Pulmonary oedema in healthy persons during scuba-diving and swimming

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ABSTRACT: The prevalence of pulmonary oedema during scuba-diving is unknown. In our referral centre for diving accidents we have observed several episodes of pulmonary oedema in four previously healthy persons while scuba-diving or swimming. Four events were documented by physical findings, typical chest radiographic changes, and arterial hypoxaemia. Four additional episodes were identified in one of the individuals by a suggestive history. No technical problems with the diving equipment were detectable and none of the individuals reported aspiration of water.

In order to gather information about the incidence of pulmonary oedema, we carried out a survey among 1,250 divers. To elucidate possible underlying mechanisms of this complication we investigated forearm vascular resistance, levels of vasoreactive hormones, and left ventricular function by Doppler echocardiography, at room temperature and during cold exposure, in four patients and in healthy control subjects.

We found only one additional person with a history suggestive of pulmonary oedema among 460 responders to the survey. We found no differences in forearm vascular resistance, left ventricular systolic and diastolic function, and plasma levels of epinephrine, norepinephrine, cortisol, aldosterone, renin and atrial natriuretic peptide between the patients with a history of pulmonary oedema and the control subjects.

We conclude that the occurrence of pulmonary oedema during scuba-diving or swimming is an extremely rare event in healthy individuals. The mechanisms responsible remain unclear.
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The occurrence of pulmonary oedema during scuba (self-contained underwater breathing apparatus) diving or swimming was reported for the first time in 1981 by WILMSHURST and co-workers [1]. These investigators reported up to seven episodes of pulmonary oedema in 11 individuals whilst diving [2] or swimming [3]. They found that these persons had a larger increase in forearm vascular resistance during cold exposure than divers who did not report such episodes [3]. They speculated that an exaggerated increase in afterload due to abnormal vascular reactivity might play a crucial role, together with the elevated preload occurring regularly during swimming or diving.

This type of pulmonary oedema seems to be a very rare event, since no other cases have been reported in the literature. We studied forearm vascular reactivity, systolic and diastolic left ventricular function, and changes in plasma hormones during cold exposure, and obtained the 24 h blood pressure profiles in four previously healthy individuals with documented episodes of pulmonary oedema occurring during scuba-diving or surface swimming, and in an additional diver with a suggestive history, whom we identified by an inquiry among 1,250 scuba divers in Switzerland.

Case reports

The hyperbaric chamber of our hospital serves as a referral centre for diving accidents occurring in the German and Italian speaking part of Switzerland (population approximately 4.8 million). From April 1991 to November 1992, we observed four persons who had experienced episodes of pulmonary oedema whilst diving or swimming. None of them had a history of asthma, heart disease, high blood pressure or Raynaud's syndrome, and all were nonsmokers. A synopsis of the symptoms, clinical findings and outcomes is presented in table 1.

Patient No. 1

A 26 year old healthy, experienced, male scuba diver was diving to 42 m in Lake Lugano (southern Switzerland).

Table 1. – Four case reports of pulmonary oedema; symptoms, findings and outcome

Pt. No.	Episode	Circumstance	Sex	Age yrs	Lowest water temperature °C	Symptoms	CXR admission	Pao₂ on admission kPa (mmHg)	Pao₂ before discharge kPa (mmHg)	Outcome
1	*	Diving	M	26	5.3	Cough, bloody froth, dyspnoea	POE	8.3 (62)	13.9 (104)	POE cleared after 12 h
2	1st⁺	Diving	F	39	4.7	Cough, rales	–	–	–	Symptoms disappeared within 4 days
	2nd*	Diving			5.2	Dyspnoea	POE	13.3§ (100)	10.7 (80)	POE cleared after 8 h
	3rd⁺	Swimming			20.6	Dyspnoea, cough white froth	–	–	–	Symptoms disappeared after 1 h
	4th⁺	Swimming			18.0	Dyspnoea, cough, white froth	–	–	–	Symptoms disappeared after 30 min
	5th⁺	Swimming			18.0	Cough, white froth	–	–	–	Symptoms disappeared after 15 min
3	*	Diving	M	27	5.6	Cough, bloody froth, dyspnoea	POE	5.9 (44)	12.0 (90)	POE cleared after 9 h
4	*	Swimming	M	23	20.6	Cough, bloody froth	POE	8.8 (66)	11.2 (84)	POE cleared after 8 h

Pt: patient; M: male; F: female; *: documented episode; ⁺: episode suggested by history; CXR: chest radiograph; POE: pulmonary oedema; Pao₂; pulmonary arterial oxygen tension. §: with oxygen 4 $l\cdot min^{-1}$ *via* nasal prongs.

