REGIONAL CUTANEOUS THERMOSENSITIVITY

by

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ABSTRACT

Thermoreceptors in the skin have been assigned a major role in autonomic thermoregulation in humans, but their population density over the body surface is not known. If thermoreceptors are distributed evenly, and if their sensitivity is equivalent, then a given thermal stimulus will elicit a similar response from all skin regions. To test this hypothesis, differential sensitivity to cooling was assessed in males by separately immersing four discrete skin regions in cold water (15°C) during head-out immersion. The response measured was gasping at the onset of immersion; the gasp response appears to be the result of a neurogenic drive from cutaneous cold receptors. Subjects, of similar body proportions, donned a neoprene "dry" suit modified to allow exposure to the water of either the arms, upper torso, lower torso, or legs, with average surface areas of 1910 cm^2, 3594 cm^2, 2358 cm^2, and 5294 cm^2, respectively. Each subject was immersed to the sternal notch in all four conditions of partial exposure, plus one condition of whole body exposure wearing only a bathing suit (average surface area of 15,296 cm^2). The five cold water conditions were matched by control immersions in lukewarm (34°C) water, and trials were randomized. The magnitude of the gasp response was determined by mouth occlusion pressure (P0.1), an indicator of respiratory drive. For each subject, P0.1 values for the first minute of immersion were integrated and control trial values, though minimal, were subtracted from their cold water counterpart to account for any gasping due to the experimental design.

Results were averaged and showed the highest P0.1 values were elicited from whole body exposure, followed in descending order by the upper torso, legs, lower torso, and arms exposures. Thus, the gasp response is not saturated during partial exposure. The addition of the 4 partial exposure responses gave a value that was similar to the whole body

exposure response, suggesting that regional thermoafferent signals interact in an additive manner.

Correcting the P0.1 response for differences in exposed surface area (SA) between regions, and comparing partial exposure conditions, showed the upper torso to have a P0.1/SA value that was significantly higher ($p \leq 0.05$) than the 3 other regions. A further correction for differences in the cooling stimulus (ΔT), gave a thermosensitivity index ($P0.1/(SA \cdot \Delta T)$) for each region, and showed that the upper torso index continued to be significantly higher than the indices for the arms or legs, but not significantly higher than the lower torso index. There was no significant difference between the thermosensitivity indices of the arms, legs, or lower torso. In general, the results suggested an increased cold receptor density, or sensitivity, in the upper torso compared with the extremities.

DEDICATION

- to Women in the Sciences

and to my father, to whom this means so much

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GLOSSARY OF TERMS

P0.1 : the mouth occlusion pressure developed 100 msec after the onset of inspiration, (Pa).

ΔP0.1 : P0.1 immersion value minus average resting P0.1, (Pa).

$\int\Delta$P0.1 : the integrated ΔP0.1 response for the first minute of immersion, (Pa·sec).

$\int\Delta P0.1_{c\text{-}w}$: the integrated one minute ΔP0.1 response to cold water immersion minus the integrated one minute ΔP0.1 response to the matching lukewarm water immersion, (Pa·sec).

$\int\Delta P0.1_{c\text{-}w}/SA$: the $\int\Delta P0.1_{c\text{-}w}$ divided by the exposed surface area, for a given region and subject, (Pa·sec·cm^{-2}).

ΔT : the skin temperature one minute post- immersion minus the pre-immersion temperature, (°C).

$\Delta\bar{T}$: the average of all ΔT for an exposed skin region, (°C).

$\int\Delta P0.1_{c\text{-}w}/\Delta\bar{T}$: the $\int\Delta P0.1_{c\text{-}w}$ multiplied by $\Delta\bar{T}^{-1}$, for a given region and subject, (Pa·sec·°C^{-1}).

$\int\Delta P0.1_{c\text{-}w}/(SA\cdot\Delta\bar{T})$: the $\int\Delta P0.1_{c\text{-}w}/SA$ multiplied by $\Delta\bar{T}^{-1}$, for a given region and subject, (Pa·sec·cm^{-2}·°C^{-1}).

TSI : thermosensitivity index for a given region, equivalent to the average value for the group of $\int \Delta P0.1_{c-w} / (SA \cdot \Delta \bar{T})$, $(Pa \cdot sec \cdot cm^{-2} \cdot {}^{\circ}C^{-1})$.

INTRODUCTION

Temperature receptors in the skin detect the status of the ambient thermal environment and transmit this information to the central nervous system, where it influences behavioral and autonomic control of thermal homeostasis. The magnitude of a selected response, to a change in skin temperature alone, is a measure of cutaneous thermosensitivity. In humans, the response (for example, sweating rate) elicited by a given thermal stimulus to one skin region, has been compared with the response from stimulation of another discrete skin region, and the results suggest that cutaneous thermosensitivity differs over the body surface (Crawshaw et al., 1975).

Sudden cold water immersion elicits a variety of physiological responses: ventilation, heart rate, blood pressure, and cardiac output, all increase. The dependency of one of these variables, the respiratory response, on the rate of skin temperature decline, has been established (Mekjavic et al., 1987). Furthermore, the hyperpnea at the onset of immersion, termed gasping, (Cooper et al.,1976) appears to be the result of a neurogenic drive from cutaneous thermoreceptors (Mekjavic et al., 1987). In this thesis, the sensitivity of four separate skin regions to cooling has been compared, using gasping as an indicator of cutaneous thermosensitivity. The magnitude of the gasp has been measured by mouth occlusion pressure (P0.1), an indicator of respiratory drive, in agreement with the methodology of Mekjavic et al. (1987).

Gasping response

The drive to increase respiration, at the onset of sudden cold water immersion, disrupts the normal control of breathing. The characteristics of this cold-associated hyperpnea have been studied under controlled experimental conditions. Results of such

studies indicate that ventilation rises approximately 450% in the first minute of head-out immersion in cold water (Martin et al., 1978; Hayward and Eckerson, 1984), and declines thereafter to near stable values within five minutes (Keatinge and Evans, 1961). The rise in ventilation occurs primarily by an increase in tidal volume and secondarily by an increase in breathing frequency, though variation occurs between individuals (Cooper et al., 1976). The change in breathing pattern is immediate; the volume of the first breath post-immersion is significantly higher than pre-immersion control values, reaching mean ventilations of 95 L/min (Goode et al., 1975).

As the immersion water temperature declines from lukewarm to 15°C, the gasping increases in magnitude, decays more slowly, and maintains a higher final value (Keatinge and Evans, 1961). A further decline in water temperature below 15°C does not, however, evoke a further increase in ventilation in the first minute of immersion, suggesting that the response has reached maximal values at 15°C (Hayward and Eckerson, 1984). Pre-heating the skin (Martin and Cooper, 1978), or wearing clothing (Keatinge and Evans, 1961; Martin et al., 1978; Mekjavic et al., 1987) significantly reduces the percentage increase in ventilation at the onset of cold water immersion.

The sensitivity of the gasp response to the pre-immersion skin temperature and to the magnitude of the decline in skin temperature, the maximizing of the response at temperatures well above 0°C, and the rapid onset and decline of the response, are all indicative of the characteristics of cutaneous cold receptors (Hensel, 1973) and thus suggest the latters' involvement in this gasping phenomenon. The reduction of the response by pre-heating of the skin, also suggests the involvement of warm receptor activity, which may act to inhibit the cold receptor excitatory drive. In addition, the gasp response occurs prior to any change in deep body temperature, therefore central thermoreceptors are not thought to be involved (Keatinge and Evans, 1961; Cooper et al., 1976).

Other possible explanations of gasping have been investigated and discarded. Keatinge et al. (1964) showered male subjects with ice-cold water and observed a similar ventilatory response as with cold water immersion, though the magnitude of change was less. Heart rate and systolic and diastolic arterial pressures increased within 2-3 sec of the start of the shower. Cardiac output also increased 59 and 100% in two subjects. The time course of the cardiovascular changes, concomitant with no significant change in the plasma level of either norepinephrine or epinephrine, suggested that the responses were not hormonally based, but due to reflex sympathetic stimulation (Keatinge et al., 1964) and independent of the hyperpnea (Keatinge and McCance,1957).

Concurrent with the increased ventilation in the first minute of an ice-cold shower, the arterial PCO_2 falls by 30% (Keatinge et al., 1964), and the arterial PO_2 rises by 22% (Keatinge and Nadel, 1965). Hayward and Eckerson (1984) report an increase in the respiratory exchange ratio from 0.8 to 1.4 in the first minute of cold water immersion in resting subjects, indicating that the increased ventilation is greater than the metabolic demand, and thus is appropriately termed hyperventilation. Keatinge and Nadel (1965) conclude that the changes in blood gas tensions are the result, and not the cause, of the increased ventilation. Furthermore, the gasp response is not associated with any change in lung mechanics (Keatinge and Nadel, 1965) or central chemoreceptor sensitivity (Cooper et al., 1976).

Since the gasp response has been implicated as a primary cause in cold water immersion drownings, many investigations have been conducted to elucidate the mechanism of the response and to identify the factors which modify it. As a result, the following factors have been reported to influence the gasp response:

(1) Behavioural. Cooper et al. (1976) observed an exaggerated hyperventilation in open, choppy water, compared to the laboratory simulations, indicating that emotional factors heighten the response. Conversely, practising techniques which

control the breathing pattern reduces the magnitude of gasping, but cannot obscure it (Goode et al., 1975). Further evidence that gasping is not purely a psychological phenomenon comes from the work of Keatinge and Nadel (1965), they report that when subjects were asked to hold their breath or to breathe shallowly at the onset of a cold shower, they were unable to do so. In addition, gasping can be elicited from high decerebrate and hypothalamic cats, thus the response appears to be mediated at the level of the midbrain, not the cerebrum (Keatinge and Nadel, 1965).

(2) Exercise. Submaximal exercising masks the gasp response, because ventilation increases immediately with the onset of both exercise and immersion, and does not decline as long as exercise continues (Keatinge and Evans 1961). However, the ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$) increases as the water temperature declines during submaximal, but not maximal, exercise, suggesting that the gasp response is still present with the former condition (Cooper et al., 1976).

(3) Hydrostatic Pressure. Ventilation is also known to increase transiently at the onset of immersion in lukewarm water, however the magnitude of change is considerably less than that seen in cold water (Keatinge and Evans, 1961; Goode et al., 1975; Mekjavic and Bligh, 1989). Mekjavic and Bligh (1989) suggest that stimulation of cutaneous pressure sensors and hydrostatic force, per se, contribute to the elevation of ventilation during immersion, independently of the water temperature. Thus, the reduced ventilatory response at the onset of a cold shower, compared with cold water immersion (Keatinge et al., 1964), may be attributed to the lack of hydrostatic pressure.

(4) Acclimation. Daily repetition of cold water immersion significantly reduces the increase in ventilation at the onset of immersion (Mittleman and Mekjavic, 1987). Keatinge and Evans (1961) likened the reduced respiratory response to the cardiovascular adaptations seen with repeated immersions of the hand in ice-water

(Glaser et al.,1959). The reduction has been attributed to an adaptation in the central, not the peripheral, nervous system.

Thus, while hyperventilation at the onset of cold water immersion is influenced by factors not uniquely associated with a change in skin temperature, most investigators conclude that the gasp response is primarily the result of a massive afferent drive from peripheral cold receptors (Keatinge and Evans, 1961; Keatinge and Nadel, 1965; Cooper et al., 1976; Mekjavic and Bligh, 1989).

Mouth Occlusion Pressure

Ventilation is a measure of the final outcome of the respiratory system, which includes both neural and muscular components. Breathing is initiated and controlled by areas in the pons and medulla, referred to as the respiratory centers, but due to the complexity of the central nervous system (CNS) assessment of the output of the respiratory center is first made, not in the CNS, but in the alpha motoneurons exiting the spinal cord. An increase in the rate of rise of inspiratory alpha motoneuron activity, termed central inspiratory activity or respiratory drive, directly increases contractile activity in the respiratory muscles (Whitelaw et al, 1975). As a result, inspiratory flow rate is increased, which is the primary cause of an increase in ventilation. Tidal volume and breathing frequency are secondarily modified by reflexes, such as the Hering-Breuer inflation reflex (Milic-Emili et al, 1981).

Because ventilation may be altered by a change in the muscular component of the respiratory system, independently of a change in the neural component (Grunstein et al., 1973), it is an inadequate measure of the output of the respiratory center alone. One practical, non-invasive measure of central inspiratory activity, that has gained wide

acceptance in recent years, is mouth occlusion pressure (Lind,1984). Grunstein et al. (1973) introduced the technique of measuring the pressure generated at the airway opening, when the muscles initiate inspiration from a position of functional residual capacity against an occluded airway. The main advantage of the occlusion technique is the removal of mechanical attributes of the respiratory system from the measurement (Whitelaw et al., 1975). Gas does not flow during occlusion and lung volume does not change appreciably, therefore the effect of resistance and compliance are negated. The pressure measurement at relaxed functional residual capacity has no contribution from elastic recoil of the lung and chest wall, so it reflects the net pressure developed by the respiratory muscles. Furthermore, the contraction against an occluded airway is close to isometric, thus for a given pre-load, force-length and force-velocity characteristics of the respiratory muscles have no significant effect.

Two measures of respiratory center output show a linear relationship with mouth (or airway) occlusion pressure: phrenic nerve activity (Eldridge, 1975), and diaphragm muscle activity (Lopata et al, 1975). By simultaneously recording electroneurograms from the phrenic nerve and electromyograms from the diaphragm, in anaethetized cats, Grunstein et al. (1973) demonstrated that the activities in the phrenic nerve and diaphragm are the same during an occluded breath as during a prior unobstructed breath. However, this equality is not maintained with conscious humans, who struggle against an occluded airway. Nevertheless, Whitelaw et al. (1975) demonstrated that the reaction time of subjects delays distortion of the inspiratory occlusion pressure wave for a minimum of 150 msec. Prior to this time, the pressure rises uniformly and is reproducible. Therefore, Whitelaw et al. (1975) suggested that a useful index of the respiratory center output could be obtained by measuring the mouth occlusion pressure generated 0.1 sec after the onset of inspiration (P0.1). Subsequent tests have supported the use of P0.1. Breath-by-breath monitoring of exercising subjects with and without P0.1 measurement, show no influence

of the occlusion procedure on ventilation or end-tidal PCO_2 (Ward et al., 1981). In addition, comparisons between P0.1 and ventilation as measures of respiratory response to an increasing workload, confirm that P0.1 is the better indicator of respiratory drive (Lind, 1984).

Mekjavic et al. (1987) measured changes in respiratory drive during sudden cold water immersion, by the method of mouth occlusion pressure. The same pattern of inspiratory response is seen using mouth occlusion pressure, as was previously reported with ventilatory indices. Also, a comparison between concurrent measurement of ventilation and P0.1 supports prior findings (Lind, 1984), that the two variables diverge from a linear relationship with each other at higher values. Most importantly, Mekjavic et al. (1987) demonstrated a high correlation between the rate of change of mean skin temperature ($d\bar{T}s/dt$) and P0.1 in the first minute of immersion. Since the gasp response occurs before any significant decline in core temperature (Mekjavic et al., 1987; Tipton and Golden, 1987) the high correlation between $d\bar{T}s/dt$ and P0.1 supports earlier conclusions that the response is the result of a neurogenic drive from cutaneous cold receptors. The correlation further suggests that gasping magnitude is an indicator of sensitivity to peripheral cold stimulation.

