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Föreningen verkar för att öka kunskapen inom de flyg- och dykmedicinska områdena och samtidigt överföra kunnandet till det kliniska arbetsgolvet.

Föreningen, som utgör en sektion i Svenska läkaresällskapet, har ett årsmöte på våren till vilket vi alltid har en inbjuden föreläsare som talar över ett aktuellt ämne. Likaledes håller föreningen en utbildningskväll dagen före Transportstyrelsens utbildningsdag och ett vetenskapligt möte Scientia et Valebat (Vetenskap och äventyr) på hösten. Programmet brukar vara balanserat mellan flyg- och dykmiljön och vår uppfattning är att flyg- och dykmedicin har flera gemensamma beröringspunkter och att samarbetet mellan dessa miljöer tillför synergieffekter för båda. På vår hemsida www.sanma.se redovisas vårt arbete för att sprida information i ämnena flyg och dykerimedicin.

Scientia et Valebat 2017 kommer handla om Immersionslungödem/SIPE

SIPE är ett tillstånd som tidigare ofta tolkats som lungbristning hos dykare och drunkningstillbud eller astma hos triathlonsimmare. Den engelska termen är ”swimming induced pulmonary edema”- SIPE. Genom en donation har Svensk Flyg- och Dykmedicinsk Förening fått en unik möjlighet att bjuda in de internationella auktoriteterna Prof. Peter Wilmshurst, England och Prof. Richard Moon, USA.

Från Sverige kommer bland andra sjukvårdsgruppen bakom Vansbrosimningen: dr Annika Braman-Eriksson och medarbetare som nyligen publicerat en omfattande artikel om SIPE i Läkartidningen 2017;114. Konferensen riktar sig till läkare såväl som instruktörer inom dykning och vattensport.

Tid och plats: torsdag 7/12-17 kl 13-17, ABF-huset. Sveavägen 41, Stockholm.

Anmälan görs via www.sanma.se

Scientia et Valebat with focus on SIPE

Time. December 7th , 1300 – 1700
Location: ABF-huset, Sveavägen 41, Stockholm

1300 Welcome	Chairman SANMA Håkan Sköldefors
1305 Markers of decompression stress during simulated high Altitude flying (No abstract)	Rickard Ånell
1320 No title, no abstract	-
1335 Why a conference on SIPE now?	Hans Örnhagen
1345 Early reports on immersion pulmonary edema in diving	Peter Wilmshurst
1425 SIPE in swimming competition in cold water	Annika Braman-Eriksson/ Maria Hårdstedt
1455 Infiniti Medical presentation	Johan Vobern
1505 Coffee/tea/fruit	Host Infiniti Medical
1525 Personal experience of SIPE	Peter Geiger
1540 Resent research on SIPE	Richard Moon
1620 – 1700 Questions to a panel	

Questions to a panel of speakers:
Is there any clinical method to identify persons prone to develop SIPE?
What are the signs/symptoms that the diver/swimmer should pay attention to?
What is the optimal treatment on site?
Is there an increased risk of SIPE if you have had one incident?

Why a conference on SIPE now?

Hans Örnhagen, MD, PhD, Consulting physician at the Swedish Sports Diving Federation.

On November 7th a Swedish couple, Ulf Pettersson and Kim Linh Pettersson, with an experience of more than 400 dives, made a dive in Thailand. This was the first dive the second day on a live aboard tour. The previous day, with two dives, had been uneventful, but Linh had aborted a dive some days earlier because of breathing problems, which resolved quickly after reaching surface. After about 8 - 10 minutes into the present dive at about 24 m Lin indicated a problem with the mouthpiece/breathing equipment and the dive guide brought the group towards the surface. At 5 m Linh got a question regarding her well-being and signaled that she still had problems with the breathing. Only seconds later pinkish foam came out of Linh´s mouth and she lost her conscience. At surface more foam came from Linh´s mouth and she was still unconscious. Five minutes later, on board the rescue boat, Linh was given mouth to mouth resuscitation. CPR was continued during transport by the coastguard and was not stopped, and Lin declared dead, until the hospital was reached 4 h later.

The devastated husband was told, by the hospital doctor, that the cause of death was decompression illness, which was difficult to understand for a diver with long experience. In the current situation, however, the diagnose was not objected to.

