การประชุมเชิงปฏิบัติการ

เรื่องสมดุลอุณหภูมิกาย

บทบาทและการตอบสนองของระบบไหลเวียนโลหิตและระบบหายใจ

ณ ห้องประชุม 1 อาคารวิชาการ มหาวิทยาลัยเทคโนโลยีสุรนารี

วันที่ 23 - 25 เมษายน 2546

จัดโดย สาขาวิชาสรีรวิทยา สำนักวิชาวิทยาศาสตร์

มหาวิทยาลัยเทคโนโลยีสุรนารี

การประชุมเชิงปฏิบัติการเรื่องสมดุลอุณหภูมิกาย

บทบาทและการตอบสนองของระบบไหลเวียนโลหิตและระบบหายใจ

ISBN: 974-533-223-2

จัดโดย สาขาวิชาสรีรวิทยา สำนักวิชาวิทยาศาสตร์ มหาวิทยาลัยเทคโนโลยีสุรนารี

การประชุมเชิงปฏิบัติการ

เรื่อง "สมดุลอุณหภูมิกาย : บทบาทและการตอบสนองของระบบไหลเวียนโลหิตและระบบหายใจ"

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หลักการและเหตุผล

ประเทศไทยเป็นประเทศที่อยู่ใกล้เส้นศูนย์สูตรและมีภูมิอากาศค่อนข้างร้อน โดยเฉพาะในภาคตะวันออกเฉียงเหนือจะพบว่าในช่วงฤดูร้อน (ประมาณเดือนเมษายนถึงพฤษภาคม) อุณหภูมิในอากาศสามารถเพิ่มขึ้นได้ถึง 41 – 42 องศาเซลเซียส และมีความชื้นสัมพัทธ์ประมาณ 75% (ข้อมูลจากกรมอุตุนิยมวิทยาตั้งแต่ปี พ.ศ. 2541 – 2543) และเราพบว่าจะมีประชากรจำนวนหนึ่งที่ต้องตายจากการที่อุณหภูมิในบรรยากาศเพิ่มสูงขึ้นมากกว่าอุณหภูมิแกนในร่างกายเกือบทุกปี แม้ว่าในทางทฤษฎีนั้นเราจะทราบคร่าว ๆ ว่าถ้าอุณหภูมิในบรรยากาศเพิ่มสูงขึ้น ศูนย์ควบคุมอุณหภูมิในร่างกายที่อยู่ในฮัยโปทาลามัส (Hypothalamus) จะส่งสัญญาณประสาทไปยังศูนย์ควบคุมที่ช่วยในการระบายความร้อนในลักษณะการนำความร้อน การพาความร้อน การแผ่รังสีและการระเหยในรูปของเหงื่อ รวมทั้งการหายใจเพิ่มขึ้นด้วย ตลอดจนหลอดเลือดในร่างกายจะมีการขยายตัวเพิ่มขึ้น เพื่อทำให้ความร้อนที่ผลิตขึ้นกับการสูญเสียความร้อนมีความสมดุลกันหรือเท่ากัน แต่อย่างไรก็ตามเราก็ยังไม่ทราบถึงกลไกในการระบายความร้อนที่เกิดขึ้นที่ผิวหนังนั้นจะเกิดขึ้นที่ตำแหน่งใดของร่างกายมากที่สุด และการตอบสนองในแต่ละแห่งนั้นจะเกิดขึ้นพร้อมกันหรือไม่อย่างไร ตลอดจนกลไกในการตอบสนองที่แท้จริงของระบบหัวใจและการหายใจนั้น ดำเนินขั้นตอนอย่างไร ซึ่งในประเทศไทยนั้นยังมีการศึกษากันน้อย ส่วนใหญ่ความรู้ความเข้าใจก็ได้จากการอ่านตำราและวารสารจากต่างประเทศ ดังนั้นคณะผู้จัดทำการประชุมครั้งนี้จึงเห็นถึงความสำคัญในการจัดประชุมเชิงปฏิบัติการ เพื่อจะได้ความรู้ทางด้าน

1. การควบคุมอุณหภูมิของร่างกายเมื่อได้รับความร้อน
2. อัตราการหลั่งของเหงื่อ
3. อันตรายที่เกิดขึ้นเมื่อร่างกายไม่สามาถปรับตัวได้

รวมทั้งได้มีการฝึกปฏิบัติเทคนิคต่าง ๆ ในการวัดพารามิเตอร์ทั้งในระบบการไหลเวียนโลหิตและระบบหายใจ

ดังนั้น ทางสาขาวิชาสรีรวิทยา สำนักวิชาวิทยาศาสตร์ จึงได้จัดการประชุมเชิงปฏิบัติการในครั้งนี้ โดยเชิญ Assoc. Prof. Dr. Nigel Taylor จากมหาวิทยาลัย Wollongong ประเทศออสเตรเลีย ซึ่งเป็นผู้เชี่ยวชาญด้านการควบคุมสมดุลอุณหภูมิกายมาร่วมการประชุมเชิงปฏิบัติการแก่คณาจารย์และผู้สนใจในสถาบันการศึกษาทุกแห่งและองค์กรต่างๆทั้งในภาครัฐและเอกชน

วัตถุประสงค์

1. เพื่อเพิ่มความรู้ ความเข้าใจทางด้านสมดุลอุณหภูมิของร่างกายให้มากขึ้น
2. เพื่อทราบถึงกลไกในการปรับตัวของสมดุลอุณหภูมิที่มีผลต่อระบบไหลเวียนโลหิตและระบบหายใจ
3. เพื่อเป็นการฝึกเทคนิคในการทดสอบระบบไหลเวียนโลหิตและระบบหายใจให้มีความเชี่ยวชาญมากยิ่งขึ้น

ประโยชน์ที่คาดว่าจะได้รับ

1. ผู้ร่วมประชุมเชิงปฏิบัติการสามารถนำความรู้ ความเข้าใจ และเทคนิคต่าง ๆ ทางด้านสมดุลอุณหภูมิของร่างกายไปใช้กับการเรียน การสอน และงานวิจัยได้
2. เพื่อพัฒนาความร่วมมือทางด้านวิชาการและงานวิจัยร่วมกับมหาวิทยาลัยต่างๆภายในประเทศ ให้มีความแข็งแกร่งเพิ่มขึ้น
3. เพื่อเผยแพร่ความรู้ทางด้านงานวิชาการอันจะเป็นแนวทางไปสู่การวิจัยให้กับสมาชิก คณาจารย์ และผู้สนใจในสถาบันการศึกษารวมทั้งชุมชน

<u>คณะกรรมการที่ปรึกษา</u>

1. ผู้ช่วยศาสตราจารย์ ดร.ทวี เลิศปัญญาวิทย์	ที่ปรึกษา มหาวิทยาลัยเทคโนโลยีสุรนารี
2. แพทย์หญิง อาภรณ์ ปราบริปูตลุง	ที่ปรึกษา มหาวิทยาลัยเทคโนโลยีสุรนารี
3. ศาสตราจารย์ ดร.ธีรยุทธ กลิ่นสุคนธ์	ที่ปรึกษา มหาวิทยาลัยรังสิต
4. รองศาสตราจารย์ ดร.ไถ้ออน ชินธเนศ	ที่ปรึกษา มหาวิทยาลัยมหิดล
5. ผู้ช่วยศาสตราจารย์ ดร.ปัญญา ไข่มุก	ที่ปรึกษา มหาวิทยาลัยมหิดล

<u>คณะกรรมการดำเนินการ</u>

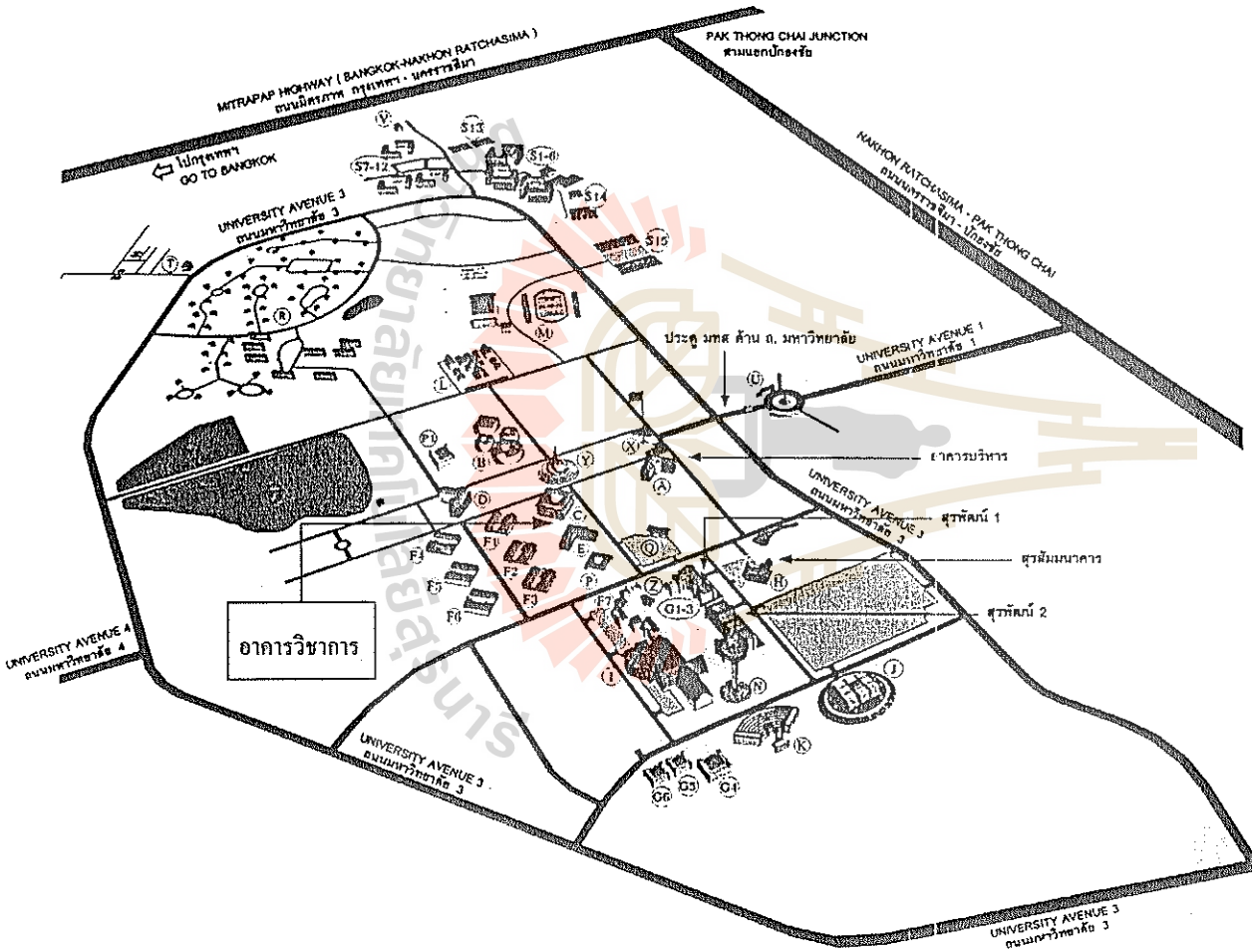
1. รองศาสตราจารย์ ดร.ประสาท สืบค้า	ประธานกรรมการ มหาวิทยาลัยเทคโนโลยีสุรนารี
2. ผู้ช่วยศาสตราจารย์ ดร.พาณี วรรณนิธิกุล	กรรมการ มหาวิทยาลัยเทคโนโลยีสุรนารี
3. ดร.ราเชนทร์ โกศัลวิตร	กรรมการ มหาวิทยาลัยเทคโนโลยีสุรนารี
4. ดร.วารี วิดจายา	กรรมการและเลขานุการ มหาวิทยาลัยเทคโนโลยีสุรนารี
5. นางกานดา สังข์สาย	ผู้ช่วยเลขานุการ มหาวิทยาลัยเทคโนโลยีสุรนารี
6. นางปลื้มจิตร กังตระกูล	ผู้ช่วยเลขานุการ มหาวิทยาลัยเทคโนโลยีสุรนารี

วัน เวลาและสถานที่

วันที่ 23-25 เมษายน 2546 ณ ห้องประชุม 1 อาคารวิชาการ และศูนย์เครื่องมือวิทยาศาสตร์และเทคโนโลยี มหาวิทยาลัยเทคโนโลยีสุรนารี

สถานที่ติดต่อ

ดร.วารี วิดจายา
สำนักวิชาวิทยาศาสตร์
มหาวิทยาลัยเทคโนโลยีสุรนารี
111 ถนนมหาวิทยาลัย ตำบลสุรนารี
อำเภอเมือง จ.นครราชสีมา 30000
โทรศัพท์ : 044-224630, 044-224633, 044-223305, 044-224187-8
โทรสาร : 044-224185
E-mail : waree@ccs.sut.ac.th



Ⓐ อาคารบริหาร / ADMINISTRATIVE BUILDING
Ⓑ กลุ่มอาคารเรียนรวม / CLASSROOM BUILDING COMPLEX
Ⓒ อาคารวิชาการ / ACADEMIC BUILDING
Ⓓ อาคารบรรณสาร / LIBRARY BUILDING
Ⓔ อาคารวิจัย / RESEARCH BUILDING
Ⓕ กลุ่มอาคารเครื่องมือ (F1-F7)
○ EQUIPMENT BUILDING COMPLEX (F1-F7)
Ⓖ กลุ่มอาคารสุรพัฒน์ 1-6 / TECHNOPOLIS ZONE (G1-G6)
Ⓗ สุรสัมมนาคาร / SURASAMMANAKHAN
Ⓘ ศูนย์ปฏิบัติการวิจัยเครื่องกำเนิดแสงซินโครตรอนแห่งชาติ
○ NATIONAL SYNCHROTRON RESEARCH CENTER
Ⓙ อาคารกาญจนาภิเษก / GOLDEN JUBILEE PAVILLION
Ⓚ อาคารสุรนิทัศน์ / SURANITAT AMPHITHEATER
Ⓛ กลุ่มอาคารกิจการนักศึกษา
○ STUDENT ACTIVITIES BUILDING COMPLEX
Ⓜ สถานกีฬาและสุขภาพ / SPORTS AND HEALTH CENTER
Ⓝ หอสุรนภา / SURANAPA SYMBOLIC TOWER
Ⓟ โรงอาหารกลาง / CANTEEN
Ⓟ1 โรงอาหารอาคารเรียนรวม / CANTEEN
Ⓠ อาคารขนส่ง / TRANSPORT STATION
Ⓡ กลุ่มอาคารบ้านพัก เรือนพักบุคลากร
○ STAFFS RESIDENCES ZONE
Ⓢ กลุ่มอาคารหอพักนักศึกษา (S1-S15)
○ STUDENTS DORMITORIES ZONE (S1-S15)
Ⓣ ฟาร์มมหาวิทยาลัย / UNIVERSITY FARM
Ⓤ สำนักงานรักษาความปลอดภัย / GUARDHOUSE
Ⓥ หน่วยผลิตน้ำประปา / WATER SUPPLY UNIT
Ⓦ อ่างสุระ 1, 2 / RESERVOIRS 1, 2
Ⓧ เสาธง / FLAG POLE
Ⓨ สัญลักษณ์สถาน / SUT SHRINE
Ⓩ บ้านไทยไฮเทค / THE THAI HIGH TECHNOLOGY HOUSE

แผนผังบริเวณมหาวิทยาลัยเทคโนโลยีสุรนารี

MAP OF SURANAREE UNIVERSITY OF TECHNOLOGY

การประชุมเชิงปฏิบัติการ

เรื่อง "สมดุลอุณหภูมิกาย : บทบาทและการตอบสนองของระบบไหลเวียนโลหิตและ ระบบหายใจ"

ณ ห้องประชุม 1 อาคารวิชาการ มหาวิทยาลัยเทคโนโลยีสุรนารี

วันที่ 23-25 เมษายน 2546

จัดโดย สาขาวิชาสรีรวิทยา สำนักวิชาวิทยาศาสตร์ มหาวิทยาลัยเทคโนโลยีสุรนารี

วันที่ 23 เมษายน 2546

08.00 – 08.30 น.	ลงทะเบียน
08.30 – 09.00 น.	พิธีเปิดโดยอธิการบดีมหาวิทยาลัยเทคโนโลยีสุรนารี
09.00 – 10.30 น.	**Thermoregulation in extreme environments: heat loss heat adaptation and thermal strain** **(Assoc. Prof. Dr. Nigel Taylor)**
10.30 – 10.45 น.	พักรับประทานอาหารว่างและเครื่องดื่ม
10.45 – 12.00 น.	**Body fluid volumes during postural, thermal and exercise stress.** **(Assoc. Prof. Dr. Nigel Taylor)**
12.00 – 13.00 น.	พักรับประทานอาหารกลางวัน
13.00 – 13.30 น.	Body composition assessment (นายบุญศักดิ์ หล่อพิพัฒน์)
13.30 -17.00 น.	**Practice I " Cardio-respiratory function during exercise) "** ที่ศูนย์เครื่องมือวิทยาศาสตร์และเทคโนโลยีอาคาร 2 (F2) 1. Assoc. Prof. Dr. Nigel Taylor 2. พ.ต.ดร.รุ่งชัย ชวนไชยะกุล 3. นายชัยสิทธิ์ ภาวิลาศ 4. ดร.วารี วิดจายา

วันที่ 24 เมษายน 2546

09.00 – 12.00 น.	**Practice II " Exercise in thermal condition"**
	ที่ศูนย์เครื่องมือวิทยาศาสตร์และเทคโนโลยีอาคาร 2 (F2)
	1. Assoc. Prof. Dr. Nigel Taylor
	2. พ.ต.ดร.รุ่งชัย ชวนไชยะกุล
	3. นายชัยสิทธิ์ ภาวิลาศ
	4. ดร.วารี วิดจายา
12.00 – 13.00 น.	พักรับประทานอาหารกลางวัน
13.00 – 15.00 น.	สรุปผลและวิเคราะห์ข้อมูล
15.00 – 15.15 น.	พักรับประทานอาหารว่างและเครื่องดื่ม
15.30 – 17.00 น.	อภิปรายผลแต่ละกลุ่ม

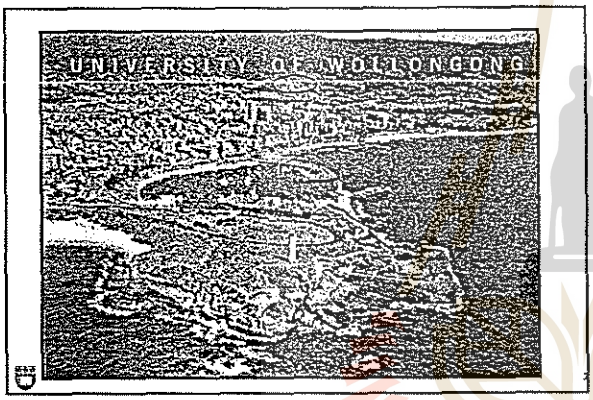
วันที่ 25 เมษายน 2546

09.00 – 10.30 น.	**Heat acclimatization, thermoregulatory failure and fluid replacement**
	(Assoc. Prof. Dr. Nigel Taylor)
10.30 – 10.45 น.	พักรับประทานอาหารว่างและเครื่องดื่ม
10.45 – 11.15 น.	สรุปและอภิปรายผล **(Assoc. Prof. Dr. Nigel Taylor)**
11.15 – 11.30 น.	พิธีปิดและมอบประกาศนียบัตร
11.30 – 12.00 น.	รับประทานอาหารร่วมกัน

WORKSHOP

Thermoregulation in extreme environments: heat loss and thermal strain.

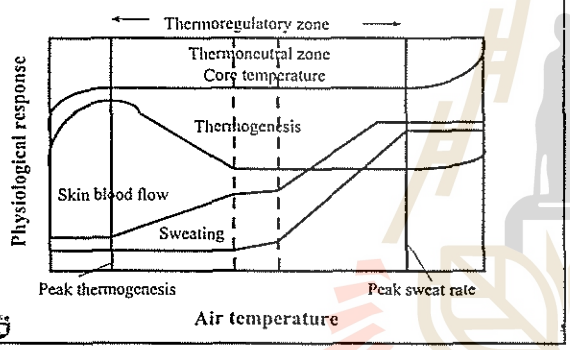
Nigel A.S. Taylor
Department of Biomedical Science
University of Wollongong

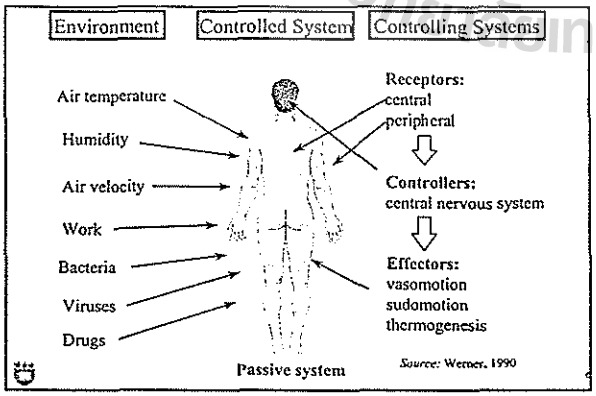


Exercising and working in extreme environments:

- Avenues for heat loss and heat gain
- Measuring body temperatures
- Physiological significance of sweat
- Measuring sweat secretion
- Factors affecting sweat evaporation
- Measuring skin blood flow
- Psychophysical measures

Heat gain and loss mechanisms:



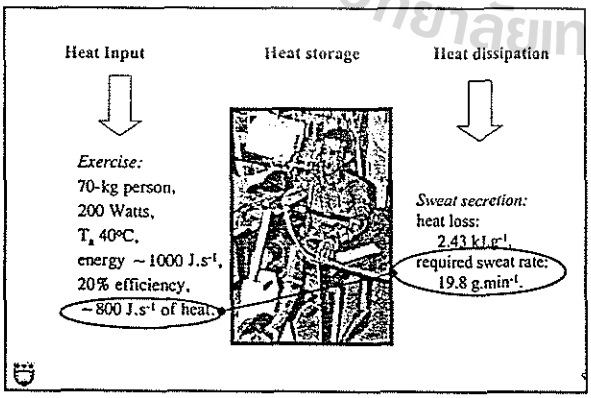


Heat balance equation

$S = M \; -(\pm W) \; \pm E \; \pm R \; \pm C \; \pm K \; [W.m^{-2}]$

- Heat storage (+=↑)
- External work (+= energy → machine)
- Metabolic heat production
- Evaporative heat loss
- Conductive heat exchange
- Convective heat exchange
- Radiative heat exchange

Mass (kg)	70.0
Height (cm)	175.0
Surface area (m2)	1.85
Core temperature (Tc)	37.0
Skin temperature (Tsk)	33.0
Mean body temperature (Tb)	35.7
Wind velocity (m/s)	0.20
Surface layer insulation (clo)	0.83
Clothing insulation (clo)	0.00
Total insulation (Icl; clo)	0.83
Air temperature	44.0
Heat Exchange Calculations:	
Rest heat production (Watts)	105.0
Rest heat production as % absolute heat exchange	19.9%
Heat Storage (Watts)	2410.5
Respiratory evap (Watts)	-14.5
Convective + Radiative exchange (Watts)	158.6
Required physiological heat exchange (Watts)	249.5
Required physiological heat loss (Watts; Resp+Sweat	263.6
Respiratory evap as % of absolute heat exchange	2.7%
C+R as % of absolute heat exchange	30.1%
Required new metabolic heat production (Watts)	0.0
% increase in metabolic heat production	0.0%
Required sweat evaporation (Watts)	249.5
Required sweat rate (ml/hr)	369.6
Sweat evap as % of absolute heat exchange	47.3%



Exercising and working in extreme environments:

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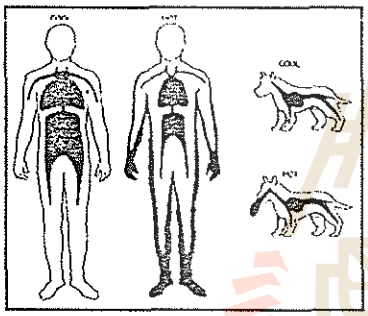
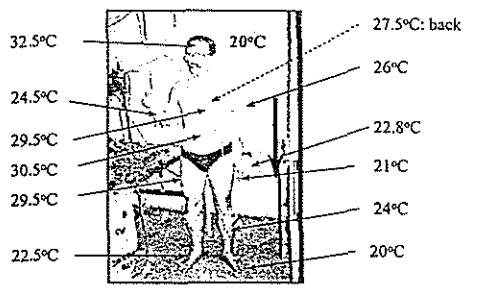
Resting blood volume distribution

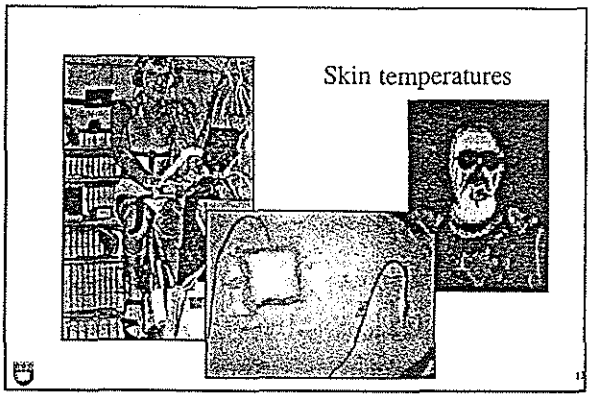


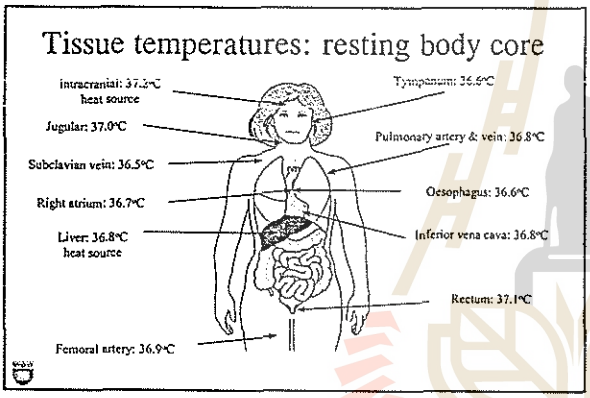
Source: Rowell (1983) *Handbook of Physiology*

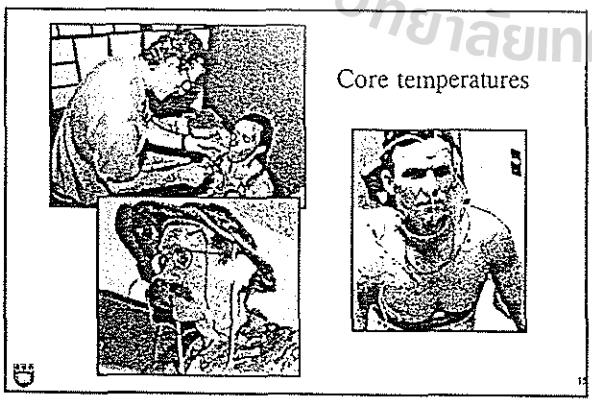
Tissue temperatures: resting skin (Δ12.5°C)

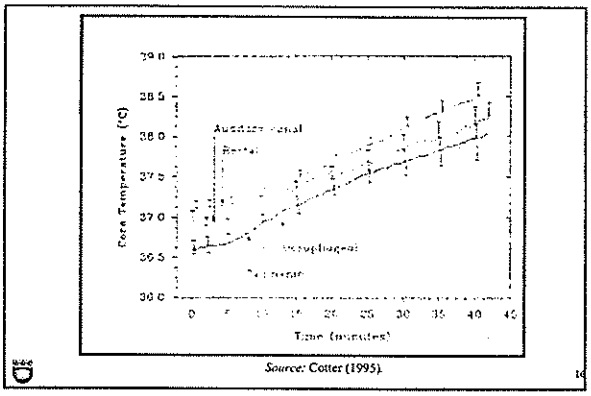


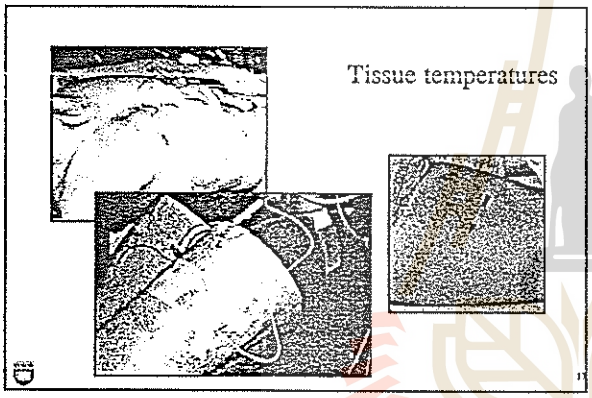
Source: Werner & Reents (1980) *Eur.J.Appl.Physiol.* 45:87-94.

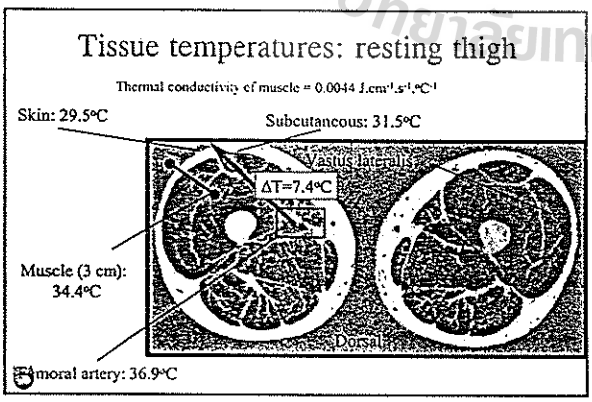












Blood flow and heat transfer

Heat flow ∝ thermal gradient.
Conduction (K) = $(k_c/l)(T_1-T_2)$:
k_c = thermal conductivity,
l = distance between sites,
T_1-T_2 = thermal gradient.

Mean T_{core} = 36.8°C & mean T_{skin} 32°C:
Thermal gradient is ≈ 5°C.

If 1 litre of blood loses 1°C in a trip from core to skin, then the core will lose 3.85 kJ of heat.

Exercising and working in extreme environments:

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  - Psychophysical measures

Fluid losses during thermoneutral rest:

Dissipate ~25% of total heat production

Kidneys:
- 1000-2000 ml.d^{-1} (urine)

Lungs:
- 750 ml.d^{-1}
- 1 ml per 10 litres of ventilation

Alimentary canal:
- 100-200 ml.d^{-1} (faeces)

Skin:
- transpiration (750 ml.d^{-1}).

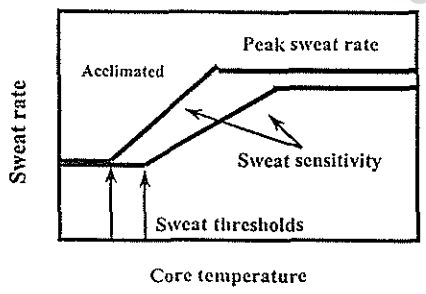
Total fluid loss:
- 3150 ml.d^{-1}.

- *Eccrine sweat glands:*
  - 1.6-4.0 million glands: lowest density on back (64 per cm^2), highest on palms and soles (600-700 per cm^2);
  - most effective means of heat dissipation in hot conditions;
  - unacclimated people:
    - extended sweating: 30 $g.min^{-1}$, or 1.5-1.8 $l.h^{-1}$;
    - severe, short-term heat stress: 3-4 litres per hour (very short period);
    - maximal daily sweat rate (heat+exercise): 10-15 litres.
  - men have higher sweat output than women in both dry and humid conditions;
  - heat acclimation enhances sudomotor function.

- *Eccrine sweat glands:*

Highest recorded sweat rate (Alberto Salazar: 1984 Olympics):

- 3.7 $l.h^{-1}$ after 19 days of heat adaptation,
- body mass: 67 kg,
- mass loss: 5.43 kg (8.1 % mass) in a 134-min marathon.

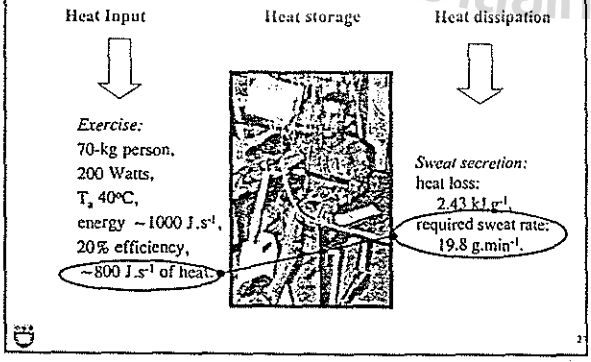


- *Sweat composition:*
  - The major composition of sweat is:
    - sodium ions (Na^+),
    - chloride ions (Cl^-), and
    - water.
  - Sweat glands have secretory and reabsorptive components.
  - Some primary sweat constituents are reabsorbed:
    - Na^+, Cl^- and bicarbonate ions.
  - This is the function of the distal portion of the gland tubule.
  - Thus, sweat is hypotonic to extracellular fluid.

Sweat composition:

Site	Sweat rate (mg/cm2/min)	Na (mmol/l)	K (mmol/l)	Cl (mmol/l)	HCO3 (mmol/l)	Lactate (mmol/l)	pH
Forehead	2.39	56.7	4.52	53.8	2.5	6.5	6.16
Chest	1.21	47.6	3.79	43.6	1.33	7.24	5.81
Scapula	0.95	41.1	3.49	38.1	1.8	7.65	5.82
Lower back	0.85	26.2	3.11	22.6	0.58	8.13	5.28
Abdomen	0.65	28.5	4.46	23.2	1.04	9.81	5.48
Arm	0.52	39.8	5.74	35	1.46	9.68	5.61
Forearm	0.75	42.2	5.93	36.1	2.38	10.52	5.88
Hand	0.91	34.4	5.65	25.9	1.07	11.02	5.68
Thigh	0.66	27	4.57	22	0.39	8.52	5.19
Calf	0.76	31.5	4.86	25.7	0.72	9.43	5.36
Foot	0.56	24.3	6.83	18.1	0.52	12.99	5.21

Source: Exp Physiol (2000) 85:869-87



Effects of exercise in the heat:

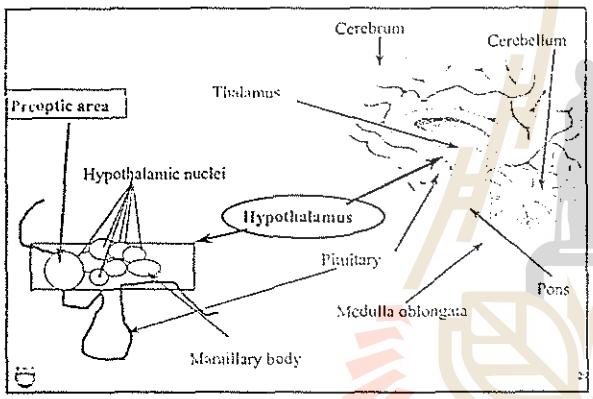
Kidneys:
- reduced renal blood flow.

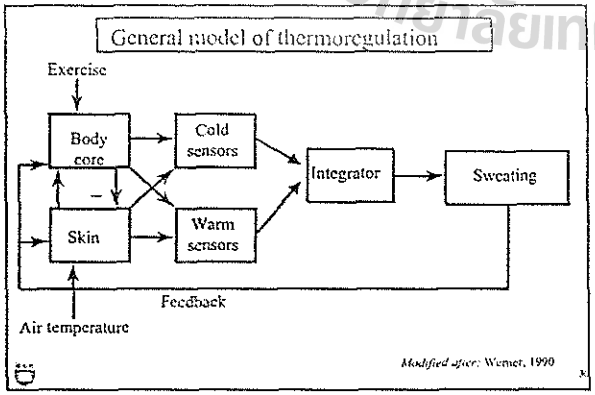
Lungs:
- increased alveolar ventilation,
- 100 l.min^{-1} → 10 ml.min^{-1} (evaporation).

