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

The underwater environment: cardiopulmonary, thermal, and energetic demands

D. R. Pendergast[1,2] and C. E. G. Lundgren[1,2]

[1]*Center for Research and Education in Special Environments and* [2]*Department of Physiology and Biophysics, University at Buffalo, Buffalo, New York*

Pendergast DR, Lundgren CE. The underwater environment: cardiopulmonary, thermal, and energetic demands. *J Appl Physiol* 106: 276–283, 2009. First published November 26, 2008; doi:10.1152/japplphysiol.90984.2008.—Water covers over 75% of the earth, has a wide variety of depths and temperatures, and holds a great deal of the earth's resources. The challenges of the underwater environment are underappreciated and more short term compared with those of space travel. Immersion in water alters the cardio-endocrine-renal axis as there is an immediate translocation of blood to the heart and a slower autotransfusion of fluid from the cells to the vascular compartment. Both of these changes result in an increase in stroke volume and cardiac output. The stretch of the atrium and transient increase in blood pressure cause both endocrine and autonomic changes, which in the short term return plasma volume to control levels and decrease total peripheral resistance and thus regulate blood pressure. The reduced sympathetic nerve activity has effects on arteriolar resistance, resulting in hyperperfusion of some tissues, which for specific tissues is time dependent. The increased central blood volume results in increased pulmonary artery pressure and a decline in vital capacity. The effect of increased hydrostatic pressure due to the depth of submersion does not affect stroke volume; however, a bradycardia results in decreased cardiac output, which is further reduced during breath holding. Hydrostatic compression, however, leads to elastic loading of the chest wall and negative pressure breathing. The depth-dependent increased work of breathing leads to augmented respiratory muscle blood flow. The blood flow is increased to all lung zones with some improvement in the ventilation-perfusion relationship. The cardiac-renal responses are time dependent; however, the increased stroke volume and cardiac output are, during head-out immersion, sustained for at least hours. Changes in water temperature do not affect resting cardiac output; however, maximal cardiac output is reduced, as is peripheral blood flow, which results in reduced maximal exercise performance. In the cold, maximal cardiac output is reduced and skin and muscle are vasoconstricted, resulting in a further reduction in exercise capacity.

respiratory; renal; immersion; exercise; aging; sex differences; submersion; pressure; energy cost

THE UNDERWATER ENVIRONMENT is unique, and while it is a magical place with great history and beauty and plant and animal life and holds a wealth of resources, it also imposes pronounced physiological stresses on humans and animals. This brief review offers an overview of how the major environmental challenges, i.e., the pressure, temperature extremes, and the unbreathable ambient medium, of the "Silent World" affect circulation, renal system and water balance, breathing, exercise, and thermal balance and how it imposes some threats due to pharmacological or toxic effects of respired gases at high pressure, i.e., great depths. Adaptations to the strains caused by the underwater environment are accomplished in some cases physiologically, while others require an artificially created microenvironment (breathing gear, diving suit, etc.), and in extreme cases adjustment cannot be achieved, and injury and even death may result. The effects imposed by water immersion, per se, and the effects of depth of submersion in humans strain the cardiovascular and urinary and respiratory systems and result in changes in systemic and regional exchange of respired gases. As most diving lasts for hours, not weeks or months like spaceflight, this review will focus on the more short-term responses that are relevant to diving.

Address for reprint requests and other correspondence: D. R. Pendergast, Center for Research and Education in Special Environments, 124 Sherman Hall, Univ. at Buffalo, Buffalo, NY 14214 (e-mail: dpenderg@buffalo.edu).

CARDIOVASCULAR FUNCTION

Head-out immersion of humans in thermoneutral water (34 or 35°C for 3–6 h) causes an immediate translocation of blood

from the dependent limbs and an increase in intrathoracic blood volume (1, 31, 41) that augments the cardiac output via increased end-diastolic and stroke volume due to the effect of increased cardiac muscle length on cardiac muscle contractile force. The stretching of the atrium also results in a compensatory diuresis.