Regional Cutaneous Thermosensitivity

Thermoreceptors are sensory neurons responding to steady-state and transient temperatures. Criteria for their classification has been established (Hensel, 1973), and their location and responsiveness investigated in numerous species. The most peripheral thermoreceptors lie in the dermal and epidermal layers of the skin as free nerve endings (Spray, 1986). Two distinct populations exist: cold receptors, which are active between skin temperatures ranging from approximately 10 to 40°C, and warm receptors, active in a

skin temperature range from approximately 30 to 45°C. The temperature range of static activity for both receptor types, however, varies considerably for different species and for different skin regions within a single species (Iggo, 1969). (Extremely low or high skin temperatures also stimulate pain receptors). Cutaneous warm and cold receptors have been found in diverse regions, but whether they are evenly distributed over the body surface remains unresolved.

Our knowledge of human cutaneous thermal sensibility has been acquired mainly by psychophysiological studies on temperature sensation, electrophysiological findings on individual thermosensitive fibers, and autonomic responses to cutaneous thermal stimulation. Using these techniques, differences in thermosensitivity between discrete skin regions have been demonstrated. For example, the results of three psychological experiments agree that per unit area the forehead has a much greater thermosensitivity than the back (Hardy and Oppel, 1937; Kenshalo et al., 1967; Stevens and Marks, 1971). While few electrophysiological recordings have been made from thermosensitive fibers in humans (Hensel and Bowman, 1960), the variation in maximal static firing rates in different thermoreceptor populations on the body surface of mammals suggests a specialization of thermal sensitivity, which may correspond to the normal temperature of that part (Iggo, 1969). Several studies, using different species, have graded the thermoregulatory effect in response to a stimulus to separate skin areas, in the absence of any significant change in core temperature (Necker, 1981). The differences found in the thermosensitivity between skin regions implies differences in the density of thermal sensors, or an uneven weighting of afferent activity by central signal processing (Nadel, 1977).

Several studies have observed a strong thermoregulatory effect in response to a temperature stimulus to the inguinal region in animals of both sexes. Heating the skin of the scrotum in rams (Waites, 1962; Hales and Hutchison, 1971) and pigs (Ingram and Legge, 1972), and the udder in goats (Linzell and Bligh, 1961) evokes vigorous panting,

equivalent to that due to thermal stimulation of the spinal cord or hypothalamus (Ingram and Legge, 1972). Heating an equivalent area of the flank skin in the ram increases the respiratory rate only a little, suggesting that flank skin is less sensitive to heating than scrotal skin (Waites, 1962).

In cooled pigeons, continuous shivering is differentially decreased by heating discrete skin regions, without altering the low body temperature. Results show that in feathered skin of equivalent area, the back is the most sensitive, the wing less so, and the breast the least sensitive (Necker, 1977). Heating the beak has little influence on shivering, while heating or cooling naked parts of the feet has no effect at all. Conversely, a similarity in thermosensitivity of two separate skin areas was reported by Kluger et al. (1972). The decline of metabolic rate, in response to heating either the skin of the ears or the back in rabbits, was similar when metabolic rate was expressed as a change per °C per unit area.

Differences between the skin tissues in humans and various other animal species (furred, feathered, hairy, and glabrous) restrict the application of findings in other animal species, to humans. Two studies have investigated the differential thermal sensitivity of human skin by comparing the sweating response to regional heating (Nadel et al., 1973), and cooling (Crawshaw et al., 1975), independently of any change in deep body temperature. In the warm environment used in these two studies, further skin heating theoretically stimulates only warm receptors (Spray, 1986), while the cooling of warmed skin primarily stimulates cold receptors (Hensel and Bowman, 1960). In addition, there may be a dynamic decline in cold receptor activity during heating, similarly cooling may reduce warm receptor activity (Hensel, 1973). Because the skin temperature change in these two studies partially falls within the area of overlapping activity of warm and cold receptors, it is impossible to attribute the response to the activity of only one receptor type (Necker, 1981). In both studies, sweating, measured at the thigh, was considered representative of a systemic response, and thermal stimulation of a small area was

considered equivalent to thermal stimulation of the whole region.While the area of stimulation varied between regions, Crawshaw et al. (1975) demonstrated a linear relationship in the abdominal region between decreases in thigh sweating rate (SR) and size of the cooled area. Another discrepancy, namely differences in the magnitude of change in the temperature of tested areas (Tsk stim), was accounted for by assuming a linear relationship between thigh sweating rate and skin temperature. Thus, sensitivity coefficients (S) for each region (i) were expressed as the change in thigh SR per unit area per °C. Both studies showed that the face was approximately three times more sensitive than any region on the torso. Nadel et al. (1973) compared the sensitivity coefficient of each region to that of the thigh (Si/Sthigh) and noted small differences in sensitivity to heating of the chest (1.2), thigh (1.0), abdomen (0.94), and upper arm (0.92), but the lower leg and lower arm had only one-half the thermal sensitivity of the thigh. Crawshaw et al. (1975) compared the sensitivity coefficient to cooling of each region, not to the thigh, but to the chest (Si/Schest). Again, the same small differences and hierarchial order in thermosensitivity were noted between the chest (1.0), thigh (0.9), and abdomen (0.8). In addition, the back sensitivity (1.2) was the highest on the torso, and the lower leg showed twice as high a sensitivity to cooling as to heating.

The finding of a high thermal sensitivity of the face by Nadel et al. (1973) and Crawshaw et al. (1975), is in agreement with the observation by Mekjavic and Eiken (1985) of an extreme sensitivity of the trigeminal region of the face to thermal stimuli. In human subjects exposed to cold air, radiant heat applied to the face significantly reduced shivering tremor, while rectal temperature was stable at normothermic values. Thus, studies of many animal species, including humans, support the concept that some skin areas have a greater thermosensitivity than others over the body surface, which implies an uneven distribution of cutaneous thermosensitive fibers and/or a weighting of

thermoafferent information by the integration center in the CNS, based on more factors than area of stimulation and magnitude of temperature change.

Gasping As A Measure Of Regional Cutaneous Thermosensitivity

Accepting the hypothesis that gasping is an autonomic response to skin cooling, the question arises whether all skin regions over the body surface contribute equally to the gasp.

Keatinge and Nadel (1965) compared the increase in pulmonary ventilation during the first 20 sec of cold showers, limited to nine skin areas. The torso was divided into six regions (three front and back) of approximately equal area.Three regions in the extremities - the arms, thighs, and lower legs, were all larger than those in the torso. The greatest responses were elicited from the three areas in the front of the torso, and the upper portion of the back. Smaller responses were initiated with showers to the middle and lower back. As well, showers to the three regions in the extremities resulted in small responses, despite their relatively larger size. Thus, in general, the torso appeared more sensitive to skin cooling than the limbs, though the only reported statistical test compared each regional response to a control shower (34°C) and not to each other. Additionally, no measurements were made of skin temperature.

In contrast to the findings of Keatinge and Nadel (1965), Tipton and Golden (1987) found no significant difference between the initial increase in minute ventilation with cooling of the torso, compared with the limbs (arms and legs) during head-out immersion in 10°C water. The two regions were of slightly different surface area. According to the theory of Hensel and Zotterman (1951), the response to a change in skin temperature is partly a function of the area of stimulation, because an increased area encompasses a greater number of afferent receptive fields, which in turn evokes an increased magnitude in the

thermosensitive response. Therefore, the area of stimulation should be controlled when studying the thermosensitive response of gasping. Another discrepancy in the study by Tipton and Golden (1987) was a temperature decline in the unexposed regions of approximately 4 - 5°C, though the temperature decline in the exposed regions was of equal magnitude (19°C). In addition, subjective measurements of comfort indicated that pain receptors were stimulated in the hands, forearms, and feet during limbs exposure, but torso exposure was more comfortable. Thus, the gasp response may have been influenced by not only the skin cooling of exposed regions, but also the cooling of unexposed regions, and by cold nociception. Furthermore, Tipton and Golden (1987) did not test the subject response to a control immersion in lukewarm water. Thus, the effect of non-thermal stimuli, such as hydrostatic pressure and apprehension, were also not taken into account.

An unequivocal test of regional cutaneous themosensitivity requires that thermal stimulation of skin regions is confined to the selected areas, and does not influence other parts of the body (Necker, 1981). Therefore, to use the gasp response as an indicator of thermosensitivity, the non-exposed skin regions must remain thermoneutral throughout testing. In addition, differences in the areal extent and stimulus intensity between regions, must be accounted for. The present study fulfills these requirements, and allows a comparison of cutaneous thermosensitivity to cooling to be made between four separate regions, excluding the head and neck. In contrast to measuring the decay of a warm-induced response by skin cooling (Crawshaw et al., 1975), this study measures the development of a cold-induced response and accounts for the influence of non-thermal stimuli on the gasp response.

METHODS AND MATERIALS

Subjects

Seven male students, aged 20 to 33, volunteered their participation in the present series of immersion trials, to be immersed to the sternal notch in 15°C water in 5 separate trials testing 5 conditions, and in 34°C (skin temperature) water for a matching set of 5 control trials. Subjects were accepted on the basis of having anthropometric measurements within one standard deviation of a student population mean, as determined by the CANREF study (Ross and Marfell-Jones, 1982). Subject's physical characteristics (mean ± SD) are presented in Table 1. The experimental protocol utilized in the present study was approved by the Ethics Review Committee of Simon Fraser University. Subjects were familiarized with the procedures and possible risks of the experiment prior to the trials.

Protocol

Thermosensitivity was evaluated for regions below the level of the sternal notch; thus the head and neck remained in an air environment (26°C). The head was excluded from the present investigation, because water on the face elicits the diving response, and ventilation may be reduced (Mukhtar and Patrick, 1986); the neck was excluded for practical reasons, to avoid water entry in the equipment measuring mouth pressure. The testing of regional cutaneous thermosensitivity requires that stimulation of selected areas does not influence other parts of the body. The task of dividing the body into segments which could be separately exposed to water was achieved by using a custom-made segmental dry suit (Fitzwright-Sine Ltd., Cloverdale, B.C.). The suit consisted of six sections (two arm covers, two leg covers, one upper torso cover, and one lower torso

cover) made from 1/2 inch neoprene. Each part was designed to provide a seal at its boundary, with either the underlying skin or an adjacent section of neoprene, except at the neck, where the suit was loose-fitting. In order to ensure a seal at the edge of a suit part, a stretchable rubber tube was placed over each border area.The three rubber seals: an arm cuff, thigh cuff, and waist girdle had vertical lengths of 11 cm, 15 cm, and 30 cm, respectively, and when placed at a suit border, approximately one-third of the rubber seal directly covered the skin. By arranging the suit in various combinations, four regions could be independently exposed, as seen in Fig. 1:

1) right and left upper limb, from the midlevel of the upper arm to the distal end of the hand; for the purposes of this study this region was referred to as the "arm".

2) upper torso, from the sternal notch to the waist (at the level of the minimal girth) and from the acromion processes to the midlevel of the upper arms.

3) lower torso, from the waist to the level of the gluteal furrow.

4) right and lower limbs, from the midlevel of the thigh to the distal end of the foot; for the purposes of this study, this region was referred to as the "leg".

In addition, a fifth exposure included all areas, see Fig. 1. Note that although the exposure was referred to as "whole body" for the purposes of this study, the whole body exposure did not include the head and neck regions.

Subjects participated in ten head-out immersion trials, five in cold water (mean ± SD =14.9 ± 0.3 °C) and five in lukewarm water (34.4 ± 0.5 °C). The lukewarm immersions were conducted to account for any non-thermal effect of the experimental protocol, in particular, hydrostatic pressure and apprehension. The conditions tested at both water temperatures were:

1 - whole body exposed

2 - arms exposed

3 - upper torso exposed

4 - lower torso exposed

5 - legs exposed

For any one subject, trials commenced at the same time of day, thus reducing circadian rhythm effects. The five cold water trials were randomly administered, and spaced one week apart to avoid acclimation to the cold. However, acclimation was not expected to occur during lukewarm immersions, therefore these five tests were split into two sets of consecutive trials. Test days in lukewarm water were interspersed between those in cold water.

Subjects were asked to avoid strenuous physical activity for the four hours preceding testing, and if they wished to eat , to have only a light meal (no caffeine or alcohol) at least two hours prior to testing. One subject was a light smoker and he agreed not to smoke on the test day.

Prior to each experiment, subjects changed into a bathing suit (Speedo nylon briefs, Warnaco Ltd., Montreal, Que.), and thermocouple probes were taped to their skin. For partial exposure conditions, the appropriate neoprene suiting was donned. The height and circumference of the end-points of exposed skin regions were measured and a weight belt was positioned around the waist to reduce flotation.

After the instrumentation procedure, the subject was assisted into a mesh chair suspended from the ceiling above the immersion tank. The subjects remained seated above the immersion tank during a five minute rest period, thus allowing pre-immersion rest values to be collected. A pulley system attached to the chair suspension allowed rapid (approximately 3 sec) lowering of the subject into an immersion tank at the onset of minute six of the trial. Subjects were immersed in the water to the level of the sternal notch for a total immersion time of five minutes. Thereafter, they were removed from the tank and disconnected from the recording instrumentation.

Instrumentation

1. Mouth pressure

Mouth pressure was detected by a bi-directional differential gas pressure transducer (Model 270, Hewlett Packard), connected to an AC carrier preamplifier (Model 17403A, Hewlett Packard). The pressure signal was filtered with a 1 Khz low-pass filter (3rd order), and the resultant signal transmitted to three peripheral devices.

The main peripheral device was a breathing monitor which detected the onset of inspiration and expiration. Onset of inspiration was defined as a decrease of mouth pressure below ambient air pressure to a threshold value of -39 Pascals (equivalent to -0.4 cm. H_2O, in agreement with Lind et al., 1984) ; whereas onset of expiration was defined as an increase in mouth pressure above ambient air pressure to 39 Pascals. The breathing monitor initiated a P0.1 measurement every alternate breath by closing a pneumatically-driven mechanical shutter, via a solenoid (No. 8345E1, Asco Electric Ltd., Brantford, Ont.). The shutter was placed on the inlet side of the mouthpiece. When a measurement was initiated, the shutter closed during expiration and opened 110 msec after the onset of inspiration. Subjects wore noise protecting ear muffs (NRR=24.0 decibels, Bilsom Comfort 74-2315, Safe-Pak Supply Canada Inc., Port Moody, BC) to reduce their awareness of the shutter closure.

The pressure signal was also transmitted to an oscillographic chart recorder (Model 7404A, Hewlett Packard), which provided a continuous analog record of the mouth pressure. During shutter closure the breathing monitor increased the speed of the chart paper to 100 mm/msec, thus allowing a more accurate determination of the mouth pressure at 100 msec following onset of inspiration.

Lastly, the pressure signal was transmitted via an analogue-to-digital converter, to a computer (Apple II+). The computer was programmed to sample the mouth pressure at 100 μsec intervals during each inspiration.

