Original Article

Thermoregulatory responses to exercise and warm water immersion in physically trained men with tetraplegia

EM Gass*[,1], GC Gass[1] and K Pitetti[2]

[1]School of Physiotherapy and Exercise Science, Griffith University, Australia, and [2]College of the Health Professions, Wichita State University, Kansas, USA

Objective: To compare thermoregulatory responses of highly trained men who are tetraplegic during 40 min exercise at 65% $\dot{V}O_2$ peak and 60 min immersion in 39°C water.

Methods: Four physically trained men who are tetraplegic participated in three laboratory visits. The first visit involved familiarisation and then determination of $\dot{V}O_2$ peak using open circuit spirometry during an incremental test to exhaustion with each man propelling his sport wheelchair on a motor driven treadmill. The order of second and third visits was randomly allocated. Visit 2 involved 40 min of exercise at 65% $\dot{V}O_2$ peak propelling each man's sport wheelchair on treadmill. Visit 3 involved sitting immersed to nipple line in 39°C water for 60 min. Venous blood was obtained pre, during and after each intervention and analyzed for haemoglobin, haematocrit and changes in plasma volume were calculated. Separated plasma was analyzed for noradrenalin and adrenalin (high performance liquid chromatography). Heart rate, rectal temperature, and sweat rate estimated from a sweat capsule placed on forehead (dew point hygrometry) were recorded throughout.

Results: $\dot{V}O_2$ peak and HR max of these subjects were 1.14 ± 0.16 l·min^{-1} and 99 ± 4 b·min^{-1} respectively. Heart rate preimmersion was 67 ± 4 b·min^{-1} rising to 75 ± 4 b·min^{-1} after 40 min and 87 ± 3 b·min^{-1} after 60 min immersion. Heart rate was 68 ± 3 b·min^{-1} pre-exercise rising to 91 ± 5 b·min^{-1} after 40 min exercise. Rectal temperature rose from 35.97 ± 0.30°C pre immersion to 37.32 ± 0.51°C after 60 min immersion, and from 36.42 ± 0.20°C pre-exercise to 36.67 ± 0.19°C after 40 min exercise. Haemoconcentration occurred during 40 min of exercise and haemodilution occurred throughout 60 min of water immersion. Three participants demonstrated no sweating on the forehead during immersion or exercise. One subject commenced sweating after 20 min exercise and after 5 min of immersion.

Conclusion: Compared to exercise, immersion was associated with a lower heart rate, a lower plasma noradrenalin concentration and an expanded plasma volume. When considering exercise or warm water immersion as therapeutic modalities in men who are tetraplegic, attention should be paid to heat gain and changes in plasma volume.
Spinal Cord (2002) **40**, 474–480. doi:10.1038/sj.sc.3101341

Keywords: tetraplegia; thermoregulation; exercise; immersion; acclimation

Introduction

Human body temperature is highly regulated.[1] Afferent signals from various body sites are integrated in the hypothalamus where appropriate effector responses regulate core temperature around a given set point.[2] The major effector responses that regulate heat storage include vasomotor changes, sweating, shivering and changes in blood flow, with the intensity of these effector responses being proportional to the core temperature.

The temperature of arterial blood reaching the hypothalamus is typically represented as the core temperature and reflects the balance between heat production and heat loss. To estimate the core temperature, temperature at sites other than the hypothalamus are recorded, including the rectum, oseophagus and tympanic membrane. Evidence suggests that temperature measured in the oseophagus at

*Correspondence: EM Gass, School of Physiotherapy and Exercise Science, Griffith University, Gold Coast Campus, PO Box 50 Gold Coast Mail Centre, Queensland 9726, Australia.

rest and during exercise is a valid estimate of arterial blood temperature and therefore temperature of the blood reaching the hypothalamus.[3–7] Rectal temperature, while having slower response times, does also track arterial blood temperature.[8–10]

Passive heating is typically described as heat exposure without exercise[11] and can include warm water immersion (hot baths).[12] Immersion in water at $39°C$ can seriously challenge efferent heat loss responses because of the high thermal conductivity of water and a limited skin area available for evaporative or convective heat loss. Prolonged immersion (60 min), in $39°C$ water can result in a rapid increase in heat storage or core temperature (Tcore). Indeed, warm water immersion or hydrotherapy is frequently used as a therapeutic modality for patients with a spinal cord lesion, often with little regard to the thermal conductivity of water and heat balance implications.