Gauer and Henry (36) provided the first evidence that stretching of the cardiac atria causes a reflex diuresis, thus showing that blood volume is regulated by intrathoracic stretch receptors via a cardiac-renal link. They (36) postulated that the increased fluid output by the kidneys in immersion would reduce plasma volume and thus return stroke volume and cardiac output to normal. However, this view has been modified, since a major transcapillary fluid shift from extravascular compartments into the blood results, initially, in plasma volume expansion, which in turn is corrected by the diuresis returning plasma volume to normal. External hydrostatic pressure decreases the capacity of the venous compartment, primarily the splanchnic veins, shifting blood into the chest (49). In addition, the autotransfusion, derived primarily from the capillaries of the legs, augments plasma volume and reduces plasma oncotic pressure (48). The fluid shift is derived from the intracellular compartment, and the role of the kidney in short-term immersion is to minimize the increase in plasma volume that would otherwise result in a much greater increase in cardiac output (54, 64, 65). Plasma volume increases initially ~7% during immersion, as tissue pressure rises above estimated capillary hydrostatic pressure favoring capillary reabsorption. As immersion depth increases, there is a linear coupling between tissue pressure, capillary pressure, and central venous pressure. While plasma osmolarity does not change, there is a decline of plasma oncotic pressure resulting from hemodilution. Interstitial fluid volume and lymph flow do not change, and amino acids and K^+ rise in the plasma, indicating that most of the fluid is shifted out of the intracellular compartment (54, 64). Plasma volume, stroke volume, and cardiac output remain elevated throughout 6- to 12-h immersions. These observations suggest that the intracellular reservoir of fluid is not significantly depleted during short-term immersion (47).

Cardiac output is reported to increase according to the water level of immersion in the erect posture, increasing from leg to midcervical level, and is sustained for several hours. The 32–62% cardiac output increase is due to an enlarged stroke volume, dominating over a decrease in heart rate while mean arterial pressure is unchanged, indicating that total peripheral resistance to blood flow declines (1, 54). Arterial pulse pressure increases consistently, secondary to the rise in stroke volume (69). Systemic O_2 consumption is constant in thermoneutral immersion so O_2 delivery exceeds the O_2 requirements of the tissues due to the hyperkinetic circulation. Normally, blood flow is autoregulated to meet the metabolic demands of peripheral tissues (41); however, autoregulation of blood flow is modified in immersion, resulting in increased systemic blood flow (18, 42). Blood flow to respiratory muscles and cardiac muscle is increased in response to the increased respiratory and cardiac work (42). Blood flow to the cerebellum increases, reflecting altered vestibular and proprioceptive inputs (42). The transition from air to thermoneutral or warmer water leads to heating of the integument such that blood flow to skin and subcutaneous fat rise. Renal blood flow is unchanged. However, there are increases in flows to the gastrointestinal tract, liver, pancreas, and spleen. During immersion, the visceral flows decline to preimmersion levels, and the increased cardiac output is redirected into nonrespiratory skeletal muscles (4).

The regional hyperemias may offset tendencies for fluid to shift out of cells in some tissues. As a reflection of overall increase in tissue blood flow, the whole body nitrogen elimination during oxygen breathing increases during immersion in thermoneutral (35°C) water compared with during nonimmersion (5).

Allowing for differences in body size, there do not appear to be sex differences in cardiovascular or renal responses to immersion in thermoneutral water (104). Thus the responses to immersion described above apply to both men and women (38).

The increase in cardiac output observed in young people is blunted in the elderly (76), although the natriuretic responses to immersion are augmented with increases in glomerular filtration rate (98) despite a decline in pressure-dependent sodium and water excretion with age (102). While there are differences in immersion responses with aging, old and young have similar physiological responses to body cooling, when all other factors, such as changes in metabolism and vascular control, are considered (39). The effects of sex and aging on the physiological response as a function of depth of submersion need further study.

An increase in ambient pressure whether in water or in a dry hyperbaric chamber does not affect stroke volume; however, a bradycardia results in similar decrease in cardiac output at all depths (62). During breath holding there is a further reduced cardiac output, as the bradycardia is part of the so-called diving response (61). This is also true when there is CO_2 retention in divers and breath holders, irrespective of depth (3). One might speculate that a reduced cardiac output could contribute to CO_2 retention.