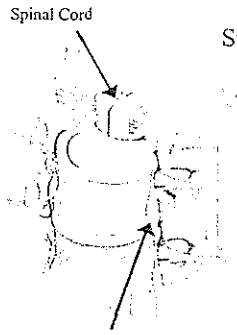
Alimentary canal:
- reduced colonic blood flow,
- increased bowel motility.

Skin:
- sweat secretion.

Total fluid loss:
- 10-15 l.d^{-1}.







Sweat gland innervation:

Spinal segments (*Quinton, 1983*):
T_2-T_4 supply glands of head and neck,
T_2-T_8 innervate the upper limbs,
T_6-T_{10} supply the trunk, and
segments T_{11}-L_2 pass to the legs.

Sweat glands

Total number of sweat glands:
- between 1.6-4.0 million.

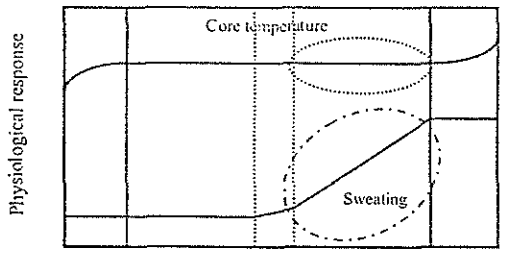
Considerable variability in gland density:
- lowest: back (64 per cm^2),
- highest: palms and soles (600-700 per cm^2).

Glands:
- single unbranched, coiled tubules (~300 μm diameter),
- 2-5 mm below the epidermis,
- connected to the epidermis via a straight segment,
- tubule contains secretory and reabsorptive components.

Sweat glands

Neural signals reach sweat glands in waves:
- sympathetic cholinergic activation,
- pulsatile sweat secretion,
- synchronised between skin regions,
- frequency of expulsion: 5-30 per minute.

Core temperature:



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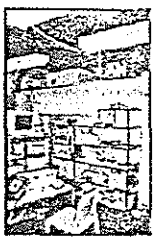
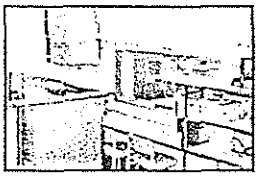
  - Psychophysical measures

Measuring thermoregulatory function: University of Wollongong.

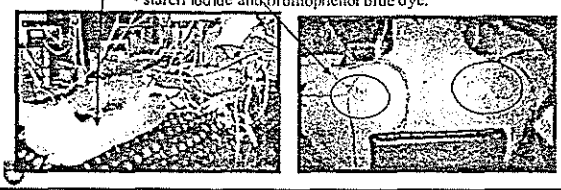




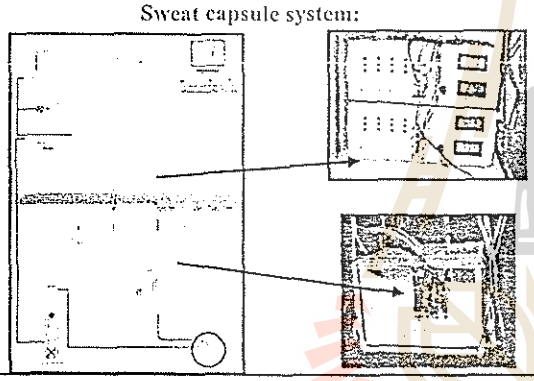
Sweat rate measurement:

Whole-body mass changes:
- scale: integrated changes,
- load cells: instantaneous changes (*Candas*).

Regional sweating:
- sweat bags: limb segment (*Maughan*),
- sweat capsules: expulsion rate and frequency (*Wollongong*),
- starch iodide and bromophenol blue dye.

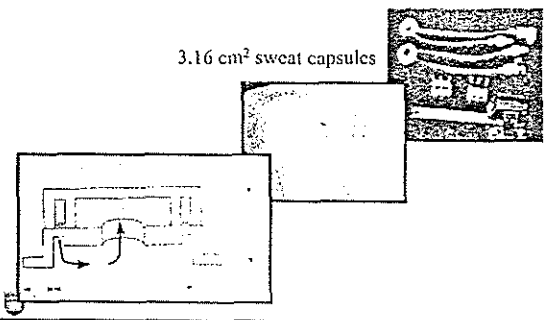


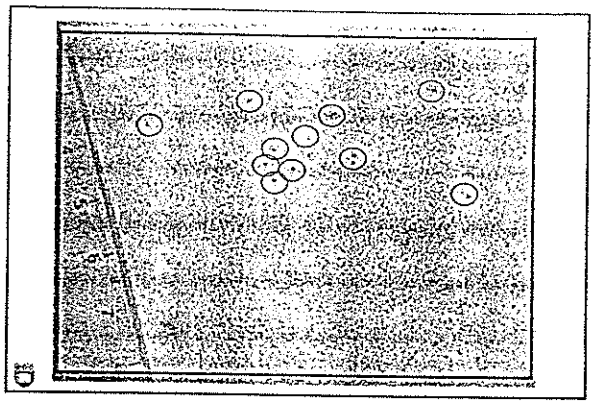
Sweat capsule system:

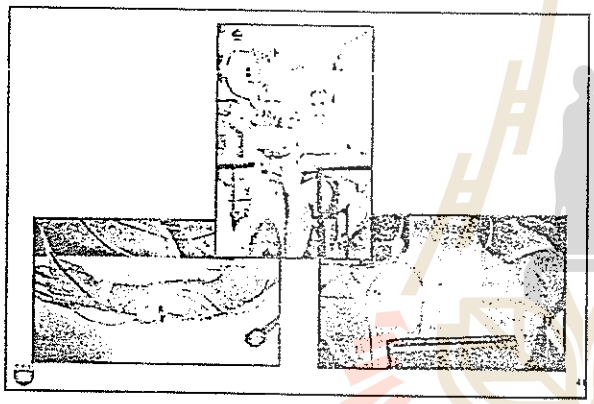


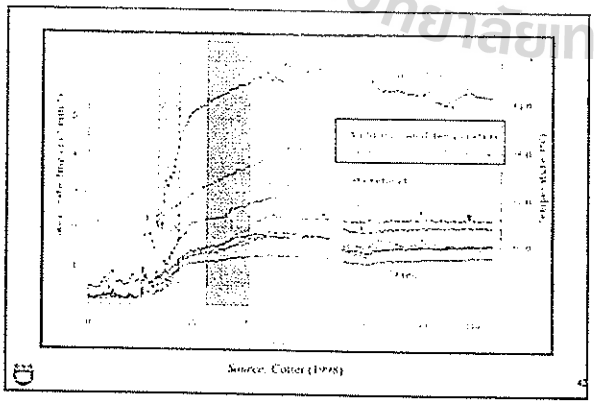
Sweat capsule system:

3.16 cm^2 sweat capsules

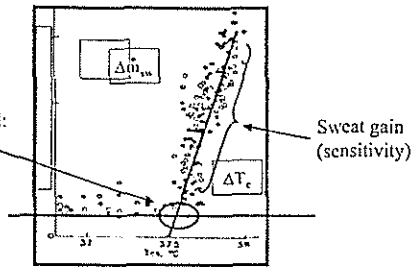








Sweating response to core temperature:



Sweat rates encountered during exercise:

Extended sweating: 30 g.min^{-1}, 1.5-1.8 l.h^{-1}.
Severe, short-term heat stress: 3-4 l.h^{-1}.
Acclimatisation: 2-3 l.h^{-1}.
Highest recorded sweat rate: 3.7 l.hr^{-1}
(Alberto Salazar: 1984 Olympic Marathon).
24-hour heavy sweating: 10-15 l.d^{-1}.

Exercising and working in extreme environments:

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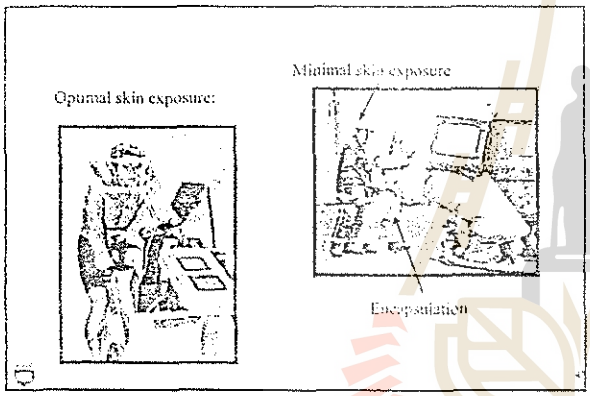
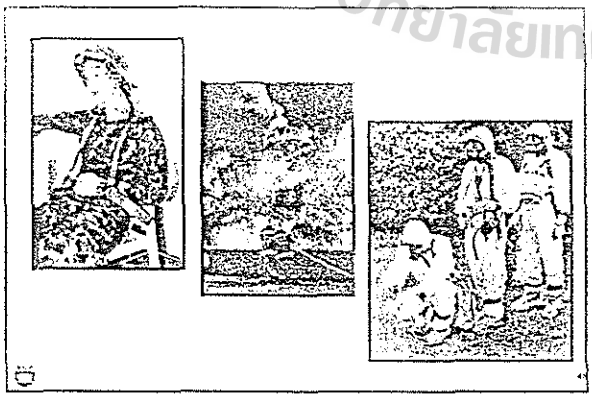
Factors affecting the evaporation of sweat:

- Water vapour pressure gradient:
  - air vapour pressure:
    - macro-climate: air,
    - micro-climate: clothing;
  - skin vapour pressure:
    - skin temperature.

  } gradient

- Surface area:
  - wetted surface area,
  - exposed surface area.
- Wind velocity:
  - macro-climate: air,
  - micro-climate: clothing ventilation.





Thermal protective clothing:

Problems:

- Reduced evaporative heat loss:
  - can be reduced by 70%,
  - solution:
    - increase skin exposure,
    - increase garment ventilation;
- Trapped metabolic heat:
  - insulates worker from radiation,
  - reduces dry heat losses;
- Increased metabolic load:
  - mass of clothing added to body,
  - reduced mechanical efficiency,
  - reduced range of motion.

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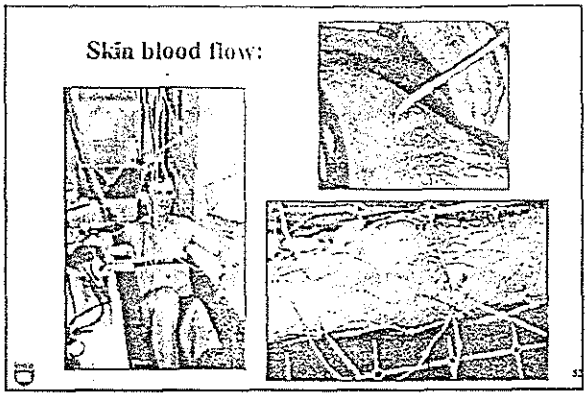
Skin blood flow:

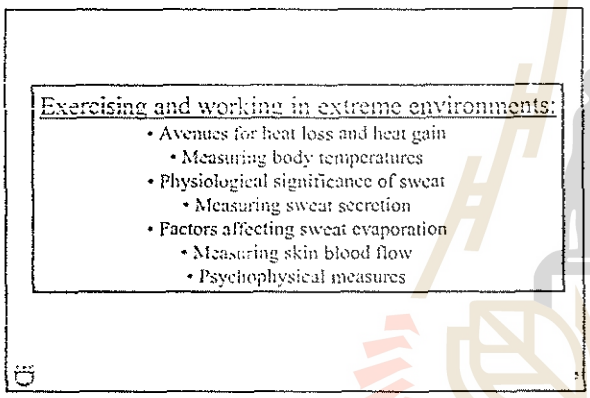
Venous occlusion plethysmography

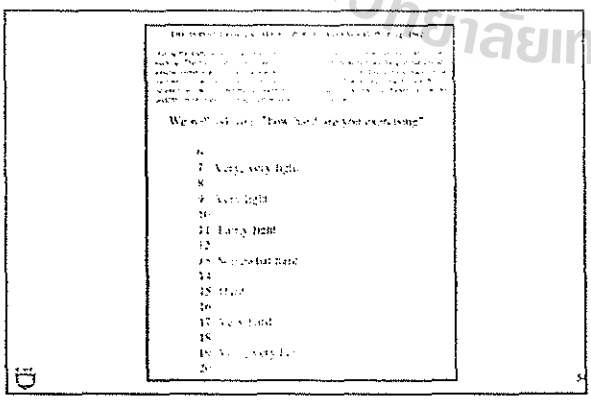
Pressure cuff: venous occlusion

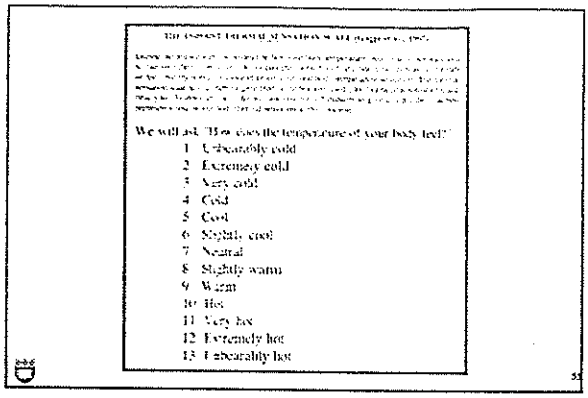
Strain-gauge

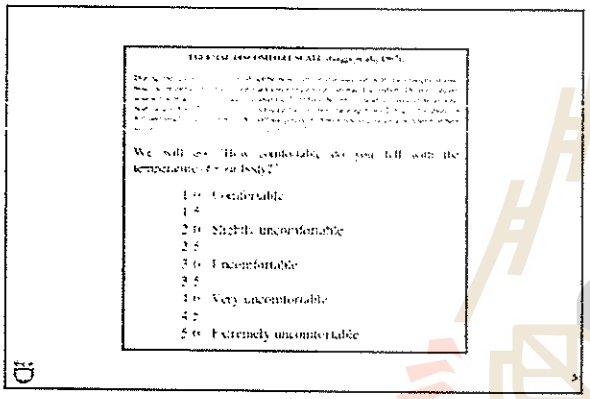
Pressure cuff: arterial pressure

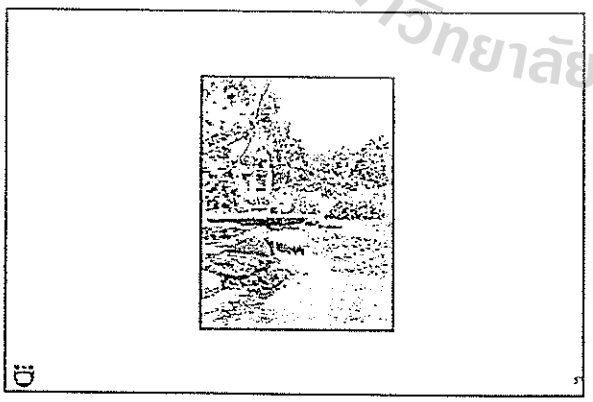












Body fluid volumes during postural, thermal and exercise stress.

Nigel A.S. Taylor
Department of Biomedical Science
University of Wollongong

Fluid volumes: postural, thermal, exercise stress:

- Body fluid compartments
- Measuring body fluid volumes
- Physiological significance of body fluids
- Body fluid shifts during postural changes
- Body fluid movements during exercise
- Body fluid volume during thermal stress

Total body water (TBW) volume:

- The human body is ~60% water
  - may range between 40-80% of body mass.
  - reasons for variability:
    - natural variability between people,
    - particularly due to adipose tissue.
  - Adipose tissue has the lowest water content.
  - Higher water content in lean people.

Relative water volumes:

Tissue	% water
Kidney	83
Heart	79
Lung	79
Skeletal muscle	76
Brain	75
Skin	72
Liver	68
Skeleton	22
Adipose tissue	10

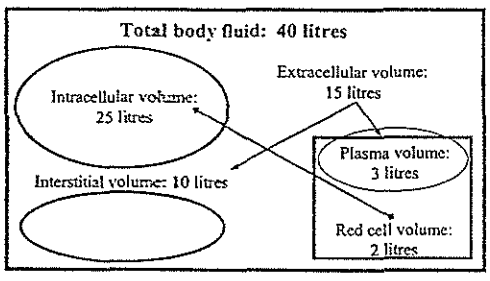
Total body water (TBW) volume:

TBW content is also influenced by:

- *Age:* decreases with age
- *Gender:* higher in women due to generally greater fat content.

Age (years)	Male (% water)	Female (% water)
newborn	80	75
1-5 y	65	65
10-16 y	60	60
17-39 y	60	50
40-59 y	55	47
60+ y	50	45

Body fluid volumes



Body fluid volumes

Eight body-fluid compartments:

- Total body fluid: 500-600 ml.kg^{-1} (female-male):
  - Intracellular fluid: 300-340 ml.kg^{-1}:
    - red cell volume: 25-30 ml.kg^{-1};
  - Extracellular volume: 200-260 ml.kg^{-1}:
    - interstitial fluid volume: 160-220 ml.kg^{-1};
    - plasma volume: 40-40 ml.kg^{-1};
  - Extravascular cellular volume: 275-310 ml.kg^{-1};
    - blood volume: 65-70 ml.kg^{-1} (RCV+PV):
    - plasma volume: 40-40 ml.kg^{-1};
    - red cell volume: 25-30 ml.kg^{-1}.

Composition of body fluids

Cells are separated from surrounding fluids by a selectively-permeable plasma membrane.

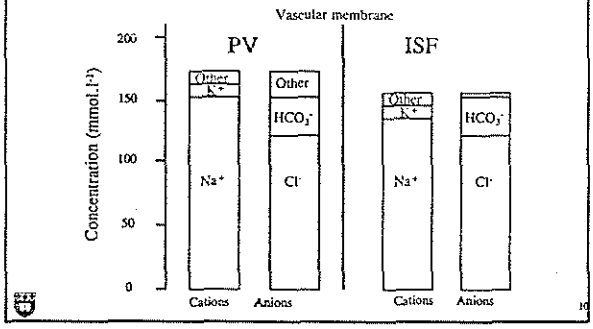
Thus, the composition of intracellular and extracellular fluids is very different.

- Extracellular fluid:
  - major compartments of ECF: PV and interstitial fluid (ISF)
  - have very similar, but not identical compositions
  - Na^+ is major cation and Cl^- and HCO_3^- the major anions
  - proteins are also present in the PV:
    - cannot easily pass through the vascular walls
    - this causes electrolyte compositions to be different.

Composition of body fluids

- **Extracellular fluid:**
  - Proteins:
    - proteins cannot generally cross the vascular membrane
    - hence, they affect distribution of ions on both sides of the vascular membrane (PV has more -ive ions than ISF):
      - cations (Na^+, Mg^+, K^+) will tend to move into PV
      - sum of anions and cations in PV and ISF is equal
        - thus, electrical neutrality within a fluid
      - but the total concentration is not equal across the membrane (*e.g.* one side is more negative).

Composition of body fluids



Composition of body fluids

Electrolytes (cations & anions)	Extracellular fluid: Plasma (mmol.l⁻¹)	Interstitial (mmol.l⁻¹)	Intracellular (mmol.l⁻¹)
sodium (Na^+)	152	143	14
potassium (K^+)	5	4	157
calcium (Ca^{2+})	2.5	2.5	
magnesium (Mg^{2+})	1.5	1.5	13
chloride (Cl^-)	113	117	5
bicarbonate (HCO_3^-)	27	27	10
protein ($Prot^{n-}$)	1		4

Units: concentrations are expressed mmol.l⁻¹, which are the same as millimolar concentrations (m*M*); some texts use mEq.l⁻¹, which are equal to mmol.l⁻¹×valance.

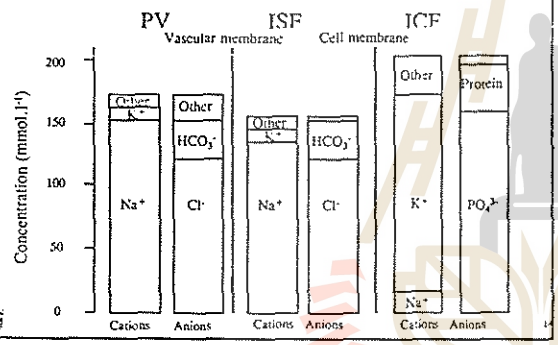
Composition of body fluids

- **Intracellular fluid:**
  - *Na^+/K^+-ATPase pump:*
    - present in all cell membranes
    - actively transports Na^+ out of, and K^+ into, the cells
      - Na^+ is the primary extracellular cation
      - K^+ is the primary intracellular cation.
  - *Limited membrane permeability:*
    - some electrolytes cannot cross the cell membrane
      - proteins and organic phosphates
    - these affect distribution of ions across cell membrane
      - inside cells there is more protein than the outside
      - cations (Na^+, Mg^+, K^+) will tend to move inside
      - so the sum of anions and cations *on each side of the membrane* is equal
      - but total concentration is not be equal across the membrane.

Composition of body fluids

- **Intracellular fluid:**
  - Results of these two factors:
    - the concentrations of small diffusible ions (K^+ and Cl^-) are not equal across the cell membrane:
      - K^+ is greater inside, Cl^- is greater outside cell
      - distribution is governed by Na^+/K^+-ATPase pump
    - total concentration of cations and anions is greater inside cells
    - there is electrical neutrality within the intracellular compartment.

Composition of body fluids



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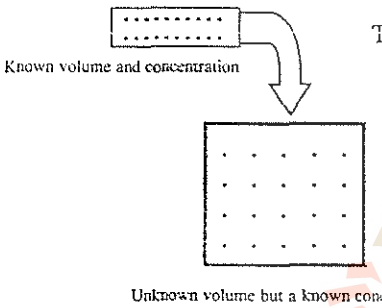
Fluid volumes: postural, thermal, exercise stress:

- Body fluid compartments
- Measuring body fluid volumes
- Physiological significance of body fluids
- Body fluid shifts during postural changes
- Body fluid movements during exercise
- Body fluid volume during thermal stress

Tracer dilution technique:



Measuring the size of fluid compartments

General equation:

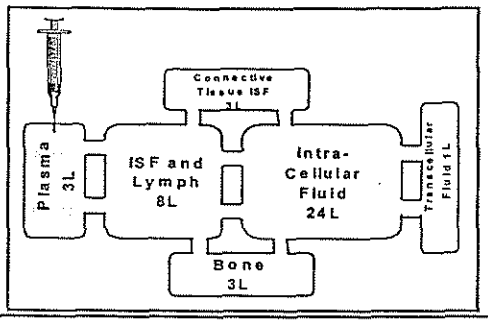
$V_1C_1 = V_2C_2$

where: V_1 = injected (known) volume
C_1 = injected (known) concentration
V_2 = unknown volume
C_2 = measured (known) concentration

Thus: $V_2 = V_1C_1 / C_2$

$$\text{Compartment volume} = \frac{\text{injected volume} * \text{injected concentration}}{\text{measured concentration}}$$

Measuring the size of fluid compartments



Measuring the size of fluid compartments

COMPARTMENT	SUBSTANCE
Total body	Tritiated water (^3H)
Extracellular	^{82}bromium ^{51}cromium-EDTA
Plasma	^{131}I-albumin Evans Blue
Blood	^{51}Cr-RBCs

Fluid volumes: postural, thermal, exercise stress:

- Body fluid compartments
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- Body fluid movements during exercise
- Body fluid volume during thermal stress

Regulating body-fluid volumes

Water balance:
Body-fluid balance requires input to match output.

- Three main sources of water input:
  - *water consumed as water* (1-2 litres per day)
  - *water consumed in foods* (0.8-1 litres per day)
  - *water generated during oxidation* (metabolism) of foods:
    - 0.3-0.4 litres per day
    - oxidation of 108 g glucose generates 108 ml of water.

Regulating body-fluid volumes

Water balance:
Body-fluid balance requires input to match output.

- Four main sources of water output:
  - *urine* (1-2 litres per day)
  - *faeces* (0.1-0.2 litres per day)
  - *sweat* (widely variable: 0.2 to 15 litres per day)
  - *insensible water losses*:
    - 50% respiratory (0.4-0.5 litres per day)
    - 50% via skin (0.4-0.5 litres per day).

Regulating body-fluid volumes

Water balance:
Body-fluid balance requires input to match output.

- Two most important factors controlled during water regulation:
  - *water consumed as water*
    - modulated by thirst
  - *water lost as urine*
    - modulated by antidiuretic hormone (vasopressin).

Regulating body-fluid volumes

Water balance:

- Working with numbers:
  - Total body water = 550 ml/kg
  - Average urine flow = 0.893 ml/kg/hr (max: 12 ml/kg/hr)
  - Average faecal loss = 0.089 ml/kg/hr (max: 2.98 ml/kg/hr)
  - Average respiratory loss = 0.3 ml/kg/hr
  - Average skin loss (rest) = 0.3 ml/kg/hr
  - Average sweat rate = 22 ml/kg/hr (1.5 l/hr for 70 kg).

Fluid losses during thermoneutral rest:

Dissipate ~25% of total heat production

Kidneys:
- 1000-2000 ml.d^{-1} (urine)

Lungs:
- 750 ml.d^{-1}
- 1 ml per 10 litres of ventilation

Alimentary canal:
- 100-200 ml.d^{-1} (faeces)

Skin:
- transpiration (750 ml.d^{-1}).

Total fluid loss:
- 3150 ml.d^{-1}.

Regulating body-fluid volumes

Resting adults

Mass (kg)	Total body fluid (ml)	Resp water loss (ml/hr)	Urine flow (ml/hr)	Skin water loss (ml/hr)	Faecal water loss (ml/hr)	Total loss per hour (ml)	Total loss per day (ml)
50	27500.0	15.000	44.650	15.000	4.450	79.100	1898.400
55	30250.0	16.500	49.115	16.500	4.895	87.010	2088.240
60	33000.0	18.000	53.580	18.000	5.340	94.920	2278.080
65	35750.0	19.500	58.045	19.500	5.785	102.830	2467.920
70	38500.0	21.000	62.510	21.000	6.230	110.740	2657.760
75	41250.0	22.500	66.975	22.500	6.675	118.650	2847.600
80	44000.0	24.000	71.440	24.000	7.120	126.560	3037.440
85	46750.0	25.500	75.905	25.500	7.565	134.470	3227.280
90	49500.0	27.000	80.370	27.000	8.010	142.380	3417.120
95	52250.0	28.500	84.835	28.500	8.455	150.290	3606.960
100	55000.0	30.000	89.300	30.000	8.900	158.200	3796.800

Required fluid intake:
- 1.6 ml/kg/hr

Effects of exercise in the heat:

Kidneys:
- reduced renal blood flow.

Lungs:
- increased alveolar ventilation,
- 100 $l.min^{-1} \rightarrow 10$ $ml.min^{-1}$ (evaporation).

Alimentary canal:
- reduced colonic blood flow,
- increased bowel motility.

Skin:
- sweat secretion.

Total fluid loss:
- 10-15 $l.d^{-1}$.

Sweat rates encountered during exercise:

Extended sweating: 30 $g.min^{-1}$, 1.5-1.8 $l.h^{-1}$.
Severe, short-term heat stress: 3-4 $l.h^{-1}$.
Acclimatisation: 2-3 $l.h^{-1}$.
Highest recorded sweat rate: 3.7 $l.hr^{-1}$
(Alberto Salazar: 1984 Olympic Marathon).
24-hour heavy sweating: 10-15 $l.d^{-1}$.

Regulating body-fluid volumes

Exercising adults: average sweat rate = 22 ml/kg/hr

Mass (kg)	Total body fluid (l)	Sweat rate (l/hr)
50	27.5	1.1
55	30.3	1.2
60	33.0	1.3
65	35.8	1.4
70	38.5	1.5
75	41.3	1.7
80	44.0	1.8
85	46.8	1.9
90	49.5	2.0
95	52.3	2.1
100	55.0	2.2

Required fluid intake:
- rest: 1.6 ml/kg/hr
- exercise: 22.2 ml/kg/hr
- total: 23.8 ml/kg/hr

Regulating body-fluid volumes

Key facts:

- The PV is the only body fluid which has its volume and composition directly regulated.
- The ECF serves as an intermediary between the cells and the external environment:
  - all exchanges between cells and environment occur through the ECF:
    - water added to body enters and leaves via the ECF.
- Because fluid and electrolyte exchanges can occur across the capillary membrane, then the volume and composition of the ISF is also regulated.
- Thus, plasma fluid and composition regulation dictates the volume and composition of the entire ECF.

Regulating body-fluid volumes

Key facts:

- Since ICF composition is influenced by the ECF, then it is largely dictated by PV regulation:
  - exception: the influence of cell membrane barriers:
    - Na^+/K^+-ATPase pump
    - protein composition of membranes.
- Two most important factors regulating body-fluid volumes:
  - ***Extracellular fluid osmolarity:***

    The maintenance of water balance is of primary importance in the regulation of the ECF osmolarity.
    - regulated to prevent swelling (reduced osmolarity):
      - hypotonicity or hyperhydration
    - regulated to prevent shrinking (elevated osmolarity)
      - hypertonicity or hypohydration.

Regulating body-fluid volumes

Key facts:

- Two most important factors regulating body-fluid volumes:
  - ***Extracellular fluid osmolarity:***
  - ***Extracellular fluid volume:***

    Regulated to help maintain blood pressure.
    - It is the concentration of electrolytes in the ECF (electrolyte balance) that is responsible for determining the ECF.

Regulating body-fluid volumes

Water balance:

Two most important factors controlled during water regulation:

- *water consumed as water*
  - modulated by thirst
- *water lost as urine*
  - modulated by antidiuretic hormone (ADH or vasopressin).

These are stimulated by two primary physiological variables:

- plasma osmolarity (concentration)
- blood pressure.

Regulating body-fluid volumes

- Osmoreceptors within the hypothalamus monitor plasma osmolality, *[and also juxtaglomerular cells of kidney]*.

- The volume and tonicity of these receptor cells change with variations in the osmolarity of plasma:
  - expand (become hypotonic, hyperhydrated) when plasma is dilute
  - shrink (become hypertonic, dehydrated) when plasma is concentrated.

Regulating body-fluid volumes

- Dehydration can be induced in three ways:
  - insufficient water intake
  - excessive water loss
  - diabetes insipidus:
    - a deficiency of ADH, which prevents the kidneys from adequately reabsorbing water; patients can produce up to 20 litres of urine per day.

- Hyperhydration can be induced in three ways:
  - renal failure
  - rapid ingestion of large volumes of water
  - retention of excess water by body without retention of solutes: due to inappropriate secretion of ADH.

Regulating body-fluid volumes

- Baroreceptors are located in two regions:
  - low pressure receptors: right and left atria
  - high pressure receptors: aortic and carotid regions
    - also at the juxtaglomerular cells of the kidney.
- Baroreceptors respond to stretching induced by either an increase in PV or blood pressure (BP):
  - increased BP or PV increases baroreceptor activity
  - decreased BP or PV decreases baroreceptor activity.

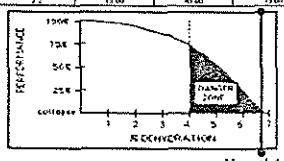
Thirst regulation

- Thirst is the *subjective sensation* that drives the consumption of water:
  - *thirst centre*: hypothalamus
- The sensation appears only after we have started to become dehydrated, and the sensation disappears before the body is adequately hydrated.
  - *Significance:*
    - drink early to minimise the risk of dehydration
    - drink to replace mass loss, not to quench thirst.

Thirst regulation

Mass (kg)	Total body fluid (l)	Water loss (l)	Loss as 1% dehydration	Loss as 3% dehydration	Loss as 5% dehydration
30	20.1	1.1	15.00	45.00	75.00
35	23.5	1.2	15.00	45.00	75.00
40	26.8	1.3	15.00	45.00	75.00
45	30.2	1.4	15.00	45.00	75.00
50	33.5	1.5	15.00	45.00	75.00
55	36.9	1.6	15.00	45.00	75.00
60	40.2	1.7	15.00	45.00	75.00
65	43.6	1.8	15.00	45.00	75.00
70	46.9	1.9	15.00	45.00	75.00
75	50.3	2.0	15.00	45.00	75.00
80	53.6	2.1	15.00	45.00	75.00
85	57.0	2.2	15.00	45.00	75.00
90	60.3	2.3	15.00	45.00	75.00
95	63.7	2.4	15.00	45.00	75.00
100	67.0	2.5	15.00	45.00	75.00

Average sweat rate:
22 ml/kg/hr
(1.5 l/hr for 70 kg)



Normal daily loss:
zero fluid replacement

Thirst regulation

- Thirst is predominantly stimulated by the *hypothalamic osmoreceptors:*
  - increased osmolarity stimulates
  - decreased osmolarity inhibits thirst.
- Thirst is also stimulated by changes in ECF, which is detected by *atrial volume or pressure receptors* and *baroreceptors*.

Thirst regulation

- Thirst is also stimulated by the presence of *angiotensin II* within the blood.
- Thirst is not stimulated by stress-related factors: pain, fear, trauma.
- It is unaffected by alcohol.
- However, thirst may be promoted by factors which do not act in the regulation of ADH secretion: *dry mouth.*

Fluid volumes: postural, thermal, exercise stress:

- Body fluid compartments
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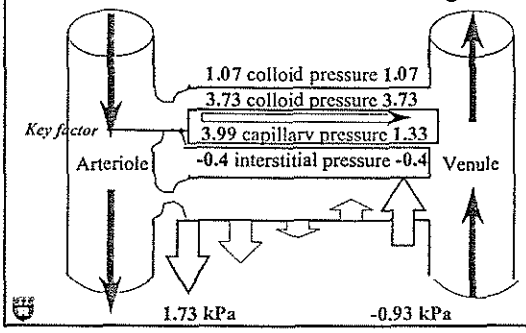
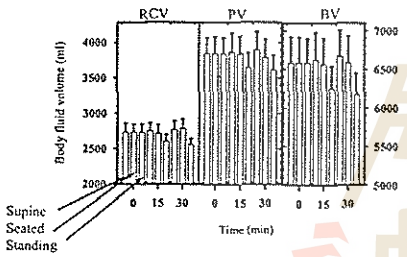
Arteriolar and venous fluid exchange:



Plasma volume changes: posture



Source: Maw et al. (1995) *Acta Scand. Physiol.* 155:157-163.

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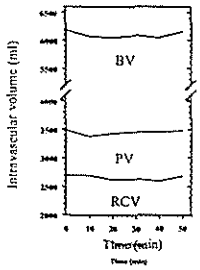
Plasma volume changes: exercise



Source: Maw et al. (1998) *Acta Scand. Physiol.* 163:297-304.

Fluid volumes: postural, thermal, exercise stress:

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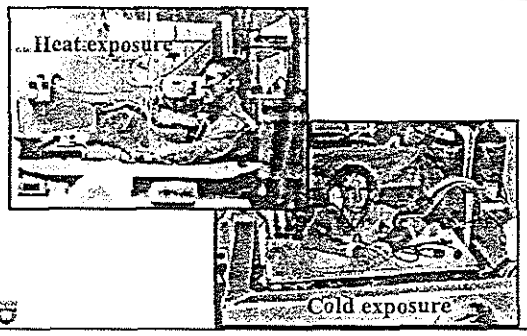
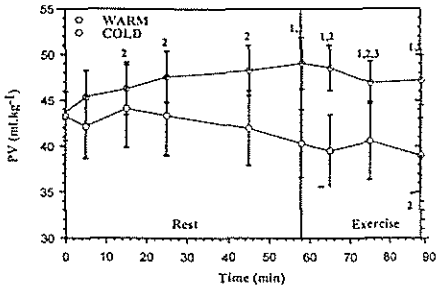
Thermal factors:

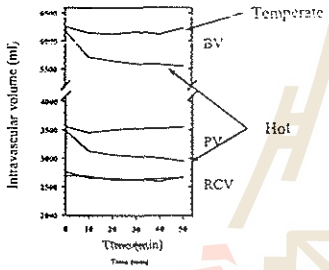


Plasma volume changes: cold immersion



Source: Regan *et al.* (1998)

Plasma volume: exercise and heat



Source: Maw et al. (1998) *Acta Scand. Physiol.* 163:297-304.