2. Skin temperature

Skin temperature was measured at 19 sites by attaching 24 gauge copper-constantan (T-type) thermocouples to the skin with waterproof tape (Elastoplast, Smith and Nephew Inc., Lachine, Que.). The 19 sites included those used in four formulae for mean skin temperature: Hardy and DuBois 7-point (1938), Nadel et al. 10 point (1971), Ramanathan 4- point (1964), and Mitchell and Wyndham's modification of Hardy and DuBois' formula to a 12- point one (Mitchell and Wyndham, 1969). In addition four unique sites were selected to represent the lower torso.

With the subject standing in the anatomical position, the 19 sites (as seen in Fig. 2) were located at:

Head: 1) mid-forehead;

Upper Extremity: 2) lateral aspect of right upper arm at the midlevel, i.e, halfway between the acromion process and the olecranon process; 3) lateral aspect of left forearm at midlevel, i.e., halfway between the midcubital fossa and the distal wrist crease; 4) central point of dorsal surface of right hand; 5) central point of palmar surface of left hand.

Upper Torso: 6) right scapula (midpoint of infraspinous fossa); 7) left scapula (midpoint of supraspinous fossa); 8) right thorax, 5 cm superior to the nipple; 9) abdomen, 2 cm superior to the umbilicus; 10) lower back, at the level of the waist at a point midway between the vertebral column and the most lateral aspect of the left side.

Lower Torso: 11) midpoint of right inguinal line; 12) 1 cm superior to the left anterior superior iliac spine; 13) midpoint of right iliac crest; 14) midpoint of left buttock, i.e., halfway between the coccyx and the most lateral aspect of the hip, at the level of the maximal gluteal protuberance.

Lower Extremity: 15) midpoint of right anterior thigh, i.e., at the level halfway between the inguinal line and the superior aspect of the patella; 16) midpoint of left posterior thigh, at the same level as thermocouple position 15; 17) midpoint of left posterior calf at the level of the maximal calf girth; 18) the lateral calf, at a level halfway between the apex of the fibula and the level of the minimal ankle girth; 19) central point of dorsum of left foot.

Skin temperatures were measured on-line every 10 sec by an HP3497A Data Acquisition System (Hewlett Packard) controlled by an HP9817 computer (Hewlett Packard).

Bath temperature was measured during the pre-immersion collection of baseline values, using a YSI 701 thermistor (Yellow Springs Instrument Co., Yellow Springs, Ohio) placed at a midpoint in the tank, and connected to a digital voltmeter (Model 5000, Dana Laboratories Inc., Irvine, Ca.). The immersion tank was constructed of plywood (86 x 89 x 115 cm), encased in a steel frame, and lined with a polyvinyl sheet. The tank was filled with 750 liters of water. A Spa Support System (Swimquip, Wicar Canada Ltd., Missassauga, Ont.) continuously stirred the water throughout immersion at a maximum flow rate of 75 L/min. The water temperature was maintained either at 15°C by a portable cooling unit (Blue M Electric Co., Blue Island, Ill.), or at 34°C by the Spa Support System.

3. Electrocardiogram

An electrocardiogram was obtained from three pre-gelled, disposable silver/silver chloride electrodes (Medi Trace, Graphic Controls Canada Ltd., Gananoque. Ont.) placed in a modified Lead I (CM5) position, and connected by an extended, shielded patient cable to an electrocardiograph (Physio-Control Systems, Seattle, Wa.). The electrodes and cable connections were protected from water by a cover of waterproof tape. The electrocardiograph was located one meter from the immersion tank and received power via a medical grade isolation transformer.

Electrocardiograms were monitored at regular intervals (in particular during entry into the water), for any irregularities.

Calibration

The pressure transducer was calibrated in the hour preceding each test. The threshold pressure for inspiration on the breathing monitor was set prior to each test using an inclined manometer (Dwyer Instruments Inc., Michigan City, Ind.) and the correct functioning of the mouth occlusion apparatus was tested manually.

Calibration of thermocouples and thermistor were conducted using a 6 liter refrigerating circulator (Lauda, Model RMT-6, Brinkmann Instruments Co., Rexdale, Ont.), which controlled a reference water bath temperature to within $\pm$ 0.1°C. Accuracy was verified with a standard reference thermometer.

Analysis

1. Calculation of exposed surface areas

For the purpose of calculating the total skin surface area exposed to the cold stimulus in each condition, the body was represented as a combination of geometrical shapes, based on the rationale that if measurements are sufficiently close together, then the limbs and torso, or portions thereof, can be represented as geometrical figures. This approach was validated by Katch et al. (1974), who used the method to determine body volume, by modelling the limbs and torso as a series of truncated cones, and the hands and feet as wedges. Modifications were made to standard geometric equations, as outlined in Appendix A, to calculate the surface area of the limbs and torso:

$$SA = H \cdot (C_1+C_2)/2 \qquad \text{..... (1)}$$

where,

H = vertical length (cm)

C_1= circumference at top of cone (cm)

C_2 = circumference at bottom of cone (cm)

Likewise, modifications were made to standard geometric equations, to calculate the surface area of the hands and feet:

$$SA(\text{wedge}) = L \cdot H + H \cdot (C/\pi) + (C/\pi) \cdot \sqrt{(H^2+L^2)} \qquad \text{..... (2)}$$

where,

L = horizontal length (cm)

H = vertical length (cm)

C = circumference (cm).

The truncated cones defined by Katch et al.(1974) did not always coincide with the exposed regions. When the uncovered skin in partial exposures did not begin and end at the boundaries of the truncated cones set by Katch et al., the surface area for portions of cones was calculated by the following equation:

$$SA(\text{cone portion}) = H_1 \cdot (C_1 + (H_1/2H_2) \cdot (C_2-C_1)) \quad \ldots\ldots\ldots (3)$$

where,

H_1 = vertical length of cone portion (cm)

H_2 = vertical length of cone proper (cm)

C_1 = circumference at top of cone proper (cm)

C_2 = circumference at bottom of cone proper (cm)

2. Calculation of P0.1

P0.1 values were determined by two methods: a computer calculation from digital mouth pressure samples, and manual derivation from analogue chart recordings of mouth pressure, as outlined in Fig. 3. A comparison between these two methods of analysis of P0.1 indicated a very good correlation ($r^2 = 0.94$). Due to the greater accuracy and resolution possible with the computer, the P0.1 values used for data analysis came primarily from the computer calculations. The chart recordings were used only as confirmation of the values determined by the computer, and also as back-up in case of computer failure.

Furthermore, the chart records were analyzed for any irregular mouth pressure responses, and these values were removed from the analysis.

For each trial, a resting P0.1 value was calculated by averaging all values between minute 2.5 and 4.0 of rest. This resting average P0.1 was taken as a baseline value, with which all immersion P0.1 values could be compared. For each trial, the baseline value was subtracted from each P0.1 immersion value, giving a ΔP0.1 value. An integrated ΔP0.1 response was derived for the first minute of immersion. The integrated one minute ΔP0.1 response, $\int\Delta$P0.1, was considered representative of the gasp response to the cold stimulation of the exposed skin surface area.

Thermosensitivity Index Determination:

1. Non-thermal stimuli

The $\int\Delta$P0.1 for lukewarm water immersions represented a response to non-thermal stimulation. To isolate the response to a drop in skin temperature alone, the lukewarm water response was subtracted from its cold water counterpart ($\int\Delta P0.1_{C-W}$), for each subject and condition.

2. Surface area

For each subject and condition, the total surface area exposed to the water was determined by adding together the surface area of the geometrical shapes representing the exposed region. To account for differences in exposed surface areas between conditions, each $\int\Delta P0.1_{C-W}$ value was divided by the exposed surface area ($\int\Delta P0.1_{C-W}/SA$) for that condition and subject, during cold water immersion.

3. Skin temperature

Initially, temperature readings were corrected according to calibration formulae. For each trial, the temperatures recorded one minute post-immersion at the 19 sites were subtracted from their last pre-immersion temperatures. The resulting ΔT values were divided into the regions they represented. Temperature sites for a given region were averaged, giving $\Delta\bar{T}_{arm}$, $\Delta\bar{T}_{upper\ torso}$, $\Delta\bar{T}_{lower\ torso}$, and $\Delta\bar{T}_{leg}$. $\Delta\bar{T}_{whole\ body}$ was the average of the regional $\Delta\bar{T}$.

For each subject and condition, the $\int\Delta P0.1_{C-W}$ response was divided by the decline in skin temperature of the exposed region ($\int\Delta P0.1_{C-W}/\Delta\bar{T}$), during the first minute of cold water immersion. Additionally, a thermosensitivity index (TSI) was calculated for each condition, which represented the variability in the gasp response between regions, after accounting for differences between conditions in exposed surface area and temperature decline. Thus, TSI equals $\int\Delta P0.1_{C-W}/(SA\cdot\Delta\bar{T})$ for any given region. The thermosensitivity index was calculated first for individual subjects, and then averaged for the group.

Statistics

Overall ANOVA for repeated measures was performed on the group data, followed by a Scheffé F-test for multiple comparisons. In addition, a Pearson product-moment correlation coefficient was used to compare $\int\Delta P0.1$ and surface area (Fig. 9).The level of significance for all tests was 95% ($p \leq 0.05$).

RESULTS

For the purpose of clarity all the pressure values in the present analysis have been expressed in absolute terms (Pascals).

The respiratory response to cold water immersion was substantially greater than the lukewarm water immersion, as illustrated in Fig.4 for a representative subject. The lack of response observed for the whole body exposure trial in lukewarm water indicates that hydrostatic force and emotional reactions to sudden immersion exerted a minimal effect. The nature of the P0.1 response for the whole body exposure in cold water is indicative of the response observed for partial exposure conditions. Specifically, following a peak response observed within seconds of immersion, the P0.1 decayed towards a pre-immersion value within the five minute immersion period. However, as depicted in Fig. 5, the magnitude and duration of the response varied between partial exposure conditions.

Several analytical approaches for the quantification of the respiratory response were reviewed, prior to accepting the procedure outlined in the Methods. Consideration for a previous finding of a strong correlation between P0.1 and dTs/dt (Mekjavic et al., 1987), necessitated that the observed immediate respiratory response to a fall in skin temperature be given full weighting. Therefore, the use of averaging techniques, which mathematically reduced the importance of the first post-immersion breath, was discarded. Similarly, representing the immediate response by a single breath, that is, the highest-valued, peak P0.1 post-immersion, was also discarded for the following reasons: 1) it was unclear which breath was the first breath post-immersion, because entry into the tank was not instantaneous; 2) P0.1 measurements were initiated every second breath, thus the first unmeasured breath might have altered the analysis, had it been measured; 3) the lack of an exact time for onset of the cold water stimulus negated the use of back extrapolation, curve-

fitting techniques to predict the peak P0.1; and 4) the change in skin temperature over the duration of a single breath could only be estimated, because the time interval between measurements of skin temperature was 10 seconds.

An improvement in the selection of a single peak P0.1 measure was considered by selecting five peak post-immersion breaths, and averaging them. However, the highest P0.1 values did not always occur as the first five breaths post-immersion. If the count began with the first highest P0.1 value, then the onset time of the respiratory response differed between trials. In addition, the duration of five breaths, and thus the stimulus duration and intensity, differed between trials.

The preferred method of analysis included the peak P0.1 values, but kept the stimulus time a constant. The chosen method integrated the area under the P0.1 curve (Figs. 4,5) for the first minute of immersion. It was observed that after one minute of immersion the P0.1 values had declined sharply from their peak, and for most partial exposure trials, they were close to their pre-immersion values. In addition, the decline in skin temperature for the 60 second interval was known. And finally, the consideration of the response during the first minute post-immersion was in agreement with previous authors (Hayward and Eckerson, 1984; Mekjavic et al., 1987; Tipton and Golden, 1987).

Tracings of mouth pressure for a single breath, showed that during occlusion, the pressure increased in a smoothly rising curve, but the shape varied depending on the magnitude of the pressure increase as illustrated in Fig. 3a (rest) and 3b (immersion). In addition, the shape of the occlusion pressure wave differed between subjects (as noted previously by Milic-Emili et al., 1981) and between trials (with the same subject). However, for any one trial waveforms were similar, therefore, irregularities were apparent and could be attributed to:

1) movement during entry into the water;

2) inadequate functioning of the rubber flaps ensuring one-way airflow

in the mouthpiece; or

3) the subject swallowing.

During the rest period, the average P0.1 values over all trials varied between individuals from a minimum of 107.0 Pa to a maximum of 443.4 Pa, with a coefficient of variation (SD/mean) of 27%. Within individuals, the difference between average resting P0.1 values on different test days, ranged for the group from a minimum of 97.5 Pa to a maximum of 194.5 Pa. Between trials, the intraindividual coefficient of variation was 20%. Within trials, the difference between the maximum and minimum P0.1 value, during rest, ranged for the group between 27.6 and 490.1 Pa. The intraindividual breath-to-breath coefficient of variation was 52%.

The magnitude of the gasping response for all immersions, in both cold and lukewarm water, are shown for each subject in Fig. 6, a-g. The response to lukewarm water immersion was of a lower magnitude than the response to the equivalent cold water immersion, for all but 3 out of the 35 comparisons. In addition, the $\int\Delta P0.1$ response to all five lukewarm conditions was similar for each individual. Therefore, subtracting the $\int\Delta P0.1$ for each lukewarm condition from the matching cold condition ($\int\Delta P0.1_{c-w}$), did not alter the observed differences in respiratory response for each cold water condition, as seen in Fig. 7, a-g.

When $\int\Delta P0.1_{c-w}$ responses for each condition were averaged for the seven subjects (see Fig. 8), the arms, lower torso, and legs exposed conditions showed a similar gasp response, but the highest of these three responses was only 18% as high as the whole body exposure response. In contrast, the response to upper torso exposure was 57% as high as the whole body exposure response, thus the upper torso response was the highest amongst the partial exposure conditions. The results indicated that the response was not saturated during partial exposure.

The exposed surface area varied between conditions as shown in Table 2. Dividing each $\int\Delta P0.1_{c-w}$ by its corresponding exposed surface area, and averaging the results, gave values of $\int\Delta P0.1_{c-w}/SA$ as shown in Fig. 9. This initial analytical step in the determination of the Thermosensitivity Index for each region, showed that the upper torso had a value significantly higher than each of the 3 other regions ($p \leq 0.05$). These three regions (the arms, lower torso, and legs) were not significantly different from each other.

The final step in the determination of the Thermosensitivity Index (TSI) accounted for variation between regions in the drop in skin temperature (see Table 3). The average decline in skin temperature in exposed skin areas ($\Delta\bar{T}$) during cold water immersions was similar between the conditions of whole body, arms, upper torso, and legs exposures. However, the decline in temperature in the exposed lower torso was less than the four other conditions.