RENAL FUNCTION

Immersion in a hydrated human leads to diuresis, natriuresis, kaliuresis, and an increase in free water clearance (C_{H_2O}) (28, 31). In nonreplete subjects the renal responses abate after 2 h, returning to control levels in 4 h, while in replete subjects, renal responses are partially sustained for the duration of immersion. In dehydrated subjects there is a smaller diuresis with an increase of osmolar clearance (8). The level of physical training also influences the character of the immersion diuresis. Claybaugh et al. (19) found that diuresis and natriuresis were reduced significantly in trained runners and swimmers compared with sedentary humans despite larger and more persistent increases in cardiac output. In subjects with experimentally induced high sympathetic nerve activity, cardiac output remains elevated for up to 6 h (54); longer time periods have not been systematically studied.

Immersion does not alter glomerular filtration rate but changes tubular mechanisms. Plasma osmolarity does not change in immersion; however, the plasma vasopressin level is depressed and is responsible for the increase in free-water clearance (10, 29, 35, 68). The primary mechanoreceptors regulating vasopressin secretion appear to be the arterial baroreceptors and cardiac receptors (34, 54, 91). In immersion, plasma renin activity (PRA) and aldosterone levels decline in a more consistent manner than plasma vasopressin and appear to be less related to the degree of hydration (30, 54). However, the diuretic and natriuretic responses are present after 40 min of immersion and

are too rapid in onset to be due primarily to suppression of aldosterone secretion. Distension of the atria releases atrial natriuretic peptide (ANP) that acts to elicit diuresis, natriuresis, vasodilatation, and a fluid shift out of the vascular compartment. Immersion elevates ANP in plasma rapidly (54, 74). However, there is a disassociation in the time courses of the ANP response, which rises immediately and is sustained for hours, and the renal response, which develops slowly and abates in 2–4 h (40, 64, 95). It can be concluded that ANP is only effective when plasma volume is expanded. It has been demonstrated that renal sympathetic neural activity elicits changes in kidney function in the absence of renal hemodynamic responses (26). Mechanical stretch of atrial and/or arterial baroreceptors causes striking reductions in renal sympathetic nerve activity and increases urine flow and sodium excretion (30).

The immersion diuresis (~500 ml/day), natriuresis, calcium excretion, and potassium secretion are greater and last longer at depth, particularly in a helium-oxygen atmosphere. However, glomerular filtration rate and hormone release are not affected by pressure (62, 72). There is a drop in free-water clearance, causing a fall in urine osmolarity when exposed to pressure (72), presumably reflecting the body's "attempt" to conserve plasma volume. These pressure-dependent changes last as long as several days (62, 72), but no longer-term controlled experiments have been conducted and the graded effect of pressure cannot be assessed with existing data.

RESPIRATORY FUNCTION

Except for short periods of breath holding, air breathing mammals, including humans, need to have free access to a suitable breathing gas, which is prevented by the underwater environment. Breath-holding time is based on the body's O_2 stores and tolerance to CO_2 accumulation and O_2 depletion and is limited to 1.5–2.0 min in normal individuals; however, it may approach 10 min in trained breath-holders (58). Past this time, the diver has to use breathing gear, of which the simplest version is the snorkel, which is limited in depth to just under the surface due to the inspiratory effort of the respiratory muscles to overcome the water pressure acting on the chest (15). At depth, a regulator-controlled flow of air from a high-pressure tank, called self-contained underwater breathing apparatus (scuba), or a closed-circuit rebreathing system (using special gas mixtures or pure O_2 in combination with a CO_2 scrubber) is used. The old style, which combined diving helmet and suit, supplied with gas from the surface, is also still in use, often in a modernized version. All three of these systems are used, to varying degrees, by professional, sport, and military divers. Breathing underwater is influenced by often-present increased hydrostatic pressure differences across the chest wall (static lung loading) (100), gas density and composition, and by exercise hyperpnea, all of which may cause increased work of breathing and fatiguing of the respiratory muscles (88).

