Effects of Spinal Cord Lesion Level upon Thermoregulation during Exercise in the Heat

MICHAEL J. PRICE[1] and IAN G. CAMPBELL[2]

[1]*School of Science and the Environment, Coventry University, Coventry, UNITED KINGDOM;* [2]*School of Sport, Health and Exercise, Staffordshire University, Stoke-on-Trent, UNITED KINGDOM*

ABSTRACT

PRICE, M. J., and I. G. CAMPBELL. Effects of Spinal Cord Lesion Level upon Thermoregulation during Exercise in the Heat. *Med. Sci. Sports Exerc.*, Vol. 35, No. 7, pp. 1100–1107, 2003. **Purpose:** This study examined the effects of the level of spinal cord injury upon the thermoregulatory responses of wheelchair athletes during prolonged wheelchair exercise in warm conditions. **Methods:** Eight tetraplegic (TP), 10 high-level paraplegic (HP), and 10 low-level paraplegic (LP) athletes exercised for 60 min at 60% $\dot{V}O_{2peak}$ in a warm environment (31.5 ± 1.7°C, 42.9 ± 8.0% relative humidity). Skin temperature and aural temperature were monitored. **Results:** Aural temperature increased gradually during the exercise period by 1.1 ± 0.3 and 1.4 ± 0.5°C for the HP and LP groups, with a more marked increase observed for the TP group (2.1 ± 0.5°C; $P < 0.05$). Upper-arm skin temperature was higher for the TP when compared with the HP and LP between 30 and 60 min ($P < 0.05$). Back skin temperature was higher for the TP when compared with the HP and LP between 45 and 60 min ($P < 0.05$). No differences were noted between groups for the thigh or calf skin temperatures. During recovery, skin temperature remained elevated for the TP group when compared with the HP and LP groups ($P < 0.05$). Heat storage was greatest for the TP athletes at the end of exercise and remained elevated throughout recovery ($P < 0.05$). **Conclusions:** All athletes completed the exercise task even though the gradual increase in aural temperature observed for the HP and LP groups suggests a degree of thermal imbalance. However, this was much less than observed for TP athletes, who demonstrated a much greater imbalance in temperature regulation. Increasing the exercise or environmental strain may result in the thermoregulatory responses of athletes with a spinal cord injury being compromised. **Key Words:** WHEELCHAIR EXERCISE, RECOVERY, BODY TEMPERATURE, AURAL TEMPERATURE, SKIN TEMPERATURE, HEAT STORAGE

The thermoregulatory responses of spinal cord-injured and able-bodied subjects to heat exposure at rest have been previously reported (9,14,21,27,28). These studies have shown that thermoregulatory responses are proportional to the level of lesion, reflecting the amount of sympathetic nervous system available for sweating and blood redistribution. In general, although paraplegics have been shown to regulate body temperature effectively at rest, they show greater increases in core temperature when compared with able-bodied subjects (9). Furthermore, paraplegics with lesions at T6 and below demonstrate smaller increases in core temperature than those with lesions above T6, who in turn demonstrate smaller increases than subjects with a cervical lesion (tetraplegics) (9). The latter subject group exhibits the least effective thermoregulation due to complete absence of sweating capacity (9,26,21,28). Despite this finding, no studies have reported the thermoregulatory responses of the tetraplegics to prolonged exercise in a warm environment.

Previous work has shown that during prolonged upper body exercise in cool conditions, trained paraplegics with low-level spinal cord lesions demonstrate similar increases in core temperature to those reported for able-bodied athletes (22). This similarity was suggested to be due to the reduced sweating capacity below the level of lesion being matched by the reduced muscle mass for heat production enabling thermal balance to be achieved. Although the above study examined responses of low-level paraplegics, little data exist relating to the responses of both high-level paraplegics and tetraplegics during exercise. Hopman et al. (12) observed greater rectal temperature responses for high-level paraplegics (T2–T6) when compared with low-level paraplegics (T7–T12) during exercise and thermal stress (40% peak power, 35°C). However, although the athletes in this study were working at similar relative power outputs,

Address for correspondence: Michael J. Price, School of Science and the Environment, Coventry University, Priory Street, Coventry, CV1 5FB, United Kingdom; E-mail: m.price@coventry.ac.uk.
Submitted for publication September 2002.
Accepted for publication February 2003.

0195-9131/03/3507-1100
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/01.MSS.0000074655.76321.D7

TABLE 1. Physical characteristics and peak test data for the TP, LP, and LP athletes.

	TP	Group HP	LP
$\dot{V}O_{2peak}$ (L·min^{-1})	1.26 ± 0.34	1.70 ± 0.33	2.15 ± 0.66*
$\dot{V}E_{peak}$ (L·min^{-1})	45.9 ± 9.6	64.9 ± 17.6	77.7 ± 26.3
HR peak (beats·min^{-1})	130 ± 20	175 ± 11*	188 ± 8*
BLa peak (mmol·L^{-1})	3.70 ± 0.83	5.66 ± 1.37*	5.66 ± 1.76*
Time since lesion (yr)	13.3 ± 10.5	16.7 ± 8.5	18.5 ± 6.6
Training sessions per week	4.1 ± 1.1	4.4 ± 1.4	4.4 ± 1.7
Hours per session	2.2 ± 0.8	2.0 ± 0.6	1.6 ± 0.4

* Significantly different from TP athletes ($P < 0.05$).

this produced different relative oxygen consumption values between groups. Consequently, the high-level paraplegics exercised at greater relative exercise intensities (63% $\dot{V}O_{2peak}$) when compared with the low-level paraplegics (43% $\dot{V}O_{2peak}$), which may have contributed to the greater thermal strain for this group. In addition, the use of rectal temperature has been suggested to be inappropriate for use in paraplegics (7). Therefore, the aim of this study was to examine the influence of the level of spinal cord injury on thermoregulatory responses at rest, and during prolonged wheelchair exercise and recovery in warm conditions.

