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TEMPERATURE REGULATION DURING UPPER BODY EXERCISE: ABLE BODIED AND SPINAL CORD INJURED

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Arm-Crank Exercise, Core Temperature, Cutaneous Blood Flow, Disabled Populations, Sweating Responses, Thermoregulation

INTRODUCTION

Human thermoregulatory responses to muscular exercise have been described by experiments that almost exclusively employed lower body (treadmill and cycle) exercise. Few investigators have examined human thermoregulatory responses to upper body exercise; this is surprising since in 1947 Asmussen and Nielsen (1) reported thermoregulatory response differences between upper (arm-crank) and lower body exercise. Humans in thermally stressful environments often engage in upper body exercise for industrial, agricultural and military tasks as well as sports activities (13,36,45). Likewise, astronauts are required to do a substantial amount of exercise with their upper body (17). In all of these situations, the individual needs to dissipate the metabolic heat as well as the environmental heat load in order to regulate body temperature. If the body temperature is not regulated within a narrow range, the individual will fatigue and discontinue the exercise task and possibly suffer a heat injury. As a result, it is important to know whether upper body exercise poses any unique thermoregulatory problems.

Disabled individuals who suffer from spinal cord injuries, such as paraplegics, must depend on their upper body musculature for locomotion via the use of wheelchairs. For these individuals, rehabilitation programs include exercise training to improve their health and capability for wheelchair exercise (6,19). Therefore, many disabled individuals will be exposed to thermal stress (metabolic and environmental) while performing upper body exercise. It is often not appreciated that the spinal cord injury not only results in the paralysis of skeletal muscle but also the impairment of the autonomic nervous system. The autonomic nervous system is important for the control of body heat loss via alterations in cutaneous blood flow as well as thermoregulatory sweating. As a result, individuals with an impaired ability to

dissipate body heat may be required to perform upper body exercise which in itself might pose several thermoregulatory problems.

This paper considers human thermoregulatory response differences between upper body and lower body exercise. In addition, the thermoregulatory problems of spinal cord injured individuals are examined.

TEMPERATURE REGULATION

Body temperature is believed to be regulated by a proportional control system (18,40). A proportional system is defined as the graded response of a controlled variable (e.g., cutaneous blood flow, sweating) to the displacement of the regulated variable (e.g., body temperature). Both peripheral and central thermal receptors provide afferent input into the hypothalamic thermoregulatory centers where this information is processed (5) with a resultant effector signal to initiate and maintain the thermoregulatory responses of cutaneous blood flow and sweating (18,40). In humans, the peripheral thermal receptors are located in the skin, and central thermal receptors are presumed to be primarily located in the hypothalamus, but other receptors are thought to be located in the spinal cord, heart, pulmonary vessels and skeletal muscle (10,11,28,34,43).

Several mathematical models could be described for human temperature regulation; however, for this paper we will use a linear additive model to describe the control of thermoregulatory effector responses by the equation:

$$R\text{-}R_o = a\ (T_c\text{-}T_{co}) + b\ (T_{sk}\text{-}T_{sko})$$

where, R is a thermoregulatory response;

R_o is a basal value of R;

a and b are proportional control constants;

T_{co} and T_{sko} are the basal values for core and mean skin temperatures, respectively.

For humans, the proportional control constants of 0.9 for a, and 0.1 for b are used for the thermoregulatory effector responses (26). This ratio of 9:1 means that a change of $1^{o}C$ in core temperature elicits about nine times as great a change in thermoregulatory effector response as a $1^{o}C$ change in mean skin temperature.

The afferent thermal information is processed in the hypothalamic thermoregulatory centers, and a resultant effector signal is generated to control heat loss. The thermoregulatory effector signal descends through the brain stem and spinal tracts to exit into the thoracolumbar division of the autonomic nervous system. The post-ganglionic sympathetic fibers which innervate the eccrine sweat gland are nonomylinated class C fibers that are primarily cholinergic. The eccrine sweat glands respond primarily to thermal stress through sympathetic cholinergic stimulation. However, it appears that circulating catecholamines, in particular epinephrine, facilitate thermoregulatory sweating, as there are α and β adrenergic receptors associated with eccrine sweat glands (40). Cutaneous blood flow is affected by local skin temperature acting directly on the vascular smooth muscle and by reflexes operating through the autonomic nervous system. During heat stress, active vasodilation of the cutaneous vasculature of the arm, thigh and calf is mediated by sympathetic stimulation. Finally, it is known that eccrine sweat secretion and cutaneous vasodilation are clearly associated, but the vasoactive substance responsible is not known (40).

During exercise, core temperature initially increases rapidly and subsequently increases at a reduced rate until heat loss equals heat production, and essentially steady-state values are achieved (40). The elevation of core temperature represents the storage of metabolic heat which is a by-product of skeletal muscle contraction. At the initiation of exercise, the metabolic rate increases immediately; however, the

thermoregulatory effector responses which enable dry (radiative and convective) and evaporative heat loss to occur increase more slowly. Eventually, these heat loss mechanisms increase sufficiently to balance metabolic heat production allowing a steady-state core temperature to be achieved. The relative contributions of dry and evaporative heat exchange to the total heat loss, however, varies with the environmental conditions. In cool environments, the large skin-to-ambient temperature gradient facilitates dry heat loss; but as ambient temperature increases the gradient for dry heat exchange diminishes, and there is a greater reliance upon evaporative heat exchange. When the ambient temperature is equal to skin temperature, evaporative heat exchange will account for essentially all of the heat loss. The capacity for evaporative heat loss is dependent upon the skin-to-ambient vapor pressure gradient.