Heat acclimatisation, thermoregulatory failure and fluid replacement.

Nigel A.S. Taylor
Department of Biomedical Science
University of Wollongong

Heat acclimation, thermal failure and hydration:

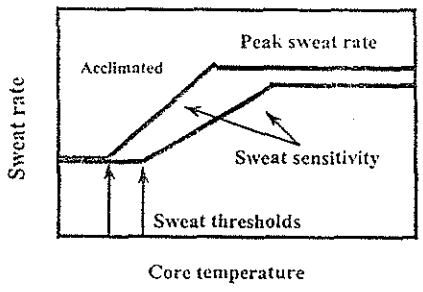
- Heat adaptation
- Heat adaptation methods
- Thermoregulatory failure
- Dehydration
- Fluid replacement

STANDARD THERMAL TERMS

- *Adaptation:*
  - change which reduces physiological strain;
  - when change occurs within the normal lifetime:
    - *phenotypic adaptation.*
  - When change is the result of genetic selection:
    - *genotypic adaptation.*
  - Two types of thermal, phenotypic adaptation are:
    - *Acclimation:* experimentally-induced adaptation.
    - *Acclimatisation:* natural exposures which induce adaptation.

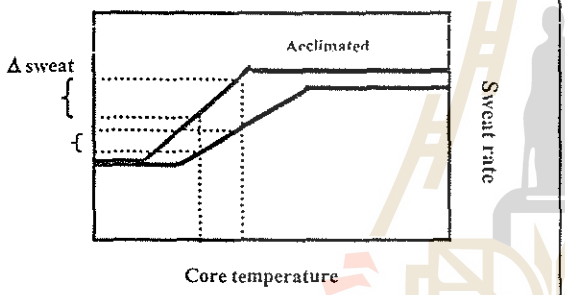
- *Eccrine sweat gland adaptation:*
- *Sweat rate:*
  - improved sweating is one of the more delayed adaptations,
  - requires 8-14 days to obtain ~95% of the final change:
    - an elevated steady-state sweat rate,
    - increased peak sweat rate.
  - result: doubling peak hourly sweat rate to 2-3 $L.h^{-1}$;
  - highest recorded sweat rate (*Alberto Salazar: 1984 Olympics*):
    - 3.7 $L.h^{-1}$ after 19 days of heat adaptation,
    - body mass: 67 kg,
    - mass loss: 5.43 kg (8.1% mass) in a 134-min marathon.

- *Eccrine sweat gland adaptation:*
  - Sweat adaptation may be of central or neural origin:
    - hypothalamus:
      - indicated by a sweat threshold change,
      - reduced T_c threshold for sweating onset,
      - hypothalamus initiates sweating at a lower T_c;
    - acclimation increases number of sweating glands:
      - increased gland recruitment.



- *Eccrine sweat gland adaptation:*
  - Adaptation is also brought about by peripheral mechanisms.
  - For a given neural drive, more sweat is produced.
  - Thus, acclimation increases sweat gland sensitivity.
  - The mechanism:
    - glandular hypertrophy,
    - larger tubular diameters,
    - longer secretory tubules,
    - greater secretory capacity of the glands,
    - increased periglandular concentrations of acetylcholine,
    - increased cholinergic sensitivity.

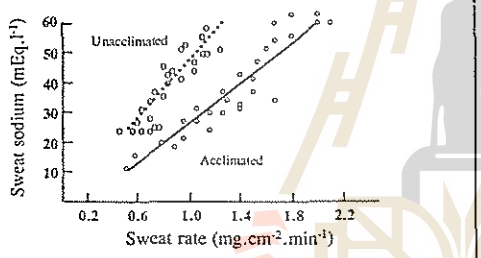
    (Source: Sato and Sato, 1974)



- *Eccrine sweat gland adaptation:*
- *Sweat composition:*
  - The major composition of sweat is:
    - sodium ions (Na^+),
    - chloride ions (Cl^-), and
    - water.
  - Sweat glands have secretory and reabsorptive components.
  - Some primary sweat constituents are reabsorbed:
    - Na^+, Cl^- and bicarbonate ions.
  - This is the function of the distal portion of the gland tubule.
  - Thus, sweat is hypotonic to extracellular fluid.

- *Eccrine sweat gland adaptation:*
- *Sweat composition:*
  - As sweat rate increases:
    - the rate of ion secretion exceeds ion reabsorption,
    - thus, the ion concentration of sweat increases, and
    - total electrolyte loss per litre of sweat increases.
  - Acclimation increases gland responsiveness to aldosterone.
  - Thus, glands reabsorb more sodium and chloride from the primary sweat, leading to a greater conservation of electrolytes.

Sweat rates and sodium loss

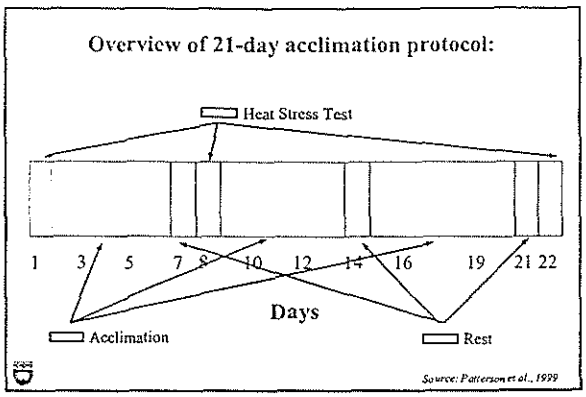


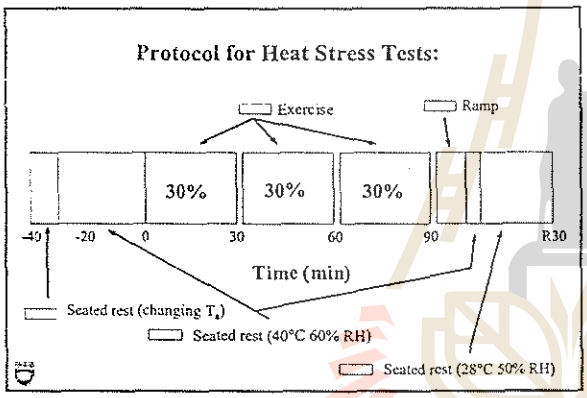
Source: Allan and Wilson (1971)

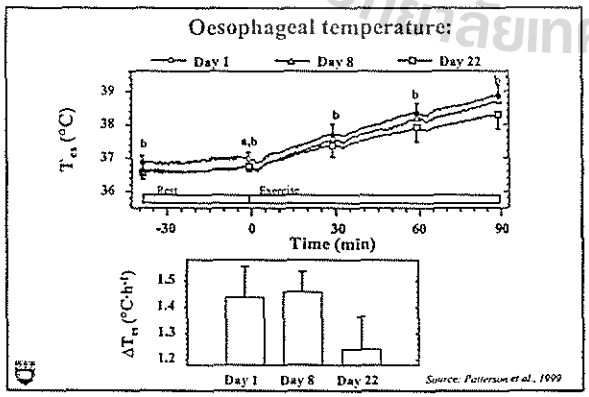
Heat adaptation:

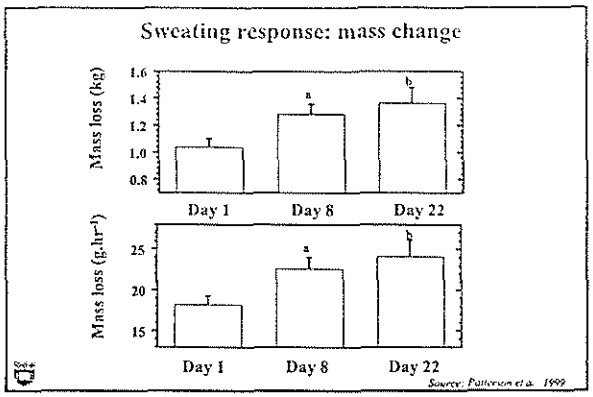
- Eleven active males
- **Heat Stress Tests:**
  - prior to acclimation
  - after 7 days
  - after 21 days
- **Task:**
  - cycling 90-min per day
  - six days a week
  - 40°C, 60% RH
  - T_{core} elevated to 38.5°C
  - then held stable.

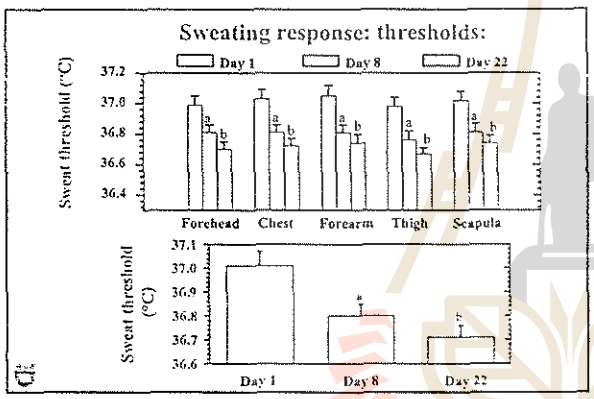
Source: Patterson et al., 1999

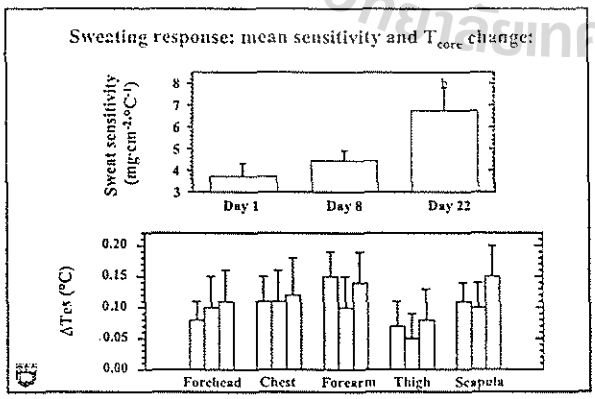




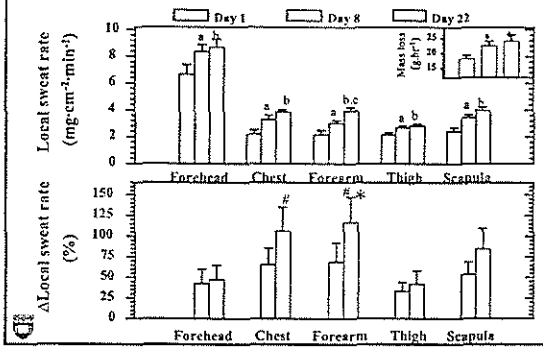








Sweating response: local sweat rates:



Sweating response:

Active sweat glands (glands·cm^{-2})		Glandular flow (nl ·gland^{-1}·min^{-1})		
		Day 1	Day 8	Day 22
Forehead	177.8 ±5.8	35.9 ±4.2*,#	46.1 ±3.1*	48.6 ±3.6*
Forearm	105.8 ±5.0	19.6 ±3.2	28.7 ±2.3	36.8 ±3.0
Scapula	80.4 ±2.7	29.3 ±3.5*	42.9 ±3.0*	50.3 ±3.4*
Chest	79.6 ±2.0	26.6 ±3.8	40.5 ±4.3*	47.9 ±2.6*
Thigh	61.6 ±6.8	33.4 ±2.8*	43.2 ±2.3*	45.6 ±2.5

Source: Montagna and Parakkal, 1974

Interpretation:

- Sweat elevations were observed at all sites.
- A trunk-to-limb redistribution was not observed.
- Why?
  - sweat changes reflect the capacity to elevate sweat secretion
  - sweat changes appear not to show altered sudomotor control.
- The reduced sweat threshold may be related to a lower resting T_{core}, and not necessarily central control.

Source: Patterson et al., 1999

Summary of human heat adaptation:

- Range of days to achieve ~ 95% of maximal response:
  - f_c decrease: 3-6 days;
  - PV expansion: 3-6 days: unless work rate is continually ↑;
  - reduced RPE: 3-6 days;
  - reduced urine Na and Cl excretion: 3-8 days;
  - reduced T_c: 5-8 days;
  - reduced sweat Na and Cl excretion: 5-10 days; and
  - increased sweat rate: 8-14 days.

Summary of human heat adaptation:

- *Key points:*
  - It takes up to 14 days for all systems to adapt completely;
  - Early adaptations involves improved CV control:
    - increased PV, reduced f_c.
  - The increase PV appears to be transient:
    - decays in 8-14 days (unless work rate is continually ↑),
    - hormonal changes also show decay,
    - both are replaced by:
      - increased sweating, and
      - reduced skin blood flow.

Heat acclimation, thermal failure and hydration:

- Heat adaptation
- Heat adaptation methods
- Thermoregulatory failure
- Dehydration
- Fluid replacement

Considerations for heat acclimation protocols:

- *Safety:*
  - monitor the T_c and f_c of each individual;
  - identify subjects most likely to experience dysthermia;
  - establish emergency procedures.
- *Efficiency:*
  - efficiency of personnel;
  - throughput;
  - acclimation duration;
  - acclimation decay;
  - individual needs;
  - availability of the acclimation facility.

Considerations for heat acclimation protocols:

- *Acclimation specifications:*
  - basal acclimation state of people;
  - worst case thermal stress expected;
  - the level of heat acclimation required;
  - critical heat exposure work durations;
  - test of heat acclimation.

Principles and practices of heat acclimation:

- Natural acclimatisation is best.
- It is usually not possible.
- Five methods:
  - Passive heat acclimation:
  - Exercise-induced heat adaptation:
  - Exercise-induced heat adaptation with solar load:
  - Exercise-induced heat adaptation with sweat clothing:
  - Combined exercise and heat stress acclimation:
    - Constant work rate protocols:
    - Self-regulated exercise protocols:
    - Controlled hyperthermia protocols:

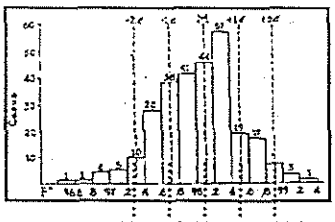
How is heat acclimation best maintained?

- The benefits of heat acclimation are transient.
- Rule of thumb:
  - one days acclimation is lost every two days
  - trained subjects lose effects more slowly.
- Decay is influenced by:
  - the magnitude of the thermal strain,
  - the duration of acclimation, and
  - the number of exposures.
- To maintain heat acclimation:
  - one exposure for each five days away from significant heat sources or exposures.

Heat acclimation, thermal failure and hydration:

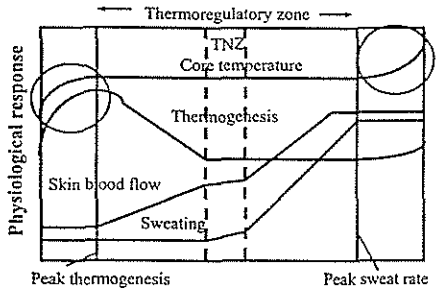
- Heat adaptation
- Heat adaptation methods
- Thermoregulatory failure
- Dehydration
- Fluid replacement

Distribution of core temperatures in young, resting adults (n=276).



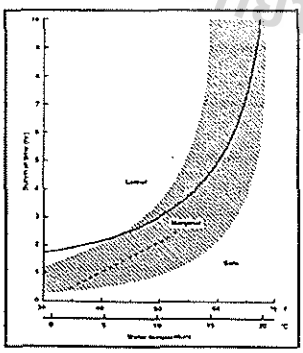
Source: Ivy, 1944

Heat gain and loss mechanisms:

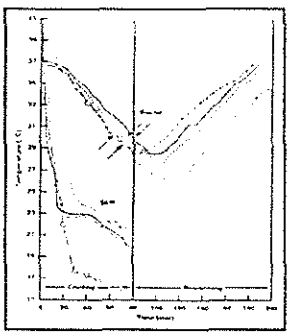


Limits of core temperature tolerance:

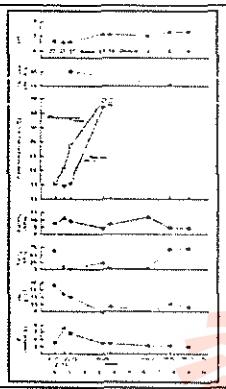
- Core temperatures:
  - 28°C: ventricular fibrillation, no pupillary reflexes
  - 30°C: loss of consciousness
  - <35°C: hypothermia
  - 35-39°C: normal range
  - >39°C: hyperthermia
  - 40-42°C: high intensity protracted endurance exercise
- Extreme values:
  - survival at 14.4°C (accidental),
  - survival at 9°C (clinical) ,
  - survival at 47°C,
  - people have died at 40°C.



Source: Hayward *et al.* (1975).



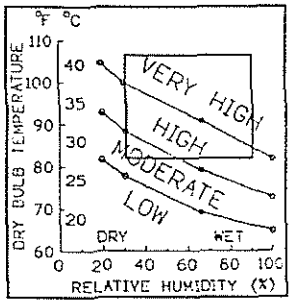
Source: Alexander (1945)

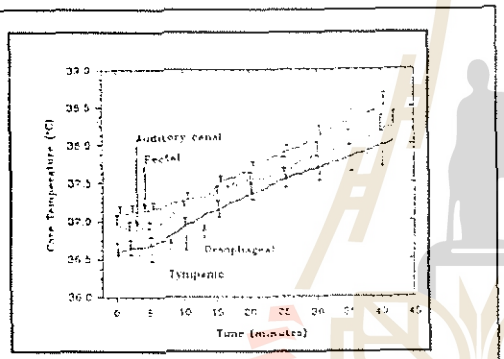


Source: Gilbert et al. (2005)

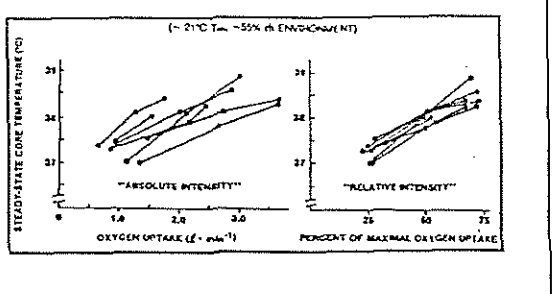
Risk of heat illness

- Environmental conditions affecting heat illness:
  - Critical factors:
    - air temperature
    - relative humidity
    - wind speed
    - radiant heat.
  - Heat stress index: wet bulb globe temperature (WBGT) index:
    WBGT=(0.7 T_{wb})+(0.2 T_g)+(0 .1 T_{db})
    - Note: humidity accounts for 70% of the index.
  - Risk categories:
    - Very high risk: WBGT above 28°C
    - High risk: WBGT 23-28°C
    - Moderate risk: WBGT 18-23°C
    - Low risk: WBGT below 18°C.





Source: Cotter (1995).



Source: Saltin and Hermansen (1966).

Heat injuries

- Heat illness occurs most frequently during first 5 days.
- Heat illness increases probability of subsequent illness.
- The following states may predispose to heat illness:
  - sleep deprivation,
  - infectious disease,
  - excess fatigue,
  - glycogen depletion,
  - sudden increases in training intensity,
  - alcohol or drug abuse,
  - obesity,
  - very low body mass: <50 kg,
  - age: >40 years,
  - poor level of fitness: <40 ml.kg^{-1}.min^{-1},
  - previous heat disorder.

- The following states may predispose to heat illness:
  - various medications:
    - diuretics,
    - anticholinesterases,
    - vasodilators,
    - antihistamines,
    - CNS inhibitors,
    - muscle relaxants,
    - atropine,
    - tranquilisers and sedatives,
    - beta-blockers, amphetamines, propranolol.
  - cardiovascular disease:
    - hypertension (SBP >160 mmHg, DBP >95 mmHg),
    - reduced CV response (lower).

- Three syndromes associated with heat exposure:
  - Heat cramps:
    - only in those that sweat profusely, and
    - occurs early before acclimatisation;
  - Heat exhaustion:
    - due to an inadequate cardiovascular response,
    - the initial increased skin blood flow is not compensated
      - by increased blood volume, or
      - reciprocal vasoconstriction elsewhere;
  - Heatstroke:
    - hyperpyrexia due to relative or absolute thermoregulatory failure.

- Heat cramps:
  - Limb or abdominal cramping associated with heat stress:
    - usually associated with vigorous physical activity, and
    - profuse sweating, but
    - the exact mechanism is uncertain.
  - Incidence:
    - ~1% of all workers.
    - Some clinical examples:
      - cane-cutter's cramp,
      - fireman's cramp,
      - miner's cramp,
      - stoker's cramp.

- Heat exhaustion:
  - Most common form of heat illness:
    - T_c greater than 40°C is defined as heat stroke.
  - Characterised by an inability to continue work, and is frequently seen among the elderly.
  - *Cause:*
    - inadequate cardiovascular response to heat stress;
    - increased skin blood flow is not compensated for by:
      - increased blood volume, or
      - reciprocal vasoconstriction elsewhere;
    - person loses heat tolerance and fatigues.
  - *Symptoms:*
    - pallor (sense of depression or gloom), headache, vomiting,
    - postural syncope, urge to defaecate, giddiness, fatigue,
    - hyperventilation, tachycardia,
    - profuse sweating.

- Heatstroke:
  - Due to relative or absolute thermoregulatory failure.
  - *Diagnosis:*
    - T_c 39.5-41.0°C or more, no sweating (most important factor);
    - *also:* hyperpnoea, altered consciousness, lethargy, stupor, coma;
    - convulsions may occur;
    - elevations in blood urea nitrogen may be present (*e.g.* 20-40 mg/100 ml common).
  - Predisposing factors:
    - use of diuretics,
    - skin diseases,
    - spinal lesions (autonomic nerves severed).

- Heatstroke:
  - Predisposing factors:
    - obesity (SA/mass and increased muscle work),
    - alcohol consumption (↑ muscular work, diuresis, vasomotor control),
    - acute viral or bacterial infections (T_c already high),
    - use of anticholinergic agents (inhibit sweating),
    - Parkinson's disease (medication inhibits sweat and muscular work is high, thus greater heat production),
    - single most important factor in the aged:
      - cardiovascular disease,
      - such patients do not ↑ heat tolerance over time.

- Heatstroke:
  - Severe cases can result in:
    - extensive muscle damage,
    - myoglobinuria (myoglobin in urine),
    - oliguria (decreased ability to form and pass urine),
    - myocarditis (inflammation of myocardium),
    - muscle and neural necroses (localised tissue death),
    - haemorrhages of the skin,
    - gastrointestinal bleeding (in some severe cases),
    - denaturation of protein and blood coagulation.

- Heatstroke:
  - *Therapy:*
    - wet towels or cold bath,
    - constant skin rubbing to ensure skin blood flow is maximised (*i.e.* heat shift from core to skin),
    - oxygen therapy,
    - avoid excessive intravenous fluid replacement unless clinical dehydration exists:
      - such therapy can produce pulmonary oedema,
    - antipyretic drugs (*e.g.* aspirin) are not effective:
      - their action requires a functioning heat loss systems, and
      - may also produce intestinal bleeding,
    - T_c may be unstable for several days, and may show secondary elevation.

- Rhabdomyolisis:
  - Derivation:
    - *rhabdos* = rod, *mys* = muscle, *lysein* = loosen.
    - Meaning: breakdown of muscle tissue.
  - Can occur in both hyperpyrexic and mildly hyperthermic people.

- Heat injury prevention:
  - Sources for detailed information:
    - ACSM position statements: *Med. Sci. Sports & Exerc. (1985).*
    - city-to-surf policy: *Med. J. Aust. (1979) 2:453-474.*
  - Summary of this information:
    - identify people at risk,
    - identify work and exercise conditions in which the risk is elevated,
    - establish work and sporting practices which are designed to minimise the incidence of heat disorders;
    - three categories of these practices:
      - worker/competitor education;
      - medical organisation; and
      - work/event organisation.

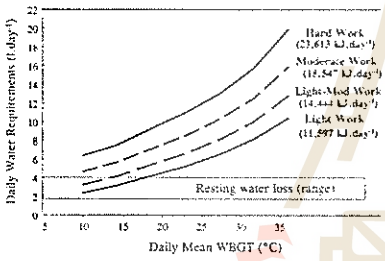
Heat acclimation, thermal failure and hydration:

- Heat adaptation
- Heat adaptation methods
- Thermoregulatory failure
- Dehydration
- Fluid replacement

Physiological consequences of dehydration:

- Increased cardiovascular strain
- Reduced general endurance (performance)
- Reduced local muscle endurance
- Increased thermal strain
- Reduced heat tolerance
- Increased risk of heat illness.

Daily water requirements



Source: Sawka and Montain (2000)

- The consequence of sweating:
  - *Progressive dehydration*
    - 1 litre of sweat equates with a 1-litre dehydration
    - 1 litre of sweat weighs about 1 kg
    - >1% dehydration increases cardiac strain
      - 70 kg person: 1% = 0.7 kg
      - limits ability to move heat to the skin (blood)
    - 2-3% dehydration increases chance of heat illness
      - 70 kg person: 3% = 2.1 kg
      - reduces blood volume
      - reduces sweating
      - increases body temperature.

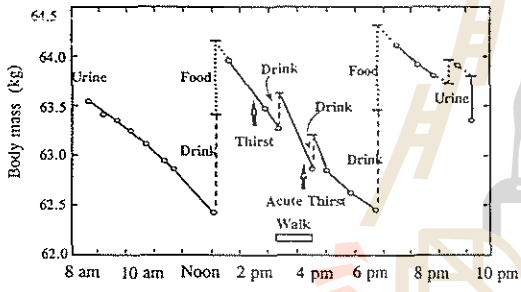
Total body-fluid losses

Exercising adults: average sweat rate = 22 ml/kg/hr

Mass (kg)	Total body fluid (l)	Sweat rate (l/hr)
50	27.5	1.1
55	30.3	1.2
60	33.0	1.3
65	35.8	1.4
70	38.5	1.5
75	41.3	1.7
80	44.0	1.8
85	46.8	1.9
90	49.5	2.0
95	52.3	2.1
100	55.0	2.2

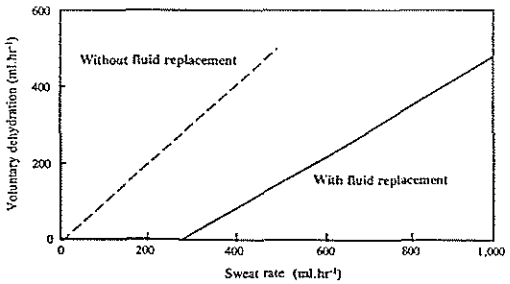
Required fluid intake:
- rest: 1.6 ml/kg/hr
- exercise: 22.2 ml/kg/hr
- total: 23.8 ml/kg/hr

Body changes (20 h) during desert march: fluid and food



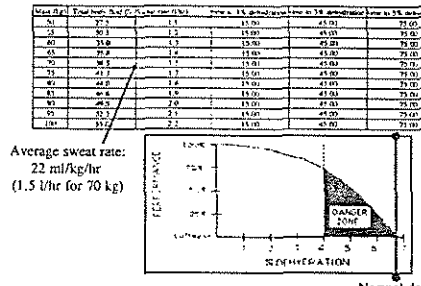
Source: Adolph *et al.* (1947)

Voluntary dehydration



Source: Adolph *et al.* (1947)

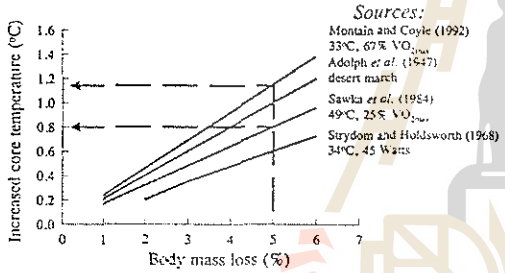
Dehydration



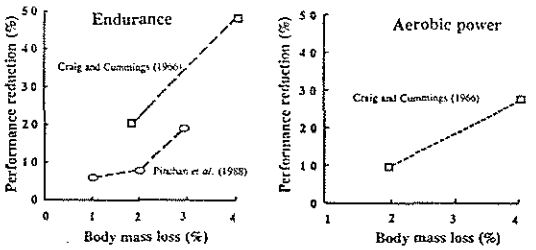
Average sweat rate:
22 ml/kg/hr
(1.5 l/hr for 70 kg)

Normal daily loss:
zero fluid replacement

Core temperature changes with progressive dehydration



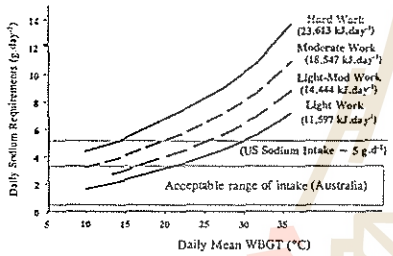
Dehydration and reduced physiological performance



Physiological consequences of substrate depletion:

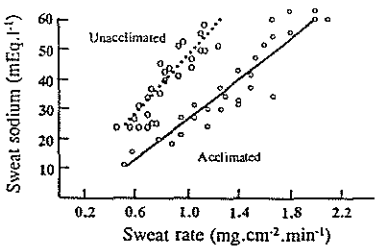
- Electrolyte loss leads to:
  - dehydration
  - hyponatremia
  - muscle cramps.
- Muscle glycogen depletion leads to:
  - reduced exercise performance
  - reduced mental performance.

Daily sodium requirements



Source: Sawka and Montain (2000)

Sweat rates and sodium loss



Source: Alan and Wilson (1971)

Heat acclimation, thermal failure and hydration:

- Heat adaptation
- Heat adaptation methods
- Thermoregulatory failure
- Dehydration
- Fluid replacement

Dehydration and heat illness

- The solution:
  - **Identify people** at risk
  - **Identify conditions** in which the risk is elevated
  - **Establish practices** to minimise heat disorders
    - Three general categories:
      - (i) *worker/competitor education:*
        - preparation for heat exposure
        - clothing
        - diet
        - water replenishment
        - alcohol and drug use
        - the hazard of exposure while ill
        - signs and symptoms of heat illnesses.

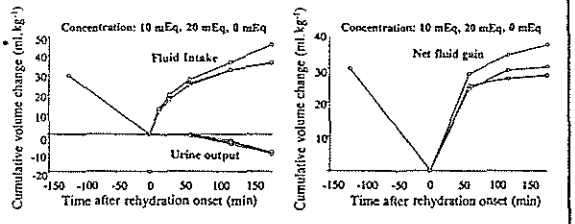
- The solution:
  - Three general categories:
    - (i) *worker/competitor education:*
    - (ii) *medical organisation:*
      - aid centres
      - personnel trained in detection of those at risk
    - (iii) *work organisation:*
      - modify duties according to environment.

- The solution:
  - Fluid replacement:
    - Before exercise/work:
      - 1.5 litres on night before
      - 0.5 litre 2 hours before
    - During exercise/work:
      - 0.5 litre per hour for short exposures
      - 1 litre per hour for long exposures
      - frequent small volumes better
      - larger, less frequent volumes less effective.
    - After exercise/work:
      - replace mass lost (non-alcoholic)
      - ensure healthy diet to replace electrolytes.

Thermal effects of fluid consumption

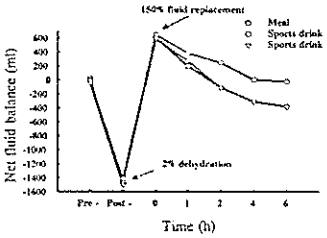
Mass (kg)	Fluid volume (ml)	Core temp (°C)	Mean body temp (°C)	Fluid temp (°C)	Change in core temp (°C)	Heat flux (kJ)
70	250	38.0	37.2	5.0	-0.14	34.49
70	500	38.0	37.2	5.0	-0.28	68.97
70	750	38.0	37.2	5.0	-0.43	103.46
70	1000	38.0	37.2	5.0	-0.57	137.94
70	250	38.0	37.2	10.0	-0.12	29.56
70	500	38.0	37.2	10.0	-0.24	59.12
70	750	38.0	37.2	10.0	-0.36	88.68
70	1000	38.0	37.2	10.0	-0.48	118.24
70	250	38.0	37.2	15.0	-0.10	24.64
70	500	38.0	37.2	15.0	-0.20	49.27
70	750	38.0	37.2	15.0	-0.30	73.91
70	2525	38.0	37.2	15.0	-1.01	248.75

Sodium concentration and beverage consumption



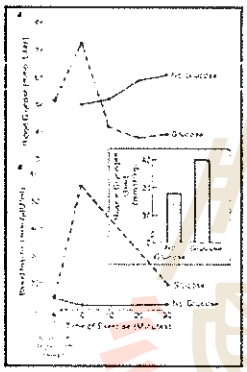
Source: Wemple *et al.* (1997)

Sports drinks are less effective than eating



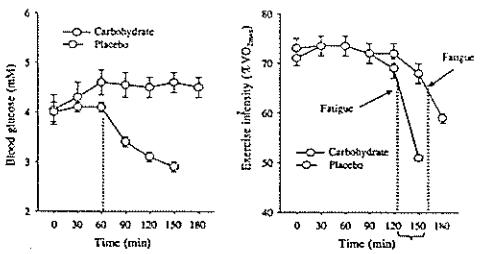
Source: Maughan *et al.* (1996)

Glucose ingestion prior to exercise



Source: Costill *et al.* (1977)

Carbohydrate ingestion (~1 g.min^{-1}): blood glucose and endurance

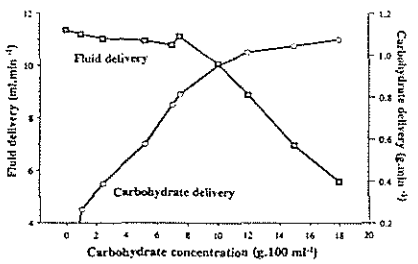


Source: Coyle *et al.* (1983)

- The solution:
  - Electrolyte replacement:
    - Short duration:
      - not required
    - Long duration (>4-5 hours):
      - may be beneficial
      - 0.5 grams per litre
      - improves palatability of fluid
  - Carbohydrate replacement:
    - Short duration:
      - not required
    - Long duration (>4-5 hours):
      - concentration between 3-8% (3-8 g per 100 ml)
  - Key point:
    - sweat is 99% water; replace with 99% water.

- The solution:
  - General fluid prescription at work:
    - start drinking early
    - do not wait for thirst sensation
      - thirst = dehydration: thirst prevention is the key
    - drink frequently: every 15 minutes
    - aim to replace weight loss
      - or maximum that can be tolerated
    - keep fluids cooler than air temperature (15-22°C)
    - flavour drinks to increase palatability
      - increases desire to drink
    - long duration:
      - carbohydrate (50 g) & electrolytes (0.5 g) each hour
      - Thus: drink a total of 1 litre of water containing 50 g glucose and 0.5 g salt each hour.

Carbohydrate concentration:
affects on fluid and carbohydrate delivery



Source: Costill (1990).