METHODS

Twenty-eight wheelchair athletes volunteered to participate in this study, which had received University Ethics Committee approval. Subjects comprised of wheelchair rugby, basketball, and track athletes who trained and competed regularly at national and international levels. More specifically, the group comprised eight tetraplegic athletes (TP; C5/C6–C7/C8), 10 paraplegic athletes with high-level spinal cord injuries (HP; T1–T6), and 10 paraplegics athletes with low-level spinal cord injuries (LP; T7 and below). The separation of paraplegic athletes into groups with high- and low-level spinal cord injury is consistent with previous studies of spinal cord-injured subjects during exercise (12). All subjects had complete lesions of the spinal cord. Subject training and lesion characteristics are shown in Table 1. Subjects were fully familiarized with testing procedures and provided written informed consent to participate.

Subjects performed all tests in their sports specific wheelchair on a wheelchair ergometer (Bromking Turbo Trainer, Loughborough, UK). The ergometer consisted of a wheelchair roller, adjustable spring-loaded bracket for holding the wheelchair in position, and an optical sensor for determination of roller velocity. The system was calibrated via a personal computer and conventional software (Kingcycle) for each individual in order to produce a rolling resistance similar to that experienced when pushing on the road. Velocity could be maintained by observation of power output from the computer screen or from a speedometer attached to the athletes' chair. To determine the propulsion speed that would elicit an exercise intensity of 60% peak oxygen uptake ($\dot{V}O_{2peak}$), subjects performed four submaximal stages of wheelchair ergometry (Bromking Turbo Trainer) and an incremental test for $\dot{V}O_{2peak}$. Each submaximal exercise stage was 4 min in duration and separated by a 2-min rest period in order to minimize local fatigue (16). The submaximal stages for athletes exercising in rugby and basketball wheelchairs were performed at propulsion speeds of 4, 5, 6, and 7 mph (8, 10, 12, and 14 W). The submaximal stages for the athletes in track wheelchairs were performed at 8, 10, 12, and 14 mph (20, 30, 40, and 50 W) (16). A 1-min expired air sample was collected between min 2:45 and 3:45 of each 4-min exercise stage via the Douglas bag technique. Samples were subsequently analyzed for oxygen and carbon dioxide content (Servomex, Crowborough, UK). A small 20-μL capillary blood sample was obtained from the earlobe for the determination of blood lactate concentration (BLa) at rest and at the end of each exercise stage (YSI, Yellow Springs Instruments, Yellow Springs, OH).

After the fourth submaximal exercise stage had been completed, subjects rested for at least 5 min to allow heart rate to return to below 100 beats·min^{-1}. Subjects then performed a continuous incremental test for the determination of $\dot{V}O_{2peak}$. This test involved increases in speed of 1.0 mph each minute (17) from an initial speed of 4.0 mph for the athletes exercising in rugby and basketball wheelchairs and 10.0 mph for the athletes exercising in track wheelchairs. The test was open ended and continued until the athlete reached volitional exhaustion. A 1-min expired air sample was collected during the final minute of the test. A small 20-μL capillary blood sample was obtained from the earlobe for the analysis of BLa once volitional exhaustion had been reached. Heart rate was continually monitored (Polar Sports Tester, Kempele, Finland).

After at least 3 h chaperoned rest from the preliminary tests, subjects began the 60-min test protocol by returning to the laboratory and resting quietly for a further 15 min (pilot work demonstrated that this was suitable recovery from preliminary tests and produced similar thermoregulatory and other physiological responses to performing the tests on separate days). At the end of this period resting heart rate was recorded along with resting thermoregulatory data. A 5-mL venous blood sample was obtained from the antecubital vein from which hemoglobin (Clandon HemoCue, HemoCue Ltd., Sheffield, UK) and hematocrit (Hawksley Reader, Hawksley & Sons, Sussex, UK) were subsequently analyzed to determine plasma volume (4). Body mass was then recorded (Seca 710, seated scales, Hamburg, Germany) after individuals had evacuated their bladder. Skin-fold measurements were taken from the biceps, triceps, subscapular, and suprailiac sites using Harpenden skin-fold callipers (British Indicators Ltd, Luton, UK), in accordance with the procedures of Durnin and Wormersley (5). Subjects then entered the environmental chamber (31.5 ± 1.7°C, 42.9 ± 8.0% relative humidity) where a resting expired air sample was obtained (time points −10 to −5 min) via the same procedures described for the preliminary tests. A small 20-μL capillary blood sample was obtained from the earlobe and analyzed for blood lactate concentration. All subjects wore lightweight tracksuit trousers and training shoes with no clothing covering the upper body.

Thermistors were positioned for measures of aural and skin temperatures. Aural temperature was measured by an aural thermistor inserted into the subjects' auditory canal (3)

and securely plugged and taped in position. Skin thermistors were placed at standard positions (7,25) on the forehead, forearm, upper arm, back, chest, abdomen, thigh, and calf in order to establish the whole-body thermoregulatory response. The insulation and use of the aural thermistor and skin thermistor preparation has been described in previous studies (22,23). Subjects then undertook a standardized 5-min warm-up (−5 to 0 min). This involved 3-min exercise on the wheelchair ergometer at a speed equivalent to the first stage of the preliminary submaximal test, followed by 2 min of light stretching. Once completed, subjects were allowed to undergo their usual stretching routine. Subjects then exercised at an intensity of 60% $\dot{V}O_{2peak}$ for 60 min and were allowed to drink plain water (~15°C) *ad libitum*. On completion of the exercise test, subjects remained in the seated position and a second 5-mL venous blood sample was obtained. Postexercise hemoglobin concentration and hematocrit were subsequently analyzed. Subjects then rested quietly for 30 min and were reweighed. As well as the absolute change in body mass, the change in body mass in relation to fluid consumed was also calculated (adjusted change in body mass: ($Mass_{pre}$ − $Mass_{post}$) − fluid consumed)).

