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# Revisiting the dermatomal recruitment of, and pressure-dependent influences on, human eccrine sweating

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ABSTRACT

Herein we describe two experiments in which the recruitment and pressure-induced modifications of human eccrine sweating were investigated. In one experiment, the longstanding belief that glandular recruitment follows a gradual, caudal-to-rostral (dermatomal) recruitment pattern was re-evaluated. The onset of sweating was simultaneously determined (ventilated capsules) from four spinal (dermatomal) segments (forehead, dorsal hand, lower chest and dorsal foot) during the passive heating of supine participants ($N = 8$). No evidence was found to support either dermatomal or simultaneous glandular recruitment patterns. Instead, the results were more consistent with individualised (random) patterns of regional activation ($P > 0.05$), with significant time delays among sites. Such delays in the appearance of discharged sweat may reflect differences in neurotransmitter sensitivity, precursor sweat production or ductal reabsorption. In the second experiment, the pressure-induced hemihidrotic reflex (contralateral sudomotor enhancement) was revisited, using pressures applied over 10 $cm^2$ areas of the chest (left side: 6 $N\,cm^{-2}$) and left heel (3 $N\,cm^{-2}$) during both supine and seated postures ($N = 12$). Participants were passively heated and thermally clamped before pressure application. Hemihidrosis was not observed from the contralateral surfaces within the same (chest) or lower spinal segments (abdomen; both $P > 0.05$) during chest pressure, but a generalised enhancement followed heel pressure when supine. We suggest that previous observations of hemihidrosis possibly resulted from elevated heat storage, rather than a neural reflex. Chest pressure significantly inhibited ipsilateral sweating (forehead, hand, chest; all $P < 0.05$), and that influence is hypothesised to result from interactions between ascending mechanoreceptor afferents and the descending sudomotor pathways.

## 1. Introduction

Thermally activated sweating in humans develops firstly through the cholinergic activation of individual eccrine sweat glands (Ichihashi, 1936; Kuno, 1938 Machado-Moreira et al., 2012), and then by elevating flow from those glands (Ichihashi, 1936; Randall, 1946; Kondo et al., 1998). Based only on visual inspection, Oehler (1904) described sudomotor recruitment as a gradual progression across the body surface. Almost 50 years later, Hertzman et al. (1952) reported a caudal-to-rostral pattern of glandular activation; a sympathetic, dermatomal recruitment. Contemporaneously, Kuno (1934) observed posture-induced reductions in sweating from the lower, along with concurrent sweating elevations from the upper, skin surfaces during lateral recumbency; the hemihidrotic reflex or a contralateral sudomotor enhancement (Kuno, 1934). When further explored, Takagi and Sakurai (1950) claimed that "*pressure upon one side of the body evokes without exception a hemihidrosis of the opposite side*" (P. 28). Since both of those sudomotor phenomena have become widely accepted, although not universally supported, components of our understanding of human thermal homoeostasis, then we designed two experiments in which the veracity of those observations, and their interpretations, might be re-evaluated.

Unlike the dermatomal recruitment of discharged thermal sweating (Hertzman et al., 1952), Kuno (1934, 1956) described an almost simultaneous regional activation, with exceptions at the glabrous (non-hairy) skin of the hands and feet, regardless of how heating was applied. On the other hand, List and Peet (1938) observed considerable inter-subject recruitment variability. For some, sweat first appeared on the face; in others, it was initiated at the axillae and inguinal folds.

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Nonetheless, for most subjects, sweat was apparent on the face and torso, before being evident at the extremities (List and Peet, 1938).

It seems that many, including the current authors, may have overlooked those earlier observations, accepting instead the interpretation of Hertzman et al. (1952), which was subsequently supported by Randall and Hertzman (1953) and McCook et al. (1965), although those observations were entirely descriptive in nature. However, closer examination of those original observations has revealed that glandular activation was estimated, but not measured, using group data obtained from separate summer and winter trials, with each point representing a trial average (Hertzman et al., 1952). In addition, there is uncertainty whether data from each site were even obtained from the same participants. A subsequent investigation provided evidence from two individuals who showed a dermatomal recruitment, and a third with a different activation sequence (Randall and Hertzman, 1953). No group data were provided. In their third contribution, evidence from two more subjects was reported (McCook et al., 1965). Unfortunately, it is impossible to resolve recruitment time differences among some skin sites, even though they are evident between the foot and forehead (Randall and Hertzman, 1953; McCook et al., 1965). Our view has been, given the extensive inter-individual variability in sweating, that such data did not unequivocally support the dermatomal recruitment hypothesis (Taylor and Machado-Moreira, 2013).

With regard to the hemihidrotic reflex, there exists both contradictory (Watkins, 1956; Ferris, 1960) and supportive evidence (van Beaumont et al., 1966; Tadaki et al., 1981; Vaidya and Dhume, 1994; Wilsmore et al., 1999), with some investigators subscribing to a spinal, segmental interaction with the descending sudomotor neurones (Kawase, 1952; Ogawa et al., 1980; Okagawa et al., 2003). While the initial evidence for that view was also descriptive rather than quantitative, those inhibitory and excitatory effects appeared to be pressure dependent, rather than being postural influences *per se* (Kuno, 1934; Takagi and Sakurai, 1950; van Beaumont et al., 1966). Nonetheless, Watkins (1956) "*concluded that the reflex does not occur, or if it does occur in some circumstances, it is certainly not physiologically significant*" (P. 269).

Since almost every experiment into the hemihidrotic reflex involved the use of passive heating, then changes in sweat secretion accompanying pressure application will continuously track heat-storage variations. Therefore, if those non-thermal treatments inhibited sweating in one region, then heat storage may have been transiently elevated, thereby eliciting more pronounced sweating from the contralateral skin surfaces. In that scenario, pressure-associated sudomotor enhancement will primarily reflect temperature regulation, and may not necessarily be the consequence of a spinal reflex. To exclude such artefactual thermal influences, whole-body thermal clamping is required before applying postural or pressure treatments (Jessen, 1981; Cotter et al., 1995; Gordon et al., 2004). Since, to our knowledge, only one group has used that technique (Wilsmore et al., 1999), then the assumption that the hemihidrotic reflex is an unequivocally verified physiological phenomenon remained in doubt.

Consequently, experiments were planned through which to evaluate the veracity of both the dermatomal recruitment and hemihidrotic reflex hypotheses. The first experiment was designed to test the hypothesis that, during the passive heating of supine individuals, the recruitment of human eccrine sweating does not follow a sequential pattern of activation that commences at the feet, and gradually moves towards the head. In the second experiment, in which the isothermal clamping of mildly hyperthermic individuals was used, it was hypothesised that locally applied pressure to a lateral skin surface (chest) would not elicit hemihidrosis from the contralateral skin surfaces.

## 2. Methods

### 2.1. Experiment one: sudomotor recruitment

#### 2.1.1. Subjects

Eight habitually active (endurance exercise more than three times weekly) and asymptomatic males were exposed to a resting air exposure (age 31.4 y [standard deviation (SD) 13.7], body mass 72.9 kg [SD 7.4], height 175.5 cm [SD 5.3 cm]), following the provision of written, informed consent. None was taking medication, nor did any have either sweating disorders or a history of cardiovascular, neurological or thermal illness. All procedures were approved by the Human Research Ethics Committee (University of Wollongong) in accordance with the National Health and Medical Research Council (Australia).