HABITUAL PHYSICAL ACTIVITY ON FLOW-RESISTIVE WORK OF BREATHING DURING EXERCISE: A REVIEW

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Heat loss during physical activity is generally via conduction, convection and radiation (60-75%) and another 25-40% via evaporation and insensible water loss (ACSM, 1984). At certain level of exercise when heat produced exceeds capability of body regulation, there is an additional cooling method, the evaporative cooling via respiratory tract through panting, taken place. As respiratory rate increases, this rapid shallow breathing obviously causes heat evaporation from upper respiratory tract, but this is associated with alkalosis of the body (Haymes, 1984). Furthermore, panting itself also requires muscular work, which produces heat in order to overcome the elastic and dynamic properties of the respiratory tract. This is one of the physiologic constraints that impact on human thermoregulatory system.

Dynamic work of breathing of the lung, to overcome flow resistance, is determined mainly by tidal volume (V_T), and pulmonary flow-resistance (R_L) (Otis *et al.*, 1964). Two types of pulmonary flow-resistance to transport of air into and out of the lung have been specified as: (a) resistance due to lung tissue deformation (lung tissue resistance or tissue viscous resistance, R_{lt}, and (b) resistance due to airway friction (airway resistance, R_{aw}, including upper airway, glottis, and tracheobronchial tree) (Fry *et al.*, 1954; Frank *et al.*, 1957).

HABITUAL PHYSICAL ACTIVITY AND DYNAMIC LUNG FUNCTION.

Habitual physical activity offers numerous physiological benefits on the ventilatory responses in young adults with increases of respiratory flow, pressure, and ventilatory capacity (Leith and Bradley, 1976; Fanta *et al.*, 1983; Clanton *et al.*, 1987). On the contrary, reductions in ventilatory capacity were reported after long-term training, which was related to the reduction of circulating lactate, epinephrine and nor-epinephrine (Casaburi *et al.*, 1987). The effect of physical training in young adults on the ventilatory work was reported by Milic-Emili and co-workers (1962), where trained subjects had smaller ventilatory costs than the untrained subjects at a given oxygen uptake.

Table 1. Changes of flow-resistance of the lung (R_L), airway (R_{aw}), and lung tissue (R_{It}) during exercise.

Source	Age (years)	Flow-Resistance
Chiang *et al.*, 1965.	15-35	Unchanged (R_L)
McIlroy *et al.*, 1954.	21-35	Decrease (R_L)
Stubbing *et al.*, 1980.	22-65	Unchanged (R_L)
Granath *et al.*, 1975.	26-37	Unchanged
Johnson *et al.*, 1991.	63-74	Increase (R_L)
Johnson and Dempsey, 1991.	70	Increase (R_{aw}, R_{It})
Butler *et al.*, 1960.	75-90	Unchanged (R_{aw})

Flow-resistance of the lung and its component during exercise has been previously reported with unequivocal results (Table 1). Some investigators indicated that increased in flow-resistance was due to imbalance between tension in the airway and alveolar walls and the opposing transmural pressure gradient (Johnson and Dempsey, 1991; Johnson *et al.*, 1991), others indicated that changes of these forces during exercise are well balance (Butler *et al.*, 1960; Chiang *et al.*, 1965).

Work against dynamic resistance at rest is about 20-40% of total ventilatory cost (McIlroy *et al.*, 1954). However, during exercise, it was reported that this flow resistive work increased as an exponential function of workload (Holmgren *et al.*, 1973). At maximal exercise intensity, Johnson and co-workers (1991) reported the increased of total respiratory work, inspiratory, and expiratory work up to 69-, 65-, and 73-fold from resting levels, in which 60-70% of total ventilatory work was spent to overcome expiratory flow limitation during exercise. At maximum intensity, ventilatory work increases by 6% from resting level in young untrained subjects and 13% in the older habitually-active subjects (Johnson and Dempsey, 1991). They also found that expiratory pulmonary resistance ($R_{L(E)}$) increases to a greater extent than inspiratory pulmonary resistance ($R_{L(I)}$), which is particularly observed in older subjects. Thus, in both young sedentary and the older habitually-active persons, increased work of breathing during exercise is primarily due to increased of expiratory flow resistance. Narrowing of small airways, with the concurrent increases of airflow resistance in the aged lung, has been reported (Niewoehner and Kleinerman, 1974), in association with the reduction of lung elastic recoil (Colebatch *et al.*, 1979). Thus, it is likely that even in physically-active person more ventilatory work is spent to overcome expiratory flow limitation during exercise. In the aged lung, the ability to resist compressing forces, the high pressure from the surrounding tissues, during expiration

is further diminished.

ESTIMATION OF DYNAMIC WORK OF BREATHING

A variety of different methods for estimating dynamic work of breathing have been previously determined. For example, respiratory work can be estimated from the estimation of the oxygen cost of breathing (Barlette *et al.*, 1958), from analysis of the pressure-volume diagram during spontaneous breathing (Otis, 1964), during mechanical ventilation (Coussa *et al.*, 1993), and from predictive equations (Otis *et al.*, 1950; Magaria *et al.*, 1960). Work of breathing obtained from the integration of the pulmonary pressure-volume loop was found to be equivalent in outcome to method which derived work using the other techniques (Sekizawa *et al.*, 1984). With the integration method, the pressure-volume relationships of inspired and expired air must be separated using two sets of low resistance two-way, non-rebreathing valves with pneumotachograph assembly (Figure 1). Intrapleural (P_{Pl}) and mouth (P_m) pressures are determined using differential pressure transducers.

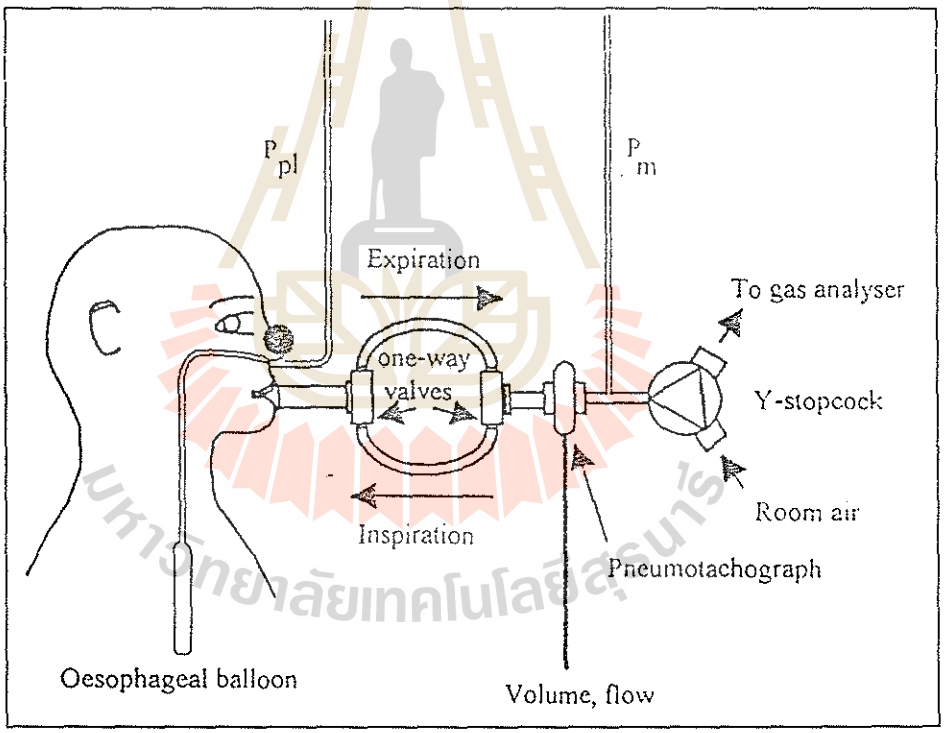

Figure 1. Schematic of breathing circuit used during exercise. Inspired and expired pressure-volume relationships are measured separately.

With the correction of phase shift between flow and transpulmonary pressure (P_{tp}) signals, total dynamic work of breathing of the lung (W_{pul}) from the integration of P_{tp} with respect to volume over a complete respiratory cycle can be derived. As spontaneous ventilation

varies between subjects, W_{pul}, is generally presented as Joule (J), and normalised to tidal volume (Joule per liter, $J.l^{-1}$) (Taylor and Morrison, 1991). It is assumed that the dynamic compliance $C_{dyn(l)}$ always shows its linearity as indicated by the line LT between zero flows at the end-inspiration and end-expiration (Figure 2).

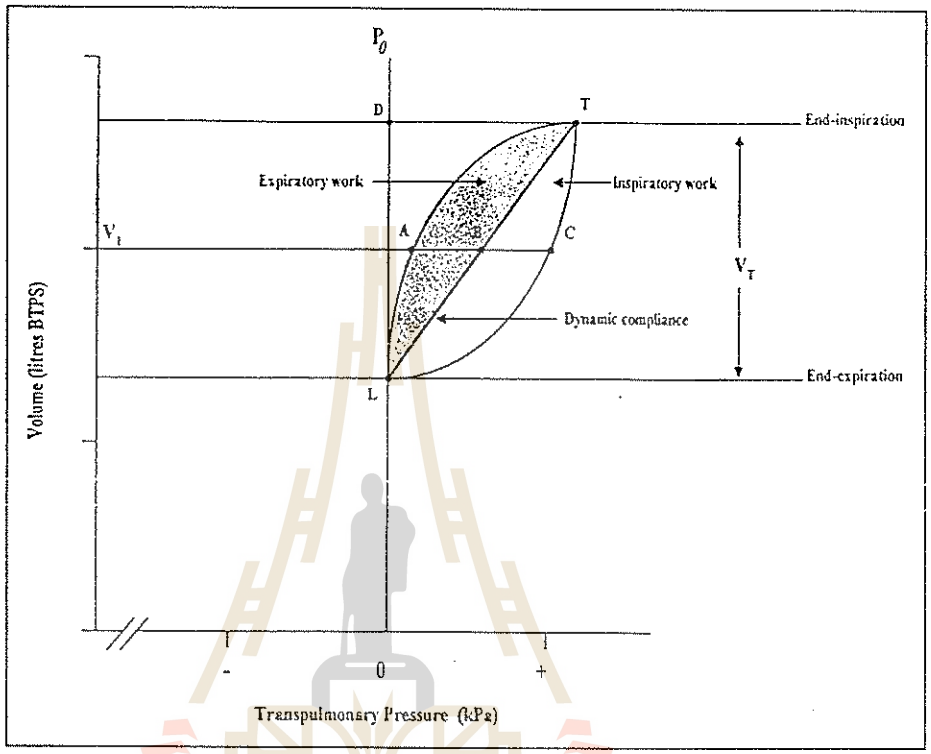

Figure 2. Schematic of the pulmonary dynamic work using transpulmonary pressure-volume loop. Line LT represents dynamic compliance of the lung during volume changes (V_T), areas LCTBL, TALBT and LCTAL represent inspiratory, expiratory and total dynamic works respectively. Lines CB and BA represent flow-resistive pressures required to inflate and deflate the lungs at volume V_1 respectively.

Pressure gradient required to overcome elastic force of the lung during inspiration equals to the horizontal distance from point B on the dynamic compliance LT to zero axis and pressure gradient to overcome inspiratory flow-resistance equals to pressure at point C minus pressure at point B. Since total mechanical work of the lung during inspiration (LCTD) is to overcome both the elastic (LTD) and flow-resistive components (LCTBL), then inspiratory flow-resistive work of the lung equal to total mechanical work of inspiration minus the elastic work (LCTD-LTD).

This is equivalent to the integration of area bounded by the inspiratory pressure-volume relation follow line LCT and the dynamic lung compliance LT. With the same principles, expiratory flow-resistive work of the lung is computed by integration of area bounded by expiratory pressure-volume curve (TALBT) and dynamic compliance LT. Because the expiratory flow-resistive work lies entirely within the elastic work of inspiration, no work is required from the expiratory muscles, and energy stored during inspiration is used to overcome flow-resistive forces of expiration. This area represents energy dissipated during the early phase of expiration where inspiratory muscles performed the lengthening contraction. Thus, inspiratory muscles performed negative work assisting expiration (Otis, 1950; Otis, 1954; Taylor and Morrison, 1991). Total dynamic work of the lung (area LCTAL) is derived from summation of inspiratory (LCTBL) and expiratory dynamic work (TALBT).

ESTIMATION OF FLOW RESISTANCE (R_L): In this review, a term pulmonary flow-resistance is used to represent the combination of airway resistance (R_{aw}) and lung tissue resistance (R_{lt}). Resistance to airflow within the lung can be expressed as a pressure drop per unit flow. Based on the dynamic pressure-volume loop, which was superimposed on the $C_{dyn(l)}$ and by assuming that $C_{dyn(l)}$ is constant during each respiratory cycle, the inspiratory and expiratory flow-resistance can be obtained. To do this, values for instantaneous flow-resistive pressures during inspiration and expiration were calculated, from the pressure difference between P_{tp} of dynamic transpulmonary pressure-volume loop and P_{tp} of the compliance, and divided by inspiratory and expiratory flows at a given volume (after Mead and Whittenberger, 1953).

For example, flow-resistive pressure during inspiration is equal CB. The ratio of CB and inspiratory flow at that particular volume (V_1) provides inspiratory flow-resistance. The pressure gradient required to overcome expiratory flow-resistance is opposite in sign (-AB) but represents by the magnitude BA. Thus, expiratory flow-resistance during expiration equals to BA/expiratory flow. Specific computer software might be developed in order to computed these flow-resistive pressures with the corresponding volume, then averaged this ratio from the starting until the end of inspiration, and repeat the same procedure from beginning of expiration toward the end of expiration. Total flow-resistance is then averaged from the algebraic summation of inspiratory and expiratory flow-resistance for the full breathing cycle. Average inspiratory resistance (R_I), average expiratory resistance (R_E) and average total pulmonary resistance (R_L) are computed

for the full breathing cycle with sampling rate of 50 Hz. Thus, this method represents the precise values of the average flow-resistance across the whole breathing cycle. Flow-resistance will be expressed as $kPa.l^{-1}.s$.

RESPIRATORY FLOW AT REST AND DURING EXERCISE.

It is generally known that airways become wider during inspiration and narrower during expiration. Lung tissue is stretched during inspiration to accommodate the increase in lung volume and, as a result, airway diameters are enlarged (Marshall and Holden, 1963; Fry *et al.*, 1954). Flow resistance generally alters in an inverse proportion with airway caliber (Granath *et al.*, 1959). Accordingly, inspiratory flow always tends to be greater than expiratory flow and some subjects may experience an expiratory flow limitation at higher ventilation.

A number of factors involved with such flow limitation have been defined. These include changes in the elastic recoil of the lung, airway diameter, differences in lung size, breathing frequency, and time dependence of maximum flow (Frank *et al.*, 1957; Briscoe and Dubois, 1958; Fry *et al.*, 1960; Chiang *et al.*, 1965; Mellisinos *et al.*, 1979; McNamara *et al.*, 1987). An important factor affecting airway diameter is transmural pressure. At normal end-expiration, alveoli and small airways are kept open by positive transmural pressure across alveolar wall (Fry *et al.*, 1954). During forced expiration, when a high intrathoracic pressure is developed, transmural pressure is diminished as expired air is pushed downstream toward the airway opening (Dubois, 1964). When transmural pressure reverses, pressure inside the airway is less than surrounding pressure, and the alveoli and some small airways, which have low air conductance and high resistance (Hughes *et al.*, 1972; McNamara *et al.*, 1987), may become smaller and possibly even compressed. As a result, some alveoli and small airways will be collapsed, while others remain open during forced expiration. Thus, as lung volume decreases, pulmonary resistance becomes progressively increased due to the resistance produced within by the air passages and alveoli, particularly at lower lung volume (Fry *et al.*, 1954; Campbell *et al.*, 1957). This effect may be magnified during exercise in persons who have an uneven alteration of the lung tissues and airway tissues (Edge *et al.*, 1964; Niewoehner and Kleinerman, 1974). As there is the reduction of pulmonary elastic recoil which is equivalently occurred at all lung volume, it is likely that, at any given lung volume, the airway diameter will be less, which results in the increased in pulmonary flow-resistance. Based on these principles, it may be assumed that a person may have lower expiratory flow at a given exercise intensity. Under the resting condition, the effect of a cyclic change in airway calibre during inspiration and expiration is

minimal, but during forced expiration, it becomes a critical factor leading to exercise intolerance (Stubbing *et al.*, 1980). Previous investigations show that as ventilation increases with exercise, the uniformity of gas distribution in untrained subjects, who were exposed to resistive loading, becomes impaired (Hanson *et al.*, 1965), and air flow is then independent of the elasticity of the lung (Hughes *et al.*, 1972). Thus, higher inspiratory and expiratory flows in habitually-active subjects, during exercise, may be the results of a uniform distribution of air within the lung.

CONCLUSION

In summary, evidence from previous literature shows that habitual physical activity enhances some aspects of ventilatory capacity. On the other aspects, other investigators indicated that this factor fails to offset the age-related alteration of the lung. Increases of dynamic work of breathing, even in the habitually-active subjects, during exercise is likely to be associated with increases of both inspiratory and expiratory flow-resistances. Narrowing of small airways in which 60-70% of total ventilatory work was spent to overcome expiratory flow limitation is indicated. With positive transmural pressure across alveolar wall, these small airways diameters are kept open even at normal end-expiration. During forced expiration or at high respiratory rate, when transmural pressure becomes gradually diminished or even reverse from high intrathoracic pressure. It is critical that pressure inside the airways is now less than surrounding pressure, airways diameters become smaller and will be collapsed. This effect may be magnified during exercise, particularly in persons who have defects of the lung tissues. At a given exercise intensity, this expiratory flow limitation becomes a critical factor leading to exercise intolerance. It is likely that habitual physical activity affects pulmonary mechanisms by increasing total, and expiratory dynamic work in younger persons, which may be related to their high minute ventilation. On the other hand, a lack of habitual physical activity was associated with an increased total pulmonary flow resistance, and expiratory flow resistance during exercise in the aged group.

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ระบบประสาทอัตโนมัติและความแปรปรวนของอัตราการเต้นหัวใจ

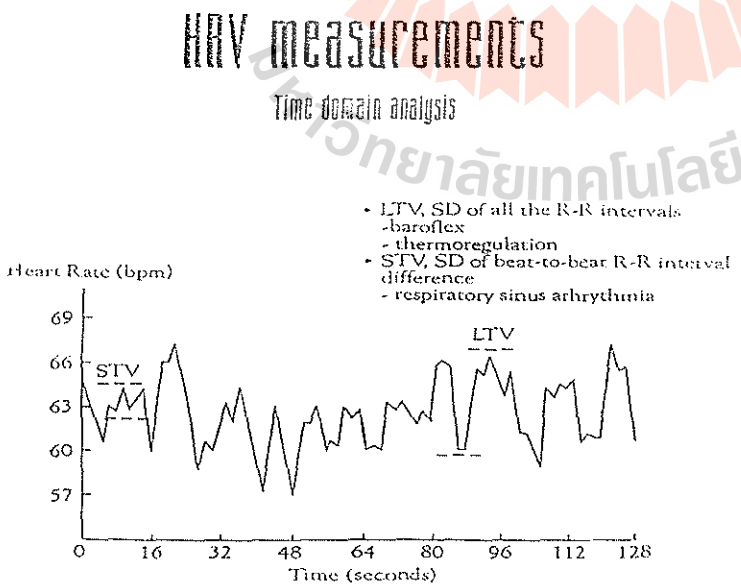
(AUTONOMIC NERVOUS SYSTEM AND HEART RATE VARIABILITY)

ชัยสิทธิ์ ภาวิลาส

ฝ่ายวิทยาศาสตร์การกีฬา การกีฬาแห่งประเทศไทย

มี.ค. 46

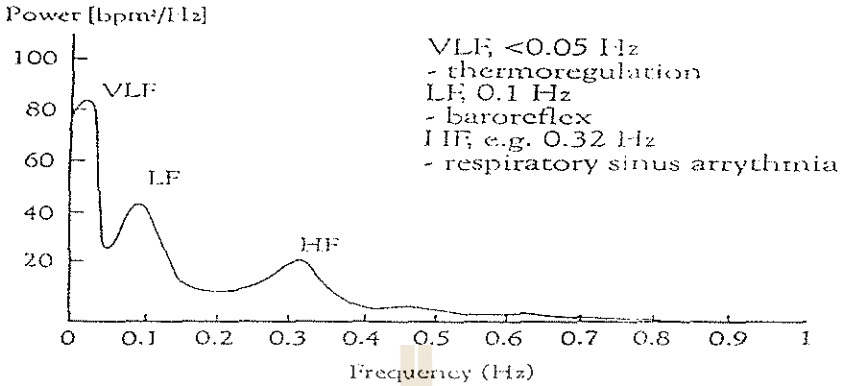
การนำผลการเปลี่ยนแปลงทางด้านสรีรวิทยา (Physiology) ของร่างกาย อาทิเช่น ความแข็งแรงกล้ามเนื้อ สมรรถภาพหัวใจและปอด มาใช้ติดตามประเมินผลประสิทธิภาพของการฝึกซ้อมในนักกีฬาด้านความสมบูรณ์ร่างกาย (Physical Fitness) นั้นเป็นไปอย่างแพร่หลาย และยอมรับกันอย่างกว้างขวางในทุกประเทศ ซึ่งประเทศมหาอำนาจทางกีฬา ได้มีการวิจัยและค้นคว้ากันอย่างต่อเนื่องตลอดเวลา ในปัจจุบันหลายประเทศได้เริ่มการศึกษาความสมดุลย์ของการควบคุมระบบประสาทอัตโนมัติในกีฬา เพื่อบ่งชี้ถึงสภาพด้านจิตใจและความเครียดของนักกีฬา ซึ่งทางด้านร่างกาย (Physiological stress) จะมีผลทำให้ได้แก่ มีการหลั่งเหงื่อเพิ่มขึ้น หายใจถี่ตื้น กล้ามเนื้อเกร็ง เป็นต้น ส่วนทางด้านจิตใจ (Psychological stress) จะเกี่ยวกับทางพฤติกรรม เช่น มือสั่น เสียงสั่น นอนไม่หลับ รวมถึงความแปรปรวนทางอารมณ์และความคิด[1] ซึ่งจะพบว่า ทั้งสองด้านจึงมีความเกี่ยวพันกันอย่างสูงมาก จึงมีคำว่า "Psychophysiology" เกิดขึ้นมา ดังนั้นความแปรปรวนของอัตราการเต้นหัวใจ (Heart rate variability: HRV) จึงเป็นตัวบ่งชี้ที่น่าสนใจ เพราะเกี่ยวข้องกับ ความสมดุลของการควบคุมของระบบประสาทอัตโนมัติ ดังรายงานการวิจัยของ โรเบิร์ต แบร์รี่ และคณะ ที่ได้ศึกษาในนักยิงปืน[2] พบว่าความต้านทานของผิวหนัง (Skin conductance) กับการเปลี่ยนแปลงของอัตราการเต้นหัวใจมีความสัมพันธ์กัน



ค่าความแปรปรวนของอัตราการเต้นหัวใจ(HRV) ได้มาจากการวัดคลื่นไฟฟ้าจากหัวใจ(EKG)โดยบันทึกแบบ R-R-interval ซึ่งจะมีค่าตรงกันข้ามกับอัตราการเต้นหัวใจ (heart rate: HR) คือ HR ($b.min^{-1}$) ต่ำ ค่า R-R (ms) จะมีค่าสูง ซึ่ง HRV ยังแบ่งการวิเคราะห์ออกเป็น 2 แบบ คือ แบบขึ้นกับเวลา (time domain analysis) และแบบขึ้นกับความถี่ (frequency domain analysis)[3,4] ดังรูป

HRV measurements

Frequency domain analysis (spectral analysis)



ค่า HRV ที่วิเคราะห์ตามความถี่ (frequency range) สามารถบันทึกได้ทั้งแบบ Long – term ใน 24 ชั่วโมง แล้วนำข้อมูลมาวิเคราะห์ ส่วน แบบ Short- term จะใช้เวลาในการบันทึกเพียง 5 นาที[5] ในคนทั่วไป สามารถวิเคราะห์ผลได้ ดังนี้

1. 5-min Total power	มีค่าความถี่ ประมาณ 0.4 Hz	
2. VLF (Very low frequency)	มีค่าความถี่ น้อยกว่า 0.04 Hz	ค่าอ้างอิง 773 ± 660 ms^2
3. LF (Low frequency)	มีค่าอยู่ระหว่าง 0.05 – 0.15 Hz	ค่าอ้างอิง 215 ± 272 ms^2
4. HF (High frequency)	มีค่าอยู่ระหว่าง 0.16 – 0.40 Hz	ค่าอ้างอิง 151 ± 165 ms^2
5. LF/HF ratio		ค่าอ้างอิง 2.0 ± 1.7

ค่าที่ได้จากการวิเคราะห์ทั้งสองแบบ จะบ่งบอกถึง การตอบสนองของความสมดุลในการควบคุมระบบประสาทอัตโนมัติ เช่น ในการเปลี่ยนแปลงของ

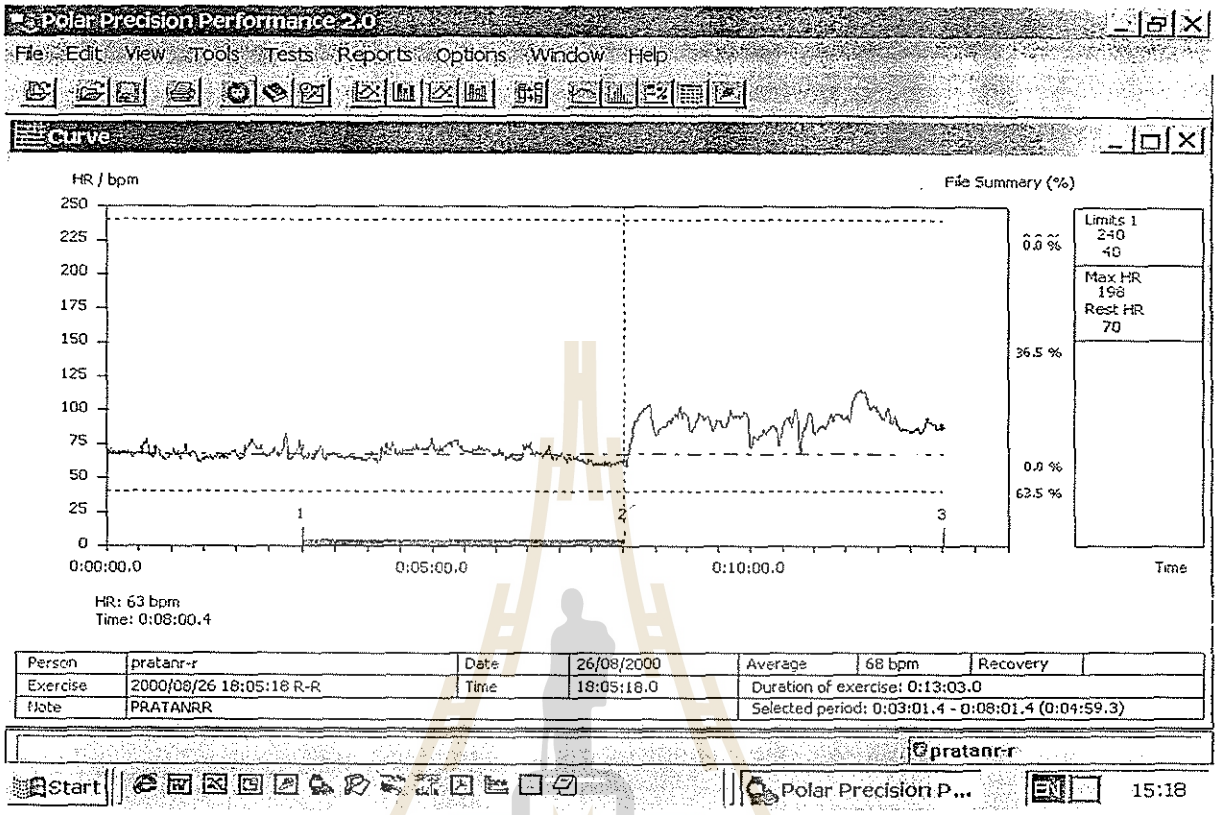
1) Baroreflex แสดงค่าโดย ค่า LF
2) Respiratory sinus arrythmia แสดงค่าโดย ค่า HF
3) Thermoregulation แสดงค่าโดย ค่า VLF

การประยุกต์ใช้ในการออกกำลังกายและเล่นกีฬา[4,5,6]

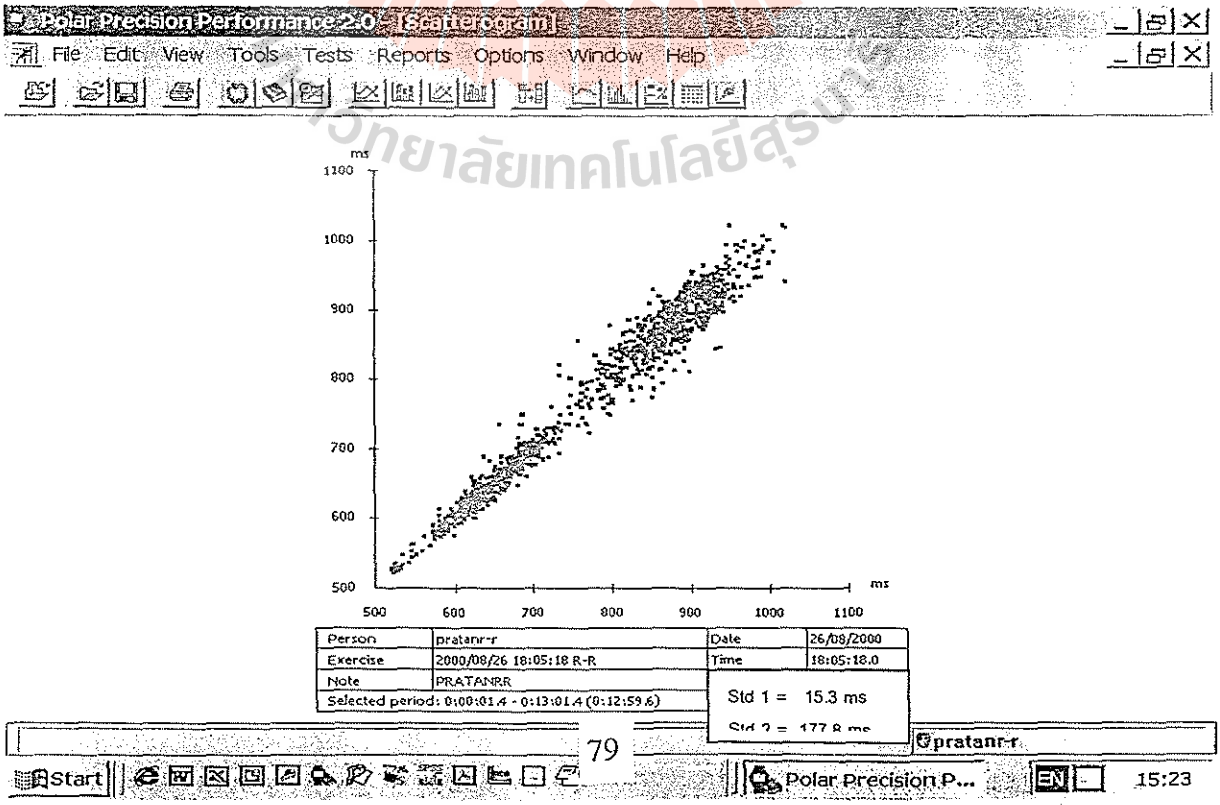
HRV วิเคราะห์แบบขึ้นกับเวลา (time domain analysis)

นักกีฬาที่มีความสมบูรณ์ของร่างกายดีขึ้นนั้น จะพบว่า ขณะพักอัตราการเต้นหัวใจจะต่ำลง และความแปรปรวนของอัตราการเต้นหัวใจ (HRV,SD1,2) มากขึ้น การวิเคราะห์ HRV ในแบบ time domain

analysis นี้ จะสามารถนำค่ามาสร้างเป็นรูปกราฟ (Scatterogram) ดังเช่น การทดสอบ Orthostatic test เพื่อทราบภาวะการฝึกซ้อมเกินหรือไม่ โดยให้นักกีฬานอน และบันทึกอัตราการเต้นของหัวใจแบบ R-R Interval 5 นาที (จาก ตำแหน่งที่ 1 – 2) และลุกขึ้นยืน 5 นาที (จาก 2 – 3) ดังรูป



กราฟแสดงการวิเคราะห์แบบ Scatterogram

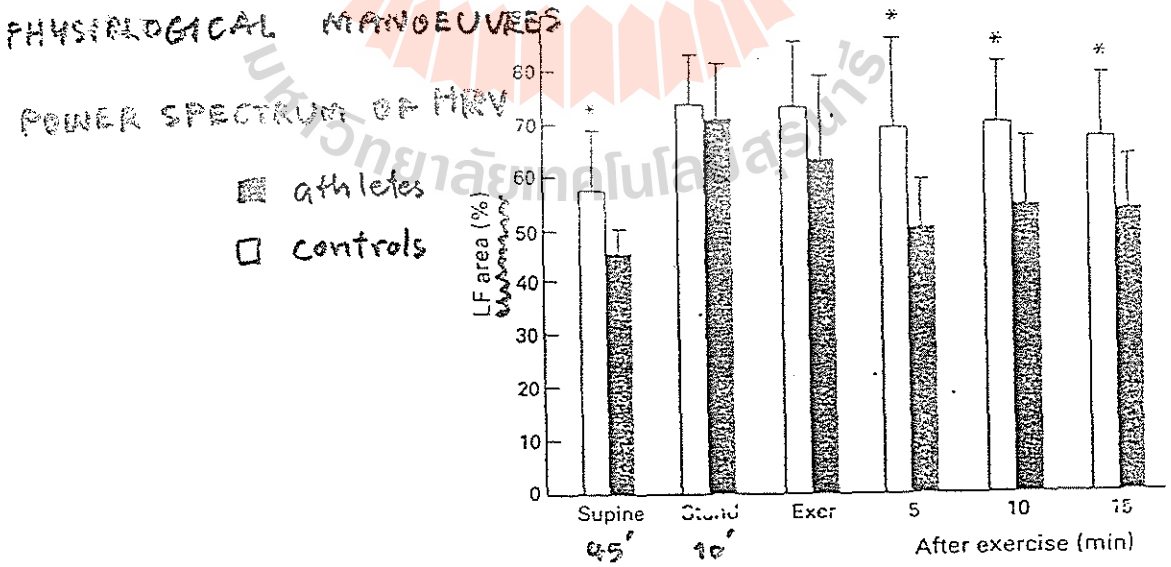


โดยกราฟ จะแสดงค่าออกมาเป็น การเปลี่ยนแปลงในแนวแกน 45 องศา (Standard Deviation, std2) และในแนวตั้งฉากกับแกนเดิม เรียก std1 ซึ่งค่า std2 จะแสดงถึงความแปรปรวนของอัตราการเต้นหัวใจที่เพิ่มขึ้นในขณะยืน นอกจากนั้นค่า std1 จะเป็นหนึ่งตัวแปรหลักที่ใช้ติดตามนักกีฬาดูการเปลี่ยนแปลงของสภาวะผ่อนคลายของร่างกาย (Relaxation rate) ซึ่งจะมีค่าปกติอยู่ระหว่าง 10-100 ms

อย่างไรก็ตาม ในขณะออกกำลังกาย ค่าความแปรปรวนจากการวิเคราะห์แบบใช้เวลานั้น จะมีค่าค่อยๆ ลดลง และเข้าใกล้ หรือเท่ากับศูนย์ เมื่อระบบ Sympathetic flow ทำงานเหนือ (over come) ระบบ Parasympathetic ณ จุดนี้ เมื่อคำนวณค่าจาก RR กลับเป็นอัตราการเต้นของหัวใจ และบวกเพิ่มขึ้นประมาณ 20 ครั้งต่อนาที จากจุดดังกล่าว (ขึ้นกับอายุ ; เฉลี่ย 10 % ของอัตราการเต้นของหัวใจสูงสุด) จะเป็นช่วงที่พอเหมาะในการออกกำลังกายเพื่อสุขภาพ หรือเรียกว่า "Own Zone" ซึ่งจะแปรผันไปตามสภาพร่างกายและจิตใจของคนเราในแต่ละวัน