During dynamic exercise in a thermoneutral environment, an additional thermal load proportional to the intensity of the exercise is imposed which may result in a higher core temperature and more intense effector responses than passive heating alone.

Traumatic tetraplegia results in motor, sensory and autonomic deficits that are dependent upon the site and extent of spinal cord damage. Complete spinal cord lesions above the fifth cervical segment, will result in the loss of motor and sensory innervation to some regions of the arms and all regions of the legs, hips and trunk. The loss of the sympathetic division of the autonomic nervous system will also affect cardio-acceleration and redistribution of blood flow.[13,14]

When people with tetraplegia exercise at high relative intensities in thermo-neutral environments, the absolute heat production from exercise is small because of the reduced amount of innervated and active muscle mass.[15] Despite limited sweating and evaporative capacity, people who are tetraplegic competing in a marathon event under neutral environmental conditions can expect convection to be the main avenue for heat loss. Accordingly, with a reduced innervated muscle mass and with the wheelchair moving at speeds of approximately 5.0 m·s^{-1}, the rise in core temperature over 2 h would be expected to be $0.50–1.00°C$.[15]

This may not be the case when people with tetraplegia use hydrotherapy and are immersed to the nipple line in warm water. The high thermal conductivity of water favours a high rate of heat gain, dependent partially on water temperature and, at $39°C$ can be characterised by a $1.0–3.0°C$ rise in core temperature within 60 min.

Given the widespread use of exercise and hydrotherapy after spinal cord injury, the aim of the present experiment was to compare the thermoregulatory responses of four highly trained men with tetraplegia during active heating via 40 min of exercise at 65% $\dot{V}O_2$ peak and during passive heating via 60 min of immersion in $39°C$ water.

Method

Four physically trained men with tetraplegia (competitors in international wheelchair marathons) volunteered to participate in the study which had been approved by an institutional ethics committee. After giving consent, each participant visited the laboratory on three occasions.

The first visit was to provide familiarisation followed by an incremental wheelchair exercise test to exhaustion on a motorised treadmill to determine peak oxygen consumption ($\dot{V}O_2$ peak). A second visit either involved 40 min of exercise at 65% $\dot{V}O_2$ peak or 60 min of warm ($39°C$) water immersion. A third visit was the alternate condition. Visitations were separated by two to three days, and were randomly allocated with each subject completing each condition.

At the first visit, each participant was weighed wearing a swimming costume and an emptied catheter bag. Skinfold thickness was measured at four sites (triceps, biceps, subscapular and suprailiac) on each subject on the right side. Electrocardiograph electrodes were placed in the CM5 placement to monitor and record heart rate and rhythm throughout the incremental wheelchair test.

After a two min seated rest period, each participant performed an incremental exercise test by pushing their track wheelchair on a motor driven treadmill until exhaustion. Participants began at a speed of 0.5 km·h^{-1} with increments of 0.5 km·h^{-1} and 0.5% grade every minute. Throughout the incremental wheelchair exercise test, participants breathed through a Hans Rudolph valve (no 2700) and expired gas passed through a calibrated pneumotach (Hewlett-Packard model HP 47304A) into a baffled mixing chamber. Oxygen and carbon dioxide analysers (Rapox and Capnograph) continuously sampled mixed expired gas from the distal end of the mixing chamber. Every 30 s calculations were made of O_2 uptake ($\dot{V}O_2$) CO_2 production ($\dot{V}CO_2$) and minute ventilation ($\dot{V}_E$). Each analyser was calibrated before and after each exercise test using a mixture of precision reference gases.

