary edema or comorbidity of SIPE and acute asthma. [17,34,35]

NPPV-treatment of SIPE has been described in seven reports in addition to our Swedish cohort. [14,23,36-41] Theoretically, NPPV should be beneficial for treatment of SIPE based on the assumption of reversal of the pathophysiology of SIPE. NPPV is also proven effective and safe for treatment of cardiogenic pulmonary edema. [42] Even though pathophysiology still remains elusive, SIPE is considered a hydrostatic edema. [35,43] The proposed mechanism is a combination of central pooling of blood and increased left ventricular afterload during immersion in cold water, increased pulmonary capillary pressure in physical exercise together with increased negative alveolar pressure during head-out immersion. The result is an increased pulmonary capillary transmural pressure gradient that leads to fluid filtration into the pulmonary interstitium or the alveoli, sometimes also to capillary stress failure. [44] NPPV is thought to augment clearance of hydrostatic pulmonary edema via reduction of left ventricular preload and afterload as well as decrease of the alveolar-capillary transmural pressure gradient. [45] Pressures of NPPV usually range from 5 to 15 cm H_2O in patients with cardiogenic pulmonary edema. [42] Adverse events of NPPV are mostly related to the face mask such as claustrophobia, air leakage or skin damage. More serious events such as hypotension, gastric aspiration, barotrauma, pneumothorax or hypercapnia are rare. [46] In our cohort from Vansbrosimningen, patients with low oxygen saturation are treated with CPAP by facial mask with pressure of 7.5 cm H_2O and an inspired oxygen fraction of 30%. Treatment

cycles of 20 minutes are followed by 10 minutes pause, breathing air to wash out oxygen before decision on further treatment or discharge. Nevertheless, efficacy of CPAP for treatment of SIPE has not been proven. SIPE resolves spontaneously when the patient is removed from water, warmed and rests. Therefore, CPAP, as well as any other SIPE treatment, should be compared to spontaneous recovery for evaluation of unbiased efficacy.

Reflections on CPAP in breathhold divers

Whether CPAP could be advisable for treatment of respiratory distress in breathhold divers is unclear. The pathophysiology of respiratory distress in breathhold diving and SIPE/IPE has several differences. [30] Moreover, differential diagnosis between pulmonary edema and barotrauma might be difficult. CPAP might be safe when there is no suspicion of arterial gas embolism or pneumothorax. CPAP has low risk of barotrauma when compared to invasive respiration, since it is applied with low pressures. [42] However, even low pressures might aggravate unidentified barotrauma, gas embolism, pneumomediastinum or a small pneumothorax.

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Discussion

I Barković: From Vitomir's presentation yesterday where he's hanging breathing oxygen on the line with head down, do you know what he does? He said that he felt much better using CPAP. Breathing from a scuba tank with regulator, head down, is a CPAP and some kind of evidence that it works.

C Seiler: This is the study that you were talking about. The regulator either on the back or on the front is a CPAP.

F Silva: It is very exciting for me to see love for some B-lines. You mentioned different kinds of B-lines when you were talking about your slide. Yesterday we briefly mentioned distribution of B-lines, but also the kind of B-lines. If you do enough lung ultrasound, you're going to see there are different kinds of B-lines. Some are brighter and better defined and more spread apart. Others are thin and a lot of them side by side. There is no paper that I'm aware of published on this. We have got to figure out what is the normal, pulmonary edema of freediving or what is lung squeeze. One definitely seems to have an inflammatory component and one is not, it's just fluid. Years ago Liechtenstein in the first edition of his book had a comment, one paragraph on B3 and B7 lines, which is the distance between the lines. One would be hydrostatic edema that he described in CHF, the other one in ARDS. I believe that we see the B7 lines in squeeze, not the B3 lines. That would be a big difference in future research. Liechtenstein stated that the inter-observer consistency was negligible, that it was impossible to teach this. There was no way to measure or to put into words that is something that you can relate to everyone he's examining. So he gave up. Maybe it's time for us to try to differentiate what the expected pulmonary edema is and what lung squeeze B-lines are.

C Seiler: I agree with that to see the nuances of B-lines, you have to note the ultrasound. We won't be able to teach this. The SIPE B-line is a very fine, thin, almost white line.

P Germonpré: Are these B-lines that you see a sign of interstitial edema?

C Seiler: I cannot tell. It might be the first stage, the interstitial edema or it might have become alveolar edema. I can't differentiate that.

P Germonpré: That was my second question. If you see the radiologic images, does that translate to B-lines? Is there any radiological imaging available somewhere? Has anybody taken x-rays or CT scans from a freediver with squeeze immediately after? What does it look like? Is it interstitial?

P Lindholm: If it can resolve in an hour, then by the time you get the diver to the hospital, it might be mostly gone.

S Hopkins: My concern about ultrasound is not that it is not sensitive or not an incredibly useful clinical tool. My concern is that you take a technique that is intrinsically qualitative and put it into a research context without appropriate validation and controls. This tries to make something that is qualitative, quantitative without doing the underlying fundamental research. The result is your metric. I'm not just saying B-lines or anything like can't be considered quantitative when the foundational work to show that hasn't been done.

C Seiler: There are some qualitative/quantitative translations for B-line count. Not too many, but there are.

V Maričić: On the CPAP usage and experience with ultrasound, I never liked it. It seems subjective for me. I didn't see a clear correlation with symptoms and the feeling of the diver with the number of B-lines. There was kind of a correlation, but it all seemed very subjective in my personal experience. I would sometimes come to research and already have B-lines without even diving. Then sometimes I would dive but have less B-lines. It was always like a guess.

C Seiler: The symptoms and the saturation are what counts for the severity.

V Maričić: We used to travel with the CPAP machine for two years and then use it extensively in recovery. I can definitely say it didn't hurt, but I also can definitely say that underwater was working much better. I have a feeling that with the exercise I showed and especially when it's combined with stronger balloons, it kind of comes down to the same thing. My objection on the CPAP was that I wanted to crank it up higher. I was using it on max already and for me it was just too light.

M Hårdstedt: Sue, when we started to use ultrasound, we actually did it blinded. The group of physicians on site did a clinical examination just relying on their ears, listening for crackles during lung auscultation and looking at the peripheral oxygen saturation. Then we had a closed room where Claudia and her friend performed ultrasound examinations. We also brought in patients or individuals that had been swimming without symptoms so we had a control group where we could see that they did not have B-lines at all.

C Seiler: No. Actually, I think there was a case of asymptomatic edema.

M Hårdstedt: Not sure that patient was completely asymptomatic, but it is true that we have seen a few cases of asymptomatic edema as well. Anyhow, we did start by comparing ultrasound results to clinical examinations. To start with we were not sure about how to interpret the ultrasound findings in this context. We wanted to make sure that we had a control group and did lung auscultation blindly and listened for crackles.

MODELING PULMONARY EDEMA IN BREATHHOLD DIVING

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Background

Breathhold (BH) diving is a popular sport. Some divers experience crackles, wheezing, or hemoptysis after dives, particularly with greater depths or cold water. We propose a clinical schema of BH diving-induced lung injury (Fig. 1) comprised of four distinct pathological processes: (i) tracheal bleeding, (ii) small airway edema, (iii) alveolar flooding, and (iv) alveolar hemorrhage. The first type arises from airway pressure at depth becoming negative relative to ambient, thus exerting traction on mucosal capillaries of the larger airways that are supplied by bronchial arteries directly off the aorta. The result is hemoptysis due to petechial hemorrhages within the upper airway. [1] The second type involves transudation of plasma fluid due to increased pressure differential between distal pulmonary capillaries and the bronchovascular interstitium, which ends at the level of terminal bronchioles. [2] Edema fluid cuffing around the small airways manifests as wheezing or lower airway congestion. [3] The third type involves alveolar ductal and septal edema in the most distal interstitial region bounded by tight low-permeable epithelium, which is separate from that of airways and contains lymphatic vessels only at corners. The precise site of alveolar leakage is controversial, but there is disruption of surfactant and flooding into the alveolar airspace. [4] The result is impaired gas diffusion, patchy consolidation, and symptomatic lung restriction. The fourth type is rare and involves direct tensile or shear stress injury to the capillary endothelium, causing alveolar hemorrhage with potentially severe or fatal consequences [5], consistent with the tragic death of freediver Nick Mevoli in 2013.

Normal BH dives involve lung gas compression and compensatory blood shift into the chest. [6] Without blood shift, there would hypothetically be a sudden painful drop in negative intrathoracic pressure around residual volume caused by loss of chest compliance due to inward rib flexion and upward diaphragm stretch. In reality, there is a more gradual transition from positive to negative airway pressure as depth increases, defining a pressure reversal depth Z_R. This is typically sensed by divers around 20 to 40 msw depth, depending on initial lung volume and chest compliance. Large changes in intrathoracic pressure due to inspiration, lung packing, and diving are transmitted differentially to the pulmonary capillaries and interstitial spaces within the lungs, increasing the gradient for fluid shift.

Considering the pathophysiology of the second category described above, altered chest pressure gradients at depth promote fluid filtration into bronchovascular bundles, which causes peri-bronchovascular edema and small airway narrowing that persists after diving. Fluid transudation into this compartment occurs much faster than clearance via lymphatic vessels. We hypothesize that severity of post-dive wheezing and lung restriction correlates with increased volume of peri-bronchiolar interstitial fluid, thus reducing airway caliber and maximum expiratory flow.

Since physiological measurements are difficult to obtain underwater and pulmonary data is very limited, we constructed an integrated systems computational model to predict effects of BH dive profiles on blood redistribution and pulmonary pressures. We focus here on studying lung fluid shifts and estimating the effect of depth and initial surface lung volume on fluid filtration into the airway interstitium.

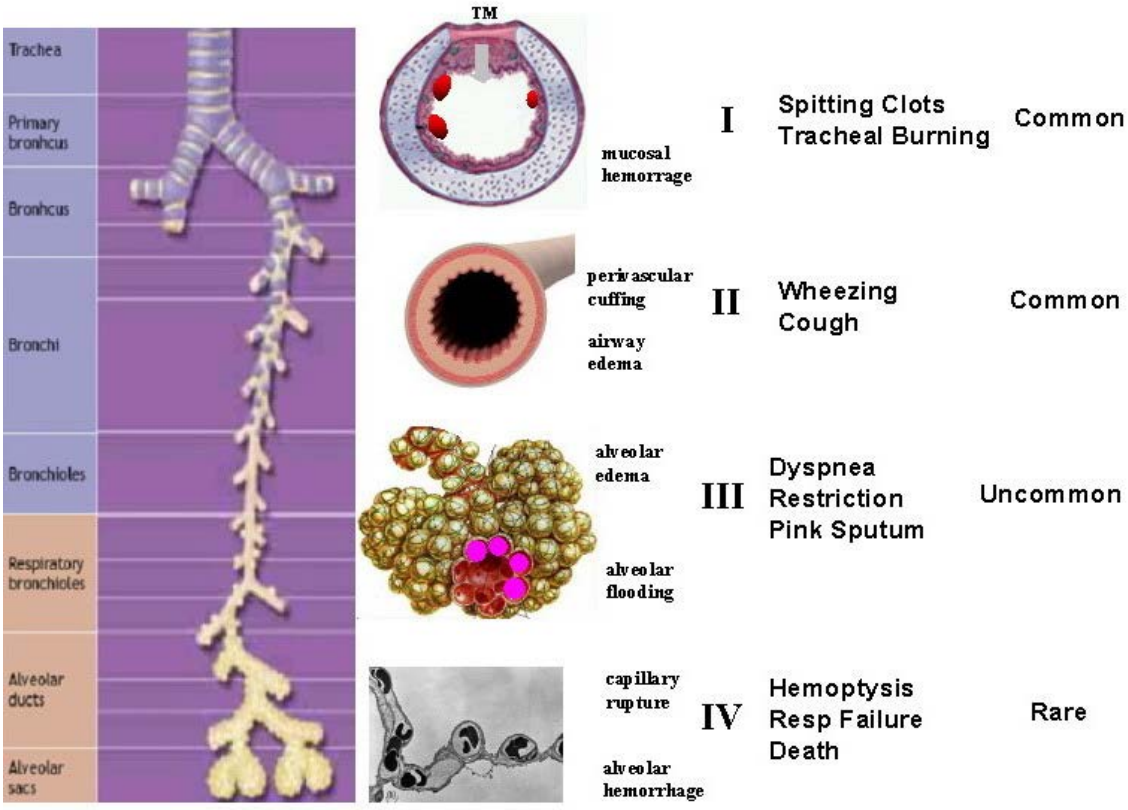

Figure 1. Four categories of potential lung injury due to breathhold diving.