#### 2.1.2. Procedural overview

This experiment involved a single, supine air exposure within a pre-warmed and thermally equilibrated climate chamber (28 °C, 2.27 kPa water-vapour pressure [60% relative humidity]). Subjects wore only a swimming costume, were blindfolded and positioned on a wire-mesh bed that maximised skin exposure to the air. When in place, participants were wrapped in a tube-only, water-perfusion garment (Paul Webb Associates, Yellow Springs, U.S.A.). In that state, sweating was not apparent, conversation ceased and other non-thermal sudomotor influences were minimised. Following preparation, supplemental heating was provided in three stages, by perfusing that garment with heated water (38-L, Grant GD120, Grant Instruments [Cambridge] Ltd., U.K.): 0–5 min (water bath temperature 40 °C), 5–10 min (bath temperature 45 °C) and beyond 10 min (bath temperature 50 °C). Those stages were aimed at the gradual activation of thermal sweating, with each trial terminated following the establishment of steady-state sweating from each of four sites.

#### 2.1.3. Experimental standardisation

Participants were required to avoid strenuous exercise and heavy alcohol consumption within the 12 h, and caffeine consumption for 2 h, prior to testing. Written instructions were provided concerning the establishment of the required pre-experimental euhydrated state, and this included ingesting 15 mL $kg^{-1}$ of additional fluid (any form) before retiring, with a further 500 mL consumed with breakfast. Low-fat, high-carbohydrate evening and morning meals were also prescribed.

#### 2.1.4. Sudomotor responses

Local (discharged) sweat rates were simultaneously measured using ventilated sweat capsules (3.16 $cm^2$; capacitance hygrometry) positioned at four right-side locations, none of which was covered by, or in contact with, the perfusion garment: forehead (first-second thoracic segments [T1-2]), dorsal hand (third-sixth thoracic segments [T3-6]), lower chest (fifth-sixth thoracic segments [T5-6]) and dorsal foot (tenth thoracic to second lumbar segments [T10-L2]; Purves et al., 2008). Those positions maximised the range of spinal segments investigated. Sweat capsules were glued to the skin to prevent local pressure influences (Collodion U.S.P., Mavidon Medical Products, U.S.A.), with pre-capsular airflow independently regulated (300 mL $min^{-1}$). The relative humidity of the inflowing air was maintained at 12% by passing room air through an enclosed container within which there was a saturated, lithium-chloride solution. The temperature of that air, as well as post-capsular (effluent) relative humidities and air temperatures were measured using capacitance hygrometers and thermistors that formed parts of an integrated sweat-monitor system (Clinical Engineering Solutions, NSW, Australia). Hygrometer calibration, performed using three saturated salt-solution standards, preceded experimentation. Data were sampled at 1-s intervals (DAS1602, Keithley Instruments, Inc., Cleveland, U.S.A.) and used to compute local sweat rates (Taylor et al., 1997).

#### 2.1.5. Tissue temperatures

Eight skin temperatures were simultaneously measured (Type EU, Yellow Springs Instruments Co. Ltd., Yellow Springs, OH, U.S.A.): forehead, right scapula, right chest, right arm, left dorsal forearm, left dorsal hand, right anterior thigh and left posterior calf. Thermistors were secured using a single layer of adhesive tape, with those data summed, using area-weighted coefficients, to derive mean skin temperature (Hardy and DuBois, 1938). In addition, insulated auditory canal temperature was monitored (Edale instruments Ltd., Cambridge, U.K.), since that method minimises artefactual influences, and thereby provides a valid, reliable and responsive index of changes in deep-body temperature (Keatinge and Sloan, 1975; Todd et al., 2014). Finally, a temperature-specific weighting of mean skin (10%) and auditory canal (90%) temperatures was used to approximate mean body temperature (Vallerand et al., 1992). Temperatures were recorded at 15-s intervals (Grant Instruments Ltd., 1206 Series Squirrel, U.K.), with thermistors calibrated against a certified reference thermometer (Dobros total immersion, Dobbie Instruments, Sydney, Australia).

#### 2.1.6. Data analysis

The variables of interests for the first experiment were the four regional sweat activation times, as determined from manual inspection of each local, discharged sweat rate. The definition of glandular activation (recruitment) was set using a local increase in sweat rate $> 0.05\,mL\,cm^{-2}.min^{-1}$ above site-specific basal values. In addition, non-continuous sweating was not included, since that primarily reflects the ever-present, non-thermal oscillations in sympathetic tone (Hagbarth et al., 1972), which even occurs during thermoneutral states. Instead, our interests were in the activation of continuous, thermally induced sweating. Those points were identified when sweating first crossed, and was thereafter sustained above that threshold. The time of that crossing point was taken to signify local sudomotor recruitment. Those data are reported in descriptive form, and include means and standard deviations (SD). In addition, a Friedman test was used to compare the order of sudomotor recruitment (four levels: forehead, hand, chest and foot), with Dunn's multiple comparison (*post hoc*) tests and *alpha* set at the 0.05 level.

### 2.2. Experiment two: pressure-dependent sweating responses

#### 2.2.1. Subjects

For the second experiment, a total of 13, similarly healthy and physically active males were recruited (age 26.1 y [SD 11.0], body mass 77.8 kg [SD 9.0], height 179.8 cm [SD 7.7 cm]), four of whom participated in experiment one. Subjects were similarly screened, and procedures were again approved by the Human Research Ethics Committee (University of Wollongong).

#### 2.2.2. Procedural overview

This experiment involved two trials within a pre-heated and equilibrated climate chamber (36 °C, 1.50 kPa water-vapour pressure [25% relative humidity]), with all but two individuals participating in both trials (repeated-measures design) and acting as their own controls. The same standardisation procedures were followed prior to each trial, and for this experiment, an isotonic drink was provided during preparation (200 mL). One trial was performed in a supine posture (head elevated 15-20° above horizontal; $N = 12$), and the other in a seated position (65° above horizontal; $N = 12$). Those trials were administered in a balanced order, and in both trials, subjects were supported by a soft underlay (~10 cm thick foam), with a double thickness beneath the buttocks during the seated trial. For all trials, subject preparation, which included the donning of a water-perfusion garment (Cool Tubesuit, Med-Eng, Ottawa, Canada), occurred within an adjacent, air-conditioned laboratory (20–21 °C), after which subjects were wheeled into the chamber.

Once inside the chamber, passive heating was initiated. The perfusion garment was supplied with water at 40 °C (38-L, Grant GD120, Grant Instruments [Cambridge] Ltd., U.K.), whilst the feet were placed into a second water bath (40 °C). The aim was to induce a clamped, auditory canal temperature approximately 0.5 °C above each person's normothermic baseline (mild hyperthermia), but without contact between the legs and feet and the water bath. Once that thermal target was achieved, thermal clamping was initiated, which restricted subsequent auditory canal temperature changes to < 0.1 °C. This open-loop design ensured that both thermoeffector feedback and centrally mediated thermoeffector drive remained constant throughout the pressure treatments that followed.

