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Physiology of Exercise in the Cold

Thomas J. Doubt
Hyperbaric Environmental Adaptation Program, Naval Medical Research Institute, Bethesda, Maryland, USA

Contents

Summary

Recreational and job requirements have increased the incidence in which humans exercise in cold environments. Understanding the physiological responses while exposed to cold entails knowledge of how exercise and cold interact on metabolic, cardiopulmonary, muscle and thermal aspects of human performance. Where possible, distinctions are made between responses in cold air and cold water.

While there is no consensus for diets most appropriate for working cold exposures, the evidence is strong that adequate amounts of carbohydrate are necessary. Carbohydrate loading appears to be efficacious, as it is for other athletic endeavours.

Contrary to conventional wisdom, the combination of exercise and cold exposure does not act synergistically to enhance metabolism of fats. Free fatty acid (FFA) levels are not higher, and may be lower, with exercise in cold air or water when compared to corresponding warmer conditions. Glycerol, a good indicator of lipid mobilisation, is likewise reduced in the cold, suggesting impaired mobilisation from adipose tissue.

Catecholamines, which promote lipolysis, are higher during exercise in cold air and water,

indicating that the reduced lipid metabolism is not due to a lack of adequate hormonal stimulation. It is proposed that cold-induced vasoconstriction of peripheral adipose tissue may account, in part, for the decrease in lipid mobilisation. The respiratory exchange ratio (RER) is often similar for exercise conducted in warm and cold climates, suggesting FFA utilisation is equivalent between warm and cold exposures. The fractional portion of oxygen consumption ($\dot{V}O_2$) used for FFA combustion may decrease slightly during exercise in the cold. This decrease may be related to a relative decrease in oxygen delivery (i.e. muscle blood flow) or to impaired lipid mobilisation.

Venous glucose is not substantially altered during exercise in the cold, but lactate levels are generally higher than with work in milder conditions. The time lag between production of lactate within the muscle and its release into the venous circulation may be increased by cold exposure.

Minute ventilation is substantially increased upon initial exposure to cold, and a relative hyperventilation may persist throughout exercise. With prolonged exercise, though, ventilation may return to values comparable to exercise in warmer conditions. Exercise $\dot{V}O_2$ is generally higher in the cold, but the difference between warm and cold environments becomes less as workload increases. Increases in oxygen uptake may be due to persistence of shivering during exercise, to an increase in muscle tonus in the absence of overshivering, or to nonshivering thermogenesis.

Heart rate is often, but not always, lower during exercise in the cold. The linear relationship between heart rate and oxygen consumption is displaced such that at a given rate oxygen uptake is higher. Cardiac arrhythmias are more frequent in the cold. Stroke volume tends to be higher than under warmer control conditions, but may decline sooner or at a rate equivalent to warm controls at heavy workloads. Cardiac output is similar to the same work done in temperature environments.

Cold-induced vasoconstriction occurs both in cutaneous and resting skeletal muscle beds, but inactive muscle provides most of the passive body insulation. With exercise insulation provided by muscle decreases as blood flow increases. Relative to warmer conditions, muscle blood flow at a given workload may be reduced if deep muscle temperature is below normal (i.e. 39°C optimum).

Respiratory heat loss is often assumed to represent 8% of the total metabolic heat production. However, during exercise this value will increase as minute ventilation increases. Loss of significant amounts of heat from the distal extremities can limit performance, even though the area is not directly involved with exercise.

Cooled muscle has a decreased capacity to generate force expressed on cross-sectional area. As a consequence, it may be necessary to recruit more fast twitch motor units. Glycolysis is higher in cooled muscle, which may account for higher lactate levels and greater rates of muscle glycogen depletion. Brief intense exercise will not raise cooled muscle temperature to normal limits, but mild exercise can maintain normal temperatures if exercise begins before the muscles become cooled.

Regional heat flux increases with exercise in the cold in direct proportion to the workload. Differing rates of heat loss can occur between active and inactive limbs, and individual rates are not constant throughout steady-state exercise. Peak rates of heat flux for inactive limbs occur during exercise, but peak flux for active limbs occurs in the postexercise period. At equal metabolic rates, more heat is lost with arm than with leg exercise.

Most of the heat generated by exercising muscle is transferred convectively to the core via the venous circulation. The amount of heat lost conductively to the environment through tissue depends upon factors such as subcutaneous fat. Thus, individuals with higher levels of fat (e.g. skinfold thickness) generally are better able to maintain their core temperature in cold environments.

Steady-state exercise $\dot{V}O_2$ values of approximately 2.0 L/min have been shown to prevent falls in core temperature in water as low as 15°C. Warmer temperatures are required in order to maintain core homeostasis during intermittent exercise.

Predicting an individual's response to exercise in the cold is quite difficult because of the interplay of many factors. Using existing data, responses to some forms of exercise and environmental stress can be estimated with reasonable accuracy. However, the number of these type

responses is small compared to the total number of possible permutations, showing that much is yet to be learned.

Interest in exercise in cold environments is no longer limited to wintertime sporting events. There is increasing participation in sports like scuba diving, long distance swimming, triathlons, running and cycling that occur on a year-round basis. In addition, there are greater numbers of men and women working in environmental extremes where cold temperatures may limit performance. As a consequence, there is more than an occasional concern about the interaction between cold stress and human performance. Understanding the physiological limits of endurance in the cold presents a challenge to exercise scientists that will likely continue into the next century.

This review presents an update on human exercise in the cold, using selected physiological topics that relate to performance. The intent is to provide both the expert and nonexpert with a sufficient overview to permit visualisation of the human response to cold stress. It is evident that there is no single response that covers every type of exposure, exercise protocol, or individual; although a generic example is offered in the conclusion to further aid the nonexpert. It is hoped that readers will also note those areas where additional research is needed to fill gaps in our present understanding.