Methods

The model is a software tool that runs on a laptop computer (Fig. 2). It contains modules to simulate the heart and cardiovascular system [7], pulmonary and abdominal mechanics, lung and airway mechanics [8], and fluid exchange with the airway interstitial space. Component properties and system dynamics are specified by equations that are solved and updated in small time steps. Model parameters were calibrated from many published sources, representing standard physiology of an average adult male.

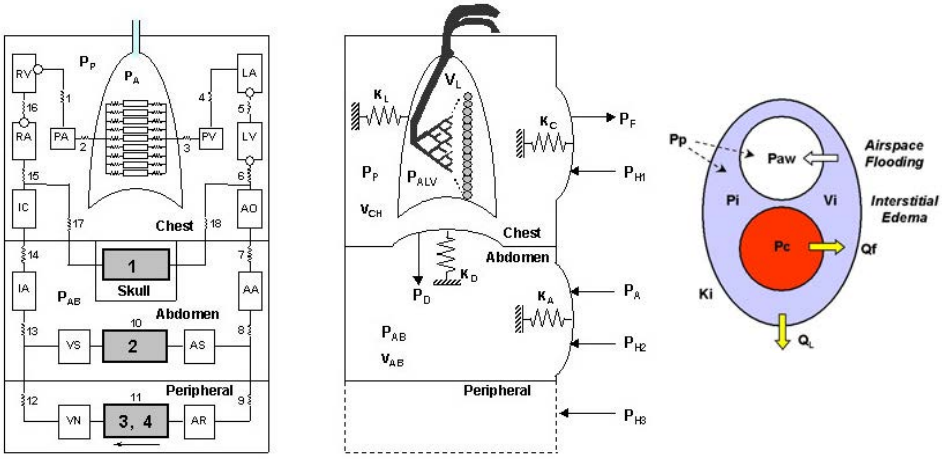

Figure 2. Computational model: Cardiovascular (left). Pulmonary (center). Bronchovascular (right).

Input is the dive depth profile and pre-dive breathing protocol, which includes initial lung volume. Blood compartments represent heart chambers, great vessels, central and peripheral vasculature. Heart rate, chamber contractility, and autonomic vascular responses are regulated by defined control rules. Immersion and depth exert added hydrostatic pressures to each compartment according to respective vertical centroid location. [9] The pharynx and airway tree were divided into 40 serial compartments. There are 42 parallel alveolar regions in a vertical column. Each element has an individual compliance curve, and extrinsic pressure depends on anatomical location. Alveolar volume responses are governed by the local pleural pressure and surfactant kinetics. Collapsed alveoli reopen during ascent according to air expansion and stochastic spectrum of opening pressures.

Airway interstitial pressure Pi changes according to a complex relationship between airway pressure Paw, pleural pressure Pp, vascular pressure Pc, and lung stretch V_L, [10] and is subject to a short first order delay time constant to account for extracellular matrix fragmentation and recovery. [11] The Starling equation governs fluid filtration from pulmonary capillaries into adjacent interstitial spaces, [12] modeled here as a single compartment, which is subject to slow lymphatic clearance that increases only marginally during the course of a short dive. Changes in vascular permeability to proteins that would modify the Starling reflection coefficient are not dealt with in this version. Resultant edema is thus of hydrostatic etiology.

Results

Dive simulations include initial immersion, inspiration, lung packing, and a triangular depth profile of descent and ascent, and post-dive recovery. Simple surface immersion raises pulmonary pressures, resulting in a small interstitial fluid shift. Lung packing above TLC shifts blood from pulmonary capillaries into the right heart, which causes a small additional fluid shift into the interstitial space. Descent compresses lungs causing blood to move from peripheral to central compartments, largely into pulmonary capillaries. Initially positive intrathoracic pressure at the surface reverses at 27 msw for a dive started at TLC, and 40 msw if preceded by lung packing (Fig. 3).

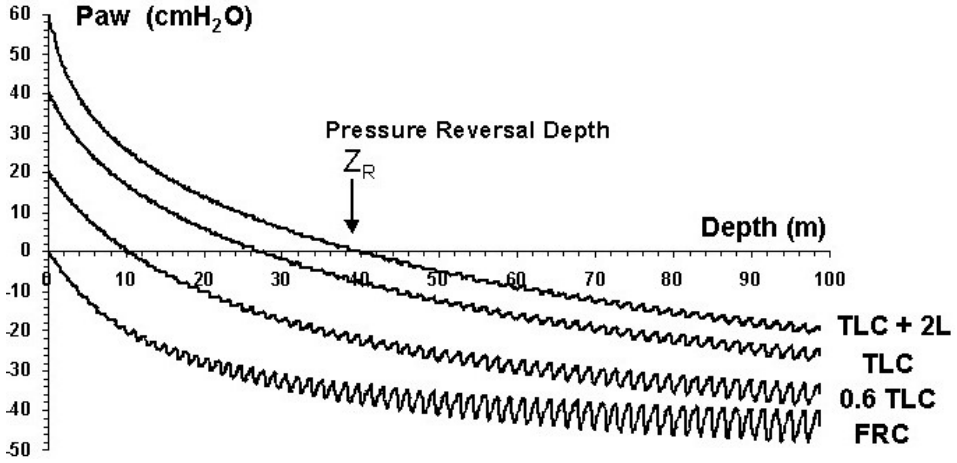

Figure 3. Predicted airway pressure versus depth for BH dives started at various lung volumes ranging from functional residual capacity (FRC) up to total lung capacity (TLC) plus 2 liters of lung packing. Airway pressure decreases from positive to negative relative to ambient at Z_R (pressure reversal depth). Pressure oscillations at depth are due to cardiac contractions within the low compliance chest.

Increasing negative pleural pressure lowers pulmonary capillary pressure Pc relative to ambient. Negative airway pressure Paw lowers the interstitial pressure Pi to a greater degree, thus favoring a net gradient for fluid filtration from the vascular to interstitial space (Fig. 4). Dives ranging from 10 to 100 msw result in greater fluid accumulation that follows a nonlinear trend (Fig. 5). The drop in interstitial pressure at maximum depth is more prominent than the drop in pulmonary capillary pressure. The former is driven mainly by the drop in pleural pressure with depth according to respiratory system mechanics. The latter is more sensitive to blood shift causing increased right heart preload. Interstitial fluid volume accumulation predicted by the model correlates with published data on depth-dependent forced expiratory volume FEV_1, forced vital capacity FVC, and oxygenation SpO_2. [3]

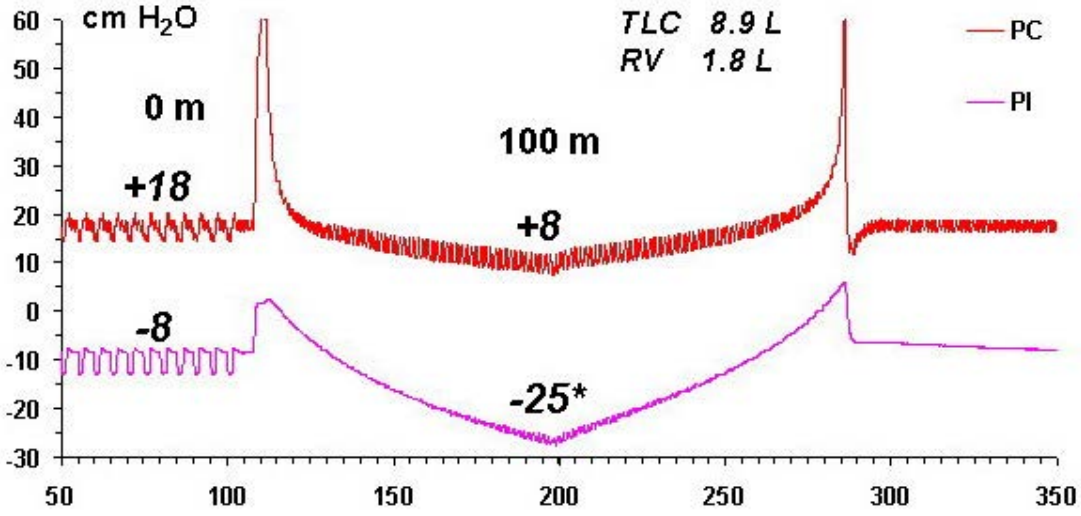

Figure 4. Breathhold dive to 100 msw. Horizontal axis is time. The difference between pulmonary capillary pressure Pc and interstitial pressure Pi drives hydrostatic filtration into the peri-bronchovascular space. It is mainly the drop of Pi to a highly negative value at depth that promotes the fluid shift.

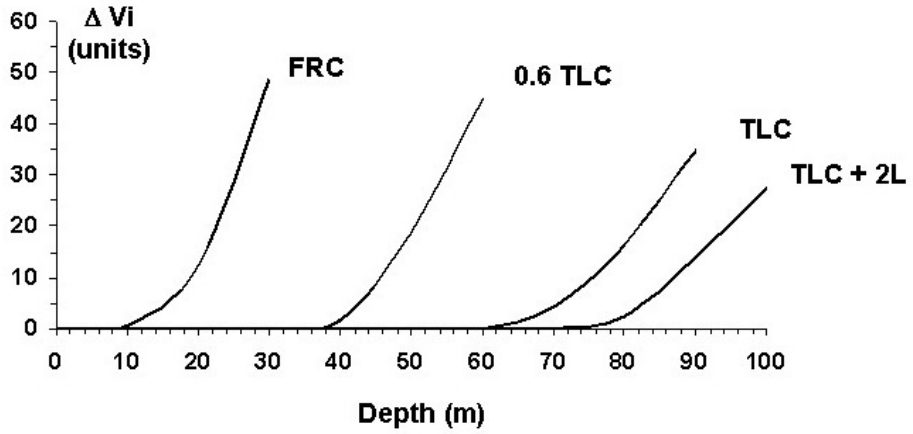

Figure 5. Increase in interstitial fluid volume from baseline for various initial lung volumes.