When back in Sweden, Ulf tried to find answers to what could have caused Linh respiratory problems at depth after only 10 minutes dive. Ulf did not find any explanation or answers in the scuba literature and he called me. My interpretation was that it was immersion pulmonary edema that had caused the tragic death, a condition that Ulf had never heard about. Ulf´s immediate reaction was that this condition should be better explained to students in diving courses, but maybe also to instructors and even diving doctors, based on Ulf´s experience from Thailand. This led to the decision to donate money in the name of his wife Linh to spread the information on SIPE to reduce the number of fatalities in the future.

The Swedish Aviation and Naval Medical Association, SANMA, received the donation with a promise to arrange the workshop/conference that we now attend. During the spring 2017 our attention was drawn to the fact that it was not only diving but also other watersports that had IPE cases. Triathlon and “Island to island runs” being among them. We will today hear a personal witness from an anesthesiologist who is also a triathlon runner. In the June issue of Läkartidningen, Dr Annika Braman Eriksson and colleagues presented medical experiences from a famous long distance cold water swim. Today Dr Braman is here to give us further details. In addition to this we, in the planning group, have done our best to meet the wish of the donor to invite international specialists through the invitation of Professor Richard Moon USA and doctor Peter Wilmshurst UK. Though Dr Wilmshurst I got my first knowledge of clinical problems with IPE in diving, and Prof Moon is today leading a group of scientists working on IPE. With this list of speakers and a panel that can answer our questions, I am convinced that we today will get a better understanding of SIPE so we then can help others to avoid SIPE and/or at least quickly abort a water activity when struck by sudden dyspnea in water.

Early reports of immersion pulmonary oedema

Peter Wilmshurst, MB, ChB, Consultant Cardiologist, Royal Stoke University Hospital, UK

I first encountered immersion pulmonary oedema (IPO) in April 1977.(1) My club was diving out of Plymouth and I was on a boat when two club members surfaced in distress. One was dyspnoeic, cyanosed and expectorating bloodstained froth. She said that she had not inhaled water and that she had had six previous episodes. I examined her and I took her to hospital where my diagnosis of acute pulmonary oedema was confirmed. In the next three years I saw two male members of my club (approximately 200 members) suffer acute pulmonary oedema when diving. None had any detectable cardiac abnormality. They were the subjects of our first report of IPO, which provided preliminary evidence of abnormal vascular reactivity in affected divers.(2) In 1984, at Underwater Physiology VIII, we reported that IPO might be quite common and at the BSAC Diving Officers' Conference, I presented evidence that IPO can be fatal and is mistaken for drowning.(1,3) Our Lancet paper in 1989 reported that IPO also affected surface swimmers.(4) Compared with controls 11 affected individuals had reproducibly exaggerated systemic vasoconstrictor responses to physical stimuli such as cold-pressor test. During the test (out of water) 9 of 11 developed signs of cardiac decompensation and one had frank pulmonary oedema requiring treatment with vasodilator infusion. Most developed hypertension during 8 years follow up. (The Lancet required that we shorten our paper.) We showed that vasodilator drugs, particularly oral nifedipine counteracted the pathological vasoconstriction induced by cold and the cardiac decompensation. In 1998 I reported our observational experience that nifedipine protects against recurrence of IPO.(5) In the absence of other data, for more than 30 years, I have recommended use of nifedipine by divers who had IPO but will not stop diving. The original case subsequently had high altitude pulmonary oedema, as have others.(6) I reported that some people had IPO and also had pulmonary oedema in other situations on land (emotional stress, sexual intercourse and exertion in a very cold environment).(6) Clearly some people who had no detectable heart disease were predisposed to IPO and pulmonary oedema in other situations. It appeared that the combination of increased filling pressures from immersion and vasoconstriction can produce IPO. Weiler-Ravell and colleagues reported that IPO occurred in eight fit military recruits during a strenuous swim in temperate water after ingestion of a large volume of water.(7) So cold-induced vasoconstriction is not required in all cases, if cardiac filling pressures are increased by both immersion and pre-hydration. Others have confirmed that hypertensive divers are particularly at risk of IPO. An increase in cardiac output by exercise or sometimes by emotional stress can be implicated. We have also found that IPO may be the first manifestation of cardiac disease, renal artery stenosis (two cases - one had seven episodes of IPO) and conditions causing fluid retention. Therefore it appears that IPO is the result of a combination of factors that increase pulmonary capillary pressure to a critical level. Recent experience suggests that use of certain types of closed circuit rebreathers may further increase the risk of immersion pulmonary oedema.