Cold exposure is defined herein as any environmental condition where the potential exists to lose a significant amount of heat from any region of the body. This distinction is necessary since certain aspects of exercise performance may be influenced by heat loss from one or more areas of the body.

1. Metabolic Changes

1.1 Diet

At present, there is no consensus as to what the most appropriate diet should be for cold exposure. Both high carbohydrate and high fat diets have been reported to increase tolerance to cold and physical endurance, but high protein diets may have negative effects on tolerance and endurance (see Askew 1989). It is difficult to define a single effect of dietary manipulation since increasing one nutrient must, as a consequence, reduce another, unless total caloric intake is also changed. Thus, an observed effect may be due to the higher intake of one nutrient, a corresponding reduction in another or simply an alteration in caloric intake.

For prolonged cold exposure, there does not appear to be an increase in the resting energy requirement, provided adequate thermal protection is available (Askew 1989). However, the energy cost of performing work will increase as a consequence of the added resistance imposed by the thermal protective gear. For example, there is about a 10% increase in the oxygen cost of walking while wearing Arctic clothing (Nunneley 1989).

The amount of dietary carbohydrate intake may not be essential at rest, but has added importance during exercise. Young et al. (1989) found similar rates of decline in rectal temperature at rest in 18°C water after either a 3-day high carbohydrate diet (65% of calories) or a high fat diet (65% of calories). The high fat diet was coupled with exercise to produce low muscle glycogen levels, but the net change in glycogen content was comparable to the high carbohydrate diet.

High carbohydrate diets, used to increase muscle glycogen, are well known to increase exercise endurance conducted in milder environments (e.g. Sherman et al. 1981). A similar benefit of a high carbohydrate diet may also occur during cold exposure. Using intermittent exercise at 80% $\dot{V}O_{2max}$ for 4 hours in 25°C water, significantly more work was achieved after a 3-day high carbohydrate diet (600 g/day) than after a 3-day normal diet (300 g/day) [Thorp et al. 1990]. It would thus appear that carbohydrate intake is an important predeterminant of exercise performance in the cold.

1.2 Lipid Metabolism

Either cold exposure or exercise alone will increase lipid mobilisation and venous free fatty acid (FFA) concentration. The combination of cold plus exercise does not, however, act synergistically on lipid metabolism. Leg exercise at 50 to 60% $\dot{V}O_{2max}$ in 0°C air resulted in smaller increases in venous triglyceride than for the same exercise in 22°C air (Sink et al. 1989). Exercise at 65% $\dot{V}O_{2max}$ in 18°C water also produced a smaller increase in FFA than the same exercise in 28°C water (Doubt & Hsieh 1991).

Increases in circulating catecholamines stimulate lipolysis from adipose tissue and enhance the intravascular breakdown of triglycerides, with adrenaline (epinephrine) being more potent than noradrenaline (norepinephrine). Both adrenaline and noradrenaline are higher with exercise in cold water (Doubt & Hsieh 1991) or air (Dolny & Lemon 1988; Therminarias et al. 1989) than in warmer trials, suggesting that any relative decrease in FFA in the cold cannot be ascribed to a reduction in the hormonal stimulus. Glycerol, which is a better indicator of lipid mobilisation than FFA, is also reduced during exercise in cold water (Doubt & Hsieh 1991) and air (Sink et al. 1989). Thus, the simultaneous reductions in FFA and glycerol may be due, in part, to a perfusion limitation of peripheral adipose tissue. It is known, for example, that cold exposure directly induces vasoconstriction in adipose tissue and potentiates adrenergic vasoconstriction (Hjemdahl & Sollevi 1978), thereby reducing blood flow. This cold effect would be less pronounced in fat depots within the core since local temperature would be higher.

The respiratory exchange ratio (RER = $\dot{V}CO_2/\dot{V}O_2$) is often used to estimate nonprotein substrate utilisation. Use of RER is confined to steady-state conditions. Initial exposure to cold or the start of exercise will transiently increase and decrease RER, respectively, due to changes in body CO_2 stores that are not related to any alteration in substrate metabolism. RER will decline during steady-state exercise, reflecting an increase in fat metabolism. Many studies have found no significant differences in steady-state RER between warm and cold conditions, indicating cold stress does not increase FFA use (Doubt & Hsieh 1991; Jacobs et al. 1985; Moore et al. 1970; Sink et al. 1989; Therminarias et al. 1989). Others have noted RER to be lower by 0.02 to 0.03 in cold air, suggesting greater FFA oxidation (Dolny & Lemon 1988; Timmons et al. 1985).

Such disparate findings in RER are not easy to reconcile. Reported changes in RER are small, and minor differences in technical approach or subject variability may complicate interpretation of data. Small changes in RER can relate to larger estimates in fractional substrate utilisation. For example, an RER of 1.00 and 0.71 will occur for oxidation of 100% carbohydrate and fat, respectively (McGilvery 1970). The fractional utilisation of fat, F_{fat}, can be estimated by:

$$F_{fat} = 1.00 - [(RER - 0.71)/0.29] \quad \text{(Eq. 1)}$$

The term in parentheses represents the fractional utilisation of carbohydrate. Analysis of equation 1 reveals that F_{fat} will change by 3.4% for each 0.01 change in RER. Furthermore, a variation in either $\dot{V}CO_2$ or $\dot{V}O_2$ of only 10 ml/min will result in a change in RER of 0.01, or a 3.4% change in F_{fat}. Thus, while RER may be a useful means to estimate nonprotein fractional substrate utilisation it clearly must be used with caution when calculating total energy expenditures during exercise.

