HIGHLIGHTED TOPIC | The Physiology and Pathophysiology of the Hyperbaric and Diving Environments

Effects of head and body cooling on hemodynamics during immersed prone exercise at 1 ATA


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Submitted 15 September 2008; accepted in final form 16 November 2008

Wester TE, Cherry AD, Pollock NW, Freiberger JJ, Natoli MJ, Schinazi EA, Doar PO, Boso AE, Alford EL, Walker AJ, Uguccioni DM, Kernagis D, Moon RE. Effects of head and body cooling on hemodynamics during immersed prone exercise at 1 ATA. J Appl Physiol 106: 691–700, 2009. First published November 20, 2008; doi:10.1152/japplphysiol.91237.2008.—Immersion pulmonary edema (IPE) is a condition with sudden onset in divers and swimmers suspected to be due to pulmonary arterial or venous hypertension induced by exercise in cold water, although it does occur even with adequate thermal protection. We tested the hypothesis that cold head immersion could facilitate IPE via a reflex rise in pulmonary vascular pressure due solely to cooling of the head. Ten volunteers were instrumented with ECG and radial and pulmonary artery catheters and studied at 1 atm absolute (ATA) during dry and immersed rest and exercise in thermoneutral (29–31°C) and cold (18–20°C) water. A head tent varied the temperature of the water surrounding the head independently of the trunk and limbs. Heart rate, Fick cardiac output (CO), mean arterial pressure (MAP), mean pulmonary arterial pressure (MPAP), pulmonary artery wedge pressure (PAWP), and central venous pressure (CVP) were measured. MPAP, PAWP, and CO were significantly higher in cold pool water (P ≤ 0.004). Resting MPAP and PAWP values (means ± SD) were 20 ± 2.9/13 ± 3.9 (cold body/cold head), 21 ± 3.1/14 ± 5.2 (cold/warm), 14 ± 1.5/10 ± 2.2 (warm/warm), and 15 ± 1.6/10 ± 2.6 mmHg (warm/cold). Exercise values were higher; cold body immersion augmented the rise in MPAP during exercise. MAP increased during immersion, especially in cold water (P < 0.0001). Except for a transient additive effect on MAP and MPAP during rapid head cooling, cold water on the head had no effect on vascular pressures. The results support a hemodynamic cause for IPE mediated in part by cooling of the trunk and extremities. This does not support the use of increased head insulation to prevent IPE.

diving; immersion; pulmonary edema; pulmonary circulation

Immersion pulmonary edema (IPE) occurs sporadically in healthy divers and swimmers and is characterized by cough, shortness of breath, decreased blood oxygen levels (17, 24, 29, 35, 39), and sometimes death (13). Symptoms may manifest early in dives, with reports of dyspnea as early as 7 min after reaching depth (13). Other cases have been reported after prolonged periods of exercise and immersion (1, 29, 30, 34). The cause for this phenomenon is unknown. Risk factors seem to include heavy exertion and cold water (24), although IPE has been reported in water close to thermoneutral temperatures without exertion (17, 24). Studies of individuals who have recovered from IPE have not identified chronic pulmonary function abnormalities or pulmonary hypertension (28). Ventricular function (24) and resting pulmonary artery systolic pressures (28) are generally normal.

Indirect evidence implicates high pulmonary vascular pressures (27). Pulmonary edema has been described in the dependent lung of special forces combat swimmers swimming in the lateral decubitus position (29, 30). One study reported that subjects who have experienced IPE have an exaggerated vasoconstrictive response to applications of cold to the head and neck (40). While the effect of cold water exposure may partly explain the condition, the factors that lead to an increase in pulmonary vascular pressures are incompletely understood. Passive redistribution of blood from the periphery or peripheral vasoconstriction as a direct reaction to cold water may play a role in this increase (25). There may also be a causal role for the mammalian diving reflex, which consists of a group of responses including apnea, bradycardia, decreased cardiac output, peripheral vasoconstriction, and increased mean arterial blood pressure (MAP) in diving mammals. Selective peripheral vasoconstriction and increased MAP allow preferential oxygen delivery to the heart and brain by reducing blood flow to visceral organs (e.g., gastrointestinal tract and kidney) (7).

In humans, some of the features of the diving reflex can be induced by facial immersion in cold water. Bradycardia, hypertension, and peripheral vasoconstriction have been observed (9, 21) associated with increased peripheral sympathetic nerve activity (14). Severe hypertension has been observed during breath-hold dives in cool water (15). It is likely that pulmonary hypertension also occurs. Indeed, exposure of the nasal vestibule and upper lip to cold air (2–5°C) has been observed to increase mean pulmonary arterial pressure by ~4 mmHg in mechanically ventilated intensive care unit (ICU) patients (10). A rise in pulmonary artery pressure has also been reported with facial immersion and apnea (6). Divers in cold water usually wear insulated suits, and this may help attenuate the body’s...
initial response to peripheral cold water exposure (36, 37). Nevertheless, IPE has been reported in divers wearing insulated suits (17, 24). It is therefore plausible that cooling of the face or head alone may be sufficient to augment the rise in pulmonary arterial pressures that occur during immersion (12), thus precipitating pulmonary edema.