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The “Vansbrosimningen” experience – incidence of swimming induced pulmonary edema (SIPE) and organization of prehospital medical resources

Annika Braman Eriksson, MD, Vansbro Primary Healthcare Center
Maria Hårdstedt, MD, PhD, Cardiology department Falun Hospital/Center of Clinical Research (CKF) Dalarna-Uppsala University

Since the Vansbro open water race, “Vansbrosimningen”, started in 1950 the number of participants has continuously increased. Today the event attracts approximately 13000 swimmers to the cold rivers of Western Dalarna in early July to swim 1000, 1500 or 3000 meter. The participants include experienced as well as unexperienced swimmers of all ages with a close to equal numbers of men and women.

Breathing problems and swimming induced pulmonary edema (SIPE)
Dyspnea with a characteristic severe cough has been a major health issue among swimmers through the years; originally thought as cold asthma or aspiration. Attempts to treat with beta2-agonist inhalation, oxygen and diuretics have been unsuccessful. Clinical findings (e.g. crepitations at lung auscultation), literature review and contacts with military divers led to the conclusion that breathing problems could be due to pulmonary edema. Since five years, continuous positive airway pressure (CPAP) has successfully been used to treat clinical SIPE at the on-site medical care unit.
In 2016, an evaluation of the medical care at “Vansbrosimningen” was performed [1]. Of 13 900 participants, 69 (0.5%) were seen at the on-site medical unit for dyspnea and/or coughing. The vast majority (n=58) were women. There was an overrepresentation of earlier diagnosed asthma in the group. The symptoms varied from coughing to fulminant pulmonary edema. At arrival, 27 patients had oxygen saturation ≤90% (≤80% (n=5), 81-85% (n=4 and 86-90% (n=18)). 47 patients were treated with CPAP. No patient had to be transferred to the hospital. In 2017, we started an organized research study at “Vansbrosimningen” with the aim of characterizing the clinical condition, study the occurrence of SIPE, evaluating the prehospital treatment and identifying risk factors. At the Stockholm SIPE conference we hope to share preliminary data from this year’s swimming event.

Organization of prehospital on-site medical care during “Vansbrosimningen”
Vansbro is a small village located 78 kilometers from the nearest emergency hospital. With a growing open water event a professional organization for security and health care was needed – nearly ten years ago there was a division between first aid and prehospital health care. The *first aid team* is spread along the riverside during the race. They identify swimmers with health problems, pull them out of the water and bring them to the healthcare unit. The staff at *the healthcare unit* consists of four doctors and four nurses in close contact with an *ambulance crew*. There is continuous contact over radio between the first aid crew, the health care unit and the ambulance. The on-site medical care unit consists of two tents and a container. The unit is equipped with oxygen, ECG, emergency medications, suturing equipment, defibrillation/intubation equipment and four CPAP units.

Conclusions
¤ Open water swimming races are increasingly popular. Knowledge about SIPE among organizers, health care providers and swimmers is crucial. Professional first aid/medical care organizations around open water races are needed.
¤ Awareness of SIPE in the organization at “Vansbrosimningen” has led to early recognition of swimmers with breathing problems and efficient prehospital care.
¤ Prehospital treatment of SIPE with CPAP has been successful.
¤ The incidence of breathing problems in a mixed population of swimmers (regarding experience, age and sex) in an open water race in Sweden was about 0.5%.
¤ Women seemed more prone to SIPE then men.

1. Braman Eriksson, A., M. Annsberg, and M. Hårdstedt, Läkartidningen, 2017. 114.

”Pappa, är allt ok?”

Peter Geiger, ÖL Anestesi och Intensivmedicin, Dalsjöfors

Hur känns det när man drabbas av akut andnöd under en simtur ute i en sjö? En berättelse: SIPE under ett vanligt träningspass, spontanförlopp.

Jag är en aktiv människa, håller på med simning, löpning, cykling och även dykning, skulle betrakta mig som vältränat idag. Men när det inträffade var jag 50 år gammal och höll jag på med uthålighetsidrott under ca 1 år: ett Göteborgsvarv var avklarat och träningen hade blivit del av min vardag, 8-12 olika träningspass/vecka. Simträning hela vintern i bassängen, 2-3 gånger per vecka, från maj även i sjön; bröstsim och crawl blandades fortfarande eftersom jag hade just lärt mig att crawla.