Protein degradation during exercise further complicates estimates of fat (or carbohydrate) utilisation since RER is about 0.82 for protein oxidation. Urea nitrogen excretion, measured over 3 days including 90 minutes of cycle exercise, was higher when exercise was conducted in 5°C air than in 30°C air (Dolny & Lemon 1988), indicating greater protein degradation occurred in the cold. This finding does not segregate the portion of protein breakdown actually used directly in support of muscle contraction (oxidation and gluconeogenesis) from other metabolic pathways or muscle damage. It does, nonetheless, illustrate the need to consider protein metabolism when attempting to

quantify energy expenditure and substrate utilisation during exercise.

1.3 Carbohydrate Metabolism

There is little doubt that circulating glucose plays an important role in tolerance to cold and in exercise endurance. Exogenous manipulation of venous glucose concentration with insulin infusion has shown that hypoglycaemia will suppress shivering (Gale et al. 1981) and significantly reduce rectal temperature (Mager & Francesconi 1983). In the absence of exogenous manipulation, Jacobs et al. (1984) found no significant change in plasma glucose following a resting immersion in 10°C water that resulted in a 1°C drop in core temperature. There was a significant decrease in insulin and a nonsignificant increase in glucagon. Likewise, no significant change occurred in glucose concentration after thermally protected subjects had been immersed in 5°C water for 6 hours, performing 9 minutes of moderate work each hour (Smith et al. 1990).

It is readily appreciated that exercise generally can not be sustained if glucose concentrations fall below 3.5 to 4.0 mmol/L. Glucose concentrations during steady-state submaximal exercise usually do not fall below this range, and cold exposure *per se* does not appear to significantly modify the rate of decline. For example, glucose concentration and its rate of decline were similar during 60 minutes of exercise at 1.5 W/kg in 18 and 28°C water (Doubt & Hsieh 1991). Whether longer exercise periods or greater thermal stress alter control of circulating glucose remains to be determined.

The rate of muscle glycogen utilisation during cold exposure depends on the interaction between cold and exercise intensity. At rest, muscle glycogen content has little effect on the change in core temperature during cold water immersion (Young et al. 1989). The combination of cold and light exercise decreased glycogen by 23% in vastus lateralis muscle following 30 minutes of exercise at 50 to 65W in 9°C air, whereas the same workload in 21°C air resulted in minimal glycogen depletion (Jacobs et al. 1985). Pre-exercise glycogen content was higher, and postexercise content lower, in subjects who performed the cold exposure. When a higher workload was used (85 to 120W) this study found equivalent decreases in muscle glycogen in both air temperatures. At the lighter workload muscle temperature may have been lower than at the higher workload, thereby increasing the reliance on muscle glycolysis. This postulate is supported by the finding that glycolysis is higher in vastus lateralis muscle cooled to 28 to 29°C prior to exercise than in muscle at 35°C (Blomstrand et al. 1986; Blomstrand & Essen-Gustavsson 1987).

1.4 Lactate Metabolism

Since there is an inverse relation between muscle temperature and the rate of glycolysis, it is not surprising that many studies have reported venous lactate concentrations to be higher with exercise in the cold than under warmer conditions (Doubt & Hsieh 1991; Jacobs et al. 1984, 1985). Absolute values of lactate appear to be higher in cold water (Goodman et al. 1985) than in cold air (Jacobs et al. 1985; Therminarias et al. 1989), but the reason is not obvious.

Increases in blood lactate are generally considered to reflect a higher anaerobic contribution to the work effort (Pendergast 1988).

The appearance of lactate in the venous circulation has, however, a time lag that may be temperature dependent. Brief high intensity exercise resulted in no difference in venous lactate during exercise using cooled *vs* normothermic muscle, although intramuscular and postexercise venous lactate values were higher in the cooled muscle (Blomstrand et al. 1986).

Therminarias et al. (1989) obtained venous lactate samples at the end of each incremental increase in workload conducted at −2 and +24°C air temperatures. Lactate values were higher in the cold air at workloads from 30 to 90W, and increased at a slower rate than in 24°C air. The rate was also slower at workloads >120W, leading to lower lactate levels in the −2°C air. A cold-induced delay in lactate release from muscle may have contrib-

uted, in part, to the observed lower lactate values as work intensity increased.

Decreases in the aerobic utilisation of lactate within muscle (i.e. lactate shuttle) or decreases in its hepatic clearance could also contribute to higher venous concentrations during exercise in the cold. A recent review addresses the difficulties in evaluating lactate metabolism solely on the basis of venous concentrations (Stainsby & Brooks 1990).

2. Cardiopulmonary Changes

2.1 Ventilation

A relative increase in minute ventilation ($\dot{V}_E$) occurs upon initial exposure to a cold environment (Doubt & Francis 1989; Mekjavic & Bligh 1989). A higher $\dot{V}_E$, compared to the same workload under warmer conditions, can persist during light exercise (Cooper et al. 1976), during continuous increases in workload (Therminarias et al. 1989), or during discontinuous incremental increases in workload (Moore et al. 1970). Differences between $\dot{V}_E$ in warm and cold trials diminish as workload increases (Therminarias et al. 1989). During steady-state exercise $\dot{V}_E$ eventually becomes equivalent to work done under warmer conditions (Doubt & Hsieh 1991; McMurray & Horvath 1979.