For the supine trial, the water temperature of the foot bath was reduced to 37 °C, and the left foot was removed from the bath and dried, prior to the pressure application. For the seated trial, both feet were removed, supported on a foam-padded shelf and wrapped in towelling to reduce heat loss. These heating phases continued until auditory canal temperature, and each of the local sweat rates were elevated and stable for at least 5 min. The mean times to achieve those outcomes were 40 min (SD 8; supine trial) and 44 min (SD 4; sitting), after which the experimental (pressure) treatments were applied.

#### 2.2.3. Experimental treatments

Mechanical pressure was applied to the skin on three occasions during each trial, using a modified dynamometer (Sundoo, Model SN-500, S.I. Instruments, Hilton, SA, Australia) fitted with a flat and smooth, rectangular contact piece (10 $cm^2$). Pressure applications were not randomised because the sudomotor responses were evaluated from different spinal segments during the first pressure application (four sites: left and right chest and abdomen), relative to those measured during the second and third applications (four sites: left side of the forehead, and the left dorsal hand, chest and calf).

Two left-side skin sites were stimulated; the lateral chest wall (subaxillary, just above nipple level) and the heel of one foot. Those sites targeted the cutaneous sensory fields broadly classified as the fourth thoracic (T4-5) and the first sacral (S1) spinal segments, and were reported to be particularly sensitive to pressure (Takagi and Sakurai, 1950; Kawase, 1952; Ogawa et al., 1980). From the literature, it was apparent that the pressure threshold necessary to induce sudomotor responses was between 1.5 and 3 $N\,cm^{-2}$ (Takagi and Kobayasi, 1955; Ogawa et al., 1980; Tadaki et al., 1981). Therefore, to ensure an adequate stimulation, a pressure of 6 $N\,cm^{-2}$ was chosen for the lateral chest. For the heel, a pressure of 3 $N\,cm^{-2}$ was applied to simulate gravitational (heel) compression for a 76-kg male when standing. If any stimulus was painful or uncomfortable, its application was immediately terminated. Occasionally, some subjects experienced altered levels of arousal and microsleep. Data associated with painful pressure applications, microsleeps or technical difficulties were excluded, resulting in the sample sizes being < 12 for some pressure treatments.

In both postures, the duration of each pressure treatment was 5 min, to outlast latent sudomotor effects reported by Takagi and Sakurai (1950). After each pressure application, there followed a 5-min recovery period, which was extended if sweating had not returned to its thermally clamped, stable baseline. The stimulation sequence was: chest (T4-5), heel (S1) and chest (T4-5). Prior to the seated pressure treatments, participants were passively moved from a supine to a sitting position by gradually elevating the backrest of the bed. To minimise the impact of non-thermal stimuli, subjects were blindfolded, instructed not to talk and extraneous noises were eliminated.

#### 2.2.4. Sudomotor responses and tissues temperatures

Ventilated sweat capsules (3.16 $cm^2$) were again used to measure local (discharged) sweat rates (as above), the same tissue temperatures were measured and all sampling rates were identical. For this experiment, however, capsules were glued to the skin of seven sites: left side of the forehead (first-second thoracic segments [T1-2]), left dorsal hand (third-sixth thoracic segments [T3-6]), left and right medial chest

(adjacent the nipple; fourth-fifth thoracic segments [T4-5]), left and right lateral abdomen (just above the iliac crest; tenth-twelfth thoracic segments [T10-12]), and the left, dorsal surface of the calf (lateral head of gastrocnemius; tenth thoracic to second lumbar segments [T10-L2]). All capsules were continuously ventilated with room air to prevent moisture accumulation on the skin surface, but only four capsules were used for data collection at any one time (the active capsules), with their airflow regulated at 400 mL $min^{-1}$. During the first pressure application (lateral chest), sweating was measured from active capsules on the left and right surfaces of both the chest and abdomen. At the conclusion of that treatment, capsules positioned on the left side of the forehead, and also at the left dorsal hand, chest and calf were connected into the sweat-monitor system (replacing the right chest and both abdominal sites). They became the active capsules for the second and third pressure applications. Only one capsule (left chest) was used during all pressures applications.

#### 2.2.5. Data analysis

For this experiment, the literal meaning of the hemihidrotic reflex was used; increased sweating (Greek: *hidrosis*) on one half (Greek: *hemi*) of the body. More precisely, this implied a unilateral sudomotor enhancement from the contralateral skin surfaces, although the original descriptions of that reflex also included ipsilateral sudomotor inhibition (Kuno, 1934; Takagi and Sakurai, 1950). To evaluate the possibility of hemihidrotic pressure effects, local sweat rates from the ipsilateral and contralateral skin surfaces were analysed for the 5 min prior to, and for 5 min during, each pressure application, using 5-s rolling averages to minimise artefactual influences. The former were used as the pre-treatment sudomotor baselines, and for each individual, baseline means, as well as the upper and lower 95% confidence intervals, were determined for each of the active sweat sites. Those confidence intervals were used to identify sudomotor thresholds, beyond which sweating responses were deemed to have been influenced by each pressure treatment. Such conservative thresholds reduced the probability of erroneous response classifications. Four classes of sweating responses were identified: (1) a lasting excitatory response: local sweat rate crossed, and continued to remain above, the upper threshold for a total time of at least 30 s; (2) a persistent inhibitory response: local sweat rate crossed, and continuously remained below, the lower threshold for at least 30 s; (3) a null response: local sweating remained between the upper and lower thresholds; and (4) the cessation of local sweating: sweating declined to the basal [transepidermal] level of water loss (Taylor and Machado-Moreira, 2013).

To facilitate statistical comparisons between the sudomotor responses observed before, and then during, each pressure application, raw sweat rates (1 Hz) from each local site were integrated over the final 2.5 min of both the baseline and pressure-treatment periods. Differences between those sudomotor responses were evaluated using paired *t*-tests, with *alpha* set at the 0.05 level. Those data are reported as means and standard errors of those means ( ± ), unless stated otherwise (SD).

## 3. Results

### 3.1. Experiment one: sudomotor recruitment

For the dermatomal recruitment hypothesis to be supported, glandular activation should commence at the foot, and then progress upwards to the chest, hand and finally to the forehead. Alternatively, the recruitment progression could occur in the opposite direction. However, no participant displayed either recruitment pattern, and data from two divergent patterns are shown in Fig. 1. Moreover, there was no evidence that the segmental recruitment of sweating was anything other than a random occurrence ($P = 0.31$). Glandular activation was most commonly observed to commence at the foot ($N = 6$), although one person did not sweat at all from the foot (age 61 y), while another commenced foot sweating last (age 38 y). There is no reason to highlight those ages, as both subjects displayed normal sudomotor responses and were asymptomatic and habitually very active; note also that the person in Fig. 1A was 37 y. Nevertheless, the age of the remaining five subjects was 25.4 y (SD 2).

When individual recruitment patterns were analysed (Table 1), it was evident that, regardless of age, sudomotor recruitment was highly individualised. The average change in auditory canal temperature at the first onset of sweating was 0.29 °C (SD 0.16), and whilst that most commonly occurred at the foot, the other three sites were equally likely to commence sweating next (Table 1). The time delay between the first activated site and the appearance of sweat on the forehead was the longest (Table 1), but its activation was last on only one occasion. Moreover, only two individuals showed the same activation sequence: foot, hand, forehead and chest. Accordingly, we accept the first hypothesis that, in our hands, human eccrine sweating does not follow a dermatomal (caudal-to-rostral) recruitment pattern.