We are aware of only one published study of the effect of head or face immersion on pulmonary vascular pressures, in which only two subjects were studied during apnea (6). If respiration is maintained, the normal bradycardic response to facial immersion is attenuated (9). Whether any pulmonary vascular effects might also be attenuated is unknown. We therefore tested the hypothesis that during prone immersion, selective exposure of the head to cold water would increase pulmonary arterial and pulmonary capillary pressures independently of body exposure. This could provide a possible mechanism for IPE, as well as suggest prophylactic measures such as insulation of the head when swimming or diving in cold water.

MATERIALS AND METHODS

Subject selection. After institutional approval and informed consent, 10 volunteer subjects were studied. Screening before the experimental day included a medical history, physical exam, 12-lead ECG, posteroanterior and lateral chest radiograph, measurement of vital capacity, forced expiratory volume in 1 s (FEV1), maximal midexpiratory flow (FEF25–75), body composition, aerobic capacity [maximal oxygen uptake (VO2max)], and hypercapnic ventilatory response (HVR) (11). VO2max < 30 ml·kg⁻¹·min⁻¹, FEV1/forced vital capacity (FVC) < 0.75, pregnancy, or an estimated body fat >3% higher than the age- and sex-based upper limits (men: <35 yr = 25%, ≥35 yr = 28%; women: <35 yr = 38%, ≥35 yr = 41%) (26) were exclusion criteria for the study. The aerobic fitness minimum and body composition thresholds were established so that the subject pool might reasonably model U.S. Navy divers.

Chamber and conditions. The experiment was conducted in a small water-filled pool (volume 4.42 m³), using an electronically braked ergometer as previously described (11).

Subjects were upright for dry exercise and prone for immersed exercise as seen in Fig. 1A. Air temperature inside the chamber was maintained at a comfortable level (22.7–27.6°C). Submersion trials were conducted in both cold (18–20°C) and warm water (29–31°C) with similar head tent temperatures. All trials were performed in the chamber at surface atmospheric pressure, typically 750 mmHg.

Equipment. A modified head tent made in-house from a standard oxygen treatment hood (model 8892, Amron International, Vista, CA) was used to control the temperature of the water surrounding the head. A latex neck dam provided a seal, such that the head and upper neck

Fig. 1. A: schematic representation of the experimental setup for the rest and exercise immersed trials. Delta chamber was pressurized to ΔP (~50 cmH₂O) to maintain zero transrespiratory pressure. B and C: schematic representation of the experimental setup for the rapid immersion trials. In B, the subject is suspended in the prone position above the pool for data collection before immersion, with transducers maintained at the midchest level. In C, the subject is completely immersed in the prone position for data collection, and transducers are at the level of the water surface. 1 ATA, 1 atm absolute.
HEMODYNAMIC EFFECTS OF HEAD AND BODY COOLING

of each subject could be bathed in water at a temperature different from the pool water. Respiratory hoses (1-in. ID, WE Collins, Bradenton, MA) were connected to the two ports in the base of the head tent (Fig. 1A), through which water was circulated from either cold or warm reservoirs (in 75-liter insulated containers). Submersible aquarium pumps (Lifeguard Quiet One 2200, Pentair Aquatics, El Monte, CA) were used to pump the water through the hoses continuously. Head tent and pool temperatures were monitored constantly using temperature probes placed inside the head tent and in the pool. Water supplying the head tent was adjusted as necessary to maintain head tent temperature within \( \pm 2°C \) of target. The head tent temperature was recorded throughout each rest and exercise trial, and the average value was used. Subjects wore swim goggles to facilitate viewing of the speed dial during exercise.

During the experiment, gases were supplied to subjects via a mouthpiece and breathing circuit, which entered the head tent through a penetrator. Breathing circuit pressure, tidal volume, and breathing frequency were recorded (11). All subjects breathed air for all trials (21% \( \text{O}_2 \), or 0.21 atm absolute (ATA) \( \text{PO}_2 \)). To assess the transient immersion response, subjects were placed in a wire basket-type stretcher (JSA-300, Junkin, Louisville, KY) lined with a PVC vinyl sheet for comfort. This basket was suspended from the ceiling of the chamber using nylon cable and pulleys, as seen in Fig. 1B. The subject, wearing a 4.5-kg weight belt, was then lowered into the water with a hand crank. With a nose clip in place, subjects breathed from a scuba regulator (XTX Regulator, Apeks Marine Equipment, Blackburn, Lancashire, UK). Apart from eye goggles, the face and head were exposed to the water.