One limitation of the segmental suit in partitioning the body was that a band of skin bordering the area between adjacent regions was never exposed in any of the partial exposure conditions. The extent of this unexposed area was equivalent to the difference between the area of whole body exposure and that of the combined regions (Table 2), and had an average value of 2,140 cm^2. Also, along the border between a suit part and exposed skin, there was a band of skin covered by a rubber seal with an approximate surface area at the arm, waist, and thigh of 112 cm^2, 764 cm^2, and 277 cm^2, respectively. The rubber seal effectively reduced water leakage to the suited regions, but did not provide sufficient insulation to prevent a drop in temperature in the underlying skin. The temperature recorded under the rubber seal was included in the calculation of $\Delta\bar{T}$ for the unexposed skin regions. As seen in Table 3 the temperature of unexposed skin during partial exposure trials did not change markedly, the average decline did not exceed 1.3°C.

The average temperature of exposed skin regions, prior to cold water immersion, was similar for all conditions; pre-immersion temperatures in Table 3 ranged from 32.2°C

to 34.0°C. Also, the average temperature of unexposed skin regions, prior to cold water immersion, was similar for all conditions; pre-immersion temperatures (Table 3) ranged from 34.8° C to 35.3° C. The difference between unexposed and exposed pre-immersion temperatures for each cold water partial exposure condition, ranged from 0.9°C to 3.0°C. The change in skin temperature ($\Delta\bar{T}$) during lukewarm water immersions was minimal in both exposed and unexposed skin regions, overall the skin temperature rose. Average values (Table 4) ranged from 0.3 to 1.6 °C in exposed regions, and from 0.1 to 0.7°C in unexposed regions.

When the $\int\Delta P0.1_{c\text{-}w}$ response was divided by the average skin temperature drop in the exposed area, $\int\Delta P0.1_{c\text{-}w}/\Delta\bar{T}$, and averaged for the group (see Fig. 10), the result showed the upper torso to have a $\int\Delta P0.1_{c\text{-}w}/\Delta\bar{T}$ value that was significantly higher than that of the arms or legs. A further correction of $\int\Delta P0.1_{c\text{-}w}$ to account for differences in both exposed surface area and temperature decline between regions, $\int\Delta P0.1_{c\text{-}w}/(SA\cdot\Delta\bar{T})$, showed that the resulting TSI for the upper torso was also significantly higher than that of the arms or legs (Fig. 11). While both the $\int\Delta P0.1_{c\text{-}w}/\Delta\bar{T}$ and TSI for the lower torso fell below the upper torso $\int\Delta P0.1_{c\text{-}w}/\Delta\bar{T}$ and TSI, there was no significant difference between the two condions with either measure. Also, there was no significant difference between either the $\int\Delta P0.1_{c\text{-}w}/\Delta\bar{T}$ or TSI of the legs, arms, or lower torso. In Fig. 12, the thermosensitivity index for each individual for the upper torso condition was ranked in order of magnitude, and this ranking order was maintained for the graphing of the individual TSI for the remaining four conditions. In addition, the conditions were positioned in descending order on the basis of their mean TSI value. The individual ranking order for the three conditions with the highest mean TSI, that is, the upper torso, whole body, and lower torso exposures, followed a pattern; one subject, PP, had the highest TSI for all three exposures and four subjects continuously ranked higher than the remaining three. There was no pattern in the individual ranking for the legs and arms exposures,

though one subject, DD, ranked either lowest or second lowest in TSI on all five conditions.

Adding the integrated P0.1 values, $\int\Delta P0.1_{C-W}$, for each subject for the four partial exposures, then averaging the values for the group, gave a mean ± S.E. of 43,000 ± 10,400 Pa·sec, which was similar to the $\int\Delta P0.1_{C-W}$ value for whole body exposure of 44,200 ± 8,700 Pa·sec. The average of the thermosensitivity indices for the four partial exposures, 0.256 ± .064 $Pa \cdot sec \cdot cm^{-2} \cdot °C^{-1}$ (mean ± SE) was also similar to the whole body exposure index, 0.223 ± .043 $Pa \cdot sec \cdot cm^{-2} \cdot °C^{-1}$.

DISCUSSION

Gasping at the onset of cold water immersion appears to be a valid indicator of regional cutaneous sensitivity to cooling because stimulation of select skin regions produced a measurable response, thus allowing a comparison to be made between regions. Gasping occurred concurrently with the decline in skin temperature, in agreement with previous observations (Hayward and Eckerson, 1984; Mekjavic et al., 1987; Tipton and Golden, 1987). Though the average range of skin temperature decline observed in this experiment (from 33.5°C to 21°C) is similar to the range (from 36°C to between 16 and 22°C) investigated by Crawshaw et al. (1975), it is suggested that in this study cold receptor excitation induced the gasp, whereas in the study by Crawshaw et al. (1975), the measured response, decline in sweating rate, was likely the result of cold receptor excitation inhibiting sweating. In both studies, warm receptor activity may have influenced the progression of the response while skin temperatures were above 30 °C (Duclaux and Kenshalo, 1980).

Present results are in agreement with the findings of Keatinge and Nadel (1965), that the upper torso is more sensitive to cooling than either the arms or the legs. The present ranking of 4 regional thermosensitivities agrees with the ranking predicted by averaging the sensitivity of the corresponding subregions tested by Keatinge and Nadel (1965). In contrast, the finding by Tipton and Golden (1987) of an equivalency in thermosensitivity of the two large skin regions, the torso and limbs, disagrees with the ranking predicted by averaging of the indices of the subregions, in either the present or the earlier study by Keatinge and Nadel (1965). Tipton and Golden (1987) suggest that spatial summation over the large areas tested may have obscured the sensitivity of the subregions (tested by Keatinge and Nadel, 1965), but this was not apparent in the present study, except perhaps for lower torso exposure.

Differences in methodology may also explain the disparity between the observation by Tipton and Golden (1987) of a similarity in thermosensitivity between the whole torso and the limbs, and the prediction by the present findings, that the thermosensitivity of the combined upper and lower torso would be greater than the sensitivity of the combined arms and legs. The ventilatory measurements of Tipton and Golden (1987) may underestimate the magnitude of the gasp response, compared to P0.1 measurements (Mekjavic et al., 1987). In addition, Tipton and Golden (1987) argued against the value of controlling the regional surface area, and ignored the 5% difference in exposed surface area between the torso and the limbs. But theoretically, the areal extent of the cooled region will influence the thermosensitive response (Hensel and Zotterman, 1951). Furthermore, Crawshaw et al. (1975) have shown that for relatively small increments in the cooled surface area in the abdominal region, there was a proportional increase in the rate of decline of sweating. In the present study, the results of statistical tests for differences between partial exposure conditions for measures of $\int \Delta P0.1_{c\text{-}w} / \Delta \bar{T}$ (Fig. 10), were the same as for measures of $\int \Delta P0.1_{c\text{-}w} / (SA \cdot \Delta \bar{T})$ (Fig. 11), indicating that correction for differences in surface area between conditions was not influential to the results. The uniformity of results, however, indicates only that the differences in response berween the upper torso, arms, and legs were sufficiently large, and the differences in surface area between the conditions were sufficiently small, that a correction for differences in the exposed surface area between conditions had an insignificant effect. It cannot be concluded that correction for differences in surface area of stimulated regions is unnecessary.

The assessment of regional cutaneous thermosensitivity requires that unexposed skin regions remain thermoneutral. The present study satisfies this requirement. Though pre-immersion skin temperatures of unexposed regions were slightly higher than those of exposd regions, prior to cold water immersion (Table 3), all pre-immersion skin temperatures lay within a range of thermoneutrality, where the cold and warm receptors are

relatively quiescent (Necker, 1981). During cold water immersion, limited cooling did occur under the rubber covers, but overall there was no substantial change in the unexposed mean skin temperature. Thus, cutaneous thermoreceptor activity in the unexposed region did not likely have a significant influence on the gasp response. In the study by Keatinge and Nadel (1960), skin temperature was not measured, but their use of showers had the advantage of limiting the cooling to the selected region. Thus, the agreement between their results and present findings would be expected. Cooling of unexposed skin regions (approximately 4 - 5°C) in the study by Tipton and Golden (1987) may have obscured a higher thermosensitivity for the torso region.

The present study observed a reduced temperature decline in the lower torso, which was attributed to the thermal protection provided by the swim suit, the seated position, and possibly, the bordering both above and below of insulated regions. Because the gasp response is highly sensitive to the rate of skin cooling (Mekjavic et al., 1987), the reduced cooling in the lower torso suggests that the response was diminished. Keatinge and Nadel (1960) observed the highest sensitivity to cooling in the lower front torso, in agreement with the higher sensitivity to heating attributed to this region in studies on other animals (Waites, 1962; Hales and Hutchison, 1971; Ingram and Legge, 1972). In contrast, the lower back torso was observed by Keatinge and Nadel (1960) to have a low sensitivity to cooling, close to that of the extremities. Thus, the lower torso, including both the front and back, might be expected to have a sensitivity higher than the extremities, as indicated by the present thermosensitivity index, which corrects for the diminished lower torso cooling.

Results are partially in agreement with the sensitivity coefficients to cooling measured by a decline in sweating rate. Crawshaw et al. (1975) observed that the thermosensitivity of the upper torso was greater than the abdomen, and thigh, but similar to the lower leg (the thermosensitivity of the lower torso and arm were not tested). However,

the regions tested in that study comprised a relatively small surface area, and it may be incorrect to assume that the sensitivity of a large region matches that of the smaller region within it. Furthermore, the reliability of comparisons is reduced because of the low sample size (n=3), the lack of statistical testing, and the large variation between individuals, in this earlier study by Crawshaw et al. (1975).

Theoretical Considerations

An increased thermosensitivity of a skin region may be attributed to either an increased density of thermoreceptors, or an increased weighting of the thermoafferent information from this area during central processing. It is impossible, with the present state of knowledge, to unravel the exact basis for differences in regional thermosensitivity. Even if a skin region was shown to have a higher density of thermoreceptors, the effect of this increase on the function of the organism would have to be evaluated. A one-to-one correspondence between the afferent and efferent nervous system cannot be assumed, because as the thermoafferent signal travels up the spinal cord it comes under a complex supraspinal control, which is neither entirely inhibitory nor excitatory (Pierau et al., 1984). Processing of non-facial ascending information appears to occur in a connected series, involving raphe nuclei, the pons, the thalamus, and the hypothalamus (Hellon, 1983). Yet the characteristics of cutaneous receptors, excluding facial and scrotal areas, seem to be represented at all levels of central processing (Hellon, 1983), suggesting that the activity/temperature relationship is retained centrally. Thus, the greater thermosensitivity of the upper torso may indicate that this region has a greater density of thermoreceptors or a greater influence centrally, than do the extremities. Similarly, the lower torso may also have a greater density of thermoreceptors, or influence centrally, as the thermosensitivity of this region was not significantly different from that of the upper torso.

The equivalency of the sum of regional responses to the whole body exposure response, suggests that regional thermoafferent signals interact in an additive manner, in accordance with the general finding that the sensitivity of the thermoregulatory response is equivalent to the sum of peripheral and central thermosensitivities (Simon et al., 1986). Though the gasp response does not appear to be a thermoregulatory response, it is sensitive to two important controls of mammalian thermoregulation: the deviation of skin temperature (Hellon, 1981), and the rate of temperature decline (Werner, 1983). Therefore, the thermoregulatory response to skin cooling may emanate from a summation of regional thermoafferent inputs, with some regions having a greater influence than others.

Individual Differences

The results of this study support earlier findings of a wide variation in the magnitude of the gasping response between individuals for both whole body (Keatinge and Evans, 1960; Cooper et al., 1976; Mekjavic et al., 1987) and regional exposures (Keatinge and Nadel, 1965; Tipton and Golden, 1987). Variability may arise because the efferent pathways for a thermal response do not emanate from one key integrative site in the CNS (Gordon and Heath, 1986); also, both thermal and non-thermal factors (for example, emotional status, and hydrostatic pressure) influence the central motor output, adding to the variability.

The respiratory system is influenced by an extensive number of stimuli (Milic-Emili et al., 1981), necessitating the use of a control for comparison with the test condition. Breath-to-breath measurements of the breathing pattern suggest that the drive component of the respiratory system has greater variability than the rhythm-generating function (Tobin et al., 1988). Measuring variation by the coefficient of variation (CV), the intraindividual breath-to-breath variation in resting respiratory drive of 52%, observed in this study, is

higher than the value of 32% reported by Tobin et al. (1988), but in both studies the day-to-day intraindividual CV, as well as the interindividual CV, were substantially less: 20% and 27%, respectively, in this study; and 9% and 22%, respectively, in the study by Tobin et al. (1988). The lower CV values reported by Tobin et al. (1988) may be associated with the method of measuring respiratory drive. Tobin et al. (1988) measured inspiratory flow (V_T/T_I), prior studies measuring interindividual variation in resting respiratory drive by mouth occlusion pressure report higher CV values of 39% (Sorli et al., 1978) and 57% (Mann et al., 1978). In this experiment, though between trial intraindividual variation in resting P0.1 was less than interindividual variation, the comparison of each immersion P0.1 with the resting value for that trial, reduced the influence of day-to-day variability.

A large interindividual variation in respiratory drive was observed, accompanying hyperventilation at the onset of cold water immersion (Fig. 6). The reason for this wide variability in response is not obvious, all subjects were young and healthy. The time interval between consecutive cold water trials was considered sufficient to avoid habituation, and none of the subjects were normally experiencing cold exposure. Because only males were tested, differences in response cannot be attributed to differences in sex, though Hayward and Eckerson (1984) report that the gasping response is not significantly different between males and females. Likewise, differences in response cannot be attributed to differences in body size and subcutaneous adiposity (as indicated by skinfold thickness), though Mekjavic et al. (1987) report no trend between the magnitude of the gasp response and a subject's surface area or subcutaneous adiposity. In addition, the experiment was designed to minimize extraneous stimuli, but despite these precautions, large individual differences persisted.

Ranking the subjects in order of magnitude of response (Fig. 12), a trend was observed, subjects with a relatively high thermosensitivity for the upper torso rank high in thermosensitivity for the lower torso, and for the body as a whole. This finding agrees with

the logical expectation that if the thermosensitivity of the individual parts is high, then the thermosensitivity of the whole body would be high as well, and vice versa. One subject tended to rank highest and one, the lowest, in magnitude of TSI for all five conditions. Thus the differences in intensity of response, noted previously for whole body immersion, appear to be a characteristic of the individual that is retained in regional immersions.

Practical Considerations

Gasping at the onset of accidental cold water immersion increases the risk of aspirating water, even for skilled swimmers (Keatinge et al., 1969). Thermal protection will reduce gasping (Mekjavic et al., 1987), but whole body protection for people engaged in high-risk occupations is not always feasible (Tipton and Golden, 1987). Furthermore, if rescue is likely to be initiated quickly, the greatest threat is not from long term exposure, but from the first few minutes of cold shock. Any method which retards immediate cooling of the skin, especially the highly thermosensitive regions, will likely enhance survival by reducing respiratory distress. Present findings suggest that for those individuals at risk, who are unable to use complete survival suits or to enter the water slowly, the most efficient protection against cold water drowning may be a close fitting lifejacket designed to provide not only flotation, but thermal insulation of the torso as well.