The increased pulmonary blood flow at rest and during exercise results in increased pulmonary arterial pressure and vascular volume, which causes an increase in residual volume and decrease in vital capacity (61). Hydrostatic compression of the chest leads to elastic loading of the chest wall and negative pressure breathing (44, 61, 100). In addition, pulmonary mechanics are altered with the increase in the earlier mentioned pressure difference between the outside of the chest and the alveolar air (static lung load) (61). The stress of increased static lung load strains the respiratory system, which may lead to changes in end-expiratory lung volume (61, 99), placing the respiratory muscles at a less than optimal muscle length, and thus reducing their ability to generate and maintain adequate force (14, 63) to cope with the increased work of breathing. There are increases in blood flow to the respiratory muscles, reflecting their increased metabolism (42). Mild hypoxemia coincident with increased lung closing volume has been observed in humans (87). It was reported that there is increased perfusion of the apical portions of the lung with improved matching of ventilation:perfusion relationships (1). However, more recent studies have shown that blood flow is increased to all zones of the lung, with the improvement in the ventilation:perfusion relationship being less than originally thought (32, 77, 90). This suggests that the lung circulation is actively regulated during immersion, and less gravity dependent, and increased flow to capillaries is more important than recruiting new ones.

Pulmonary edema, which is not uncommon during higher intensity exercise, immersed or submersed, appears to be due to increased pulmonary blood flow and pressure, resulting in capillary engorgement with increased transmural pressures (94). Pulmonary edema has also been suggested to occur in shallow water breath-hold dives with low lung volume (59), but only in deep dives, compared with shallow dives, when the lungs are inflated (60).

Although the hydrostatic pressure is transmitted to the tissue compartments of the body comprised primarily of water, the normally closed air spaces such as middle ears and sinuses must be vented during changes in depth so as to avoid barotraumas. Similarly, too deep a breath-hold dive can lead to "lung squeeze" with hemorrhage and possibly pulmonary edema (59, 60). By contrast, as long as a diver is breathing on a diving apparatus, breathing gas is provided at essentially the same pressure as the water pressure on the chest. If, however, the diver were to ascend after inhalation of air at depth and neglects to let the expanding air in the lungs escape, the very real risk of pulmonary barotrauma and gas embolism might materialize (15). High gas pressure in the lungs is connected with increase in the partial pressures of the component gases, introducing the potential for pharmacological (nitrogen narcosis) and toxic effects (oxygen poisoning) (15).

OXYGEN BALANCE

As opposed to altitude, hypoxia is not an issue in diving with breathing gear as the increased total pressure also increases P_{O_2}. However, the elevated P_{O_2} causing tissue hyperoxia may cause toxic effects in divers (2) particularly on pulmonary (9, 22) and nervous tissues (25). In contrast to the rich spectrum of systemic reactions to low oxygen, adaptive response to hyperoxia appears limited as exposure to high P_{O_2} is an "artificial" environment created by increased pressure underwater or in a hyperbaric chamber. Peripheral vasoconstriction and a moderate decrease in cardiac output, which together moderate the rise in cellular P_{O_2} (22), appear to be the adaptations to high P_{O_2}. At the cellular level chemical reactions scavenge superoxide and associated products (22). However, the extent of these adaptations is limited, and oxygen toxicity is a potential risk of the

underwater environment (2) and other hyperoxic conditions (9, 13, 25).

CARBON DIOXIDE BALANCE

Alveolar ventilation is typically tightly regulated to maintain arterial P_{CO_2} (Pa_{CO_2}) at ~40 mmHg in normal healthy subjects (43). However, diving is conducive to CO_2 retention due to spontaneous or voluntary hypoventilation (routinely practiced by some divers to save breathing gas) or to the inability to perform the increased respiratory work secondary to weak or fatigued respiratory muscles. Diving-induced CO_2 retention may cause CO_2 narcosis (e.g., 7, 55, 103) and may secondarily enhance central nervous system oxygen toxicity and nitrogen narcosis (16, 55). It has furthermore been reported that CO_2-retaining divers may convulse while still within what is generally considered to be safe limits for hyperoxic exposure (55). High external breathing resistance in the diving apparatus may cause incapacitating CO_2 retention even without prodromal symptoms (no dyspnea) (55, 103). The mechanisms behind CO_2 retention are still unclear, but one factor involved in hypoventilation in divers may be low respiratory CO_2 sensitivity (51, 55, 92). One potential mechanism of hypoventilation is inadequate respiratory muscle performance due to poor function or fatigue, which may acutely attenuate the ventilatory responses to CO_2 and chronically lead to CO_2 retention.