Heart rate (HR) was monitored throughout and recorded every minute. All experiments were conducted in a controlled environment ($23°C$ Dry Bulb, $18°C$ Wet Bulb).

Exercise at 65% $\dot{V}O_2$ peak

A thin, flexible, calibrated rectal probe (AD590) was inserted approximately 14 cm beyond the anal sphincter to estimate core temperature (Trec). Skin temperature (Tsk) was recorded from the forehead and the calf using AD590 technology.

A sweat capsule, using the principle of dewpoint hygrometry,[16] was secured to the forehead of each participant and sealed with a thin layer of paraffin jelly at the skin-capsule interface. Changes in dewpoint temperature were used to detect the onset of sweating, and calculate sweat rate.

A surflo catheter was inserted into either a vein on the dorsum of the hand, or the lower third of the extensor aspect of the forearm with patency maintained by flushing with 15 IU.ml^{-1} of sterile heparinised saline. Venous blood was withdrawn at the beginning of the pre-exercise period (-20 min), immediately before exercise (0 min) and 30–90 s after the 65% $\dot{V}O_2$ peak test finished, and analysed for haemoglobin (Hb) and haematocrit (Hct). Relative changes in plasma volume were calculated according to the formula of Greenleaf et al (1979)[17]. Separated plasma was analyzed for noradrenaline (NAD) and adrenalin (AD) using high performance liquid chromatography (HPLC).

After a 20 min seated pre-exercise rest period, each participant pushed their wheelchair at approximately 65% $\dot{V}O_2$ peak on the motor driven treadmill for up to 40 min. $\dot{V}O_2$, $\dot{V}CO_2$ and HR were measured at 15–20 and 30–35 min of the exercise test as described for Visit 1.

Warm water immersion (39°C)
Rectal and skin temperature probes, sweat capsule, surflo catheter and ECG electrodes were placed as previously described. Venous blood was withdrawn 20 min before immersion (-20 min), just prior to immersion (0 min) and just before coming out of the pool (60 min). Venous blood was analyzed as previously described.

Each participant sat in a wheelchair in a thermoneutral environment (23°C DB; 18°C WB) while pre-immersion values ($-20,0$ min) were obtained. Once the 0 min seated pre-immersion venous blood sample was obtained, the participant was wheeled to the next room and transferred in the sitting position to a lightweight plastic chair and lowered into the 39°C water to the nipple line. Trec, Tsk and forehead dew

point temperature were monitored continuously and recorded each 30 s. HR was monitored continuously and recorded every 2 min. The participant remained in the pool for up to 60 min, or until Trec reached 38.5°C.

Results

Participants' descriptive statistics, including sporting achievements, are presented in Table 1. All participants became tetraplegic as a result of trauma, and had established themselves as elite wheelchair athletes.

The results from the incremental treadmill wheelchair test to exhaustion are presented in Table 2. The $\dot{V}O_2$ peak (l·min^{-1}, ml·min^{-1}·kg^{-1}) and peak heart rate (HR) (b·min^{-1}) were 1.14 ± 0.16 l·min^{-1}, 16.7 ± 1.8 ml·kg^{-1}·min^{-1} and 99 ± 4 b·min^{-1} respectively.

Heart rate and rectal temperature (Trec) recorded for each participant during the exercise test at 65% $\dot{V}O_2$ peak and during water immersion is shown in Figure 1.

Pre-immersion heart rate (0 min) was 67 ± 4 b·min^{-1} rising to 75 ± 4 b·min^{-1} and 87 ± 3 b·min^{-1} after 40 and 60 min, respectively. The pre-exercise rate heart rate was 68 ± 3 b·min^{-1} rising to 91 ± 5 b·min^{-1} after 40 min of exercise at 60–65% $\dot{V}O_2$ peak. Rectal temperature (Trec) rose from 35.97 ± 0.30°C at rest to 36.46 ± 0.40°C after 40 min and to 37.32 ± 0.51°C after 60 min of immersion. The Trec increased from 36.42 ± 0.20°C at rest to 36.67 ± 0.19°C after 40 min of exercise.