Time spent at the bottom was 16 min. During a normal ascent he experienced cough and mild breathing discomfort. He performed regular decompression stops at 6 and 3 m. On surfacing, he noticed bloody froth around his mouth. He had no chest pain. On admission to hospital, a diagnosis of pulmonary oedema was made based on clinical findings and on chest radiographs.

Patient No. 2

A 39 year old, well-trained female diver experienced two episodes of pulmonary oedema whilst scuba-diving, and three episodes during surface swimming. The first episode occurred after a dive to 25 m, of a duration not requiring decompression stops. She experienced mild cough and noticed rales over her chest after reaching the surface. These symptoms disappeared within a few days and she did not seek medical help. The second episode occurred 3 months later, when the patient made a dive to 39 m. During a slow ascent, she experienced shortness of breath at a depth of 13 m, but was able to continue the ascent, without requiring decompression stops, in a controlled manner. She complained of severe shortness of breath but no chest pain. On admission to hospital, a diagnosis of pulmonary oedema was established by physical findings and by chest radiograph. A Doppler echocardiogram was normal a few hours after the event. Pulmonary oedema cleared almost completely within 8 h. The third episode occurred 11 months later when the patient was participating in the traditional swim across Lake Zürich (distance 1.2 km). After a distance

of about 1,000 m, she became short of breath and had a mild cough. She noticed rales over her chest but did not expectorate froth, and she shivered. The patient recovered without treatment within a few hours. The fourth episode occurred 2–3 months later when the patient was performing her regular weekly swimming training in a public indoor swimming pool. After a distance of 600 m she experienced shortness of breath and coughed small quantities of white froth. Again she felt very cold. Her symptoms disappeared with no treatment within 30 min. The fifth episode occurred when the patient was swimming in the same swimming pool and at the same water temperature. Suddenly, she started to cough and expectorated white froth but recovered after a short time.

Patient No. 3

A 27 year old police diver was performing rescue exercises with repetitive dives to depths not more than 24 m and not requiring decompression stops. The dive was uneventful and the patient noticed no equipment failure. Shortly after ascent, he experienced shortness of breath, coughed and expectorated reddish froth. He had no chest pain. On admission to hospital, he was slightly hypothermic, with a rectal temperature of 35.4°C, and bilateral rales were audible over his chest. Pulmonary oedema was confirmed by chest radiograph. A Doppler echocardiogram was normal one day after the event. Twelve hours later, chest radiograph, arterial blood gases and physical findings were unremarkable.

Patient No. 4

A 23 year old man, in previously good health, was taking part in the same traditional swim across Lake Zürich as patient No. 2. After about 600 m (half the distance) he started to cough and had to climb into a rescue boat because of worsening of symptoms within 10 min. He coughed up pink froth, but had no shortness of breath and no chest pain. On admission to hospital, a diagnosis of pulmonary oedema was established by clinical findings and by chest radiograph. Doppler echocardiography was normal one day later. The lung oedema cleared within 8 h.

The follow-up of the individuals reported (Autumn 1994) revealed no evidence of evolution of heart or lung disease meanwhile.

Methods

During 1991, we carried out a survey, amongst 1,250 recreational divers using questionnaires asking for a history of health problems in association with diving, particularly cardiorespiratory symptoms. According to the number of members of 70 sections of the Swiss Underwater Sport Association the address of every third to fifth member was randomly selected. In order to detect further possible incidents of pulmonary oedema, questions were asked about history of events with bloody expectoration, frothy sputum, shortness of breath, laboured breathing and cough during and/or after diving or swimming.

All six control subjects were nonsmoking healthy males aged 33–46 yrs and had a level of physical fitness comparable to the patients.

The following experiments were performed in the four patients, in one diver (M.B.) recruited from the survey and in six healthy control subjects. All patients and subjects gave informed consent. The study was approved by the hospital's Local Ethics Committee. None of the subjects was on a regular drug treatment. The studies were conducted at a room temperature of 20°C. The cold packing of head and neck was performed with towels soaked in iced water and lasted for 3 min.

Forearm vascular resistance

A 21-gauge Venflon® catheter was inserted into the antecubital vein of the right arm. The subjects were in a supine position and relaxed for 30 min before the start of the experiment. The electrocardiogram was displayed on an oscilloscope (Servomed®, Ruegge Medical, Switzerland). Blood pressure was measured on the right arm by an automatic sphygmomanometer (Tonomed Electronic®; Speidel & Keller KG, Germany).