HRV ที่วิเคราะห์ตามความถี่ (frequency range) [3,5,6]

ค่า Power spectrum of HRV ขณะพัก ในนักวิ่งระยะไกล (long distance runners) จะมีค่า LF ต่ำกว่าคนปกติ และมี ค่า HF สูงกว่าคนปกติ ดังนั้น ค่า LF/HF ratio ในนักกีฬาจะต่ำกว่าอย่างเห็นได้ชัด ซึ่งจะพบค่าดังกล่าวคล้ายกันในขณะยืนขึ้นและออกกำลังกาย รวมถึงหลังจากการออกกำลังกายในนาทีที่ 5, 10 และ 15 ดังกราฟ



Malik M. 1996

Figure 2 As fig 1 except that the effect is on the fractional area contained in the low frequency (LF) band. White columns=controls; black columns=athletes.
**p<0.05 athletes v controls*

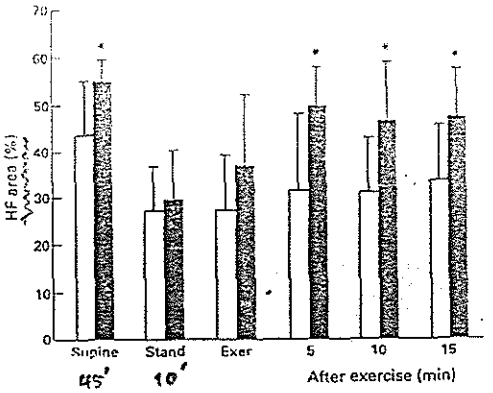


Figure 1 Effect of orthostatic stress (stand) and steady state exercise (exer) on the percentage of total spectral energy contained in the high frequency band (HF area). White columns=controls; black columns=athletes. Columns are means, bars=SD.
**p<0.05 athletes v controls*

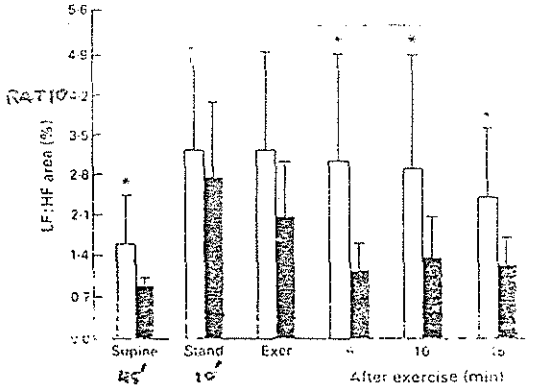


Figure 3 As fig 1 except that the effect is on the ratio of the low (LF) to high (HF) frequency areas. White columns=controls, black columns=athletes.
**p<0.05 athletes v controls*

ทั้งนี้ การตอบสนองของค่า HRV ในขณะพักและออกกำลังในนักกีฬายกน้ำหนัก ซึ่งมีการฝึกกล้ามเนื้อเป็นหลัก จะมีค่าแตกต่างจากนักวิ่งระยะไกล [6] โดยกลุ่มนักวิ่งระยะไกล จะมีการทำงานของ Parasympathetic ที่ควบคุมการเต้นของหัวใจเพิ่มขึ้นในขณะพักมากกว่ากลุ่มนักกีฬาที่ฝึกกล้ามเนื้อ

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การวัดส่วนประกอบร่างกาย
Body Composition Assessment

บุญศักดิ์ หล่อพิพัฒน์
สำนักวิชาวิทยาศาสตร์การกีฬา
จุฬาลงกรณ์มหาวิทยาลัย

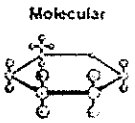
ส่วนประกอบร่างกาย ระดับต่าง ๆ

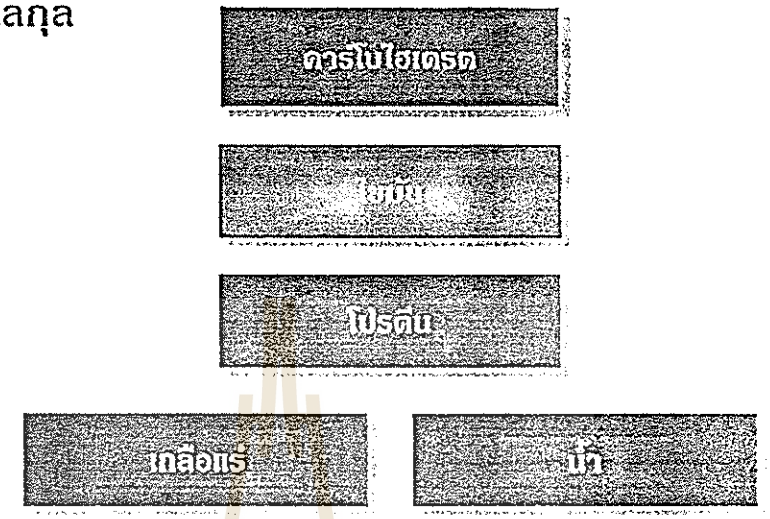
- ระดับอะตอม

ธาตุ	จำนวน (กิโลกรัม)	ร้อยละของมวลร่างกาย
ออกซิเจน	43.0	61.4
คาร์บอน	16.0	22.9
ไฮโดรเจน	7.0	10.0
น	1.8	2.6
แคลเซียม	1.0	1.4
อื่น ๆ	1.2	1.7

ส่วนประกอบร่างกาย ระดับต่าง ๆ

- ระดับโมเลกุล





ส่วนประกอบร่างกาย ระดับต่าง ๆ

- น้ำหนักน้ำในร่างกายทั้งหมด Total Body Water, TBW เป็นส่วนประกอบของร่างกายในระดับโมเลกุล คือ โมเลกุลของน้ำ ซึ่งประกอบด้วย ไฮโดรเจน 2 อะตอม และ ออกซิเจน 1 อะตอม
- น้ำหนักร่างกายทั้งหมดที่ปราศจากน้ำ Dry Weight, DW จึงมีค่าเท่ากับ น้ำหนักตัว ลบด้วย น้ำหนักน้ำใน ร่างกายทั้งหมด
- DW = BW – TBW

ส่วนประกอบร่างกาย ระดับต่าง ๆ

- ระดับเซล

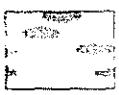
Cellular

- เซลไขมัน
- มวลเซลร่างกาย Body Cell Mass, BCM
- ของเหลวในร่างกาย ICF + ECF
- ของแข็งภายนอกเซล อินทรีย์และอนินทรีย์ ECS

ส่วนประกอบร่างกาย ระดับต่าง ๆ

- ระดับเนื้อเยื่อ

Tissue



- เนื้อเยื่อกล้ามเนื้อ
- กระดูก
- เลือด และ อวัยวะภายในต่าง ๆ

ส่วนประกอบร่างกาย ระดับต่าง ๆ

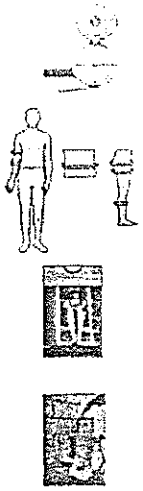
- ระดับทั้งร่างกาย

วัดความหนาของชั้นไขมัน

วัดขนาดเส้นรอบวงส่วนต่าง ๆ

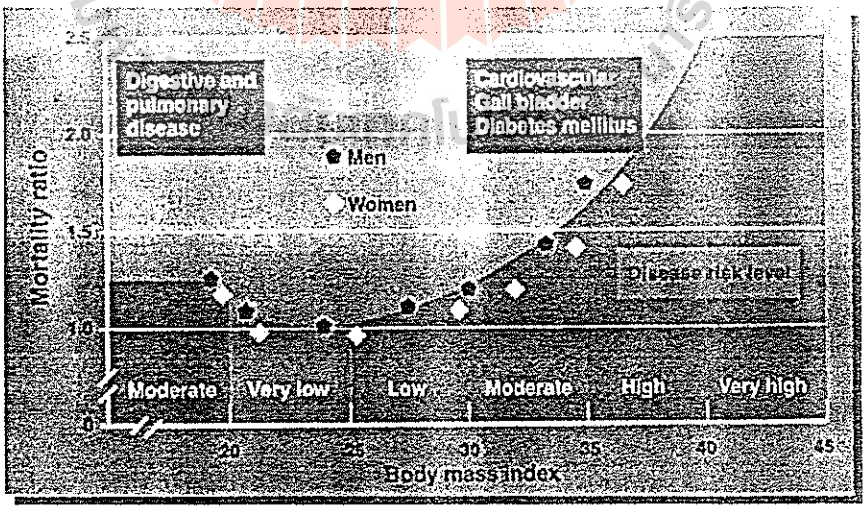
วัดความหนาแน่นของน้ำหนักใต้น้ำ

วัดปริมาตร ทีละส่วน ๆ



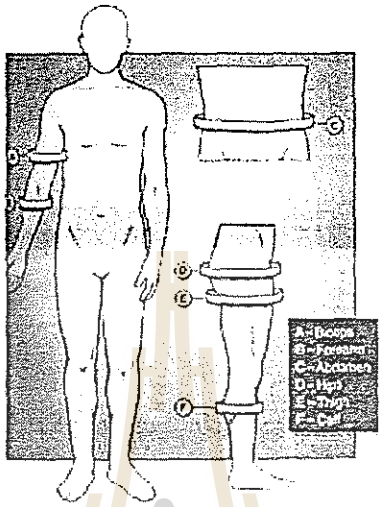
การวัดส่วนประกอบร่างกาย ระดับทั้งร่างกาย

- ดัชนีมวลกาย = น้ำหนักตัว (กก.) / ความสูง (ม)^2



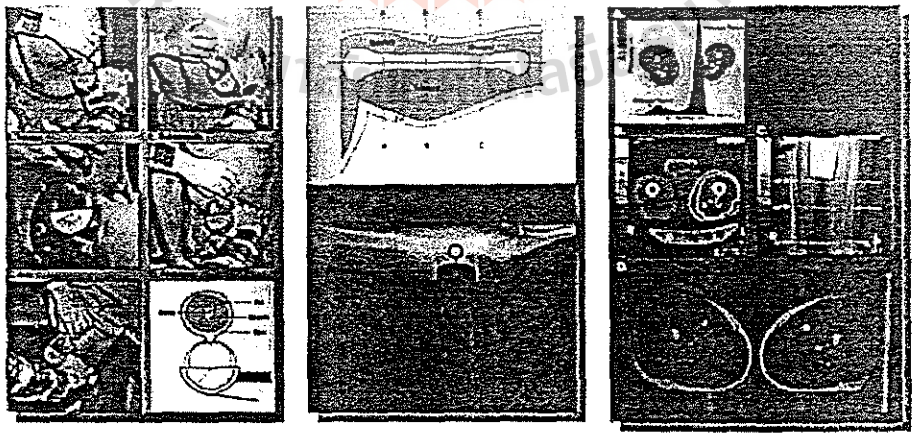
การวัดส่วนประกอบร่างกาย ระดับทั้งร่างกาย

- การวัดขนาดเส้นรอบวงส่วนต่าง ๆ ของร่างกาย



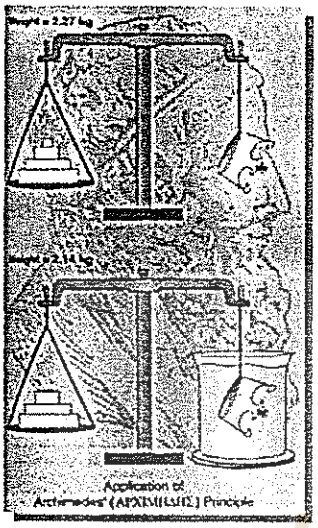
การวัดส่วนประกอบร่างกาย ระดับทั้งร่างกาย

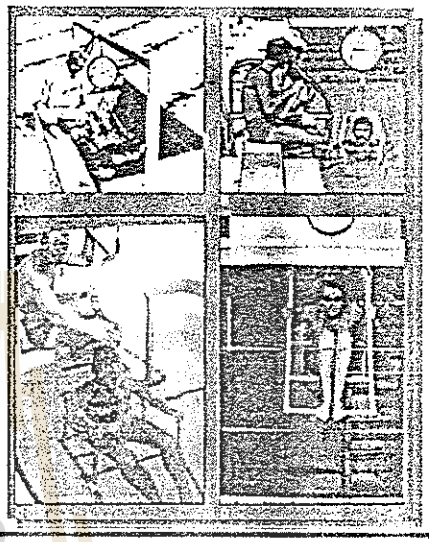
- การวัดความหนาของชั้นไขมัน (Skinfold Fat)



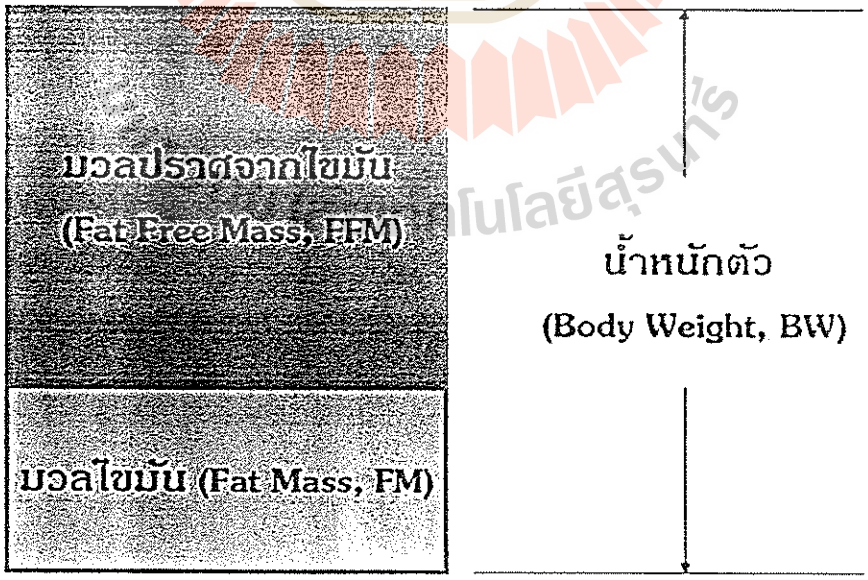
การวัดส่วนประกอบร่างกาย ระดับทั้งร่างกาย

- การวัดความหนาแน่น ชั่งน้ำหนักใต้น้ำ





ส่วนประกอบร่างกาย ระดับทั้งร่างกาย



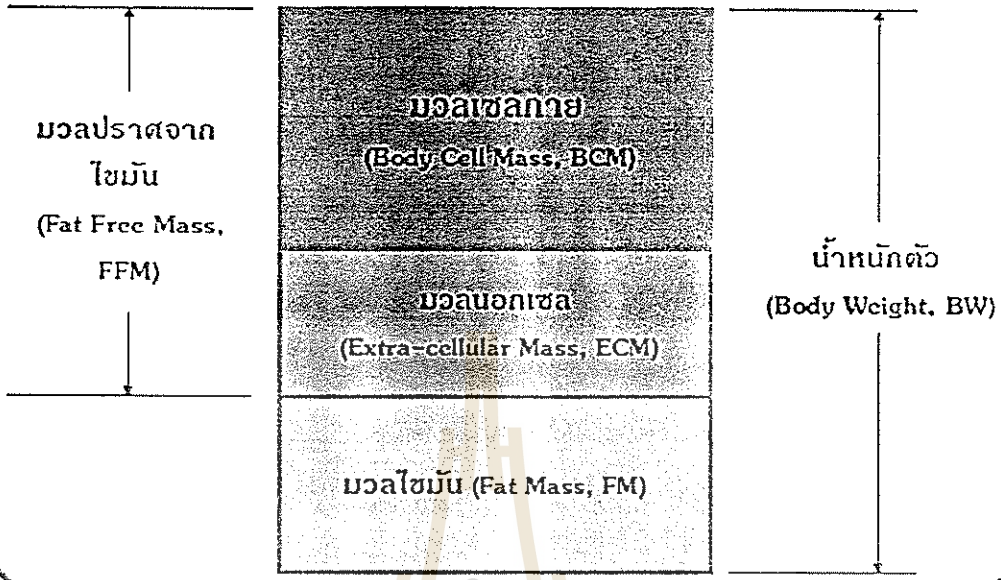
ส่วนประกอบร่างกาย ระดับทั้งร่างกาย

- **มวลปราศจากไขมัน (Fat Free Mass, FFM)** คือ น้ำหนักของร่างกายทั้งหมด ลบด้วย มวลของไขมันที่สามารถสกัดออกมาได้ (extractable fat) ทั้งหมด
- BW = FFM + FM
- ส่วนสำคัญของมวลปราศจากไขมัน (FFM) ได้แก่ กล้ามเนื้อ อวัยวะสำคัญต่าง ๆ กระดูก และของเหลวภายนอกเซล เป็นต้น
- ไขกระดูก ระบบประสาทส่วนกลาง และไขมันภายในเซล ไม่นับรวมอยู่ใน FFM แต่อยู่ใน Lean Body Mass, LBM

ส่วนประกอบร่างกาย ระดับทั้งร่างกาย

- **Lean Body Mass, LBM** คือ น้ำหนักของร่างกายทั้งหมด ลบด้วย เนื้อเยื่อไขมัน (adipose tissue)
- Lean Body Mass, LBM จะรวม ไขมันจำเป็น (essential fats and lipids) กระดูก กล้ามเนื้อ หัวใจ ตับ ไต ด้วย
- Fat Free Mass, FFM จะประกอบด้วย น้ำประมาณ 73%, โปรตีน 20%, แร่ธาตุต่าง ๆ 6%, และขี้เถ้า 1%
- **มวลไขมัน (Fat Mass, FM)** คือ น้ำหนักของไขมันทั้งหมดในร่างกาย

การวัดส่วนประกอบร่างกาย ระดับเซล



ส่วนประกอบร่างกาย ระดับเซล

- มวลเซลกาย Body Cell Mass, BCM เป็นส่วนประกอบของร่างกายในระดับเซล ประกอบด้วย Protoplasm เป็นส่วนที่สร้างพลังงานให้ร่างกาย และทำหน้าที่ต่าง ๆ ที่สำคัญทางด้านการเผาผลาญอาหาร (metabolic functions)
- BCM เป็นดัชนีที่แม่นยำในการแสดงสภาวะโภชนาการ ระดับของทุพโภชนาการ (degree of malnutrition) และใช้ปรับระดับการใช้พลังงาน (energy expenditure) และการเผาผลาญอาหารให้เป็นพลังงาน (metabolism)

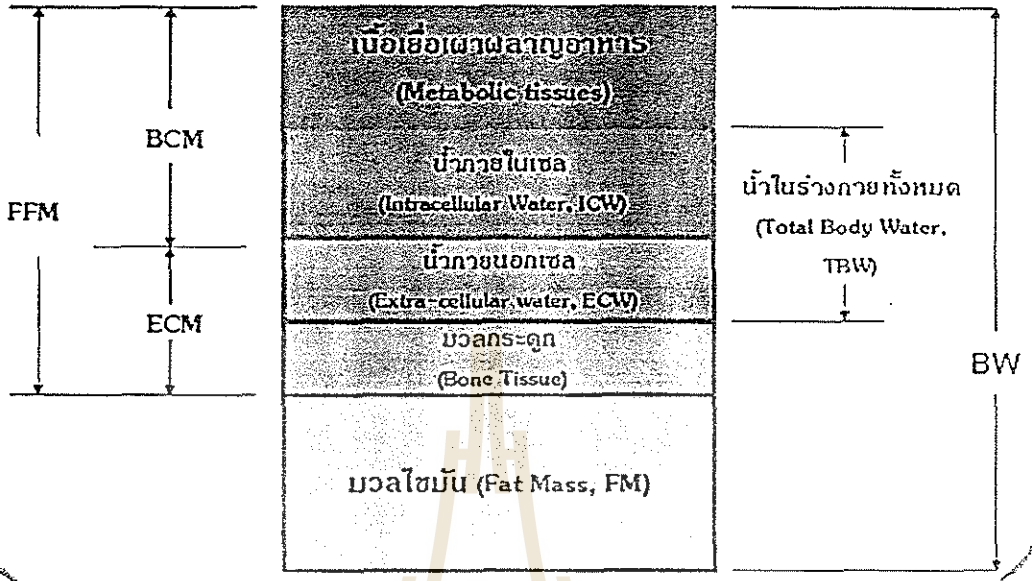
ส่วนประกอบร่างกาย ระดับเซล

- มวลเซลกาย Body Cell Mass, BCM ของผู้ชายปกติ มีค่าระหว่าง 41% - 45% ของน้ำหนักตัว Total Body Weight
- มวลเซลกาย Body Cell Mass, BCM ของผู้หญิงปกติ มีค่าระหว่าง 30% - 33% ของน้ำหนักตัว Total Body Weight

ส่วนประกอบร่างกาย ระดับเซล

- น้ำในร่างกายทั้งหมด Total Body Water, TBW เป็นส่วนประกอบของร่างกายในระดับโมเลกุล มีความสัมพันธ์กับ BCM เนื่องจาก TBW ในระดับเซล จะอยู่ในรูปของ Total Body Fluid ซึ่งเป็นผลบวกระหว่างของเหลวภายในเซล (Intra-cellular Fluid, ICF) กับของเหลวภายนอกเซล (Extra-cellular Fluid, ECF)
- ของเหลว (Fluid) ต่างจากน้ำ (water) ตรงที่มีอิเล็คโตรไลท์ (electrolyte) และสารอื่นละลายอยู่ด้วย
- BCM ประกอบด้วย ICF กับ ICS

การวัดส่วนประกอบร่างกาย ระดับเซล



ส่วนประกอบร่างกาย ระดับเซล

- ของแข็งระหว่างเซล Extra-cellular Solid, ECS ประกอบด้วยแร่ธาตุของร่างกายเกือบทั้งหมด เนื่องจากภายใน ของเหลวภายในเซล (Intra-cellular Fluid, ICF) และของเหลวระหว่างเซล (Extra-cellular Fluid, ECF) มีแร่ธาตุอยู่น้อยมาก
- นอกจากนี้ Extra-cellular Solid, ECS ยังประกอบด้วยของแข็งที่เป็นอินทรียวัตถุ (organic solids) เช่น elastic fibers, collagen, และ reticular fibers เป็นต้น

PRINCIPLES AND PRACTICES OF HEAT ADAPTATION.

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Running head: *Heat acclimation.*

PRINCIPLES AND PRACTICES OF HEAT ADAPTATION.

ABSTRACT:

During prolonged exercise or work in the heat, human thermal homeostasis is first challenged, and eventually lost, as one moves from a compensable state through to uncompensable heat stress. During the first week of such exposure, work and athletic performance is most affected, and the threat of heat illness is greatest. However, given adequate time, the body will undergo a three-phase adaptation to better tolerate the heat. In this review, the principles and practices of the six primary methods by which such heat adaptation may be achieved are evaluated. One technique involves repeated exposure to both heat and exercise, and is designed to elevate and maintain a target body temperature, by varying the intensity of the work rate during the acclimation period: the controlled-hyperthermia (isothermal) technique. It is recommended that this method provides the most dependable, and least hazardous, means of adapting workers and athletes for heat stress.

1. INTRODUCTION:

Body tissues operate as thermal energy reservoirs, with local tissue temperatures varying as a function of the nett heat exchange between the body and environment. Semi-clothed humans employ both autonomic and behavioural mechanisms by which to regulate the thermal status of the *miliue intérieur*. Heat dissipation is primarily mediated via its transportation to the skin surface within the blood. Since the specific heat of blood is relatively high, it is an ideal medium by which to transport heat to the skin surface. On reaching the skin, thermal energy is used to convert sweat into water vapour, thereby removing heat from the body.

Under hot conditions, particularly during prolonged exercise, human thermal homeostasis is first challenged, and may eventually be lost, as one moves from a compensable state through to uncompensable heat stress. This transition is dictated by the combined effects of air temperature and its water vapour pressure, exercise intensity, clothing and its permeability to water vapour, body composition, hydration status, long-term endurance fitness, and state of heat adaptation (Cheung *et al.*, 2000). During heat stress, the cardiovascular system must subserve both the metabolic demands of active skeletal muscle, and the demand for blood flow at the skin, to dissipate metabolically-derived heat. In the short term, most people, particularly endurance-trained athletes, can satisfy this dual demand. Nevertheless, during prolonged exercise in the heat, thermal homeostasis is compromised, resulting in positive heat storage, and eventually uncompensable heat strain. If the heat loading is high enough, or if the exposure duration is long enough, cardiovascular function is compromised, and it can no longer serve both skin and muscle blood flow demands (Kenney and Johnson, 1992). In this situation, blood pressure declines, and cutaneous vasoconstriction ensues. While sweating can be still remain functional, continued exercise will rapidly elevate body core temperature, eventually leading to heat exhaustion and heat illness. Regardless of whether such fatigue is associated with the attainment of a critical body temperature (Fuller *et al.*, 1998; González-Alonso *et al.*, 1999), reduced central drive (Brück and Olschewski, 1987), or reduced metabolic function triggered by oxidative stress (Mills *et al.*, 1996), the end result has the same practical outcome: impaired work performance.

During the first week of an exercising heat exposure, worker and athlete performance is most affected, and the threat of heat illness is greatest (Armstrong and Maresh, 1991). From a

practical perspective, it is during these early days of repeated exposure that workers, and their supervisors, must exercise greatest caution. However, just as humans can adapt to increased physical work, so can they modify the function of both autonomic and behavioural mechanisms for heat dissipation and conservation, to overcome altered thermal stresses. Herein, we shall briefly overview the principal physiological adaptations to heat stress, then emphasise how such modifications may best be achieved for military, industrial and sporting applications.

While transformations within the autonomic control of skin blood flow and sweating are perhaps most frequently associated with heat adaptation, a host of readily observed physiological changes are also apparent. For instance, there is an expansion of the plasma volume, associated with an elevation in, or a superior maintenance of, the osmotic potential of the blood, brought about by either a reduction in the heat-induced loss of plasma protein to the interstitial space (Senay, 1972; Harrison, 1985), or a greater retention of the extracellular electrolyte content (Patterson *et al.*, 1999). This plasma expansion typically wanes with progressive exposures, as adaptation to a constant exercise and thermal stress proceeds (Bass *et al.*, 1955; Wyndham *et al.*, 1968). However, when the physiological strain is maintained during adaptation, using small but progressive increments in exercise intensity, this expanded plasma volume is sustained, with elevations being reported for up to three weeks (Patterson *et al.*, 1998a). Indeed, the entire extracellular space may be significantly enlarged during this type of heat adaptation (Patterson *et al.*, 1998a).

Typically, during an exercise-heat stress, the interstitial and plasma volumes contract, as fluid is drawn to fuel sweating. In the unadapted state, this can result in a reduced stroke volume and elevated heart rate, to preserve cardiac output and systemic blood pressure. However, an expanded plasma volume is better able to withstand this fluid loss without compromising stroke volume. Therefore, it is often seen that, following heat adaptation, and when working at a given intensity, the stroke volume is larger and the cardiac frequency lower (Mitchell *et al.*, 1976; Shapiro *et al.*, 1981; Cadarette *et al.*, 1984). The combined effects of these changes permit an elevation in skin blood flow during heat exposure, and a lowering of the vasodilatory threshold (Fox *et al.*, 1963). Both these mechanisms facilitate a more rapid transference of heat to the periphery for dissipation. Accordingly, people report being less stressed, via thermal and effort sensations, and are better able to tolerate the exercise-heat stress.

Sweating provides our most effective means of heat dissipation within hot environments. This heat dissipation is a form of "mass transfer", because fluid is lost from the body, down the water vapour pressure gradient, and with it, heat is transferred up the thermal energy gradient. That is, heat moves from the cooler skin to the warmer air. Therefore, it is not surprising to observe an enhancement of the sweating response accompanying heat adaptation. In fact, these modifications potentially represent our most potent adaptive responses.

Specifically, there is an increased steady state sweat rate (Libert *et al.*, 1983; Sato *et al.*, 1990; Patterson *et al.*, 1998b), an increased sweat gland sensitivity relative to body temperature changes, and a reduced body temperature threshold for sweating onset (Nadel *et al.*, 1974; Patterson *et al.*, 1998b). These adaptations can produce a two-fold increase in sweat rate, increasing from 1.5 to 3 $l \cdot h^{-1}$. The eccrine sweat glands appear to reabsorb more sodium and chloride from the primary sweat, leading to a better conservation of the extracellular electrolyte content (Allan and Wilson, 1971). These changes may also be induced by endurance training, even when conducted in cool-temperate environments (Nadel *et al.*, 1974; Avellini *et al.*, 1982; Henane *et al.*, 1977). Nevertheless, such changes are universally found to be less than elicited through similar training in the heat.

Unfortunately, a more robust, heat-adapted sudomotor system, while advantageous to the semi-clothed endurance athlete, does not implicitly bestow a physiological benefit upon the clothed worker. Consider the worker wearing chemical protective clothing. Typically, this clothing is impermeable to water molecules. Thus, the microclimate will rapidly attain a water vapour pressure which will prevent the evaporation of sweat. Under such conditions, the continued secretion of sweat will not facilitate cooling, but will instead elevate thermal discomfort (Hensel, 1981), and lead to a more rapid dehydration and work performance decrement (McLellan *et al.*, 1993). While it is beyond this review to fully address the interaction of heat adaptation and clothing (see: Cheung *et al.*, 2000; Goldman, 1994), readers should consider these implications with some circumspection.

When combined with the above cardiovascular changes, the sweating adaptations act to minimise the affects of exercise and heat stress upon thermal homeostasis, permitting reduced physiological strain and elevated heat tolerance. Collectively, these transformations permit a greater potential for heat flow from the core and active muscles to the skin, a greater potential

for dissipation of this heat via the evaporation of sweat, and result in a reduction in both the average skin temperature and core temperature for a given combination of exercise and heat stress (Mitchell *et al.*, 1976; Pandolf *et al.*, 1977; Houmard *et al.*, 1990). Thus, in the semi-clothed state, and following heat adaptation, one may move from an exercising state which may be deemed to be physiologically uncompensable, and into a state in which compensation is physiologically attainable.

Classically, the above physiological modifications have been shown to be elicited in response to naturally-occurring climatic changes (acclimatisation: Hellon *et al.*, 1956; Wells *et al.*, 1980), artificial heat exposure (acclimation: Nadel *et al.*, 1974; Patterson *et al.*, 1998a, 1998b, 1999), or endurance training which produces significant elevations in body temperatures (Gisolfi and Robinson, 1969; Pandolf *et al.*, 1977). In addition, a number of techniques have arisen from which thermal adaptation may also be derived. These methods may confer various levels of pre-exposure heat adaptation, and we shall now discuss several of these methods, whilst providing a critical assessment of their relative merits.

2. HEAT ADAPTATION PROCEDURES:

2.1 Natural acclimatisation:

It is generally recognised that natural heat acclimatisation forms the most effective means by which to increase heat tolerance (Edholm *et al.*, 1963). In climates which undergo considerable seasonal change in air temperature, physiological adaptation occurs via a gradual transition, as air temperatures climb from the cooler to the hotter months (Shapiro *et al.*, 1981; Inoue *et al.*, 1995). In this situation, the simple completion of daily physically-demanding tasks confers some degree of thermal adaptation. Indeed, from an occupational perspective, these subtle changes allow the thermal tolerance of workers and athletes to gradually develop. Thus, long-term residents, and some indigenous groups, appear to be better adapted to the heat than do transient visitors (Hellon *et al.*, 1956; Duncan and Horvath, 1988). Nevertheless, such an extended adaptation procedure is not always possible. For instance, military personnel, athletes and some workers are constrained by the need to rapidly move from one environment to another, and must do so without compromising either their physical performance or their health. Thus, while natural adaptation is the most effective technique, its practical limitations minimise its relevance to many occupational and sporting groups.

2.2 Passive heat acclimation:

This method represents an extension of natural acclimatisation. Since external (exogenous) heat sources can invoke physiological adaptation (Hensel, 1981; Wells *et al.*, 1980), one may achieve some degree of heat adaptation by passively raising body tissue temperatures, at rest, via the external application of heat. Various means have been employed by which to apply heat: water baths, saunas, climate chambers and vapour-barrier suits (Fox *et al.*, 1963, 1964, 1967; Fox, 1968; Sugenoya *et al.*, 1986; Ogawa *et al.*, 1988; Inoue *et al.*, 1995). Fox and colleagues extensively explored the use of vapour-barrier suits to maintain an elevated body temperature. In this controlled, passive hyperthermia technique, body temperature was rapidly increased using exercise in the heat, then held stable (2 h) by dressing subjects vapour-barrier suits, placing them within a heated chamber, and controlling their rate of heat loss. The authors reported this technique to be very effective in achieving an acclimation effect. Notwithstanding, passive acclimation is less effective than methods which incorporate an active, exercise stress into the acclimation procedure (Shapiro *et al.*, 1981; Wyndham, 1973), and might generally be considered to be a technique of last resort.

2.3 Exercise-induced heat adaptation in temperate climates:

Exercise elevates muscle and deep tissue temperatures in proportion to the intensity of the exercise stress (Nielsen, 1938; Saltin and Hermansen, 1966). In addition, once an exercise steady state is achieved, and sustained, the body may be placed under a significant thermal load, which very slowly decays when exercise is terminated. If this exercise-induced thermal load is applied on a regular basis, then exercise can induce heat adaptation responses. This association was first recognised by Bean and Eichna (1943), Robinson *et al.* (1943), and Bass *et al.* (1955). Greenleaf (1964) and Piwonka *et al.* (1965) described their endurance-trained subjects as behaving as if they were already heat acclimatised.

An elevated aerobic power, which typically accompanies endurance training in more sedentary people, permits a greater cardiovascular stability (Gisolfi and Robinson, 1969; Wells *et al.*, 1980), and more favourable body-fluid dynamics when exposed to the heat (Senay, 1979). In addition, improved heat tolerance is ascribed to the results of repeated simultaneous activation of cutaneous blood vessels and sweat glands to dissipate metabolic (endogenous) heat (Piwonka *et al.*, 1965). Indeed, endurance-trained subjects display an earlier sweat onset (Nadel, 1979) and a greater sweat sensitivity to increments in body temperature (Wells *et al.*, 1980).

Pandolf *et al.* (1977) and Cohen and Gisolfi (1982) reported that, not only does temperate exercise induce heat adaptation, but it also facilitates a more rapid response to the traditional heat acclimation protocols (combined exercise and heat stress; r=-0.68). Pandolf *et al.* (1977) found that subjects with an aerobic power >65 ml·kg^{-1}·min^{-1} could be acclimated within about four days, to achieve more stable cardiac frequency and body temperatures during a subsequent heat stress. Moreover, subjects who display an insufficient acclimation response during heat adaptation, have been found to have a low basal fitness level (Kok, 1973). Consequently, the early part of the traditional acclimation regimen might be considered to act more to elevate fitness than acclimation state, with less trained subjects requiring a longer acclimation regimen to achieve a comparable acclimation status.

It is important to note that it is not the aerobic power *per se* which enhances the heat acclimation response, but the method in which aerobic power was elevated, and in particular, the endogenous thermal load accompanying that elevation. This relationship is perhaps best exemplified within the experiments conducted on trained swimmers (Hennane *et al.*, 1977), and the sweatless training work of Hessemer *et al.* (1986). These projects have shown that exercise *per se* is not a sufficient stimulus for heat adaptation. Instead exercise must induce, and hold, an elevated body temperature to elicit thermal adaptation.