Conclusions

An improved technique was used in the present study to assess regional cutaneous thermosensitivity by the gasping response: differences in surface area and temperature decline of exposed regions were accounted for, and thermoneutrality of non-exposed regions was confirmed. From a theoretical point of view, a dynamic change in the activity

of cutaneous thermoreceptors would have occured only in exposed regions. In addition, the constancy of pre-immersion skin temperature among exposed regions, prior to cold water immersion (Table 3), ensured that the magnitude of dynamic thermoreceptor activity would not have been affected by a difference in the adaptive temperature (Kenshalo and Duclaux, 1977). Thus, the greater thermosensitivity of the upper torso, compared to the extremities, appears valid. Additionally, the lower torso may also be a region of increased thermosensitivity.

The gasp response has been shown to be influenced by non-thermal factors (Cooper et al., 1976; Mekjavic and Bligh, 1989), therefore, the matching of cold water conditions with control immersions was necessary, if the influence of thermal factors was to be evaluated. The experimental design and the method of analysis chosen for this study successfully isolated the component of the respiratory response due to the cooling stimulus alone. The use of a segmental neoprene suit to direct water to a specific skin region, while keeping the remainder of the body dry and thermoneutral, was unique. The results of this study suggest that the segmental suit was better able to achieve this goal than previous techniques reported in the literature (Tipton and Golden, 1987).

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Table 1. Physical characteristics of subjects

Variable	Mean	SD	Range (min - max)
age (yr)	25	4.5	20 - 33
mass (kg)	73.2	2.7	69.8 - 77
standing height (cm)	180.4	2.5	176.9 - 183.9
upper extremity length (cm)	78.5	2.5	73.5 - 81.1
lower extremity length (cm)	85.7	2.2	81.1 - 87.8
sitting height (cm)	94.7	1.8	92.1 - 96.6
Girths (cm): arm	30.4	1.4	28.1 - 32.2
chest	97.7	2.3	94.5 - 101.2
waist	76.4	1.7	74.6 - 79.2
thigh	55.4	2.0	52.4 - 58.4
calf	37.2	0.7	36.1 - 38
Sum of 6 skinfolds* (mm)	49.2	10.6	37.4 - 64.4

* triceps, subscapular, abdominal, supraspinale, front thigh, medial calf

Table 2. Exposed surface area for the 5 experimental conditions

Condition of exposure	Mean (cm^2)	SD	Range (min - max)
arms	1,910	157	1,636 - 2,078
upper torso	3,594	403	3,035 - 4,231
lower torso	2,358	517	1,828 - 3,319
legs	5,294	307	4,823 - 5,841
Sum of 4 regions	13,156		
whole body	15,296	532	14,518 - 15,796

Table 3. Skin temperature during cold water immersion conditions: the temperature of the exposed region at the end of the pre-immersion period; the temperature of the unexposed region at the end of the pre-immersion period; the change in the temperature of the exposed region from the pre-immersion value, at the end of the first minute of immersion; the change in the temperature of the unexposed region from the pre-immersion value, at the end of the first minute of immersion. Values are mean ± SD. See Methods and Materials section for details of calculation of $\bar{T}$.

Condition of exposure	Pre-immersion $\bar{T}$ exposed region (°C)	Pre-immersion $\bar{T}$ unexposed region (°C)	Immersion $\Delta\bar{T}$ exposed region (°C)	Immersion $\Delta\bar{T}$ unexposed region (°C)
whole body	33.5 ± 0.9		-13.0 ± 0.7	
arms	33.9 ± 0.9	35.2 ± 1.0	-14.2 ± 0.4	-0.3 ± 0.2
upper torso	33.9 ± 0.8	34.8 ± 0.3	-14.0 ± 1.6	-1.3 ± 0.6
lower torso	34.0 ± 0.5	35.3 ± 0.6	-9.7 ± 1.6	-0.7 ± 0.4
legs	32.3 ± 0.8	35.3 ± 0.7	-13.3 ± 1.2	-0.8 ± 0.6

Table 4. Skin temperature during lukewarm water immersion conditions: the temperature of the exposed region at the end of the pre-immersion period; the temperature of the unexposed region at the end of the pre-immersion period; the change in the temperature of the exposed region from the pre-immersion value, at the end of the first minute of immersion; the change in the temperature of the unexposed region from the pre-immersion value, at the end of the first minute of immersion. Values are mean ± SD. See Methods and Materials section for details of calculation of $\bar{T}$.

Condition of exposure	Pre-immersion $\bar{T}$ exposed region (°C)	Pre-immersion $\bar{T}$ unexposed region (°C)	Immersion $\Delta\bar{T}$ exposed region (°C)	Immersion $\Delta\bar{T}$ unexposed region (°C)
whole body	32.7 ± 1.0		+1.6 ± 1.0	
arms	34.2 ± 0.8	35.1 ± 0.6	+0.6 ± 0.5	+0.2 ± 0.1
upper torso	33.7 ± 1.0	33.5 ± 1.4	+0.8 ± 0.8	+0.7 ± 0.4
lower torso	34.0 ± 1.2	34.0 ± 1.2	+0.3 ± 0.8	+0.6 ± 0.6
legs	33.3 ± 1.0	35.0 ± 0.6	+1.2 ± 0.7	+0.1 ± 0.1

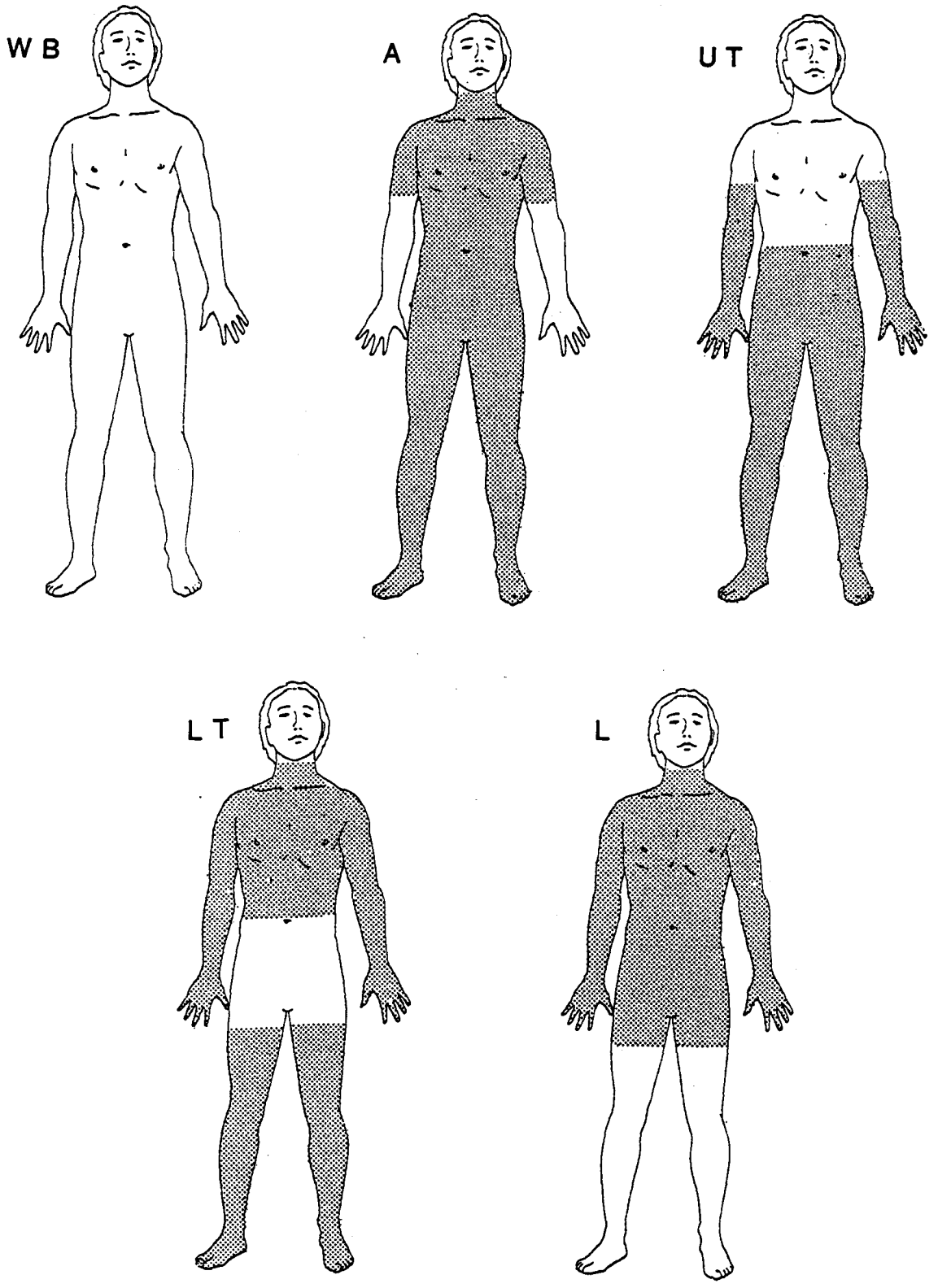


Fig. 1. Diagram of 5 conditions of exposure: whole body exposure (WB); arms exposure (A); upper torso exposure (UT); lower torso exposure (LT); and legs exposure (L). The shaded area represents the segmental neoprene suit.

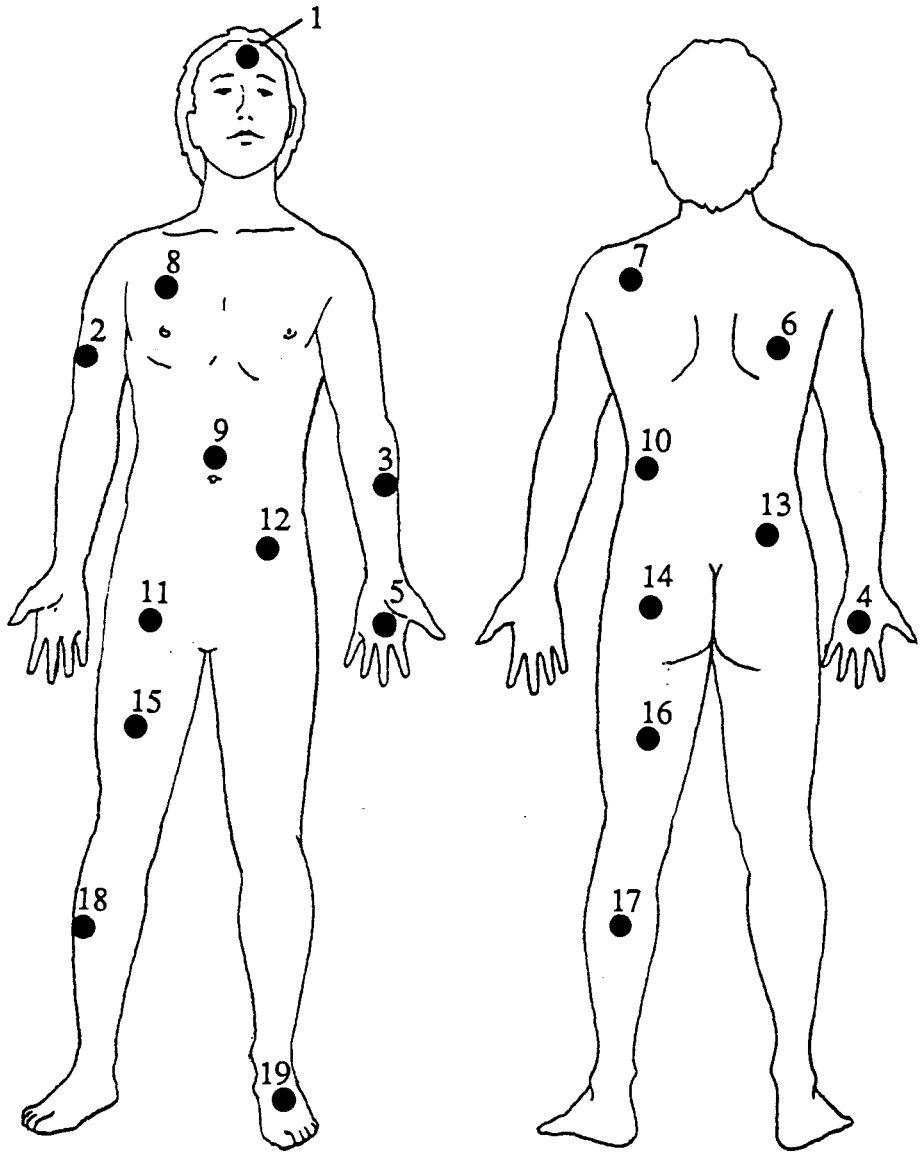


Fig. 2. Position of skin temperature measurement at 19 sites. For detailed definition of anatomical landmarks see Methods and Materials section.

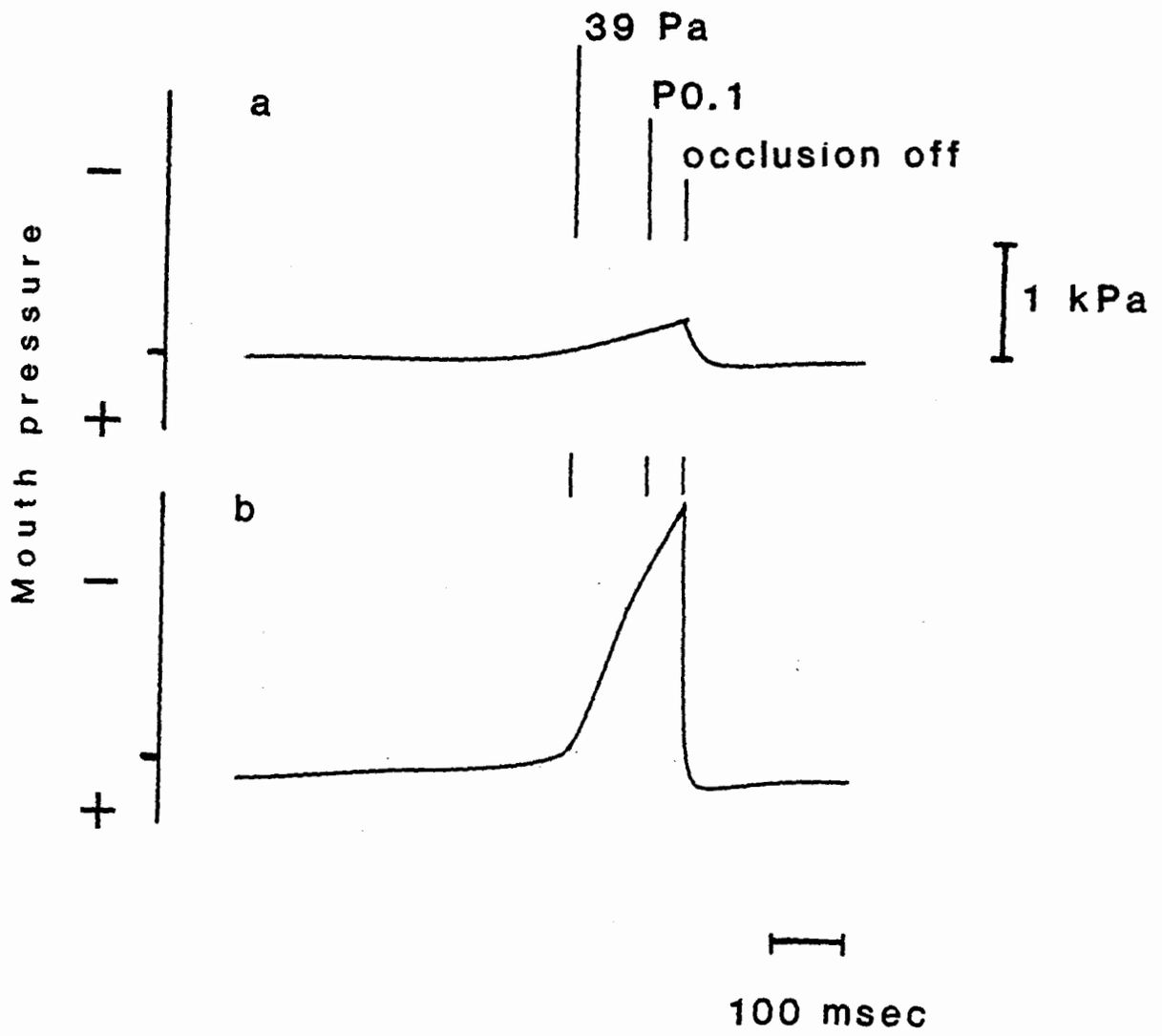


Fig. 3. Representative traces of single P0.1 measurement from one trial, during rest (a) and during immersion (b).