During exercise, the elevation in core temperature is proportional to the metabolic rate and nearly independent (particularly in low humidity conditions) of environmental temperature (22,29). The range of ambient conditions that core temperature increases in proportion to the metabolic rate, and independent of environment, is called the "prescriptive zone" (22). As the metabolic rate increases, the upper limit of the "prescriptive zone" decreases (40). The relationship between metabolic rate and core temperature is strong for a given individual (40) but does not always hold well for comparisons between different individuals. Several investigators (2,35) have reported that the use of relative intensity (percent of maximal oxygen uptake), rather than actual metabolic rate (absolute intensity), removes much of the intersubject variability for the core temperature elevation during exercise.

UPPER BODY EXERCISE

There is debate as to whether upper body exercise results in different core temperature values than those elicited by lower body exercise at the same metabolic

rate. Arm-crank exercise is often used as the mode of upper body exercise because it is the least physiologically complex form of upper body exercise (36). Several (1,27,28) investigators suggested that arm-crank exercise elicited different thermoregulatory responses and core temperature values than lower body exercise. Examination of those early studies, however, indicate that small sample size, technical problems and inconsistent results make any conclusions tenuous. Table 1 provides an outline of factors which might result in different thermoregulatory responses for upper body exercise.

TABLE 1 ABOUT HERE

Maximal effort arm-crank exercise elicits an oxygen uptake that is approximately 70% of that obtained during maximal effort cycle exercise (36). It can be argued that if core temperature elevations during exercise are determined by relative intensity (with respect to the musculature employed) then arm-crank exercise would be expected to elicit a higher core temperature for a given metabolic rate than would lower body exercise. There is also a different source of metabolic heat during upper than lower body exercise: as a result, temperatures measured within a given body region may change relative to other body regions. Therefore, different indices of core temperature, such as esophageal or rectal, might provide disparate values. The surface area-to-mass ratio of the arms would be expected to be greater than the legs. A greater surface area-to-mass ratio for the exercising limb could facilitate heat loss and alter thermoregulatory responses during exercise.

There are several neural factors that might modify thermoregulatory responses to upper body exercise. Robinson and colleagues (34) theorized that thermal receptors located in skeletal muscle and in draining veins may provide an afferent input to the

thermoregulatory centers Likewise, mechanoreceptors and metaboreceptors within the skeletal muscles might provide afferent thermoregulatory information (28). Since upper body exercise employs a relatively smaller skeletal muscle mass than lower body exercise (36), a greater metabolic rate and heat production per unit of muscle must occur in order to perform exercise at a given oxygen uptake level. Thermoregulatory afferent information should therefore be somewhat quantitatively and qualitatively different during upper than lower body exercise. This logic has led several investigators to suggest that there is a different thermoregulatory setpoint during these two exercise types (1,41). Finally, Davies and colleagues (8) reported that plasma catecholamine concentrations are inversely related to the skeletal muscle mass used during submaximal exercise at a given oxygen uptake. Therefore, vasoconstrictor drive would be expected to be greater during upper than lower body exercise.

The cardiovascular system may have greater difficulty in supporting the thermoregulatory system during upper than lower body exercise. For example, during upper body exercise the legs are inactive, so there is less skeletal muscle pump activity to facilitate venous return. If upper body exercise were performed in the heat, the large blood volume displaced in the cutaneous vasculature combined with minimal skeletal muscle pump activity could make it difficult to maintain cardiac output. In addition, there is a greater total peripheral resistance and myocardial afterload during upper body exercise at a given oxygen uptake level (36). Finally, there is a greater hemoconcentration at a given oxygen uptake level during upper than lower body exercise (25,31). It is known that a reduced blood volume will result in less efficient thermoregulatory responses during leg exercise (37). As a result, the greater hemoconcentration might result in greater body heat storage during upper than lower body exercise.

Table 2 provides a summary of the investigations which have examined the core temperature and thermoregulatory responses to upper body exercise. During the following paragraphs, an attempt will be made to explain some of the discrepancies between these investigations.

TABLE 2 ABOUT HERE

In 1947, Asmussen and Nielsen (1) studied two subjects' core (rectal) temperature responses to arm-crank and cycle exercise at the same metabolic rates. They found that after the fortieth minute of exercise, the elevation in rectal temperature was 0.28°C less during arm-crank than cycle exercise. The authors noted that the subjects did not achieve steady-state rectal temperature levels by forty minutes, but were unable to exercise longer because of local fatigue. Asmussen and Nielsen (1) were concerned that rectal temperature values may have been spuriously high during cycle exercise because of the warm venous blood returning from the leg muscles. They conducted additional experiments in which they measured stomach temperature. In agreement with their rectal temperature data, the stomach temperature values were consistently lower during arm-crank than cycle ergometer exercise. Since both indices of core temperature were lower during arm crank exercise, they concluded that upper body exercise results in a reduced thermoregulatory setting than lower body exercise.

In 1968, Nielsen (27) examined two subjects' core (rectal and esophageal) temperature responses to arm-crank and cycle exercise over a range of metabolic rates. Rectal temperature values (mean of values obtained at four depths) were found to be lower (0.20 to 0.40°C) during arm-crank than cycle exercise. In contrast, the esophageal temperature values were not different between the two exercise types.

Figure 1 presents the steady-state esophageal temperature responses in relation to metabolic rate during arm-crank and cycle exercise. In addition, the subjects' sweating rates and thermal conductance were not different between the two exercise types. They concluded that thermoregulatory control during exercise was not modified by the muscle groups employed.

