REVIEW ARTICLE

Sports Med 2006; 36 (9): 747-765
0112-1642/06/0009-0747/$39.95/0

Physiological Response to Water Immersion

A Method for Sport Recovery?

Ian M. Wilcock,[1] *John B. Cronin*[1] and *Wayne A. Hing*[2]

1 Institute of Sport and Recreation Research New Zealand, Division of Sport and Recreation, Faculty of Health and Environmental Sciences, Auckland University of Technology, Auckland, New Zealand
2 School of Physiotherapy, Faculty of Health and Environmental Sciences, Auckland University of Technology, Auckland, New Zealand

Contents

Abstract

Recovery from exercise can be an important factor in performance during repeated bouts of exercise. In a tournament situation, where athletes may compete numerous times over a few days, enhancing recovery may provide a competitive advantage. One method that is gaining popularity as a means to enhance post-game or post-training recovery is immersion in water. Much of the literature on the ability of water immersion as a means to improve athletic recovery appears

to be based on anecdotal information, with limited research on actual performance change. Water immersion may cause physiological changes within the body that could improve recovery from exercise. These physiological changes include intracellular-intravascular fluid shifts, reduction of muscle oedema and increased cardiac output (without increasing energy expenditure), which increases blood flow and possible nutrient and waste transportation through the body. Also, there may be a psychological benefit to athletes with a reduced cessation of fatigue during immersion. Water temperature alters the physiological response to immersion and cool to thermoneutral temperatures may provide the best range for recovery. Further performance-orientated research is required to determine whether water immersion is beneficial to athletes.

Water immersion has been used in some cultures for centuries as a means of health restoration.[1,2] Recently, water immersion has gained popularity as a means to improve recovery from exercise, although much of its use is based on anecdotal information.[2] For example, Netball New Zealand[3] recommends that during the course of tournament-type events, players should perform 5 minutes of water immersion (contrast therapy) after games to aide recovery and performance. There is some basis for the use of water immersion (non-exercising) to enhance recovery from exercise as it can produce beneficial physiological changes within the body. These physiological changes have been attributed principally to effects of hydrostatic pressure and temperature. Exercise in water is also used as a recovery session by some sport teams. However, the focus of this article is the physiological response of the body during non-exercise immersion. This article will briefly describe the water immersion recovery strategies used in the laboratory and in the field. Thereafter, the effect that hydrostatic pressure may have on the body in thermoneutral water, and the effect that cold and hot water temperature has on the body, will be examined. This information may assist athletes, trainers, physiotherapists and coaches in determining whether to adopt water immersion as a recovery strategy or assist in developing water immersion recovery protocols.

1. Water Immersion as a Recovery Strategy

There are four different methods of using water immersion in recovery: (i) cryotherapy; (ii) thermotherapy; (iii) contrast therapy; and (iv) water immersion *per se*. Cryotherapy, thermotherapy and water immersion *per se* are immersion in water at a constant temperature, whereas contrast therapy is immersion in alternating extremes of temperature.

1.1 Cryotherapy

Cryotherapy is immersion in cold water. No specific water temperature range has been determined for cryotherapy. Low and Reed[4] state that the sensation of cold pain begins at 15°C, and some research has used temperatures of ≤15°C for the study of cold-water immersion compared with thermoneutral water[5,6] and in performance studies.[7-9] Therefore, for the purpose of this article, cryotherapy was considered to be immersion in water of ≤15°C. In the field, cryotherapy normally consists of putting bags of ice in a container (such as a plastic drum) full of water in which an athlete stands to immerse their legs. In performance research, the duration of immersion time varies from 15 to 20 minutes.[7,8,10] However, in the field, immersion time may be as little as 30 seconds as a result of the ability of an individual athlete to withstand cold discomfort. For

a large number of athletes, or team sports, this method can be impractical because of the time required to treat all of the athletes.

1.2 Thermotherapy

Thermotherapy refers to immersion in water that raises the core body temperature. This increase in core temperature occurs in water with a temperature >36°C.[5,11,12] Facilities that provide a heated pool are required to perform thermotherapy. Anecdotally, there are numerous teams that are based in facilities that provide heated baths/spas and who perform thermotherapy after training. However, compared with thermoneutral immersion, little research has been conducted on the physiological or performance effect of hot water immersion. An immersion duration of 10–20 minutes has been suggested by Brukner and Khan[13] to aide athletic recovery and rehabilitation, although this time period does not appear to be based on research and is unsubstantiated.

1.3 Contrast Therapy

Contrast therapy is a post-exercise recovery method that has recently gained popularity.[14] Contrast therapy necessitates alternating temperature immersion, from a hot to cold bath and vice versa. Protocols vary (see table I) but generally consist of 30–300 seconds of one temperature extreme, immediately followed by 30–300 seconds of the contrasting temperature. This is repeated a number of times and lasts for 4–30 minutes. Vascular 'pumping' caused by the variation in temperature has been proposed as the mechanism that could improve recovery.[14-16] Whether such a contention is supported by the literature will be discussed in section 3.3.

1.4 Water Immersion *per se*

Water immersion *per se* is both the easiest method of application in the field and, compared with the other water immersion modes, widely researched (physiologically). No resources are required to heat or cool the water, only a container, bath or pool in which to immerse athletes. The

Table I. Examples of contrast therapy protocols

Study	Temperature (°C)		Application time	Repeats	Order
	cold	hot			
Research					
Coffey et al.[17]	10	42	1 min cold: 2 min hot	5	Start cold, end hot
Cote et al.[18]	10–15	39–41	1 min cold: 3 min hot	4	Start hot, end hot
Hamlin and Magson[19]	8–10	38	1 min cold: 1 min hot	3	Start cold, end hot
Hamlin and Sheen[20]	8–10	38	1 min cold: 1 min hot	3	Start cold, end hot
Higgins and Kaminski[21]	15	40	10 min hot, then 1 min cold: 4 min hot	5	Start hot, end cold
Kuligowski et al.[22]	13	39	3 min hot: 1 min cold	6	Start hot, end cold
Sanders[16]	15	38	3.5 min hot: 30 sec cold	3	Start hot, end cold
Vaile et al.[23]	8–10	40–42	1 min cold: 2 min hot	5	Start cold, end hot
Text					
Briggs[24]	10–15	NA	3 min hot: 1 min cold	5 ×/h	Start hot, end cold
Brukner and Khan[13]	15	40	4 min hot: 1 min cold	3–7	Start hot, end cold
Clover[25]	13–18	38–43	4 min hot: 1 min cold	15–20 min	
Walsh[26]	10–18	38–44	10 min hot, then 1 min cold: 4 min hot	30 min total	Start hot, end hot
Zuluaga et al.[27]	NA	45	3 min hot: 1 min cold	NA	Start cold, end cold

NA = data not available.

temperature widely used in this mode ranges from cool to thermoneutral, which for this review is considered to range from 16 to 35°C. Research into the physiological effect of water immersion generally has concentrated on the use of thermoneutral immersion and has ranged in immersion time from 5 minutes to 6 hours. This article only considered the effect of immersion over a maximum of 30 minutes to replicate a time similar to post-exercise recovery sessions. Unlike cryotherapy, thermotherapy or contrast therapy, the main effect of water immersion *per se* comes from the effect of hydrostatic pressure and perhaps to a lesser degree buoyancy, rather than temperature. The few studies into water immersion as a performance recovery method have concentrated on cryotherapy, thermotherapy and contrast therapy, combining both temperature and hydrostatic pressure effects.[7,8,16,17,28,29] To gain greater understanding of water immersion, the effects of hydrostatic pressure and temperature would be best studied in isolation and provide the focus for the remainder of this article.

2. Hydrostatic Pressure

When a body is immersed, water exerts a compressive force on the body called hydrostatic pressure. This pressure can cause the displacement of fluids within a person from the extremities towards the central cavity. This displacement of fluid may increase the translocation of substrates from the muscles, increase cardiac output, reduce peripheral resistance and increase the ability of the body to transport substrates. Additionally, the antigravity effect caused by buoyancy may reduce perception of fatigue and aide energy conservation. The following sections (sections 2.1–2.7) will explore the physiological effects of hydrostatic pressure during thermoneutral immersion.

2.1 Water Pressure

Air exerts pressure equally on all sides of the body. At sea level, the pressure exerted around the body equates to approximately 1013Pa. Water is >800 times denser than air; consequently, at an equal depth, water will create a greater pressure than air.[30] Due to this greater density, water produces the same pressure at a depth of only 10m as the entire atmosphere of air at sea level.[30]

When immersed in water, hydrostatic pressure acts on the body in relation to the depth of immersion. The amount of pressure that acts on a body is equal to:

$$P = P_{atm} + g \bullet \rho \bullet h$$

where P = water pressure; P_{atm} = atmospheric pressure (standard sea level 1013 hPa); g = gravity (9.81 m/sec^2); ρ = water density (1000 kg/m^3) and h = height of the water (m).