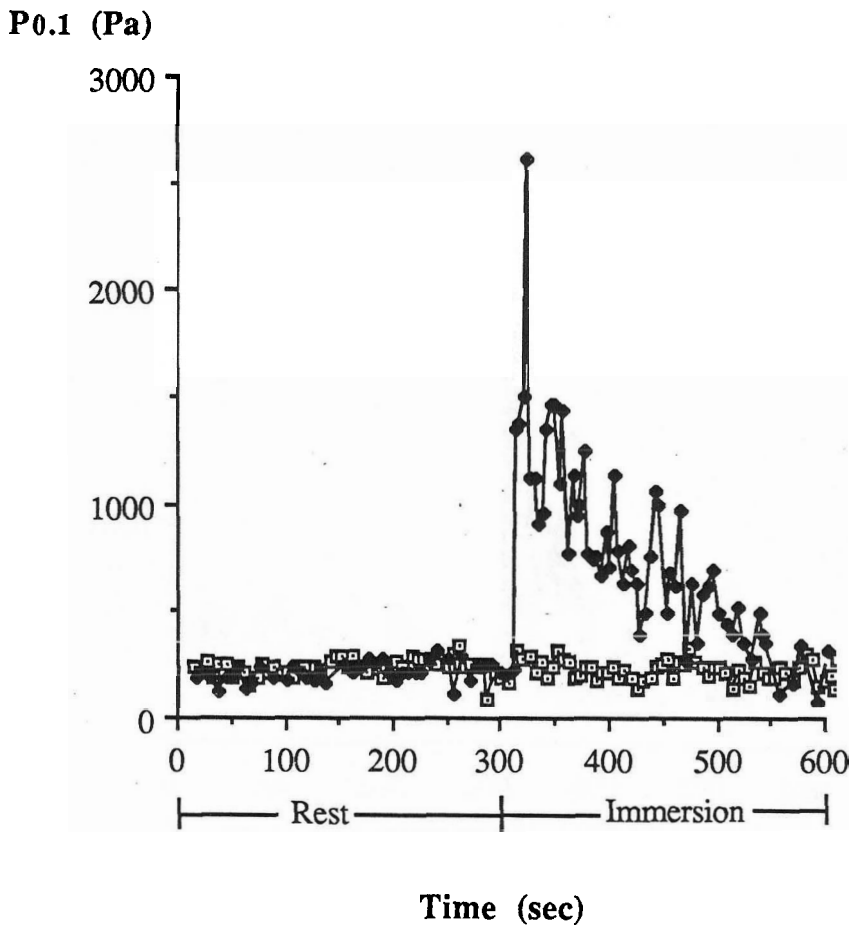


Fig. 4. The Po.1 measurements for one subject (RV), during whole body exposure in cold (●), and lukewarm (▣) water.

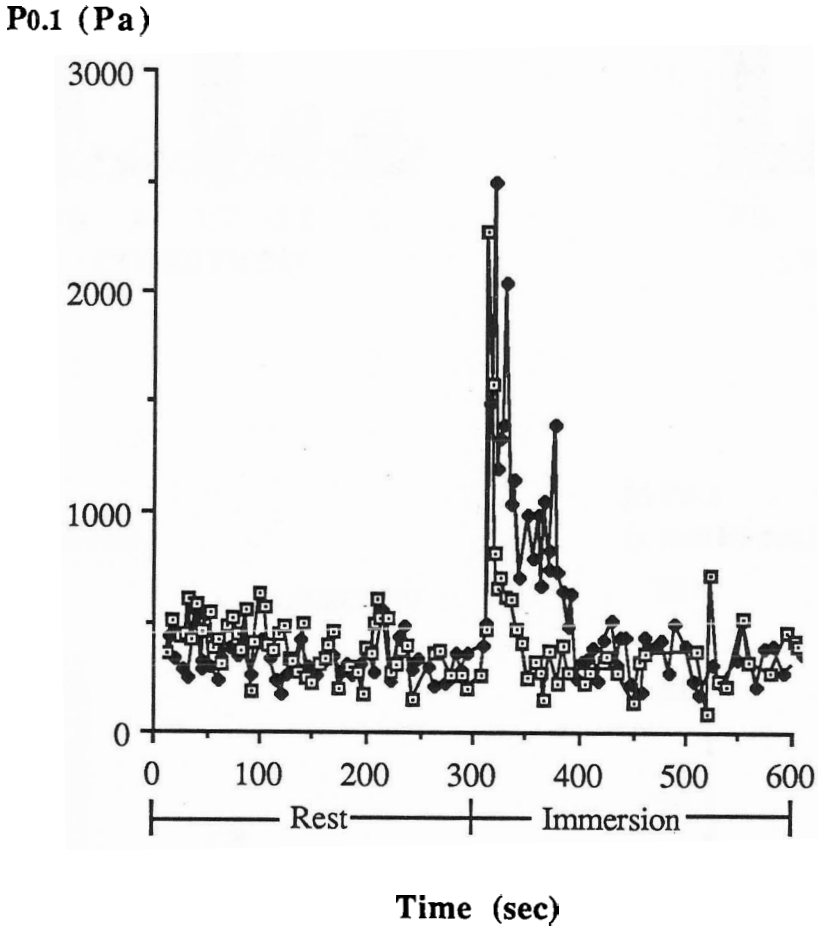


Fig. 5. The Po.1 measurements for one subject (AR), during 2 cold water trials: upper torso exposure (•); and lower torso exposure (▫).

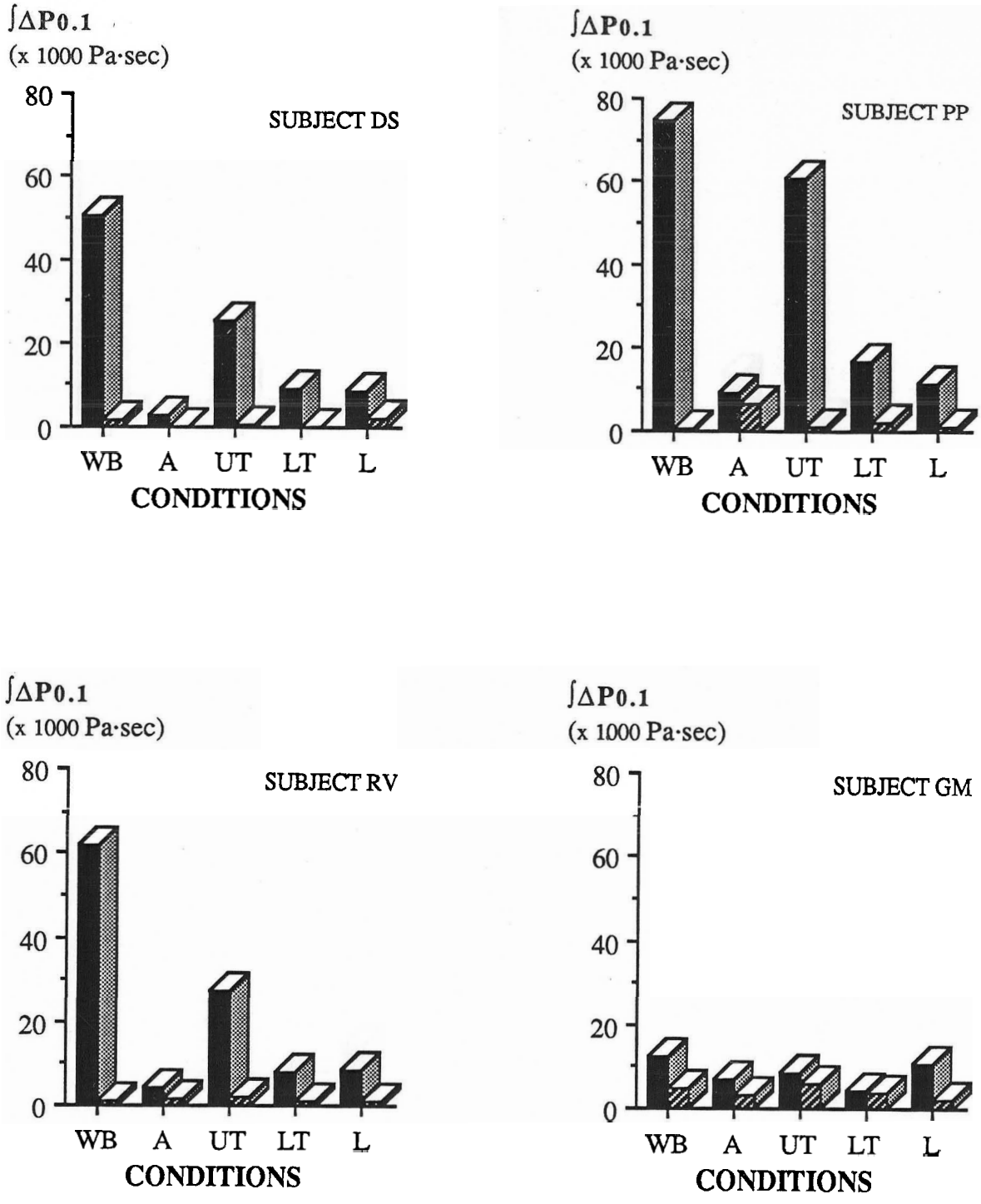
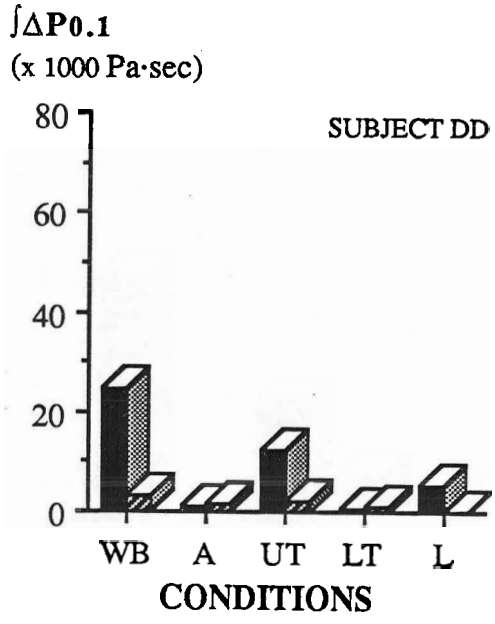
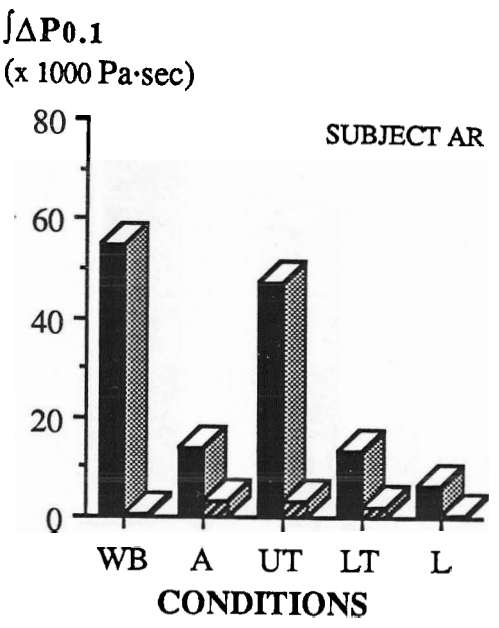


Fig. 6. The ∫ΔP0.1 measurements for the first minute of immersion for each of 7 subjects, for all trials: cold (solid bar) and lukewarm (striped bar). Conditions are whole body exposure (WB); arms exposure (A); upper torso exposure (UT); lower torso exposure (LT); and legs exposure (L).





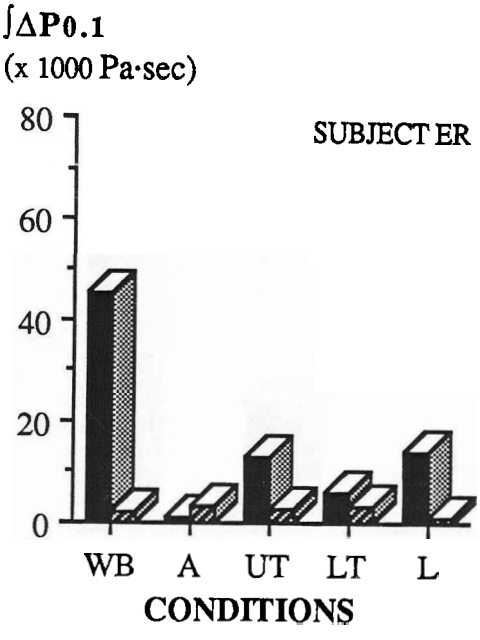


Fig. 6. Continued.