Increases in $\dot{V}_E$ may reduce end-tidal CO_2 (ET_{CO_2}). At rest or with light to moderate exercise, the reduction in ET_{CO_2} can result in mental confusion in persons working in cold environments (Cooper et al. 1976), probably secondary to cerebral vasoconstriction induced by a low arterial P_{CO_2}. This effect may, in fact, be a contributing factor in fatal or near-fatal accidents in cold water (Doubt & Francis 1989).

It is likely that the increase in $\dot{V}_E$ upon initial cold exposure is due, in large measure, to afferent input from cutaneous thermal receptors (Mekjavic & Bligh 1989). Gradual reductions in $\dot{V}_E$ at rest or during exercise in cold environments may reflect habituation of the peripheral thermal receptors. A psychogenic component to the relative hyperventilation may also occur under some circumstances. For example, open water swimming resulted in a higher $\dot{V}_E$ than when a similar workload was performed in the laboratory at a similar water temperature (Cooper et al. 1976).

2.2 Oxygen Consumption

Oxygen consumption ($\dot{V}O_2$) will depend on the interplay of exercise intensity and duration with the overall thermogenic requirements. In general, thermogenesis at rest will increase $\dot{V}O_2$ to a maximum value of about 1.5 L/min. Exercise is the only effective means to increase heat production (e.g. $\dot{V}O_2$) above this level. Given the wide range of rest and exercise paradigms, and environmental temperatures, it is not entirely surprising to find studies where exercise $\dot{V}O_2$ in the cold increased (Doubt & Hsieh 1991; McArdle et al. 1976; Therminarias et al. 1989; Timmons et al. 1985) or did not change (Doubt & Smith 1990; Jacobs et al. 1985; McArdle et al. 1976) relative to warmer conditions or time of exposure. Leaner subjects tend to have a higher exercise $\dot{V}O_2$ during cold exposures than subjects with more body fat (McArdle et al. 1984).

Studies employing incremental increases in workload have noted a convergence of the $\dot{V}O_2$ *vs* workload curves for warm and cold trials, suggesting that the absolute temperature-related difference in $\dot{V}O_2$ decreases as work intensity increases (McArdle et al. 1976; Moore et al. 1970; Therminarias et al. 1989).

Two mechanisms (apart from percentage body fat) can account for higher exercise $\dot{V}O_2$ values in the cold. First, total body heat flux increases during exercise in the cold (Nadel 1984; Park et al. 1984; Rennie 1988; Sagawa et al. 1988). If the rate of heat loss is sufficiently large then one would anticipate activation of nonexercising thermogenic mechanisms. For example, overt shivering can persist during dynamic exercise in cold water (Hoar et al. 1976; McArdle et al. 1976). While shivering does not contribute to the dynamic work effort, its presence will result in a higher exercise $\dot{V}O_2$. On the other hand, overt shivering was absent in one of McArdle's subjects during exercise, and in other studies was noted to be diminished or absent during

exercise (Craig & Dvorak 1968; Jacobs et al. 1985). We also did not observe shivering during exercise in 18°C water, although exercise $\dot{V}O_2$ was higher than in 28°C water (Doubt & Hsieh 1991). The absence of overt shivering in the presence of a higher $\dot{V}O_2$ may reflect either nonshivering thermogenesis or a subtle persistence of muscular shivering.

The second mechanism to consider relating a higher exercise $\dot{V}O_2$ to cold exposure is that net mechanical efficiency may be decreased (Pendergast 1988). The kinetic energy required to perform external work should not change as a function of temperature (e.g. bike workload or running speed). Therefore, the oxygen cost for this work should remain independent of environmental temperature. However, since cooled muscle has a reduced contractile force per cross-sectional area, it has been proposed that more motor units might be recruited in order to meet the required work output (Blomstrand et al. 1986), potentially raising $\dot{V}O_2$. In addition, if shivering were present, it might involve both agonist and antagonist muscle groups, increasing the effort to conduct dynamic exercise. Even in the absence of overt shivering, muscle agonist and antagonist groups could have increased activity that opposes movement and raises the oxygen cost of work. Support for this latter notion comes from the study of Pozos (1981) where synchronised EMG activity increased during cold exposures in the absence of overt shivering.

2.3 Ventilatory Equivalent

The ratio of $\dot{V}_E$ to $\dot{V}O_2$ (ventilatory equivalent) provides a measure of how well ventilation can be matched to the oxygen uptake requirement. Therminarias et al. (1989) found this ratio to be higher in −2°C air than in 24°C air up to workloads of 150W with no temperature-related differences noted at higher intensities, suggesting that ventilation is mismatched with oxygen demand at lower workloads. Nearly the opposite effect is found during immersion, although thermal stress does not exert a significant influence. At rest and during exercise up to a $\dot{V}O_2$ of about 2.0 L/min immersion in water from 5 to 35°C does not substantially alter the ventilatory equivalent when compared to exercise in the dry (Doubt & Hsieh 1991; Moore et al. 1970; McArdle et al. 1976; Newstead 1987). Data from these studies reveals a tendency for the ventilatory equivalent to be higher during immersion when $\dot{V}O_2$ exceeds 2.0 L/min, but not in a temperature-dependent manner. Figure 1 illustrates this point using data from several of our laboratory studies. The exponential fit of the immersed data begins to increase faster than the fit of the dry data at $\dot{V}O_2$ values greater than 2.0 L/min, such that $\dot{V}_E$ exceeds $\dot{V}O_2$. This mismatch of ventilation may be due to immersion effects on alveolar-arterial O_2 gradients or ventilation-perfusion matching that become apparent at high work intensities.