Monitoring and data collection were as previously described (11). ECG, arterial pressure, pulmonary arterial pressure, and central venous pressure (CVP) were continuously recorded, with intermittent pulmonary arterial wedge pressure (PAWP) measurement. MAP, mean pulmonary arterial pressure (MPAP), and mean CVP were obtained using digital averaging.

Instrumentation. At the start of the experiment, a radial artery catheter (20 gauge, Arrow International, Reading, PA) and, via an antecubital vein, pulmonary artery (PA) catheter (model 131HF7 standard 4-lumen monitoring catheter, Edwards Lifesciences, Irvine, CA) were inserted. PA catheter placement was facilitated by intermittent radiographic imaging, which confirmed that the tip was in a pulmonary artery.

Procedure. Each subject was studied at rest and during exercise under the following conditions: 1) dry; or submerged \( \sim 50 \) cm in 2) cold water with cold water in the head tent, 3) cold water with warm head tent water, 4) warm water with warm head tent water, or 5) warm water with cold head tent water. Rapid immersion trials were also performed in both cold and warm water.

Each rest and exercise trial consisted of 6 min of resting measurements followed by 6 min of exercise measurements. Exercise levels were set at 150 W for dry exercise and 100 W for underwater exercise, to account for increased resistance due to leg movement in water. For the resting portion of each trial, expired gas was collected during the third to sixth minute. Arterial (from an indwelling catheter in a radial artery) and mixed venous (from an indwelling pulmonary artery catheter) blood samples were collected anaerobically over a 15- to 20-s period during the sixth minute of rest.

Expired gas was collected during the fifth minute (bag 1) and sixth minute (bag 2) of each exercise period. Arterial and mixed venous blood samples were collected anaerobically in heparinized glass syringes over a 15- to 20-s period during the sixth minute. Values from the fifth and sixth minutes were compared to ensure that the subject was in steady state. Blood gas and expired gas values from the sixth minute of exercise were used in all analyses.

Before each underwater resting period, the temperature of the head tent water was set, and hemodynamic measurements (HR, MAP, MPAP, PAWP, and CVP) were obtained during transition from either warm to cold or cold to warm. The change in temperature typically occurred over 30–60 s, and measurements were taken for the 45–60 s preceding the initiation of the transition and again for 45–60 s once the head tent temperature reached its new goal. When this change was performed after an exercise period, the subject was allowed several minutes to return to baseline resting heart rate (HR). Data for all four possible transitions (warm to cold head tent and cold to warm in both warm and cold pools) were available for 3 of the 10 subjects, with partially complete data reported for the remaining 7 subjects. Most of the missing data points were PAWP measurements, as it was not always possible to obtain a reliable measurement within the time frame of the transition. We decided not to repeat any of these measurements in the interest of reducing subject discomfort as much as possible.

One rapid immersion trial was done for each water temperature (cold and warm). For these, the subject was in the face-down prone position in the stretcher. Each trial began with cardiovascular data collection (HR, MAP, MPAP, PAWP, CVP) for approximately 45–60 s while the subject was in the air above the water (Fig. 1B). The subject was then lowered into the water, and continued data collection took place for the 60 s following complete immersion (Fig. 1C). Cardiac output could not be measured during either head tent transitions or rapid immersion because of the lack of steady state.

For logistical reasons, trials involving cold water in the pool were always performed before those with warm pool water. However, each subject was given time to rest and warm after the cold pool trials, and all were returned to their baseline core temperatures by the start of the warm water trials. Head tent water temperatures were randomized.

Measurements. Pressure transducers (Hospira, Lake Forest, IL) were positioned 5 cm caudal to the sternal angle during upright dry rest and exercise and at water surface level for submerged activities. During transition from dry to immersed the pressure transducers were maintained at the level of the subject’s midaxillary line until immersion, after which they were maintained at the water surface level. The pressure of the chamber containing the breathing gases (Fig. 1A) was maintained equal to the hydrostatic pressure at the subject’s midchest level. Core body temperature was monitored by the pulmonary artery catheter and recorded during the experiment. At the end of each experiment the digital output from the PA catheter was calibrated in water against an analytical thermistor.

Samples of arterial and mixed venous blood (4–7 ml) were drawn and kept on ice until analysis (10 min or less) (11). Mixed expired \( \text{O}_2 \) and \( \text{CO}_2 \) concentrations were measured using mass spectrometry and confirmed using gas chromatography (GC) (model 3800, Varian, Palo Alto, CA) (11).