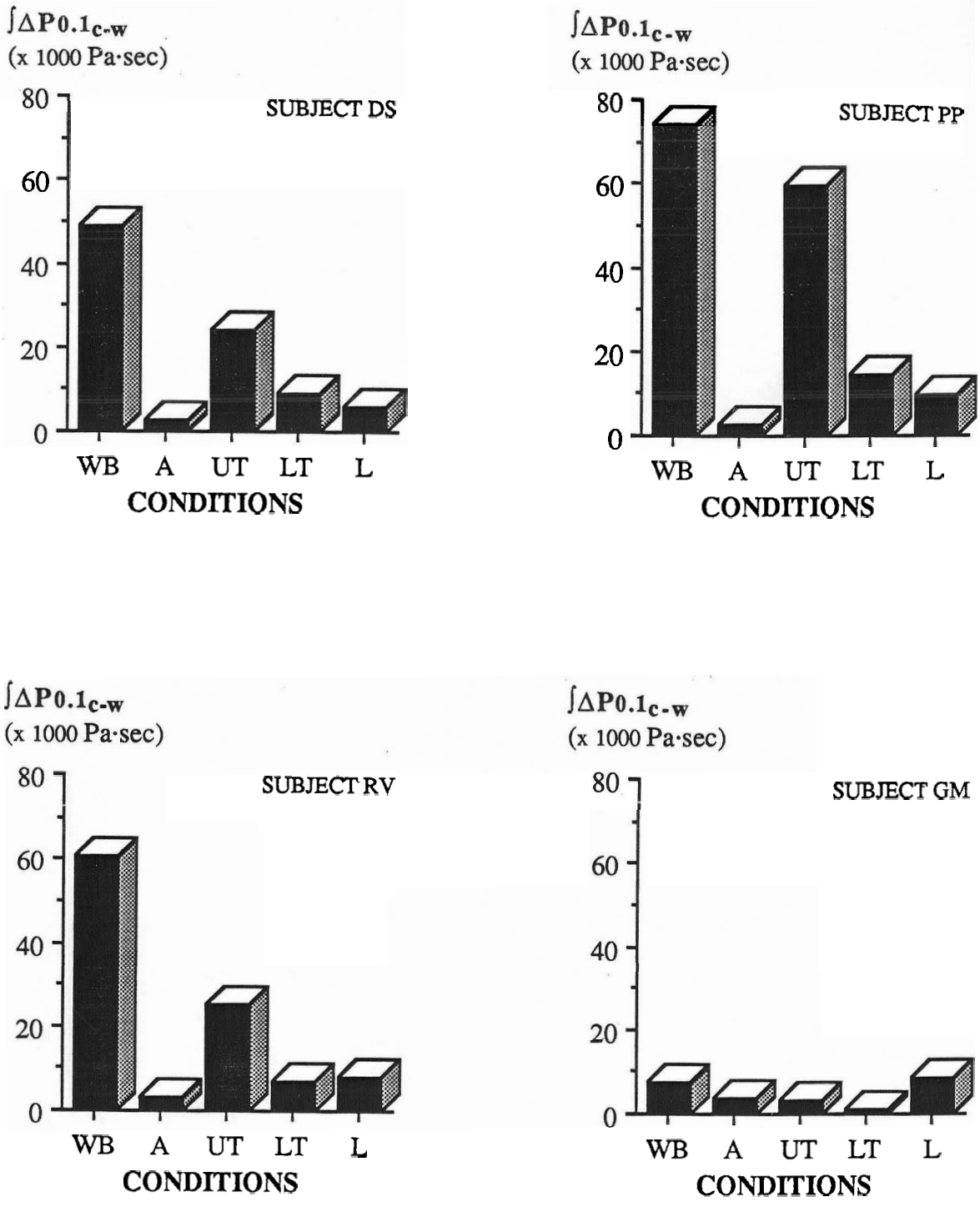
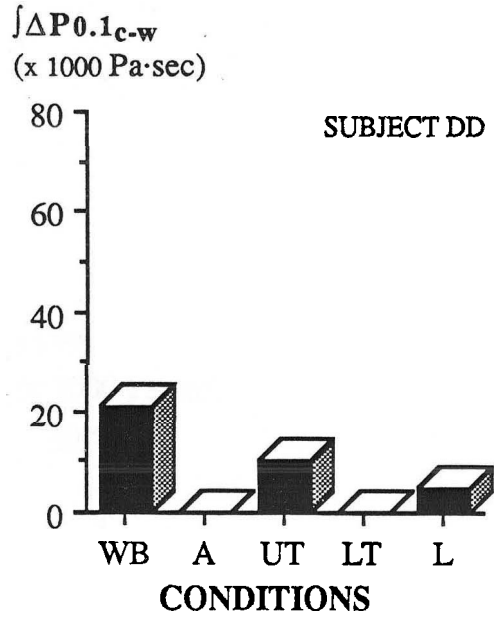
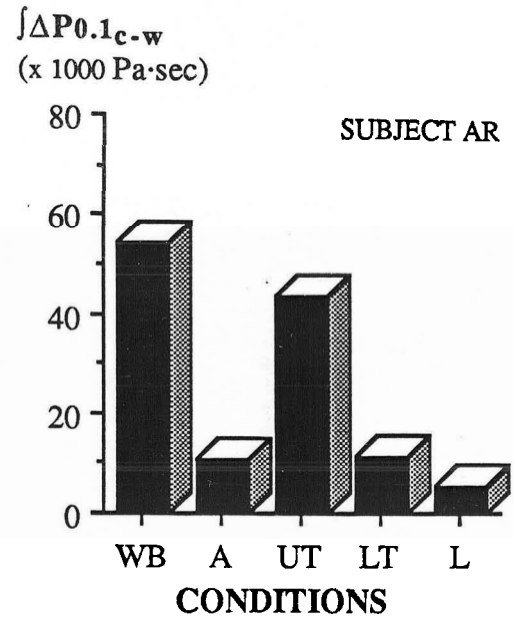


Fig. 7. The integrated one minute ΔP0.1 response to cold water immersion minus the integrated one minute ΔP0.1 response to the matching lukewarm water immersion ($\int\Delta P0.1_{c\text{-}w}$) for each of 7 subjects. Conditions are whole body exposure (WB); arms exposure (A); upper torso exposure (UT); lower torso exposure (LT); and legs exposure (L).





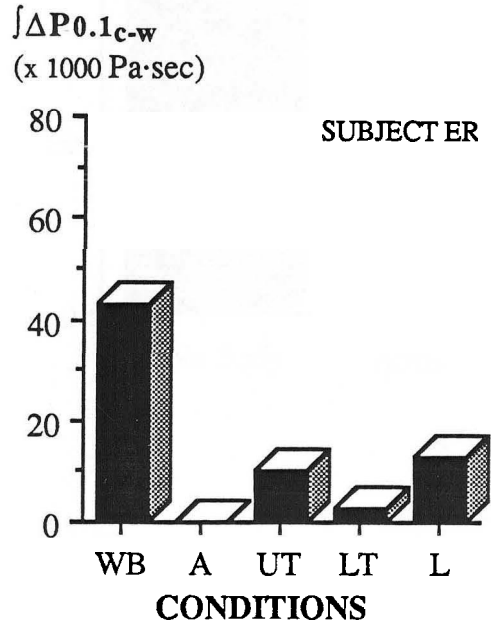


Fig. 7. Continued.

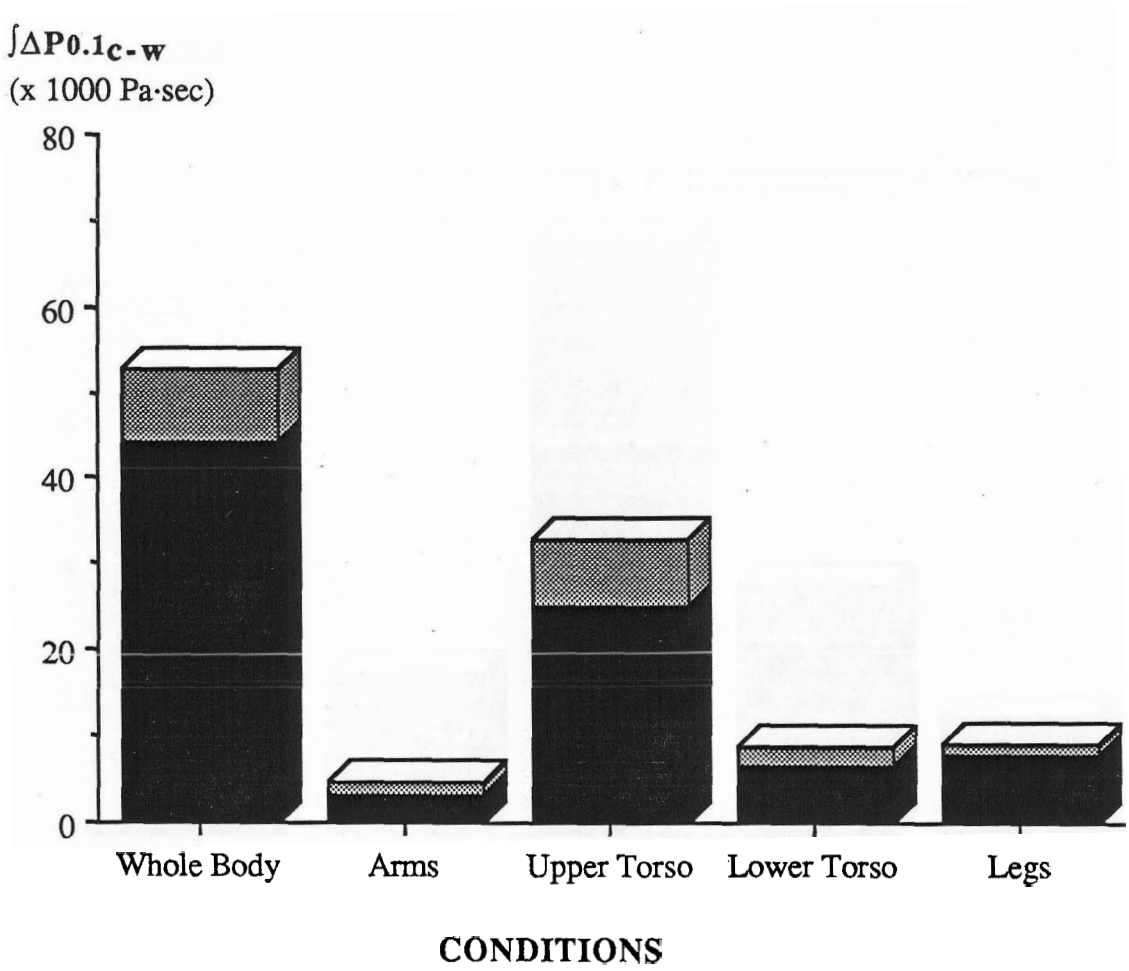


Fig. 8. The average $\int\Delta P0.1_{c-w}$ response. Conditions are whole body exposure (WB); arms exposure (A); upper torso exposure (UT); lower torso exposure (LT); and legs exposure (L). Values are mean (solid bar) + SE (shaded bar).

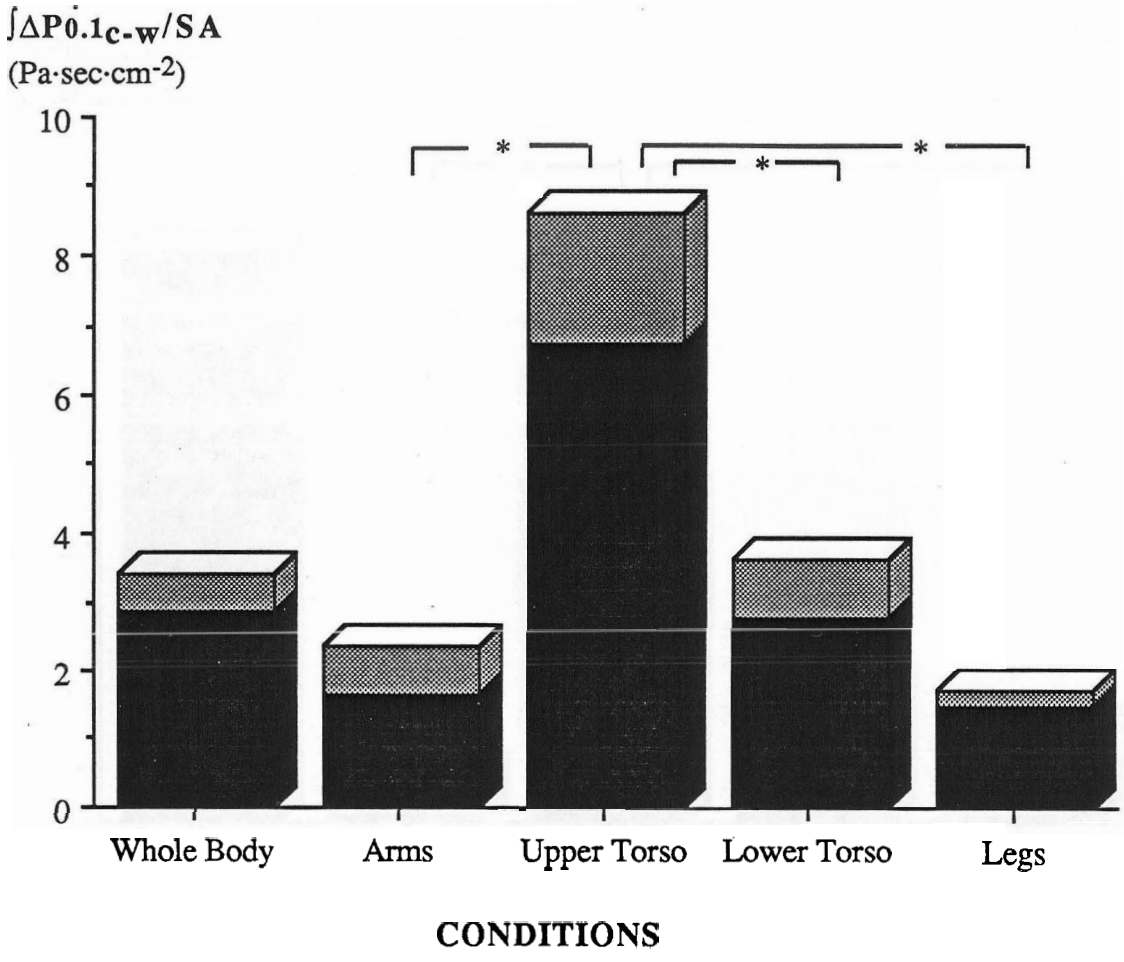


Fig. 9. The $\int \Delta P0.1_{c-w}$ value for each subject is divided by the exposed surface area for that condition. Values are mean (solid bar) + SE (shaded bar). The asterisk denotes a significant difference between partial exposure conditions, $p \leq 0.05$.