It is worth considering that ultrasound comets or B-lines detectable after dives [13] would not necessarily correlate with volume of airway interstitial fluid because comets likely indicate fluid in the

most distal peri-pleural space, which drains centrally via pumping within interlobular septa, and are potentially in a different compartment. [14] Distribution of fluid would be better characterized by computed tomography, although this would be impractical for most diving studies. Fluid dynamics of active and passive lymphatic drainage involving multiple lung compartments is a complex topic beyond the scope of this analysis but may be a future key to understanding post-dive edema clearance.

Conclusions

Simulation results provide a physiological rationale for typical fluid shifts driven by highly negative interstitial pressure. Cases that might involve major departures from model predictions would suggest additional presence of abnormally high pulmonary capillary pressure Pc responses. Model predictions therefore reveal a mathematical boundary that distinguishes two modes of diving-induced edema: (i) a nominal mode dominated by lower Pi, typical of most dives, and (ii) a pathological cardiovascular mode dominated by high Pc. The reasons why divers experience varying degrees of symptoms and results in lung function studies after equivalent dives remain unknown.

Further development of the model may include pressure-induced opening of capillary gap junctions at high vascular pressures that would allow protein shifts via exudation involving an increased Starling reflection coefficient altering the colloid osmotic gradient. The degree to which this might occur in diving is presently unknown, and at this time would be speculative.

Desirable experiments needed to validate these concepts would include measurement of negative airway pressures *in situ* at depth, and correlation of model-predicted increases in interstitial fluid volume with observed ultrasound lung comets to establish a dose-response relationship with depth and time.

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Discussion

B Smith: Wonderful talk, I love the simulations. I was imagining measuring deep in the airway. Could you just have them hold a transducer in the mouth? As long as the airways are open, can it be communicated?

J Fitz-Clarke: That's a good question. You'd have to ask the excellent divers in the room here but would need to have patency between the mouth and the lower airway.

B Smith: Could we just model mice and then "squeeze" the mice, do a model comparison there?

J Fitz-Clarke: We need to talk about that.

B Smith: That seems pretty doable. Finally, from thinking about this, the cuffing and ventilator-induced lung injury, we always put a lot of blame on the tension around the airway dropping that pulmonary interstitial pressure. I wonder if that might be a factor that's exacerbating worse than the predictions, if the alveoli are tethering that airway in place.

J Fitz-Clarke: It could be. You'd probably see a lot of tethering in the beginning of the dive, when there's high lung volume and then that tethering would decrease. Maybe that loss of tethering also contributes to loss of airway caliber.

B Smith: Squeezing the edema out, maybe that, too. Interesting thought.

P Lindholm: When Steve Loring measured airway pressures in the Brigham study, divers could open their airways and keep them open so we could measure the airway pressure. One of the things on my wish list for a long time, adding to yours, is how about doing a deep dive and then switching from freedive to a scuba dive so you open up your airways at that point and you do some measurements and then you probably have to go up by scuba because you use up your oxygen stores. That would be one way of doing it. If we have collapse it wouldn't get all the airways but it could give you a relaxed airway pressure of some kind. Maybe we need an underwater habitat as well.

J Fitz-Clarke: That's a great idea, I like that.

P Lindholm: Your third wish list item, we actually did some of those measurements this summer. We've been starting to do B-line data on competitive divers. It's been done in the past, but we're adding to more data.

J Fitz-Clarke: I look forward to seeing that.

V Maričić: We just discussed quickly about taking a measurement with a sensor in the mouth. While freedivers go down, they always have their mouths closed. Either you do a reverse pack or a mouth fill to get the air from the lungs back up to the mouth. These guys can definitely do dives to 100 m plus without keeping the glottis closed all the time. They can basically have a pause where they open the glottis and then they are equalized and you get a reading. Then they do the next equalization when needed and continue to dive, so there is a way.

LUNG DAMAGE IN SQUEEZE: IS IT LEAKING OR IS IT BROKEN?

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Pulmonary barotrauma of descent (lung squeeze) in freediving is traditionally seen primarily as a hydrostatic phenomenon, where the pressure differential between the blood inside the alveolar capillaries and the air spaces overcomes the structural integrity of the alveolar-capillary interface, resulting in alveolar hemorrhage.

It is believed that the risk of lung squeeze can be decreased by factors that minimize the alveolar-capillary pressure differential, like increased chest wall and diaphragm flexibility, ability to relax the pectoral girdle, chest wall, diaphragm and abdomen, ability to relax into hypercapnic contractions (or even actively move towards the direction of contraction), promote engagement or the mammalian dive response through training and warmup dives, and slow gradual depth progression in training.

Another mechanism that may be involved in the pathophysiology of lung squeeze is shear stress. It is known in freediving that certain movements that cause traction on the lungs and respiratory tract may cause injury, like neck extension, torso extension and energetic elevation of the arms. These movements may inflict shear stress on the pulmonary tissue, which at depths beyond RV-equivalent may have decreased tolerance to deformation due to decreased flexibility as a consequence of deaeration secondary to blood shift and volume loss.

It has also been suggested that the presence of respiratory infections may facilitate the development of lung squeeze. A case will be reported where an elite freediver performed a dive in competition after having had a sonographically detected pneumonia two days before the dive. While this dive was well within the ability of this diver, he did develop a lung squeeze. The author suspects that the presence of a pulmonary consolidation inflicted intense shear stress on the surrounding parenchyma during descent, causing structural rupture and hemorrhage.

There may be a synergistic interaction between the hydrostatic and sheer stress mechanisms. There are multiple reports of freedivers who at some point in their depth progression start to present frequent lung squeezes. Some of these freedivers eventually overcome this, but many are simply unable to progress in depth without squeezing, in spite of great efforts to improve their technique and increase chest wall flexibility. It is suspected from the autopsy of Nicholas Mevoli that recurrent squeezes cause pulmonary scarring, likely both secondary to inflammation and to fibrotic regeneration of ruptured tissue. These areas of scarring/fibrosis may act as anchor points during the parenchymal deformation during a dive beyond RV-equivalent depths, inflicting shear stress and tissue rupture. Therefore, it would not be unreasonable to assume that recurrent hydrostatic squeezes (e.g., beginner freedivers with poor relaxation, or diving beyond their abilities) could potentially promote inflammatory scarring/fibrosis, and then develop points of tissue anchoring, which in turn would be sources of shear stress independently of the development of good diving technique, relaxation or chest wall flexibility.

The understanding of these two potentially synergistic mechanisms involved in the pathophysiology of lung squeeze reinforces the importance of efficient strategies for depth progression and attention to avoidance of recurrent squeezes.

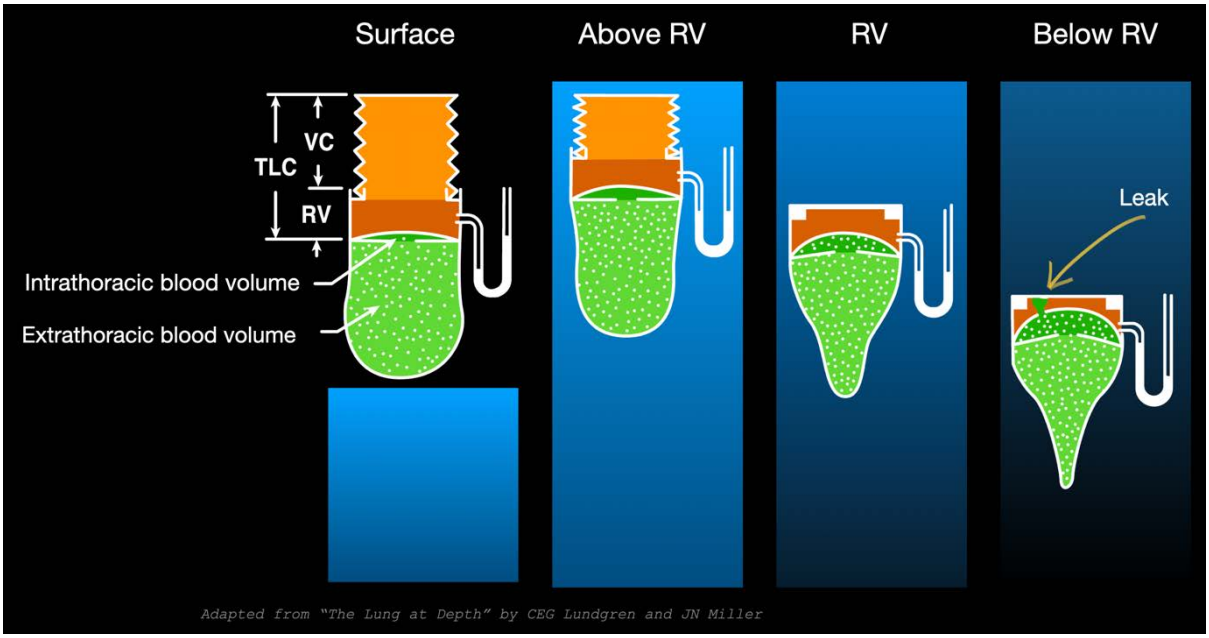

Figure 1. Diagram representing the changes in volumes and fluid shift causing hemorrhage in lung squeeze.

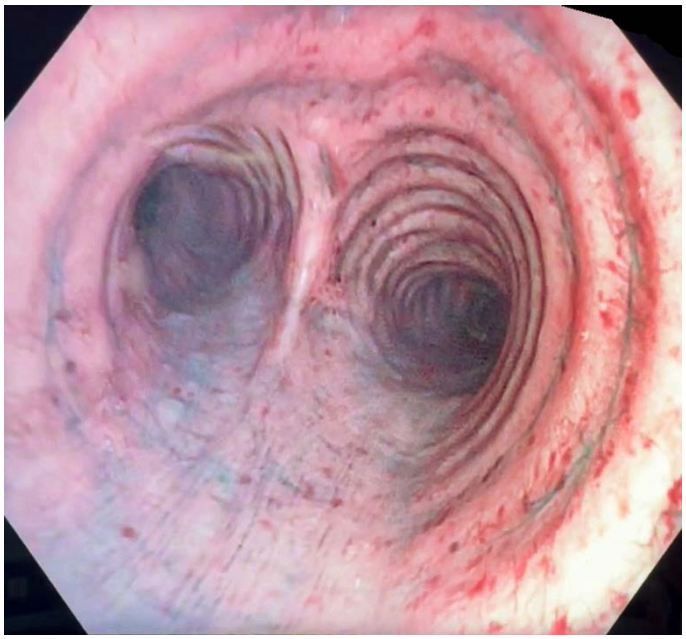

Figure 2. Petechiae may be seen on the respiratory mucosa, reflecting the intense negative pressure involved in lung squeeze. Mucosal petechiae and hemorrhage in pulmonary barotrauma of descent in breathhold diving: direct observation of a suspected pathophysiologic mechanism (Submitted for publication; Silva F, Gouin E, Lindholm P. 2023).

Figure 3. Hyperextension has been suggested as a cause of lung squeeze by promoting shear stress.

Discussion

V Maričić: How fast was the bronchoscopy done after that?

P Lindholm: The diver had consecutive dives. The bronchoscopy was a day after.

V Maričić: Do you remember whether it was bilateral or one side more affected?

F Silva: It was bilateral, the petechia were seen on both sides.

V Maričić: From personal experience, I was sensitive and logical that by overextending extremities and chest on very deep dives would mean that you can get squeezed more easily. I actually did a test on myself without the neck part, but just the positions of the arms and stretching and depth I've tried that on a 120 m dive, which is for me still a challenge. I did not see a difference, which was really interesting because I always advise athletes to make more gentle pulls, and to try to keep the chest relaxed and closed. I say, I'm never going to know if I don't do a test on myself. It went pretty good.