FIGURE 1 ABOUT HERE

Subsequent investigators have found no difference in either tympanic (7) or esophageal (38) temperature values between upper and lower body exercise performed at a given metabolic rate. These observations confirmed Nielsen's thesis that exercise type does not modify the thermoregulatory control. Nielsen's data, however, did raise the possibility that rectal temperature might provide systematically low values for upper body exercise. This possibility was consistent with some indirect observations made by Nielsen and Nielsen in 1962 (28). These authors (28) found that during leg exercise esophageal temperature was lower than rectal temperature, but that during arm exercise esophageal temperature was equal to rectal temperature values. The investigators measured rectal temperature at four depths (12,17,22 and 27 cm) during either cycle or arm-crank exercise. They found that during cycle exercise the measurement depth did not influence rectal temperature values, but that during arm-crank exercise the deeper rectal measurements (22 and 27 cm) tended to produce lower temperature values. Therefore, rectal temperature measurements at a depth of 20 cm or greater may result in spuriously low core temperature estimates during upper body exercise.

Several investigators (32,39,47) recently have compared rectal temperature (10 cm) responses between upper and lower body exercise. Sawka et al. (39) measured

nine subjects' rectal temperature responses during arm crank and cycle exercise at the same metabolic rate as well as relative intensity (% of peak $\dot{V}O_2$ for specific exercise type). During the experiments matched for metabolic rate, the subjects' steady state rectal temperatures and total body sweating rates were the same for both exercise types. On the other hand, during the experiments matched for relative intensity, the subjects' rectal temperatures and total body sweating rates were lower during arm-crank than cycle exercise. Pivarnik and colleagues (32) measured eight subjects' rectal temperature responses to arm-crank and cycle exercise at the same metabolic rate in both a 22°C and 33°C environment. They found that the rectal temperature responses were the same for both exercise types. Similarly, Young et al. (47) found that subjects had the same rectal temperature response for both arm-crank and cycle exercise in a 38°C environment while wearing microclimate cooling over the torso. It seems clear that rectal temperature values measured at 10 cm are the same for both upper and lower body exercise.

The question remains as to why the deep (>20 cm) rectal temperature measurements are systematically lower during upper body exercise? For lower body exercise, the rectal measurement is not influenced by depth (after 5 cm), and these values are equivocal to shallower (10 cm) measurements during upper body exercise. The problem seems to be that deep rectal areas are not warmed as much during upper body exercise (28). For an average adult, the rectum and anal canal length is approximately 12-16 cm long (26); therefore, the deep (20-27 cm) measurements were obtained well into the sigmoid colon. The rectum receives its blood supply from the inferior mesenteric, as well as branches of the iliac and internal pudendal arteries. The sigmoid colon receives its blood supply only from the inferior mesenteric artery. During upper body exercise, the greater sympathetic output should cause a greater constriction of splanchnic beds and result in reduced blood supply from the mesenteric artery. In theory, this compensatory vasoregulation may reduce the supply of warm

blood to the sigmoid colon more than the rectum. Also, the mucous membrane in the rectum is thicker and more vascular than in the colon (20), therefore should receive a richer supply of warm blood during exercise. Finally, it is possible that the sigmoid colon area could be influenced more than the rectum by warmed venous blood from the legs during lower body exercise (24,28); however, we have not found any anatomical evidence to support this notion.

The preceding studies all examined core temperature responses but did not attempt to quantitate the regional differences in evaporative and dry heat exchange between upper and lower body exercise. Sawka and colleagues (38) examined the relative contribution of local evaporative, radiative and convective heat exchange between arm-crank and cycle exercise at the same metabolic rate. These experiments were conducted in an 18°C/78% rh environment, which facilitated dry heat exchange, and in a 35°C/28% rh environment, which facilitated evaporative heat loss. In both environments, esophageal temperatures were not different between exercise types. Figure 2 illustrates the torso net radiative energy flux values during exercise in the two environments. In each environment, these values increased over time for both exercise types, with arm-crank exercise eliciting greater values than cycle exercise. Although this was a new finding, it was not unexpected. During upper body exercise, the muscles of the back torso areas (i.e., latissimus dorsi, trapezius, infraspinatus) are employed to a greater extent than during lower body exercise. Therefore, these skeletal muscle groups would release a greater amount of metabolic heat that would be conducted directly through the surrounding tissues to the overlying skin (46).

FIGURE 2 ABOUT HERE

These investigators found that torso and arm evaporative heat loss as well as arm dry heat exchange were not different between exercise types in each environment

(38). Leg dry heat loss was greater during cycle than arm-crank exercise in the 18°C environment; likewise, leg evaporative heat loss was greater during cycle than arm-crank exercise in the 35°C environment. These data indicate that to compensate for greater torso dry heat loss during upper body exercise, lower body exercise elicits additional dry or evaporative heat loss from the legs. The avenue for this compensatory heat loss depends upon the differential heat transfer coefficients which influence tissue conductivity and mass transfer.

Sawka and colleagues (38) attempted to determine if exercise type altered the control of thermoregulatory sweating. They found that the sweating threshold and slope were not significantly different between arm-crank and cycle exercise. Therefore, local sweating rate (back) appears to be independent of the skeletal muscle mass employed but wholly dependent on the thermal drive. Previously, Tam et al. (41) suggested that arm crank exercise might elicit a non-thermal drive to sweating through increased sympathetic activation. Their experiments, however, were performed on only two subjects (one was a spinal subject) and only during arm-crank exercise.