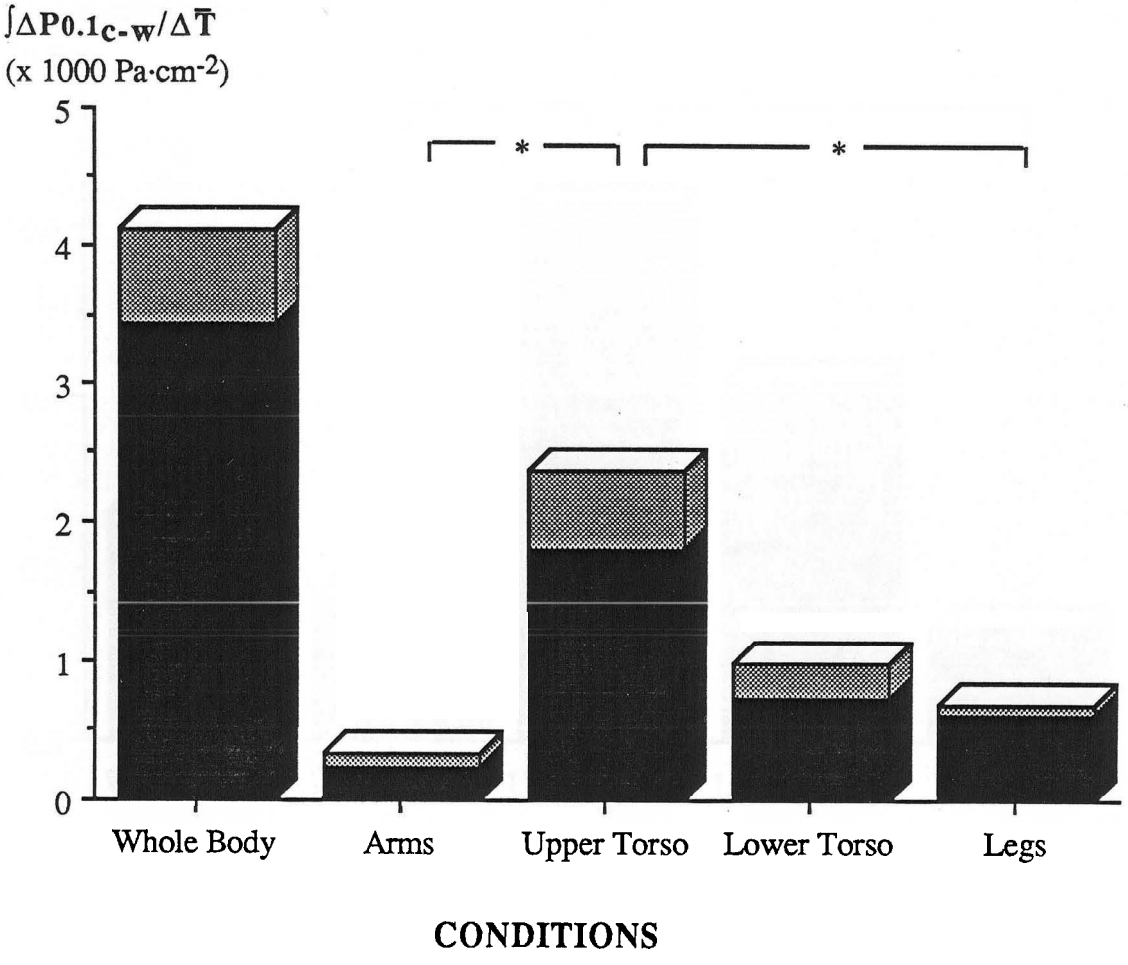


Fig. 10. The $\int \Delta P0.1_{c-w}$ value for each subject is divided by the exposed skin temperature decline for that condition. Values are mean (solid bar) + SE (shaded bar). The asterisk denotes a significant difference between partial exposure conditions, $p \leq 0.05$.

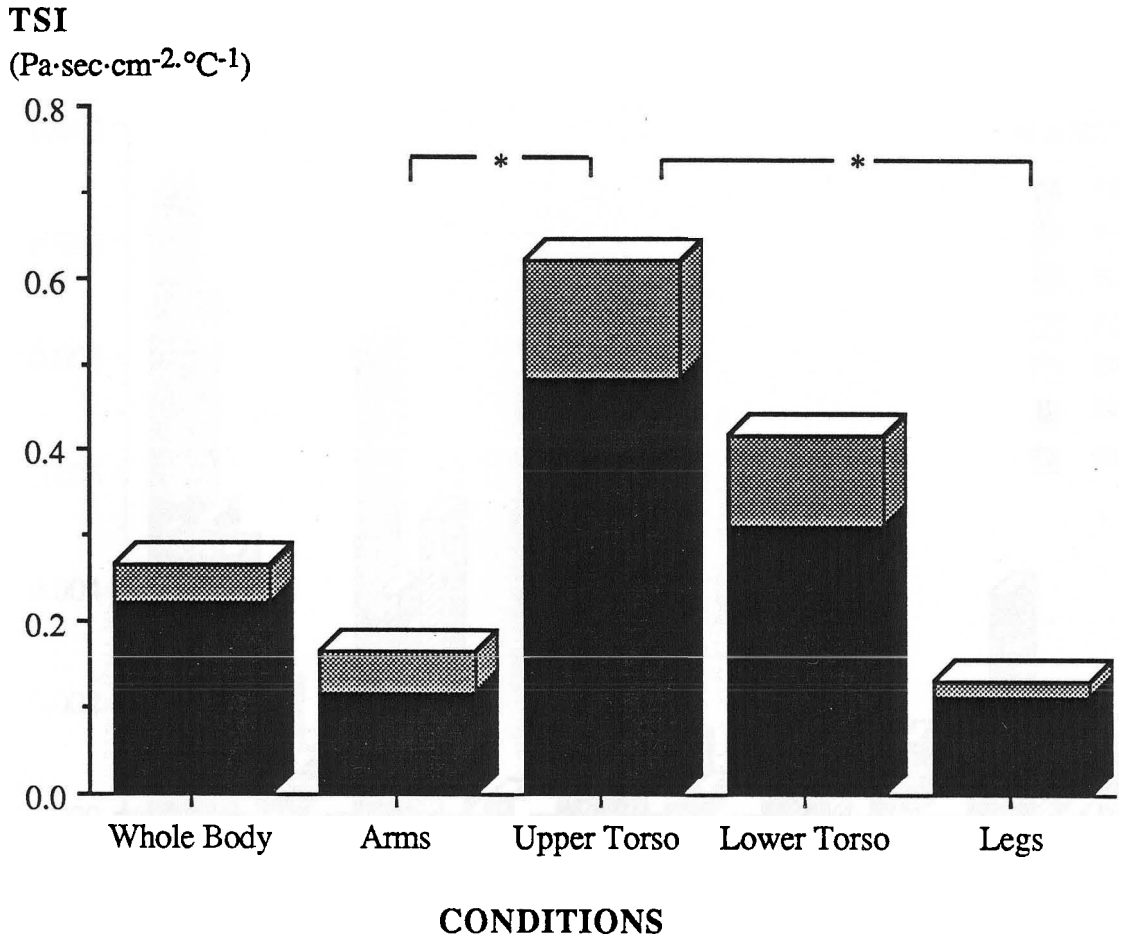


Fig. 11. Thermosensitivity index (TSI). The $\int \Delta P0.1_{c-w}/SA$ value for each subject is divided by the skin temperature decline in the exposed area $\int \Delta P0.1_{c-w}/(SA \cdot \Delta \bar{T})$. Values are mean (solid bar) + SE (shaded bar). The asterisk denotes a significant difference between partial exposure conditions, $p \leq 0.05$.

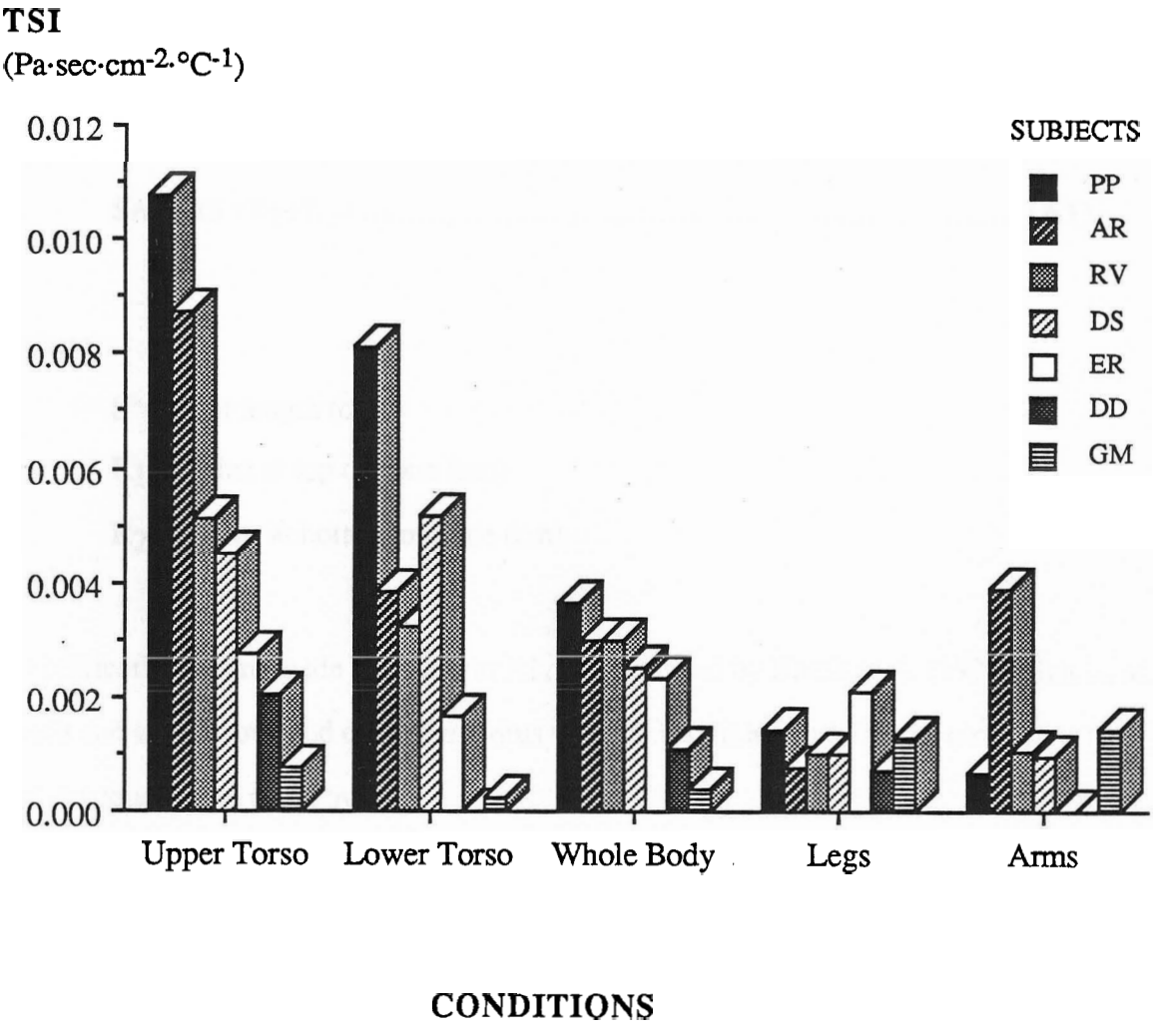


Fig. 12. Individual thermosensitivity indices (TSI) are ranked in descending order for upper torso exposure. This ranking order is maintained for the 4 other conditions.

APPENDIX A :

DEVELOPMENT OF SURFACE AREA EQUATIONS

Truncated Cone

The equation to calculate the lateral surface area of a truncated cone, as depicted in Fig. 13a is (Riddle, 1979):

$$SA = \pi S (R_1+R_2) \quad \text{(A1)}$$

where,

S = slant length (cm)

R_1= radius at top of cone (cm)

R_2 = radius at bottom of cone (cm)

Modifications were made to equation A1, as suggested by Katch et al. (1974), that is, at each end of the truncated cone, the radius was calculated by dividing the circumference (C) of the cone at that point by 2π:

$$SA = S \cdot (C_1+C_2)/2 \quad \text{(A2)}$$

The vertical length (H) of the cone was substituted for the slant length (S). Calculations of S showed there was a negligible difference between it and H. Thus, equation A2 was modified to:

$$SA = H \cdot (C_1+C_2)/2 \quad \text{(A3)}$$

Wedge

The equation to calculate the surface area of a wedge (Fig 13b) is:

$$SA\ (wedge) = L \cdot H + H \cdot B + B \cdot \sqrt{(H^2+L^2)} \quad \text{..... ..... (A4)}$$

where,

L= horizontal length (cm)

H= vertical length (cm)

B= breadth (cm)

Again, modifications were made to equation A4, as suggested by Katch et al. (1974). Breadth was calculated by dividing the circumference (C) of the ankle or wrist by π:

$$SA(wedge) = L \cdot H + H \cdot (C/\pi) + (C/\pi) \cdot \sqrt{(H^2+L^2)} \quad \text{..... ..... (A5)}$$

Portions Of Truncated Cones

The surface area for portions of truncated cones (shaded area in Fig. 13c), with unknown radius and circumference at point 3, was calculated by modifications to equation A1. The radius (R_3) has a linear relationship with the radii (R_1 and R_2), as diagrammed in Fig. 13d. R_3 can be calculated as:

$$R_3 = ((R_2-R_1)/H_2) \cdot H_1 + R_1 \quad \text{.....(A6)}$$

where,

R_3 = radius at point 3, Fig. 13c (cm)

R_1 = radius at top of cone proper, as per Katch et al., 1974 (cm)

R_2 = radius at bottom of cone proper (cm)

H_2 = vertical length of cone proper (cm)

H_1 = vertical length of cone portion (cm)

Each radius is calculated by dividing the circumference at that point by 2π, thus, equation A6 is modified to:

$$C_3 = (H_1/H_2) \cdot (C_2-C_1) + C_1 \quad \text{..... (A7)}$$

To calculate the surface area of the portion of the truncated cone (shaded area in Fig. 13c), equation A7 is substituted for C_2 in equation A3:

$$SA(\text{cone portion}) = H_1 \cdot (C_1 + (H_1/H_2) \cdot (C_2-C_1) + C_1)/2 \quad \text{..... (A8)}$$

$$= H_1 \cdot (C_1 + (H_1/2H_2) \cdot (C_2-C_1)) \quad \text{..... ..... (A9)}$$

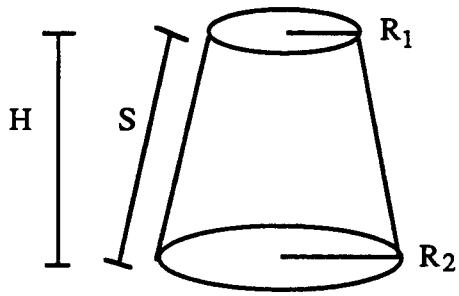
where,

H_1 = vertical length of cone portion (cm)

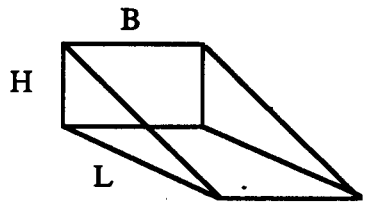
H_2 = vertical length of cone proper (cm)

C_1 = circumference at top of cone proper (cm)

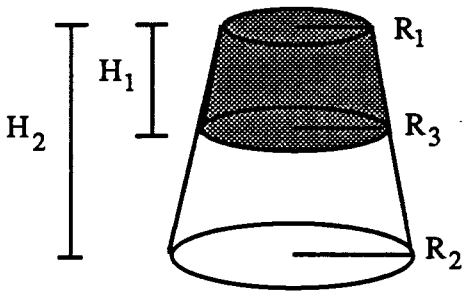
C_2 = circumference at bottom of cone proper (cm)



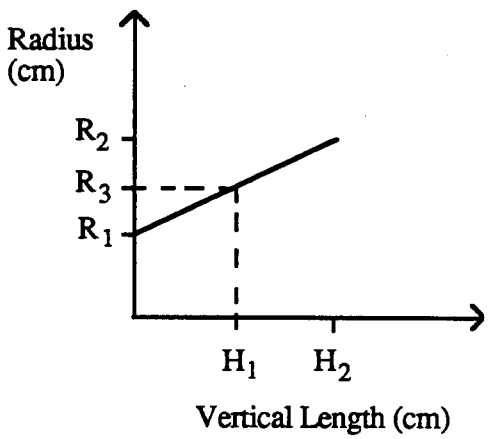
a. Truncated cone



b. Wedge



c. Portion of truncated cone



d. Radius as a function of vertical length

Fig. 13. Diagram of geometric shapes used in surface area calculations