Mean arterial blood pressure was calculated by adding one third of the pulse pressure to the diastolic pressure. Forearm vascular resistance (units) was calculated by dividing mean blood pressure by forearm blood flow. Blood flow was measured by a venous occlusion plethysmograph (Periquant 3800®; Gutmann-Medizinelektronik, Germany) according to standard techniques [4]. The left arm was supported comfortably just above the level of the heart. A pneumatic venous occlusion cuff was wrapped around the left upper arm and a mercury-in-rubber strain gauge was applied to the left forearm at the point of maximum girth. The forearm blood flow was measured by instantaneous inflation of the cuff to 60 mmHg, whilst recording the rate of increase in the forearm circumference (*i.e.* plethysmographic forearm volume expressed as ml·dl tissue^{-1}·min^{-1}).

Measurements of pulse rate, arterial blood pressure and blood flow were obtained at 1 min intervals, for 7 min before and 7 min after cold-packing of the neck with ice packs. Repeat studies in the six healthy controls showed that the results were highly reproducible.

Plasma hormones

Blood samples were collected from the subjects after they had rested for 30 min in supine position at 3 min before, and 3 and 7 min after cold exposure. The tubes were immediately placed in iced water and centrifuged within 30 min at 4,000 rpm for 10 mins at 4°C. The samples were stored at -70°C until analysis. Epinephrine and norepinephrine were measured with high performance liquid chromatography (HPLC) separation and electrochemical detection [5]. Cortisol [6], aldosterone [7], plasma renin activity (Plasma Renin Activity Radioimmunoassay Kit®; INCSTAR Corp., USA) and human atrial natriuretic peptide (HANP Kit®; Eiken Chemical Co. Ltd, Japan) were measured by radioimmunoassay kits.

Doppler echocardiography

On a separate day, a 21-gauge Venflon® catheter was inserted into the antecubital vein of the left arm. The patients were supine and rested quietly for 30 min before the examination began. Cold packing was performed as described above. Transthoracic two dimensional echocardiography, colour Doppler flow, continuous and pulsed Doppler measurements were performed using Hewlett-Packard® equipment (Andover, Massachusetts, USA) with a 2.5 MHz transducer. Structural abnormalities of the heart were sought, and diameters of the left chambers, and systolic and diastolic cardiac function were evaluated before and during cold exposure. The leading-edge to leading-edge technique was used to measure the cardiac diameters [8]. A syringe containing 8 ml normal saline at body temperature was forcefully shaken by hand, and the fluid containing microbubbles was rapidly injected into the antecubital vein. This standardized technique comprising a Valsalva manoeuvre (*i.e.* coughing immediately after injection) is used to exclude a patent foramen ovale or other communications between the right and left heart. The diastolic function was analysed, measuring the transmitral blood flow velocities with pulsed Doppler echocardiography assessed by apical position of the transducer [9, 10]. The analysis of the Doppler

flow profiles was performed shortly before and 2 min after cold-packing of the neck, since vascular changes peaked between 1–3 min. The following parameters were measured: early peak diastolic mitral flow velocity (cm·s⁻¹), atrial peak diastolic mitral flow velocitiy (cm·s⁻¹) and their ratio (E/A ratio), as well as isovolumetric relaxation time (ms).

24 h blood pressure profile

The blood pressure was measured throughout 24 h, at 20 min intervals during the day and at hourly intervals during the night, by means of an ambulatory blood pressure monitor (ABD-Monitor 90207®; Space-Labs, USA).

Statistical analysis

Baseline for all parameters was defined as the mean of the respective measurements taken at 1 min intervals during 7 min before cold exposure. The results during the cold exposure were expressed as the mean of the measurements at 1, 2 and 3 min, since the changes peaked between the first and third minute, thereafter steadily decreasing in both groups.

Since we observed only four patients with documented episodes and one additional person with a suspected episode of pulmonary oedema, the individual results of the experiments performed are presented, without statistical calculations. The results of the control subjects are presented as median values and ranges and the nonparametric sign test was used, when appropriate (StatSoft® 1994; Statistica for Macintosh). Differences were considered significant when p was less than 0.05.

Results

All four patients who had episodes of pulmonary oedema were healthy nonsmokers with no history of pulmonary or cardiac disease. Clinical examinations were normal. Doppler echocardiograms revealed no heart disease, and none of the patients had an open foramen ovale. The patients gave reliable histories and aspiration of water could be excluded. Their diving profiles were regular. A failure of the diving equipment could be ruled out by history and examination in our laboratory. A detailed report of the events is presented in the previous section and summarized in table 1.