A Luks: In thinking about some of the discussions going on about the pathophysiology here, it's going to be important that as we try to sort through this to make distinctions between the pulmonary circulation and the bronchial circulation. The pressure dynamics are very different in these areas. The bleeding that you're describing in these videos that you've shown this morning, this is all proximal airway. This is all a pressure differential between the bronchial circulation and the trachea and the bronchi. What we saw yesterday was that the mean partial pressures and, therefore, the pressures in the bronchial circulation are sky high when divers are at these great depths. Then in the alveolar space

you've got pulmonary capillary pressures that are going up, but the pulmonary circulation is a far lower pressure circulation than the systemic circulation. The pressure differential between the pulmonary capillary and the alveolar space may be much different than what you're dealing with in the proximal airways. Granted, these vessels are different. It's a much thinner alveolar capillary barrier than in the alveolar space, but that's a big distinction that needs to be made when you're trying to think about pressure differentials that are leading to blood and everything else leaking out of various spaces.

S Hopkins: I like that kind of distinctions, but I wonder if hydrostatic is the best word. When you say hydrostatic, I think hydrostatic edema and pressures in the pulmonary capillaries. With the petechiae I wonder if we should just call it petechial predominant to describe what it actually is rather than hydrostatic, which raises more possibilities for confusion with other things.

F Silva: I entirely agree with that. Two disclosures here: one, before being a doctor, I was a mechanical engineer so I think hydraulics. Second, this presentation was first prepared for a 100% nonmedical audience, and I thought hydrostatic was more accessible. I entirely agree it's time to change that.

R Moon: Thanks for a very nice talk and some wonderful videos. There's one other potential mechanism for this, and that is, essentially, heart failure. There have been some direct arterial measurements of blood pressure at University of Buffalo during breathhold diving. They have recorded some astronomically high blood pressures on the order of 300/100. I wonder if transient heart failure might also contribute to hemoptysis and, essentially, the syndrome of squeeze.

F Silva: Yes, super pertinent, but wouldn't that also fall into the to-be-renamed hydrostatic category of damage?

R Moon: Exactly, sort of like SIPE.

F Silva: The leak would be from the pressure differential.

I Barković: If I saw it right on your video, you also have from right upper lobe more bleeding coming out, not only petechial. It was fast, but maybe you have something similar to a leak.

F Silva: There’s not a mention in the report specifically, but we can review the video together.

B Smith: What you see on the ultrasound on the pneumonia patient, was that also present after the dive away from the pneumonia or was it focal to the region that had the pneumonia to start with?

F Silva: The B-lines were all over but were more prominent on the left side. I didn't do the counting of B-lines, but they were more prominent on the side where the pneumonia was.

B Smith: So a systemic effect.

F Silva: Correct, yes.

ARE THERE LONG-TERM CONSEQUENCES OF BREATHHOLD DIVING AND LUNG SQUEEZE?

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Introduction

Freediving is a ubiquitous activity with prehistoric origins that involve highly integrative physiology and extreme responses to both exercise and asphyxia during progressive elevations in hydrostatic pressure (Fitz-Clarke, 2018; Patrician *et al.*, 2021a). Upon immersion, the lungs are exposed to a combination of forces; 1) the increase in hydrostatic pressure compresses lung volume (Fitz-Clarke, 2007); 2) peripheral vasoconstriction centralizes blood volume and increase blood pressure (Ferrigno *et al.*, 1997); and, 3) hypercapnic hypoxia progressively develops with exertion to instigate involuntary diaphragmatic breathing movements (Bain *et al.*, 2018). Pulmonary gas exchange relies on the thin and delicate pulmonary capillary interface (West *et al.*, 1991), which is susceptible to excessive strain. Consequently, it is perhaps not surprising that deep diving can transiently impair pulmonary gas exchange efficiency and promote pulmonary edema in a depth dependent manner (Patrician *et al.*, 2021d). In normal dives, these transient impacts to pulmonary gas exchange resolve within 2.5 hours and small changes to airway resistance and reactance are suggestive of an improvement of lung compliance.

Lung squeeze is a form of lung injury that is primarily an affliction of freedivers who dive to depth. Despite almost 70 years of interest (Carey *et al.*, 1956) the mechanisms have yet to be elucidated. What is known, however, is that lung squeeze manifests shortly after surfacing and is broadly characterized by hemoptysis (Boussuges *et al.*, 1999; Lindholm *et al.*, 2008; Patrician *et al.*, 2021b), and is often associated with a productive cough, dyspnea, and chest tightness (Cialoni *et al.*, 2012; Patrician *et al.*, 2021c) that lead to an impairment in pulmonary gas efficiency (Patrician *et al.*, 2021b) and reduced oxygen saturation (Linér & Johan, 2008). Following a severe lung squeeze, pulmonary gas exchange has been shown to be severely impaired for at least up to 4 hours (Patrician *et al.*, 2021d). Computed tomography (CT) imaging performed within 24 hours of a lung squeeze (in otherwise healthy individuals) have identified patchy bilateral lung opacites (Boussuges *et al.*, 1999; Kalemoglu & Keskin, 2006; Prediletto *et al.*, 2009; Cialoni *et al.*, 2012), pleural effusion (Kalemoglu & Keskin, 2006), bilateral interstitial infiltrates (Gempp *et al.*, 2013), bilateral alveolar infiltrates (Kiyan *et al.*, 2001) and a pneumomediastinum (Henckes *et al.*, 2011). Consistently in all cases where CT scans were repeated within 3 weeks-3 month findings were unremarkable (Kalemoglu & Keskin, 2006; Prediletto *et al.*, 2009; Henckes *et al.*, 2011).

However, each of these studies on CT analysis focus on overt clinically relevant findings and lack nuanced metrics of lung status (e.g., structure, tissue density and airway pendulosity). Thus, leaving a large gap in our understanding of the subclinical, but likely physiologically relevant, impacts of repeated bouts of hydrostatic induced lung compression, and lung squeeze. Therefore, our objective is to explore quantifiable metrics of lung squeeze on lung structure and airway pendulosity.

Methods

Low-dose computed tomography (CT) was performed in at full inspiration and full expiration in 14 divers with a history of lung squeeze. Divers (N=14, 1 female, 13 males; 37±8 years, 82±11 kg, 184±6 cm) had FVC of 7.1±1.0 L, FEV1 of 5.3±0.7 L (0.75±0.06 FEV1/FVC), and TLC of 9.4±1.7 L. CT images were analyzed by trained radiologists. Airway diameter and collapse were quantified across the tracheal tree using calipers (with matched location), and lung density measurements were performed (Imbio). Lung voxels were classified using pre-determined HU thresholds of normal (inspiration HU

above −950, and expiration HU above −856), functional low density (inspiration HU above −950, expiration HU below −856), and persistent low density (inspiration HU below −950, and expiration HU below −856). Control scans (n=17) were collected at full inspiration only due to imaging constraints. The protocol was approved by the ethics committee at the University of Split School of Medicine and both informed oral and written consent was obtained, and conformed to the Declaration of Helsinki, except for registration in a database.

Conclusion

The lungs of divers with a history of lung squeeze show no evidence of air trapping or interstitial fibrosis, and the tracheobronchial tree demonstrated preserved patency on CT – which aligns with previous studies. The main purpose of this study is to utilize novel analytics of airway diameter, airway collapse, and tissues density to explore the nuances of lung structure and assess the sub-segmental region. The main findings are still preliminary but suggest there may be subtle changes to lung structure. However, their physiological and/or structural relevance require further elucidation.

Funding

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Discussion

G Boswell: Thank you, very nice presentation. I love the CT data that you're showing, it's good. It is really problematic to do the research that you're trying to do because there are so many clinical scans. Everyone's program scanner is just a little bit different so all the data you're getting is not all on the same standards, and one of the big caveats being how you control for the depth. How do you get maximum expiration, trying to figure out what airway times are and to add to that, airway wall thickness. But as we look at the small, < 2 mm in diameter, it gets problematic because of how close you can do that. One of the ways would be air trapping to see what the lucency of everything distal to that small airway is. That's where inspiration and expiration comes out. We still don't have a good idea what normal is for full expiration in the normal population, and that's going to vary some based on patient geometry, size, gender. Until we have good data on that, it's going to be hard to really answer this. If you can have one individual before a dive and after a dive and then do it right away, so you have an internal control, then it's going to be difficult to define that.

A Patrician: We're hoping to repeat all of this. We will have an in-person comparison. But at least we'll have some sort of direction.

G Boswell: Right. If you can do them all on one scanner and thinly sliced as possible and train the technologists to the maximum inspiration and expiration.

A Luks: Dynamic CT is better than a static CT just with a breathhold.

G Boswell: With dynamic CT you're not going to be able to do a study of the entire lung with the dynamic. There's going to be some heterogeneity throughout the lung. Ideally, you do dynamic 20 frames through breathing in and breathing out through the entire chest. Perhaps you can do a subset of dynamic, maybe three or four slices through the chest, but do the rest of the chest full inspiration and the rest of the chest full expiration to have full ex and ex volumes.

A Patrician: What's the radiation load for dynamic versus static or a low dose?

G Boswell: The nice thing about the lungs is that there is such a difference in contrast between lung tissue and air that you can lower radiation dose quite a bit. You can make up for low radiation dose with some mathematical methods and perhaps even lowering the KV a little bit could do that. You can do it at an acceptable low dose with that, but not for 20 frames of the entire lung. The key thing is getting the setup properly before you start with the first patient and making sure you've got the algorithm with as little image noise as possible that's acceptable on a late model machine. Then feed it into a computer algorithm that reads it for you.

R Moon: Nice work, Alex. You mentioned earlier low FEV1/FVC in some of your subjects and that can be due to the low FEV1 and a normal FVC, but it can also be due to a normal FEV1 with a high FVC, which was described by Jerry Meade as disynaptic pattern. He interpreted this as during growth the alveoli grew faster than the airways meaning it can be a completely normal phenomenon. That's suggested by you with a TLC measurement 9 L in one of your subjects. It may well be just a normal variant rather than anything suggesting an obstruction.

A Patrician: Yes. Divers typically have higher lung volumes and we don't know if their lung volumes have increased over time because of the training that they're doing and whether that's going to affect anything.

A Luks: We see a lot of young, fit, healthy people. You do spirometry on them and they have normal FEV1 and FVC, but they appear obstructed based on the ratio of these two parameters. It's unclear what to do with that and whether they really have obstructive lung disease. The other thing I would say is if you're going to be looking at FEV1/FVC you've got to remember that what constitutes normal varies based on age so depending on how narrow or wide the age range is for your divers, what would be considered abnormal is going to vary.

A Patrician: That would be an average of their predicted values based on their age, yes.

M Hårdstedt: These are all male divers?

A Patrician: Mostly male, but we have some females.

M Hårdstedt: You were referring to that one diver that was kind of sticking out as a female.

A Patrician: Yes, we had a mix of both men and women, but we had more men than women.