Over the past 50 years, many research groups have endeavoured to determine which method of exercise-induced heat adaptation best facilitates improved heat tolerance. With hind sight, it is apparent that one cannot make such an assessment without simultaneously quantifying the thermal loading imposed by the exercise. For example, Edholm *et al.* (1963) and Turk and Worsley (1974) relied upon traditional military training regimens to evaluate the efficacy of exercise-induced heat adaptation, finding minimal benefit. Similarly, Cohen and Gisolfi (1982) appraised the impact of differing endurance training intensities upon subsequent heat tolerance, but again without monitoring thermal strain. While such reports are often cited, it is impossible to draw firm conclusions from these investigations, as between-trial differences in thermal strain will result is differences in the stimulus for adaptation. Notable exceptions include studies conducted by Shvartz *et al.* (1973) and Regan *et al.* (1996), wherein exercise-induced heat adaptation was found to be less effective than traditional heat acclimation. Nevertheless, exercise under temperate conditions can foster heat adaptation, and the following conclusions would appear valid: (a) exercise-induced heat adaptation depends upon the capacity of the

exercise to elevate body temperatures, and hold that elevation for an extended duration; (b) body heat storage is proportional to the intensity of the exercise; (c) the total endurance-training volume appears to be more critical than the intensity, once an adequate intensity threshold has been obtained; (d) continuous exercise will more reliably hold an elevated body temperature than will intermittent exercise; (e) heat tolerance is best improved by more prolonged endurance training.

In spite of the benefits of endurance training, it only appears to provide a thermoregulatory benefit during heat exposures of $\leq$2 h (Wyndham, 1973). Once an adequate basal endurance fitness has been attained, there appears to be little additional thermoregulatory advantage that may accrue from continued endurance training (Bean and Eichna, 1943; Eichna *et al.*, 1945). In addition, on its own, it appears that endurance exercise is an inadequate substitute for heat acclimation (Armstrong and Pandolf, 1988; Gisolfi, 1973; Lind and Bass, 1963; Pandolf, 1979; Regan *et al.*, 1996). It may not be sufficient to simply elevate deep tissue temperatures. Instead, the elevation of peripheral tissue temperatures may be needed to provide a critical thermal stimulus for complete heat adaptation (Regan *et al.*, 1996). Finally, one must note that endurance training and heat adaptation subserve vastly different physiological and psychological outcomes, and require quite different exercise programmes. While endurance exercise may improve heat tolerance, exercise-induced heat adaptation may have minimal beneficial impact upon the endurance fitness of some trained people, particularly the endurance athlete, and may even be detrimental, due to the reduced work load which may be tolerated in the heat. Thus, it must be remembered that heat acclimation training merely acts to enhance heat tolerance, and should not be used as a substitute for endurance training. For these athletes, heat acclimation is a form of supplementary training.

2.4 Exercise-induced heat adaptation in hot climates with solar radiation:

While some degree of heat adaptation may be derived from training in temperate conditions, one might expect these adaptations to be less effective than those which might be induced by short-term training in hotter conditions, in which solar radiation constitutes a significant part of the thermal load. Very few studies have addressed this issue, and such studies are very hard to interpret, due to the inability of the researchers to control environmental conditions. Nevertheless, since this type of exposure most closely approximates the conditions that would obtain during natural acclimatisation, it would seem reasonable to suggest that this method

would be superior to temperate endurance training. Furthermore, direct solar radiation more readily elevates skin temperature, than does an equivalent increase in air temperature, and Jessen (1990), using the goat model, has shown that the thermoregulatory responses, at a given air temperature, are markedly different when solar radiation forms part of the thermal stimulus. Earlier, Chen and Elizondo (1974), investigating differences between the impact of changes in body and local skin temperature upon heat acclimation, determined that an increased central thermal stimulus, in conjunction with elevated skin temperatures, was necessary for heat acclimation. Since physiological systems adapt to stresses, with some degree of adaptation specificity, then it is therefore reasonable to suggest that endurance training, with solar loading, would elicit a more specific heat adaptation.

This premise was first supported by Edholm *et al.* (1963), who compared the thermal tolerance of soldiers living in two vastly different environments: the Aden (6 weeks) and Scotland. The naturally-acclimatised soldiers displayed superior heat tolerance, and had fewer field-related heat casualties. Similarly, Wells *et al.* (1980) found their endurance-trained males, who regularly exercised under sunny desert conditions, were better adapted than were their acclimatised counterparts. The investigators interpreted this observation to indicate that both endogenous and exogenous heat loads were required for optimal heat adaptation (Wells *et al.*, 1980), a proposition also advanced by Regan *et al.* (1996).

Armstrong *et al.* (1987) investigated highly-trained distance runners during both spring and summer training. They observed equivalent heat tolerance both before and after summer training. Thus, the additional summer training, and its concomitant solar loading, did not elevate heat tolerance above that which existed at the end of spring. Therefore, the state of heat adaptation of the athletes did not improve over summer. This does not mean these athletes were optimally heat adapted, just that summer training did not provide an adequate additional stimulus for heat adaptation. There are two practical implications from this work. First, highly-trained endurance athletes or workers, do not need special thermal preparation to facilitate heat tolerance during seasonal climatic changes. Second, when athletes or workers are required to travel from their winter to compete or work in the summer months of the other hemisphere, additional heat exposure may be required, due to the inadequacy of the solar load during winter exposures.

2.5 Combined exercise and heat stress acclimation:

We have established that, prior to artificial heat adaptation (acclimation), endurance-trained people will have a thermal tolerance advantage over the untrained. However, Cadarette *et al.* (1984) has shown that this advantage subsides after both trained and untrained subjects undergo heat acclimation, and their thermal tolerance is more homogenous. It is widely acknowledged that, for practical objectives, heat acclimation produces a reasonable and close approximation of the physiological benefits which accompany natural acclimatisation.

One of the paramount advantages of heat acclimation is that one may choose a combination of air temperature and relative humidity which most closely matches the environmental conditions which might reasonably be expected to be encountered. While the experimental evidence is somewhat sparse, it does appear that some degree of specificity exists relating to the transference of acclimation benefits between hot-dry and hot-humid conditions (Goldman *et al.*, 1965). Humid heat acclimation generally results in a greater elevation in sweating than does dry heat acclimation (Henane, 1980; Shvartz *et al.*, 1973), and dry adaptation does not provide optimal protection for humid exposures (Armstrong *et al.*, 1987). However, Shvartz *et al.*, (1973) found, when subjects were exposed to hot-dry conditions (50°C) following either hot-humid or hot-dry heat acclimation, that the hot-dry acclimation regimen resulted in less thermal strain (see also: Eichna *et al.*, 1950; Gisolfi and Robinson, 1969; Piwonka and Robinson, 1967). Therefore, careful consideration of the projected environment is critical to successful heat acclimation. The conventional heat acclimation regimen involves moderate-to-heavy exercise (*e.g.* walking, running, cycling, bench stepping) within a temperature- and humidity-controlled chamber. These methods may be grouped into one of three general modes: (a) constant work-rate methods; (b) self-regulated exercise methods; and (c) controlled-hyperthermia methods.

2.5.1 Constant work-rate methods:

For this heat-acclimation procedure, subjects exercise at a fixed rate, such that heat production exceeds heat dissipation, with body temperatures tracking nett heat storage. This is the most common heat-acclimation method (see: Greenleaf and Greenleaf, 1970 and Sciaraffa *et al.*, 1980), and is the typical model used by military-based researchers, who are tasked with evaluating performance at various fixed work rates, such as the standard marching rate of soldiers (Pandolf *et al.*, 1977, 1988). While this method has wide-ranging application, the

observations from this work must be applied with caution. First, since all subjects are forced to exercise at the same absolute intensity, there may be considerable variability in the relative load imposed upon various subjects, thus physiological strain may vary widely between subjects. Such variance within pre-adaptation data will inherently drive these methods towards the more conservative statistical outcomes. Second, since the work rate is held constant throughout adaptation, the strain during subsequent heat exposure will decline progressively. Thus, the physiological adaptations which derive from this type of regimen may not be identical to those which obtain from either of the two methods described below, and the universal application of these observations must be treated with caution.

2.5.2 Self-regulated exercise methods:

This method allows subjects to select their own work rates, during prescribed work:rest intervals, on the basis of their endurance fitness or perceived exertion, during heat acclimation exposures (see: Miller, 1984; Armstrong *et al.*, 1986). The between-subject variability is minimised with this approach, but the technique suffers in that it can become quite difficult to control the endogenous thermal load. Accordingly, this method has greater practical utility than is does research application.

2.5.3 Controlled-hyperthermia methods:

In this isothermal model, exercise in the heat is used to elevate and maintain a steady-state body temperature above the sweating threshold. Most previous research, which has evaluated the efficacy of exercise-heat exposure regimens, has simply compared heat adaptation responses elicited by those methods, and not the differential thermal forcing functions applied via each method. Since we have herein established that exercise, in the absence of an elevation in body temperature, is an inadequate stimulus for heat adaptation (Hennane *et al.*, 1977; Hessemer *et al.*, 1986), then it follows that, to compare various heat-acclimation methods, one must control and equate the thermal strain imposed upon subjects. Therefore, it is considered more germane to compare experimental conditions on the basis of the thermal potency of the methods, rather than on the basis of their secondary impact upon physiological function. This fundamental methodological limitation has prevented the unequivocal interpretation of most experimental observations, and has made verification of the physiological mechanisms which drive heat adaptation hard to discriminate. Indeed, one may argue that both the constant work-rate and controlled-hypothermia models invoke qualitatively similar, yet quantitatively different

physiological outcomes.

Fox *et al.* (1961) first utilised the isothermal approach, controlling a constant and elevated body temperature by regulating air and microclimate temperatures, using vapour-barrier suits. Subsequently, Turk (1974), and Turk and Worsley (1974), employed the method to acclimate soldiers, exercising them in the heat whilst monitoring body temperatures, and adjusting the work rate to maintain a predetermined thermal strain. Havenith and van Middendorp (1986) revised this procedure, adapting it to a work-rest protocol. More recently, we have further developed this technique, using it to explore acclimation-induced changes sweating and body-fluid balance (Regan *et al.*, 1996; Cotter *et al.*, 1997; Patterson *et al.*, 1998a, 1998b, 1999). Two key observations from these studies are applicable to the current discussion.

First, based on significant post-acclimation reductions in core temperature, forehead skin blood flow and perceived exertion, and elevated forehead sweating, Regan *et al.* (1996) concluded that, when a controlled-hyperthermia regimen is performed in the heat, it will invoke a more complete heat acclimation than when controlled-hyperthermia is used under temperate conditions, even when both elicit equivalent core temperature changes during adaptation. Second, we have shown that the plasma volume expansion, previously considered to be only a transitory outcome of short-term heat acclimation, is preserved, with such elevations lasting as long as 21 days (Patterson *et al.*, 1998a). It is concluded that the controlled-hyperthermia model induces a more complete heat adaptation than either the constant or the self-regulated work rate techniques.

3.6 Exercise-induced heat adaptation in combination with sweat clothing:

An overview of this topic would be incomplete without noting the use of insulated clothing and vapour-barrier clothing to help induce heat adaptation. Interest in this procedure may be traced back to the work of Bass (1963) and Gisolfi and Robinson (1969). Clearly, if this technique was successful, it would have a great practical advantage over the more traditional heat acclimation methods. Unfortunately, there is little empirical evidence to indicate that this procedure is any more beneficial than is endurance training (Allan *et al.*, 1965; Crowdy and Haisman, 1965; Marcus, 1972; Dawson, 1994). Furthermore, it is evident that researchers have generally failed to use appropriate experimental controls or to equate the dependent variables between experimental conditions. For instance, if one treatment elevates body temperatures more than

another, then between-condition differences could be predicted *a priori*. In the absence of adequate research, one must concluded at this time, that minimal physiological benefit that can arise from the use of sweat clothing during physical training.

3. RECOMMENDED HEAT ACCLIMATION PROCEDURES:

Before discussing the practical recommendations for heat acclimation, it is important for the scientist, supervisor or coach to consider matters related to the acclimation specifications, subject safety and efficiency. These points are listed within Table 1, and will serve to help better decisions to be made concerning the principles and practices of the chosen heat acclimation regimen.

Table 1: Operational considerations for determining methods of heat adaptation:

Acclimation specifications:

- Operational definition of heat acclimation.
- Basal acclimation state of workers.
- Anticipated worst case thermal stress.
- Level of heat acclimation required.
- Specificity of the exposure: dry versus humid conditions.
- Critical heat exposure work durations.
- Test for attainment of heat acclimation.

Safety:

- Monitor body temperature and cardiac frequency.
- Establish criteria for exposure termination.
- Identify subjects most likely to experience dysthermia.
- Establish adequate emergency procedures.

Efficiency:

- Efficiency of technical personnel.
- Individual variations in pre-exposure heat adaptation.
- Throughput of subjects.
- Acclimation duration required.
- Acclimation decay and its minimisation.

Availability of heat acclimation facility.

It is the author's view that heat acclimation which employs exercise in the heat, in combination with the controlled-hyperthermia (isothermal) technique, provides the most dependable, and least hazardous, means of heat acclimation. That is, this method permits one to prescribe the thermal load, in terms of the magnitude of the core temperature elevation, and the duration for which the elevation is sustained. However, the technique also produces more uniform body temperature elevations across subjects, which are supervised by both the subjects and trained observers. The following points are noted for careful consideration by the experimenters.

(i) Select an air temperature which is at least equivalent to that which is anticipated.

(ii) The upper temperature limit should be ~40°C for humid exposures, and ~50°C for dry exposures.

(iii) Choose the relative humidity based upon the highest anticipated within the target environment.

(iv) The core temperature elevation should be ~1°C, but core temperatures should not exceed 39.5°C. Within these constraints, the magnitude of the core temperature elevation during acclimation should be determined by the following factors: the other duties that the subjects are required to perform between acclimation sessions; the extent of medical coverage available; the ratio of the acclimation subjects to supervisors.

(v) The mode of exercise used is only an important consideration for athletic groups. For all other groups, one should consider: cost, subject monotony, and the number of people to be simultaneously heat acclimated.

(vi) The controlled-hyperthermia technique permits the supervisor to focus upon exposure duration, while the subjects themselves modify their work intensity to hold the target core temperature. While there is little empirical data available, the literature indicates that a minimal heat acclimation to expected heat exposure ratio would be 1:2 (Wyndham, 1973; Avellini *et al.*; 1980). When it is anticipated that heat exposures will be relatively short (<2 h), and separated by full recovery periods, then it is recommended that acclimation exposures be of at least equal duration to the anticipated heat exposures. For exposures >3 h, also separated by a full recovery, acclimation exposures should be at least 2 h, or 50% of the anticipated duration, whichever is longer. If consecutive exposures are not planned to provide full recovery periods, then recovery should be considered incomplete, and

successive durations should be summed to determine the duration of the acclimation exposure.

(vii) It appears that the length of discrete acclimation exposures is a more important consideration than is the cumulative exposure duration within a day. For instance, Lind and Bass (1963) found that a single 100-min exposure was superior to two 50-min exposures (morning and afternoon). The critical consideration is the time spent at the target body temperature. Therefore, since each exposure has an inherent warm period during which tissue temperatures rise towards the target temperature, then longer exposures permit greater time at, or above, the critical temperature.

(viii) Given enough time, heat acclimation will progress through three distinct phases (Candas, 1987): the acute phase elevates body temperature; short-term acclimation enhances the sweat response; and long-term acclimation results in sudomotor habituation and improved sweat efficiency, with a reduction in sweat secretion and drippage. The aim of heat acclimation is to drive the subject into the short-term phase. This cannot occur when exposures are at one-week intervals (Barnett and Maughan; 1993). Furthermore, since not all physiological variables respond at identical rates (Armstrong and Maresh, 1991), it may take up to fourteen days for all systems to enter the short-term heat adaptation phase. However, since there are decreasing returns with each additional day of exposure, once the first week has elapsed, then one must often consider financial factors when determining the duration of the acclimation regimen. For instance, while heat acclimation may not be complete, and will vary between subjects, Turk and Worsley (1974) found that 86% of their subjects could be acclimated within four days, using a one-hour controlled hyperthermia procedure.

(ix) Finally, we must consider acclimation decay. Williams *et al.* (1967) reported that about 25% of the thermoregulatory adaptation was lost after 6-7 days without heat exposure. As a general guide, it is considered that one loses the equivalent of one days acclimation every two days (Givoni and Goldman, 1973). Nevertheless, Pandolf *et al.* (1977, 1998) found that subjects with greater endurance fitness, or some degree of pre-existing acclimation state, lost acclimation effects more slowly. Retention seems to last longer following dry acclimation than humid heat acclimation (Pandolf, 1998). While yet awaiting definitive research, it is recommended that one additional heat exposure be used for each five days away from significant exposures.

4. CONCLUSION:

It is well established that humans can adapt to many forms of physical stress, including altered thermal environments. While is generally recognised that natural heat acclimatisation forms the most effective means by which to increase heat tolerance, its practical limitations minimise its relevance to many occupational and sporting groups. The passive exposure to external heat, while mimicking natural thermal exposures, is less effective than are methods which incorporate an active, exercise stress, and might generally be considered to be a technique of last resort. Unquestionably, endurance-trained subjects behave physiologically as if they were already heat acclimatised, but endurance training only appears to provide a thermoregulatory benefit for short-duration heat exposures. Once an adequate basal endurance fitness has been established, there appears to be little additional thermoregulatory advantage from such training. However, when training is combined with a solar load, one can more closely approximate natural acclimatisation, and such conditions may elicit superior heat adaptation, though the evidence for this is not unequivocal. Therefore, it has become widely acknowledged that heat acclimation produces a close approximation of the physiological benefits which usher natural acclimatisation. Of the conventional heat acclimation methods, it may be concluded that the constant work-rate and controlled-hypothermia models invoke qualitatively similar, yet quantitatively different physiological outcomes. Indeed, the technique which elicits the more complete adaptation, and which provides the more dependable, and less hazardous, means of heat acclimation is the controlled-hyperthermia (isothermal) technique.

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BODY TEMPERATURE REGULATION IN THE HEAT. HEAT ADAPTATION, BODY-FLUID DISTRIBUTION AND THERMAL STRAIN.

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INTRODUCTION

Body tissues behave as reservoirs of thermal energy, through which heat passes, altering local tissue temperature. In turn, excitable cells within these tissues display some degree of thermosensitivity, mediated via temperature-dependent modification of ion fluxes. However, only within certain cells (thermoreceptors) does this change elicit a neural response, to which both autonomic and behavioural mechanisms act to regulate the thermal status of the *miliue intérieur*. These processes operate to regulate body temperature which, due to the convergence of thermoafferent pathways, is considered to be the algebraic summation of thermal information throughout the body (Bligh, 1976). Thus, effector function is driven following the integration of all thermoafferent messages. Of particular attention here will be thermal states outside the upper critical temperature, where sudomotor (sweat gland) function dominates autonomic thermoregulation, and where external thermal stimuli are applied via the skin surface.

CENTRAL THERMOSENSITIVITY

Thermoresponsive cells exist throughout the central nervous system (CNS; Hellon, 1983; Simon, 1974), and are also located within extra-CNS deep-body structures (Mercer and Jessen, 1978). While in many avian species, the spinal cord appears to dominate the hypothalamus in temperature regulation (Caputa, 1984), this pattern does not universally obtain in mammals, where the reverse generally occurs (Simon, 1974), with the hypothalamus contributing ~50% of the total thermoafferent flow (Jessen, 1996). From an autonomic perspective, it is the mammalian preoptic anterior hypothalamus within which peripheral and central thermal afferents converge and are integrated. More than 60% of its neurons are temperature-insensitive, while ~30% are warm- and ~10% cold-sensitive (Boulant, 1996); coincidental with the general dominance of core temperature changes in most warm-defence strategies.

CUTANEOUS THERMOSENSITIVITY

Cutaneous thermoreceptors are positioned in a three-dimensional configuration (Ivanov *et al.*, 1986), appearing as overlapping fields of variable sensitivity and size, with few insensitive zones (Melzack *et al.*, 1962). Hensel (1976) has suggested their role is to warn of changing, more so than of constant, tissue temperatures. This notion is appealing when one considers that cutaneous cold thermoreceptors are more numerous, and possibly related to the exogenous nature of cold thermal stress.

Within any skin region, peripheral thermosensitivity is the product of the number of afferent neurons and their discharge frequency, with the intensity of a thermal stimulus dictating discharge frequency. However, cutaneous afferents do not project directly to the hypothalamus. Indeed, they pass via the spinothalamic tracts, through which spinal cord afferents also proceed. Simon (1972) has demonstrated almost 100% convergence between cutaneous and spinal afferents within these tracts. Such convergence acts in an additive function, with two warm afferents producing a more powerful signal than either on its own, and the convergence of warm and cold afferents resulting in a less powerful signal, or even signal inhibition. Such convergence is not apparent for thermal afferents travelling via trigeminal neurons, which maintain their integrity through the trigeminal ganglion, medulla and thalamus (Poulos *et al.*, 1976).

In non-facial skin, convergence results in spatial summation, demonstrated for thermal sensation, increasing with the size of the area stimulated. At the same time, the smaller the area stimulated, the greater is the thermal stimulus required to elicit a thermal sensation (Hardy *et al.*, 1938). Sensation also displays temporal variations, with changes occurring primarily at the boundaries of thermosensory fields, while the central portion remains stable (Melzack *et al.*, 1962). That is, not all receptive spots associated with an afferent fibre are functional at any given time. Finally,

there is strong evidence of thermal adaptation (Melzack *et al.*, 1962), with mapped areas revealing the same sensitivity patterns, but with a reduced overall sensitivity. While generally lacking statistical support, these psychophysical data provide the neural basis for within-dermatome differences in cutaneous thermosensitivity.

Cutaneous thermal sensation differs between regions (Kenshalo *et al.*, 1961). Recently, we have investigated local and whole-body thermal sensations to locally applied heat and cool stimuli, across ten skin regions (different dermatomes), while the core and untreated skin temperatures were clamped (Cotter *et al.*, 1996). In general, the skin was found to be of uniform thermal sensitivity. However, the hands and feet were highly sensitive to local stimuli, but did not modify whole-body sensation. Moreover, the face displayed a significantly greater local thermal sensitivity, which also affected whole-body thermal sensation. As facial afferents pass via the trigeminal path, this observation could be explained on the basis of minimal afferent convergence (Poulos and Molt, 1976).

On a neural basis, regional cutaneous thermosensitivities vary as a function of the number of afferent neurons radiating from any given site, the number of active nerve endings within that site, their state of thermal adaptation, the magnitude and direction of the thermal stimulus, and the extent of neural convergence between the site of stimulation and the hypothalamus. These peripheral neural messages are integrated with those arising within the CNS, thereby giving rise to both vascular and sweating responses. We shall now focus our attention more closely on the control of the latter.

THE PHYSIOLOGICAL SIGNIFICANCE OF SWEAT

The evaporation of sweat is an extremely powerful cooling process. When totally evaporated from the skin surface, sweat can remove body heat at a rate of 2.43 $kJ \cdot g^{-1}$. Humans therefore control sweat secretion to maintain thermal homeostasis. Since humans are capable of extended sweat rates approximating 30 $g \cdot min^{-1}$, it is possible to remove heat at rates $\approx$73 $kJ \cdot min^{-1}$. Assuming a 20% metabolic efficiency, such a heat loss rate will support a normothermic total energy use $\approx$1520 W. This equates with an external work rate of 304 W, eliciting an oxygen consumption >3.5 $l \cdot min^{-1}$. However, while man has a great capacity to both work and dissipate metabolically-derived heat, exercise under various environmental extremes may impede heat dissipation. Under such conditions, the cumulative effects of metabolic and environmental thermal loads may represent an uncompensable heat stress, predisposing to hyperthermia, and impairing physiological and cognitive performance.

Evaporative cooling in terrestrial beings is perpetual, occurring from the respiratory tract with every breath, and via water-permeable membranes. Resting normothermic man, within a cool-temperate environment, evaporates 30-33 $g \cdot h^{-1}$ from each surface, with the corresponding cooling effect accounting for the dissipation of about 25% of the resting metabolic heat production. This occurs without our awareness: insensible evaporation. When faced with an external heat load, thermosensitive cells within the skin, and eventually those within deeper tissues, communicate this altered thermal status to the hypothalamus for interpretation. Hypothalamic integration of thermal messages results in the generation of a thermal error message, to which a proportional sympathetic response is elicited: sweating from eccrine glands.

Apart from man, a number of species possess the ability to sweat actively in response to thermal stress. In man, considerable inter-individual differences are apparent for sweat gland densities and secretion rates (Montagna and Parakkal, 1974; Sato and Sato, 1981). Indeed, men generally sweat more than women (Janowitz and Grossman, 1950), and even racial differences exist (Gibson and Shelley, 1948). While euhydrated people can sustain insensible losses indefinitely, dehydration will occur during active sweating, if water replacement is not elevated proportionately. For instance, daily sweat rates can increase from 300-400 ml, to 10-15 litres during prolonged heat and exercise exposure. Extended-duration sweating of 1.5-1.8 $l \cdot h^{-1}$ (30 $g \cdot min^{-1}$) is commonly observed and, under severe heat stress, glands can secrete up to 3-4 $l \cdot h^{-1}$.

It has been estimated that we have between 1.6-4.0 million sweat glands (Szabo, 1962), with considerable variability in gland density between regions. Each gland consists of a secretory coil, connected to the skin surface. As sweat moves through the duct, sodium, chloride and

bicarbonate ions are reabsorbed (Quinton, 1983). Sweating typically starts by recruiting groups of glands innervated by the same sympathetic nerve. At rest, sweating generally starts at the extremities, moving towards the head as thermal strain increases (Randall and Hertzman, 1953). However, we have shown that, with the exception of an earlier lower torso sweat onset, between-site sweat recruitment is generally uniform during upright exercise (Cotter *et al.*, 1995). During sustained sweating, sweat is secreted across the body surface in a cyclic pattern, reflecting the rhythm of sympathetic activity. As thermal strain rises, the frequency of glandular stimulation is elevated. During the phase conversion from liquid to gas, the water molecules do not themselves change temperature, but simply absorb thermal energy to drive evaporation. With the evaporation of 1 g of sweat, 2.43 kJ of heat is removed.

FACTORS WHICH AFFECT SWEAT EVAPORATION

The evaporation of sweat is influenced by the water vapour pressure of the surrounding air, but is mainly determined by that of the microclimate above the skin, with water passing down the vapour pressure gradient. Thus, any factor which affects this gradient will impact upon evaporative cooling.

Immediately above the skin is a very thin layer of air, which behaves as though it was trapped in permanent contact with the skin: the boundary layer. For evaporation to occur, the water vapour pressure of the boundary layer must be less than that of the skin surface. Since both the size and composition of the boundary layer are an inverse function of **air velocity** (relative or absolute), both **environmental water vapour pressure** (reflected within relative humidity) and wind speed can modify evaporative cooling. The single greatest impact upon the boundary layer is brought about by the use of **clothing**. While some clothing ensembles allow air to pass through the fabric and apertures, less permeable garments trap air. In such ensembles, locomotion largely determines garment ventilation (Havenith *et al.*, 1990), and trapped air water vapour pressure.

The above factors represent physical changes. Evaporative cooling also depends upon physiological variations. Continuous sweating, at high flows, leads to sweat accumulation, and eventually sweat suppression (**hidromeiosis:** Collins and Weiner, 1962). This is generally attributed to water-induced swelling of subcutaneous tissues, leading to pore blockage. **Hydration status** affects heat loss by reducing the core temperature threshold for sweating onset, the sensitivity of the sweat response to such changes, and local sweat rates (Sawka and Coyle, 1999). On the other hand, **adaptation** to both endurance training and heat have been shown to increase sweat rates (Regan *et al.*, 1996).

SWEATING AND THE BODY-FLUID COMPARTMENTS

The human body is about 60% water (500-600 $ml.kg^{-1}$: female-male), which, while stored within various compartments, is free to move between these sites. Water, the primary substrate for sweat, is drawn from this reservoir. The total volume of water stored is a function of hydration state (Sawka and Coyle, 1999), body composition (Pace and Rathbun, 1945), and endurance exercise adaptation (Maw *et al.*, 1996). For instance, a low adiposity is associated with the storage of a larger water volume relative to body mass, due to the higher water content of lean tissue relative to fat (Pace and Rathbun, 1945). During exercise, we have shown that fluid losses are drawn almost equally from the intracellular and extracellular compartments (Maw *et al.*, 1998). The plasma volume forms a sub-division of the extracellular volume, and is generally defended in cool and temperate, but not within hot environments (Maw *et al.*, 1998). If not replaced, fluid losses result in progressive dehydration, impaired physiological function and dysthermia.

SWEATING AND HEAT ADAPTATION

It was believed that heat adaptation (acclimation) might influence the regional distribution of sweating, favouring greater limb sweating. Past experiments from our laboratory have demonstrated that, while adaptation enhances sweating by elevating sweat rate and lowering its onset threshold (Regan *et al.*, 1996; Patterson *et al.*, 1998), it was not associated with a sweat redistribution (Patterson *et al.*, 1998). For any site, the post-adaptation sweat responses appeared more closely related to differences in sweat gland density, than to altered control of the sweat glands.

Heat adaptation is known to affect body-fluid volumes. Typically, post-adaptation elevations in body fluid, including the plasma compartment, are observed (Patterson *et al.*, 1998). However, until recently, it was accepted that the plasma lost during combined heat and exercise stress would be diminished following heat adaptation. We now know this to be somewhat imprecise. Instead, the post-adaptation plasma volume increase is also associated with a greater post-adaptation plasma loss (Patterson *et al.*, 1998). We have observed that plasma losses, during heat and exercise stress, increase from 16% prior, to 22% following, extended heat adaptation (Patterson *et al.*, 1998). Thus, the plasma volume was not preferentially defended. Instead, the heightened sweat losses and elevated resting plasma volume, resulted in an elevated plasma contribution to fluid losses following heat adaptation.

CONCLUSION

We have seen that, while the evaporation of sweat serves a very powerful cooling function, it may be impeded by changes in the boundary layer air and hydration state. On the other hand, endurance training and heat adaptation enhance sweat secretion. However, such changes need not necessarily be associated with greater evaporative cooling, which remains dependent upon the water vapour pressure gradient between the skin and the boundary layer air.

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THE REGULATION OF HUMAN ECCRINE SWEATING.

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1. Introduction

The *First Law of Thermodynamics* dictates that energy can change from one form to another, but its total magnitude will remain constant. Heat is thermal energy. During exercise, chemical energy is converted into both kinetic and thermal energy in a ratio of about 1:4. For example, a 70 kg person cycling at 200 Watts in a thermoneutral environment would consume approximately 2.5 litres of oxygen per minute, and experience a total metabolic energy use of ~1000 $J \cdot s^{-1}$. About 800 $J \cdot s^{-1}$ of energy would be converted to heat energy. Since a storage of ~3.5 kJ of heat per kilogram body mass causes tissue temperature to rise 1°C (mean specific heat of tissues), then heat storage at a rate of 800 $J \cdot s^{-1}$ (48 $kJ \cdot min^{-1}$), causes the tissue temperature to rise by 1°C in just over 5 min. If we assume a pre-exercise average body temperature of 36°C, and a maximal upper limit of 41°C, then our person would reach this point in ~25.5 min. Yet, people frequently exercise for several hours at this work rate without becoming hyperthermic. This is possible because of our well-developed heat loss mechanisms. All animals dissipate heat, however, humans rely heavily upon evaporation cooling. This paper focuses upon the regulation of thermal energy dissipation via eccrine sweat gland (sudomotor) secretion.

An understanding of heat balance is derived by examination of the avenues for heat gain or loss. This relationship is illustrated by the *heat balance equation:*

$$S = M - (\pm W) \pm E \pm R \pm C \pm K \qquad [W \cdot m^{-2}]$$

where:

S = heat storage (+ = storage; - = loss) $[W \cdot m^{-2}]$
M = internal heat production (metabolism) $[W \cdot m^{-2}]$
W = work performed (+; leaving) or entering (-) system $[W \cdot m^{-2}]$
E = heat exchange via evaporation (-) or condensation (+) $[W \cdot m^{-2} \cdot kPa^{-1}]$
R = heat exchange via radiation (- = loss; + = gain) $[W \cdot m^{-2}]$
C = heat exchange via convection flow (- = loss; + = gain) $[W \cdot m^{-2}]$
K = heat exchange via conductance (- = loss; + = gain) $[W \cdot m^{-2}]$

During exercise in the heat, avenues for non-evaporative heat dissipation are impeded and even reversed. For instance, under full solar load, the body experiences radiative heat gains from the sun and nearby surfaces. Similarly, natural convective losses cease when air temperature approximates that of the skin surface. Under these conditions, the body becomes heavily, if not totally reliant upon evaporative heat loss. While some

people suffer hyperthermia when exercising in the heat, the majority of people do not. In fact, our 200 Watt work rate can be maintained for some time when air temperature equals skin temperature. The body experiences an elevation in tissue temperature, but evaporative cooling generally enables the maintenance of thermal equilibrium; evidence of the cooling power of the sudomotor system.

Evaporation of 1 g of sweat dissipates 2.43 kJ of heat (latent heat of evaporation). In our situation, dissipation of 48 kJ of heat each minute requires the evaporation of ~19.8 g of sweat. Extended duration sweat rates ($\dot{m}_{sw}$) up to 30 g·min^{-1} are possible, permitting heat removal rates of ~73 kJ·min^{-1}, or a total metabolic energy use of about 1520 J·s^{-1} without hyperthermia. Since thermal equilibrium (S=0) frequently occurs at states other than thermoneutrality, it is within the mild hyperthermic state that we shall explore the regulation of human eccrine sweating.

2. Eccrine sweat glands

Humans, and some animal species, possess sweat glands, of which there are two functional variations: apocrine and eccrine glands. From an evolutionary perspective, primitive sweat glands (apocrine) are found in the dog. These glands lack neural innervation, relying on circulating epinephrine to initiate sweat secretion (Robertshaw, 1975), and play a minimal thermoregulatory role. An evolutionary step found in the cow is the termination of nerves close to sweat glands, providing superior glandular regulation (Robertshaw, 1971). However, the apocrine glands of the horse are under direct neural regulation (adrenergic), and are functionally similar to human eccrine glands. In man, and some primates, apocrine glands open into hair follicles, having a secretory function only after puberty. They are located in the axilla, external auditory meatus, the areolar and genital regions, and primarily serve non-thermal functions (tactile sensation, frictional contact and possible sexual attractant: Robertshaw, 1971).

Human eccrine glands are primarily thermoregulatory in function, and are found over virtually the entire body surface. Unlike the adrenergic apocrine glands, eccrine glands are cholinergically activated, and discharge a fluid containing various electrolytes and metabolites directly onto the skin surface. The total number of eccrine glands has been estimated between 1.6-4.0 million (Kuno, 1956; Szabo, 1962), with a considerable inter-regional variability in gland density: lowest on the back (64·cm^{-2}), and highest on the palms and soles (600-700·cm^{-2}: Kuno, 1956; Sato and Dobson, 1970). While eccrine glands exist in other mammals (cat, dog, rat, mouse and opossum), they tend to be restricted to the paws (Sato, 1977).