Aural and skin temperatures were recorded at rest, post warm-up, and every 5 min during the exercise period and during the first 30 min of recovery. Values were recorded by a Grant Squirrel meter logger (1250 Series, Grant Instruments, Cambridge, UK) via Edale thermistors (Edale Instruments, Cambridge, UK). Heat storage was calculated from the following formula employed by Havenith et al. (10), where:

$$\text{Heat Storage} = (0.8\ \Delta\text{Tcore} + 0.2\ \Delta\text{Tskin}) \cdot C_b,$$

and C_b is the specific heat capacity of the body tissue (3.49 $J \cdot g^{-1} \cdot {}^{\circ}C^{-1}$). Values were calculated from changes in aural and skin temperature (24) from resting values at 5, 15, 30, 45, and 60 min of exercise and 15 and 30 min of recovery. Small 20-μL capillary blood samples were obtained from the earlobe at 5, 15, 30, 45, and 60 min during the exercise period and 5 min postexercise. One-minute expired air samples were collected via the Douglas bag technique at 5, 15, 30, 45, and 60 min during the exercise period. Ratings of perceived exertion (RPE; Borg scale) were obtained after each expired air collection. Heart rate was continually monitored.

The peak physiological responses to incremental wheelchair exercise were compared using one- way ANOVA. Physiological data (0, 5, 15, 30, 45, and 60 min) and thermoregulatory data (rest, post warm-up, 5, 10, 15, 30, 45, 60 min of exercise and 5, 10, 15, and 30 min for recovery) were compared using two-way ANOVA with repeated measures (group × time). Significance was accepted at the $P < 0.05$ level. Where significance was obtained, Scheffé *post hoc* analysis was undertaken. All data was analyzed using a standard statistical package (Statistica, Statsoft Inc).

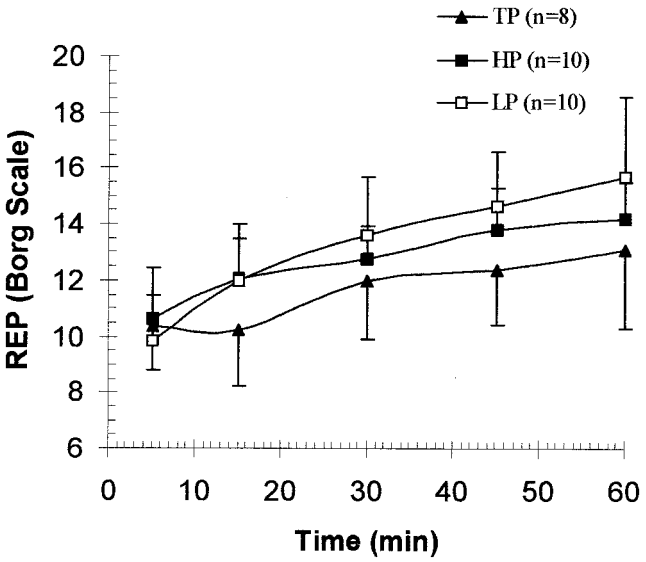

FIGURE 1—Ratings of perceived exertion (RPE) for the TP, HP, and LP athletes during exercise in warm conditions.

RESULTS

The peak physiological responses to incremental wheelchair ergometry are shown in Table 1. $\dot{V}O_{2peak}$ and peak ventilation rate ($\dot{V}E_{2peak}$) values for the LP athletes were greater than those for the HP and TP athletes. Peak heart rate and peak blood lactate for the HP and LP athletes were similar but greater than those for the TP athletes.

Oxygen consumption ($\dot{V}O_2$), heart rate, and ventilation rate ($\dot{V}E$) were lower for the TP athletes throughout the exercise period when compared with the HP and LP athletes ($P < 0.05$). No differences were observed between groups for percent $\dot{V}O_{2peak}$ (62.3 ± 13.2, 54.5 ± 8.0, and 57.4 ± 6.6%, for the TP, HP, and LP at 5 min of exercise, respectively), BLa, or RER responses during exercise. After 30 min of exercise, HR for the HP athletes (146 ± 14 $beats \cdot min^{-1}$) was elevated from values at 5 min of exercise (125 ± 24 $beats \cdot min^{-1}$; $P < 0.05$) and remained elevated until the end of the exercise period (153 ± 27 $beats \cdot min^{-1}$; $P < 0.05$), whereas for the LP athletes, HR was elevated from values at 5 min of exercise by 45 min of the exercise protocol (140 ± 40 and 159 ± 16 $beats \cdot min^{-1}$, respectively; $P < 0.05$) also remaining elevated until the end of the exercise period (168 ± 17 $beats \cdot min^{-1}$; $P < 0.05$). An increase in HR from 5 min of exercise (98 ± 13 $beats \cdot min^{-1}$) was not observed for the TP athletes during exercise with values reaching 109 ± 15 $beats \cdot min^{-1}$ at the end of the exercise period. RPE throughout the exercise period is shown in Figure 1. Perceptions of effort were elevated above those reported at 5 min of exercise ($P < 0.05$) by 45 min for TP (12.4 ± 2.1), 30 min for HP (12.8 ± 1.2), and 15 min for LP (12.0 ± 2.0). Values then remained elevated until the end of the exercise period ($P < 0.05$) for all groups (13.2 ± 2.3, 14.2 ± 1.3, and 15.7 ± 2.8). When compared with the TP athletes, RPE for the LP athletes was greater at 45 min of exercise until the end of the exercise period ($P < 0.05$). No differences were observed