Recent work has shown that specific training of the respiratory muscles can prevent the failure of respiratory function and substantially (>85%) enhance runners' and divers' exercise endurance (57, 88, 106). Studies have also shown that respiratory muscle training can improve respiratory muscle function and surface and underwater swimming performance (57, 88, 100) and "normalize" CO_2 sensitivity (82) in hypo- and hyperventilators as well as reduce respiratory muscle fatigue and CO_2 retention (88).

NITROGEN BALANCE

In addition to P_{O_2}, other gases such as N_2 may exert high pressures at depth and are taken up during compression and eventually would saturate the body at the partial pressure of that specific gas. During decompression the inert gas taken up must be eliminated, which is primarily a perfusion-dependent process and if not done properly can result in bubble formation in tissues and blood, causing decompression sickness (15). If blood perfusion could be reduced during compression and facilitated during decompression by selective changes in cardiac output, such as may be achieved by negative-pressure breathing (23), in a pressure chamber, by head down tilt (12), and immersion (5), the tissue load of nitrogen or other inert gases at the end of the dive would be reduced, which potentially would lower the risk of decompression sickness. Recent studies have shown that exercise carried out in the predive period can reduce bubble formation and decompression sickness and have related this to a potential role of nitric oxide (11, 27). Previous studies have also suggested that nitric oxide may play an important role in decreasing perfusion during cold stress (78) and increasing perfusion during exercise (46), the latter effect potentially explaining the earlier observed enhancement of N_2 elimination during immersion (5).

Changes in peripheral circulation may be accomplished by changes in water temperature (75), and warming of the body has been shown to reduce bubble formation (5) and the risk of decompression sickness (37). At higher pressures N_2 has a narcotic effect, thus limiting the depth at which air can be breathed (15). At deeper depths, nonnarcotic inert gases are therefore breathed, such as helium mixed with oxygen.

THERMAL PROBLEMS

Most of the waters of the world are well below thermoneutral (34–35°C) temperature for humans and, when combined with the high thermal conductance and capacity of water, present the danger of hypothermia. By definition, there is little effect on resting metabolism by submersion in thermoneutral water. However, as water temperature decreases and time passes, metabolism increases in proportion to the drop in skin and core temperatures (75). In waters that are above the thermoneutral temperature (warm or hot), significant risk of hyperthermia exists as the ability to eliminate heat from the body in this environment is limited. The potential physiological and performance effects of hyperthermia, particularly during exercise, are not fully understood.

Cold Water

Immersion in cold water elicits an increased translocation of blood to the chest and reductions in intracellular fluid, although the increase in plasma volume is reduced (99), resulting in similar increases in stroke volume and cardiac output (75). Cold-induced changes are mainly due to increased sympathetic nerve activity (96). Heart rate is depressed in cold submersion such that stroke volume is elevated and cardiac output is similar to what is observed in thermoneutral water. By contrast, cardiac output is typically reduced during breath-hold diving. Arterial blood pressure has been reported not to be elevated during immersion in cold water because the vasoconstriction in skin, subcutaneous tissues, and muscle is offset by vasodilatation in other areas, and total peripheral resistance is unchanged (75). Other studies have demonstrated an increase in diastolic blood pressure and afterload (13), so that cardiac output in cold water is not increased over thermal neutral water, despite the increased translocation of blood to the chest (75). In cold water, cutaneous vasoconstriction is initiated by decreased skin temperature. However, a reduction in core temperature greatly augments sympathetic activity with further vasoconstriction during sustained cold exposure and cold acclimatization (70). Nitric oxide has been shown to be a mediator of temperature regulation (93) and is depressed in expired gas during cold stress (78).