Water pressure does not correlate to the total weight of the water in a vessel, only to the depth. If the wall of a container is solid then the walls of the container exert a pressure on the water equal to the pressure of the water at that depth. This means that water pressure is a force per unit area and is transmitted equally throughout the water at a given level. On a body immersed in water, the pressure varies relative to depth. A body part such as a foot immersed at a depth of 1m would have 981Pa extra pressure acting on it, whereas at hip level (0.1m), only an extra 98.1Pa. To relate this external pressure to blood pressure measurement, for every 1cm depth of immersion, the pressure increases by 0.74mm Hg. The proportional change in pressure with depth causes an upward squeezing action on the body, which at 1m depth (74mm Hg) is almost equal to normal diastolic blood pressure (80mm Hg).

The human body is mostly water with the addition of some oil (fats) and proteins. Because water is essentially non-compressible, it occupies the same volume regardless of pressure.[31] When external pressure on the body increases, gas and fluid sub-

stances are displaced to lower pressure areas.[30,32,33] Therefore, a person standing in water experiences compression on the body acting inwards and upwards. During hip-level immersion, this 'squeezing' causes the displacement of fluid from the lower extremities of a person into the thoracic region. During head-out immersion, hydrostatic pressure on the central cavity reduces the residual air volume of the lungs increasing the displacement of fluids into the thorax.[32] It is the movement of these fluids that may enhance the ability of an athlete to recover from exercise.

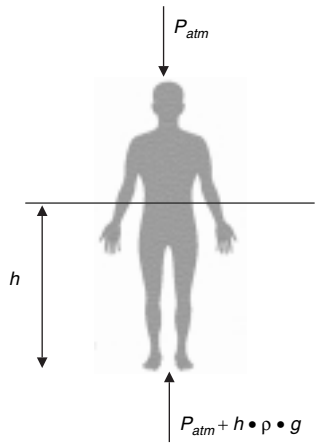

Fig. 1. Water upthrust. ***g*** = gravity; ***h*** = height; ρ = water density; P_{atm} = atmospheric pressure (standard sea level 1013 hPa).

2.2 Weightlessness and Perceived Fatigue

One significant consequence of water pressure being proportional to the immersion depth is that the body will weigh less when immersed in a liquid i.e. it is easier to lift a rock in water than it is on dry land. This is because water exerts a net upward force on the body immersed in it. This upward force helps to support all or part of the weight of the body immersed in it. The upward force exerted by a fluid on any object placed in it is called buoyancy or hydrostatic upthrust.

The force created by upthrust is calculated as:

$F = h \bullet \rho \bullet g \bullet A$

taking the calculation further, $F = V \bullet \rho \bullet g$

$F = m/g$

where h = height; ρ = water density; g = gravity; A = base area; V = immersed volume; and m = mass.

Hence there is a net upward pressure, giving rise to an upward force equal to upward pressure times horizontal base area (as shown in figure 1). In other words, any body partly or wholly immersed in a liquid, experiences an upthrust that is equal to the weight of the liquid displaced (Archimedes Principle). The greater the body density, the less buoyancy a person has, which is why people with higher fat mass (less density) are more buoyant than those who are lean. The effect of buoyancy is a reduction in the gravitational forces that act on the musculoskeletal system, allowing for a greater relaxation of gravitational muscles and conservation of energy. Such greater relaxation would appear to reduce perceived fatigue. A number of studies (see table II) have observed lower perception of fatigue after exercise during and after water immersion. While two authors[17,23] did not observe any significant difference in perceived fatigue, both did observe a moderate effect of lower sense of fatigue with water immersion compared with other recovery modes (Cohen's effect size = 0.50–0.89).

The decrease in the perception of fatigue may also be due to reduced neuromuscular responses during water immersion.[35-37] During water immersion, electromyographic activity produced during maximal contractions have been observed to reduce by 11–35%.[35,36] In their 2002 study, Pöyhönen and Avela[35] also observed a 13% decrease in maximal voluntary contraction force during immersion. Immersion may modify the peripheral processes associated with contraction and change central and/or neural command contractions,[37] or trigger inhibitory mechanisms. The reduced perception of fatigue may come then not only from a reduction in the neuromuscular activation required to maintain posture but also due to an overall reduction in neural

Table II. Water immersion and perception of fatigue

Study	Scale	Exercise	Recovery	Measurement timing	Main findings
Coffey et al.[17]	20-point RPE recovery scale	Maximal sprints	15 min contrast therapy, active or passive	4, 8, 12, 16, 20 min post-sprint. Pre- and post-second set of sprints	No significant difference between recoveries
Kuligowski et al.[22]	12cm graphic pain scale	DOMS-inducing arm curl	24 min warm water, cold water, contrast therapy or control	0, 24, 48, 72, 96h post-exercise	↓ pain perception with cold water and contrast therapy
Nakamura et al.[34]	5-point fatigue scale	10 min of submaximal cycling	10 min in 30°C or 38°C water bath, or control	Not specified	↓ fatigue for 30°C bathing
Sanders[16]	10-point CR scale	Repeated Wingate test	12 min contrast therapy, active or passive	3 and 7 min during recovery Post-recovery Post second exercise bout	↓ fatigue at all times with contrast therapy
Vaile et al.[23]	10cm VAS score	DOMS-inducing leg press	15 min contrast therapy or control	(i) 0 (ii) 24h (iii) 48h (iv) 72h	Both groups had increased pain. No significant difference
Viitasalo et al.[28]	10cm VAS score	Strength, plyometric and sprint training over 3d	20 min warm water immersion or control	Over 2 days post-training	↓ DOMS during immersion week

CR = Borg category ratio scale; **DOMS** = delayed onset of muscle soreness; **RPE** = rating of perceived exertion; **VAS** = visual analogue scale; ↓ = decreased.

transmissions. However, more research is required on whether water immersion does reduce neural efficiency, whether such reductions are due to weightlessness or hydrostatic pressure[35] and if there are any post-immersion effects.

2.3 Fluid Shifts

Under normal conditions, the body is comprised of 50–60% fluid, which is located either in the intracellular, interstitial (between cells) or intravascular (blood plasma) space (see figure 2). Typical intracellular, interstitial and plasma volumes are 35–40%, 11–15% and 4–5%, respectively, of bodyweight.[38] Fluid within these compartments acts as a vehicle for the transport and exchange of materials, such as metabolic wastes and nutrients, between the body and the external environment.

Movement of fluid and materials between the intravascular and extravascular space occur in the vascular capillaries. Fluid and substance movement across the capillaries occur via three processes: (i) diffusion; (ii) vesicular transport; and (iii) filtration-reabsorption. Diffusion is the movement of fluid/substances from a high concentration to a low concentration, whereas vesicular transport is active transport (requiring adenosine triphosphate) of substances across the vascular membrane. Diffusion occurs along all of the capillary membrane and accounts for the largest exchange of fluids and substances, whereas vesicular transport and filtration account for a small portion of fluid movement.[39] Filtration-reabsorption is the net movement of fluid due to the capillary-interstitial pressure gradient. Filtration is the net movement of fluid into the interstitial space at arteriolar ends of the capillaries, which is then reabsorbed at the venular ends of the capillaries. Approximately 2–4L of this fluid per day is not reabsorbed by the capillaries through filtration-reabsorption, but moves through the lymphatic vessels and drains into the subclavian veins.[40] Disruption in the balance of filtration-reab-

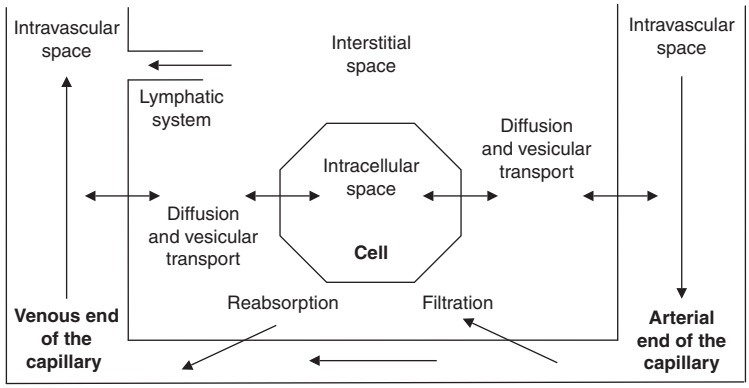

Fig. 2. Schematic diagram showing the intracellular-intravascular movement of fluid.

sorption, through effects such as physical trauma, lymph blockage or changes in pressure gradients, can cause an abnormal increase in interstitial fluid in localised areas, a condition called oedema, swelling, or inflammation.