From the 1,250 questionnaires, 37% (460) were returned for analysis from 135 female and 325 male divers. We invited 18 persons who gave a history consistent with a previous episode of pulmonary oedema for a thorough interview and a clinical examination. Only one 24 year old diver reported symptoms evocative of pulmonary oedema (included as patient M.B. in table 2). After a strenuous dive to 20 m, within a time not requiring decompression stops, he experienced an obstinate cough and became short of breath. He coughed reddish froth and noticed rales over his chest after reaching the surface. The symptoms resolved spontaneously within 3 h.

During the experiments, all patients as well as the healthy volunteers experienced a local cold sensation during the first minute after application of the ice packs. This was followed by a sensation of pain, peaking after 2 min and progressively decreasing thereafter. Preliminary experiments had shown that covering a more extended area by cold-packing could not be tolerated, and induced bradycardia and nausea in some individuals. No laboured breathing was reported, and the heart rate did not change

Table 2. – Vascular response to cold exposure in patients and in healthy volunteers

	BPsys mmHg	BPdias mmHg	HR b·min⁻¹	Forearm Q̇ ml·dl tissue⁻¹·min⁻¹	BP mean mmHg	VR Units
Patients n=5						
Baseline						
No. 1	115	86	64	2.5	95	40
No. 2	143	101	66	2.5	115	47
No. 3	127	72	51	5.4	91	18
No. 4	126	70	61	1.9	88	51
M.B.	122	78	58	2.9	93	35
Cold exposure						
No. 1	121	90	65	1.6	100	66
No. 2	145	107	61	1.5	120	85
No. 3	130	81	48	2.7	98	38
No. 4	136	76	63	1.6	96	92
M.B.	129	84	55	1.7	99	59
Volunteers n=6						
Baseline						
Median	119	82.5	58.5	3.0	96.5	33
Range	110–131	77–87	50–60	1.3–4.5	88–99	22–70
Cold exposure	NS	NS	NS	*	NS	*
Median	122	88.5	60	1.6	98	66
Range	112–140	81–101	48–61	0.8–2.4	92–114	40–136

NS: nonsignificant; *: p<0.05. M.B.: patient with pulmonary oedema suggested by history; BP: blood pressure; Q̇: blood flow; BPsys: systolic blood pressure; BPdias: diastolic blood pressure; Forearm Q̇: see methods; VR: vascular resistance.

during cold exposure in either group of subjects. No cardiac dysrhythmias were detected at any stage, with all patients and controls remaining in sinus rhythm.

Median systolic and diastolic blood pressure were equal in the patients and the normal volunteers and remained unchanged (table 2). Since the calculated mean blood pressure did not change significantly and the median blood flow decreased by 53% from 3.0 to 1.6 ml·dl tissue^{-1}·min^{-1}, the median vascular resistance increased by 100% from 33 to 66 U. Reduction in forearm blood flow and a corresponding increase in forearm vascular resistance occurred in patients and controls, but no differences were noticed between the two groups (table 2).

The Doppler echocardiograms were normal. A patent foramen ovale or other structural abnormalities were excluded in patients and healthy volunteers. Analysis of systolic function and of transmitral pulsed Doppler flow profiles before and 2 min after cold-packing of the neck found no abnormal changes. In the volunteers, the median left ventricular shortening fraction was 33% (range 29–36%), the ratio of early peak diastolic to atrial peak diastolic mitral flow velocity (E/A-ratio) was 1.6 (range 1.4–2.1) and isovolumetric relaxation time was 86 ms (range 75–100 ms) and did not change during cold exposure. These indices were comparable to those of the patients.

Serum levels of epinephrine, norepinephrine, aldosterone, cortisol, and atrial natriuretic peptide did not change during cold exposure (data not shown). The baseline levels of these hormones were not different between patients and healthy volunteers. No consistent reactions could be detected in the individuals with a history of pulmonary oedema.

The only diver from our survey who had a history suggestive of pulmonary oedema, a 24 year old man (M.B.), was not different with regard to vascular response, echocardiographic parameters, 24 h blood pressure readings and plasma hormone profiles from the patients and the healthy volunteers (table 2).