K Van Hoesen: Fascinating data. I loved the plot grams with the difference between the right and the left lung. Yesterday I reached out to my friend Peter Hackett who is one of the high altitude experts in hypoxia and altitude sickness. I asked him why they think there's a predilection for high altitude pulmonary edema to start in the right middle lobe and maybe only be there or that's spread from there? He replied because of two reasons: one, there's greater hydrostatic pressure in the right middle lobe, and second, it's also the only lobe without collateral ventilation. Maybe going forward and look at some of your plots and the B-lines whether that has anything to do with either SIPE and its presentation or in lung squeeze.

V Maričić: Great data. Just wanted to say the release of your lung squeeze guy that was diving to 91 m, it was 92 m.

CURRENT STATE OF LUNG SQUEEZE GUIDELINES: THE GOOD, THE BAD, AND THE UGLY

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Introduction

Lung barotrauma, commonly known as lung squeeze, is a poorly understood medical condition. No granular data is available in freediving as of 2023. Much of what we know is from basic scientific research and experience from the field: training camps, competitions, and our own personal training experiences. This description provides the current state (of-the-art) of lung squeeze classification, severity perception, and return-to-dive guidelines currently in use.

Materials and Methods

Current AIDA International and United States Freediving Federation lung squeeze guidelines were reviewed along with recent AIDA incident reports and return-to-dive protocols designed by the author.

Conclusion

"All I know is that I know nothing" - Socrates. The current knowledge foundation on lung squeeze in freediving is extrapolated from basic scientific research and is anecdotal at best. We finally recognize the huge blind spots we have on this topic. Much clinical research and data collection is needed. Until we have more information, perhaps the safest answer is to wait longer and just say no.

Discussion

R Moon: Juan, that was a nice talk. What you were showing us was, in fact, hypoxia, which may have been contributed to by lung squeeze, but it could also be due to the fact that you're underwater, not breathing for a long period of time. What percentage of cases of this syndrome, people losing consciousness upon surfacing, do you think actually have some lung squeeze as we talked about five minutes ago?

J Valdivia: I'm not sure, perhaps 50%. That's a good question for Vito and Alexey. The one time I had my worst, my last competition, I had a squeeze. I had a feeling I was ascending already squeezed. I couldn't ventilate or recover and then I blacked out. I squeezed at the bottom and was coming up already abnormal.

V Maričić: You said that maybe it would be good to include in the competition form any previous incidents and how much they repeated. I don't think it makes sense. Everyone is just going to lie. You can follow your example, why would you put yourself into trouble? Why would you put any trust in people who you know don't know anything? Now, since all the experts are here, we can all agree that we don't know, why would an athlete risk his budget and time and everything for some people's opinion who don't know and who are very often with conflict of interest? That's a huge issue. The most fair and the best thing would be to, in case of incidents or reported incidents, have an onsite assessment on the day of the dive. If someone is healthy and can be cleared, a normal medical examination that they would go through anyway to get to a competition, then everything else other than that would be discrimination and subject to subjectivity, which is problematic. The same thing is with the grading that's suggested by the American standards that you were working on. The accumulation of points, while practical, you need to hide something and the symptoms do not necessarily reflect the severity of the event, is not objective or protective for an athlete, and it can be a tool that's going be used against the athlete. It creates fear and stress, which is going to create more disease. You have to hide them better. I have a feeling if we're not open about this and being realistic, which will then hopefully gain the trust of the freedivers, we're just not going to go anywhere. I know

it's hard and there's insurance and so many things involved, but this is the only way. When we compare this to other sports it feels sometimes like we're trying to put limitations on someone's personal attempt when they go into competition, which does not exist in any other competition, e.g., telling a marathon runner, if you become too exhausted, you're just going to be disqualified.

P Lindholm: We can discuss a few cases from a treatment and return to dive perspective. With yourself acknowledging that you lied in competition, and Vito's support of that kind of notion, I just put signs. Maybe we shouldn't talk about symptoms. If we're looking at this from the medical perspective, we can see what we can come up with as just signs. That's something we can measure because there are so many symptoms in respiratory distress.

O Christen: In the freediving competition environment it's a very specific, rare, special situation. 99.5% of freediving happens outside of competition situations. What can someone say who had an injury or suffered from symptoms that we until today call squeeze, and from tomorrow on call something different? The self-assessment is interesting because in regression freediving injuries do happen and freedivers would like to know whether they can continue diving. Juani, you just introduced something like being able to perform without discomfort. Every master freediver should be able to do that. We create a negative pressure in the lungs, is that actually a good thing to do or is that a crazy thing to do or would that be one of the self-assessment tools that we should discuss?

J Valdivia: If you feel it's going to cause harm or discomfort, don't. You don't have to go through all the maneuvers.

O Christen: If there's an injury and you perform the Valsalva maneuver you might actually aggravate the injury.

V Maričić: If it's an edema or a bleeding, it's like a hickey if you provide pressure.

A Molchanov: The period of recovery is really fast, on the order of 10 minutes. During the breaks you don't want to do vacuum exercises within 5 - 10 min after surfacing, but 1 or 2 hour afterwards should be fine.

S Hopkins: They're closed in 10 minutes, which doesn't mean they're healed in 10 min.

A Molchanov: It's an open question, something we should do in 1 hr, 2 hrs, or the next day.

V Maričić: I agree with that as well. When you say that people call and ask for advice and you don't know what to tell them this also happens to me many times, and I don't know how to assess it. That's why we cannot discard fear and stress because they are one of the major contributors. Because of all this hiding and unknown questions and no answers in the community, there is a lot of fear and stress. It would be a little bit demystified if everyone would get more relaxed about squeezing, they would squeeze much less.

DISCUSSION ON TERMINOLOGY

Moderator: ***Peter Lindholm***

P Lindholm: The problem with the definitions, today it's immersion pulmonary edema. Then, for some reason somebody swam when they were doing this and the term SIPE caught on. Scuba divers get immersion pulmonary edema because they don't swim or they do swim, but most of them don't exercise. Then there are military divers and the freedivers. We have a suggestion here to start broad because we do have symptoms from the lung. Immersion pulmonary edema is more of a generic term. The problem with 'DIPS' is we are looking into breathhold diving symptoms mostly and that would sort of bring in the high-jump divers and scuba divers a bit. I don't know if Freediving-Induced Pulmonary Syndrome would make that okay? We can talk about one overall term, but then also agree on something? Should we talk about tracheal or tracheobronchial problems compared to the further out problems? John, you had categorized four different kinds, but the two in the middle are overlapping a little bit.

J Fitz-Clarke: Yes, there's controversy over whether airway edema and alveolar edema are a continuum or are separate diseases. The first thought that came to my mind was Freediving Lung Injury. The second thing that came to mind is the similarity with a concussion. We call a concussion a concussion, but it could be a dozen different symptoms, headache, dizziness, nausea, confusion, poor concentration. It can have all those features, but we still call it a concussion. It's just a catch-all term that embodies a lot of different symptoms.

P Lindholm: We also have 'squeeze.'

P Germonpré: I'm not sure whether a general term like Freediving-induced Lung Injury covers exactly what we're talking about. Freediving is also surface-level freediving, static apnea, deep freediving. I would favor keeping the term squeeze even if it's a bit of a strange term to medical people who are not divers who might be remiss of their lungs being squeezed out. Basically, this is part of it.

P Lindholm: What do you think about whether it is two separate things? We discussed that, too. Can we say that at least we think it is two separate things? One is the hydrostatic or Starlings Law edema fluid shift, and the other one is maybe some shear stress or contusion.

R Moon: Peter, that's confusing pathophysiology with the description of the disease. Since we don't really know what's going on with squeeze, we should keep it simple, call it squeeze or whatever, at least a term that differentiates it from SIPE. Later, perhaps at the next meeting in five years, maybe we'll have more information on pathophysiology. With regard to SIPE, as you well know, tradition in medicine counts much more than logic. Since SIPE is now ingrained in the literature, it would be reasonable to keep it notwithstanding the British call it SIPO.

K Krack: The freedivers would like you all to know that freediving is one word. It's not free and diving. It's freediving. From here forward, it's freediving.

V Maričić: I think the way it happens, it's quite clear that there are these two major components or maybe even more. But it's almost impossible to distinguish early on. In most cases it's going to be a combination of both, it's a syndrome. Maybe something like concussion fits most. Since squeeze is already colloquial everywhere, just keep it simple. Freediving squeeze syndrome is something that encompasses everything.

O Christen: I like the term DIPS for the sound of it. I see a problem in freediving. Oh, he DIP'd, but that's a different thing in freediving already. So it will create confusion with the surface DIP of an athlete.

S Hopkins: These are in fact different things that happen. We've seen petechial hemorrhages at airways. We've seen evidence for bleeding from the right upper lobe. Those are not the same mechanism. Some of them could be related to squeeze, but some could be related to getting basically a hickey of your airways. For a broad catch term I would envision that there would be some categories of that. Just like in the altitude universe, there's altitude illness, but we have high altitude pulmonary edema and high altitude cerebral edema. Athletes have always referred to things colloquially but when you're trying to define science then we need to be more precise.

J Valdivia: Freediving-induced respiratory or pulmonary edema. People do dry settings and they go through the limit. Squeeze, would you exclude or include them from the definition?

V Maričić: Dry static should be included just like SIPE. Even if you take speedos and go for running or stair climbing in extremely cold weather, you don't have to be swimming, you're going to have the same result. This is a special case, but it's totally included freediving and the mechanics are going to be the same so, definitely, yes.

P Lindholm: If we call it a syndrome and we have an overview, colloquially people use different terms, but the medical literature needs to have some definition that makes sense in relation to all the other definitions that are in textbooks. We could say we have a syndrome, and then have categories going down. I don't have a chart, but we have immersion pulmonary edema, which can be tracheal issues, we could have capillary failure, we can have an immersion and that can go into, we don't know, but it could be airway or capillary. You would just sort of filter that down, and there will be maybe some unknown, some overlap, what do you have when you have 100 boxes and all these arrows?

A Luks: The way we do it in the altitude world is acute altitude illness, as opposed to chronic altitude illness. You can almost have acute freediving illness, these are all acute processes. Then you could have subcategories of that as you subsequently begin to define the entities.

P Lindholm: The problem is that there are other freediving issues besides the lung. Just saying acute freediving would be a little too broad. I want to keep it on the lung because you have ear and brain issues as well.

P Germonpré: You also could have it at the surface without even being in the water by just breathholding, exhaling, waiting for the contractions, you can squeeze. So it's not just diving, it's breathholding. So BIPs.

P Lindholm: That's good. You don't need to dive to squeeze.

S Hopkins: The counterpoint to that is the vast majority of this happens with diving and immersion. If we just call it breathhold, then don't we run the risk of confusing it with some random person on the street holding their breath?

P Lindholm: We do get into the very common sleep apnea problem if we call it just breathhold.

A Lussier: Are there any cases of sleep apnea with waking up with edema? No? I wonder. Because you wake up before contractions.

P Lindholm: Breathhold-induced pulmonary syndrome is still in the running

F Tillmans: Just let me know which ones to delete.

J Fitz-Clarke: My feeling is diving needs to be in there even if it's surface diving related.

K Krack: In the freediving community, obviously, we know that freediving is about time, depth, and distance. By default we know static apnea we know distance is all part of those subcategories of freediving, just like DCI and then AGE and DCS and on from there.

S Hopkins: In the interest of simplifying this list, I'm going to propose we take out the DIPS. For me, personally, FIPS is better than DIPS. Now we can talk about the nuances of the other two.

P Lindholm: If we say freediving induced, if you hold your breath on empty lungs dry, it's part of freediving training. That would probably catch more of those than if we say Breathhold Induced Pulmonary Syndrome.