The previous studies also indicate that differences in the surface area-to-mass ratio between the exercising arms and legs have nominal thermoregulatory effects in air. Water, however, has a heat conduction approximately 25 times greater than still air. It seems that during cool water immersion, exercise performed with the arms (relatively large surface area-to-mass ratio) and with the legs (relatively small surface area-to-mass ratio) would be expected to have different heat exchanges. Toner et al. (44) examined the thermal responses of subjects performing 45 minutes of arm-crank or cycle exercise while immersed in stirred water at 20, 26 and 33°C. Metabolic rate was not different between exercise types at each water temperature. Rectal temperature values (10 cm) were lower for arm-crank than cycle exercise. These lower core temperature values were supported by both mean weighted skin temperature and mean weighted heat flow values, which were greater during arm-crank

than cycle exercise at each water temperature. These data indicate that individuals are at a thermoregulatory disadvantage (for hypothermia) when performing upper body exercise in environments with a high convective heat transfer coefficient.

SPINAL CORD INJURED

A spinal cord injury will impair the control of man's cardiovascular system (23) and his ability to thermoregulate (9). The magnitude of impairment is related to the level and completeness of the lesion. The higher the spinal injury the greater the thermoregulatory impairment (30). Normell (30) has defined the areas with loss of cutaneous vasomotor and sweating function for a given lesion level. The consequences of the loss of sympathetic control for heat loss (via vasomotor and sudomotor adjustments) over large areas of skin is the inability to regulate body temperature during exposure to environmental extremes. Figure 3 presents a group of able-bodied and disabled subjects' core (rectal) temperature responses during passive exposure to a range (15 to 40^{o}C) of ambient temperatures. The disabled subjects had suffered spinal core transections (C_7 to S_1) during the preceding 6 to 8 years. As expected, the able-bodied subjects could regulate their core temperature throughout the range of ambient temperatures. The spinal subjects, however, had higher core temperatures in the heat and lower core temperatures in the cold. This association between core temperature and ambient temperature in spinal cord injured individuals has resulted in them being referred to as "partial poikilotherms" (3).

FIGURE 3 ABOUT HERE

Several investigators have performed detailed analyses of spinal subjects' thermoregulatory sweating and cutaneous blood flow responses to passive heat exposure (14,21,33,42). During heat exposure, some sweating can occur over the insensate skin (33), but it is sparse (33) and not synchronous with sensate skin sweating (21). Individuals with spinal injuries will have a reduced sweating response for a given core temperature (Figure 4A) during passive heat exposure (21,42). In addition, Tam and colleagues (42) have reported that sweating would not occur without a rise in core temperature in their spinal (T_6) subject. Local skin heating will elicit a sweating response in able-bodied subjects; but remember from a previous section that skin temperature only provides ~10% of the drive for thermoregulatory effector responses. Similar to the sweating response, cutaneous (forearm) blood flow is lower (than for able-bodied) for a given core temperature (Figure 4B) during passive heat exposure (14,42). Freund and colleagues (14) found that when insensate skin was heated there was no increase in forearm blood flow (FBF), but the heating of sensate skin increased forearm blood flow (but by a relatively small amount).

FIGURE 4 ABOUT HERE

From the preceding paragraphs, it can be concluded that spinal cord injured individuals are at a thermoregulatory disadvantage during heat exposure because of: (a) loss of vasomotor and sudomotor control over areas of the skin; and (b) a reduced thermoregulatory effector response for a given core temperature. Several investigators (14,42) have suggested that the latter observation may be indicative of reduced afferent input into the hypothalamic thermoregulatory centers resulting in a reduced effector drive for thermoregulatory responses. Earlier we described a linear additive model for human temperature regulation in which skin temperature (10%) and

core temperature (90%) provided the afferent inputs to elicit a given thermoregulatory effector response. Freund and colleagues (14) have argued that the magnitude of reduced thermoregulatory response ($\Delta FBF:\Delta T_{es}$) suggests that a reduced central afferent input may be present. These central thermal receptors could be located in isolated (below lesion) areas of the spinal cord and paralyzed muscles.

The spinal cord injured individual's thermoregulatory responses to exercise have not been thoroughly studied, and Table 3 provides a summary of those investigations. Surprisingly, only one investigation has compared the spinal cord injured thermoregulatory responses to a control group of able-bodied subjects (12). Fitzgerald and colleagues (12) compared the thermoregulatory responses of spinal cord injured (T_9-L_4) and able-bodied individuals to 90 minutes of wheelchair ergometer exercise (50% $\dot{V}O_2$ max) in a moderate environment. They found that the spinal subjects had a greater increase in oral (~0.5°C) and mean skin (~1.2°C) temperature than the control group (12, personnel communication). In addition, after the initial increased (from rest to exercise) cardiac output in the spinal subjects' cardiac outputs decreased by 14% during the exercise bout, whereas the control group values remained constant. These data indicate that the able-bodied subjects were able to get considerable evaporative cooling, as evidenced by the decreasing skin temperature values. The spinal cord injured subjects, however, had to rely on dry heat exchange as evidenced by the high skin temperatures. In order to get sufficient dry heat exchange for thermal equilibrium, more blood had to be shunted to the skin to increase skin temperature to achieve a better (T_{sk}-T_a) gradient. The greater amount of blood displaced to the skin (increased cutaneous flow and volume) resulted in greater circulatory strain as evidenced by elevated heart rates. The combination of more blood displaced to the skin and perhaps reduced skeletal muscle pump activity from the legs (due to paralysis) resulted in a less facilitated venous return and the reduction in cardiac output during exercise for the spinal subjects.