Discussion

The occurrence of pulmonary oedema during scuba-diving or swimming is very rare in healthy individuals. The underlying mechanisms remain elusive. According to the diving profile as well as the clinical presentation, our patients' complaints and findings cannot be attributed to the pulmonary manifestation of decompression sickness This condition, known among divers as"chokes", occurs when excessive numbers of gas bubbles liberated during decompression are trapped in the pulmonary circulation and cause severe retrosternal pain, accompanied by extreme fatigue, dry cough and, eventually, respiratory distress [11–14]. That "chokes" are not the cause of pulmonary oedema in the cases observed is supported by our observation of the same clinical picture in a healthy young man, who developed pulmonary oedema whilst swimming, and a 39 year old athletic woman, who experienced repetitive episodes of pulmonary oedema not only during scuba-diving but also during swimming.

The clinical presentations of our cases are identical to those of WILMSHURST and co-workers [1–3], who have reported the occurrence of pulmonary oedema in persons during scuba-diving as well as surface swimming. This group described 11 divers, some with up to seven episodes of pulmonary oedema whilst scuba-diving and breathing air, and two with similar episodes during swimming at the surface. Surprisingly, we did not find further published observations, but have been told of a very few isolated, nonpublished cases by experts in diving medicine. Thus, the occurrence of pulmonary oedema during diving and swimming seems to be an extremely rare event in healthy persons. This notion is also supported by our survey amongst 1,250 recreational divers, where we found only one single additional suspicious case.

The mechanisms of pulmonary oedema occurring during scuba-diving and swimming remain unknown and speculative. Obvious causes, such as aspiration of water, pulmonary decompression illness, forced inspirations against an increased resistance, or pre-existing or evolving heart disease could be excluded by history and circumstantial evidence. WILMSHURST and co-workers [3] demonstrated that divers with a history of pulmonary oedema had a larger increase in forearm vascular resistance during a cold pressure test compared to normal persons. Some of the divers even developed a third heart sound and functional mitral regurgitation, and in one diver left ventricular failure, causing severe dyspnoea and basal crepitations, was noticed during the experiments. We were not able to confirm these findings in our patients (table 2). The forearm vascular resistance doubled and the blood flow decreased by 53% in our volunteers, changes which are in accordance with the haemodynamic reactions in the normal divers studied by WILMSHURST and co-workers [3]. However, in contrast to their findings, our patients exhibited normal values of mean arterial blood pressure and the amount of vasoconstriction of their forearm vessels was not different from the reaction of the healthy volunteers. We have no explanation for this discrepancy. The intensity of our cold exposure tests seems to be comparable, since the degree of changes that we observed in our volunteers were similar to those of the British group.

The introduction of the 24 h ambulatory blood pressure monitoring has contributed greatly to our understanding of hypertension. However, there is yet no clear definition of hypertension based on this procedure [15]. Some authors propose that hypertension be defined as the presence of at least 50% of awake blood pressure readings over 140/90 mmHg [16]. Given these criteria, one patient and two controls in our study were hypertensive.

Although significant and reproducible increases in forearm vascular resistance were inducible, changes in plasma norepinephrine levels, known to occur during cold immersion [17], could not be observed. Obviously, cold-packing of the neck is different from immersion of the forearm in iced water. Our cold exposure test was either not intense enough or induced counterregulatory mechanisms. This might be the reason why no changes in the plasma hormone profile were detectable. We

could not strengthen the cold stimulus by packing a more extensive area of the head, because this was not endured by our test persons. Since no results with a similar experimental set-up are reported in the literature, we are unable to speculate further on these negative findings.

There have been anecdotal reports of acute pulmonary oedema developing in apparently healthy elite athletes during marathon running [18]. However, it has not been shown that interstitial pulmonary oedema develops during strenuous exercise in normal humans at sea level [19]. Immersion is known to considerably increase preload [20]. Cold exposure increases both preload and afterload by vasoconstriction [21]. It is, therefore, conceivable that a combination of these mechanisms, as occurs during diving or swimming, together with an increase in cardiac output could be responsible for an excessive increase in pulmonary capillary pressure in certain susceptible individuals. This might explain why we were unable to induce measurable changes in left ventricular systolic and diastolic function in our experimental set-up, consisting of cold exposure without immersion and physical activity.

The role of cold exposure as an important stimulus for exaggerated vascular response and as a prerequisite for the development of pulmonary oedema during scuba-diving or swimming remains open to debate. According to the observations of WILMSHURST and co-workers [3], all episodes of pulmonary oedema have occurred in cold British waters with temperatures below 12°C, whereas some of our individuals developed pulmonary oedema during swimming at higher water temperatures (table 1).

It seems plausible, that this type of pulmonary oedema, as observed by WILMSHURST and co-workers [1–3] as well as our group, is more likely to be caused by a transient increase of the pressure than due to permeability changes of the pulmonary capillaries, although no data exist to exclude the latter mechanism.

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