Resistance to heat flux is provided by the subcutaneous fat layer and increased peripheral vasoconstriction, in particular in acclimatized individuals (72). Acclimatization increases maximal tissue insulation by peripheral vasoconstriction and may be either centrally or locally controlled (56). Nitric oxide has been suggested to play an important role in the thermally induced vasoconstriction (93). An additional response to reduced skin and core temperatures is the initiation of shivering, generating more metabolic heat (56). Originally it was reported that reductions in skin and core temperatures resulted in vasoconstriction of the skin; however, it was subsequently shown that there is also a vasoconstriction of muscle and reduced maximal cardiac output, thus limiting exercise capacity in the

cold (75), despite the absence of reduced neural recruitment of muscle (21).

Depth has a significant effect on thermal stress as the hydrostatic pressure compresses the insulating layer of the diving suit material and increases thermal conductivity, thus increasing body heat uptake or loss (6), although the physiological responses are similar to what would be observed for similar decreases in skin and core temperature near the surface. High inspired CO_2 or CO_2 retention, often observed in divers, has been reported during cold water immersion to the neck to further reduce forearm blood flow compared with cold alone (35). Thermal responses in helium-oxygen at 11 atmospheres absolute have been reported to result in reduction in skin temperature due to increase in heat flux (62).

In addition to the physiological stress there are significant reductions in cognitive performance in cold water (24). Thus there are impacts on simple arithmetic, logical reasoning, word recall, word recognition, and manual dexterity, these changes being related to skin and core temperatures and not depth, per se (24).

During immersion in cold water there are, after correction for body fat and surface area, no sex differences in physiology or metabolic responses (101). Although studies have suggested alterations in thermal response to cold in the elderly, studies during immersion or submersion are lacking.

Warm Water

Although the strain caused by cold immersion is well studied, diving also occurs in hot water, such as encountered in some tropical areas, which poses an extreme stress, particularly during exercise, the adaptations to which are not well documented. Immersion in warm water causes an immediate drop in temperature gradient, or reversed gradient in hot water, across the skin and inability to evaporate sweat, causing a rapid increase in skin and core temperature. This condition is exacerbated when additional heat is produced by exercise. Increased sweating can lead to reductions in plasma volume (~7%), and increases in hemoglobin concentration, hematocrit, and serum proteins after 4-h dives (45). In fact a single warm-water scuba dive, independent of depth, can significantly increase hematocrit (105). When the reduced plasma volume is combined with associated vasodilatation of the skin and increased heart rate, cardiac output, and skeletal muscle blood flow, performance can be significantly decreased (13). In addition, hot water immersion causes significant decreases in total peripheral resistance (13). The vasodilatation in response to body heating in warm water is mediated by nitric oxide (95). The ability of the diver to recognize CO_2 retention is blunted by immersion in warm water (34°C) (35), while wearing a wet suit elevated anxiety during exercise (52).

Although underwater data are not available, experiments in air where there were increases in skin and core temperature may well predict what can be expected in warm or hot water. The nondiving studies have shown that muscle temperature, epinephrine, and glycogen levels increase, while exercise time is significantly shorter in hot than in thermoneutral or cold water (73). In addition, carotid baroreceptor stimulation of cutaneous vascular conductance is exaggerated in whole body heating in air (50). Muscle sympathetic nerve activity is increased during heat stress in air at rest, as is skin blood flow, heart rate, and cardiac output, while blood pressure is reduced (66, 89). Sympathetic nerve activity may play a role in both thermal and blood pressure regulation during heat stress (66). The increased sympathetic nerve activity during heat stress at rest is also evident during exercise (89). It is reasonable to speculate that the responses to increased skin and core temperature in air would be similar in water and lead to the same reductions in diver performance as seen in air.

Acclimatized individuals may be at increased risk due to increase sweating and vasodilatation, risking reduced plasma volume and exercise blood flow. The effects of sex and aging have not been investigated in warm water. These areas of underwater physiology need additional study.