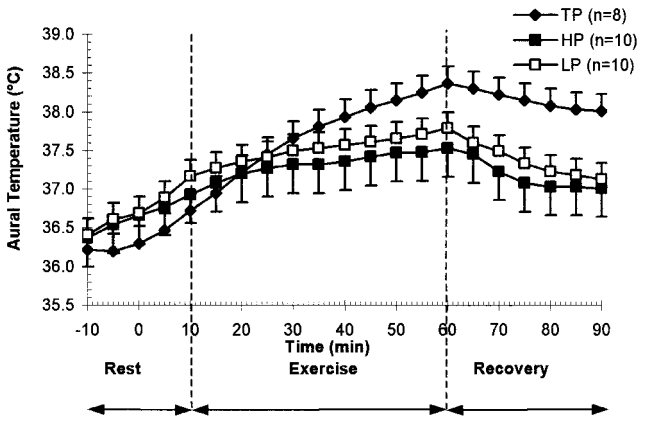

FIGURE 2—Aural temperature for the TP, HP, and LP athletes at rest, during exercise, and recovery in warm conditions.

between ratings of perceived exertion for the HP and LP athletes, or the HP and TP athletes.

At rest in a cool environment, before entering the environmental chamber, aural temperatures for the TP, HP, and LP athletes were similar (36.2 ± 0.2°C; 36.4 ± 0.4°C, and 36.4 ± 0.2°C, respectively). After 60 min of exercise, aural temperature increased from resting values by 2.1 ± 0.5°C, 1.1 ± 0.3°C, and 1.4 ± 0.5°C for the TP, HP, and LP athletes, respectively ($P < 0.05$; Fig. 2). Aural temperature was elevated from rest after 5 min of exercise ($P < 0.05$) until the end of recovery for LP, and after 10 min of exercise for the TP and HP athletes ($P < 0.05$). Aural temperature for the TP athletes was greater than that observed for the HP and LP athletes after 45 and 60 min of exercise, respectively, remaining elevated until the end of recovery ($P < 0.05$). No differences were observed between the aural temperature responses for the HP and LP athletes during exercise or recovery. After 30 min of recovery aural temperature remained elevated when compared with rest for the TP, HP, and LP athletes by 1.9 ± 0.5°C, 0.6 ± 0.2°C, and 0.8 ± 0.3°C, respectively ($P < 0.05$).

Skin temperature responses for the TP, HP, and LP athletes at rest, during prolonged exercise, and recovery in warm conditions are shown in Figures 3 and 4. Forehead, forearm, upper-arm, back, chest, and thigh skin temperatures increased from rest in the environmental chamber until the end of exercise for each group ($P < 0.05$). After 5 min and 15 min of exercise, respectively, abdomen skin temperature for the TP and LP athletes was elevated above values observed at rest in the environmental chamber until the end of recovery ($P < 0.05$). Abdomen skin temperature for the HP athletes remained unchanged from rest during both exercise and recovery. Calf skin temperatures for the TP, HP, and LP athletes were elevated from rest in the environmental chamber from 60, 15, and 10 min of exercise until the end of recovery, respectively ($P < 0.05$).

No differences were observed between the HP and LP athletes for any of the skin temperature responses during exercise or recovery. However, upper-arm skin temperature for the TP athletes was higher than for both the HP and LP groups after 30 and 60 min of exercise until the end of

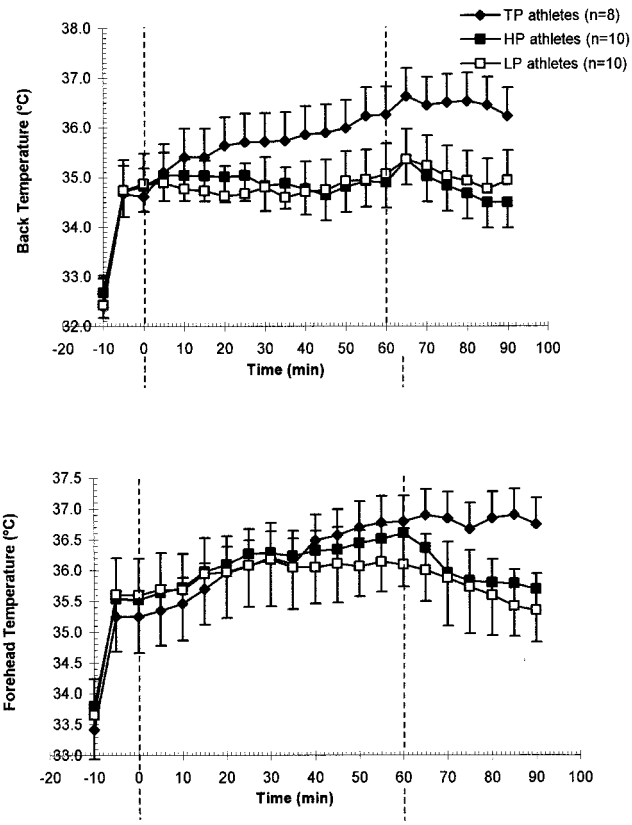

FIGURE 3—Forehead and back skin temperatures for the TP, HP, and LP athletes at rest, during exercise, and recovery in warm conditions.

recovery, respectively ($P < 0.05$). Back skin temperature for the TP athletes was higher than observed for the HP and LP athletes from 45 min of exercise until the end of recovery ($P < 0.05$). Chest and forehead skin temperatures were higher for the TP athletes when compared with the HP and LP athletes throughout recovery from exercise only ($P < 0.05$). Abdomen skin temperature for the TP athletes was higher than observed for the HP athletes during recovery ($P < 0.05$) but no different from the LP athletes during exercise or recovery. No differences were noted between groups for forearm, thigh, and calf skin temperatures during either the exercise or recovery periods.