It is well documented that water immersion causes a rise in central blood volume, which increases with the depth of immersion.[33,41-43] The increase in central blood volume is due to two effects: haemodilution (increased diffusion and reabsorption) and blood displacement. During immersion at hip level, haemodilution occurs as a result of negative transcapillary pressure in the legs. This pressure gradient causes a fluid shift from the interstitial to intravascular space in the legs.[42,44,45]

With immersion above hip level, additional increases in central blood expansion results as blood from the abdomen, which acts as a blood reservoir, is displaced.[42] Norsk et al.[46] studied changes in plasma concentration during head-out immersion and observed significant decreases in haematocrit and haemoglobin concentration with an associated 6.5 ± 1.9% (mean ± standard error [SE]) increase in plasma volume. Hinghofer-Szalkay et al.[47] observed similar results when using plasma densitometry to measure transvascular fluid shifts during immersion to the neck. After 30 minutes of thermoneutral immersion, the six men in the study had plasma volume increases of 11 ± 3%, with decreased blood and haematocrit densities (−1.5% and −1.0%, respectively). While plasma dilution occurred in these studies, the intravascular fluid shift was also accompanied by a plasma protein shift of albumin.

Stocks et al.[44] suggested that the increase in the extracellular fluid volume ultimately comes at the expense of intracellular fluid, although further investigation and verification of this hypothesis is required. Such fluid shifts would increase the intracellular-intravascular osmotic gradients and some intracellular constituents, such as metabolic wastes, may leave the cells and interstitial space to maintain an osmotic balance.[44,47] It is possible then that immersion may cause improvements in the translocation of substrates, which may help to increase the ability of an athlete to recover, as reduced transportation time could increase clearance of waste substrates.[16,17,34,48]

2.4 Exercise-Induced Muscle Oedema

Apart from assisting the possible removal of substances, the gradient between internal tissue hydrostatic pressure and capillary filtration pressure may also improve the reabsorption of interstitial fluids, reducing oedema.[29,49] An increase in the pressure gradient between the interstitial compartment of the legs and the intravascular space caused by hydro-

static pressure should reduce oedema in a similar fashion to compression stockings.[50,51]

Exercise causes a shift of plasma from the blood into the muscles, with this fluid movement being relative to the intensity of the exercise.[52-57] The mode of exercise does not appear to be a factor but rather the respondent increase in mean arterial pressure.[53] Researchers have observed that cycling at intensities of 30–120% of maximal oxygen uptake decreased blood plasma by 5–17% as fluid shifted intramuscularly.[54-58] However, during resistance training, plasma decreases of 8–14% have been observed in relation to an intensity range of 40–70% one repetition maximum (1RM).[53] The decrease in plasma volume observed by Knowlton et al.[53] during the resistance training correlated highly with an increasing mean arterial pressure ($r = -0.98$). However, fluid shifts from the vascular space reflected movement into active but not inactive muscle during exercise.[59] Using magnetic resonance imaging, pre- and post-resistance exercise (six sets of 10RM squats) Ploutz-Synder et al.[59] observed an increase in the cross-sectional area of the vasti and adductor muscle groups coinciding with a 22% decrease in plasma volume (measured by Evans blue dye). The coefficient of determination between the plasma decrease and volume increase in the adductor and vasti muscle groups was strong ($r^2 = 0.75$, $p = 0.0157$). Muscles that were less active during squats (rectus femoris and the hamstring muscle groups) had smaller non-significant increases in their cross-sectional area.

Oedema in response to exercise or muscle damage may increase both the transport route and compression of localised capillaries, reducing oxygen delivery to localised cells. With excessive muscle oedema, such an increase in transportation time can cause an increase in cellular damage or death.[49,60-62] A positively increased pressure gradient can reduce cellular infiltration by leukocytes and monocytes decreasing further tissue degeneration.[23,63,64] Such reduction in muscular inflammation may improve contractile function as well as lowering the levels of inflammatory cells and muscle enzymes circulating in the blood.[23,65] Reducing oedema may, therefore, decrease secondary damage to tissue, which in turn may increase the ability of an athlete to recover from muscle damaging exercise.[23,66]

2.5 Cardiac Response

The predominant effect of water immersion and the associated increase in central blood volume expansion is an increase in cardiac pre-load. Central blood volume expansion increases atrial pre-load and stroke volume. Increasing the depth of immersion causes greater stroke volume increases. Compared with non-immersion, at the level of the hips stroke volume has been reported to increase by 12–37%,[32,33] this increases to 38–67% at the level of the xiphoid process[5,12,32,33,45] and 28–95% during head-out immersion.[32,33,41,67-69] The effect size between each level of immersion from these studies ranged from moderate to very large (0.75–3.95).

When immersed in thermoneutral water to the level of the hips, heart rate has a tendency to decrease by approximately 4–6%.[32,33] Increasing depth of immersion to the xiphoid process has decreased heart rates by 11–18% compared with non-immersion.[5,12,32,33,45,70,71] However, rather than a linear decrease in heart rate with increasing immersion depth, the decrease in heart rate during head-out immersion (3–15%)[6,32,33,41,42,67-70] is less than that observed during immersion to the xiphoid process.

Individual heart rate response to immersion varies and decreases in heart rates have been non-significant in some studies.[5,41,42,68] While non-significant, a negative effect on heart rate was still apparent in the immersed subjects of these studies (see table III). The explanation for heart rate variance may be due to conflicting physiological feedback systems. Increasing mean arterial pressure causes arterial baroreceptors to bring about a reflex

Table III. Cardiac response of thermoneutral immersion compared with non-immersion ($p < 0.05$ unless otherwise stated)

Study	Immersion duration (min)	Change in stroke volume (%)	Change in heart rate (%)	Change in cardiac output (%)
Hip level immersion				
Farhi and Linnarsson[32]		11.9	−3.9	14.0
Löllgen et al.[33]		37.0	−5.7	29.2
Xiphoid process immersion				
Farhi and Linnarsson[32]		64.2	−10.5	48.0
Löllgen et al.[33]		67.1	−11.4	48.1
Bonde-Petersen et al.[5]	15	38.7	−14.5 NS	19.1
Gabrielsen et al.[45]	10	50.8	−10.6	32.6
Gabrielsen et al.[70]	10		−14.1	
Watenpaugh et al.[71]	30		−18.3	
Weston et al.[12]	15	50.0	−11.0	31.5
Head-out immersion				
Arborelius et al.[41]	10	28.3	−3.3 NS	28.9
Farhi and Linnarsson[32]		79.1	−6.6	66.0
Löllgen et al.[33]		79.5	−11.4	59.1
Gabrielsen et al.[70]	10		−15.3	
Johansen et al.[42]	5		−6.9 NS	
	10		−8.6	
	15		−8.6	
Park et al.[67]	30	54.7	−1.4	53.2
Shiraishi et al.[69]	30	62.1	−8.6	52.4
Sramek et al.[6]	10		−8.0	
Yun et al.[68] a	20	52.5	−1.7 NS	49.4
Yun et al.[68] b	20	56.4	−6.3 NS	48.7
Yun et al.[68] c	20	95.3	−2.3 NS	101.7

a Subjects = breath-hold divers (mean age 55y).

b Subjects = housewives (mean age 55y).

c Subjects = housewives (mean age 22y).

NS = non-significant.

to slow the heart, most likely to prevent abnormally high blood pressure levels. Opposing this sympathetic response, an increased atrial stretch caused by the greater central blood volume (most notably when the water level rises above the hips) stimulates atrial stretch receptors and increases heart rate through a neural reflex called the Bainbridge reflex.[72] Individual physiological variables (such as heart size) would then determine the dominant reflex. Generally speaking, however, the mean heart rate of subjects appears to decrease during short-term thermoneutral immersion.

Regardless of an individual's heart rate response, the increase in stroke volume ultimately causes an increase in cardiac output. Observed cardiac outputs (see table III) vary but have been reported to be approximately 14–29% at hip level,[32,33] 19–48% at the height of the xiphoid process[5,12,32,33,45] and 29–66% at head-out immersion.[32,33,41,67,69] Yun et al.[68] observed larger increases in cardiac output (102%) during head-out thermoneutral immersion, but was the only study in which the subjects were both female and Korean. Subjects used in immersion research have for the most part been male and European.