Calibrations. Before each experimental run, the pressure transducers, mass spectrometer, gasometer, blood gas analyzer, CO-oximeter, and gas chromatograph were all calibrated using an anaerobic gauge, which had been previously calibrated against a mercury manometer.

Calculations. Tidal volume (VT) was calculated using measurements of minute ventilation (\( \text{V}_\text{E} \)) and ventilatory frequency (f) and was converted to BTPS. Oxygen consumption (\( \text{VO}_2 \)) and \( \text{CO}_2 \) elimination (\( \text{VO}_2 \)) rates were determined from standard equations using GC measurements. Arterial and venous \( \text{pH} \), \( \text{PO}_2 \), and \( \text{PCO}_2 \) were corrected for body temperature using algorithms approved by the National Committee for Clinical Laboratory Standards. The measured dead space heparin volume (0.3 ml) was corrected for in arterial and venous hemoglobin and hematocrit values.

Statistics. Data are presented as means \pm SD. The analysis of the cardiovascular and respiratory effects of warm and cold water on the head and body was accomplished using two-way ANOVA (JMP 7, SAS Institute, Cary, NC). The effect of exercise was analyzed with one-way ANOVA. Parameters found significantly different between exposure condition (dry, cold body/cold head, etc.) were analyzed by pairwise comparison using the Student’s \( t \)-test. For the head tent transition data, the changes in the various cardiovascular parameters during each head tent temperature change were compared using two-factor ANOVA. The cardiovascular changes were also compared
for the rapid immersion data (by subtracting values obtained before immersion from those obtained after), and a paired t-test was used for analysis. The significance level for all these tests was set at $\alpha = 0.05$.

RESULTS

Subjects. Ten subjects completed the study. Their characteristics are seen in Table 1. Two of these subjects were unable to complete all the cold pool trials, one due to a noseclip falling off during exercise and the other because of excessive shivering and discomfort. Shivering during cold water immersion occurred in 9 of the 10 subjects. Mean baseline core temperature was 36.8°C and at the end of cold water exposure it was 36.2°C. None of the subjects exhibited any signs of pulmonary edema during any of the trials.

Cardiovascular parameters during rest and exercise trials. Wave forms appropriate to catheter location (arterial, pulmonary artery, CVP) were obtained from all subjects. PAWP could be obtained for the vast majority of experimental runs. Hemodynamic data are depicted in Figs. 2 and 3. Exercise induced a statistically significant increase in all hemodynamic parameters ($P \leq 0.02$) except for CVP, which did not change. Overall, there was a statistically significant effect of immersion on all hemodynamic parameters ($P \leq 0.0009$) except HR ($P = 0.6$). Specifically during exercise, there was an effect of immersion on MPAP, PAWP, and CVP ($P \leq 0.04$) but no effect on HR, MAP, or CO.

Resting HR, MAP, MPAP, PAWP, CVP, and CO were all higher during the cold vs. warm pool trials ($P = 0.004$). MPAP and CVP remained significantly higher in cold water during exercise than in warm water ($P = 0.02$). There was no significant effect of head tent temperature on any of these cardiovascular parameters. The highest individual mean MPAP and PAWP values during cold/cold were 37 and 20 mmHg, respectively. These values during cold/warm were 36 and 28 mmHg. During warm/warm the highest individual MPAP and PAWP values were 31 and 19 mmHg, and during warm/cold they were 31 and 18 mmHg, respectively. All of these maximum values occurred during exercise trials. We examined the relationship between baseline PAWP in the dry and PAWP during rest and exercise while immersed. In neither cold nor warm water was there a correlation.

Systemic vascular resistance (SVR) decreased with immersion and was significantly lower in cold pool water compared with warm ($P = 0.003$). During periods of dry rest the average SVR was 1,760 ± 640 dyn·s·cm⁻⁵. In cold pool water it was 863 ± 193 dyn·s·cm⁻⁵ with cold head tent water and 925 ± 159 dyn·s·cm⁻⁵ with warm. In warm pool water the SVR was 1,167 ± 398 dyn·s·cm⁻⁵ with warm head tent water and 1,085 ± 274 dyn·s·cm⁻⁵ with cold. There was no significant difference existed between conditions during exercise for HR or CO.

Respiratory parameters during rest and exercise trials. Mean values for breathing rate (f), VT, VO₂, VE, VCO₂, end-tidal PCO₂ (PETCO₂), and dead space-to-tidal volume ratio (VD/VT) are shown in Table 2. The resting values for f, VE, VO₂, and VCO₂ were 25.3 ± 0.4, 17.6 ± 0.3, 25.7 ± 0.4, and 12.5 ± 0.3, respectively. During exercise, there was a statistically significant increase in all of these respiratory variables ($P \leq 0.003$) except HR or CO.