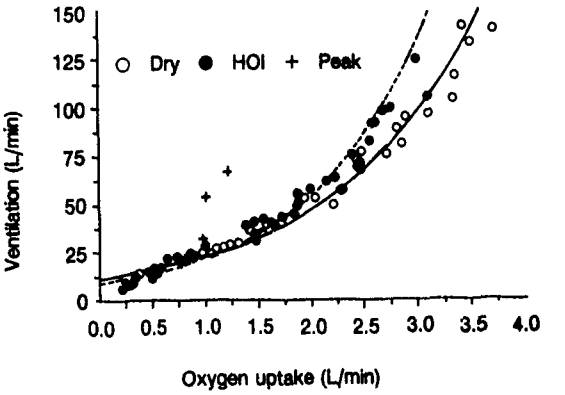

Fig. 1. Relationship of minute ventilation to oxygen consumption obtained in dry and head-out immersion (HOI) in 18 to 31°C water, from rest to exhaustive workloads. HOI resulted in significant shift in $\dot{V}_E$ to $\dot{V}O_2$ ratio at oxygen consumptions higher than 2.0 L/min. Crosses are resting data from Mekjavic & Bligh (1989) 1 minute after immersion in 10 to 40°C water; with 10 and 20°C data points appreciably above the standard regression curve.

Figure 1 also contains peak resting values of $\dot{V}_E$ *vs* $\dot{V}O_2$ obtained during the first minute of immersion in water from 10 to 40°C (Mekjavic & Bligh 1989). At temperatures below 20°C there is an obvious mismatch in $\dot{V}_E/\dot{V}O_2$, since ventilation is markedly higher than oxygen consumption. If exercise were to begin at this point it would likely require some time for the ventilatory equivalent to return to the expected range.

2.4 Heart Rate

Exposure to cold air or water causes a significant peripheral vasoconstriction that results in an elevation of blood pressure. In theory, baroreceptor reflexes would attempt to lower pressure through a parasympathetically mediated reduction in heart rate. However, it is far from universally observed that resting or exercise heart rate will be lower with cold exposure.

Males exposed to 5°C air had lower heart rates at rest and during exercise than in 22°C air, but no significant changes were observed in females during exercise (Stevens et al. 1987). Other studies, using males, have noted heart rate to be relatively lower during submaximal exercise in cold air (Sink et al. 1989; Therminarias et al. 1989); while others have found no difference between warm and cold air exposures (Jacobs et al. 1985).

Decreases in heart rate upon head-out immersion have often been noted, with a relative bradycardia persisting throughout exercise compared to dry conditions (Craig & Dvorak 1968; Dressendorfer et al. 1976). Although these type studies have used water temperatures below thermoneutral, a lower heart rate during immersion is not *a priori* evidence of a cold effect. We have, for example, found no difference in exercise heart rate for the same workloads in 28 *vs* 18°C water (Doubt & Hsieh 1991) or in 31 *vs* 20°C water (unpublished data). Dissimilar studies comparing warm with cold water have noted that exercise heart rate is reduced (Craig & Dvorak 1968; Dressendorfer et al. 1976; McArdle et al. 1976; McMurray & Horvath 1979), unchanged (Pirnay et al. 1977) or even increased (Cooper et al. 1976) in cold water. Such disparate results may reflect differences in level of fitness, relative workload, sympathetic nervous system arousal, psychogenic awareness of stress, shivering, increased muscle tonus or immersion times.

The slope of the linear relationship between heart rate and exercise workload is little changed as a function of environmental temperature (McArdle et al. 1976; Moore et al. 1970) or duration of exposure (Doubt & Smith 1990). This indicates that cardiac responsiveness to increases in workload is largely unaffected by cold exposures where hypothermia is absent.

The oxygen pulse (amount of O_2 delivered per beat of the heart = $\dot{V}O_2$/HR) may or may not change with exercise in the cold. Relative to warm control conditions, the oxygen pulse has been observed to increase in studies where exercise $\dot{V}O_2$ increased with no change in heart rate (Doubt & Hsieh 1991), no change in $\dot{V}O_2$ with decreases in heart rate (Sink et al. 1989), or where exercise $\dot{V}O_2$ increased and heart rate decreased (McArdle et al. 1976; Moore et al. 1970; Therminarias et al. 1989). A decrease in the oxygen pulse can occur where there is no change in $\dot{V}O_2$ coupled with an increase in heart rate, as observed in thermally protected subjects immersed for long periods in cold water (Doubt & Smith 1990). McArdle et al. (1976) have shown that the relationship of $\dot{V}O_2$ to heart rate is shifted leftward in cold water, such that at a given heart rate the $\dot{V}O_2$ is higher. The variability of the oxygen pulse data obtained during cold exposures also illustrates the difficulty of reliably predicting $\dot{V}O_2$ from measures of heart rate alone.

2.5 Cardiac Arrhythmias

Cardiac arrhythmias appear to be more frequent with exercise in colder environments than in warmer environments (Doubt & Francis 1989). We have noted, for example, that cold water immersion increases the incidence of sinus dysrhythmias in healthy males; probably as a consequence of altered autonomic tone (Doubt et al. 1987). The incidence of ventricular extrasystoles is also noted to be higher in colder environments (Keatinge 1972; Okada 1984); possibly related to increases in ventricular preload or afterload, or to increased sensitivity to higher catecholamine levels.

The possibility exists that exercise in cold water may produce fatal cardiac arrhythmias. Eldridge (1979) did a retrospective study of scuba diving accidents and found a higher incidence of cardiac deaths occurred in cold water. Furthermore, she noted a positive correlation between the age of the diver and the occurrence of fatalities due to cardiac events. Although aging and incidence of cardio-

vascular disease are positively correlated for the general population, there is no definitive predictor as to how much an individual's risk of a serious arrhythmia might increase with exercise in the cold. Factors such as level of aerobic fitness will modulate any age-related risk.