Calf and head skin temperature obtained during exercise and water immersion are shown in Figure 2.
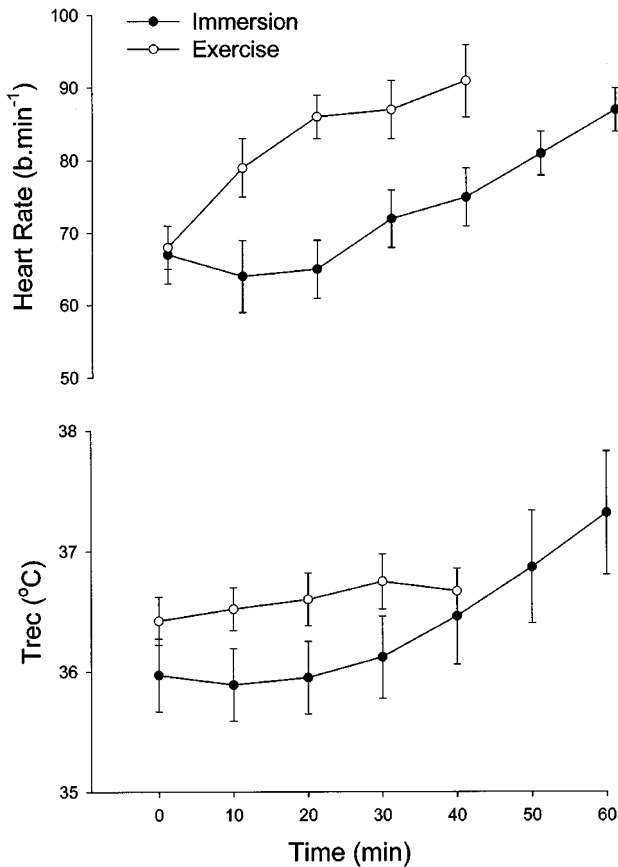
Participants 1, 2 and 4 demonstrated no centrally driven forehead sweating during exercise or immersion. Participant 3 (C 5/6) commenced sweating after 20 min of exercise (0.1 mg·min^{-1}·cm^2) and reached

**Table 1** Personal and anthropometric characteristics of participants

Subject	Age (years)	Lesion level	Time in chair (years)	Sporting achievements at time of experiment	Body mass (kg)	Sum of skinfolds (mm)
1	37	C6/7	14	World record holder 100 m Silver Medal 4×100 m Paralympics	77.5	42
2	33	C6	11	Gold Medal 100 m Stoke Mandeville Games Personal best 100 m (26.0 s) Paralympics Personal best 200 m (56.1 s) Paralympics	51.1	19
3	44	C5/6	19	Gold Medal Marathon + World Record (2.29.58) Paralympics Gold Medal 400, 800, 1500 and 5000 m	74.7	28
4	38	C6/7	18	Personal best marathon time 2.02.00 Silver medals, marathon, 200 and 5000 m Paralympics	71.3	28 (3 sites)

Table 2 Peak physiological data from incremental wheelchair test to exhaustion

Subject No	$\dot{V}O_2$ (l·min^{-1})	$\dot{V}O_2$ (ml·min^{-1}·kg^{-1})	$\dot{V}O_2$ (l·min^{-1})	$\dot{V}E$ (l·min^{-1})	HR (b·min^{-1})
1	1.0	12.9	0.94	35.2	94
2	0.78	15.4	0.75	24.3	97
3	1.27	17.1	1.27	56.5	100
4	1.52	21.3	1.58	64.8	104
$\overline{X}$	1.14	16.7	1.14	45.2	99
SE	0.16	1.8	0.18	9.3	4