A Molchanov: Exercise is to prepare for breathhold in freediving we can call it as well. We had some cases where guys were doing breathhold stretches with packing, plus stretches and get lung collapse. There was a case in the last 10 years. He was stretching, not really breathhold, but packing, doing their breach with packing. His lung collapsed and instead of competitions was in the hospital for a week. All was fine and everything healed. But that's another trauma, does it fit there?

P Lindholm: You can get pneumomediastinum from playing the trumpet or just jumping up and down in the gym. There are reasons to get all kinds of traumas and diseases. We need something that encompasses most of these. That would be a catch phrase and a little more acceptable to the medical community than a squeeze. It is not really a squeeze, it's a pulmonary edema.

C Seiler: I would like to know from you divers, is it important for you to get the depth into the syndrome or doesn't it matter if someone holds his breath and dives flat just to make it clear for me to follow? Because it makes a big difference if it's hydrostatic pressure at depth.

F Silva: The term freediving infers depth.

C Seiler: It's not important to me. Does depth play any role? Otherwise, are those included?

P Germonpré: They're included. Breathhold diving seems more logical than freediving. What would the freediving community prefer, to have breathhold diving or freediving, which is simpler, but breathhold diving, to me, sounds more real than freediving.

P Lindholm: We could have breathhold diving induced pulmonary syndrome, and then just have the short. How do we distinguish freediving from spearfishing and is spearfishing freediving?

K Krack: Freediving is the travel mode for the spearfishing.

A Patrician: When you talk about it, do you say, I'm a spearfisher or I'm a freediver?

J Valdivia: It's freediving.

A Patrician: If you ask a spearfisher, I'm not a freediver. I only spearfish.

P Lindholm: We'll try to see if we can move on and talk also a little bit about what we think is included in this syndrome. What could we have there? John had four entities. Do we all agree on that or not? Can we agree on a few things? I think you'd like to start here.

B Smith: We can take something from the definition of the acute respiratory distress syndrome, that they have an acute onset, non-cardiogenic. They have three categories based on oxygen, inspired oxygen to arterial oxygen content. Probably those are not applicable here, but it is an acute onset. It has to be post-diving or dive training activity. Some sort of severity gauge based on physiologic function, pulse oximetry maybe at rest and after a specified period of exercise to have an easily classified level of severity independent of symptom.

P Lindholm: Is there a difference between the larger airways and the more peripheral capillary? Do we know? Do we have data on that?

F Tillmans: At this point I want to say we don't have data on that really. It's also something that what we want to do is try to get something that is distinguishable on site at a competition. I'm not sure if we need a bunch of measurement techniques that are not on site that we're going to get anywhere with these different classifications. We need to try to keep this more practical.

R Moon: I totally agree with you, Frauke. The difference between what we're talking about in terms of where in the lung and what the pathophysiology is different from altitude illness where you can tell right away what the person has, nausea, vomiting, pulmonary edema, or he's got cerebral edema. We're not ready yet to distinguish different subtypes of breathhold diving induced pulmonary injury because we just don't know. We don't have the tools yet to distinguish them. To come up with a term that is an acute onset event that occurs during breathhold diving is probably good enough at this point with hypoxemia, coughing up blood, whatever symptom you want to put in there. We don't want to get down into the weeds yet because we don't have any way of telling, at least on site, as you pointed out, Frauke, what's going on.

P Lindholm: I agree with that. We had 30 minutes, if we don't agree, we're not going to agree on more. There seems to be a need for a term for a syndrome that we also agree that we don't know. We have sort of a framework for future research work. We can suggest this term as part of the workshop findings. Maybe some people are going to call it squeeze, we'll see if it works. You come up with a term and if it has a functionality, it's going to catch on and it's going to be used.

P Germonpré: I noticed, Richard, that you avoided the term respiratory distress. You said acute onset, and then you said pulmonary whatever. Does anybody object to having respiratory distress there, even if it's just coughing up some blood? It's a respiratory event, and it's something distressing. I would define further on to Brad, acute onset respiratory distress induced by diving or freediving or breathhold diving activity.

A Luks: I would not include the term distress. People with ARDS are truly in respiratory distress. And most of these people if not intubated or get some high level therapy very quickly, they'll meet their demise. As opposed to a lot of people coming out of the water who are symptomatic but not in

distress. While it may be distressing to them, they're not in a similar situation. Building on the term I was proposing before, you could just say 'acute freediving pulmonary illness.'

P Lindholm: Freediving acute respiratory trauma syndrome. I'm not so sure, acronyms are always going to be a problem.

M Lang: Acute freediving illness. But then we're going to call it breathhold. Either you have compressed air on your back that's supplied by rebreather, scuba cylinder, or umbilical from the surface, or you don't have compressed air on your back. What's wrong with the term freediving?

P Lindholm: Isn't freediving an American term used mostly when we talk about breathhold diving in the rest of the world?

Comment: If you breathe underwater, you're not freediving. It's breathhold.

M Lang: I'm saying that's a distinction. Either you have compressed gas on your back or you don't.

P Lindholm: If you're not in the diving community, if you're a medical doctor and you read breathhold something, you understand that they're breathholding. Freediving, is that somebody jumping off a cliff or you free swim, you're not tendered by a surface supplied gas?

Comment: I think freediving is the name of the sport and the name of the practice now. You can go and say football. You do not play with your foot. People call it football in the United States.

J Valdivia: Every freediver in the world knows the word "freediving."

O Christen: Back to the education part. Freediving is one word, not two, and how we use it in every agency internationally. There's also the Spanish version, apnea is used in the Spanish-speaking part of the world. The Germans have Apnoe Tauchen (Freitauchen), two names for the same group of sports and that's it.

M Lang: We're trying to get freedivers to recognize Divers Alert Network as a repository for incidents and such. Won't they recognize 'freediving' better than 'breathhold diving' when we're trying to collect this data? Yes.

P Lindholm: It seems like freediving is a better word for us for what we can come up with here. It's good that we had this round of discussion to see where we stand. We'll leave it at that for now and see if it sticks or not. We'll call it FIPS instead of squeeze for the moment to be a little bit more medically or scientifically open to that it's not a squeeze. Some of it might be, but we don't know that. FIPS is a syndrome of pulmonary symptoms that could include, but not limited to, edema, barotrauma of descent, barotrauma of ascent, aspiration, or dyspnea out of proportion.

PROCEDURES ON LUNG SQUEEZE IN FREEDIVING COMPETITIONS: TREATMENT AND RETURN TO DIVING

Moderators: ***Oliver Christen-Drew, Michael Lang***
Panelists: ***Alexey Molchanov, Kirk Krack, Vitomir Maričić, Juan Valdivia***

O Christen: My first question to you guys is, why are there no cases of severe squeezes in female freedivers? It's always the guys who have these accidents.

A Molchanov: There are some but fewer cases. Maybe Vito has some examples. It also could be because of the development of their chest and muscles and just the effort we can produce. I'm trying to remember when the depth was 100 m and the men were already diving deeper. Monofin is much shallower, with fins, it's less. The difference between men and women is monofin. It's 136 to 130 something. It could be, if it's contributor to their squeeze, tension.

K Krack: Anatomically, women tend to be a lot more flexible than men in general. Men tend to throw caution to the wind a lot more than women. Women will be more conscious in their approach to their diving and have a higher self-preservation component than a man. We'll just go for it, squeeze be damned. A woman will tend to be more self-protective and family-protective, than a guy will.

V Maričić: There are just fewer women in the percentages so less cases by the raw numbers. But there are severe cases with girls as well when they push hard enough. There's maybe a little bit less competitiveness and a little bit different approach to the whole freediving sport in general. In other sports there are also less severe injuries.

J Valdivia: I don't have experience with females, but there is a difference in gender. In the blackout statistics published two years ago the highest risk discipline for blackout was for males.

C Seiler: Now that we agreed on FIPS you still say squeeze, we should test the new term. Does it work?

O Christen: Let's run with FIPS.

A Molchanov: I want to add to the topic of how fast lungs can heal. I have a good example from my accident in 2013. I was diving daily to see if I'm recovered enough. It was six or seven days before the finals of the world championships. I participated in an attempt to do the world record. Then I got reverse block at the bottom and I lost the rotation. I was dizzy and came back from 108 m. I was not blacked out yet, but I was spinning and not swimming up in a straight line. Because of all this, abrupt and little movements on the camera I could see that I go up, 128 to 115 m. Then I went 90 degrees off the line, stopped at the lanyard, went off the line a bit, stopped. I could see this at least three times from the bottom camera at 115, 110, 100 m. Then I got a very strong squeeze, likely the strongest squeeze in my career and I was bleeding until the evening for many hours. Finals were in a week. This was the year before everybody was so careful with FIPS. There were not any restrictive measures yet to stop freedivers from doing this and it was actually fine. Everybody was going around and spitting blood as part of the routine and nobody was really worried about it. Could I get ready for these finals in one week? For me the decision was that I would compete and try to win anyway the next day. I could feel the soreness in my lungs. That morning I did light breathing exercises and a bit of diaphragm stretch. By then it was lessened. On the third day, the second day after the accident, I tried a 10-m, 15-m, and 20-m dive. I stopped at 25 to 30 m. That was the deepest dive of the day and I felt pain. I felt I gave enough information to my body to sort of give it enough impulse to promote recovery. Then I left for home, rested, did breathing exercises again. When I returned the next day I was able to progress to 45 to 50 m, and I felt better than on the previous day and also rested again. Lots of fluids, lots of food, almost a full recovery, and then breathing exercises. On day number 5, I did 70 m, 75 m, and I felt completely fine and fully recovered. Day number 6 was rest day and day number 7 was finals when I was able to win world championships. I had no squeeze on this day seven days later.

P Lindholm: I have a slightly separate question about adaptation. In scuba diving in the northern countries people drown and have accidents in the spring because you haven't dived for six months. Most of the world championships in freediving are in the fall or the late summer because a lot of the activity

comes from Europe and it's much nicer diving in Europe in the summer. Many of the Swedish divers reported squeezes in the spring when they were not adapted. There has been some speculation or questions that if you start diving deep after not having done it for a while, is there deconditioning? Is there a conditioning phase? Can you feel that if you haven't dived for one week, a month, six months? Can you train your lungs to tolerate the depth, or train something in the pulmonary system beyond just getting your flexibility in the ribcage up? Is there some depth adaptation?

V Maričić: I was thinking about that a lot actually. Adaptation period is a very common term that's used in freediving. I was testing a lot with myself and others but cannot provide factual evidence on that. I concluded that most of this adaptation is mental to not be stressed and also neurological learning related, not necessarily to physiologically change the tissue, but to learn how to coordinate and relax on this micro level, which is not sometimes consciously available. With exposure and time our bodies learn how to go deeper in this micro-coordinated way to collapse, reopen the lungs, and so on. Maybe it had something to do with secretion of surfactant? From my experience, I don't see how I could have made such small changes where I completely changed the way I squeeze in such a short period. I see it with others and I see it between how under narcosis you are and the type of narcosis symptoms you have. For me it literally changed in a week and just changed my perception to narcosis. My squeezing since was far less, more comfortable and far better. Petar, who I'm training with and I have a very similar story. The difference between that was six days for his biggest squeeze. He was bleeding the next day filling up a cup with a mushy mix of saliva and not just blood. We agreed on 24 hours full rest and CPAP. He then went to a medical assessment. Two days on everything was fine. He then did a CPAP to see how it feels. From day five you feel amazing and great and on day six, did a 40-m dive with no squeeze. I always try to put 24 to 48 hours, in between. But there was this one competition, world championships, the pre was Friday. The next day I was supposed to dive deeper and I was late for my official time. The transfer boat was late. I had literally a minute to jump in the water. I really squeezed a lot. I remember lying down on the bed and filling up a cup with blood that afternoon. The next day I woke up and stretched a little bit and felt okay. If there's no reason not to go I did a deeper dive with no squeeze, one of the best dives ever. It managed to heal in less than 24 hours. The crucial point was on the day of the dive when I woke up, if I didn't feel amazing I was not going to do the dive.