2.6 Cardiac Output

Exposure to cold air increases intrathoracic blood volume secondary to peripheral vasoconstriction. Immersion in thermoneutral water increases thoracic blood volume by a hydrostatic effect (Pendergast 1988). Cold water induces peripheral vasoconstriction that further increases the thoracic volume, as implied by larger increases in stroke volume (McArdle et al. 1976) or total body insulation (Rennie 1988).

The increase in thoracic blood volume resulting from immersion has been shown to increase both left ventricular end-diastolic and end-systolic dimensions at rest and during exercise (Sheldahl et al. 1984). Such increases in cardiac dimensions are presumptive evidence that wall tension increases, boosting myocardial oxygen demand. Elevations in afterload can also alter cardiac function independently of other physiological changes. For example, immersing a forearm in ice water raised blood pressure 20 to 25% (i.e. afterload), and resulted in a 7% decrease in left ventricular ejection fraction during an isometric handgrip test (Jones et al. 1986). Thus, myocardial oxygen demand would be expected to increase during exercise in the cold as functions of increased preload (i.e. increased filling pressure, increased wall tension) and increased afterload (i.e. increased blood pressure).

Although absolute values may vary from laboratory to laboratory, stroke volume will increase on exposure to either cold air (Graham 1988; Raven et al. 1975) or cold water (McArdle et al. 1976; Pendergast 1988; Rennie et al. 1971). During light to moderate exercise, stroke volume will generally remain higher than equivalent dry or warm conditions, and heart rate will be lower, thereby resulting in little change in cardiac output compared to control conditions. At higher workloads, however, stroke volume has been found to decline such that it may not differ significantly from control conditions (Graham 1988; McArdle et al. 1976). In these studies heart rate concurrently increased (McArdle et al. 1976) or did not change (Graham 1988). Interestingly, Graham (1983) found that women show little or no difference in stroke volume or heart rate during exercise in 5 *vs* 22°C air.

There are several factors that can influence changes in stroke volume in the cold. First, any increase in ventricular end-diastolic volume would result in a rightward shift in the Starling curve, potentially to the point where stroke volume might subsequently decline as the exercise demand increased. Second, increases in myocardial oxygen demand mentioned above may exceed cardiac reserve at high workloads, thereby reducing stroke volume. Third, the positive inotropic or chronotropic effects of higher catecholamine levels may support cardiac output.

Mild decreases in core temperature do not adversely affect cardiac performance in healthy individuals. Olschewski and Bruck (1988) reported that a 15-minute exposure to 5 to 10°C air prior to exercise lowered core temperature by 0.2°C. During light exercise stroke volume increased with no change in cardiac output relative to trials without precooling. During exercise at 80% $\dot{V}O_{2max}$ decreases in stroke volume were comparable to control values although core temperature was about 0.5°C lower with precooling. If this mild drop in core temperature had a negative influence on cardiac performance, one would have expected to see more of a decrease in stroke volume. Larger decreases in core temperature, though, may result in a relative decline in cardiac output during exercise (Pendergast 1988).

2.7 Peripheral Blood Flow

At rest, skeletal muscle blood flow is low. Consequently, muscle mass accounts for the major portion of passive body insulation at rest during cold exposures (Hayward & Keatinge 1981; Rennie 1988; Toner et al. 1986). With the onset of exercise, muscle blood flow increases and reduces insulation

in direct proportion to workload. Blood flow to active muscle may be lower in cold environments, however, at subnormal muscle temperatures (Blomstrand et al. 1986; Pendergast 1988).

The effects of cold on peripheral circulation are somewhat more complex than usually thought. There is unanimous agreement that cold exposures result in peripheral vasoconstriction. Exercise produces a neurally mediated vasoconstriction that will persist up to maximal workloads (Johnson et al. 1986). Cold exposure magnifies the neurally and humorally induced constriction of superficial cutaneous veins, while the opposite effect occurs in deep limb veins (Johnson et al. 1986). It has also been noted that cold exposures appear to primarily influence the α_2-adrenoreceptor constrictor response to noradrenaline (Flavahan et al. 1985). Data are currently lacking on how cold and exercise might modify the various types of adrenoreceptor responses in each of the peripheral circulatory beds, including skeletal muscle.

3. Thermal Aspects

3.1 Respiratory Heat Loss

Measurements of net thermal balance usually assume that respiratory heat loss represents about 8% of total body heat production (Ferretti et al. 1988; Rennie 1988; Sagawa et al. 1988; Toner et al. 1986). Estimates of respiratory heat loss have used measures of $\dot{V}_E$ and assumed values for inspired and expired gas temperatures (Hayward & Keatinge 1981). Respiratory heat loss during exercise can be higher than at rest. For example, dry and evaporative respiratory heat loss represented 8% of total heat production at rest in 22°C water, but increased to 11 to 14% (concomitant with increases in $\dot{V}_E$) while swimming at speeds of 0.3 to 0.5 m/sec (Rapp 1970). This finding indicates that assuming a strict 8% value for respiratory heat loss may underestimate net thermal balance during exercise in cold environments.

3.2 Peripheral Thermoregulation

The head, hands, feet, external genitalia and skin are often not considered when discussing exercise in the cold, since they do not participate directly in the exercise. However, thermal balance in these regions often will play a significant indirect role in overall performance. As an example, 14 of 63 exposures of thermally protected subjects in 5°C water were terminated prematurely because low finger or toe temperatures produced undue discomfort even though total body heat flux averaged only 125 W/m^2 and rectal temperature declined only 1.3°C in 6 hours (Doubt & Smith 1990). In like fashion, one can envision how athletic performance would be impaired if severe cold exposure of the digits reduced dexterity, or if heat loss through the head exacerbated body cooling.