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Sex</th>
<th>Age, yr</th>
<th>Ht, cm</th>
<th>Weight, kg</th>
<th>Body Fat, %</th>
<th>VO₂max, ml·kg⁻¹·min⁻¹</th>
<th>HR, slope (1·min⁻¹·Torr⁻¹)</th>
<th>FVC, liters</th>
<th>Order</th>
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<tr>
<td>1</td>
<td>M</td>
<td>33.7</td>
<td>191</td>
<td>99.5</td>
<td>17.3</td>
<td>43.6</td>
<td>1.31</td>
<td>6.7</td>
<td>C-W-W-C</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
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<td>175</td>
<td>69.7</td>
<td>14.4</td>
<td>37.4</td>
<td>0.95</td>
<td>5.7</td>
<td>C-W-W-C</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>40.1</td>
<td>173</td>
<td>82.8</td>
<td>16.5</td>
<td>35.5</td>
<td>1.02</td>
<td>4.4</td>
<td>W-C-C-W</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>33.2</td>
<td>183</td>
<td>90.8</td>
<td>16.9</td>
<td>37.9</td>
<td>1.09</td>
<td>5.9</td>
<td>C-W-C-W</td>
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<tr>
<td>5</td>
<td>M</td>
<td>25.4</td>
<td>168</td>
<td>78.4</td>
<td>21.9</td>
<td>50.3</td>
<td>1.36</td>
<td>5.4</td>
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<td>6</td>
<td>M</td>
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<tr>
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<td>49.7</td>
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<td>6.8</td>
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<tr>
<td>8</td>
<td>F</td>
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<td>68.3</td>
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<td>9</td>
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<tr>
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<td>M</td>
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<td>113.0</td>
<td>17.0</td>
<td>43.7 ± 6.4</td>
<td>1.13 ± 0.19</td>
<td>6.0 ± 0.8</td>
<td>C-W-C-W</td>
</tr>
</tbody>
</table>

VO₂max, maximal O₂ uptake; HVR, hypercapnic ventilatory response; FVC, forced vital capacity; M, men; F, women. The order of head tent temperature exposures is shown using C (cold, 18-20°C) or W (warm, 29–31°C).

J Appl Physiol • VOL 106 • FEBRUARY 2009 • www.jap.org
were all higher during cold pool trials compared with warm ($P \leq 0.01$). Resting $V_{D}/V_{T}$ was lower during immersion than in the dry ($P < 0.009$); it was lower in cold pool trials vs. warm ($P = 0.0006$). These differences were not present during exercise, where no significant effect of pool temperature was found. There was a significant effect of head temperature on resting $V_{E}$, which was higher during warm and cold pool trials with cold water in the head tent vs. warm ($P = 0.02$). Head tent temperature had no effect on $V_{O_{2}}$, $V_{E}$, $V_{T}$, $V_{O_{2}}$, $V_{C_{O_{2}}}$, and $P_{ETCO_{2}}$, all increased with exercise ($P \leq 0.01$). $V_{D}/V_{T}$ decreased with exercise ($P < 0.0001$).

**Blood gas values during rest and exercise trials.** Mean arterial and mixed venous values for $pH$, $P_{O_{2}}$, $P_{C_{O_{2}}}$, and oxygen content ($Ca_{O_{2}}$, $C_{v}O_{2}$) are shown in Table 2. Arterial and mixed venous mean $pH$ values were lower during all cold vs. warm pool trials ($P \leq 0.02$), with the difference more marked when exercising. Resting trials in the cold pool showed a lower mixed venous $P_{O_{2}}$ and $O_{2}$ content when compared with warm pool values, and these differences were not apparent during exercise. There were no significant differences in blood gas values attributable to head tent temperature. Arterial and mixed venous $pH$ values decreased with exercise for all conditions ($P < 0.0001$). Arterial and mixed venous $P_{O_{2}}$ values also decreased with exercise ($P \leq 0.02$), while arterial and mixed venous $P_{C_{O_{2}}}$ values increased ($P < 0.0001$). Mixed venous oxygen content decreased with exercise ($P < 0.0001$).

Transitions. Changes in HR, MAP, MPAP, PAWP, and CVP during each head tent water temperature transition are shown in Fig. 4. When changing the head tent temperature from warm to cold, average MAP values increased 5–6 mmHg ($P < 0.0001$) and MAP increased by an average of 1 mmHg ($P = 0.02$). Head tent transitions had no statistically significant effect on any of the other hemodynamic parameters.