O Christen: It's interesting to hear your perspective on Petar's recover from such a FIPS event. I saw this from a distance through social media. This situation caused a worldwide uproar and was the biggest scandal in freediving that year. How was it possible that AIDA as the organizing agency allowed a guy that nearly died on a dive to dive again three days later.

V Maričić: That was political and personal interest and they were referring to a dive on the pre-comp this year, which wasn't even severe yet overly exaggerated and full of lies. Those reports are just in support of someone's personal interest and we can't take them seriously. In fact they actually went beyond what is a normal procedure by testing. They tested us five times for doping and they checked us five or six times medically, independently, outside of AIDA and inside AIDA by different physicians on the same competition. They wanted to protect themselves with our approval, of course, to make sure that everything is absolutely perfectly fine. But with social media, people will keep pushing and saying whatever they want. This is another reason why we need objective rules from a third party so that these cannot really happen.

K Krack: When we look at the competition it's the practitioners and their overall experience of how quickly they can recover and get into it versus the public's perception of what they see. We come to the surface, the person blacks out and right away we're declared dead. Or we come up and are spitting lots of blood and we're going to die. Now we have The Deepest Breath on Netflix. None of that is in support of a positive image towards freediving. The practitioners who know what they can do take their own personal risks versus the larger organizing bodies that are trying to protect the sport for the whole, not just the one or two people who want to do a dive. What are the competition potential protocols and procedures we would follow versus the recreational person? In the competition you have highly experienced athletes and a doctor. You could have a complicated point

score system and all the testing and equipment you want versus the recreational freediver who's taken a basic course, doesn't have that level of knowledge, is out there just with their self and their buddy and needs actually very simple, easy to remember steps from their course training. It's okay to say in competition we can probably dive quicker and we can have a more complicated scoring system and testing, but at the recreational level it kind of has to be really dumbed down for the public and has to be easy to remember. It has to default to the safer side of things because they don't have primaries and secondaries and counterbalances and all the oxygen and de-fibs and everything that you need.

P Germonpré: That was exactly my question. Your experience and your personal feeling and how quickly you can recover is extremely interesting because it teaches us something on what the possibilities of recovery are in persons that can evaluate themselves and their own condition in a reliable and as objective way as possible. When does a freediver actually get to that point where he can evaluate himself? The recreational freediver can't evaluate his or her own condition the same way that you guys can. There's a complete difference between how you put out the rules and how you put the evaluation out.

K Krack: From my personal experience and the people I've worked with in the past, it's one thing to be an adapted athlete to be able to decide the problem resolved, I know what I did wrong I can go into this dive, I'm more relaxed, it's a different day. The recreational freediver who isn't adapted is still dealing with very simple technique errors that they're doing that complicates things. It's that recreational person who's just come out of a course, playing in that 20 to 30 m zone who gets to that point where they could start to get into a FIPS situation. They're still complicated by poor techniques that are going to contribute to those problems. I go back to this idea that we should default to simpler and longer recovery times versus adapted athletes who can very quickly analyze their situation, correct problems, and go into the next case a lot more comfortably.

A Luks: This has been a very interesting discussion for me to listen to because I'm a complete outsider when it comes to the world of not only scuba diving but also freediving. This discussion nicely encapsulates why groups like DAN and others need to get out there and get the research done and get guidelines out there. What you have right now is eminence-based, not evidence-based medicine. The challenge is while you guys being world champion freedivers spend a lot of time thinking what works for you, the general public who's doing this, whether it's in competition or on a recreational basis, are going to put tons of faith in the world champion's personal experience with this, which does not necessarily apply on a much broader scale. Until you get good evidence-based recommendations out there from societies that have a lot of respect, that's going to predominate. Then the risk is you have people adopting your personal practices, which may not be suitable for them at all because they don't have your physiology, your skill, your experience. That's the challenge where the field stands now. The way you try to sort things out and listen to your body and figure out what works, that's great. That does not apply to the person who's in their third month of freediving to start doing all this stuff.

K Krack: I think that's also where I think that AIDA and CMAS being competitive, working with select individuals, high-level people with a lot of support is one component versus a recreational training agency, which is another component. Where we're defaulting, we need to default to the limited liability model. What I want to default to is what does DAN say. What has DAN done in the research, how do they default to the conservative aspect of this? Then we can say we follow what DAN has recommended much like we've done in flying after diving recommendations in scuba diving. We just went to the professional medical researchers to say, what is our recommendation? CMAS and competition is one thing, versus that recreational training agency who needs to default to the general public. The sooner DAN figures it out, the sooner we live up to adopt it.

A Molchanov: I would support it and agree with Kirk.

S Hopkins: I agree for the general public you need simple, easy to follow guidelines. But people kill themselves all the time doing stupid things in sport. One guy tied 17 helium balloons to a lawn chair and flew over LAX. People rappel off their ropes, crash their mountain bikes. I've worked a lot over the years with elite athletes and am a bit concerned with the idea that the elite athlete is the person

who should decide the risk they take for competition. You guys have an inherent conflict of interest. There was a study a few years ago related to doping where elite athletes were asked the question, if you could take a performance-enhancing drug that guaranteed a gold medal and you had a 50/50 chance of being dead in three years, would you do it? Half of them said yes. I would really encourage you for different reasons to develop a series of objective measurements that are administered by people external to you as athletes that are guidelines for returning to competition rather than just 'feeling okay.' We have an obligation not to kill our elite athletes by allowing them to push themselves to the point that they die.

V Maričić: I agree. I've been injured in my career in other sports multiple times and so many times I've been disappointed with the medicine. I would come there and they would just say, don't do that. What do you mean don't do that? It's part of my athletic performance. I'm still going to do it regardless of the pain. You are there to help me. But they're also there to protect themselves. If you're going to do that, then it's going to hurt you. My job would be much easier if you just quit sports. On occasion, they sent me to talk with other people and didn't want to fix my injury. There is a little bit of conflict of interest there as well. That's why I think it should be something that people can agree on that is objective and not that, like DAN for example, is just trying to protect themselves. Well, if you have a squeeze, just don't dive for a year, which is overly conservative. Athletes reply if you have a squeeze in the morning, why wouldn't you dive in the evening? It also doesn't make sense. It should be something that's objective and resistant to bias. Then athletes are going to say, there's been some thought put into it, let's respect it.

O Christen: It's the same eight years ago when I took over the AIDA education part. What was in there was more or less world champions putting something together and teaching it to beginners. There were breathe-ups and packing and everything was there jumbled into various levels. There was no system to it. That's the same job that we have now on that topic. That's where we learn from world champions and very few specialists on the medical side and have to break that down to the appropriate level. That's going to hopefully not take 15 years as we just discussed regarding the concussion protocol, but that's the job that we're facing.

P Lindholm: If you squeeze above 40 m, it's bad technique. Basically, take a course. If it's a squeeze in a recreational situation, FIPS, then you need to probably work on the technique. The competitive situation is different. If we have a case and somebody comes with signs of something it depends on how deep they actually were. If it's one of you guys on a 110-m dive, it's different than if it were a 10-m dive. We have somewhere along the lines of where the recreational freediving courses are, 30, 40, 50 m. It coexists a bit with the residual volume feeling where you start getting fairly good lung collapse. That's where you get that difference. As Kirk said, you need the rules that are good for the uneducated or the masses. Then what we also hope to achieve with this is maybe a starting point for things like the DAN hotline. We can't solve all the things that exist with the competitive organizations. Maybe we should make a distinction between the recreational and competitive freediving worlds.

F Silva: That's pretty much what I was going to say. Nothing prevents two different recommendations, one is the recreational public with DAN, and then the competition side with CMAS or AIDA or whatever the organization is. When we're dealing with the high-performance athletes in that situation we can provide a different recommendation. For everyone else, DAN should have probably the wider net and safer recommendations.

R Moon: You brought up the Netflix documentary, The Deepest Breath, where there was a death due to a mismatch in timing and location between the breathhold rescue diver and the breathhold woman who was trying to do something unusual. In the movie the issue of having scuba rescue divers was brought up and it was dismissed as being impossible or not safe because of the risk of depth-time exposure, which is not correct. Why is it that in these competitions there aren't scuba rescue divers who can hang out for much longer periods of time at reasonable depths and prevent that kind of death?

Comment: There used to be. Why are there not anymore?

K Krack: The freediving competition that I run is by AIDA and is the only competition that's allowed to employ scuba divers. From the freediving point of view, being non-educated in scuba, let alone technical diving, it's more dangerous for the technical divers than it is for the freedivers down there because they don't understand technical diving. My argument as a 170 m mixed gas technical diver and an accomplished freediver is that's misinformed, they do have the ability to operate safely. Do we need scuba divers down to 40 m? No. Do we need scuba divers to 60 m? No. What we're trying to accomplish is eyes on and to know with immediacy that we have a situation. Back in the day if Alexey is diving to 120 m, there was a zone before we had video where there's no scuba diver down there. It's a zone where he's sinking, he's out of sight, he's gone. The freedivers are going to come down. They are going to know a time where they will meet him or have eyes on, but that could be after 2.5 minutes that could go by and we don't realize that he's still blacked out on the bottom. Now we have to bring him to the surface at 1 m/min at the slowest, 1.5 m/min the fastest. Now you're talking about a delay from when potential blackout at bottom occurs to the time you're going to actually be able to close the airway. That's roughly 2 minutes, which is potentially in the anoxic zone where lungs are going to flood. That's been my rationale to it. What I've always strived to is past certain depths where we couldn't communicate to the surface, that the counterbalance should be enacted so that we can recover within that 2-min potential anoxic zone, terminal gas zone. That is what we should have eyes on. What we do have nowadays is a diver so we can see the athlete the whole way down. We can determine that they're out of timing, they're hooked up on the plate, whatever it is. We can then from the surface start bringing them up. We still don't collect the airway or protect it until they reach a diver, but with immediacy we've been able to get them on their way. We're hopefully still within that 2-minute zone where protective reflexes like laryngospasm is still hopefully doing the job. Is that the reason why because it was more expensive, and they were harder to find? You have freedivers not in the community of divers wondering how do I find scuba divers. Then I have to pay for their gases and it's more expense. I don't understand why it's more dangerous to have divers down there, when honestly it isn't.

M Lang: The issue is the long 6-7 hours of deco time for the safety divers at depth and there is more than one competitor during the competition.

K Krack: When I run Deja Blue, I don't put divers all the way to depth, only where they can still see the person at the plate, where they can do a sound signal that can be heard on the surface. I've had divers 136 m and no limits 20 years ago being able to signal that they knew something was happening. I've run Deja Blue with 6 to 8 rebreather divers who fly themselves down, buy their own hotels. All I do is give them scrubber and gas. They're looking for good reasons to dive. Whatever puts mask, fins and snorkel on a person under the water, we're all part of the diving community, freediving, scuba diving, technical diving.