Distal extremities depend on blood flow to maintain suitable local temperature in the cold since their intrinsic capacity to generate heat is limited. Cold induced vasoconstriction dramatically reduces flow of warm arterial blood into the region and local temperature falls. Prolonged low temperatures in local regions can result in nonfreezing cold injury or frostbite (Doubt & Francis 1989).

Neurally mediated vasoconstriction is inhibited at very low local temperatures, leading to vasodilation (Rusch et al. 1981). The resulting influx of warm arterial blood establishes a negative feedback loop (often termed 'hunting relfex') where alternating vasoconstriction and vasodilation results in corresponding decreases and increases in local skin temperature. During exercise, heat flux from hands and feet will increase (Ferretti et al. 1988), indicating an increase in blood flow. Finger and toe temperatures commonly rise and the magnitude of the 'hunting reflex' will diminish with exercise.

3.3 Skeletal Muscle Temperature

Skin and subcutaneous fat account for <30% of total body insulation at rest in cold water (Hayward & Keatinge 1981; Nadel 1984; Rennie 1988). Muscle provides most of the insulation at rest. Toner et al. (1986), comparing males of similar body fat resting in 22°C water, measured higher body insulation values in individuals with a higher muscle mass. With the onset of exercise, insulation values were similar for low and high muscle mass groups as would be expected if increased blood flow

decreased the passive insulation value of skeletal muscle (Park et al. 1984; Rennie 1988; Sagawa et al. 1988).

A number of studies have shown that body insulation decreases as workload increases (Rennie 1988; Sagawa et al. 1988; Toner et al. 1984; Veicsteinas et al. 1982). In water colder than about 25°C cutaneous vasoconstriction is near maximum, and any exercise-related decreases in insulation would presumably reflect an increase in muscle blood flow. Total body insulation for a given workload will be higher at lower water temperatures which suggests a relative underperfusion of muscle at colder temperatures.

Cooling of skeletal muscle will reduce contractile force and increase the rate of fatigue. Petrofsky et al. (1981) found that maximum voluntary contractions and isometric endurance time in forearm muscles decreased at water temperatures below 20°C. In order to perform dynamic exercise in muscle cooled from 35 to 29°C, Blomstrand et al. (1986) speculated that more fast twitch fibres might be recruited. The greater participation of fast twitch glycolytic fibres could contribute to the higher rates of lactate production and shorter endurance times often noted with exercise in the cold.

It is evident that deep muscle temperature depends both on environmental temperature and the intensity and duration of exercise. In vastus lateralis muscle cooled to 29°C exercise at 370W for 1.5 minutes increased muscle temperature to only 33°C, compared to 36°C for muscle starting exercise at 35°C (Blomstrand et al. 1986). Thus, brief intense exercise will not raise precooled muscle to normal operating temperatures.

If exercise begins before muscles become cooled it appears that muscle temperature can increase to near normal levels. As an example, subjects who began exercising shortly after immersion in 20°C water had deep quadriceps muscle temperatures of 38°C after 15 minutes of work at $\dot{V}O_2$ values of 1.0 to 2.0 L/min (Pirnay et al. 1977). This study also confirmed that $\dot{V}O_{2max}$ occurs at a deep muscle temperature around 39°C. One can conclude, therefore, that although exercise will increase muscle temperature, certain combinations of environmental temperature and exercise protocol will result in lower than optimal temperatures that could limit performance. One might also expect intramuscular temperature gradients to exist during cold exposures, the magnitude of which could influence local muscle blood flow, motor unit recruitment or endurance.

3.4 Core Temperature Homeostasis

Preservation of core temperature in cold environments depends on the ability to generate enough heat to offset avenues of heat loss. There is no doubt that exercise, while increasing metabolic heat production, also increases the rate of heat loss. Not only is the rate of heat loss proportional to workload (Ferretti et al. 1988; Rapp 1970; Toner et al. 1985) but the ratio of limb to trunk heat loss also increases directly with exercise intensity (Sagawa et al. 1988).

Regional heat flux is not necessarily constant with exercise time, and varies considerably between active and inactive limbs. Toner et al. (1985), for example, reported arm heat flux decreased from 65 to 60 W/m^2 during 60 minutes of leg exercise in 30°C water, whereas in 18°C water the flux decreased from 205 to 75 W/m^2. Leg heat flux in this study increased from 50 to 74 W/m^2 and decreased from 158 to 114 W/m^2 for 30 and 18°C, respectively. Peak values for active and inactive limb heat fluxes may not occur simultaneously. During intermittent leg exercise in 25°C water, peak values for arm heat flux occurred during exercise while leg flux peaked during the subsequent postexercise rest period (Thorp et al. 1990). The high peak fluxes in the postexercise period might explain why intermittent exercise in cool water often results in *declines* in core temperature (Hoar et al. 1976).

At equivalent metabolic rates, arm exercise or a combination of arm-leg exercise resulted in greater decreases in core temperature than leg-only exercise in 20 and 26°C water (Toner et al. 1984). Since arm muscle mass is less than the leg, the equivalent metabolic rate was presumably achieved at a greater arm muscle blood flow. This would decrease insulation and promote greater heat loss, as was ev-

ident from higher rates of total heat flux when arm exercise was employed.