At rest in the environmental chamber, no differences in heat storage were observed between groups (1.06 ± 0.53 $J \cdot g^{-1}$, 1.52 ± 0.53 $J \cdot g^{-1}$, and 1.81 ± 0.76 $J \cdot g^{-1}$ for the TP, HP, and LP athletes, respectively). After 15 min of exercise, heat storage was greater for all groups when compared with rest ($P < 0.05$), increasing further until 60 min of exercise (8.64 ± 1.81 $J \cdot g^{-1}$, 5.45 ± 1.33 $J \cdot g^{-1}$, and 6.71 ± 1.30 $J \cdot g^{-1}$ for the TP, HP, and LP athletes respectively; $P < 0.05$). No differences were observed between heat storage values for the HP and LP athletes during exercise, although values for the LP athletes tended to be greater. The TP athletes demonstrated greater increases in heat storage than those observed for the HP and LP athletes from 30 min of exercise until the end of recovery ($P < 0.05$). At the end of 30 min of recovery, heat storage for the HP and LP athletes

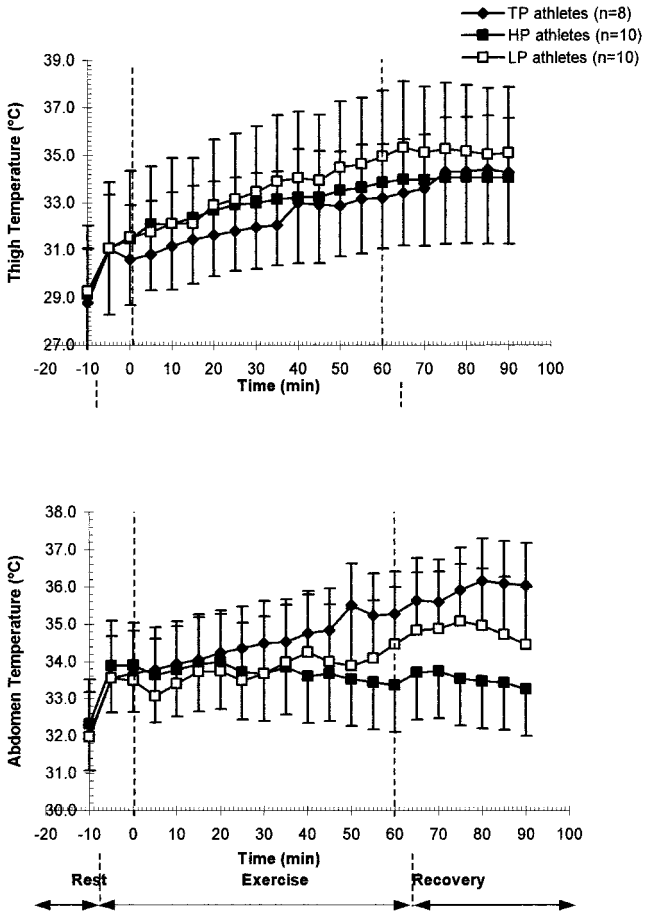

FIGURE 4—Abdomen and thigh skin temperature for the TP, HP, and LP athletes at rest, during exercise, and recovery in warm conditions.

was lower than observed at the end of exercise (3.88 ± 1.37 $J \cdot g^{-1}$ and 5.07 ± 0.97 $J \cdot g^{-1}$, respectively; $P < 0.05$), whereas values for the TP athletes were no different (7.89 ± 2.10 $J \cdot g^{-1}$). Heat storage at the end of exercise in relation to the level of spinal cord injury, for the athletes with known spinal cord lesion level, is shown in Figure 5.

Changes in plasma volume during exercise were greater for the LP athletes when compared with the TP and HP athletes, respectively ($P < 0.05$; Table 2). Changes in body

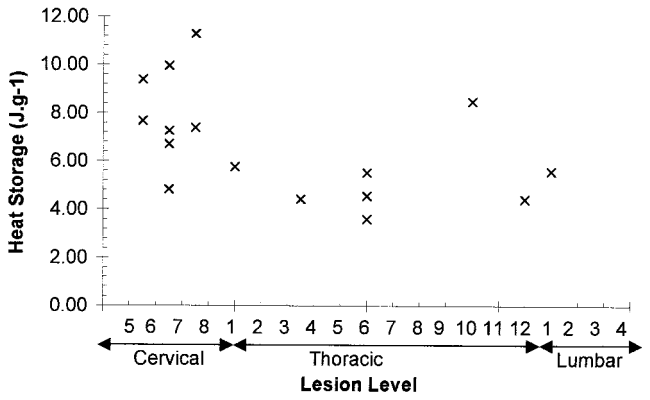

FIGURE 5—Heat storage at the end of exercise in relation to lesion level for athletes with specifically known injury level.

TABLE 2. Fluid consumption and changes in plasma volume and body mass during prolonged wheelchair exercise in the heat for TP, HP, and LP athletes.

	TP	HP	LP
Change in plasma volume (%)	0.76 ± 0.48	−3.47 ± 6.20	−6.30 ± 3.81§
Change in body mass (kg)	0.45 ± 0.60	−0.24 ± 0.38*	−0.31 ± 0.44*
Fluid consumed (mL)	764 ± 342	472 ± 252	381 ± 251*
Adjusted change in body mass (kg)	−0.31 ± 0.42	−0.71 ± 0.33	−0.69 ± 0.53

* Significantly different from TP at $P \leq 0.05$ level.
§ Significantly different from TP and HP at $P \leq 0.05$ level.

mass were greater for the HP and LP athletes during exercise when compared with the TP athletes ($P < 0.05$). Greater volumes of fluid were consumed by the TP athletes during exercise when compared with the LP athletes ($P < 0.05$), with the HP athletes consuming a similar volume of fluid to the LP athletes. When these values were combined to calculate total weight loss, no differences were observed between groups.