2.6 Peripheral Resistance and Blood Flow

Accompanying the increased cardiac output during immersion is a decrease in peripheral resistance implying that peripheral vasodilation occurs.[5,12,41,67,68] Total peripheral resistance (TPR) has been measured indirectly using the following calculations:

$TPR = (MAP - CVP)/\dot{Q}$[67,68]

$TPR = (MAP - \text{right atrial pressure})/\dot{Q}$[41]

$TPR = MAP/\dot{Q}$[5,12]

where MAP = mean arterial pressure, CVP = central venous pressure and $\dot{Q}$ = cardiac output.

Decreases in peripheral resistance of 27–51% have been reported[41,67,68] during head-out water immersion. Immersions at lower depths do not seem to reduce peripheral resistance. Gabrielsen et al.[70] determined intramuscular blood flow using a counting signal between two cadmium-telluride detectors to measure the washout of injected ^{113}Xenon-saline (k). From the determined blood flow, peripheral resistance was determine as TRP = MAP/k. Muscular vascular resistance did not change significantly with immersion to the xiphoid process but decreased by approximately 15% with immersion to the neck. Similarly, Gabrielsen et al.[70] observed that during immersion to the xiphoid process, blood flow did not significantly increase but increased by 49 ± 16% (mean ± SE) during head-out immersion. Bonde-Petersen et al.[5] and Weston et al.[12] also observed no significant decrease in total peripheral resistance during immersion to the xiphoid process.

A direct method of measuring venous tone during immersion is occlusion plethysmography. Echt et al.[43] used this method to determine venous elasticity during a 3-hour immersion to the neck of subjects. In the first 15 minutes of immersion, the venous volume elasticity coefficient reduced from 16.6 to 13.5mm Hg/mL/100g tissue, a decrease of 19%. Venous tone slowly reduced a further 30% by the third hour of immersion and persisted for 1 hour post-immersion. However, reduction in peripheral resistance would seem to occur during head-out immersion only.[41,43,67,68] While studies of immersion at a depth under chin level have shown a decrease in peripheral resistance and increased blood flow (with small to very large effect sizes; −0.59 to −2.62) the findings of these studies compared with non-immersion have been non-significant.[5,12]

With the increase in cardiac output, some reduction in peripheral resistance and vasodilation, increased blood flow may result throughout the body. During erect head-out immersion, dogs have responded with large increases (>50%) in blood flow through the liver, intestinal tract, pancreas, spleen, renal cortex and skeletal muscle.[73] If the responses displayed by dogs can be extrapolated to humans, greater organ and muscle blood flow may allow improved removal of metabolites and an increased ability to replenish energy stores. However, such extrapolation may not be possible. Blyden et al.[74] observed that the clearance of lidocaine (lignocaine) in humans was unaltered by immersion to the neck, indicating no change in splanchnic blood flow. Epstein et al.,[75] using the clearance of p-aminohippuric acid and inulin to determine changes in renal plasma flow and glomerular filtration rate, supported the findings of Blyden et al.[74] With greater cardiac output, lower peripheral resistance and vasodilation (at head-out immersion at least), it could be logically assumed that an increase in blood flow through the muscles and perhaps the organs would occur. Results from Epstein et al.[75] and Blyden et al.[74] imply that renal blood flow is unaffected, although their research only analysed the clearance of certain chemicals not actual blood flows. Other metabolites, notably blood lactate, have an increased clearance rate when subjects have been partially immersed in water,[16,17,19,34] indicating that blood flow through the muscle beds increase.[74] Whether blood flow increases to organs other than

the kidneys is unknown and requires further investigation.

2.7 Summary of Findings

Hydrostatic pressure during water immersion aids the return of fluid from the muscles into the blood.[29] If plasma volume increases due to a fluid shift from the interstitial space, the translocation of metabolic waste may be improved due to blood dilution and improved diffusion gradients.[38] Additionally, the increase in blood volume increases stroke volume and cardiac output, increasing blood flow through the body. Improved diffusion gradients and increased blood flow through the body could increase the ability of an athlete to metabolise waste products and enhance recovery from exercise by reducing transport time of substrates.[76] The increased clearance of blood lactate in subjects that have been immersed in water following exercise[16,17,19,20,34] would support such a theory. Sports that cause a large depletion in muscle energy stores or cause large increases in metabolites (high-intensity anaerobic power-endurance or endurance sports) may therefore benefit from water immersion, especially in tournament situations where an athlete may compete a number of times within a few days. Hydrostatic pressure may also aide the reduction exercise-induced muscle oedema. Excessive muscle oedema may cause capillary constriction and increase substrate transportation time leading to greater cellular damage or death. Reduction of oedema may therefore improve nutrient delivery and speed recovery from exercise that has induced muscle damage. Where fluid shifts could improve athletic recovery physiologically, buoyancy may provide a psychological enhancement to recovery as some studies have observed a lower perception of fatigue in immersed subjects.

3. Temperature

The previous sections have considered the physiological response that thermoneutral immersion in water can have on a person. This article has concentrated on the immersion of subjects in thermoneutral water because of the abundance of research in this area. Colder, warmer or a variation in water temperature may alter these physiological responses, providing either additional benefits or detriment to any possible recovery enhancement.

3.1 Cold Temperature Effects

Thermoneutrality is considered to occur in a small range (35°C) in which subjects can maintain their core temperature for at least 1 hour.[77] Critical cold temperatures at which an individual cannot maintain core temperature for an hour ranges from 30 to 34°C depending on cutaneous fat.[78] However, core temperatures can be maintained during head-out immersion at temperatures as low as 18°C for up to 30 minutes.[78,79]

Cooler temperatures do have some effect on the physiological responses of the body. As water temperature decreases, heart rate reduces,[6,12] which decreases cardiac output.[5,6,67] Additionally, arterial blood pressure and peripheral resistance also increase.[5,6,67] The increase in peripheral resistance is due to blood being redirected from the periphery to maintain core temperature.[5,80] Oxygen consumption and metabolism also increase to maintain core temperatures.[6,67]

Reduced permeability of cellular, lymphatic and capillary vessels due to localised vasoconstriction reduces fluid diffusion into the interstitial space.[81,82] This reduced fluid diffusion can assist in reducing acute inflammation from muscle damage.[18] This in turn can reduce pain, swelling and the loss of force generation that is also associated with inflammation.[83] Hence, cold is often used in the

treatment of inflammation to improve the rehabilitation process.[18]

One metabolite that is used as a marker of muscle damage is the level of creatine kinase in the blood.[84] Exercise-induced injury is thought to increase the permeability of cells increasing the diffusion of myoproteins such as creatine kinase into the extracellular space.[81,82,85] Cold water immersion decreases the level of creatine kinase in the blood after exercise-induced muscle damage.[81,86] Lower creatine levels are attributed to a decrease in cellular, lymphatic and capillary permeability caused by vasoconstriction induced by the cooler temperature.[81,82] However, caution is warranted when applying the presence of creatine kinase in the blood as an indication of muscle damage. Levels of creatine kinase in the blood reflect not only creatine kinase release rate but also the removal rate. Exercise-induced haemoconcentration or haemodilution and alterations of tissue clearance due to blood-flow or function will affect creatine concentration in the blood. Creatine kinase may not then accurately indicate muscle damage or fatigue.[85]

Neural components are also affected by the cold. Cooling of tissue decreases the rate of transmission along neurons by decreasing the production of acetylcholine[87] and possibly stimulates superficial inhibitory cells that regulate the impulse of pain perception to the CNS.[88] Reduction of nerve impulse transmission by cold has two effects: (i) reduced level of pain perception (analgesia); and (ii) reduction in muscle spasm.[88,89] While reduction in pain may be of benefit, a reduced neural transmission may decrease muscular contractile speed[87,90,91] and force-generating ability of an athlete post-application.[91-93] Performance may then be initially inhibited if exercise is performed shortly after cold immersion.