Rapid immersion. Subject data for MAP, MPAP, PAWP, and CVP after rapid immersion are shown in Fig. 5. After immersion in cold water, mean HR increased from 79 ± 13.1 beats/min, dry, to 94 ± 25.8 beats/min, immersed ($P = 0.02$). After immersion in warm water, there was no significant change in HR (75 ± 8.4 beats/min, dry; 74 ± 11.0 beats/min, immersed). Cold water immersion also caused significant increases in MAP (101 ± 10.3 mmHg, dry; 125 ± 13.0 mmHg, immersed) and MPAP (16 ± 5.6 mmHg, dry; and 22 ± 3.4 mmHg, immersed). Neither cold nor warm rapid immersion had any significant effect on any of the other parameters.

There were greater increases in HR, MAP, and MPAP immediately after cold water immersion compared with warm ($P \leq 0.04$). Rapid immersion in warm water caused only small changes in HR and measured pressures. However, immersion in cold water caused relatively large increases in average MAP and MPAP. Average MAP increased 24 mmHg in cold water vs. no change in warm; MPAP increased 6 mmHg in cold water vs. 2 mmHg in warm. After rapid immersion, systolic PAP values were transiently as high as 91 mmHg in cold water vs. 55 mmHg in warm water (Fig. 6). Average peak systolic PAP was 53.7 ± 14.3 mmHg after cold immersion vs. 39.8 ± 8.7 mmHg after warm immersion ($P = 0.009$).

**DISCUSSION**

Many reported cases of IPE have implicated capillary stress failure caused by high pulmonary vascular pressures (1, 24, 34, 35). Hemoptysis often occurs (24), and bronchoalveolar lavage...
of five cases has revealed a high protein content implicating breakdown of the blood-gas barrier (27).

While none of the subjects in this study developed cough, dyspnea, or other manifestations of pulmonary edema, in several subjects PAWP (an estimate of left atrial pressure) reached levels where a capillary leak is likely to occur. Interstitial edema can occur with left atrial pressures in the range of 18–25 mmHg; alveolar flooding is likely at higher left atrial pressures (16, 38). The fact that some can swim in water at temperatures without IPE attests to the resilience of the capillary barrier. The prevalence of IPE has been estimated at 1.8% in young (18–19 yr old) healthy fitness trainees during swimming trials greater than 2 km, in water temperatures similar to those used in this study (1). The lack of evidence of pulmonary edema in the present study is likely due to the brief exposures to cold water and exercise and small sample size.

In this study we observed independent effects of immersion, cold temperature, and exercise on pulmonary vascular pressures. Immersion by itself causes redistribution of blood from the periphery and a rise in central vascular pressures (2). A study of skin surface cooling observed that cooling caused an increase in MAP, MPAP, PAWP, and CVP (41). While we are unaware of any studies of the effect of water temperature on pulmonary vascular pressures, it has been reported that cold augments the reduction in vital capacity caused by immersion in warm water (25), consistent with active peripheral vasoconstriction augmenting central translocation of blood from the periphery due to immersion.

Published case series suggest that IPE is more likely in cold water (24). Our results provide a mechanism for this by demonstrating an accentuated rise in MAP, MPAP, PAWP, and CVP with cold water. The effect was significant for MPAP and CVP during both rest and exercise. High resting pulmonary and systemic pressures were also observed during rapid immersion in cold water (Figs. 5 and 6). It has been observed that fluid loading may be a factor contributing to IPE (29, 39), but we did not observe a correlation between PAWP while immersed and baseline PAWP resting in the dry condition.

In this study, selective exposure of the trunk and limbs to cold water increased MAP, MPAP, PAWP, and CVP independently of the temperature of the water bathing the head. Rapid cooling of the head caused a transient rise in MAP and MPAP, but otherwise had no significant effect on pulmonary or systemic pressures. The hemodynamic changes we observed were consistent with redistribution of blood from the periphery due to immersion and active peripheral vasoconstriction. Since the observed changes occurred very rapidly, we feel these are most consistent with local vasoconstrictive effects of cold. Since there was some degree of core cooling, we cannot exclude the possibility that thermoregulatory mechanisms may have contributed.