DISCUSSION

The main finding of the present study was that during 60 min of wheelchair exercise in warm conditions, both trained paraplegics and trained tetraplegics were able to complete the exercise test. However, differing degrees of thermal imbalance were observed between groups. Furthermore, during recovery from exercise, all athletes demonstrated positive heat storage, with this being considerably greater for the tetraplegic group.

Aural temperatures for the TP, HP, and LP athletes at rest were similar to those reported for able-bodied athletes and paraplegics with low-level lesions (22,23). A lower resting body temperature has been observed previously for the spinal cord injured when compared with able-bodied subjects (1,15). However, such a decrease in resting body temperature between able-bodied (24) and spinal cord-injured individuals and between subjects of different lesion levels is not supported by the results of the present study.

The LP athletes demonstrated an earlier increase in aural temperature during exercise when compared with the TP and HP athletes. This is most likely due to the greater recruitable muscle mass and absolute metabolic heat production of these athletes. However, by 20 min of exercise, similar increases in aural temperature were observed for all groups. After this point, aural temperatures for the HP and LP athletes increased at much slower rates when compared with the TP athletes. The gradual increase in aural temperature indicates that although the HP and LP were not able to dissipate heat as quickly as it was gained, the cooling mechanisms initiated by these groups were more effective than for the TP group. The greater and continued increase in aural temperature for the TP group is most likely to be due to the loss of evaporative cooling and effective heat dissipation observed in this population (21).

At the end of the exercise period, no differences were observed between the increases in aural temperature for the HP and LP athletes. These results are inconsistent with previous reports of resting heat exposure where HP subjects were unable to effectively regulate body temperature

(14,25). The current study involved trained individuals regularly participating in exercise durations and intensities that would result in increased body temperature. Consequently, a partial acclimatization involving earlier sweating onset and greater sweat output of the sensate area may have existed as observed for able-bodied subjects (11). A more effective dissipation of heat from the sensate skin of the paraplegic athletes in the present study, as a result of training, may have contributed to the ability of these athletes to successfully complete prolonged exercise in a warm environment. Furthermore, although sweating capacity would be significantly reduced in such subjects (21), it would be balanced by the amount of recruitable muscle mass for heat production. The results of the present study suggest that both trained HP and LP groups can regulate body temperature as effectively as each other during prolonged exercise in warm conditions but not as effectively as able-bodied individuals during arm exercise (24).

The TP athletes in the present study were also able to successfully complete prolonged exercise in warm conditions even though large increases in body temperature occurred. It is interesting to note that Guttman (8) reported tetraplegic patients who were conditioned to cool or warm environmental temperatures demonstrated improved tolerance during cool and warm stressors, respectively. Although the mechanisms of such a response have not been reported, it is possible that regular increases in body temperature during training may have conditioned the TP athletes in the present study to thermal stress in a similar way, thus improving heat tolerance. Whether or not heat tolerance was improved in the TP group, the differences in heat dissipation and heat storage between paraplegic and tetraplegic athletes during exercise were also evident during recovery, with the TP athletes maintaining most of the heat stored during exercise.

Forehead skin temperature was similar during exercise for each group of athletes indicating similar dissipation of heat from this area for all athletes. Large forehead sweat rates and increased evaporative cooling have been previously reported for both paraplegic and tetraplegic subjects during exercise in hot conditions (22). However, Petrofsky (21) reported that local forehead sweat rates for tetraplegic subjects were so large that sweat dripped from the skin surface and was of no thermoregulatory benefit. Such large dripping sweat rates were not observed in the present study. Although the present observations are only subjective and not quantitative, effective forehead cooling has been suggested to be related to selective brain cooling (30). If this is indeed true and a degree of forehead cooling did occur in the TP group, similar forehead skin temperatures for all groups during exercise may suggest a physiological attempt to maintain a functional brain temperature. Although this would not be consistent with the greater aural temperature of the TP group, it would provide a relatively large thermal gradient for direct heat conductance to the surface of the head, possibly improving thermal tolerance and completion of the exercise test in the presence of thermal imbalance.

Back, chest, and also upper-arm skin temperatures for the TP athletes demonstrated a gradual increase when compared with the relatively steady state skin temperatures for the paraplegic groups. Similar skin-temperature responses to these have been reported for resting heat exposure (9) and demonstrate differences in heat dissipation of insensate and sensate skin. As sweating does not occur in areas of insensate skin, heat cannot be dissipated, resulting in continual increases in skin temperature. Therefore, as the majority of the upper-body skin of TP athletes is insensate, this may explain the greater increases in skin temperature when compared with the sensate upper-body skin of the HP and LP athletes.

The present study examined athletes employing both basketball and track wheelchairs in an attempt to provide greater specificity and ecological validity to the results. However, due to differences in chair design and athlete positioning within them, some site-specific differences in skin temperature may have occurred, for example, the back and abdomen skin sites. When considering the back skin temperature site, further analysis of the data demonstrated little difference between the temperature responses of any group in relation to chair design. This may not be unexpected as neither chair design incorporates a high backrest, which would potentially cover the skin thermistor. However, when considering the abdomen site, the chair design may have a greater impact on the thermoregulatory responses. For example, two LP athletes exercising in basketball wheelchairs demonstrated slight decreases in abdomen skin temperature during exercise. These values were within the cooler range of responses observed for the athletes employing track wheelchairs. However, for the HP group, where wheelchair design was evenly matched, no differences were observed between chair types. This would lead us to conclude that chair design may be an important consideration for the abdomen skin site where subjects with lower-level spinal cord lesions are examined. It is not anticipated that other skin temperature sites would be affected by wheelchair type. Differences between groups based on the degree of thermal dysfunction though were robust and consistent whatever the design of wheelchair. A more detailed comparison of chair design on the full complement of thermoregulatory measures would appear to be pertinent for future study. It must though be considered that individuals employing wheelchairs of different design to their habitual or sporting wheelchairs may exhibit variations in pushing economy and heat exchange which, in such a study, may be difficult to control for.