### 3.2. Experiment two: pressure-dependent sweating responses

#### 3.2.1. Thermal clamping

Since this experiment was performed under open-loop conditions, it was first necessary to establish the precision of the thermal clamp, and then to characterise the physiological baselines from which the pressure-dependent sweating responses were induced. Those thermal and cardiovascular data are summarised in Table 2, from which it may be concluded that the pre-treatment baselines were consistent with healthy, normothermic individuals. Furthermore, thermal clamping elevated and stabilised both the auditory canal and mean skin temperatures throughout subsequent pressure treatments. Therefore, it was assumed that the pressure-induced sudomotor responses were successfully isolated from variations in heat storage and thermoafferent feedback.

Prior to commencing those treatments, local sweat rates (baselines) during the supine trials averaged 0.81 mg $cm^{-2}.min^{-1}$ ( ± 0.25) at the left side of the chest, 0.84 mg $cm^{-2}.min^{-1}$ ( ± 0.21) for the right chest, 0.51 mg $cm^{-2}.min^{-1}$ ( ± 0.12) at the left side of the abdomen and 0.61 mg $cm^{-2}.min^{-1}$ ( ± 0.06) for the right abdomen. In the sitting trials, those sweat rates averaged 0.63 mg $cm^{-2}.min^{-1}$ ( ± 0.14; left chest), 0.74 mg $cm^{-2}.min^{-1}$ ( ± 0.12; right chest), 0.35 mg $cm^{-2}.min^{-1}$ ( ± 0.04; left abdomen) and 0.60 mg $cm^{-2}.min^{-1}$ ( ± 0.08; right abdomen). The pressure-induced sudomotor changes were then overlaid onto those stable, basal states.

#### 3.2.2. Sudomotor responses: qualitative outcomes

Fig. 2 displays variations in sweat rates for two individuals from the ipsilateral (left) chest site during the first pressure application (chest) of the supine trials. During those trials, eight subjects showed a clear inhibitory effect on sweating from that surface (Table 3; Fig. 2A), and for six individuals, that progressed to a total sudomotor cessation (Fig. 2B). For those participants, oscillations in the sweat traces, which are generally synchronised across skin regions (Fig. 1; van Beaumont et al., 1966; Taylor and Machado-Moreira, 2013), were no longer visible for the left chest. However, evidence of neither inhibition nor excitation was apparent from the left abdominal surface (Table 3). When an identical treatment was applied in the seated posture, that inhibitory effect occurred in more participants (Table 4).

On the contralateral skin surface (right chest), sweating increased in some subjects following pressure application, but that response was quite variable (Tables 3 and 4). Instead, those responses were more consistent with random variations. From the contralateral abdominal surface, however, an excitatory effect occurred more frequently in the supine than the seated posture (Tables 3 and 4). Thus, hemihidrosis did not occur when seated, but there was some evidence for elevated sweating in the supine posture.

When pressure was applied to the left side of the chest for the

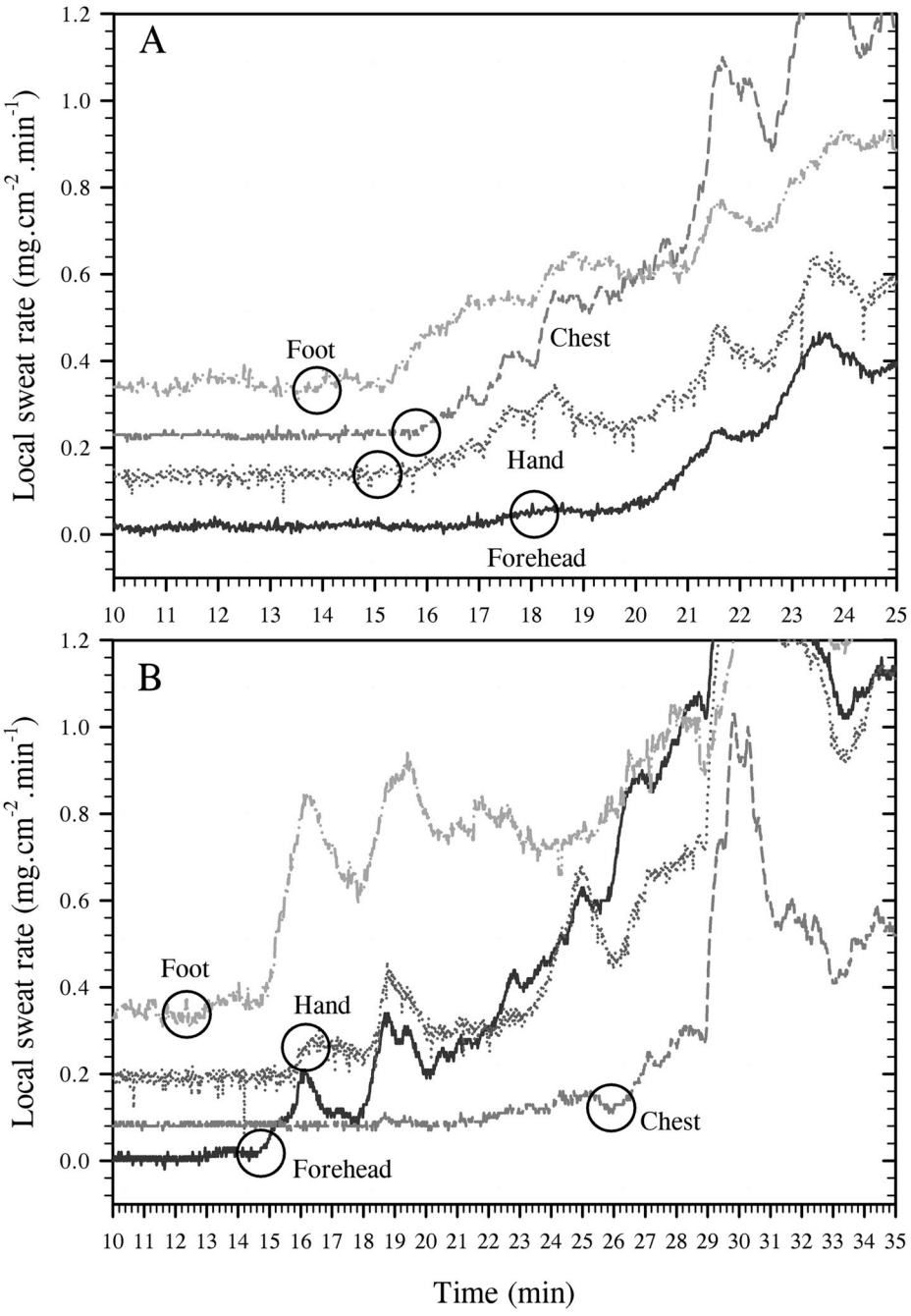


**Fig. 1.** Raw sweat traces for two participants from Experiment One. The local sweat rates have been artificially offset so that individual traces and differences in recruitment time (indicated by the circles) might become clearer. Non-zero secretion rates for the forehead reflect transepidermal water losses (Taylor and Machado-Moreira, 2013). The glandular activation sequence that would be consistent with the dermatomal recruitment hypothesis would be: foot, chest, hand and forehead. However, that pattern was not observed in any individual. For the subject in Fig. 1A (37 y), the activation sequence was: foot, hand, chest and forehead. In Fig. 1B (age 25 y), the sequence was: foot, forehead, hand and chest.