Eccrine sweat glands are usually single units, embedded in the loose matrix of subdermal connective tissue. Each gland consists of a single unbranched tubule coiled into a bolus (~300 μm diameter), 2-5 mm below the epidermis, and connected to the epidermis via a straight segment (Quinton, 1983). This tubule contains secretory and reabsorptive components. The former is located in the tubule coil, and is responsible for sweat formation. Two distinct cell types (clear and dark) are found in this tubule. The clear cells rest either directly on the basement membrane or on myoepithelial cells, and contain abundant mitochondria and an infolded plasma membrane. It is thought the clear

cells are responsible for the secretion of water and electrolytes (Sato, 1977). The reabsorptive component removes sodium, chloride and bicarbonate ions from the fluid as it passes towards the skin surface.

3. Neural networks

To appreciate sudomotor regulation, we must not only understand gland function, but also the mechanisms which dictate that function. We must first determine the regulated variable. This is generally, though not unequivocally, recognised to be body tissue temperature (Cabanac, 1975). However, some suggest that body heat content or flow is regulated (Webb, 1995). Space does not permit entry into this debate. However, one may ask that if temperature was not the regulated variable, then how could one explain the remarkable constancy of body temperature within both intact and spinal patients, across a huge range of ambient conditions? If temperature is the key variable, then which temperature is regulated and how is it monitored? Is it body core temperature (T_c), since it is the most stable temperature? Such stability may, however, merely result from regulating another body temperature (Mitchell *et al.*, 1972). Is it an average tissue temperature? Since thermoreceptors have been identified in deep and superficial tissues (Hellon, 1983), and since thermal stimulation of non-hypothalamic sites elicits an hypothalamic response (Hellon, 1970), one may hypothesise that the regulated temperature is an integrated function of afferent input from all thermosensitive sites (Hensel, 1981). How then are these tissue temperatures quantified?

3.1 Thermoreceptors and afferent pathways: Thermosensitive neurones have been located in the skin, viscera, spinal cord, midbrain, medulla oblongata, and the anterior and posterior hypothalamus (Hensel, 1981; Hellon, 1983). Afferents generated by cutaneous thermoreceptors in the limbs and trunk enter the spinal cord through the spinal ganglion and dorsal root, synapsing in the dorsal horn with second order afferents, which ascend via the lateral spinothalamic tract (Willis *et al.*, 1974; Brück and Hinkel, 1990). It is believed that the subcoeruleus region, within the pontine reticular formation (Hinkel and Schröder-Rosenstock, 1981), and the raphè system, located in the pons and midbrain (Dickenson, 1976), relay these thermal afferents to the hypothalamus for integration. Thermal afferents from the cutaneous thermoreceptors of the face pass through the trigeminal ganglion, to the hypothalamus, synapsing with second order neurones in the trigeminal nucleus caudalis (Brück and Hinkel, 1990).

3.2 Central integration: The hypothalamus has long been associated with thermoregulation (Isenschmidt and Krehl, 1912), with the preoptic anterior hypothalamus (POAH) established as a dominant site in the regulation of eccrine sweating (Hensel, 1981). Thermosensitive neurones of the POAH receive and integrate the central and peripheral thermal signals (Boulant, 1981). However, data indicates that such integration is also possible at both the spinal cord and brain stem (Simon *et al.*, 1986). This integration enables the generation of a thermal `load error' signal, to which the hypothalamus produces an autonomic response. Increased evaporative loss accompanying heat storage reveals that sweat regulation operates via a proportional control mechanism (Hammel, 1968), with an approximately linear increase in heat loss with elevations in body temperature (Smiles *et al.*, 1976; Mercer and Jessen, 1980).

3.3 Efferent pathways: From the POAH, efferent sweat impulses descend through the brain stem and the spinal tract, crossing at various levels, and terminating in the lateral horn, where new neurones start (Sato, 1977). Eventually these neurones synapse with post-ganglionic sympathetic fibres, which innervate the glands. Quinton (1983) estimated that spinal cord segments T2-T4 supply eccrine sweat glands on the head and neck, T2-T8 innervate glands of the upper limbs, T6-T10 supply the trunk, and segments T11-L2 pass to the lower extremities. Efferent signals reach the sweat glands in waves, resulting in pulsatile sweat secretion, which is well synchronised between regions. The periodicity of these neural impulses, which may approach 0.25 Hz in very hot conditions (Bini *et al.*, 1980), causes alternating sweat peaks and troughs. Such fluctuations may result from an oscillation within the regulatory system, created by the superimposition of transient rises and falls in skin temperature upon a constant central drive for heat loss (Bini *et al.*, 1980).

While sweat glands are innervated by cholinergic sympathetic fibres, they also respond to norepinephrine and low concentrations of adrenaline (Sato and Sato, 1981). The glands also respond to both α and β stimulation, and appear to have a separate adrenergic pathway (Sato, 1977; Uno and Montagna, 1975). Large inter-individual differences in cholinergic sensitivity are apparent, and such variation accounts for some of the observed differences in sweat function between people (Sato and Sato, 1983). Accordingly, cholinergic sensitivity is generally greater in males (Janowitz and Grossman, 1950), tropical ethnic races (Gibson and Shelley, 1948), and young adults (Montagna and Parakkal, 1974).

4. Eccrine sweat gland secretion

4.1 Eccrine secretion: The proximal, secretory portion of the sweat gland tubule produces a primary secretion, the concentrations and the components of which are modified as the fluid passes through the reabsorptive duct to the skin surface. The exact sequence of events following the cholinergic activation of the sweat gland has not been ascertained. However, the process of fluid secretion probably involves the active transport of electrolytes (sodium/potassium-ATPase) through the basal-lateral membranes of the secretory cells (Quinton and Tormey, 1976). The most probable scheme for this process was postulated by Field (1982). The intra-cellular Na/K pump creates an electrochemical gradient which draws sodium ions (Na^+) into the sweat gland cells. This flux is accompanied by chloride ions (Cl^-), due to their coupling with Na^+. The Cl^- can only leave the cell by moving into the lumen of the sweat duct. Such unidirectional movement is due to the presence of a Cl^- permeable apical membrane, combined with a Cl^- impermeable basal-lateral membrane and paracellular shunt. The electrochemical gradient thus created, draws Na^+ across the paracellular barrier and into the gland cells. Water then moves passively into the lumen of the gland, to maintain an osmotic equilibrium, and primary sweat is thus formed. Accordingly, the intra-lumen hydrostatic pressure increases, forcing the primary sweat down the tubule, through the reabsorptive duct, and eventually onto the skin surface (Field, 1982).

Before sweat actually reaches the skin surface, various primary sweat components are preferentially reabsorbed, helping ensure the conservation of essential solutes. This

reabsorption is the function of the distal portion of the sweat gland tubule. Na^+, Cl^- and bicarbonate ions are the primary reabsorbed solutes. The concentration of Na^+ in the reabsorptive duct cells is kept at a low level by the action of sodium/potassium-ATPase within the cell membranes of the gland (Quinton and Tormey, 1976). The electronegativity of the interior of the cell, in conjunction with the low cytoplasmic Na^+ concentration, allows Na^+ to passively move from the lumen into the cell via the apical membrane (Quinton, 1981), and Cl^- follows passively.

Increases in thermoregulatory sweating closely parallel rises in body temperature, and as sweating increases towards maximal levels, there is a recruitment of progressively more sweat glands, followed by an increase in sweat secretion per gland (Randall, 1946). A caudal-to-rostral pattern of sweat onset has been demonstrated in resting (Hertzman *et al.*, 1953), but not in exercising subjects (Nadel *et al.*, 1971; Cotter *et al.*, 1995). However, while there is considerable $\dot{m}_{sw}$ variation between subjects and a powerful postural influence, steady state sweating at rest tends to generally be greatest on the forehead, and least on the arms, hands or chest.

4.2 Maximal eccrine sweat secretion: Given that humans have relatively sparse hair coverage, and that the entire skin surface is covered with eccrine sweat glands, humans have an unique ability to utilise evaporative heat loss as a means for heat dissipation. Typically, during combined moderately heavy exercise and heat stress, sweat rates ($\dot{m}_{sw}$) will range between 1.0-3.0 $mg \cdot cm^{-2} \cdot min^{-1}$, depending upon the site of measurement (Cotter *et al.*, 1995). Under severe heat stress, glands can secrete up to four litres of sweat per hour, for short periods (Quinton, 1983).

5. Eccrine sweat gland regulation during exercise

The thermoregulatory system is comprised of four components: a passive system (tissues experiencing altered thermal states); thermal sensors (inhomogenously distributed); a controller; and effector units (blood vessels and eccrine sweat glands). Numerous cybernetic models have been developed to help clarify the inter-relationships between these system components (see Hammel, 1968; Hensel, 1981; Werner, 1990). For our purposes, we shall restrict ourselves to closed-loop regulation based upon a single feedback loop, which provides information concerning the thermal state of the whole body, and which elicits proportional sweat regulation (Hensel, 1981; Werner, 1990). Working from the combined assumptions that human heat balance is a function of temperature regulation, and that the regulated temperature is an average body tissue temperature, derived using inputs from all available thermosensitive sites, it becomes apparent that thermal sweating should be induced when one or more of these sites experiences a supra-threshold local temperature elevation of sufficient duration.

It is well established that both core and cutaneous temperatures are involved in the regulation of sweating (Robinson, 1949; Nadel *et al.*, 1971; Patterson *et al.*, 1995). We know that T_c changes can produce large sweat responses (Benzinger *et al.*, 1963). Similarly, we know that high skin temperatures alone may elicit sweating (McCaffrey *et al.*, 1979). Furthermore, the relative importance of these two inputs has generally been determined. For example, Proppe *et al.* (1976) demonstrated that a core temperature

elevation in baboons produced a ten-fold greater elevation in iliac blood flow than did skin temperature elevation. In humans, similar central:peripheral thermal sensitivities appear to govern skin blood flow (Wenger *et al.*, 1975) and $\dot{m}_{sw}$ (Nadel *et al.*, 1971). However, our understanding of the contributions, and the thermal sensitivities of various central and peripheral thermosensitive sites in humans is relatively poor. While we know that core and skin temperatures are fundamental inputs of the sudomotor regulatory system, we know relatively little about the sensitivities of tissues within these zones. Thus, our understanding of human thermoregulation is incomplete, with most investigators considering both the body core and skin as single sources of thermal input. Animal research tells us that not all deep body thermosensitive sites provide equally effectual sources of afferent input, with the POAH dominating inputs from the midbrain, medulla oblongata, spinal cord and visceral sites (Hensel, 1981). Investigations of this type are almost impossible to perform in humans, however, we are able to evaluate the relative thermal sensitivities of different skin regions.

Four groups have attempted to identify the role of local skin temperature (T_{skl}) in the regulation of sweating. The earliest work came from an American group. Nadel *et al.* (1973) irradiated several skin surfaces (N=2), evaluating thermal sensitivity according to site-specific capacities to invoke sweat responses at the untreated thigh. T_{skl} heating initiated sweating, with the face showing the greatest thermal sensitivity. Crawshaw *et al.* (1975) applied cooled thermodes to the skin, investigating the inhibitory effect of T_{skl} cooling on the sweat response of an untreated thigh (N=5). Forehead cooling resulted in the strongest inhibition of sweating. In neither study was there an attempt to control T_c, mean skin temperature, or thermal stimulus intensity. Furthermore, the treated surface areas were not uniform between treatment sites. Accordingly, data from both groups must viewed with some caution.

The French approached this question using sealed limb chambers and a climate chamber, to independently control steady-state T_{skl} of the arms and legs, and the head and torso, respectively (Libert *et al.*, 1984: N=5). The affect of T_{skl} changes was measured from its impact on right arm sweating. Head-torso T_{skl} changes displayed the greatest influence upon sweating. This project again highlighted the significance of T_{skl} on sudomotor function. However, the value of the inter-regional comparisons of thermal sensitivity is limited, since the surface areas of treated skin were not equal, and they did not control T_c, mean skin temperature or the temperatures of the untreated skin regions.

A group from Germany also used a limb chamber paradigm. They independently heated and cooled the legs, showing that an elevated T_c could nullify the influence of T_{skl} on sweating (Heising and Werner, 1987: N=3). More recently, they used this technique to investigate the effects of controlling arm and leg temperatures on sweating (Werner and Heising, 1990: N=7). It is uncertain to what extent the limb chambers affected the microclimate, and therefore sudomotor function. However, their results confirmed sweating was regulated by both T_c and T_{skl}. They also observed differences in cutaneous thermal sensitivity, and provided evidence that central processing of skin temperature information was different between heating and cooling. However, these observations relate only to limb skin temperatures. Again, this group did not clamp T_c or skin

temperature, and neither study adequately controlled the untreated skin temperature, or the surface area of treated skin.

Finally, recent research in this area comes from the laboratory of the present authors (Patterson *et al.*, 1995). The influence of T_{skl} on the regulation of sweating was evaluated, while a water-perfusion suit (held at 37°C) and climate chamber (36.5°C ±0.8; relative humidity 60.3 ±1.6%) were used to clamp skin and core temperatures (N=8). Individual water-perfusion patches (249.0 ±0.2 cm^2) were used to elevate and reduce T_{skl} of four upper body skin surfaces (face, upper arm, forearm and hand). The sweat response was evaluated simultaneously from eight body segments, using sweat capsules. Heating promoted, while cooling suppressed $\dot{m}_{sw}$, both locally and at the other skin segments. However, local $\dot{m}_{sw}$ was not altered to a greater extent than $\dot{m}_{sw}$ at either the adjacent or contralateral skin surfaces ($p>0.05$). Face and hand heating produced a greater elevation in whole-body sweat output than did forearm heating ($p<0.05$). Upper arm cooling was least effective in suppressing sweating compared to face, hand and forearm cooling ($p<0.05$). These results indicated that independent changes to T_{skl} result in $\dot{m}_{sw}$ modifications, with differences in the $\dot{m}_{sw}$ response varying between upper body treatment sites, which generally revealed an hierarchical thermal sensitivity between skin regions. These observations are currently being extended to include the torso and the lower body (N=12). Therefore, thermal sensitivity of the skin is not uniform, possibly reflecting differences in thermoreceptor density, convergence of afferent signals, or differences in hypothalamic responses to these signals.

Conclusion

The thermoregulatory system is made up of four main components: a passive system; thermal sensors; a controller; and effector units. In closed-loop sweat regulation, sensors provide information concerning the thermal state of the passive system. The POAH (controller) integrates this input, comparing it with a reference temperature (or some analogue thereof), and generates a load error signal (feedback). The POAH then invokes a proportional sweat response to elevate heat dissipation and regulate mean body temperature. We know very little of the roles of the deep body thermal sensors in human sweat regulation. However, we do know that their inputs dominate that of the skin. We also know that cutaneous thermal sensitivity is not uniform, displaying an apparent hierarchical pattern in their regulatory roles.

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HUMAN TEMPERATURE REGULATION: PARTITIONAL CALORIMETRY

Introduction

Approximately 75-80% of the energy converted into ATP from carbohydrates, fats and proteins is lost as heat during energy conversion processes in humans (this "loss" does not mean an energy destruction, but an energy conversion). The science of measuring heat production is called calorimetry, and Antoine Lavoisier (1743-1794) first measured body heat production in small animals using **direct calorimetry**: the direct measurement of heat production. Lavoisier also used the relationship between metabolism and oxygen consumption ($\dot{V}_{O2}$) to develop a second method for measuring metabolic rate: **indirect calorimetry**.

In this workshop, we will use the method of **partitional calorimetry** to compute body heat storage during exercise in the heat. This method is another form of indirect calorimetry. The principle of partitional calorimetry is based upon derivation of the amount of heat stored within the body from the separate analysis of the avenues by which the body gains or loses heat.

Heat is thermal energy transported from one site to another due to a temperature difference between two sites (*i.e.* the body and its surroundings). This heat transfer ends when the temperatures of the two sites are equal (*thermal equilibrium*). By summing the heat fluxes from these avenues, we are able to indirectly arrive at the amount of heat the body will store.

The technique of partitional calorimetry is based upon the *First Law of Thermodynamics*[1], which is a form of the *law of conservation of energy*. In general terms, this law states that:

> *The total energy in a closed system (e.g. the universe) is constant. Energy is neither created nor destroyed. Energy can change from one form to another (e.g. work into heat), but its total magnitude remains the same.*

From our perspective, we shall consider our closed system to be a laboratory (or climate chamber) in which our subjects will be exercising. Now, within this system, the total amount of energy will be conserved, but not the energy in its various forms. That is, chemical potential energy (carbohydrates, fats and proteins) will be converted into mechanical energy (external work performed on a cycle ergometer), and heat energy within the body. The mechanical energy passes to the ergometer or treadmill from the subject, where it is converted into several other forms (*e.g.* heat energy via friction within the ergometer; kinetic energy via movement of the ergometer and the air surrounding it), and dissipated and dispersed (but not lost) within the laboratory (chamber) system. In humans, approximately 75-80% of the energy which is converted into *adenosine triphosphate* (ATP) is subsequently converted into heat energy, and the body has two options: it can store this heat, or it can pass (dissipate) this heat to the surrounding environment (laboratory or chamber system). If this heat energy is stored, the

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[1]Hermann von Helmholtz (1812-1894) was the first person to convince the scientific community of the validity of the law.

body's temperature will rise at the rate of 1°C for each 3.47 kJ of heat stored per kilogram of body mass[2]. An average 70 kg person will change mean body temperature (T_b)[3] by 1°C for each 243 kJ change in heat energy content (3.47 · 70 = 242.9).

During exercise at 200 Watts ($J \cdot s^{-1}$) in a thermoneutral environment, our hypothetical person would consume approximately 2.5 litres of oxygen per minute, and experience a total metabolic energy use of ~1000 $J \cdot s^{-1}$. About 800 $J \cdot s^{-1}$ of this energy would be converted to heat energy. This would cause the T_b to rise by 1°C in just over 5 min. If we assume a pre-exercise T_b of 36°C, and a maximal upper limit of 41°C, then our person would reach this point in ~25.5 min. Yet, people frequently exercise for several hours at this work rate without becoming hyperthermic. Clearly, heat energy is being dissipated to the environment.

Purpose:

The aim of this laboratory activity is to introduce workshop participants to human **temperature regulation measurement**, and to quantify the avenues of **heat flux** using partitional calorimetry.

Workshop methods:

Apparatus:

(i) treadmill or cycle ergometer;
(ii) equipment for measuring body temperatures: thermistors and recording devices.
(iii) electronic scales;
(iv) SportTester heart rate monitor;
(v) stopwatch;
(vi) calculators;
(vii) container of Cidex solution to sterilise apparatus;
(viii) tissues, paper towel, thermistor tape, vaseline, alcohol wipes, gloves;
(ix) subject Informed Consent packages;
(x) drinking cups and sports drink.

Procedures:

The laboratory activity involves strenuous exercise, and your subject must first be screened using the attached questionnaire, and he/she must also provide informed consent for this activity. Part of that release is the Par-Q Questionnaire, which is designed to identify people at risk of cardiovascular problems during exercise.

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[2] Specific heat for the body tissues averages 0.83 $kcal \cdot kg^{-1} \cdot °C^{-1}$, or 3.47 $kJ \cdot kg^{-1} \cdot °C^{-1}$. *Specific heat* (*c*: $J \cdot g^{-1} \cdot °C^{-1}$, or thermal energy capacity) is the quantity of heat required to raise the temperature of 1 gram of matter by 1°C. Examples: water 4.186; air 0.963; ice (-10°C) 2.219; wood 1.758; human tissues 3.470-3.558 ($J \cdot g^{-1} \cdot °C^{-1}$). Thus, for humans, it takes approximately 3.5 kJ of heat to elevate 1 kg of tissue 1°C.

[3] Mean body temperature (T_b) is ~35-36°C. This is computed from two assumptions: (i) that core temperature is ~37°C and mean skin temperature (T_{sk}) is ~33°C; and (ii) that the contribution of the core to mean body temperature in comfortable conditions is about 70%. Thus, $T_b = (0.7 \cdot T_r) + (0.3 \cdot T_{sk})$ [°C].

(1) Subject preparation:

The subject will require a swimming costume, running shoes and towel.

(i) Record your subject's pre-exposure mass and height. Compute body surface area:

$A_D = 0.202 \cdot \text{mass}^{0.425} \cdot \text{height}^{0.725}$

where: mass is in kg, height is in metres, and area is in m^2.

(ii) Subject goes to the toilet, then inserts the sterilised rectal thermistor to a depth of 10 cm beyond the anal sphincter.

(iii) Subject is instrumented with the skin thermistors and a SportsTester. The thermistors will measure skin temperature, while the SportsTester is a cardiac frequency monitor. For the purpose of this workshop, we will use a four-site (Ramanathan[4]) method for measuring mean skin temperature (T_{sk}). Ideally, we should measure skin temperatures at more sites, with eight being the standard number of sites used in our research laboratory. Sites: chest ($T_{sk\text{-}1}$: mid-pectoral), forearm ($T_{sk\text{-}2}$: mid-dorsal), thigh ($T_{sk\text{-}3}$: mid-ventral), and calf ($T_{sk\text{-}4}$: mid-dorsal).

(iv) Drinking: You will perform two trials. In the first trial, your subject is to drink 200 ml (1 cup) of water every 15 minutes. In the second trial, there is no drinking.

(2) Data collection:

(i) Start stopwatch. Record one baseline data point for each variable after 10 min of rest: core temperature (T_c), skin temperatures and cardiac frequency (f_c). Enter these data into Table 1 (this Table is at the end of this document). Collect resting f_c, skin and rectal temperatures (T_{re}) for each minute of the first 10 minutes within the chamber. You have 5 temperatures to read from the telethermometer, so you should allow 10 seconds for each reading.

(ii) Record baseline values for three psychophysical indices. Enter these data into Table 1.

(1) THE 15-POINT BORG SCALE OF PERCEIVED EXERTION (Borg, 1962):

During the exercise period we want you to pay close attention to how hard you feel you are working. This feeling should indicate the total amount of exhaustion and fatigue that you are sensing, combining all possible sensations, physical stress, effort, and fatigue (no matter what their source). Do not concern yourself with any one factor such as leg pain, shortness of breath or exercise intensity, but try to concentrate on your total, inner feeling of exertion. Do not underestimate or overestimate, just be as accurate as you can.

We will ask you: "how hard are you exercising".

6
7 Very, very light
8
9 Very light
10
11 Fairly light
12
13 Somewhat hard

[4] Ramanathan, N.L. (1964). A new weighting system for mean surface temperature of the human body. *J. Appl. Physiol.* 19:531-533.

14
15 Hard
16
17 Very hard
18
19 Very, very hard
20

(2) THE 13-POINT THERMAL SENSATION SCALE (Gagge et al., 1967):

During the test we want you to describe how your body temperature feels: that is, we want you to rate your thermal sensation. Do not concern yourself with any one area, such as your hands or feet, but try instead to concentrate on your total body temperature sensation. The thermal sensation scale has numbers ranging from 1 (unbearably cold), to 7 (a neutral sensation), and finally to 13 (unbearably hot). We will ask you every 5 minutes to give us a number that best represents your whole-body thermal sensation at that moment.

We will ask " How does the temperature of your body feel?"

1 Unbearably cold
2 Extremely cold
3 Very cold
4 Cold
5 Cool
6 Slightly cool
7 Neutral
8 Slightly warm
9 Warm
10 Hot
11 Very hot
12 Extremely hot
13 Unbearably hot

(3) THERMAL DISCOMFORT SCALE (Gagge et al., 1967):

During the test we want you to describe how comfortable you feel with the changes in your body's temperature. That is, we want you to rate your thermal discomfort. Do not concern yourself with any one area, such as your hands or feet, but try instead to concentrate on total body discomfort. The thermal comfort scale has numbers ranging from 1.0 (comfortable), to 5.0 (extremely uncomfortable). We will ask you every 5 minutes to give us a number that best represents your whole-body thermal comfort at that moment.

We will ask "How comfortable do you fell with the temperature of your body?".

1.0 Comfortable
1.5
2.0 Slightly uncomfortable
2.5
3.0 Uncomfortable
3.5

4.0 Very uncomfortable
4.5
5.0 Extremely uncomfortable

(iii) At minute 11, your subject can commence exercising.
(iv) Collect f_c, skin and T_{re} at 5-minute intervals during exercise (during last minute of each interval). Also record the three psychophysical variables at 5-minute intervals.
(v) Towel subject dry and collect mass data at 15-minute intervals. Record these data into Table 1.
(vi) Terminating a trial: trials will terminate at exhaustion, or if T_{re} reaches 39.0°C, or if a subject's f_c reaches 190 b·min^{-1}, or if your subject desires to terminate the trial prematurely.
(vii) Towel subjects dry, and record body masses at exercise termination. Gently remove thermistors. Subject removes rectal thermistor in the toilet, washes and dries the probe, and returns it to for sterilisation.

(3) Calculations:
You will be calculating components of the heat balance equation, using data collected from your subject. This equation is derived from the first law of thermodynamics.

$$S = M - (\pm W) \pm E \pm R \pm C \pm K \quad [W \cdot m^{-2}]$$

where:
S = heat storage (+ for storage; - for loss) [W·m^{-2}],
M = internal heat production (metabolism) [W·m^{-2}],
W = work performed (+: energy leaving system) or received (-: energy entering system) [W·m^{-2}],
E = heat exchange via evaporation (-) or condensation (+) [W·m^{-2}·kPa^{-1}],
R = heat exchange via radiant exchange (loss -; gain +) [W·m^{-2}],
C = heat exchange via convective heat flow (loss -; gain +) [W·m^{-2}],
K = heat exchange via conductance (loss -; gain +) [W·m^{-2}].

(i) Compute T_{sk} for each of the recording intervals.

$$T_{sk} = (0.3 \cdot T_{sk-1}) + (0.3 \cdot T_{sk-2}) + (0.2 \cdot T_{sk-3}) + (0.2 \cdot T_{sk-4}) \quad [°C]$$

(ii) Compute $\bar{T}_b$ for each of the recording intervals:
$\bar{T}_b$ may be derived using the weighted sum of T_r & T_{sk}:

$$\bar{T}_b = (0.8 \cdot T_r) + (0.2 \cdot \bar{T}_{sk}) \quad [°C]$$

(iii) Compute body heat storage (S) at 10-minute intervals, for the full trial (*i.e.* commencing at time zero (pre-exposure thermoneutral)). During thermal equilibrium: $S = (M - (\pm W) \pm E \pm R \pm C \pm K \pm S = 0)$. However, during this workshop, T_c and $\bar{T}_b$ both experienced elevations, so S must have been some value other than zero, and it must have had a positive sign. We can now compute the change in heat storage (ΔS in the units of kJ·m^{-2}·hr^{-1}) from the equation:

$$\Delta S = 3.47 \cdot \text{mass} \cdot (\bar{T}_{b1} - \bar{T}_{b0}) / A_D / \text{time} \quad [kJ \cdot m^{-2} \cdot hr^{-1}]$$

where:

3.47 = average specific heat of body tissues ($kJ \cdot kg^{-1} \cdot °C^{-1}$),
mass = subject's mass at recording interval (use pre-exposure mass) (kg),
$\bar{T}_{b1} = T_b$ at the current recording interval (°C),
$\bar{T}_{b0} = T_b$ at the previous recording interval (°C),
[thus: $\Delta \bar{T}_b = \bar{T}_{b1} - \bar{T}_{b0}$ (°C)],
A_D = surface area (m^2) [read metabolism laboratory concerning the use of A_D], and
time = the sampling duration (hr: 10 min ∴ 0.167 hr).

(iv) Compute metabolism (M: in the units of $kJ \cdot m^{-2} \cdot hr^{-1}$) at 10-minute intervals, for the full trial (*i.e.* commencing at time zero). Assume the data at time zero were also computed over a 10-min period. Due to space and equipment restrictions, we will not measure metabolic heat production during this activity. However, if we did do so, we would use *open circuit, indirect calorimetry (spirometry)* to derive $\dot{V}_{O2}$. If it is possible to measure the $\dot{V}_{O2}$ for our resting subject, the we will use those data. However, we can also assume that $\dot{V}_{O2}$ for our resting subject is 0.3 $l \cdot min^{-1}$ (STPD). During exercise, we could assume that $\dot{V}_{O2}$ increases linearly, and that the $\dot{V}_{O2}$ for our exercising subject (at each 10 minute mark) is: 1.25, 1.50, 1.75, 2.00, 2.25, 2.50, 2.75, 3.00, 3.25, 3.50, 3.75, and 4.00 $l \cdot min^{-1}$ (STPD). We will also assume an RQ of 0.90 during exercise (see Table 2): at rest, use 0.83 (during heavy work RQ is closer to 1.00).

$$M = \dot{V}_{O2} \cdot H_c \cdot 60.0 / A_D \quad [kJ \cdot m^{-2} \cdot hr^{-1}]$$

where:
$\dot{V}_{O2}$ = oxygen consumption ($l \cdot min^{-1}$),
H_c = the thermal equivalent of O_2 for the non-protein respiratory quotient ($J \cdot l^{-1}$),
60.0 = to convert $\dot{V}_{O2}$ from $l \cdot min^{-1}$ to $l \cdot hr^{-1}$,
A_D = surface area (m^2).

(v) Compute the external work performed on the ergometer (W: in the units of $kJ \cdot m^{-2} \cdot hr^{-1}$) at 10-minute intervals, for the trial (*i.e.* commencing at time zero). During the rest period, this will be zero.

$$W = \text{work rate} \cdot 0.06 \cdot 60.0 / A_D \quad [kJ \cdot m^{-2} \cdot hr^{-1}]$$

where:
work rate = load setting on the ergometer (Watts or $J \cdot s^{-1}$),
0.06 = to convert $J \cdot s^{-1}$ into $kJ \cdot min^{-1}$,
60.0 = to convert $kJ \cdot min^{-1}$ to $kJ \cdot hr^{-1}$,
A_D = surface area (m^2).

(vi) Compute the evaporative heat loss (E: in the units of $kJ \cdot m^{-2} \cdot hr^{-1}$) at 10-minute intervals, for the trial (*i.e.* commencing at time zero).

$$E = \Delta mass \cdot 2430 / A_D \quad [kJ \cdot m^{-2} \cdot hr^{-1}]$$

where:
Δmass = mass change over sampling duration (kg),
2430 = latent heat of evaporation ($kJ \cdot l^{-1}$: 1 litre ≈ 1 kg),
A_D = surface area (m^2).

Table 2: The thermal equivalent (kJ·l^{-1}) of oxygen for the non-protein respiratory quotient (RQ).
From: Zuntz, N. (1901). *Pfluger's Arch. Physiol.* 83:557.

Non-protein RQ	kJ	Non-protein RQ	kJ	Non-protein RQ	kJ
0.707	19.616	0.81§	20.147	0.92‡	20.712
0.71	19.632	0.82§	20.197	0.93‡	20.767
0.72	19.683	0.83§	20.252	0.94	20.817
0.73	19.733	0.84	20.302	0.95	20.867
0.74	19.787	0.85	20.352	0.96	20.922
0.75	19.837	0.86	20.407	0.97	20.972
0.76	19.888	0.87	20.457	0.98	21.022
0.77	19.942	0.88	20.457	0.99	21.077
0.78	19.992	0.89	20.557	1.00	21.127
0.79	20.043	0.90‡	20.612		
0.80§	20.097	0.91‡	20.662		

Notes: § = typical non-protein respiratory quotient at rest; and ‡ = typical non-protein respiratory quotient during steady-state exercise.

(vii) The computations of radiative, convective and conductive heat losses are technically complex, and, for simplicity, shall be treated as a single value. Compute this collective value ($\pm R \pm C \pm K$: in the units of kJ·m^{-2}·hr^{-1}) at 10-minute intervals, for the trial (*i.e.* commencing at time zero).

$$S = M - (\pm W) - E \pm R \pm C \pm K \quad [W \cdot m^{-2}]$$
$$\therefore (\pm R \pm C \pm K) = S - M + (\pm W) - E \quad [W \cdot m^{-2}]$$

Laboratory report:

Table 1: Raw data collection sheet.

Time (min)	Mass (kg)	f_c	T_r	T_{sk-1}	T_{sk-2}	T_{sk-3}	T_{sk-4}	T_{sk}	T_b
0 (rest)									

Laboratory report:

Questions:

(i) Heat is lost from the body core by moving down existing thermal gradients. The key gradient is from the core to the skin. From here heat moves along the skin surface where it is passed to the air via the evaporation of sweat. Compute the core-skin thermal gradient (T_r-T_{sk}) for each 10-minute interval.

(ii) Produce the following graphs using you data:

(a) cardiac frequency against time,
(b) core temperature against time,
(c) mean skin temperature against time,
(d) REP against time,
(e) thermal sensation against time, and
(f) thermal discomfort against time.

(iii) Prepare an oral report to interpret the graphs produced above. You should pay particular attention to the time course of your data.

(iv) This experiment has been also performed on an elite athlete. Here is a summary of that experiment. Steady-state cycling for 90 min at 40°C and 40% humidity. Trial one was performed in the euhydrated state, while trail 2 was performed on the next day, with the subject drinking only 500 ml of water between the two trials. So the subject was very dehydrated on this trial. You will be provided with several graphs from this experiment. Prepare an oral report to interpret these graphs. You should pay particular attention to the time course of these data.

THE 15-POINT BORG SCALE OF PERCEIVED EXERTION (Borg, 1962):

During the exercise period we want you to pay close attention to how hard you feel you are working. This feeling should indicate the total amount of exhaustion and fatigue that you are sensing, combining all possible sensations, physical stress, effort, and fatigue (no matter what their source). Do not concern yourself with any one factor such as leg pain, shortness of breath or exercise intensity, but try to concentrate on your total, inner feeling of exertion. Do not underestimate or overestimate, just be as accurate as you can.

We will ask you: "how hard are you exercising".

6
7 Very, very light
8
9 Very light
10
11 Fairly light
12
13 Somewhat hard
14
15 Hard
16
17 Very hard
18
19 Very, very hard
20

THE 13-POINT THERMAL SENSATION SCALE (Gagge et al., 1967):

During the test we want you to describe how your body temperature feels: that is, we want you to rate your thermal sensation. Do not concern yourself with any one area, such as your hands or feet, but try instead to concentrate on your total body temperature sensation. The thermal sensation scale has numbers ranging from 1 (unbearably cold), to 7 (a neutral sensation), and finally to 13 (unbearably hot). We will ask you every 5 minutes to give us a number that best represents your whole-body thermal sensation at that moment.

We will ask "How does the temperature of your body feel?"

1 Unbearably cold
2 Extremely cold
3 Very cold
4 Cold
5 Cool
6 Slightly cool
7 Neutral
8 Slightly warm
9 Warm
10 Hot
11 Very hot
12 Extremely hot
13 Unbearably hot

THERMAL DISCOMFORT SCALE (Gagge et al., 1967):

During the test we want you to describe how comfortable you feel with the changes in your body's temperature. That is, we want you to rate your thermal discomfort. Do not concern yourself with any one area, such as your hands or feet, but try instead to concentrate on total body discomfort. The thermal comfort scale has numbers ranging from 1.0 (comfortable), to 5.0 (extremely uncomfortable). We will ask you every 5 minutes to give us a number that best represents your whole-body thermal comfort at that moment.

We will ask "How comfortable do you fell with the temperature of your body?".

1.0 Comfortable
1.5
2.0 Slightly uncomfortable
2.5
3.0 Uncomfortable
3.5
4.0 Very uncomfortable
4.5
5.0 Extremely uncomfortable

Informed consent for subjects:

Participation as a subject within workshop activity is voluntary. Before being a subject, you should familiarise yourself with the following features of the relevant laboratory:

* The workshop objectives.
* The rationale for the workshop.
* The test procedures.
* Possible risks and discomforts.

