Pulmonary oedema in healthy persons during scuba-diving and swimming


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.

Eur Respir J., 1995, 8, 762–767.

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).
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.

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Episode</th>
<th>Circumstance</th>
<th>Sex</th>
<th>Age yrs</th>
<th>Lowest water temperature °C</th>
<th>Symptoms</th>
<th>CXR admission</th>
<th>PaO2 on admission kPa (mmHg)</th>
<th>PaO2 before discharge kPa (mmHg)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>*</td>
<td>Diving</td>
<td>M</td>
<td>26</td>
<td>5.3</td>
<td>Cough, bloody froth, dyspnoea</td>
<td>POE</td>
<td>8.3 (62)</td>
<td>13.9 (104)</td>
<td>POE cleared after 12 h</td>
</tr>
<tr>
<td>2</td>
<td>1st*</td>
<td>Diving</td>
<td>F</td>
<td>39</td>
<td>4.7</td>
<td>Cough, rales</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Symptoms disappeared within 4 days POE cleared after 8 h</td>
</tr>
<tr>
<td></td>
<td>2nd*</td>
<td>Diving</td>
<td></td>
<td></td>
<td>5.2</td>
<td>Dyspnoea</td>
<td>POE</td>
<td>13.3§ (100)</td>
<td>10.7 (80)</td>
<td>Symptoms disappeared after 1 h</td>
</tr>
<tr>
<td></td>
<td>3rd*</td>
<td>Swimming</td>
<td></td>
<td></td>
<td>20.6</td>
<td>Dyspnoea, cough white froth</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Symptoms disappeared after 30 min</td>
</tr>
<tr>
<td></td>
<td>4th*</td>
<td>Swimming</td>
<td></td>
<td></td>
<td>18.0</td>
<td>Dyspnoea, cough white froth</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Symptoms disappeared after 15 min</td>
</tr>
<tr>
<td></td>
<td>5th*</td>
<td>Swimming</td>
<td></td>
<td></td>
<td>18.0</td>
<td>Cough, white froth</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>*</td>
<td>Diving</td>
<td>M</td>
<td>27</td>
<td>5.6</td>
<td>Cough, bloody froth, dyspnoea</td>
<td>POE</td>
<td>5.9 (44)</td>
<td>12.0 (90)</td>
<td>POE cleared after 9 h</td>
</tr>
<tr>
<td>4</td>
<td>*</td>
<td>Swimming</td>
<td>M</td>
<td>23</td>
<td>20.6</td>
<td>Cough, bloody froth</td>
<td>POE</td>
<td>8.8 (66)</td>
<td>11.2 (84)</td>
<td>POE cleared after 8 h</td>
</tr>
</tbody>
</table>

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·min⁻¹ via nasal prongs.
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 carried out at 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 transmirtal 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 velocity (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.

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

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

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

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...
during cold exposure in either group of subjects. No cardio-
diac dysrhythmias were detected at any stage, with all pa-
tients 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 pres-
sure 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 correspond-
ing 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 ex-
cluded 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 med-
ian 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 expo-
sure. These indices were comparable to those of the patients.

Serum levels of epinephrine, norepinephrine, aldost-
erone, cortisol, and atrial natriuretic peptide did not
change during cold exposure (data not shown). The base-
line 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 pul-
monary oedema.

The only diver from our survey who had a history sug-
uggestive of pulmonary oedema, a 24 year old man (M.B.),
was not different with regard to vascular response, echo-
cardiographic 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 sick-
ness. This condition, known among divers as “chokes”,
occur when excessive numbers of gas bubbles liberated
during decompression are trapped in the pulmonary cir-
culation and cause severe retrosternal pain, accompanied
by extreme fatigue, dry cough and, eventually, respiratory
distress [11–14]. That “chokes” are not the cause of pul-
monary 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 expe-
rienced 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 dur-
ing 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 evolv-
ing heart disease could be excluded by history and cir-
cumstantial evidence. WILMSHURST and co-workers [3]
demonstrated that divers with a history of pulmonary
oedema had a larger increase in forearm vascular resis-
tance during a cold pressure test compared to normal per-
sons. 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 pat-
ients (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 find-
ings, our patients exhibited normal values of mean arte-
rial blood pressure and the amount of vasoconstriction
of their forearm vessels was not different from the reac-
tion 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 pres-
sure monitoring has contributed greatly to our under-
standing 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 hyper-
tensive.

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.

Acknowledgements: The authors thank B. Küffer, Division of Arterial Hypertension and J. Schalk, Division of Angiology, Department of Internal Medicine, University Hospital of Zurich for technical assistance. They also thank K. Bloch, Pulmonary Division, for valuable discussions.

References