**Table 1**
Descriptive data for individual sudomotor recruitment patterns during Experiment One ($N$ = 8). Data in the first four rows show the number of occasions on which glandular activation occurred first, second, third or fourth within each site. The average activation position for each site, when averaged across subjects, reveals a generalised recruitment sequence. Finally, the average delays (with standard deviations in parenthesis) show the elapsed time between sweating first commencing (regardless of the site) and its subsequent appearance at each of the other three sites within each individual. No significant difference in the recruitment order was observed among sites ($P$ = 0.31).

| Variable | Foot | Chest | Hand | Forehead |
|---|---|---|---|---|
| Times this site was activated first | 6 | 2 | 0 | 0 |
| Times this site was activated second | 0 | 1 | 4 | 3 |
| Times this site was activated third | 0 | 2 | 2 | 4 |
| Times this site was activated fourth | 1 | 3 | 2 | 1 |
| Average activation position | 1.3 | 2.8 | 2.8 | 2.8 |
| Average delay from first onset (min) | 3.53 (8.80) | 8.82 (9.63) | 8.11 (9.25) | 9.85 (8.65) |

**Table 2**
Thermal and cardiovascular baselines for both the supine and seated trials of Experiment Two ($N = 13$). Data were collected before (normothermia) and during each of three, left-side pressure applications (5 min), which occurred in the presence of isothermal clamping. Data are means with standard errors of the means in parenthesis.

| Trial | Phase | Tissue temperatures (°C) | | | Heart rate |
|---|---|---|---|---|---|
| | | Auditory canal | Mean skin | Mean body | (beats.min$^{-1}$) |
| Supine | Normothermia | 36.7 (0.1) | 32.1 (0.1) | 35.8 (0.1) | 61 (2) |
| | Chest pressure | 37.1 (0.1) | 36.3 (0.1) | 37.0 (0.1) | 68 (3) |
| | Foot pressure | 37.1 (0.1) | 36.3 (0.1) | 37.0 (0.1) | 68 (3) |
| | Chest pressure | 37.1 (0.1) | 36.2 (0.1) | 37.0 (0.1) | 67 (3) |
| Seated | Normothermia | 36.7 (0.1) | 32.0 (0.1) | 35.8 (0.1) | 61 (2) |
| | Chest pressure | 37.1 (0.1) | 35.8 (0.1) | 36.8 (0.1) | 74 (3) |
| | Foot pressure | 37.1 (0.1) | 35.7 (0.1) | 36.8 (0.1) | 74 (3) |
| | Chest pressure | 37.1 (0.1) | 35.8 (0.1) | 36.8 (0.0) | 75 (3) |

second time, the ipsilateral sudomotor responses were measured from the forehead, medial chest, dorsal hand and calf. That permitted the segmental impact of the treatment to be more fully explored. Inhibitory influences were seen from all sites except the calf during both postures (Tables 3 and 4). These observations lend support to the possibility that sudomotor inhibition occurred within some, but not across all, spinal segments. During the application of heel pressure, those same responses were measured. When supine, most participants experienced greater sweating across all sites (Table 3), with a much less convincing, albeit similar, response observed when seated (Table 4).

### 3.2.3. *Sudomotor responses: quantitative outcomes*

Initially, the between-postures, baseline sweat rates were evaluated (Fig. 3A and B), with only the calf revealing a significant difference between trials, with its local sweat rate being lower when seated ($P < 0.05$). Statistical comparisons were then performed between the baseline and pressure-induced sudomotor responses of both trials.

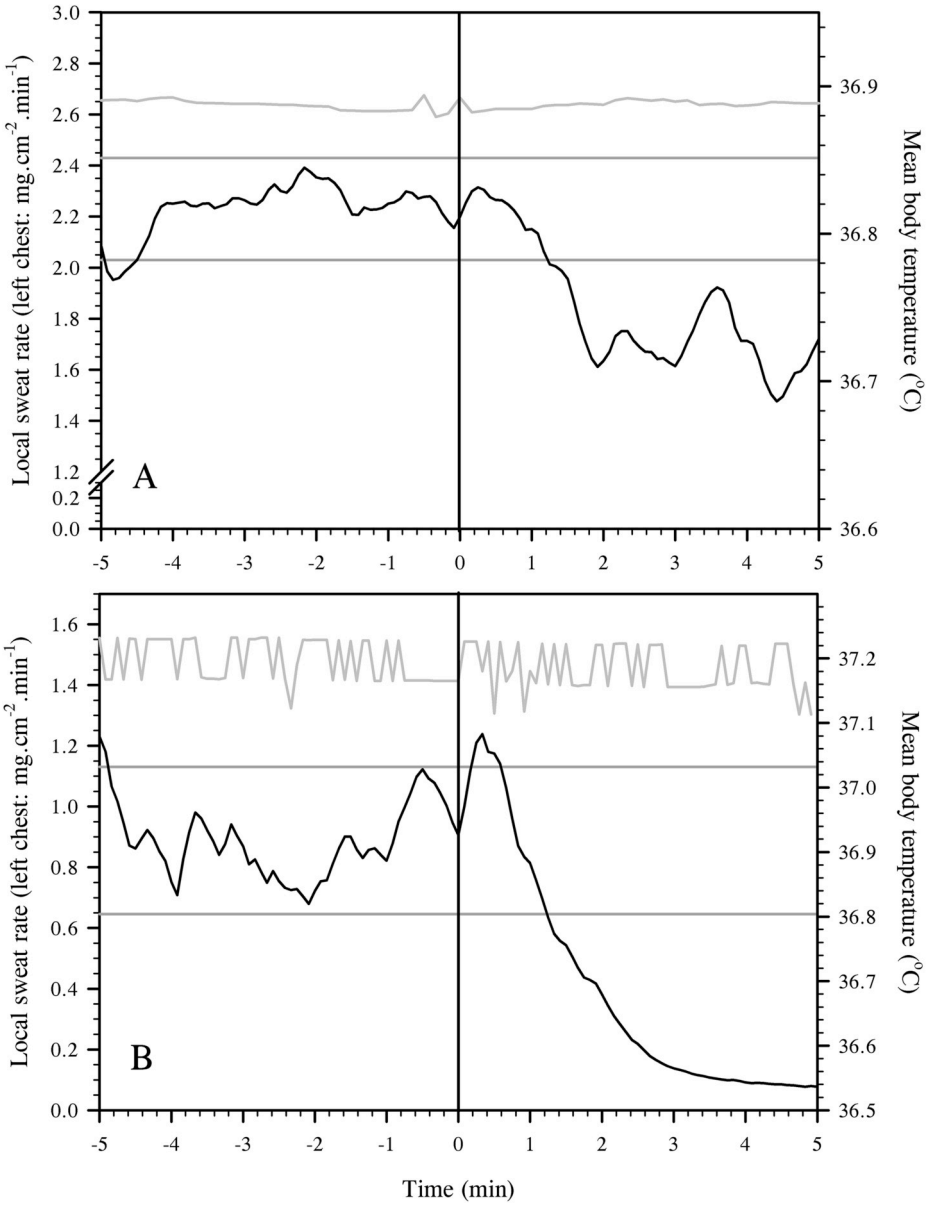


**Fig. 2.** Sudomotor responses (black traces: 5-s, rolling averages) for two individuals, measured from the medial aspect of the ipsilateral (left) chest for 5 min before (−5 to 0 min), and then over 5 min during pressure application (0–5 min) within the same spinal segment (6 N cm$^{-2}$; fourth thoracic dermatome). Both examples illustrate sudomotor inhibition, with the second progressing to the cessation of sweating. The horizontal (dark grey) lines define the upper and lower 95% confidence intervals for each participant. Also shown are mean body temperature data (grey traces).