There are risks to athletes whom may be immersed in cold water, dependent on the temperature extreme and amount of the body immersed. Generally, sudden severe cold immersion of a large portion of the body can produce hyperventilation, which may cause ventilation to increase up to five times the resting rate.[94] The decrease in arterial carbon dioxide caused by hyperventilation may lead to blood acidosis and impaired consciousness, even in fit young people.[94,95] Additionally, sudden cold immersion can cause tachycardia and acute peripheral vasoconstriction producing sudden loss of consciousness, convulsions, ventricular ectopy, cardiac arrest and death.[94,95]

While rare, some people also have cold hypersensitivity and can be at risk if body parts are suddenly immersed in cold water. Conditions consist of allergic and possible anaphylactic reactions, Raynaulds phenomenon, and paroxysmal cold haemoglobinuria.[95,96] Allergic reactions can consist of rashes and wheals, which may advance into anaphylaxis. The signs and symptoms of fully developed anaphylaxis include hypotension, syncope and vascular collapse and can lead to death.[95,96] Raynaulds phenomenon is peripheral vasoconstriction that leads to numbness, tingling and burning pain,[97] while paroxysmal cold haemoglobinuria is a rare and potentially life-threatening affliction that causes the release of haemoglobin from red blood cell into the urinary system causing acute transient anaemia.[98]

While cold hypersensitivity is rare, care should be taken when using cold immersion on athletes. Very cold water temperatures may be best only in a localised manner to treat acute injuries and reduce inflammation, rather than being used as a recovery strategy.

3.2 Hot Water Temperatures

Considering the use of thermotherapies such as hot baths in physiotherapy, there is a lack of research-based literature on the effect that superficial heat application has on a person. Apart from basic physiological responses, much of the literature

comes from texts that cite other texts or is based on anecdotal information.

Superficial application of heat increases subcutaneous and cutaneous tissue temperature while tissue temperature at depths >2cm remains unchanged.[99] An increase in superficial tissue temperature causes an increase in the cutaneous blood flow, over short durations, due to peripheral vasodilation.[5,80] Heart rate also increases in response to hot water immersion.[5,12] This increase in heart rate may reduce stroke volume due to lack of cardiac filling time, but overall cardiac output increases compared with thermoneutral immersion.[12]

The increase in cardiac output and a lower peripheral resistance allows an increase in subcutaneous and cutaneous blood flow.[5,12,100] An increase in subcutaneous and cutaneous blood flow increases the permeability of cellular, lymphatic and capillary vessels.[101] Increased permeability increases metabolism, nutrient delivery and waste removal from the cells that can increase healing.[18,102] However, for short-duration superficial application, these changes are not likely to occur within the muscle, but rather within the skin.[5,103] Additionally, Bonde-Petersen et al.[5] observed that while subcutaneous and cutaneous blood flow increased, blood flow through the muscle may decrease compared with thermoneutral immersion. Lower water temperatures may then have greater benefits in substrate transportation within a muscle. However, the effect size Bonde-Petersen et al.[5] observed in blood flow difference between thermoneutral and hot water immersion was small (0.29).

Superficial heat may also increase neural transmission[104] proprioception and improve reaction time.[9] Other proposed benefits of thermotherapy include increased muscle elasticity, joint extensibility, analgesia and reduction of muscle spasm.[13,17,102,105-107] While a large amount of anecdotal support is available, little research-based evidence has been found to support these claims. Of studies that have analysed the effect of the superficial application of heat, flexibility was not enhanced unless accompanied with stretching.[108-111] A report by Bigos et al.[112] on back pain and thermal applications concluded that not enough data existed to recommend the use of heat in pain reduction. More recent research[113-115] has observed that pain may be reduced if heat is applied continuously (8 hours/day) over a long term (2–5 days). However, research is lacking in the short-term application of heat and possible effects on pain.

There are contraindications to immersion in hot water. The most obvious is the possibility of burns due to high water temperatures. At 45–50°C protein denaturation occurs and immersion water temperatures should be below this range.[102] Superficial heat application also causes an inflammatory response and swelling,[18,116-118] which may prolong recovery time.[18,91] Cote et al.[18] observed increased oedema in 30 patients with first- and second-degree ankle sprains when 20 minutes of hot water immersion was applied each day over 3 days. Volumetric increase in ankle size was 25% with hot water immersion of the foot compared with 3% in patients receiving cold water immersion. The effect size of this difference was large (1.95). If heat increases oedema in sprains, it can be speculated that in may also increase muscle inflammation.[119]

Hot water immersion of a large portion of the body can produce a potentially dangerous strain on the cardiovascular system causing ectopic beats, hypotension, heat syncope, excessive tachycardia and even death.[120,121] Heat syncope is fainting and giddiness due to the collapse of vasomotor control and a decrease in blood pressure owing to rapid vasodilation.[11] Care is warranted with athletes who have acute injuries, oedema, vascular disease, wounds or infections as these can be exacerbated with heat application and increase potential risks.[102]

3.3 Contrasting Temperature

Contrast therapy has been considered to enhance athletic recovery through:[14-17]

- stimulating area-specific blood flow;
- increasing blood lactate removal;
- reducing inflammation and oedema;
- stimulating circulation;
- relieving stiffness and pain;
- increasing range of motion;
- reducing delayed onset of muscle soreness.

One reason behind such possible benefits to recovery is that contrast therapy may mimic one of the mechanisms attributed active recovery without the same energy demands.[2,14,29,122] Recovery using active low-intensity exercise is considered to enhance recovery compared with passive modalities.[123-125] One theory for increased recovery is alternating muscular contractions acting in a pumping/squeezing action. Low-intensity repetitive mechanical 'squeezing' by the muscles during contraction-relaxation may increase the translocation and removal of metabolites, such as lactate, and reduce intracellular fluid volume.[125] Much of the research and literature regarding contrast therapy perpetrate the theory of contrast therapy causing a similar action with vaso-pumping. Alternating vasoconstriction and vasodilation is thought to act in a comparable way to muscle pumping, increasing blood flow and metabolite removal, enhancing recovery.[14,29,122] However, vaso-pumping would seem unlikely to occur at a level that could act effectively in this manner.

During contrast therapy, each alternation of temperature generally lasts for 30–120 seconds and is repeated 2–5 times. Vaso-pumping would then occur at a slow rate and only with 2–5 'pumps' over a period of around 2–10 minutes (at a low frequency such as 0.03–0.008Hz). Under active recovery, such as light-running, muscular pumping would occur at a rate of around 2Hz. If vaso-pumping does occur during contrast therapy, it would seem unlikely to cause a great effect at such a slow frequency.

Another point to consider with vaso-pumping is that intramuscular temperature has not been observed to change with alternating contrasts, only subcutaneous temperature.[15,99] A study by Higgins and Kaminski[21] observed that at a 4cm depth, intramuscular temperature also did not fluctuate with repeated 1-minute immersions into cold water (after 4 minutes of warm whirlpool therapy), but gradually increased by 0.85 ± 0.60°C over 31 minutes. If temperature does not change at deep tissue levels with alternating immersion, any vaso-pumping would then be likely to occur at a subcutaneous level only. To aide recovery and intramuscular waste removal by vaso-pumping, temperature changes would surely be required at a deeper tissue level. Additional to the unlikely vaso-pumping within deep tissue, the sudden immersion into an icy bath from heat may not cause vasoconstriction. During high body temperatures, as may occur after intense athletic exertion and hot water immersion, the sudden immersion into cold may cause cutaneous vasodilation rather than vasoconstriction in a shock response.[5,126]

To date, no research was found that had observed or measured any form of alternating vasodilation-constriction caused by contrast therapy. If vaso-pumping does not occur, another explanation for elevated blood lactate removal during contrast therapy must exist. One area that is little discussed in contrast therapy literature that may explain increased removal of wastes from the body is simply the hydrostatic pressure caused by immersion in water.

Similar to heat application, contrast therapy may be harmful to athletes by causing inflammation. Compared with cold water immersion, Cote et al.[18] observed a 26.5% increase in oedema using contrast therapy on patients with first- and second-degree ankle sprains. The effect size of this difference was

large (2.05). However, Vaile et al.[23] observed that 15 minutes of contrast therapy after muscle damaging eccentric leg presses reduced thigh volume oedema significantly over 72 hours compared with passive recovery. Mean (± standard deviation) thigh measurements increased by 2.3 ± 0.8% with passive recovery compared with 0.6 ± 0.6% in the contrast therapy group. The reason for the benefit observed in the study of Vaile et al.[23] may be due to the large portion of the body that was immersed (immersion to the gluteal fold), rather than the effect of temperature. A greater physiological response would have occurred due to the higher hydrostatic pressures compared with the study of Cote et al.[18] where only the foot was immersed. Alternatively, in the study of Cote et al.,[18] subjects had acute injuries rather than induced muscle damage, which may account for the possible effect difference of the contrast therapy. More research is required before any conclusion can be drawn on whether contrast therapy is harmful or beneficial to oedema. Other contraindications of contrast therapy are likely to include those of both the hot and cold temperatures.[102,120] Care is warranted when using extremes of temperature in contrast immersion.