HR and CO would be expected to decrease with cold water head immersion (the diving reflex) (4, 5). Bergman et al. (4) observed an average decrease of 24 beats/min at rest and even greater decreases during dynamic (bicycle) exercise. One common feature of these studies is apnea during cold water facial immersion trials. In studies where the subjects were breathing during facial immersion, either a smaller decrease in HR (8, 20, 32) or no significant change has been observed (6, 18). Transtition from head-out immersion to total submersion also resulted in no significant HR change (33). Kawakami et al. (21) observed an immediate HR decrease during facial immersion with normal breathing, but after ~25 s the HR increased toward baseline. It has been hypothesized that the diving reflex may cause activation of both parasympathetic and sympathetic activity, and the latter may outlast the former (31). A consideration is that our measurements were all obtained at least 5

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### Table 2. Rest and exercise trials results

<table>
<thead>
<tr>
<th>Table 2. Rest and exercise trials results</th>
<th>Body Temperature/Head Temperature</th>
<th>Respiratory</th>
<th>Arterial blood gas</th>
<th>Mixed venous blood gas</th>
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<tr>
<td>f</td>
<td>13 ± 3.9</td>
<td>26 ± 6.9</td>
<td>19 ± 8.4</td>
<td>25 ± 4.8</td>
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<td>0.99 ± 0.13</td>
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<tr>
<td>Ve</td>
<td>13 ± 2.5</td>
<td>70 ± 15.9</td>
<td>33 ± 8.0</td>
<td>72 ± 7.2</td>
</tr>
<tr>
<td>VCO2</td>
<td>0.29 ± 0.07</td>
<td>2.39 ± 0.35</td>
<td>0.85 ± 0.17</td>
<td>2.40 ± 0.23</td>
</tr>
<tr>
<td>PtcO2</td>
<td>33 ± 1.7</td>
<td>38 ± 5.0</td>
<td>29 ± 6.2</td>
<td>33 ± 7.8</td>
</tr>
<tr>
<td>Vd/Vt</td>
<td>0.40 ± 0.11</td>
<td>0.0083 ± 0.22</td>
<td>0.19 ± 0.10</td>
<td>0.029 ± 0.09</td>
</tr>
<tr>
<td>pH</td>
<td>7.44 ± 0.03</td>
<td>7.33 ± 0.05</td>
<td>7.44 ± 0.05</td>
<td>7.31 ± 0.06</td>
</tr>
<tr>
<td>PO2</td>
<td>106 ± 15</td>
<td>102 ± 12</td>
<td>105 ± 34</td>
<td>105 ± 10</td>
</tr>
<tr>
<td>PCO2</td>
<td>33 ± 4</td>
<td>32 ± 8.8</td>
<td>29 ± 5.5</td>
<td>31 ± 4</td>
</tr>
<tr>
<td>CaO2</td>
<td>197 ± 20.7</td>
<td>199 ± 25.8</td>
<td>195 ± 15.2</td>
<td>200 ± 14.7</td>
</tr>
<tr>
<td>pH</td>
<td>7.36 ± 0.02</td>
<td>7.21 ± 0.06</td>
<td>7.37 ± 0.04</td>
<td>7.20 ± 0.08</td>
</tr>
<tr>
<td>PO2</td>
<td>31 ± 3</td>
<td>21 ± 3.3</td>
<td>25 ± 6</td>
<td>20 ± 3</td>
</tr>
<tr>
<td>PCO2</td>
<td>37 ± 6</td>
<td>54 ± 6.6</td>
<td>36 ± 5.6</td>
<td>53 ± 8</td>
</tr>
<tr>
<td>CaO2</td>
<td>126 ± 16.7</td>
<td>62 ± 13.4</td>
<td>100 ± 23.5</td>
<td>60 ± 11.7</td>
</tr>
<tr>
<td>Vd/Vt</td>
<td>0.40 ± 0.11</td>
<td>0.0083 ± 0.22</td>
<td>0.19 ± 0.10</td>
<td>0.029 ± 0.09</td>
</tr>
</tbody>
</table>

Values are means ± SD. Rest, resting values; Ex, exercising values; f, respiratory rate (min⁻¹); Vt, tidal volume (liters); VO2, oxygen consumption (l/min STPD); Ve, minute ventilation (min⁻¹ BTPS); VCO2, CO2 elimination (l/min STPD); PtcO2, end-tidal PO2 (mmHg); Vd/Vt, dead space-to-tidal volume ratio; CaO2, arterial oxygen content (ml/l); Vd/Vt, mixed venous oxygen content (ml/l). PO2 and PCO2 values are in mmHg.

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J Appl Physiol • VOL 106 • FEBRUARY 2009 • www.jap.org
min after head cooling. However, we did not observe even transient bradycardia after facial cooling.

Rapid total-body immersion in cold water increased HR, MAP, and pulmonary arterial pressure to a greater degree than transient bradycardia after facial cooling. However, we did not observe even transient bradycardia after facial cooling.

Fig. 4. HR, MAP, MPAP, PAWP, and CVP changes during head tent temperature transitions. Individual observations and mean values are shown. Head tent temperature is designated C (cold, 18–20°C) or W (warm, 29–31°C). *Statistically significant changes that occurred with transitions (MAP, $P < 0.0001$; MPAP, $P = 0.02$).