**Table 3**
Sudomotor response summaries during sequential pressure applications to the left, lateral chest (6 N cm$^{-2}$; fourth thoracic dermatome), left heel (3 N cm$^{-2}$; first sacral dermatome) and the left chest of heated and thermally clamped, supine participants. Numbers are counters for four possible response classifications (see methods). Parenthetical counts identify responses lasting < 30 s. Since sudomotor inhibition resulted in either the partial or total cessation of sweating, then some responses are counted in both rows. Sample sizes varied due to data exclusion criteria (pain, altered arousal state).

| Classification | Pressure to the fourth thoracic dermatome (left) | | | |
|---|---|---|---|---|
| | Left chest ($N$ = 10) | Right chest ($N$ = 10) | Left abdomen ($N$ = 10) | Right abdomen ($N$ = 10) |
| Excitatory: upper threshold | 1 (2) | 4 (1) | 3 (3) | 6 (2) |
| Inhibitory: lower threshold | 8 (0) | 3 (0) | 4 (1) | 1 (1) |
| Null response | 1 | 3 | 2 | 1 |
| Total cessation of sweating | 6 | 0 | 0 | 0 |
| | Pressure to the first sacral dermatome (left) | | | |
| | Left chest ($N$ = 11) | Left forehead ($N$ = 11) | Left hand ($N$ = 11) | Left calf ($N$ = 11) |
| Excitatory: upper threshold | 7 (1) | 5 (2) | 8 (0) | 4 (2) |
| Inhibitory: lower threshold | 1 (1) | 3 (1) | 2 (0) | 1 (2) |
| Null response | 3 | 3 | 2 | 5 |
| Total cessation of sweating | 0 | 0 | 0 | 0 |
| | Pressure to the fourth thoracic dermatome (left) | | | |
| | Left chest ($N$ = 11) | Left forehead ($N$ = 11) | Left hand ($N$ = 11) | Left calf ($N$ = 11) |
| Excitatory: upper threshold | 1 (1) | 1 (1) | 2 (0) | 3 (2) |
| Inhibitory: lower threshold | 9 (0) | 8 (0) | 9 (0) | 1 (0) |
| Null response | 0 | 2 | 0 | 5 |
| Total cessation of sweating | 7 | 0 | 7 | 0 |

Those data are summarised in Fig. 3 for pressures applied to the lateral chest during both postures, revealing the following outcomes. Firstly, and notwithstanding the preceding descriptive outcomes (Tables 3 and 4), those lateral pressures did not result in hemihidrosis (right-side sudomotor enhancement) in either posture ($P > 0.05$; Fig. 3A and B). Secondly, pressure application significantly inhibited ipsilateral sweating at the same segmental level (chest) during both postures ($P < 0.05$; Fig. 3A and B). Significant inhibition was also seen at the forehead and hand when supine ($P < 0.05$; Fig. 3A), but only at the chest and hand when seated ($P < 0.05$; Fig. 3B). Thirdly, neither the ipsilateral abdominal nor the ipsilateral calf sudomotor responses differed significantly during either posture when chest pressure was applied ($P > 0.05$; Fig. 3A and B).

Prior to pressure being applied to the left heel, significantly lower sweating was observed at the calf ($P < 0.05$; Fig. 4B), but none of the other between-postures, baseline sweat rates was different ($P > 0.05$). When analysed during heel pressure, there were no significant left-side sweating responses when participants were seated ($P > 0.05$; Fig. 4B).

**Table 4**
Sudomotor responses accompanying pressure applied to the left, lateral chest (6 N cm$^{-2}$; fourth thoracic dermatome), the left heel (3 N cm$^{-2}$; first sacral dermatome) and the left chest of heated and thermally clamped, seated subjects. Numbers are counters for four sudomotor response classifications (see methods). Parenthetical values identify responses lasting < 30 s. Since an inhibitory effect can produce either partial or total sudomotor cessation, then those outcomes are included in both rows. Sample sizes varied due to data exclusion (pain, altered arousal state).

| Classification | Pressure to the fourth thoracic dermatome (left) | | | |
|---|---|---|---|---|
| | Left chest ($N$ = 11) | Right chest ($N$ = 11) | Left abdomen ($N$ = 11) | Right abdomen ($N$ = 11) |
| Excitatory: upper threshold | 1 (4) | 4 (1) | 7 (1) | 6 (1) |
| Inhibitory: lower threshold | 11 (0) | 6 (1) | 3 (2) | 3 (1) |
| Null response | 0 | 2 | 1 | 2 |
| Total cessation of sweating | 7 | 0 | 0 | 0 |
| | Pressure to the first sacral dermatome (left) | | | |
| | Left chest ($N$ = 10) | Left forehead ($N$ = 11) | Left hand ($N$ = 11) | Left calf ($N$ = 11) |
| Excitatory: upper threshold | 6 (2) | 5 (1) | 6 (2) | 4 (3) |
| Inhibitory: lower threshold | 3 (2) | 3 (1) | 4 (0) | 4 (3) |
| Null response | 0 | 2 | 1 | 1 |
| Total cessation of sweating | 0 | 0 | 0 | 0 |
| | Pressure to the fourth thoracic dermatome (left) | | | |
| | Left chest ($N$ = 11) | Left forehead ($N$ = 12) | Left hand ($N$ = 12) | Left calf ($N$ = 12) |
| Excitatory: upper threshold | 1 (0) | 1 (1) | 3 (1) | 7 (0) |
| Inhibitory: lower threshold | 11 (0) | 10 (0) | 11 (0) | 2 (1) |
| Null response | 0 | 1 | 0 | 2 |
| Total cessation of sweating | 7 | 1 | 5 | 0 |