EXERCISE

Moving in water is different from moving in air as the density of the fluid is significantly greater. Thus both external and internal work are increased (79, 80, 107). Drag in water is velocity dependent (80) and increases as a function of kV^n, where k is a constant, V is velocity, and n is the exponent of V, with $n = 1$ for friction between the body and water, $n = 2$ for the pressure to separate the water as the body moves through it (pressure drag), and $n = 4$ for wave generation (67). Estimating the efficiency of underwater work is complicated, compared with terrestrial work, using a traditional approach (only considering external work) and yields a very low efficiency (about 5–8%) (80); during swimming the swimmer has to accelerate water when moving through it and also perform internal work moving body parts around the body center of gravity. Once these factors are accounted for, the efficiency is similar (i.e., ~25%) during fin swimming (at the surface, and underwater) as when exercising on land (107). The implication of this is that the energy cost of movement in water increases exponentially as a function of velocity and is independent of body weight, as opposed to walking or running where it increases linearly and is weight dependent. The exponent of the rise in energy cost and drag with velocity is determined by the type of drag, which is velocity dependent, and the exponent(in kV^n) typically ranges from ~1.5 to 1.8 for surface fin swimming and from 1.2 to 1.5 for underwater fin swimming (80). Another interesting observation is that the energy cost of locomotion in water, being more technique dependent, is highly variable among individuals (i.e., ~25%), which is not the case for walking or running.

When subjects exercise in water the baseline characteristics of circulatory function are altered with increased cardiac output and stroke volume and perfusion to nonmuscle tissues, reduced heart rate, and total peripheral resistance (43). However, blood pressures are similar to those in air. Despite this different baseline, the responses of the cardiopulmonary system to increased metabolism are similar in thermoneutral water and air, after attaining an oxygen consumption of ~1.0 l/min and up to ~80% of maximal oxygen consumption ($\dot{V}O_{2max}$) (75, 85, 86). At low levels of exercise, cardiac output and stroke volume do not increase from rest as oxygen consumption increases, while muscle blood flow and ventilation do. Maximal cardiac output, heart rate, blood flow, and oxygen transport and anaerobic threshold and power output are ~15% lower during immersion than in air, even in thermoneutral water (75). During underwater swimming with fins the $\dot{V}O_{2max}$ is significantly less than

in air and surface swimming (17). In addition, the energy cost of underwater fin swimming is influenced by the types of fins worn, which in turn tends to vary with sex and the technical ability of the diver (83, 84).

Cycle exercise at various depths does not affect peak power, while maximal oxygen consumption (−9.5%) and heart rate (−7%) are minimally reduced, and maximal minute ventilation is significantly reduced (−41%) (20). These types of data for free swimming are available for near the surface as discussed above; however, technical limitations of hyperbaric chambers usually preclude data collection at depth. Maximal cardiac output appears to be conserved at depth, as urine osmolarity is increased and free water is conserved (20). Despite the conserved central cardiovascular response at depth, the increased work of breathing most likely requires a higher respiratory muscle blood flow and presumably diverts blood from exercising muscle, which would explain the reduced submaximal sustained fin swimming time (70–80% of $\dot{V}_{O_{2max}}$) observed at depth (88).

During exercise at a depth of 5 m in 18°C water at an oxygen consumption of 1.0 and 1.4 l/min, body insulation increased by 37%, while insulation provided by a wet suit was 63% of the total insulation. Therefore, physiological gains in insulation are important but often insufficient. In the study just mentioned, the wet suit did not prevent a continuous drop in core temperature (2). As previously reported for nude immersed subjects, divers' initial drop in core temperature was associated with their subcutaneous fat thickness (2). Other studies in submersed fin-swimming divers have shown that the energy cost of locomotion was 20% higher and $\dot{V}_{O_{2max}}$ 25% lower in cold water than in thermoneutral water and that this was due to a reduced net mechanical efficiency and not drag (75, 79).

SUMMARY

The stress and strain imposed by the underwater environment are unique and challenging, and in extreme cases dangerous. Future work in this area can resolve some of the physiology of the stress of inert gas uptake and elimination, the benefits and risks of high O_2 pressure, risk of elevated Pa_{CO_2}, hypothermia, and hyperthermia on control of central and peripheral circulation, risks of breath-hold diving, and exertion. Many insights to be expected from such research are likely to benefit not only diving but also basic cardiorespiratory function and the growing field of clinical hyperbaric oxygen therapy.

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