Figure 1 Heart rate and rectal temperature (Trec) during exercise and immersion
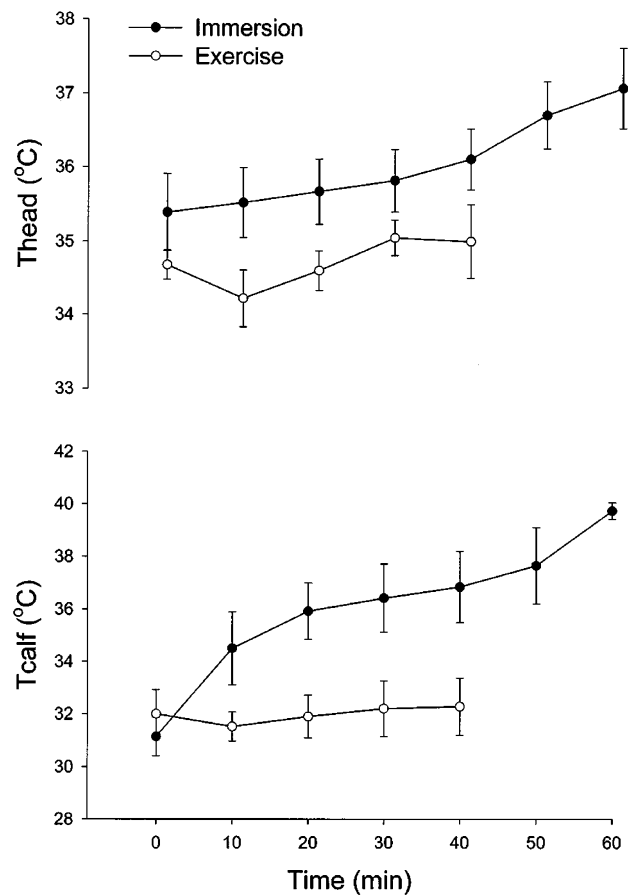
Figure 2 Calf and head skin temperatures (Tcalf, Tskin) during exercise and immersion

0.4 mg·min^{-1}·cm^{-2} after 35 and 40 min of exercise. This same participant (no.3) commenced sweating after 5 min of immersion (0.2 mg·min^{-1}·cm^{-2}) and maintained this sweat rate until the 50 min after which sweat rate increased to 0.3 mg·min^{-1}·cm^{-2} until the end of water immersion (60 min). For participant 3, sweating onset during exercise and pool immersion occurred at Trec 36.8°C and 36.12°C, respectively.

Plasma adrenalin (AD) and noradrenalin (NAD) concentrations measured during water immersion and exercise are presented in Table 3.

Haemoglobin and haematocrit values are presented in Table 3. Haemoconcentration occurred during

40 min of exercise (Δ% PV:-8.6 ± 2.3) and haemodilution occurred throughout 60 min of water immersion (Δ% PV: + 2.2 ± 1.8). Changes in plasma volume volume during immersion and exercise are shown in Figure 3.

Discussion

The participants in the present study were classified as having clinically complete tetraplegia and were successful elite athletes. The absolute (l·min^{-1}) and relative (ml·kg^{-1}·min^{-1}) $\dot{V}O_2$ peak for the present participants, is consistent with values reported for other

Table 3 Plasma noradrenalin (NAD) and adrenalin (AD) concentration, haemoglobin (Hb), and haematocrit (Hct), during exercise (Ex) and water immersion (WI) (mean ± SE)