Fig. 5. MAP, MPAP, PAWP, and CVP values during rapid immersion in cold (18–20°C) and warm (29–31°C) water. Individual observations and mean values are shown. *Statistically significant changes (MAP, $P < 0.0001$; MPAP, $P = 0.04$).
sudden immersion in warm water. This increase in HR has been observed by others (22). Ice-cold showers of water on the chest and abdomen also increase HR, as well as peripheral arterial pressures (23). The increased peripheral arterial pressures could be expected with peripheral sympathetic activation due to cold sensation or the diving reflex, but bradycardia would be expected immediately after rapid immersion if the diving reflex were playing a major role in these hemodynamic changes. In our study we calculated SVR, which decreased in cold water. Our results appear to conflict with those of Wilmshurst et al. (40), who observed an increase in forearm vascular resistance with application of cold towels to the head and neck. Our observation of decreased calculated SVR probably does not reflect active vasodilatation but rather increased CO and CVP due to translocation of blood from the periphery.

When transitioning from warm water bathing the head to cold, mean arterial pressures and pulmonary arterial pressures exhibited a small but significant increase, but there was no change when transitioning from cold head tent water to warm. Since the volume of blood in the soft tissues of the head and neck is small, this rise in pulmonary and systemic pressures is probably not due to central redistribution of blood but is rather reflexive in nature. These observations are consistent with the dive reflex playing a small role in increasing both pulmonary arterial and mean arterial pressures. However, the temperature of the water bathing the head did not have any significant effect on the steady-state cardiovascular parameters during rest or exercise. Bodily immersion in cold water was the main factor driving the increases in pulmonary pressures seen in this study. Presumably, insulation of the trunk and extremities would attenuate these changes in pulmonary vascular pressures. However, immersion even in thermoneutral water did produce an increase in MPAP, PAWP, and CVP. Moreover, thermal insulation is not uniformly effective. This might explain the occurrence of IPE in swimmers or divers in warm water, or even those wearing insulated suits in cold water.

\[ V_{O2} / V_{T} \] decreased during immersion and was lower in cold pool trials compared with warm. This is consistent with more uniform perfusion of the lung during immersion, due to redistribution of blood from the extremities. We speculate that \[ V_{O2} / V_{T} \] was lower in cold water compared with warm because of enhanced blood redistribution due to active peripheral vasoconstriction in the cold. This is consistent with the observations of Kurss et al. (25), where there was indirect evidence for greater pulmonary vascular engorgement for cold vs. thermoneutral water.

There are limitations to this study. In particular, only two ranges of water temperature were studied. It is possible that more extreme selective cooling of the face and head could induce larger changes in pulmonary artery pressure. However, studies of the hemodynamic effects of facial immersion published thus far have shown significant effects on HR at water temperatures of 25°C and 35°C (3, 15). In a study of facial cooling, systematic evaluation of water temperatures (0–35°C) failed to show any temperature effect on HR (19). Another shortcoming is the fact that not all subjects completed all of the head temperature transitions. However, the small magnitude of the effects observed suggests that a larger data set would not have materially altered the results. The absolute values of MPAP, CVP, and PAWP in the upright positions are dependent on the accuracy with which the vertical position of the atria can be estimated. Using the arbitrary position 5 cm caudad to the sternal angle may have produced pressure measurements that were higher or lower than the true values. However, relative changes would have been accurately recorded. We confined our study to prone immersion. This is arguably the most relevant to swimmers and divers as this is their position. We cannot exclude positional effects, in particular changes in blood volume distribution in the air-containing thorax, as suggested by Mahon et al. (30) and Lund et al. (29). We also cannot exclude the possibility that pulmonary arterial and pulmonary capillary hypertension might have been even higher after more prolonged exercise. Indeed, after 15–20 min of immersed exercise, changes in respiratory
pattern have been observed that are consistent with the onset of metabolic acidosis (42). Acidosis could contribute to pulmonary hypertension.

In summary, this study examined the individual effects of head and trunk/limb cooling on the rise in pulmonary and systemic pressures during immersed prone rest and exercise at 1 ATA. HR, MAP, MPAP, PAWP, and CO all increased with exercise. MPAP and CVP in this study were significantly higher during exercise in cold water rather than in warm. The observations indicate that the major effect of cold water in this regard is on the trunk and limbs, with possible reflexive effects of head and face cooling contributing only to a minor degree. In situations where IPE is more common, such as naval combat swimmer training, use of improved head insulation is unlikely to offer any benefit for IPE prevention, although trunk and extremity insulation is likely to be at least somewhat effective. We conclude that bodily cold water immersion and exercise both increase mean arterial, pulmonary arterial, and pulmonary artery wedge pressures during prone immersion and have additive effects.

**REFERENCES**