TABLE 3 ABOUT HERE

Gass and colleagues (15,16) performed two investigations on spinal cord injured individuals' responses to prolonged (80 min) wheelchair exercise. Essentially, they found that core (rectal) temperature and insensate skin temperature increased, and sensate skin temperature remained constant or decreased during prolonged exercise. Those investigators, however, suspected that the rectal temperature values may have underestimated the thermal strain experienced by these disabled populations. Gass and colleagues tested this hypothesis by simultaneously measuring the rectal (12 cm) and esophageal temperatures of spinal subjects during prolonged (35 - 45 min) wheelchair exercise (16). They found a greater increase in esophageal (1.4°C) than rectal (0.7°C) temperature during exercise; this finding was surprising since steady-state esophageal values are generally lower (~0.3°C) than rectal values for able-bodied subjects (40). Gass and colleagues (16) concluded that rectal temperature values may underestimate the thermal strain on disabled individuals during wheelchair exercise.

The question arises as to why the spinal cord injured subjects had a greater rise in esophageal than rectal temperature during exercise. Our analysis of their data indicates that the magnitude of disparity between esophageal and rectal temperature was not related to the lesion level. The reduced rectal temperature values are probably the result of nominal venous return from the paralyzed legs and the subjects' posture in their track wheelchairs. The lower body paralysis means that the pelvis and leg skeletal muscles' metabolic rate would not increase above basal levels during the exercise bout. This would result in no local heating and nominal venous return from the legs through the pelvic regions. In these experiments, the subjects used track wheelchairs which required a full flexion of the hips and knees, this posture

might also impede venous return from the legs to the pelvic region. Regardless of the anatomical/physiological mechanism(s), this paper raises the important issue of what is the most valid measurement of core temperature in the exercising paraplegic.

It seems that more research needs to be focused on the spinal cord injured individual's thermoregulatory responses to exercise. Future studies need to delineate the avenues of heat exchange for individuals with different lesion levels. In addition, technologies need to be developed that might help the disabled to thermoregulate during exposure to environmental heat stress and/or stressful exercise. One such technology might be the use of *micro*climate cooling vests that would facilitate torso heat loss (47). Young et al. (47) have recently demonstrated the efficacy of this technology for upper body exercise.

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FIGURE LEGENDS

Figure 1 - Relationship of steady-state esophageal temperature responses to arm-crank and cycle exercise at a given metabolic rate (redrawn from reference 28).

Figure 2 - Torso radiative energy flux values, measured by net radiometer, during submaximal arm-crank and cycle exercise. *$P<0.05$, $P<0.01$ (from reference 38).

Figure 3 - Spinal cord injured and able-bodied subjects' core temperature responses for passive exposure to a range of ambient temperatures (redrawn from reference 3).

Figure 4 - Spinal cord injured and able-bodied subjects' sweating rate (42, top) and forearm blood flow (14, bottom) responses for passive heat exposure.

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TABLE 1. Factors possibly resulting in different thermoregulatory responses for upper body than lower body exercise.

Exercise Factors
Relative intensity
Heat production from different muscles
Surface area-to-mass ratio of active limbs
Neural Factors
Local thermal information
Thermoregulatory set-point
Sympathetic output
Cardiovascular Factors
Skeletal muscle pump facilitation of venous return
Cardiac afterload
Plasma volume

TABLE 2. Comparison of thermoregulatory responses between upper and lower body exercise at a given metabolic rate

	Study	Year	n	Environ.	Ex. Mode	Core Temp.	Finding	Comment
1.	Asmussen & Nielsen	1947	2	20-20°C db	AC vs CY	Rectal (20 cm)	↓ T_{re}	AC not steady-state
2.	Nielsen	1968	2	27°C db 25-48% rh	AC vs CY	Rectal (12-27 cm) Esophageal	↓ T_{re} = T_{es} = $\dot{M}_{sw}$ = K_{sk}	
3.	Davies et al.	1971	2	19°C db 75% rh	AC vs CY & TM	Tympanic	= T_{ty} = $\dot{M}_{sw}$ = K_{sk}	
4.	Sawka et al.	1984	9	24°C db 20% rh	AC vs CY	Rectal (10 cm)	= T_{re} = $\dot{M}_{sw}$	T_{re} = T_{es} for AC
5.	Sawka et al.	1984	4	18 & 35°C db 78 & 28% rh	AC vs CY	Esophageal	= T_{es}	Avenues of Heat Loss Differ
6.	Young et al.	1987	6	38°C db 30% rh	AC vs CY	Rectal (10 cm)	= T_{re}	Microclimate Cooling
7.	Pivarnik et al.	1988	8	22 & 33°C db 75 & 57% rh	AC vs CY	Rectal (10 cm)	= T_{re}	

db is dry bulb temperature; rh is relative humidity; AC is arm-crank; CY is cycle; T_{re}, T_{es} and T_{ty} are rectal, esophageal and tympanic temperatures, respectively; $\dot{M}_{sw}$ is total body sweating rate; K_{sk} is thermal conductance; ↓ is lower than CY; = is equal to cycle

TABLE 3 Spinal cord injured subjects' thermoregulatory responses to upper body exercise.