Det var juli månad, vattentemperatur ca 17 grader, jag hade en triathlonvåtdräkt på mig; ett vanligt träningspass i en insjö, jag var ensam i vattnet. De första 250m gick bra, jag försökte att öka tempot! Men sedan: Andfåddhet! Hostretningar!

Jag trodde först att jag hade aspirerat vatten, pauserna under simturen blev allt tätare, andfåddheten blev inte bättre, hostretningar värre, rassel tillkommer. Jag simmade i land, tog mig hem (1km), ladde mig rätt snabbt, fortsatt andfått, huvudändan upphöjd, tachykard med 100/min (vilopuls 60/min). Saturation? Trots att jag är läkare med anestesiologi som specialitet: ingen saturationsmätare hemma! Under natten fick jag även rosafärgade upphostningar så att dottern vaknade: ”Pappa, är allt ok?”.

På morgonen var jag fortsatt påverkad, men rassel hade försvunnit, jag tog mig till jobbet. Att det var en SIPE jag hade råkat ut för förstog jag först i efterhand….

Det upprepades aldrig trots fortsatt intensiv träning och olika riskmoment: Swim-run i omgånger (Ö-loppet sprint senast augusti 2017), simning i 3 gradigt vatten på julafton, olika triathlon i sprintdistanser etc.

Vad har det gjort med mig? Funderingar, konsekvenser, tips och råd.

Present research on swimming-induced pulmonary edema (SIPE)

Richard E. Moon, MD, Duke University Medical Center, Durham, NC 27710, USA

Plausible rationale and most evidence suggests that SIPE is a form of hemodynamic pulmonary edema. Its onset is usually rapid; it generally resolves quickly once the victim is out of the water; when it occurs in swimmers in the lateral position (side stroke) the dependent lung tends to be involved. These observations all suggest that SIPE is due to high pulmonary trans-capillary pressure. Therefore, conditions that impair cardiac systolic or diastolic function or increase intrathoracic blood volume are likely to predispose to SIPE. Indeed a recent review of 292 published cases of SIPE reported that almost half of civilian cases had a cardiac or pulmonary abnormality that could provide a possible mechanism for SIPE, or a plausible risk factor for such an abnormality.[13] These include hypertension, cardiomyopathy, myocardial ischemia, valve disease and obstructive lung disease. Gempp reported 54 consecutive divers admitted for SIPE. Of these, 15 (28%) manifested myocardial dysfunction defined as the presence of elevated cardiac troponin T levels, EKG changes and/or wall motion abnormalities on TTE.[5] Several cases have been reported of SIPE precipitated by Takatsubo cardiomyopathy.[2-4,12] These cases are examples of acute myocardial dysfunction as a precipitating cause.

These examples may explain some cases, but what could be the mechanism for SIPE in military populations[1,7,14,15] or triathletes?[9] To investigate the mechanism in a healthy population we performed two studies. In the first we instrumented with arterial and pulmonary artery catheters a group of normal volunteers and a cohort of healthy individuals with a SIPE history. We studied them during 6-10 minutes of moderate exercise while submersed in 20°C water. We observed that for a given cardiac output the SIPE-susceptible individuals had higher pulmonary artery (PAP) and wedge (PAWP) pressures. After administration of sildenafil 50 mg, PAP and PAWP were lower during the same submersed exercise conditions.[11] One individual with multiple SIPE episodes has continued to take sildenafil prescribed by her doctor before triathlon races and has had no recurrences.[8]

This provided the mechanism but it doesn't explain why the pressures were higher. We then studied a group of normal volunteers and SIPE-susceptible individuals in whom risk factors had been excluded during head-out immersed exercise. We performed transthoracic echocardiography in the dry before exercise and during rest and exercise in the water. All had normal resting dry echo studies, however compared to the controls, in the SIPE-susceptible volunteers an index of diastolic left ventricular stiffness (E/e') was higher. This suggests that in susceptible individuals the triple combination of immersion/submersion (causing central redistribution of blood, increased preload), heavy exercise (high cardiac output) and less compliant left ventricle results in higher LV end-diastolic pressure. This then leads to capillary pressures that exceed their elastic limit, causing rupture or allowing fluid to cross the alveolar capillary membrane. Further observations using the same model have implicated fluid loading as a risk factor for SIPE.

Recently published case series make the argument that many triathlon deaths could be due to SIPE.[6,10] This is based on the high prevalence of markers of SIPE susceptibility found at autopsy, especially left ventricular hypertrophy.[10]

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