		Time −20	0	End Immersion/ End Exercise
NAD	WI	2.48 (0.29)	2.35 (0.40)	2.67 (0.63)
(nmol·l^{-1})	Ex	2.58 (0.32)	2.15 (0.51)	3.03 (0.57)
AD	WI	0.40 (0.21)	1.53 (0.13)	1.00 (0.43)
(nmol·l^{-1})	Ex	0.82 (0.33)	1.6 (0.40)	0.87 (0.38)
Hb	WI	13.9 (0.4)	13.6 (0.5)	13.5 (0.6)
(g·dl^{-1})	Ex	13.6 (0.1	13.4 (0.2)	13.8 (0.2)
Hct	WI	39.1 (1.3)	38.4 (1.4)	37.7 (1.6)
(%)	Ex	38.7 (1.0)	38.4 (0.9)	40.6 (1.6)
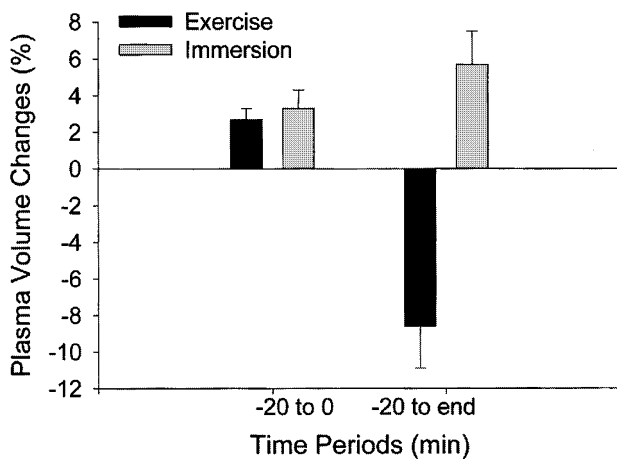
Figure 3 Changes in plasma volume during exercise and immersion

athletes with tetraplegia[18] and reflects not only their highly trained state but also the reduced amount of innervated muscle mass. Despite the low absolute value, $\dot{V}O_2$ peak (l·min^{-1}) was approximately 50% higher than similarly aged but sedentary persons with tetraplegia.[19] The maximum heart rate and minute ventilation rate recorded at exhaustion during the incremental exercise test are consistent with the physiological consequences of cervical spinal cord injury and with values reported for other tetraplegic athletes.[18] The maximum minute ventilation reflects the reduced level of innervation to the intercostal and other accessory respiratory muscles resulting from injury to the cervical spinal cord.

Trec recorded prior to exercise at 65% $\dot{V}O_2$ peak (36.42 ± 0.2°C) and prior to immersion (35.97 ± 0.3°C) is consistent with the values reported for persons with tetraplegia.[15]

The low resting Trec, typically found in persons with tetraplegia does, provide an added margin of safety when exposed to hot, but not cold environments. The present study indicates that a rise in Trec of 2.5–3.0°C would need to occur before raising the

spectre of heat stress/heat stroke. In those situations where resting core temperature may be elevated due to bladder infection, travel immunisation or prior hydrotherapy a shorter immersion time will be required before reaching 38.5–39.0°C. The continued use of hydrotherapy with people who are tetraplegic should include consideration of their initial core temperature, time of immersion and water temperature.

By comparison, the rise in Trec after 40 min of exercise at 60–65% $\dot{V}O_2$ max was 0.35 ± 0.12°C and reflects a low heat storage resulting primarily from a reduced absolute metabolic heat production (eg 15 kJ·min^{-1}) and the raised convective heat loss by propelling a wheelchair at approximately 5 m·s^{-1}.

The Tcalf and Thead recorded at rest and prior to immersion and exercise were consistent with values reported by others.[15] The direction and magnitude of change in Tcalf and Thead primarily reflects the nature of the two stressors used to raise Tcore. The temperature recorded at the calf during immersion would be primarily a reflection of the water temperature rather than a centrally driven increase in skin blood flow.

During 40 min of exercise Tcalf remained essentially unchanged and is consistent with the results of other trained men with tetraplegia.[15] It has been suggested[6,20] that an intact pathway from above C5 to at least T11 is required to mediate active vasodilation in the lower limb. Accordingly, participants in the present study, because of their cervical cord lesions, could not be expected to actively redistribute blood-flow to the skin and raise Tcalf. Skin blood flow is an important contributor to skin temperature[21] and on the basis of Tcalf recorded during exercise it is reasonable to conclude that in the present participants there was no centrally mediated vasodilation in the calf.

The consistency of Thead recorded during exercise reflects the absence of sweating and evaporation and the convective effects of pushing a wheelchair on a motor driven treadmill at approximately 10 km·h^{-1}.