Study	year	n	Environ.	Ex. Mode	Bout	Findings
1. Fitzgerald et al	1982	5 spinal (T_9-L_4) 5 able-bodied	24°C db 44% rh	Wheelchair Ergometer	90 min @ 0.7 l·min⁻¹	↑ T_o = $\bar{T}_{sk}$ } for Spinal than Able-Bodied
2. Gass & Camp	1984	4 spinal (T_7-T_{12})	24°C db 60 %rh	Wheelchair	80 min @ 1.74 l·min⁻¹	↑ T_{re} (0.6°C) ↑ T_{sk}(calf) = T_{sk} (chest & arm)
3. Gass et al	1988	6 spinal (T_5-L_3)	22°C db 60%rh	Wheelchair	80 min @ 1.75 l·min⁻¹	↑ T_{re} (0.7°C) ↓ T_{sk}(arm) = T_{sk} (thigh & calf)
		5 spinal (T_5-T_{11})	22°C db 60 %rh	Wheelchair	35-45 min @ ? l·min⁻¹	↑ T_{re} (0.7°C) ↑ T_{es} (1.4°C)

db = dry bulb temperature; rh = relative humidity; T_o, T_{re} and T_{es} are oral, rectal and esophageal temperatures, respectively; $\bar{T}_{sk}$ is mean skin temperature; T_{sk} is skin temperature

Fig. 1.