V Maričić: I think it's possible, but I don't think it's completely necessary. In the Netflix, we cannot see that as a freediving situation. That's completely unrelated to any competition. It's something like all of us take cars and go on the highway and say, let's drive really fast and see who is the fastest without safety, without proper protection. The dive plan, the way it was executed, it was a fun dive, a snorkeling adventure. The safety plan on that fun day, even though it involved elite athletes, was bad and poorly executed. Of course, these things happen all the time. People die in the water spearfishing but we cannot connect this with freediving. It's literally like the four of us go there and freedive for fun. Everyone can and does make mistakes.

K Krack: Adam from Australia went down to 130 m, suffered narcosis, sat on the plate for 40 seconds. He was beyond depth sounder range. It was not in competition.

V Maričić: They should have sonar.

K Krack: The sonar at the time did not reach that deep, it was only good to 90 m.

WORKSHOP SUMMARY AND CONCLUSIONS

Peter Lindholm, Michael Lang, Frauke Tillmans

What to do with a diver reporting a FIPS? Can we give some general medical guidance on treatment and/or return to dive? There was a general discussion on how to handle various cases but with no conclusion. The general consensus was that there is not enough data available to create standards of medical advice or treatment recommendations. The problem remains on what to do and when to return to diving from both the medical and legal aspects. There are also variations among healthcare systems depending on location so in any acute situation a local practitioner is needed.

There was some agreement that advice by telephone would not be sufficient although contact with DAN could help assisting a local physician. DAN can't assume the responsibility of the medical care of someone on site but can give advice and evaluate the urgency of a situation to reach medical care.

This field could benefit from more data on various pathologies and questions like site of bleeding, risk of recurrence? Added risk of loss of consciousness with edema etc. There is a need to collect data on the natural history of this from divers and swimmers suffering FIPS, or SIPE, did they go back in the water, recurrence? And what guidelines did they follow if such exists in their training or competitive organization.

Scoring systems, mild moderate severe, points for various pathologies were suggested but also need more data to be meaningful as well as validation. There was also consensus on the fact that guidance may differ between the recreational freediver and a competitive diver on the day of competition.

We also agreed on trying a new terminology FIPS Freediving Induced Pulmonary Syndrome as an overarching heading, and then we collect data to better define the subcategories of that like immersion pulmonary edema, aspiration, and barotrauma of descent, etc.

We could not agree on a more granular terminology, instead we agreed that it would be advisable to use freediving induced pulmonary syndrome (FIPS) as terminology for a freediver that surfaces with pulmonary symptoms. FIPS possibly including components of SIPE/IPE, Barotrauma (ascent or descent), aspiration and other still unresolved pathophysiological mechanisms.

On recommendations to return to diving we would leave that up to the local physician who oversees an event/competition. More data is needed to make any evidence-based conclusion in regard to this issue.

APPENDIX A - List of Acronyms and Unit Conversions

AIDA	Association for the International Development of Apnea
ARDS	Adult respiratory distress syndrome
ATA	Atmosphere absolute
BH	Breathhold
CMAS	Confédération Mondiale d'Activités Subaquatiques
CNS	Central Nervous System
CO_2	Carbon dioxide
CPAP	Continuous Positive Airway Pressure
CT	Computed tomography
DAN	Divers Alert Network
DCI	Decompression Illness
DCS	Decompression Sickness
ECG	Electrocardiogram
EEG	Electroencephalogram
ENT	Ears, Nose and Throat
ETBV	Extrathoracic blood volume
FIPS	Freediving Induced Pulmonary Syndrome
FRC	Functional residual capacity
GE	Glossopharyngeal exsufflation
GI	Glossopharyngeal insufflation (or lung packing)
HAPE	High Altitude Pulmonary Edema
Hb	Hemoglobin
Hct	Hematocrit
ITBV	Intrathoracic blood volume
LMC	Loss of Motor Control
LOC	Loss of Consciousness
MRI	Magnetic Resonance Imaging
N_2	Nitrogen gas
O_2	Oxygen gas
P_aO_2	Partial pressure of arterial oxygen
P_AO_2	Partial pressure of alveolar oxygen
PBT	Pulmonary barotrauma
PCO_2	Partial pressure of carbon dioxide
PET	Positron Emission Tomography
PFI	Performance Freediving International
PN_2	Partial pressure of nitrogen
PO_2	Partial pressure of oxygen
RER	Respiratory Exchange Ratio
RV	Residual Volume
SEAL	Sea, Air and Land (U.S. military special forces personnel)
SIPE	Swimming-Induced Pulmonary Edema
S_aO_2	Arterial oxygen saturation
S_pO_2	Arterial oxygen saturation (measured by pulse oximetry)
SWB	Shallow water blackout

Unit Conversions

1.0 m = 3.28084 ft	1.0 mm Hg = 0.13332 kPa	1.0 kg = 2.204623 lb	°C = (°F - 32) / 1.8
1.0 ft = 0.30480 m	1.0 kPa = 7.50064 mm Hg	1.0 lb = 0.4535924 kg	°F = °C * 1.8 + 32

APPENDIX B - Workshop Participants

Igor Barković - The Clinical Hospital Center, Rijeka, Croatia
Gilbert E. Boswell - San Diego Naval Medical Center, USA
Oliver Christen-Drew – SME Freediving, Washington DC, USA
John R. Fitz-Clarke – Dalhousie University, Canada
Peter Germonpré - Military Hyperbaric Center Brussels, Belgium
Ian Grover – UC San Diego, USA
Maria Hårdstedt – Vansbro, Sweden
Susan R. Hopkins – UC San Diego, USA
Kirk Krack – Performance Freediving International, Canada
Michael A. Lang – UC San Diego, USA
Peter Lindholm – UC San Diego, USA
Andrew M. Luks – University of Washington, USA
Anna Lussier – UC San Diego, USA
Vitomir Maričić – AIDA, Croatia
Alexey Molchanov – AIDA, Russia
Matías Nochetto – Divers Alert Network, USA
Richard E. Moon – Duke University, USA
Alex Patrician – University of British Columbia Okanagan, Canada
Timothy Patron – Divers Alert Network, USA
Benjamin A. Sebreros – US Navy Special Warfare Center, USA
Claudia Seiler – Örebro University, Sweden
Fernando Silva – Kaiser Permanente, USA
Bradford J. Smith – University of Colorado Anschutz, USA
Frauke Tillmans – Divers Alert Network, USA
Juan M. Valdivia – Baycare Medical Group Tampa, USA
Karen B. Van Hoesen – UC San Diego, USA
Elaine D. Yu – UC San Diego, USA

APPENDIX C – Workshop Agenda

BAROTRAUMA AND SIPE IN FREEDIVING

FRIDAY, 27 October 2023

8:00 Registration opens/Coffee
8:45 Welcoming Remarks:
Dr. Michael A. Lang, Dr. Peter Lindholm, Dr. Frauke Tillmans

SESSION 1 Chair: **Dr. Michael Lang**
9:00 **Dr. Michael Lang**, UCSD, Introduction: San Diego Center of Excellence in Diving
9:15 **Dr. Peter Lindholm**, UCSD, Workshop Introduction, Questions, and Goals
9:30 **Oliver Christen-Drew -** Lung Squeeze and DCI in Freediving Education
10:00 **Dr. Matías Nochetto -** A review of DAN calls involving freedivers
10:15 **Dr. Elaine Yu** - Current medical procedures at competitions
10:30 Coffee Break

SESSION 2 Chair: **Dr. Peter Lindholm**
10:45 **Dr. Richard Moon** Swimming-Induced Pulmonary Edema: What do We Know About Pathophysiology, Prevention and Treatment?
11:15 **Dr. Susan Hopkins -** Pulmonary capillary stress failure: from the thoroughbred racetrack to the summit of Everest?
11:45 **Dr. Benjamin Sebreros** - Is infection a risk factor for SIPE?
12:15 Lunch Break

SESSION 3 Chair: **Dr. Frauke Tillmans**
1:00 **Dr. Frauke Tillmans,** Divers Alert Network, *Freediving survey*
1:15 **Vitomir Maričić** - Lung squeeze - key factor in deep diving
1:45 **Dr. Peter Lindholm** - Is there an inflammatory component in SIPE and Squeeze.
2:00 **Dr. Bradford Smith** - The Biomechanics of SIPE and Squeeze: Lessons Learned from Acute and Ventilator-Induced Lung Injury
2:30 Coffee Break

SESSION 4 Chair: **Dr. Peter Lindholm**
2:45 **Dr. Maria Hårdstedt** - SIPE in recreational swimmers – potential mechanisms for sex and age difference in prevalence
3:15 **Dr. Gilbert Boswell** - SIPE: Is it more than just edema? CXR patterns and possible mechanisms.
3:45 **Dr**. **Andrew Luks** - HAPE: Pathophysiology and Clinical Course
4:30 Discussion Day 1
Moderators: **Dr. Frauke Tillmans and Dr. Peter Lindholm**
- Questions from the floor
- Challenges in data collection (accident and incident reporting – DAN, training agencies, freediver community)

5:15 Workshop Day 1 close

5:30 Reception at Fathom Bistro, Shelter Island

7:00 Dinner at Miguel's Mexican Restaurant

SATURDAY, 28 October 2023

8:00 Registration opens/Coffee
8:45 Day 1 recap: **Dr. Peter Lindholm and Dr. Frauke Tillmans**

SESSION 5 Chair: **Dr. Peter Lindholm**
9:00 **Dr. Peter Germonpré** - PFO and freediving
9:30 **Dr. Igor Barkovic** Hemoptysis as a symptom in squeeze; where does it come from, why and when?
10:00 **Dr. Claudia Seiler** - Diagnosis and treatment of SIPE in recreational swimmers – the use of lung ultrasound and prehospital CPAP
10:30 Coffee Break

SESSION 6 Chair: **Dr. Frauke Tillmans**
10:45 **Dr. John Fitz-Clarke -** Biophysical View of Lung Injury in Breath-Hold Diving
11:15 **Dr. Fernando Silva -** Lung damage in squeeze: is it broken or is it leaking?
11:45 **Dr. Alex Patrician** - Are there long-term consequences of breath-hold diving and lung squeeze?
12:15 Lunch Break

SESSION 7 Chair: **Dr. Michael Lang**
1:00 **Dr. Juan Valdivia** - Current procedures for lung squeeze: AIDA/USFF: the good, the bad and the ugly.
1:30 PANEL Discussion: Current procedures on lung squeeze in freediving competitions and training, Panel Moderators: **Dr. Michael Lang & Oliver Christen-Drew**
- **Alexey Molchanov**
- **Kirk Krack**
- **Dr. Juan Valdivia**

2:30 Coffee Break

SESSION 8 Panel Discussions:
3:00 Terminology medical/physiology: Moderator: **Dr. Lindholm**
3:45 Treatment (cpap, oxygen, drugs, dangers? Moderator: **Dr. Germonpré**
4.15 Recommendation on return to dive or return to swim. Moderator: **Dr. Nochetto**
4:45 Conclusions **Dr. Lindholm and Dr. Tillmans**
- Lessons learned & future initiatives.

5:30 Workshop close **Dr. Michael Lang**