The amplitude and direction of change in heart rate during exercise is consistent with the results of others.[15] No previous studies report heart rate changes

during immersion in warm water in people with tetraplegia. Compared to exercise, the lower heart rate recorded after the end of immersion was accompanied by a lower plasma noradrenalin concentration and an expanded plasma volume. Using impedance methodology to estimate cardiac output, immersion has been associated with increases in cardiac output of 25% and 35% in participants with tetraplegia after 60 and 120 min.[22] Whether the decrease in HR observed during immersion in the present study was accompanied by an increase in stroke volume is not known, as cardiac output was not measured.

The pre-exercise expansion in plasma volume found in the present participants has also been reported in other adults with paraplegia.[23] It has been suggested that the pre-exercise plasma volume expansion in persons with paraplegia may be a reflection of an exercise anticipatory sympathetic response affecting vascular constriction and favouring inward filtration and plasma volume expansion.[23] Given that all participants in the present study were assessed as having clinically complete cervical cord lesions, it is difficult to attribute the pre-exercise plasma volume expansion to a centrally derived sympathetic vascular constriction. The expansion of plasma volume during immersion is consistent with the results of others using subjects who are able bodied[24] and has been attributed to the depth related pressure generated by immersion in water.

The reduction in plasma volume ($-8.6 \pm 2.3\%$) that occurred after 40 min of exercise at 65% $\dot{V}O_2$ max was larger than the plasma volume reduction reported for adults with paraplegia (T4 – T12) after 40 min of exercise at 50% $\dot{V}O_2$ peak (-2.5%) but similar to the reduction (-9%) in men with paraplegia (T5 – T12) after 40 min of exercise at 60 – 65% $\dot{V}O_2$ peak.[23,25]

The reduction in plasma volume during exercise has been ascribed to various factors such as decreased lymph flow, methodological problems related to changes in posture, raised capillary hydrostatic pressure and sweating.[26-28] It was expected that posture, capillary hydrostatic pressure and sweating would have minimal effect on the plasma volume reduction observed during 40 min of exercise in the present study. All participants remained seated throughout the exercise and immersion, the increase in capillary hydrostatic pressure was expected to be small, and only one participant exhibited modest sweating (0.4 mg·min^{-1}·cm^{-2}) after 40 min of exercise.

The seated posture, low absolute $\dot{V}O_2$/cardiac output and the absence of consistent central control of sweating provides little guidance as to the mechanism for the plasma volume reduction in tetraplegics during 40 min of exercise at 65% $\dot{V}O_2$ max.

In summary, the present study found a higher rate of heat gain (ΔTrec) and an increased plasma volume expansion after 40 min of water immersion (39°C)

than after 40 min of exercise at 65% $\dot{V}O_2$ max in highly trained men with tetraplegia.

References

1 Sawka NM, Wenger BC, Pandolf KB. Thermoregulatory responses to acute exercise-heat stress and heat acclimation. In: Fregly MJ, Blatteis CM (eds). *Handbook of Physiology: Section 4 Environmental Physiology*. **Vol. 1.** New York: Oxford University Press, 1996, pp 157 – 185.

2 Sawka MN, Wenger BC. Physiological responses to acute exercise – heat stress. In: Pandolf KB, Sawka MN, Gonzales RR (eds). *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, IN: Benchmark, 1988, pp 97 – 151.

3 Cranston WI, Gerbrandy J, Snell ES. Oral, rectal and oesophageal temperatures and some factors affecting them in man. *J Physiol* 1954; **126:** 347 – 358.

4 Aikas E, Karvonen MJ, Puronen P, Ruosteenoja R. Intra-muscular, rectal and oesophageal temperature during exercise. *Acta Physiol Scand* 1972; **54:** 366 – 370.

5 Carlsten A, Grimby G. Rapid changes in human right heart blood temperature at variations in venous return. *Scand J Clin Lab Invest* 1958; **10:** 387 – 401.

6 Cooper KE, Kenyon JR. A comparison of temperatures measured in the rectum, oesophagus and on the surface of the aorta during hypothermia in man. *Br J Surg* 1957; **44:** 616 – 619.