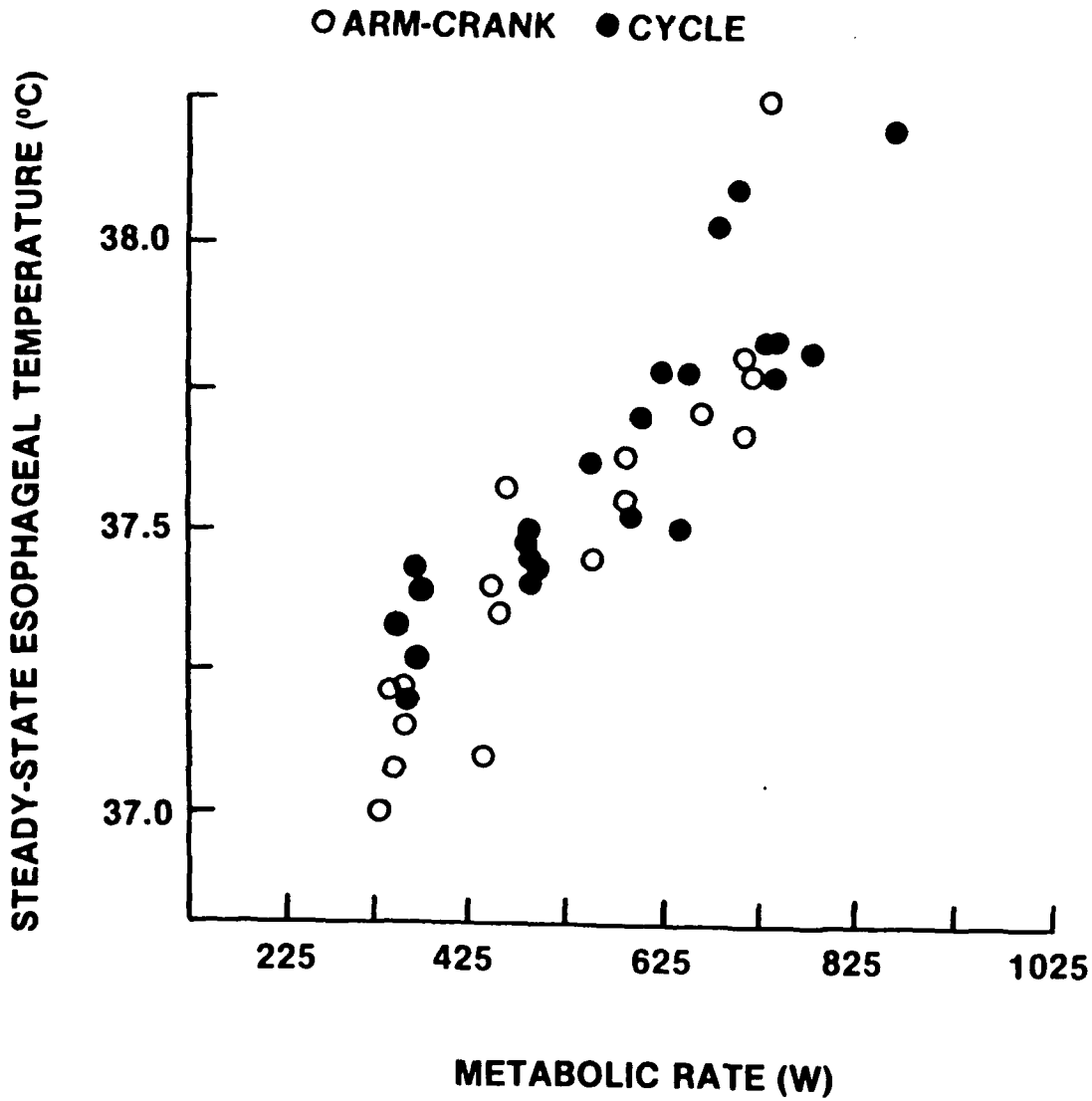
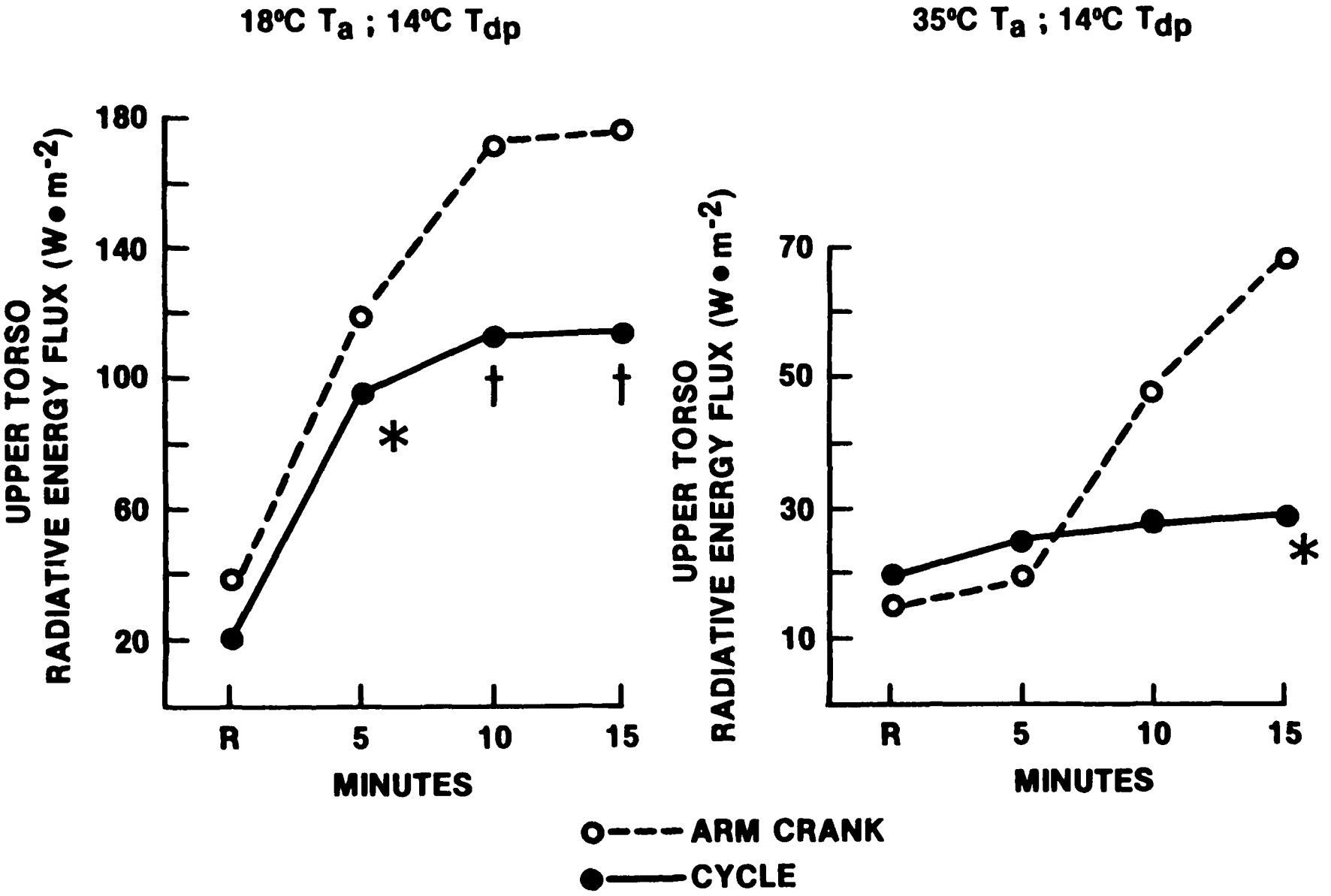
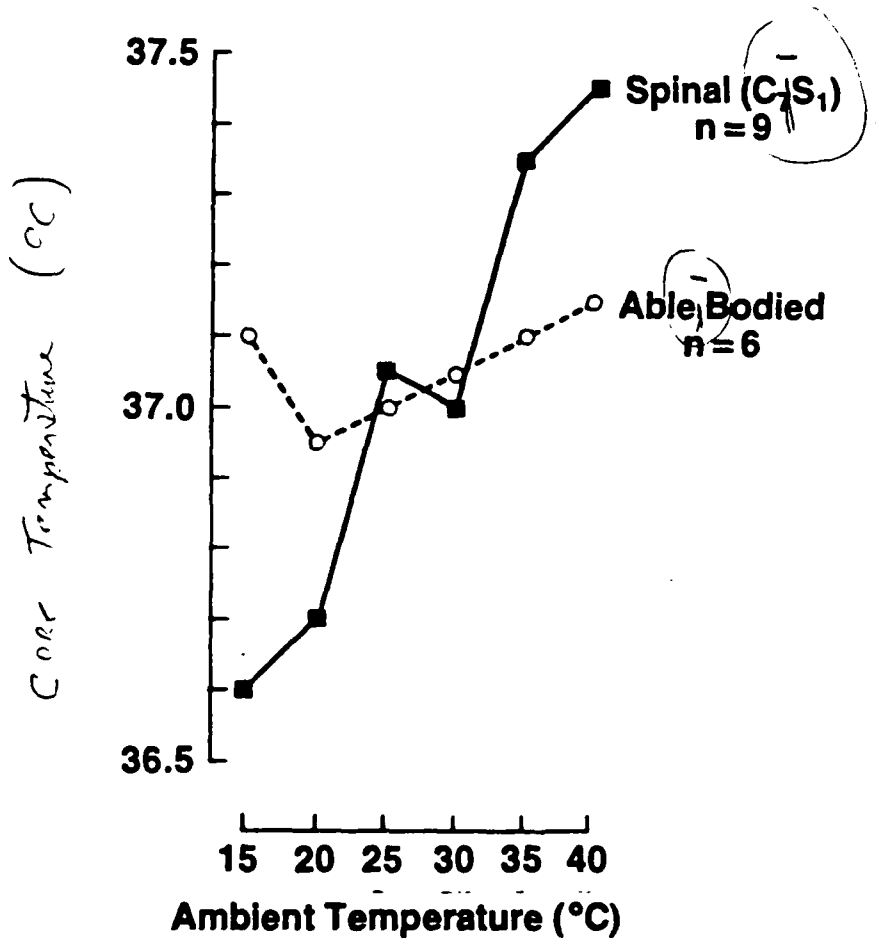