Calf skin temperatures for both paraplegic groups were elevated within the first 15 min of exercise, whereas the TP demonstrated a much later elevation at 60 min of exercise. Although the lower limb was insensate for all athletes and local metabolic heat production would be severely reduced, the paraplegic athletes exercised at a much greater whole body metabolic rate. In addition to greater heat production, as vasomotor disruption is less with lower levels of spinal cord injury (13), this would mean that the potential for greater convective heat transfer within the body was also

greater, resulting in an earlier increase in lower body skin temperatures. The more gradual increase in calf skin temperature for the TP group is therefore more likely to have a larger, passive environmental component. However, all athletes would have experienced heat gain from the environment due to the colder lower limbs (22) being much cooler than the environmental temperature employed in the present study.

As the metabolic rate of the TP group was lower than for the paraplegic groups, the primary source of whole-body heat gain for this group, as noted for the lower body, would appear to be external rather than metabolic in origin. In conjunction with this, if we consider the increase in back and chest skin temperature reported earlier, it appears that it is the area below the spinal cord lesion, which is a potent site of for heat storage, rather than the lower limb alone as reported previously for LP athletes (22). Such skin temperatures for all groups were greater than the environmental temperature and would have provided a possible avenue for dry heat exchange (6). However, as evaporative cooling is the most potent form of heat loss (29), such dry heat exchange was probably unable to effectively dissipate large quantities of heat from the skin, resulting in the continual increases in skin temperature and heat storage for the TP group.

Lower body skin temperatures for spinal cord-injured subjects at rest in a cool environment are cooler than those for able-bodied subjects (22). Furthermore, as precooling athletes before exercise results in reduced heat storage during exercise (17), a cooler lower body may be beneficial to spinal cord-injured subjects. A cooler lower body secondary to spinal cord lesion may enable heat storage to occur within the lower body during exercise, without excessive increases in aural and skin temperatures.

The LP athletes demonstrated greater changes in plasma volume and body weight during exercise when compared with the TP and HP athletes. The greater body surface area available for sweating and greater total sweat output of the LP (6) would contribute to greater body fluid losses, dehydration, and weight loss when compared with athletes with a smaller area of sensate skin. Consequently, the HP athletes, with a greater reduction in sweating capacity than the LP athletes (9), demonstrated smaller changes in plasma volume and body weight during exercise. Furthermore, the TP athletes, with the greatest reduction in sweating capacity, demonstrated little change in plasma volume. However, the TP athletes consumed the largest amount of fluid during exercise, which, in the absence of sweating and fluid loss, may explain the increase in body weight. Although the TP group did not need to replenish large fluid losses during exercise, they were under the greatest thermal strain, which would explain the intake of large amounts of fluid. These results suggest that changes in plasma volume and body weight for spinal cord-injured athletes during exercise are related to the surface area available for sweating and, therefore, to the level of spinal cord injury (12).

Although the LP demonstrated greater fluid loss, the HP athletes demonstrating an increase in heart rate early in exercise, reminiscent of cardiovascular drift. Paraplegics with high-level spinal cord injuries demonstrate greater reductions in stroke volume than paraplegics with low-level spinal cord injuries due to greater vasomotor disruption (13). Therefore, the earlier increase in heart rate for the HP during exercise may have been due to a greater reduction in stroke volume due to peripheral displacement of blood rather than fluid losses. Further studies of fluid shifts in this population are warranted.

At the end of the exercise period, the LP athletes demonstrated greater perceptions of effort than the TP athletes. This may be due to the greater level of dehydration for the LP athletes, which is an important factor contributing to the perceptions of effort during exercise (18,20). However, although plasma volume for the TP athletes remained unchanged during exercise and lower perceptions of effort were reported, the TP athletes were under a much greater thermal strain than the LP athletes throughout the exercise period. As fluid consumption during exercise has been shown to attenuate perceptions of effort during exercise (2,19), it is possible that perceptions of effort for the TP athletes may have been reduced by the consumption of larger volumes of fluid when compared with the HP and LP athletes during exercise. Such behavior may have also contributed to them being able to complete the exercise task although under a much greater thermal strain. Furthermore, the TP athletes commented upon the sensation of heat building up only across their forehead, faces, and shoulders during the exercise period. Perceptions of effort for these athletes may therefore have been attenuated further by the reduced sensory input to the brain from the hyperthermic area below the level of spinal cord injury.

The results of this study have shown that tetraplegic and paraplegic athletes with high-level and low-level spinal cord injury are able to complete 60 min of wheelchair ergometry in warm conditions. However, although the effectiveness of body temperature regulation for the paraplegic groups was better than for tetraplegic athletes, this was not as effective as previous reports of able-bodied athletes in similar conditions. For all athletes, the area below the spinal cord lesion, in particular the lower limb, was a potent site for heat storage. Increasing exercise duration or intensity, or environmental temperature or humidity, may result in the thermoregulatory responses of paraplegic and tetraplegic athletes being compromised.

REFERENCES

1. ATTIA, M., and P. ENGEL. Temperature regulatory set point in paraplegics. In: *Thermal Physiology*, J. S. Hales (Ed.). New York: Raven Press, 1984, pp. 79–82.

2. BARR, S. I., D. L. COSTILL, and W. J. FINK. Fluid replacement during prolonged exercise effects of water, saline or no fluid. *Med. Sci. Sports Exerc.* 23:811–817, 1991.