It has been a longstanding concern as to whether there is a crucial water (or air) temperature, below which exercise will be detrimental to the maintenance of core temperature (Keatinge 1972; Sagawa et al. 1988). However, since factors such as skinfold thickness (Rennie 1988; Veicsteinas et al. 1982) can modify responses to cold water exposure, it is difficult to define a discrete water temperature. An exercise $\dot{V}O_2$ of 1.6 to 1.7 L/min for 60 minutes was more effective than rest in maintaining core temperature in 18 to 30°C water (McArdle et al. 1984; Toner et al. 1985). We noted no significant change in rectal temperature during 60 minutes of exercise at a $\dot{V}O_2$ of 2.0 to 2.2 L/min in 18°C water (Doubt & Hsieh 1991). Golden and Tipton (1987) reported that an exercise $\dot{V}O_2$ of about 1.8 L/min prevented significant changes in rectal temperature during 40 minutes in 15°C water. For continuous exercise, it would appear that core temperature can be maintained in water as cold as 15°C with an exercise $\dot{V}O_2$ around 2.0 L/min. The crucial water temperature for intermittent exercise is likely higher since even 25°C can result in reductions in core temperature (Hoar et al. 1976).

It should be readily appreciated that skeletal muscle is the heat generator during exercise, and active muscle temperatures exceed core temperature by at least 0.5 to 1.0°C (Pirnay et al. 1977). Since the generator is located in the periphery, any heat transferred to the core occurs convectively through venous return. It is equally obvious that not all heat generated within the muscle will be transferred to the core, since a certain amount will be lost to the environment by conductive heat transfer through the tissue (Rennie 1988). For example, Ferretti et al. (1988) estimated that the heat production of thigh muscles working at 79W in 30°C water was 49% greater than the measured heat flux from the thigh. A majority of this difference between local production and loss represents heat transferred convectively to the core. Different muscle to environment temperature gradients, circulatory responses and cutaneous insulation values could modify the amount of heat transferred to the core for a given metabolic rate.

4. Conclusion

No single set of physiological responses can describe exercise responses in cold environments. There are simply too many circumstances to yield one discrete result. However, a useful conclusion can be offered using a generic example that may be useful to many readers. Table I will be used as a guide, employing one rest : work protocol. The generic subject is not classified by age, gender or dietary history. The cold exposure is also unclassified with respect to water or air.

After a brief resting exposure, the subject begins to exercise. Vasoconstriction results in a relative reduction in blood flow that, coupled with slightly cooled muscle, increases the reliance on glycolysis to provide energy. As a consequence, lactate accumulates in the muscle at a higher rate and subsequently raises venous lactate levels.

Increased muscle tonus and shivering raise oxygen consumption above that of a warmer climate. Increases in cardiac preload and afterload increase myocardial oxygen demand. Stroke volume is larger, but heart rate is reflexly reduced and cardiac output is near normal. Circulating catecholamines provide positive inotropic support to the heart, although an occasional irregular beat occurs.

As exercise continues the muscle temperature increases towards normal and blood flow increases. Anaerobic metabolism declines. Lipid mobilisation increases, but due to cold induced vasoconstriction in peripheral adipose tissue, the rate is less than in warmer conditions. Utilisation of fat is reduced somewhat, but blood glucose levels remain within normal limits.

Peripheral thermoreceptors, initially stimulated by the cold, become adapted. The relative hyperventilation decreases. The rate of heat loss increases with the start of exercise, but enough metabolic heat is generated by muscle to prevent a drop in core temperature. Skin blood flow remains low.

The subject stops exercising momentarily. Large amounts of heat are lost and core temperature falls.

Table I. General changes occurring with exercise during thermoneutral (TN) *vs* cold exposure

Variable	Thermoneutral[a]	Cold[b]
Metabolic substrates		
Lipid mobilisation	Gradual increase	Less than or similar to TN
Free fatty acid use	Gradual increase	Less than TN
Venous FFA levels	Gradual increase	Less than or similar to TN
Glucose use	Higher at start; gradual decrease with time	Slightly higher than or similar to TN
Venous glucose levels	Decrease with time	Similar to TN
Muscle glycogen use	Increases with increase in workload	Higher in cool muscle
Lactate productior	Increases with increase in workload	Higher than TN
Cardiopulmonary changes		
Ventilation	Increases with increase in workload	Higher than TN
Oxygen consumption	Increases with increase in workload	Higher than TN for lower workloads, similar to TN for high workloads
Heart rate	Increases with increase in workload	Lower or similar to TN
Cardiac output	Increases with increase in workload	Similar to TN
Peripheral blood flow	Increases with workload or time	Lower in skin, decrease or no change in muscle
Thermoregulation		
Respiratory heat loss	Increases with increase in ventilation	Greater than TN
Peripheral heat loss	Increases with workload or time	Greater than TN
Muscle temperature	Increases	Lower, similar to TN
Core temperature	Increase or no change	Decrease, similar to TN

a Conditions of light-moderate workloads, thermoneutral ambient temperature, steady-state exercise.
b Relative to that of thermoneutral condition.

Upon resuming exercise, at a higher workload, the $\dot{V}O_2$ is closer to what it was in milder environments. Although heat flux is increased further, the additional metabolic heat prevents a decline in core temperature. Cardiac stroke volume exhibits a decline, but increases in heart rate maintain cardiac output at values similar to warm conditions. The subject finds it increasingly difficult to continue at this pace and stops exercise. Assistance is needed to remove the subject from the cold exposure because hands and feet have become numb.

Obviously, this scenario can be modified in any number of ways by manipulating the factors covered in this review. While the outcome of some manipulations is known with reasonable certitude, others will require additional research.

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Correspondence and reprints: Dr *Thomas J Doubt*, Hyperbaric Environmental Adaptation Program, Naval Medical Research Institute, Bethesda, MD 20814, USA