4. Conclusions and Recommendations

Hydrostatic pressure from water immersion causes an inward and upward displacement of body fluid. This action reduces oedema, increases extracellular fluid transfer into the vascular system and increases cardiac output. Greater cardiac output increases blood flow through the body and in response to increased arterial pressure vasodilation may occur. Increased blood flow through the body may assist in the metabolism of waste products that accumulate during exercise by reducing transport time. Additionally, reductions in oedema due to fluid shifts may assist short-term in maintaining muscle function and assist muscular repair. Weightlessness when immersed in water decreases the perception of fatigue, which may be due to reduced neuromuscular signal magnitudes and energy conservation.

Decreasing water temperature may reduce some of the physiological responses associated with hydrostatic changes. The body responds to cold by reducing heart rate and cardiac output, and inducing vasoconstriction. This response reduces peripheral blood flow and conserves body core temperatures. Additionally, central metabolism increases to maintain the core temperature, which increases the production of waste products and erodes energy stores. However, cold also assists the reduction of oedema by increasing vasoconstriction and lowering peripheral metabolism, which may reduce secondary cellular death due to muscular damage. The analgesic effect of cold is likely due to reduced neural transmission magnitude and speed. Heat and contrast therapy provide lesser possible benefits to the recovery process by increasing oedema and increasing the energy requirement due to an increased metabolic rate. Immersion in cool to thermoneutral water may provide the best option for recovery unless muscle sprains or strains have occurred, in which case cold water immersion may provide greater benefit. Extremes of water temperature have contraindications and cool to thermoneutral immersion may provide both a safer and more beneficial immersion temperature range.

Physiologically speaking, hydrostatic pressure would seem the mechanism that could benefit exercise recovery. Ultimately, the aim of the recovery process is to enhance future performance. To date, a small amount of research has been conducted into the use of water immersion as an exercise recovery strategy.[7,8,16,17,23,28,76] The varied methodology and observations of these studies provide an unclear picture of whether water immersions could provide a benefit to exercise recovery. Numerous factors such as duration, water temperature, immersion depth, exercise type and intensity, and timing between exercise and recovery sessions require study.

Of the studies conducted, there would appear to be no harm in using water immersion as a recovery strategy and a possible benefit[7,23,28] to future performance. Increases in blood plasma fraction (movement of interstitial-intravascular fluid) during immersion have been observed to take least 10 minutes;[42,47] therefore, as a possible recovery strategy, immersions should be of at least 10 minutes duration. However, more research incorporating performance measures and water immersion needs to be conducted to determine if water immersion post-exercise causes any worthwhile performance benefit.

Acknowledgements

The authors would like to thank the Division of Sport and Recreation and the Alumni Association of Auckland University of Technology, Auckland, New Zealand, for financial support during the writing of this study. The authors have no conflicts of interest that are directly relevant to the contents of this review.

References

1. Bender T, Karagülle Z, Bálint GP, et al. Hydrotherapy, balneotherapy, and spa treatment in pain management. Rheumatol Int 2004 [online]. Available from URL: http://springerlink.metapress.com [Accessed 2004 Aug 5]
2. Calder A. Recovery strategies for sports performance. USOC Olympic Coach E-Magazine 2003; 2003 Aug 1-Oct 31 [online]. Available from URL: http://coaching.usolympicteam .com [Accessed 2004 Aug 17]
3. Netball New Zealand. Recovery strategies for a tournament environment [online]. Available from URL: http://images.tvnz.co.nz/netballnz/thegame/tournament_recovery.pdf [Accessed 2004 May 1]
4. Low J, Reed A. Electrotherapy explained: principles and practice. 2nd ed. Oxford: Butterworth and Heinemann, 1994
5. Bonde-Petersen F, Schultz-Pedersen L, Dragsted N. Peripheral and central blood flow in man during cold, thermoneutral, and hot water immersion. Aviat Space Environ Med 1992; 63: 346-50
6. Sramek P, Simeckova M, Jansky L, et al. Human physiological responses to immersion into water of different temperatures. Eur J Appl Physiol 2000; 81: 436-42
7. Lane KN, Wenger HA. Effect of selected recovery conditions on performance of repeated bouts of intermittent cycling separated by 24 hours. J Strength Cond Res 2004; 18 (4): 855-60
8. Burke DG, MacNeil SA, Holt LE, et al. The effect of hot or cold water immersion on isometric strength training. J Strength Cond Res 2003; 14 (1): 23-5
9. Burke DG, Holt LE, Rasmussen RL, et al. Effects of hot or cold water immersion and modified proprioceptive neuromuscular facilitation flexibility exercise on hamstring length. J Athl Train 2001; 36 (1): 16-9
10. Clarke DH. Effect of immersion in hot and cold water upon recovery of muscular strength following fatiguing isometric exercises. Arch Phys Med Rehabil 1963; 44: 565-8
11. Greenleaf JE, Kaciuba-Uscilko H. Acclimatization to heat in humans. Moffett Feild (CA): National Aeronautics and Space Administration, Ames Research Centre, 1989: 41
12. Weston CEM, O'Hare JP, Evans JM, et al. Haemodynamic changes in man during immersion in water at different temperatures. Clin Sci 1987; 73: 613-6
13. Brukner P, Khan K. Clinical sports medicine. 2nd ed. Sydney: McGraw-Hill, 2001
14. Cochrane R. Alternating hot and cold water immersion for athlete recovery: a review. Phys Ther Sport 2004; 5: 26-32
15. Myrer JW, Draper DO, Durrant E. Contrast therapy and intramuscular temperature in the human leg. J Athl Train 1994; 29 (4): 318-24
16. Sanders J. Effect of contrast-temperature immersion on recovery from short-duration intense exercise [dissertation]. Canberra: University of Canberra, 1996
17. Coffey V, Leveritt M, Gill N. Effect of recovery modality on 4-hour repeated treadmill running performance and changes in physiological variables. J Sci Med Sport 2004; 7 (1): 1-10
18. Cote DJ, Prentice WE, Hooker DN, et al. Comparison of three treatment procedures for minimizing ankle sprain swelling. Phys Ther 1988; 68 (7): 1072-6
19. Hamlin MJ, Magson P. The effects of post-exercise hydrotherapy on blood lactate and performance recovery in netball players. In: 2002 Conferences of the Australasian College of Sports Physicians, Sports Medicine New Zealand, and Sport Science New Zealand; 2002 Oct 30, Christchurch, New Zealand
20. Hamlin MJ, Sheen AM. The effect of contrast-temperature water therapy on performance recovery of rugby players. In: Pre-Olympic Congress: sport science thru the ages: challenges in the new millennium; 2004 Aug 5-11; Thessaloniki, Greece: International Council of Sport Science and Physical Education, Aristotle University of Thessaloniki, Department of Physical Education and Sport Science, 2004: 235-6
21. Higgins D, Kaminski TW. Contrast therapy does not cause fluctuations in human gastronemius intramuscular temperature. J Athl Train 1998; 33 (4): 336-40
22. Kuligowski LA, Lephart SM, Giannantonio FP, et al. Effects of whirlpool therapy on the signs and symptoms of delayed-onset muscle soreness. J Athl Train 1998; 33 (3): 222-8
23. Vaile J, Blazevich AJ, Gill N. The effect of contrast therapy on symptoms of delayed onset muscle soreness. Hamilton, New Zealand: Waikato Institute of Technology, 2004
24. Briggs J. Sports therapy: theoretical and practical thoughts and considerations. Champaign (IL): Human Kinetics, 2001
25. Clover J, editor. Sports medicine essentials: core concepts in athletic training and fitness instruction. Orange (CA): Carrer Publishing, 2001
26. Walsh MT. Hydrotherapy: the use of water as a therapeutic agent. In: Michlovitz SL, editor. Thermal agents in rehabilitation. Philadelphia (PA): FA Davis Company, 1996