7 Shiraki K, Konda N, Sagawa S. Esophageal and tympanic temperature responses to core blood temperature changes during hyperthermia. *J Appl Physiol* 1986; **61:** 98 – 102.

8 Benzinger TH. Heat regulation: homeostasis of central temperature in man. *Physiol Rev* 1969; **49:** 671 – 659.

9 Mairiaux P, Sagot JC, Candas V. Oral temperature as an index of core temperature during heat transients. *Eur J Appl Physiol* 1983; **50:** 331 – 341.

10 Nielsen B, Nielsen M. Body temperature during work at different environmental temperatures. *Acta Physiol Scand* 1962; **56:** 120 – 129.

11 Armstrong LE, Maresh CM. The induction and decay of heat acclimatisation in trained athletes. *Sports Medicine* 1991; **12:** 302 – 312.

12 Belding HS *et al.* Use of a water bath for study of human heat tolerance. Proceedings of Human Adaptability and its Methodology, 1965, IUPS, Japan.

13 Davis GM, Shephard RJ. Cardiorespiratory fitness in highly active versus inactive paraplegics. *Med Sci Sports Exerc* 1988; **20:** 463 – 468.

14 Coutts KD, Rhodes EC, McKenzie DC. Maximal exercise responses of tetraplegics and paraplegics. *J Appl Physiol* 1983; **55:** 479 – 482.

15 Gass EM, Gass GC, Gwinn TH. Sweat rate and rectal and skin temperatures in tetraplegic men during exercise. *Sports Med Training and Rehab* 1992; **3:** 243 – 249.

16 Graichen H, Rascati R, Gonzales RR. Automatic dew-point temperature sensor. *J Appl Physiol* 1982; **52:** 1658 – 1666.

17 Greenleaf JE, Convertino VA, Mangseth GR. Plasma volume during stress in man: osmolality and red cell volume. *J Appl Physiol Resp Environ Exerc Physiol* 1979; **47:** 1031 – 1038.

18 Hopman M, Dallmeijer G, van der Woude L. The effect of training on cardiovascular responses to arm exercise in individuals with tetraplegia. *Eur J Appl Physiol* 1996; **74:** 172 – 179.

19 Gass GC *et al*. The effects of physical training on high level spinal lesion patients. *Scand J Rehab Med* 1980; **12:** 61 – 65.

20 Gass GC *et al*. Rectal and rectal vs oesophageal temperatures in paraplegic men during prolonged exercise. *J Appl Physiol* 1988; **64:** 2265 – 2271.

21 Normell LA. Distribution of impaired cutaneous caso-motor and sudomotor function in paraplegic man. *Scand J Lab Invest* 1974; **138** (Suppl. 30): 25 – 41.

22 Tajima F *et al*. Cardiovascular, renal and endocrine responses in male quadriplegics during head-out water immersion. *Am J Physiol* 1990; **258:** R1424 – R1430.

23 Gass GC *et al*. The effects of prolonged exercise in spinally injured subjects. *Med Sci Sports Exerc* 1981; **13:** 277 – 283.

24 Greenleaf JE. Physiological responses to prolonged bed rest and fluid immersion in humans. *J Appl Physiol* 1984; **57:** 619 – 633.

25 Gass GC, Camp EM. Effects of prolonged exercise in highly trained traumatic paraplegic men. *J Appl Physiol* 1987; **63:** 1846 – 1852.

26 Sjoggard G, Saltin B. Extra- and intra-cellular water spaces in muscles of man at rest with dynamic exercise. *Am J Physiol* 1982; **243:** R271 – R280.

27 Nadel E. Temperature regulation and prolonged exercise. In: Lamb D, Murray R (eds). *Perspectives in Exercise Science and Sports Medicine*. Vol 1. *Prolonged Exercise*. Benchmark Press, 1988; pp 135 – 136.

28 Mack GW, Nadel ER. Body fluid balance during heat stress in humans. In: Fregley MJ, Blatteis CM (eds). *Handbook of Physiology*. Section 4. *Environmental Physiology*. Oxford University Press, 1996, pp 192 – 195.