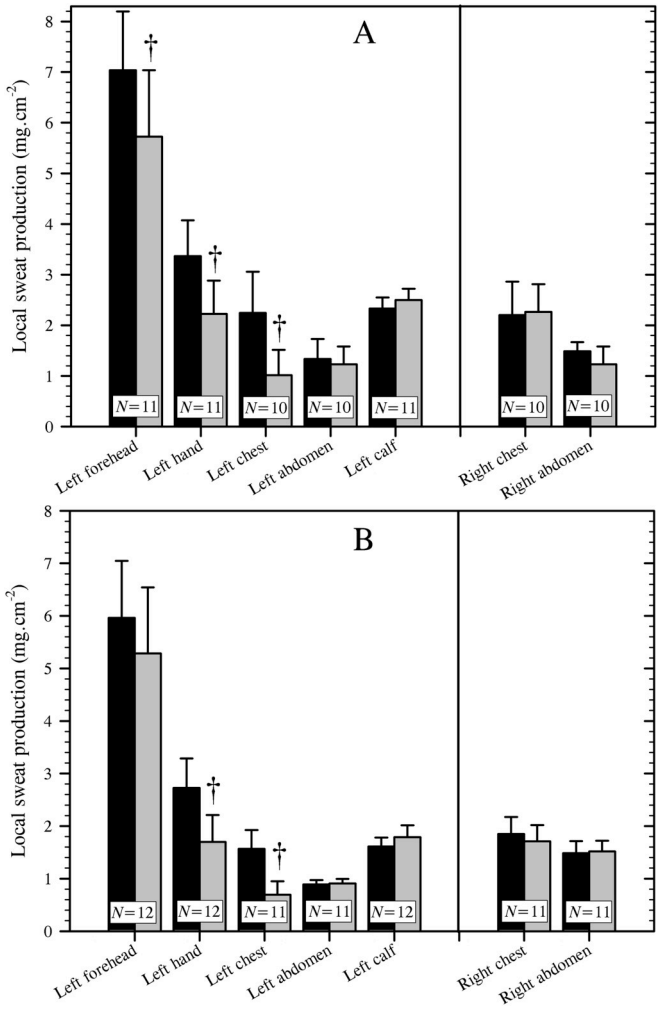


**Fig. 3.** Local sudomotor responses immediately preceding (baseline: black bars), and during (grey bars), a 5-min pressure application (6 N cm$^{-2}$) to the left side of the chest (fourth thoracic dermatome) during both supine (Fig. 3A) and seated postures (Fig. 3B). The vertical lines separate the left- from the right-side sweat capsule locations. Data are total sweat productions computed by integrating sweat rates with respect to time over the last 2.5-min period of each phase, and are shown as means with standard errors of the means. Sample sizes are indicated for each location, and significant differences between the baseline and pressure-related sweat secretions are indicated using symbols (†; $P < 0.05$).

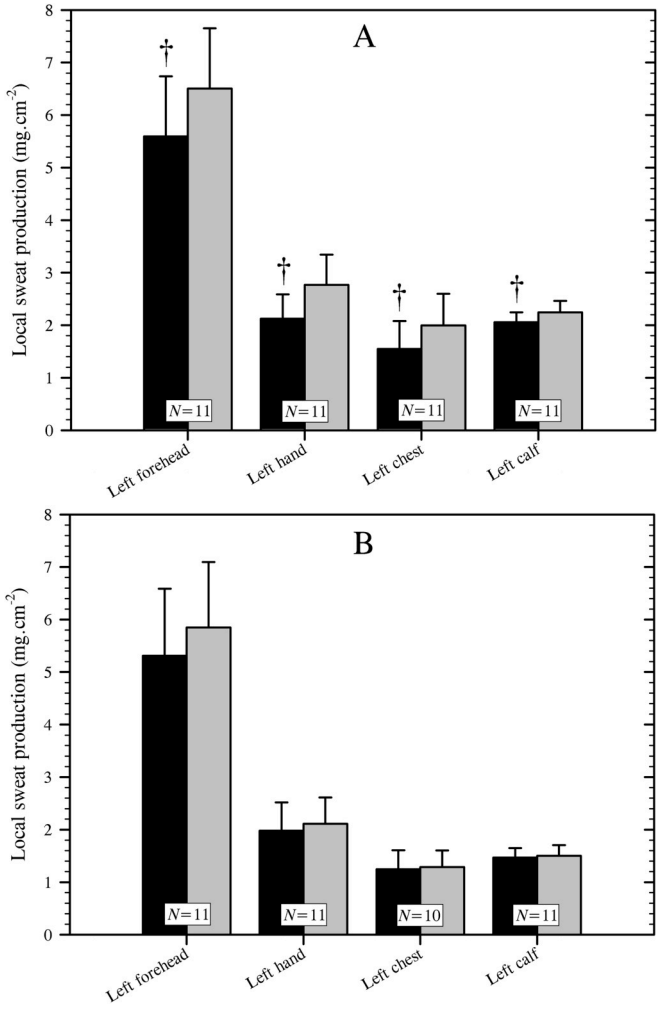


**Fig. 4.** Sudomotor responses preceding (baseline: black bars), and during (grey bars), a 5-min pressure application (3 N cm$^{-2}$) to the heel of the left foot (first sacral dermatome) during supine (Fig. 4A) and seated postures (Fig. 4B). Data are means, plus standard errors of the means, for integrated (total) sweat productions over the last 2.5-min period of each phase. Sample sizes and significant differences are shown (†; $P < 0.05$), but none of differences between the baseline and pressure-induced sweat secretions were significant in the seated posture ($P > 0.05$).

Conversely, when supine, significant excitatory effects were seen at every site ($P < 0.05$; Fig. 4A). That is, unlike the inhibited, or null responses, observed when chest pressure was applied to supine subjects (Fig. 3A), sweating was now elevated across all spinal segments investigated.

## 4. Discussion

From these experiments, four principal outcomes have arisen. Firstly, no evidence was found to support the dermatomal (caudal-to-rostral) recruitment hypothesis (Hertzman et al., 1952). Indeed, the current observations are more consistent with individualised and random patterns of regional sudomotor activation (List and Peet, 1938), with significant time delays between the sequential activation of sites within all individuals (Table 1). That intra-individual variability demonstrates that simultaneous regional activation, as proposed by Kuno (1934, 1956), was also not observed. Secondly, locally applied pressure to the chest of mildly hyperthermic individuals did not elicit a significant, unilateral sudomotor enhancement from the contralateral skin surfaces (Fig. 3). That is, under the current, thermally clamped experimental conditions, and as defined within this experiment, hemihidrosis did not occur. Thirdly, those same pressure applications exerted a significant inhibitory influence on sweat secretion from some, but not all, ipsilateral skin surfaces when participants were supine (Fig. 3A). Finally, when pressure was applied to the heel of supine, but not seated, participants, a significant sudomotor enhancement was observed across the ipsilateral sites (Fig. 4A).

It was not surprising that the dermatomal recruitment hypothesis was not supported, as, following years of indirect observation, that recruitment pattern was observed infrequently. Moreover, the original observations arose from methods ill-suited to that purpose (Hertzman et al., 1952), with Randall and Hertzman (1953) and McCook et al. (1965) presenting supporting data from only four individuals (two each). It was surprising, however, to see significant time delays among skin regions, for they could not be explained by delays in neural transmission.

From the current observations, it appears that regional sweat-recruitment sequences represent random and apparently individualised phenomena (Table 1), but which mechanisms might explain those onset

delays and differences in activation sequence? In our hands, simultaneous recruitment did not occur. However, those sudomotor data were obtained as discharged sweat rates, and it is known that precursor sweat production precedes its discharge onto the skin surface (Darrow, 1934; Thomas and Korr, 1957; Bullard, 1971). Therefore, the precursor and discharged sweat thresholds are not equivalent (Machado-Moreira et al., 2015), with regional differences in the appearance of discharged sweat possibly reflecting variations in neurotransmitter sensitivity, precursor sweat production or ductal reabsorption, even if those regions were simultaneously activated. That possibility is favoured, but requires further investigation.