27. Zuluaga M, Briggs C, Carlisle J, et al., editors. Sports physiotherapy: applied science and practice. Melbourne: Churchill Livingstone, 1995
28. Viitasalo JT, Niemela K, Kaappola R, et al. Warm underwater water-jet massage improves recovery from intense physical exercise. Eur J Appl Physiol 1995; 71: 431-8
29. Vaile J. The effect of recovery strategy on symptoms of delayed onset of muscle soreness (DOMS). Hamilton, New Zealand: Waikato Institute of Technology, 2003
30. Bove AA. Medical disorders related to diving. J Intensive Care Med 2002; 17: 75-86
31. Chaplin M. Water structure and behaviour [online]. Available from URL: http://www.lsbu.ac.uk/water/index.html [Accessed 2005 Mar 14]
32. Farhi LE, Linnarsson D. Cardiopulmonary readjustment during graded submersion in water at 35°C. Respir Physiol 1977; 30: 35-50
33. Löllgen H, Nieding GV, Koppenhagen K, et al. Hemodynamic response to graded water immersion. Klin Wochenschr 1981; 59: 623-8
34. Nakamura K, Takahashi H, Shimai S, et al. Effects of immersion in tepid bath water on recovery from fatigue after submaximal exercise in man. Ergonomics 1996; 39 (2): 257-66
35. Pöyhönen T, Avela J. Effect of head-out water immersion on neuromuscular function of the plantarflexor muscles. Aviat Space Environ Med 2002; 73 (12): 1215-8
36. Pöyhönen T, Keskinen KL, Hautala A, et al. Human isometric force production and electromyogram activity of knee extensor muscles in water and on dry land. Eur J Appl Physiol Occup Physiol 1999; 80 (1): 52-6
37. Koryak Y. 'Dry' immersion induces neural and contractile adaptations in the human triceps surae muscle. Environ Med 2002; 46 (1-2): 17-27
38. Lassiter WE, Gottachalk CW. Volume and components of the body fluids. In: Mountcastle VB, editor. Medical physiology. St Louis (MO): CV Mosby, 1980
39. Pappenheimer JR. Passage of molecules through capillary walls. Physiol Rev 1953; 33 (3): 387-423
40. Milnor WR. Capillaries and lymphatic vessels. In: Mountcastle VB, editor. Medical physiology. St Louis: CV Moby, 1980
41. Arborelius M, Baildin UL, Lilja B, et al. Hemodynamic changes in man during immersion with the head above water. Aerospace Med 1972; 43: 592-8
42. Johansen LB, Jensen TUS, Pump B, et al. Contribution of abdomen and legs to central blood volume expansion in humans during immersion. J Appl Physiol 1997; 83 (3): 695-9
43. Echt M, Lange L, Gauer OH, et al. Changes of peripheral venous tone and central transmural venous pressure during immersion in a thermo-neutral bath. Pflugers Arch 1974; 352: 211-7
44. Stocks JM, Patterson MJ, Hyde DE, et al. Effects of immersion water temperature on whole-body fluid distribution in humans. Acta Physiol Scand 2004; 182: 3-10
45. Gabrielsen A, Pump B, Bie P, et al. Atrial distention, haemodilution, and acute control of renin release during water immersion in humans. Acta Physiol Scand 2002; 174: 91-9
46. Norsk P, Bonde-Petersen F, Warberg J. Central venous pressure and plasma arginine vasopressin during water immersion in man. Eur J Appl Physiol 1985; 54: 71-8
47. Hinghofer-Szalkay H, Harrison MH, Greenleaf JE. Early fluid and protein shifts in men during water immersion. Eur J Appl Physiol Occup Physiol 1987; 56 (6): 673-8
48. Tomasik M. Effect of hydromassage on changes in blood electrolyte and lactic acid levels and haematocrit value after maximal effort. Acta Physiol Pol 1983; 34 (2): 257-61
49. Friden J, Lieber RL. Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components. Acta Physiol Scand 2001; 171: 321-6
50. Partsch H, Winiger J, Lun B. Compression stockings reduce occupational leg swelling. Dermatol Surg 2004; 30 (5): 737-43
51. Jonkera MJ, de Boera EM, Adèrb HJ, et al. The oedema-protective effect of lycra support stockings. Dermatology 2001; 203 (4): 294-8
52. Collins MA, Cureton KJ, Hill DW, et al. Relation of plasma volume change to intensity of weight lifting. Med Sci Sports Exerc 1989; 21 (2): 178-85
53. Knowlton RG, Hetzler RK, Kaminsky LA, et al. Plasma volume changes and cardiovascular responses associated with weight lifting. Med Sci Sports Exerc 1987; 19 (5): 464-8
54. Miles DS, Sawka MN, Glaser RM, et al. Plasma volume shift during progressive arm and leg exercise. J Appl Physiol 1983; 54: 491-5
55. Hildebrandt W, Schutze H, Stegemann J. Cardiovascular limitations of active recovery from strenuous exercise. Eur J Appl Physiol 1992; 64: 250-7
56. Gillen CM, Lee R, Mack GW, et al. Plasma volume expansion in humans after a single intense exercise protocol. J Appl Physiol 1991; 71 (5): 1914-20
57. Green HJ, Thomson JA, Ball ME, et al. Alterations in blood volume following short-term supramaximal exercise. J Appl Physiol 1984; 56 (1): 145-9
58. Mohsenin V, Gonzalez RR. Tissue pressure and plasma oncotic pressure during exercise. J Appl Physiol Respir Environ Exerc Physiol 1984; 56 (1): 102-8
59. Ploutz-Snyder LL, Convertino VA, Dudley GA. Resistance exercise-induced fluid shifts: change in active muscle size and plasma volume. Am J Physiol 1995; 269: R536-R43
60. Tiidus PM. Radical species in inflammation and overtraining. Can J Physiol Pharmacol 1998; 76 (5): 533-8
61. Northoff H, Berg A, Weinstock C. Similarities and differences of the immune response to exercise and trauma: the IFN-γ concept. Can J Physiol Pharmacol 1998; 76 (5): 497-504
62. Shephard RJ, Shek PN. Immune responses to inflammation and trauma: a physical training model. Can J Physiol Pharmacol 1998; 76 (5): 469-72
63. Mishra DK, Friden J, Schmitz MC, et al. Anti-inflammatory medication after muscle injury: a treatment resulting in short-term improvement but subsequent loss of muscle function. J Bone Joint Surg Am 1995; 77 (10): 1510-9
64. Lecomte JM, Lacroix VJ, Montgomery DL. A randomized controlled trial of the effect of naproxen on delayed onset muscle soreness and muscle strength. Clin J Sport Med 1998; 8 (2): 82-7