3. BENZINGER, T. H., and G. W. TAYLOR. Cranial measurements of internal temperature in man. In: *Temperature: Its Measurement and Control in Science and Industry. 3, Biology and Medicine*, J. R. Hardy (Ed.). New York: Reinhold, 1963, pp. 111–120.
4. DILL, D. B., and D. L. COSTILL. Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. *J. Appl. Physiol.* 37:247–248, 1974.
5. DURNIN, J. G. V. A., and J. WORMESLY. Body fat assessment from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged 16–71 years. *Br. J. Nutr.* 32:77–97, 1974.
6. FITZGERALD, P. I., D. A. SEDLOCK, and R. G. KNOWLTON. Circulatory and thermal adjustments to prolonged exercise in paraplegic women. *Med. Sci. Sports Exerc.* 22:629–635, 1990.
7. GASS, G. C., E. M. CAMP, E. R. NADEL, T. H. GWINN, and P. ENGEL. Rectal and rectal vs. oesophageal temperature in paraplegic men during prolonged exercise. *J. Appl. Physiol.* 64:2265–2271, 1988.
8. GUTTMAN, L. *Spinal Cord Injuries, Their Management and Treatment.* London: Blackwell Scientific Publications, 1976, pp. 327–329.
9. GUTTMAN, L., J. SILVER, and C. H. WYNDHAM. Thermoregulation in spinal man. *J. Physiol.* 142:406–419, 1958.
10. HAVENITH, G., Y. INOUE, V. LUTTIKHOLT, and W. L. KENNEY. Age predicts cardiovascular, but not thermoregulatory responses to humid heat stress. *Eur. J. Appl. Physiol.* 70:88–96, 1995.
11. HENANE, R., and R. FLANDROIS, and J. P. CHARBONNIER. Increase in sweating sensitivity by endurance conditioning in man. *J. Appl. Physiol.* 43:822–828, 1997.
12. HOPMAN, M. T. E., B. OESEBURG, and R. A. BINKHORST. Cardiovascular responses in persons with paraplegia to prolonged arm exercise and thermal stress. *Med. Sci. Sports Exerc.* 25:577–583, 1993.
13. HOPMAN, M. T. E., B. OESEBURG, and R. A. BINKHORST. Cardiovascular response in paraplegic subjects during arm exercise. *Eur. J. Appl. Physiol.* 65:73–78, 1992.
14. HUCKABA, C. E., D. B. FREWIN, J. A. DOWNEY, H.-S. TAM, R. C. DARLING, and H. Y. CHEH. Sweating responses of normal, paraplegic and anhidrotic subjects. *Arch. Phys. Med. Rehabil.* 57:268–274, 1976.
15. ISHII, K., M. YAMASAKI, S. MURAKI, et al. Tympanic temperature and skin temperatures during upper limb exercise patients with spinal cord injury. *Jpn. J. Phys. Fitness Sports Med.* 44:447–455, 1995.
16. KERK, J. K., P. S. CLIFFORD, A. C. SNYDER, et al. Effect of an abdominal binder during wheelchair exercise. *Med. Sci. Sports Exerc.* 27:913–919, 1995.
17. KRUK, B., H. PEKKARINEN, M. HARRI. K. MANNINEN, and O. MANNINEN. Thermoregulatory responses to exercise at low ambient temperature performed after pre-cooling or preheating procedures. *Eur. J. Appl. Physiol.* 59:416–420, 1990.
18. MONTAIN, S. J., and E. F. COYLE. Influence of graded dehydration on hyperthermic and cardiovascular drift during exercise. *J. Appl. Physiol.* 73:1340–1350, 1992.
19. MURRAY, S. R., T. J. MICHAEL, and P. D. MCCLELLAN. The influence of fluid replacement on heart rate and RPE during exercise in a hot, humid environment. *J. Strength Cond. Res.* 9:251–254, 1995.
20. NOAKES, T. D. Fluid replacement during exercise. *Exerc. Sports Sci. Rev.* 21:297–330, 1993.
21. PETROFSKY, J. S. Thermoregulatory stress during rest and exercise in heat in patients with a spinal cord injury. *Eur. J. Appl. Physiol.* 64:503–507, 1992.
22. PRICE, M. J., and I. G. CAMPBELL. Thermoregulatory responses of able-bodied and paraplegic athletes to prolonged upper body exercise. *Eur. J. Appl. Physiol.* 76:552–560, 1997.
23. PRICE, M. J., and I. G. CAMPBELL. Thermoregulatory and physiological responses of wheelchair athletes to prolonged arm crank ergometry and wheelchair ergometry. *Int. J. Sports Med.* 20:457–463, 1999.
24. PRICE, M. J., and I. G. CAMPBELL. Thermoregulatory responses during prolonged upper-body exercise in cool and warm conditions. *J. Sports Sci.* 20:519–527, 2002.
25. RAMANATHAN, N. L. A new weighting system for mean surface temperature of the human body. *J. Appl. Physiol.* 19:531–533, 1964.
26. RANDALL, W. C., R. D. WURSTER, and R. J. LEWIN. Responses of patients with high spinal transection to high ambient temperatures. *J. Appl. Physiol.* 21:985–993, 1966.
27. RAWSON, R. O., and J. D. HARDY. Sweat inhibition by cutaneous cooling in normal, sympathectomised and paraplegic man. *J. Appl. Physiol.* 22:287–291, 1967.
28. TOTEL, G. L., R. E. JOHNSON, F. A. FAY, J. A. GOLDSTEIN, and J. SCHICK. Experimental hyperthermia in traumatic quadriplegia. *Int. J. Biometeorol.* 15:346–355, 1971.
29. WALLERSTRÖM, B., and I. HOLMÉR. Efficiency of sweat evaporation during exercise in the heat. In: *Thermal Physiology*, J. S. Hales (Ed.). New York: Raven Press, 1984, pp. 433–436.
30. WHITE, M.D., and M. CABANAC. Physical dilatation of the nostrils lowers the thermal strain of exercising humans. *Eur. J. Appl. Physiol.* 70:200–206, 1995. .0