Alternatively, each region may be independently controlled, with a range of unique critical temperatures (thresholds). It is improbable, but not impossible, that such specific regional controllers and thresholds exist at the hypothalamic level. A third possibility is that variations in neural activation may result from downstream inhibitory influences, with their staged removal accompanying elevations in body temperatures. It is beyond the scope of this investigation, and the tools available to the authors, to expand upon that speculation. However, it is hoped that others might find these observations worthy of verification and further investigation.

Under the thermally clamped conditions of experiment two, local pressure applications to the chest did not elicit either contralateral excitatory (hemihidrotic) or inhibitory sudomotor influences in either posture (Fig. 3). That outcome is consistent with the observations of Watkins (1956) and White et al. (1995). However, it is at odds with the evidence of Kuno (1934), Takagi and Sakurai (1950) and Kawase (1952), with Ferris (1960) reporting postural hemihidrosis in supine subjects, but not during lateral pressure applications.

Previous experiments reporting hemihidrosis were mostly performed without thermal clamping. The exception was Wilsmore et al. (1999), although that study involved a postural change and not pressure application. We therefore interpret the current observations to imply that the reported enhancement of contralateral sweating was a thermoregulatory compensation, as suggested by Takagi (1960). That is, since a concurrent, ipsilateral inhibition was generally reported, then increased heat storage, unless countered by greater evaporation elsewhere, would increase thermoafferent feedback, and further stimulate sweating from sites that were not under pressure-induced inhibitory influences. Indeed, both van Beaumont et al. (1966) and Ogawa et al. (1979) reported an elevation of the ipsilateral skin temperatures accompanying reduced local and elevated contralateral sweating.

Let us now consider those local inhibitory (hypohidrotic) influences (Fig. 3). Many others have similarly reported such a response (Kuno, 1934; Takagi and Sakurai, 1950; Kawase, 1952; Watkins, 1956; van Beaumont et al., 1966; Ogawa et al., 1979; White et al., 1995), although none used thermal clamping, and most were descriptive rather than statistically based. Such inhibition is believed to result from a spinal reflex (Ogawa et al., 1980; Shen et al., 1986; Okagawa et al., 2003; Wilsmore, 2007), rather than being hypothalamically mediated, since the latter would elicit similar responses from all skin surfaces. The appearance of a gradual reduction in sweating in Fig. 2B (1–3 min) does not signify a progressive inhibition. Instead, it merely reflects time delays accompanying skin drying and the flushing of residual water vapour, while the lack of oscillations within the sweat trace (beyond 1 min) is indeed consistent with an inhibitory neural mechanism.

Such a spinal reflex would commence with cutaneous mechanoreceptor activation, with the resulting sensory feedback entering the spinal cord via the dorsal root ganglia, and ascending ipsilaterally via the dorsal columns (Purves et al., 2008). It is believed that segmental variations in the pressure-dependent sudomotor inhibition are mediated by interactions between those afferents and the descending sympathetic neurones (Ogawa et al., 1980; Shen et al., 1986; Wilsmore, 2007). The current results support that description, with sudomotor inhibition observed within the same spinal segment (chest), and also within segments above (forehead and hand), but not below (abdomen and calf; Fig. 3A and B), the level of stimulation. Nonetheless, the possibility of nociceptor participation cannot be excluded, even though instances of reported pain were excluded.

Since sweating was measured from only five spinal segments, and since responses varied within and between postures, then caution is required regarding their interpretation, even though those data represent the most complete analysis yet performed. It is suggested that the inhibitory sudomotor effects are restricted to spinal segments upstream of the pressure application, matching the path followed by the mechanoreceptor feedback. Because those afferents ascend ipsilaterally, whilst the sudomotor preganglionic neurones descend bilaterally (Purves et al., 2008), and because there is clear evidence of an upstream, ipsilateral sudomotor inhibition (Fig. 3), then it is suggested that this pressure-induced spinal reflex occurs within the spinal cord, affecting only the spinal segments above the pressure application. That is, it appears to be a segment-specific reflex. Evidence for interneurones that connect dorsal root ganglia with preganglionic neurones exists within primates (Petras and Cummings, 1972), so this mechanistic interpretation is founded upon neuroanatomical observations. However, we attribute the lack of unanimous statistical support for this interpretation (Fig. 3), in the presence of consistent sudomotor reductions (Table 3 and Fig. 3), to the inherent, and sometimes artefactual, variability of these sweating responses.

When pressure was applied to the left heel of supine participants, a significant, excitatory sudomotor influence was apparent from all ipsilateral sites (Fig. 4A). The aim of that treatment was to mimic area-specific cutaneous pressures that accompany standing ($3\ N\ cm^{-2}$), but without actually changing posture or introducing other non-thermal influences. Unlike the pressure-induced inhibition associated with chest pressure (Fig. 3A), an apparent whole-body excitatory influence resulted during the supine trial (Fig. 4A), which differs from the response seen during standing (Shen et al., 1986). Why was that so?

In considering that question, we highlight several between-trial differences in the current experiment. Firstly, the absolute pressures were different: 6 (chest) versus $3\ N\ cm^{-2}$ (heel), with the latter being much lower than encountered during either walking or running. Secondly, regional differences in mechanoreceptor densities and sensitivities may exist. Thirdly, since humans are an endurance-dependent, bipedal species that evolved in the tropics (Ruben, 1995; Lieberman, 2015), then a spinal reflex that inhibited sweating with each load-bearing stride would be counter-productive, and unlikely to survive natural selection. Thus, Figs. 3A and 4A are not necessarily in conflict, but may reflect different neural interactions. For instance, we have previously described elevations in upper-body sweating (forehead, dorsal hand and abdomen) and expulsion frequency (forehead) when thermally clamped subjects were passively moved from a supine to a seated posture (Wilsmore et al., 1999; Wilsmore, 2007).

In the supine posture, enhanced sweating is solely attributed to the unilateral pressure application (heel), the mechanism of which must differ from that accompanying the less-familiar chest pressure. When seated, the evenly distributed dorsal pressure (when supine) changed so the bulk of the body mass was borne through the buttocks (sacral segments one-three). If that local, bilateral pressure acted in the same segmental manner as chest pressure, then upstream sudomotor inhibition might have resulted, possibly suppressing the sudorific affect of the subsequent heel pressure.

## 5. Conclusion

The need to revisit past, and sometimes entrenched, teachings is often inadequately appreciated. Nevertheless, scientific progress relies not just upon novel mechanistic research, but upon constantly challenging ideas, and, when appropriate, offering new interpretations. As a consequence of these experiments, two long-held tenets of thermal physiology have been modified. In the first instance, the sequential secretion of discharged eccrine sweat across the body surface can no longer be regarded as following either a sympathetic dermatomal pattern or a simultaneous appearance. Secondly, the general acceptance of

the hemihidrotic reflex can no longer be sustained. Thus, those widely accepted teachings seem to have reached their expiry dates, and are now untenable. We must now seek to explain why significant time delays in sudomotor recruitment occur among skin regions. We also need to increase our understanding of the neural mechanisms that modulate unilateral, pressure-induced sudomotor inhibition.

## Conflicts of interest

There are no conflicts of interest.

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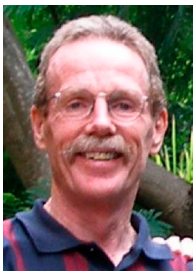

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