65. Cesari M, Penninx BW, Pahor M, et al. Inflammatory markers and physical performance in older persons: the InCHIANTI study. J Gerontol 2004; 59A (3): 242-8
66. Sayers SP, Clarkson PM, Lee J. Activity and immobilization after eccentric exercise I: recovery of muscle function. Med Sci Sports Exerc 2000; 32 (9): 1587-92
67. Park KS, Choi JK, Park YS. Cardiovascular regulation during water immersion. Appl Human Sci 1999; 18 (6): 233-41
68. Yun SH, Choi JK, Park YS. Cardiovascular responses to head-out water immersion in Korean women breath-hold divers. Eur J Appl Physiol 2004; 91: 708-11
69. Shiraishi M, Schou M, Gybel M, et al. Comparisons of acute cardiovascular responses to water immersion and head-down tilt in humans. J Appl Physiol 2002; 92: 264-8
70. Gabrielsen A, Videbæk R, Johansen LB, et al. Forearm vascular and endocrine responses to graded water immersion in humans. Acta Physiol Scand 2000; 169: 87-94
71. Watenpaugh DE, Pump B, Bie P, et al. Does gender influence human cardiovascular and renal responses to water immersion? J Appl Physiol 2000; 89: 621-8
72. Hakumaki MO. Seventy years of the Bainbridge reflex. Acta Physiol Scand 1987; 130 (2): 177-85
73. Khosla SS, DuBois AB. Fluid shifts during initial phase of immersion diuresis in man. J Appl Physiol 1979; 46 (6): 703-8
74. Blyden G, Silverstein F, Epstein M, et al. Lidocaine pharmacokinetics during water immersion in normal humans. J Appl Physiol 1989; 66 (1): 57-60
75. Epstein M, Levinson R, Loutzenhiser R. Effects of water immersion on renal hemodynamics in normal man. J Appl Physiol 1976; 41 (2): 230-3
76. Clark MG, Rattigan S, Newman JMB, et al. Vascular control of nutrient delivery by flow redistribution within muscle: implications of exercise and post-exercise muscle metabolism. Int J Sport Med 1998; 19: 391-400
77. Craig AB, Dvorak M. Thermal regulation of man exercising during water immersion. J Appl Physiol 1968; 25 (1): 28-35
78. Toner MM, Sawka MM, Holden WL, et al. Effects of body mass and morphology on thermal response in water. J Appl Physiol 1986; 60 (2): 521-5
79. Tikuisis P, Jacobs I, Moroz D, et al. Comparison of thermoregulatory responses between men and women immersed in cold water. J Appl Physiol 2000; 89: 1410-1
80. Knight KL, Londeree BR. Comparison of blood flow in the ankle of uninjured subjects during therapeutic applications of heat, cold, and exercise. Med Sci Sports Exerc 1980; 12 (3): 76-80
81. Eston R, Peters D. Effects of cold water immersion on the symptoms of exercise-induced muscle damage. J Sports Sci 1999; 17: 231-8
82. Enwemeka CS, Allen C, Avila P, et al. Soft tissue thermodynamics before, during, and after cold pack therapy. Med Sci Sports Exerc 2001; 34 (1): 45-50
83. Smith LL. Acute inflammation: the underlying mechanism in delayed onset muscle soreness. Med Sci Sports Exerc 1990; 23 (5): 542-51
84. Rawson ES, Gunn B, Clarkson PM. The effects of creatine supplementation on exercise-induced muscle damage. J Strength Cond Res 2001; 15 (2): 178-84
85. Warren GL, Lowe DA, Armstrong RB. Measurement tools used in the study of eccentric contraction-induced injury. Sports Med 1999; 27 (1): 43-59
86. Howatson G, Van Someren KA. Ice massage. J Sports Med Phys Fitness 2003; 43 (4): 500-5
87. Abramson DI, Chu LSW, Tuck S, et al. Effect of tissue temperatures and blood flow on motor nerve conduction velocity. JAMA 1966; 198 (10): 156-62
88. Sauls J. Efficacy of cold for pain: fact or fallacy? The Online Journal of Knowledge Synthesis for Nursing 1999; 6: 8 [online]. Available from URL: http://www.stti.iupui.edu/library/ojksn/articles/060008.pdf [Accessed 2002 Oct 15]
89. Washington LL, Gibson SJ, Helme RD. Age-related differences in the endogenous analgesic response to repeated cold water immersion in human volunteers. Pain 2000; 89: 89-96
90. Howard RL, Kraemer WJ, Stanley DC, et al. The effects of cold water immersion on muscle strength. J Strength Cond Res 1994; 8 (3): 129-33
91. Rutkove SB. Effects of temperature on neuromuscular electrophysiology. Muscle Nerve 2001; 24: 867-82
92. Yona M. Effects of cold stimulation of human skin on motor unit activity. Jpn J Physiol 1997; 47 (4): 341-8
93. Johnson DJ, Leider FE. Influence of cold bath in maximal handgrip strength. Percept Mot Skills 1977; 44: 323-6
94. Wittmers L, Savage MV. Cold water immersion. In: Wenger CB, Pozos RS, editors. Medical aspects of harsh environments. Vol. 1. Washington, DC: The Office of the Surgeon General at TMM Publications, Borden Institute, Walter Reed Army Medical Center, 2001
95. Lloyd EL. ABC of sports medicine: temperature and performance I -cold. BMJ 1994; 309: 531-4
96. Terrell T, Hough DO, Alexander R. Identifying exercise allergies: exercise-induced anaphylaxis and cholinergic urticaria. Phys Sportsmed 1996; 24 (11) [online]. Available from URL: www.physsportsmed.com/issues/1996/11_96/terrell.htm [Accessed 2005 Jul 17]
97. Paz JC, West MP. Acute care handbook for physical therapists. 2nd ed. Boston (MA): Butterworth Heinemann, 2002
98. Lippman SM, Winn L, Grumet FC, et al. Evans' syndrome as a presenting manifestation of atypical paroxysmal cold hemoglobinuria. Am J Med 1987; 82 (5): 1065-72
99. Myrer JW, Measom G, Durrant E, et al. Cold- and hot-pack contrast therapy: subcutaneous and intramuscular temperature change. J Athl Train 1997; 32 (3): 238-41
100. Whitney JD, Wickline MM. Treating chronic and acute wounds with warming: review of the science and practice implications. J Wound Ostomy Continence Nurs 2003; 30 (4): 199-209
101. Robertson VJ, Duck FA. A review of therapeutic ultrasound: biophysical effects. Phys Ther 2001; 81: 1351-8
102. Michlovitz SL, editor. Thermal agents in rehabilitation. 3rd ed. Philadelphia (PA): FA Davis Company, 1996
103. Wyper DJ, McNiven DR. Effects of some physiotherapeutic agents on skeletal muscle blood flow. Physiotherapy 1976; 62: 83-5
104. Cotts BE, Knight KL, Myrer JW, et al. Contrast-bath therapy and sensation over the anterior talofibular ligament. J Sport Rehabil 2004; 13: 114-21

105. Wilk KE, Macrina LC, Reinold MM, et al. Team physician's corner: common modalities in sports medicine. SportsMedicine Update 2004; July-August [online]. Available from URL: http://www.sportsmed.org [Accessed 2005 Jul 17]

106. Kaul MP, Herring SA. Superficial heat and cold: how to maximise the benefits. Phys Sportsmed 1994; 22 (12): 65-74

107. Tonnessen D. Strains and sprains: hot and cold therapy. In: Aldred HE, editor. Sports injuries sourcebook: basic consumer health information about common sports injuries. Detroit (MI): Omnigraphics, 1999

108. Sawyer PC, Unl TL, Mattacola CG, et al. Effects of moist heat on hamstring flexibility and muscle temperature. J Strength Cond Res 2003; 17 (2): 285-90

109. Taylor BF, Waring CA, Brashear TA. The effects of therapeutic application of heat or cold followed by static stretch on hamstring muscle length. J Orthop Sports Phys Ther 1995; 21 (5): 283-6

110. Henricson AS, Fredriksson K, Persson I, et al. The effect of heat and stretching on the range of hip motion. J Orthop Sports Phys Ther 1984; 6: 110-5

111. Prentice WE. An electromyographic analysis of the effectiveness of heat and cold and stretching for inducing relaxation in injured muscle. J Orthop Sports Phys Ther 1982; 3 (3): 133-40

112. Bigos SJ, Bowyer OR, Braen GR, et al. Acute low back problems in adults. Washington, DC: US Department of Health and Human Services, 1994

113. Nadler SF, Steiner DJ, Erasala GN, et al. Continuous low-level heat wrap therapy provides more efficacy than ibuprofen and acetaminophen for acute low back pain. Spine 2002; 27 (10): 1012-7

114. Nadler SF, Steiner DJ, Petty SR, et al. Overnight use of continuous low-level heatwrap therapy for relief of low back pain. Arch Phys Med Rehabil 2003; 84: 333-42

115. Nadler SF, Steiner DJ, Erasala GN, et al. Continuous low-level heatwrap therapy for treating acute non-specific low back pain. Arch Phys Med Rehabil 2003; 84: 329-34

116. Wallace L, Knortz K, Esterson P. Immediate care of ankle injuries. J Orthop Sports Phys Ther 1979; 1: 46-50

117. Barnes L. Cryotherapy: putting injury on ice. Phys Sportsmed 1979; 7 (6): 130-6

118. Magness J, Garrett TR, Erickson DI. Swelling of the upper extremity during whirlpool baths. Arch Phys Med Rehabil 1970; 51 (5): 297-9

119. Feibel A, Fast A. Deep heating of joints: a reconsideration. Arch Phys Med Rehabil 1976; 57 (11): 513-4

120. Turner B, Pennefather J, Edmonds C. Cardiovascular effects of hot water immersion (suicide soup). Med J Aust 1980; 2 (1): 39-40

121. Nagasawa Y, Komori S, Sato M, et al. Effects of hot bath immersion on autonomic activity and hemodynamics: comparison of the elderly patient and the healthy young. Jpn Circ J 2001; 65 (7): 587-92

122. Stanton DB, Bear-Lehman J, Graziano M, et al. Contrast baths: what do we know about their use? J Hand Ther 2003; 16 (4): 343-6

123. Connolly DAJ, Brennan KM, Lauzon CD. Effects of active versus passive recovery on power output during repeated bouts of short term, high intensity exercise. J Sports Sci Med 2003; 2 (2): 47-51

124. Thiriet P, Gozal D, Wouassi D, et al. The effects of various recovery modalities on subsequent performance, in consecutive supramaximal exercise. J Sports Med Phys Fitness 1993; 33 (2): 118-29

125. Signorile JF, Ingalls C, Tremblay LM. The effects of active and passive recovery on short-term, high intensity power output. Can J Appl Physiol 1993; 18 (1): 31-42

126. Kauppinen K. Sauna, shower, and ice water immersion: physiological responses to brief exposures to heat, cool and cold: part II -circulation. Arctic Med Res 1989; 48 (2): 64-74

Correspondence and offprints: *Ian M. Wilcock*, Institute of Sport and Recreation Research New Zealand, Division of Sport and Recreation, Faculty of Health and Environmental Sciences, Auckland University of Technology, Private Bag 92006, Auckland, New Zealand.
E-mail: ianwil@xtra.co.nz