○ ARM-CRANK ● CYCLE
STEADY-STATE ESOPHAGEAL TEMPERATURE (°C)
38.0
37.5
37.0
225
425
625
825
1025
METABOLIC RATE (W)

Fig. 2

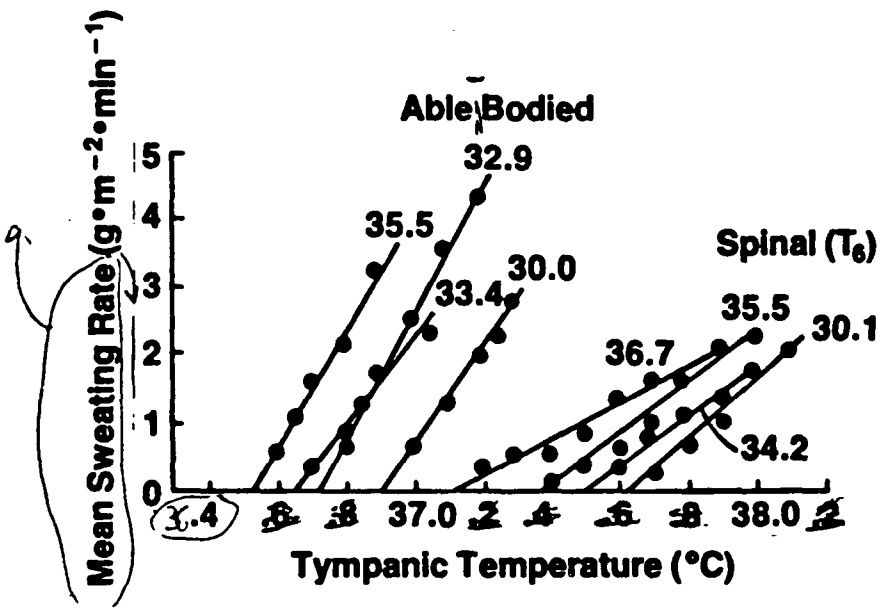

18°C T_a ; 14°C T_{dp}
35°C T_a ; 14°C T_{dp}
UPPER TORSO
RADIATIVE ENERGY FLUX (W • m^{-2})
180
140
100
60
20
70
50
30
10
*
†
†
*
R
5
10
15
MINUTES
ARM CRANK
CYCLE



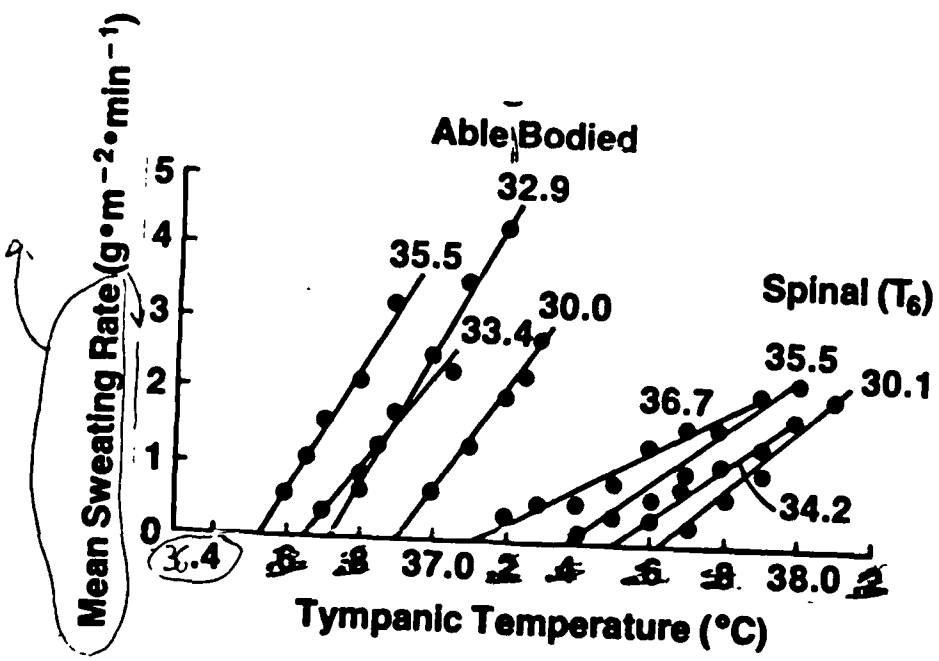
(Attia & Engel Thermal Physiol. 9:79-82, 1984)

Fig. 4

A.


Able Bodied
32.9
35.5
30.0
33.4
Spinal (T_6)
35.5
30.1
36.7
34.2
Mean Sweating Rate ($g \cdot m^{-2} \cdot min^{-1}$)
0
1
2
3
4
5
37.0
38.0
Tympanic Temperature (°C)

B.


Able Bodied
32.9
35.5
30.0
33.4
Spinal (T_6)
35.5
30.1
36.7
34.2
Mean Sweating Rate ($g \cdot m^{-2} \cdot min^{-1}$)
0
1
2
3
4
5
37.0
38.0
Tympanic Temperature (°C)