Inquiries:
Questions concerning the procedures, or rationale, used in this workshop are welcome at any time. Please ask for clarification of any point which you feel is not explained to your satisfaction.

Freedom of Consent:
Your participation is entirely voluntary. You are free to withhold such consent before or during the experiment. In the latter case, such withdrawal of consent should be performed at the time you specify, and not at the end of a particular trial. Your right to withdraw will be preserved over and above the goals of the workshop.

INFORMED CONSENT

The staff running this workshop support the principles governing both the ethical conduct of experiments and laboratories, and the protection at all times of the interests, comfort and safety of all people involved in such activities.

Your signature below indicates five things:

(1) You have read the relevant workshop material.
(2) You have been given the opportunity to discuss the contents, and any possible risks and hazards prior to commencing the workshop.
(3) You clearly understand these procedures and possible risks.
(4) You voluntarily agree to participate in the workshop.
(5) Your participation may be terminated at any point in time.

To screen out people at risk, please answer the following seven questions:

(1) Has your doctor ever said that you have a heart condition and recommended only medically approved physical activity?

Yes: _____ No: _____

(2) Do you have chest pain which is brought on by physical activity?

Yes: _____ No: _____

(3) Have you developed chest pain at rest during the last month?
Yes: _____ No: _____

(4) Do you lose consciousness or lose your balance as a result of unexplained dizziness?
Yes: _____ No: _____

(5) Do you have a bone or joint problem that could be aggravated by the proposed physical activity?
Yes: _____ No: _____

(6) Is your doctor prescribing medication for you for blood pressure or heart problems?
Yes: _____ No: _____

(7) Are you aware, through your own experience, or a doctor's advice, of any other reason why you should not perform exercise without medical consultation?
Yes: _____ No: _____

[*Source:* Thomas, S., Reading, J., and Shephard, R.J. (1992). Revision of the physical activity readiness questionnaire (PAR-Q). *Can. J. Sport Sci.* 17:338-345.]

I agree to participate in the workshop described in the accompanying documentation.

Last name: _____ Given name: _____ Date of Birth: __/__/__

Address: _____

Name and phone number of contact person in case of an emergency:

Name: _____ Phone: _____

Family doctor: _____ Phone: _____

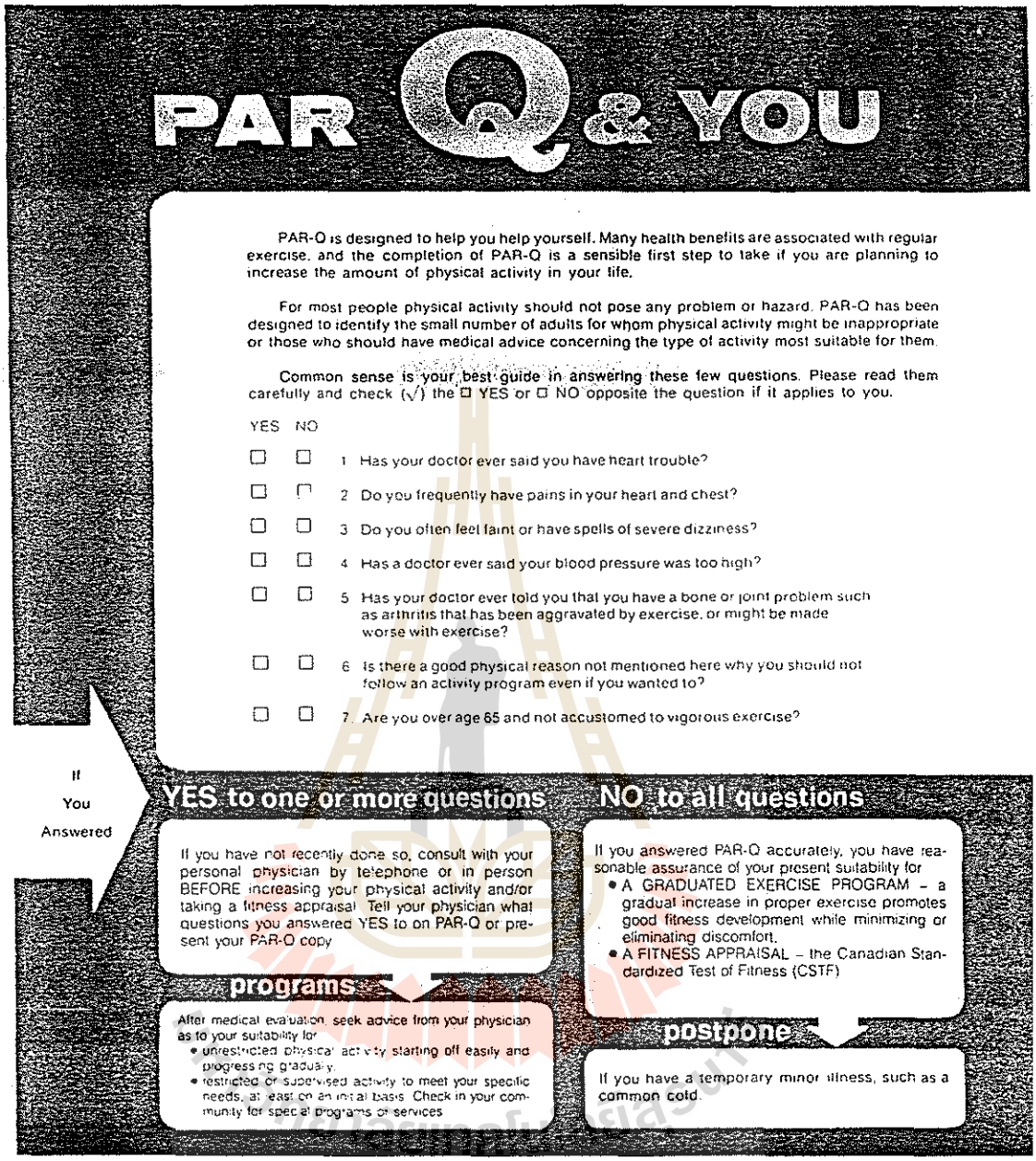
Signature: _____ Date: __/__/__

Witness: Name _____ Signature: _____

Physical Activity Readiness Questionnaire (PAR-Q)*

NAME OF PARTICIPANT _____

DATE _____



* Developed by the British Columbia Ministry of Health. Conceptualized and critiqued by the Multidisciplinary Advisory Board on Exercise (MABE). Translation, reproduction and use in its entirety is encouraged. Modifications by written permission only. Not to be used for commercial advertising in order to solicit business from the public.
Reference: PAR-Q Validation Report, British Columbia Ministry of Health, 1976.
* Produced by the British Columbia Ministry of Health and the Department of National Health & Welfare. MR-1127

APPENDIX

Eur J Appl Physiol (1995) 71:549–554

ORIGINAL ARTICLE

James D. Cotter · Mark J. Patterson
Nigel A. S. Taylor

The topography of eccrine sweating in humans during exercise

Accepted: 24 May 1995

Abstract The purpose of this study was to investigate the distribution of steady-state sweating rates ($\dot{m}_{sw}$), during stressful exercise and heat exposures. Six men completed 42-min trials: 2-min rest and 40-min cycling at 40% peak power in 36.6°C (relative humidity 46.0%). The $\dot{m}_{sw}$ was monitored using ventilated capsules at the forehead, and at three additional sites. Repeat trials allowed monitoring from eleven skin surfaces. Auditory canal temperature (T_{ac}) and 11 skin temperatures were measured. After normalising $\dot{m}_{sw}$ to the forehead response within subjects, differences in T_{ac} and onset time thresholds, and transient and steady-state $\dot{m}_{sw}$ were examined. The pooled, lower torso $\dot{m}_{sw}$ onset [mean 45.5 (SEM 42.0) s] preceded that of the head [mean 126.5 (SEM 34.8) s, $P<0.05$], but was not significantly different from the legs [mean 66.6 (SEM 25.7) s], upper torso [mean 80.2 (SEM 36.8) s] or arms [mean 108.6 (SEM 31.2) s]. Transient $\dot{m}_{sw}$ did not differ among regions ($P=0.16$). Mean, steady-state forehead $\dot{m}_{sw}$ [3.20 (SEM 0.51) mg·cm^{-2}·min^{-1}] was not significantly greater than the scapula, forearm, hand, stomach and lower back $\dot{m}_{sw}$ (in descending order), but was greater than the chest [1.6 (SEM 0.2)], upperarm [1.6 (SEM 0.2)], calf [1.5 (SEM 0.3)] and thigh $\dot{m}_{sw}$ [1.0 (SEM 0.2), $P<0.05$ for all comparisons]. The results did not support the caudal-to-rostral sweat onset evident during supine, resting heat stress. Equivalent T_{ac} sweat thresholds existed between sites, while steady-state $\dot{m}_{sw}$ topography varied among subjects and was not dominated by central regions.

Key words Exercise · Heat stress · Sweating · Sweat onset · Thermoregulation

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Introduction

Heat dissipation during exercise in hot air is primarily facilitated by eccrine sweating. The ability of this mechanism to minimise heat storage is enhanced by its early initiation, a rapid increase in the rate of sweat excretion ($\dot{m}_{sw}$) relative to core temperature change (high gain), the attainment of an adequate steady-state $\dot{m}_{sw}$ and an optimal distribution of this excretion. Acute and habitual physical exercise, and chronic heat exposure are known to affect these factors. For instance, acute exercise has been shown to lower the sweat onset point (Crockford et al. 1971), while physical training has been shown to lower the core temperature (T_c) sweat threshold and elevate the gain of the sudomotor response (Schvartz et al. 1979). Similarly, heat acclimation lowers the T_c threshold (Elizondo and Bullard 1971; Schvartz et al. 1979), elevates the gain (Libert et al. 1983; Regan et al. 1994), and induces greater steady-state $\dot{m}_{sw}$ (Candas et al. 1983). Furthermore, a possible redistribution of sweating towards peripheral skin regions has been observed (Höfler 1968; Schvartz et al. 1979). This paper addresses the issue of differences in inter-regional sweating rates, and focuses on the onset and steady-state sweating distributions accompanying thermal strain during endogenous and exogenous thermal stresses.

A caudal-to-rostral pattern of sweat onset has been demonstrated in resting (Hertzman et al. 1953; Randall and Hertzman 1953; Seckendorf and Randall 1961; Park and Tamura 1992), but not in exercising subjects (Nadel et al. 1971; Stolwijk et al. 1971; Schvartz et al. 1979). Steady-state $\dot{m}_{sw}$ has been found generally to be greatest on the forehead, and least on the arms (Hertzman et al. 1953), hands (Weiner 1945) or chest (Park and Tamura 1992). However, this distribution is dependent upon the onset thresholds and gain characteristics of each skin region (Stolwijk et al. 1971), and displays considerable interindividual variability (Weiner 1945; Hoffler 1968; Stolwijk et al. 1971).

The literature concerning the distribution of eccrine sweating appears to have been confined to studying a limited number of sites (Sato and Dobson 1970; Schvartz et al. 1979; Ayling 1986), exercise transients (Nadel et al. 1971; Stolwijk et al. 1971), low to moderate $\dot{m}_{sw}$ (Hertzman 1957; Seckendorf and Randall 1961; Tam et al. 1976; Park and Tamura 1992), or procedures which may have promoted varying degrees of localised hidromeiosis (Weiner 1945; Hoffler 1968) or hemihidrosis (Park and Tamura 1992). Therefore, while evidence from these studies is often taken as implicit, an awareness of the topography of high, steady-state sweating is not yet available. Thus, the purpose of this study was to attempt to provide this information for humans exposed to combined exercise and environmental heat stress.

Methods

Subjects

Six healthy, aerobically trained, but otherwise unacclimated males (Table 1) participated in exercising heat stress tests (HST) conducted within a temperature and humidity controlled room [mean ambient temperature 36.6 (SD 0.6)° C, black globe temperature 37.4 (SD 0.2)° C, relative humidity 46.0% (SD 2.3)% and air velocity <0.1 m·s^{-1}]. The subjects completed four 42-min HST, each consisting of 2-min rest and 40-min cycling at steady-state exercise intensity approximately equal to 40% peak power [166 (SD 19) W]. This study was approved by the Human Experimentation Ethics Committee (University of Wollongong), and the subjects were familiarised with the purposes, demands and procedures before providing informed consent.

Protocol

Peak power was determined from an initial cycle ergometer test (Quinton Excalibur, Quinton Instrument Company, USA), with the exercise intensity increasing by 3 W every 5 s until exhaustion. The subjects abstained from strenuous activity (24 h) and food (2 h) prior to HST, and reported for instrumentation approximately 40 min prior to data collection. The HST were undertaken at the same time of day for each subject, and were separated by a minimum of 3 days. Four HST, presented in balanced order, were used to determine regional sweat onsets and steady-state $\dot{m}_{sw}$ for each of the following regions: forehead (all HST), medial lower chest, lateral abdomen, lateral scapula, medial lower back, anterior mid-thigh, anterior-lateral calf, dorsal foot, dorsal hand, anterior upper-arm, and three forearm sites (distal-ventral, mid-ventral and mid-dorsal). The mid-ventral site was used to represent the forearm response during between-region comparisons of $\dot{m}_{sw}$. The use of the two medial and two lateral torso sites was designed to evaluate sweating in relation to distance from the principal sagittal plane, since it has previously been indicated that sweat rates are greater medially (Hertzman 1957).

Table 1 Subject characteristics. A_D DuBois surface area

Subject	Age (years)	Mass (kg)	Peak power (W)	A_D (m^2)
1	36	79.5	375	2.01
2	40	79.7	381	2.01
3	24	84.1	462	2.08
4	24	87.2	402	2.06
5	28	70.5	486	1.81
6	22	81.1	381	1.98
Mean	29.0	80.3	414.5	1.99
SD	7.3	5.7	47.6	0.10

The $\dot{m}_{sw}$ was determined from body mass changes (uncorrected for metabolic or respiratory losses: A&D electronic balance, model no. fw-150k, USA), with mass determined prior to instrumentation and after towelling dry at the completion of each HST, and locally using four ventilated sweat capsules (each 2.19 cm^2). The latter system was based upon the principles of capacitance hygrometry (Sweat Monitor, Clinical Engineering Solutions, Australia). Each capsule was secured with an elastic strip, which was adjusted to apply an approximately constant contact pressure among sites, and ventilated with air at 0.4 l·min^{-1} which had been passed above a saturated lithium chloride solution, providing a relative humidity standard within the range of 11%–12% in air temperatures from 25 to 50° C. The response time of this system was approximately 2 s at a flow of 0.4 l·min^{-1}. The test-retest correlation was 0.85 (SD 0.10), while the intrasubject coefficient of variation of the procedure was 15.0% (SD 11.8%), as assessed from the steady-state $\dot{m}_{sw}$ on the forehead[1]. The humidity sensors were calibrated at 36° C, by introducing relative humidity standards collected over saturated solutions of lithium chloride (11.5%), sodium iodide (33.0%), potassium chloride (42%), sodium chloride (75%) and potassium di-phosphate (93.5%). The $\dot{m}_{sw}$ was derived from changes in the relative humidity and temperature of the air passing through the capsule and over the skin surface. Although an attempt was made to provide consistent pressure when securing the sweat capsules, there appeared to be greater pressure exerted upon the capsule on the dorsal foot in two subjects. Postonset $\dot{m}_{sw}$ for the dorsal foot were therefore excluded from the statistical analyses.

The T_c was approximated from the auditory canal temperature (T_{ac}) during all HST using zero-gradient thermometry (after Keatinge and Sloan 1975). A servo-heating unit warmed and maintained an outer ear pad at the temperature of the auditory canal, thus isolating the canal and its thermistor from external thermal artefacts. Three additional indices of T_c were obtained during the second HST for each subject. Tympanic temperature (T_{ty}) was obtained using a thin and flexible thermistor (Yellow Springs Instruments, FF mini thermistor, USA), inserted through a wax ear mould, and secured in contact with the tympanic membrane, as indicated by an initial sharp pain, and subsequent noise with movement. Placement was periodically checked by gently tapping on the thermistor lead. Oesophageal temperature (T_{oes}) was monitored using a thermistor (Yellow Springs Instruments, type 401), inserted via the nose following the method used by Mekjavic and Rempel (1990). Rectal temperature (T_{re}) was measured 11 cm beyond the anal sphincter (Yellow Springs Instruments, FF mini thermistor). The T_{ac} correlated well with T_{ty} ($r=0.974$), T_{oes} ($r=0.940$) and T_{re} ($r=0.965$), and exhibited comparable response characteristics (Fig. 1), further validating the T_{ac} index of T_c. The T_{ac} was therefore used in subsequent data analyses.

Skin temperature was measured using surface thermistors (Yellow Springs Instruments, EU mini thermistor), secured to the skin with a single layer of waterproof tape. Temperatures were monitored at the sweat capsule sites, plus at sufficient sites to derive an eight-site mean skin temperature ($\bar{T}_{sk}$; according to the International Organisation for Standardisation (ISO) 1992).

All thermistors were calibrated (±0.05° C) against a certified reference mercury thermometer (Dobbie Instruments, Dobros total immersion, Australia). Temperature data, with the exception of T_{ac}, were recorded at 0.2 Hz with a portable data logger (Grant Instruments Ltd., 1206 Series Squirrel, UK). Cardiac frequency (f_c) was monitored from ventricular depolarisation and recorded at 0.2 Hz (Polar Electro SportTester, model PE3000, Finland).

[1] These data are equivalent to those obtained for pilocarpine iontophoresis (Buono et al. 1991), despite differences in the means by which sweating was elicited

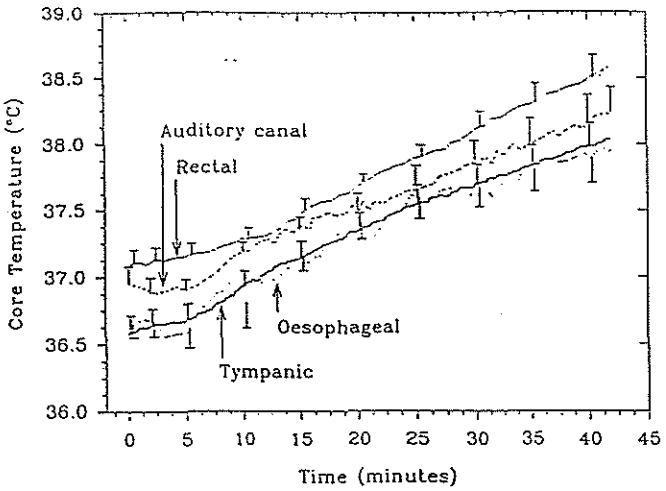

Fig. 1 Auditory canal, tympanic, oesophageal and rectal temperature during a 42-min heat stress trial, consisting of 2-min rest and 40-min cycling at 40% peak power ($n=6$; ambient temperature 36.6° C, relative humidity 46.0%). Data are means with standard errors of the means

The T_{ac} and hygrometry data were converted to digital signals (Computer Boards Inc., PPIO-A18, USA), and recorded at 1.0 Hz on a mains-isolated laptop computer (Total Peripherals, Notebook 386SX, Australia) for later analyses.

Sweat onset was determined as the point from which a continuous, supra-baseline $\dot{m}_{sw}$ was maintained for a 5-min period, without returning to the baseline. For data in which the initial deflection was not clearly apparent, linear regressions were fitted to the pre- and post-onset periods and were solved simultaneously to isolate the onset time. Since forehead $\dot{m}_{sw}$ was measured during each HST, and since this $\dot{m}_{sw}$ was variable among HST within most subjects, it was considered that sweat onset times and $\dot{m}_{sw}$ should be normalised against each subject's forehead sweat response. The sweat onset times were normalised to the earliest within-subject forehead sweat onset. Similarly, each regional $\dot{m}_{sw}$ for a given subject was normalised to the largest within-subject forehead $\dot{m}_{sw}$ response recorded for that subject. Accordingly, statistical analyses were restricted to normalised data, and reported sweat onset and rate data are normalised, unless otherwise stated.

Sweat onset data from adjacent regions were pooled for analysis to produce five skin regions: lower limbs (three sites); upper limbs (three sites); lower torso (two sites); upper torso (two sites); and the head. The average initial $\dot{m}_{sw}$ following sweat onset was termed the *transient* $\dot{m}_{sw}$, and was calculated as the mean $\dot{m}_{sw}$ over the first 5 min following sweat onset. Steady-state $\dot{m}_{sw}$ was the mean $\dot{m}_{sw}$ for the 5-min period 18.5–23.5 min after onset. This period was chosen since data from the calf were considered questionable in two subjects beyond 23.5 min, due to pressure artefacts accompanying an adjustment of the pressure on these capsules, while attempting to ensure an airtight seal. Non-normalised $\dot{m}_{sw}$ were used, in conjunction with surface area weighting factors (ISO 1992), to provide an estimate of the whole body sweat loss for the steady state, and the entire sweating period.

Statistics

Differences in sweat onset time, and the T_{ac}, T_{oes}, T_{ty} and local skin temperature (T_{skl}) thresholds at sweat onset, transient $\dot{m}_{sw}$ and steady-state $\dot{m}_{sw}$ were examined using ANOVA for repeated measures, with Tukey's HSD procedure ($\alpha=0.05$). Pearson's correlation coefficient was used to evaluate intervariable relationships.

Results

Figure 2 shows the $\dot{m}_{sw}$ responses, for four skin regions, for one subject throughout the course of one HST. These data were selected to illustrate both typical $\dot{m}_{sw}$ response patterns and inter-regional $\dot{m}_{sw}$ differences. In this HST, sweat onset times for three lower body sites were relatively synchronous, occurring at about min 2, while that of the forehead was delayed by approximately 2 min. The onset of a rapid, approximately linear rise in $\dot{m}_{sw}$ occurred concomitantly with the elevation in T_c (Fig 1; T_{ac}, T_{oes}, T_{ty}), after which a phase of reduced gain occurred, at varying $\dot{m}_{sw}$ among skin regions. The trace for the shank shows a clear pressure artefact at about 30 min.

The process of normalising data within-subjects was designed to enable valid between-HST comparison of sweat onset and $\dot{m}_{sw}$. The raw onset times were 55.6 (SEM 3.8)s later than normalised onset times, and the raw sweat rates were 83.0% (SEM 3.1)% of the normalised sweat rates (Table 2). Thus, although this procedure altered the absolute magnitudes of the sweat onset times and $\dot{m}_{sw}$ obtained, it did not substantially alter the inter-regional patterns for these variables (Table 2).

The lower torso sweat onset [mean 45.5 (SEM 42.0)s] preceded that of the head [mean 126.5 (SEM 34.8)s, $P<0.05$], but was not significantly different from the onset times of the legs [mean 66.6 (SEM 25.7)s], upper torso [mean 80.2 (SEM 36.8)s] or arms [mean 108.6 (SEM 31.2)s. Table 2]. The T_{ac}, T_{oes} and T_{ty}, at sweat onset, were equivalent across regions: 37.0 (SEM 0.03)° C ($P=0.30$), 36.7 (SEM 0.01)° C ($P=0.86$) and 36.6 (SEM 0.01)° C ($P=0.20$), respectively. The T_{sk} was obtained for 60 of the 96 sweat onsets and ranged from 34.8° C (SEM 0.22; for forearm sweat onset) to 35.2° C (SEM 0.10; for abdomen and scapula onsets), averaging 35.0° C (SEM 0.02) for sweat onsets among the 11 re-

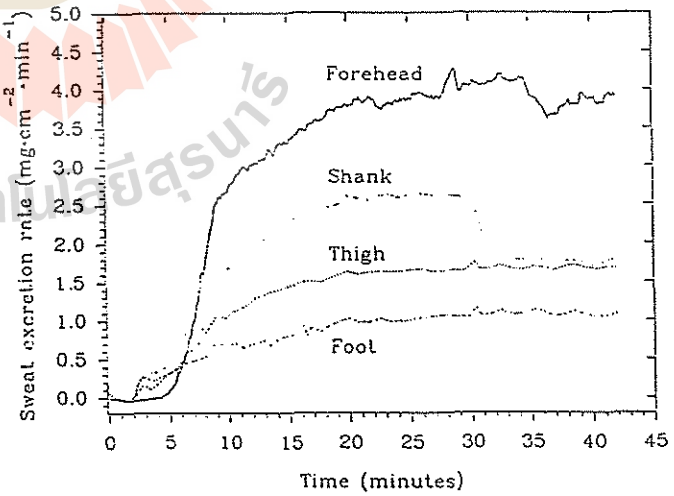

Fig. 2 Sample sweat rate patterns from one subject during a 42-min heat stress trial, consisting of 2-min rest and 40-min cycling at 40% peak power (ambient temperature 36.6° C, relative humidity 46.0%)

Table 2 Regional sweat onset times and steady-state sweat rates during constant work rate cycling (40% peak power, ambient temperature 36.6° C, relative humidity 46.0%). Data are untreated (*Raw*) and normalised (*Norm*) means with standard errors of the means (SEM). Steady $\dot{m}_{sw}$ steady-state sweat rate, [a,b] indicates steady-state $\dot{m}_{sw}$ differs significantly from that of the forehead and scapula, respectively. Postonset data for the foot were excluded due to pressure artefacts in two subjects. Statistical comparisions between individual regions were restricted to steady-state, normalised $\dot{m}_{sw}$

Region	Onset-norm (s)		Onset-raw (s)		Steady $\dot{m}_{sw}$-norm ($mg \cdot cm^{-2} \cdot min^{-1}$)		Steady $\dot{m}_{sw}$-raw ($mg \cdot cm^{-2} \cdot min^{-1}$)	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Head	126.5	34.8	182.6	35.4	3.20	0.26	2.72	0.33
Chest	90.2	36.2	133.3	26.4	1.62	0.17[a]	1.48	0.16
Scapula	70.2	42.8	144.0	51.5	2.89	0.33	2.10	0.14
Abdomen	49.2	45.5	123.0	52.2	1.99	0.33	1.42	0.16
Low back	41.9	39.1	115.8	47.0	1.91	0.24	1.39	0.09
Upperarm	100.2	33.6	143.4	23.9	1.58	0.14[a]	1.46	0.18
Forearm	111.8	37.4	170.6	56.9	2.15	0.41	1.51	0.20
Hand	113.5	31.6	156.7	26.4	2.05	0.32	1.83	0.26
Thigh	65.7	26.7	114.3	3.8	1.03	0.15[a,b]	0.95	0.20
Calf	68.2	25.4	116.8	4.6	1.48	0.28[a,b]	1.39	0.34
Foot	66.0	25.0	114.7	4.6	–		–	
Mean	82.1	8.4	137.8	30.3	1.99	0.21	1.63	0.15

gions. The T_{skl} at sweat onset was lower at the foot and calf than at either the scapula or forehead. Intrasegmentally, the distal-ventral, mid-ventral and mid-dorsal forearm sites exhibited equivalent sweat onset times: 98.0 (SEM 22.8)s, 111.9 (SEM 34.7)s, 112.0 (SEM 35.3)s, respectively ($P=0.38$).

The transient $\dot{m}_{sw}$-responses from the hand [1.06 (SEM 0.34)], forearm [1.00 (SEM 0.14)], forehead [0.86 (SEM 0.15)] and scapula [0.95 (SEM 0.22) $mg \cdot cm^{-2} \cdot min^{-1}$] tended to display greater transient $\dot{m}_{sw}$ responses than those of the legs [calf=0.40 (SEM 0.09) and thigh=0.39 (SEM 0.09); $P>0.05$]. Consequently, the transient $\dot{m}_{sw}$ for the pooled sites ranged from 0.91 $mg \cdot cm^{-2} \cdot min^{-1}$ (SEM 0.25), on the upper limb, to 0.39 $mg \cdot cm^{-2} \cdot min^{-1}$ (SEM 0.09), on the lower limb. However, intersite comparisons proved nonsignificant ($P=0.12$). Similarly, within the forearm, the mid-dorsal site tended towards a lower transient $\dot{m}_{sw}$ [0.53 (SEM 0.08) compared to 1.00 (SEM 0.14), mid-ventral, and 0.93 (SEM 0.15) $mg \cdot cm^{-2} \cdot min^{-1}$, distal-ventral], with differences again being nonsignificant ($P=0.08$).

The steady-state regional $\dot{m}_{sw}$ were considered to be close to the peak steady-state $\dot{m}_{sw}$ attainable under these conditions. For each core temperature index, and within each subject, the HST were sufficiently stressful to prevent the attainment of thermal homeostasis, while eliciting a mean f_c, during the last 5 min of the HST, equal to 84% of the peak f_c. The final T_{ae} averaged 38.2° C (SEM 0.2) across trials, with the subjects reporting that a considerable effort was required to complete each trial. Under these conditions, $\dot{m}_{sw}$ plateaued in all sites during each trial, thereby permitting $\dot{m}_{sw}$ to be described as *steady state*. Steady-state sweat distributions were significantly correlated with transient $\dot{m}_{sw}$ observed across 10 skin regions (excluding the foot: $r=0.76$ (SEM 0.05); $P<0.05$), and with the estimated mean gland density[2] ($r=0.75$, $P<0.05$). However, the magnitude and distribution of sweat was variable among the subjects, even though the subjects generally displayed higher $\dot{m}_{sw}$ on the forehead and scapula, particularly when compared to sweat rates from lower limb sites (Table 2, Fig. 3). The mean steady-state $\dot{m}_{sw}$ on the forehead [3.20 (SEM 0.51) $mg \cdot cm^{-2} \cdot min^{-1}$] was not significantly greater than $\dot{m}_{sw}$ of the scapula, forearm, hand, stomach or lower back (listed in descending order; $P>0.05$), but was greater than the $\dot{m}_{sw}$ of the chest, upperarm, calf and thigh (Table 2; $P<0.05$). The mean medial torso (chest and lower back) $\dot{m}_{sw}$ was 1.77 $mg \cdot cm^{-2} \cdot min^{-1}$ (SEM 0.10), which did not significantly differ from the mean lateral torso (scapula and abdomen) $\dot{m}_{sw}$ [2.44 (SEM 0.34) $mg \cdot cm^{-2} \cdot min^{-1}$; $P=0.06$]. Between-site forearm, steady state $\dot{m}_{sw}$ differences were not significant ($P=0.17$).

The mid-ventral and distal-ventral forearm steady-state $\dot{m}_{sw}$ [2.15 (SEM 0.41) and 1.59 (SEM 0.31) $mg \cdot cm^{-2} \cdot min^{-1}$, respectively] correlated most closely with the estimated mean body, steady-state $\dot{m}_{sw}$ [1.82 (SEM 0.11) $mg \cdot cm^{-2} \cdot min^{-1}$; $r=0.89$ and 0.85, respectively; $P<0.05$]. However, this strong correlation was not retained for total mass loss. Only the chest steady-state $\dot{m}_{sw}$ was related to total mass loss over the entire HST [1.36 (SEM 0.08) $mg \cdot cm^{-2} \cdot min^{-1}$; $r=0.91$; $P<0.05$], with the time integrated $\dot{m}_{sw}$ of this site accounting for 80% of the variability in total mass loss ($P<0.05$), and being of equivalent magnitude [1.32 (SEM 0.13) $mg \cdot cm^{-2} \cdot min^{-1}$, raw data; $P>0.05$]. When total body $\dot{m}_{sw}$ was derived from rates measured at the eleven local sites, the $\dot{m}_{sw}$ [1.31 (SEM 0.09) $mg \cdot cm^{-2} \cdot min^{-1}$, raw data] did not significantly differ

[2] Regional gland densities were taken from the data of Roberts et al. (1970)

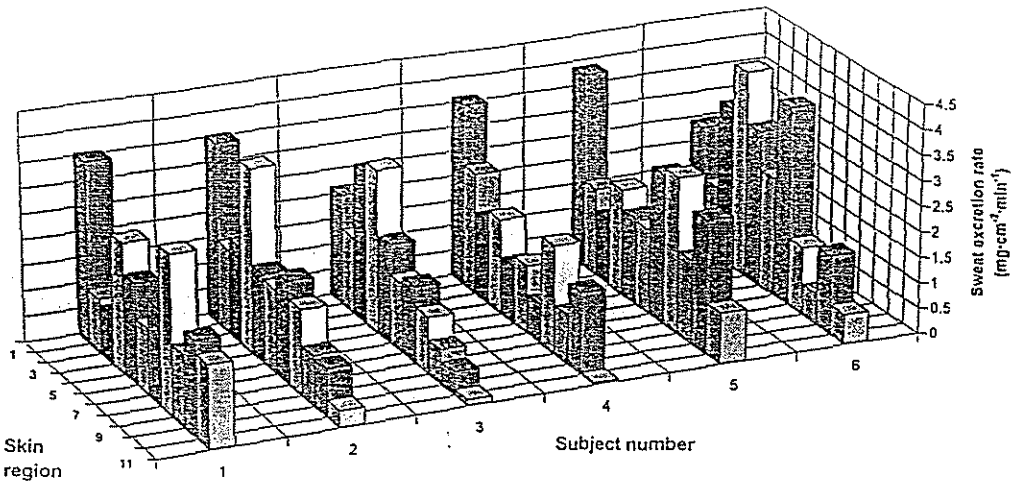

Fig. 3 Mean steady-state sweat rates for 11 skin regions during heat stress trials consisting of 2-min rest and 40-min cycling at 40% peak power ($n=6$; ambient temperature 36.6° C, relative humidity 46.0%). The 11 regions tested were: 1 forehead, 2 medial chest, 3 lateral abdomen, 4 scapula, 5 medial lower back, 6 anterior upper arm, 7 ventral forearm, 8 dorsal hand, 9 anterior thigh, 10 posterior lower leg, 11 dorsal foot

from, or correlate with, the total mass loss ($r=0.36$, $P>0.05$).

Discussion

Only the pooled, lower torso (abdomen and lower back) sweat onset significantly preceded that of the forehead. Thus, for seated subjects exercising in the heat, we found little evidence to support the caudal-to-rostral sweat recruitment which has been reported for supine, resting subjects (Hertzman et al. 1953; Seckendorf and Randall 1961; Park and Tamura 1992). Other groups have also failed to observe this distribution pattern during cycling (Stolwijk et al. 1971; Schvartz et al. 1979), although the early onset of the lower torso has been noted (Nadel et al. 1971). The current sweat onset distribution was not due to the T_{skl} distribution, since, as has been observed for supine subjects (Hertzman et al. 1953; Randall and Hertzman 1953; Seckendorf and Randal 1961), there was a trend toward an inverse relationship between T_{skl} and onset times.

While our data cannot refute the existence of a caudal-to-rostral sweat recruitment pattern, it appears that this pattern is either less pronounced or abolished in the seated posture (Park and Tamura 1992), and with the rapid imposition of a heat load (Seckendorf and Randall 1961). The caudal-to-rostral pattern of onset distribution, seen with supine rest, may possibly be restricted to instances where heat stress is of exogenous origin in combination with a sufficiently high $\bar{T}_{sk}$, since sweating is reported to be initiated from most regions within seconds of exercise onset in a warm environment (van Beaumont and Bullard 1965). Such recruitment is presumably due to a generalised, nonthermal neural outflow, as it has recently been demonstrated that the magnitude of this non-thermal portion of central sweat drive is considerable (Yamazaki et al. 1994).

Assuming that the core temperature indices were representative of the time course of temperature changes within central thermosensitive structures, it was evident that sweat onsets were not dependent on core temperature elevations. The T_{ac} was decreasing, at a rate in excess of $0.1° C \cdot min^{-1}$, concomitantly with 41% of the regional sweat onsets, and it was stable for a further 38% of the sweat onsets. This dissociation of sweating onset has been observed previously (Randall and Hertzman 1953), and did not appear to be an artefact of the T_{ac} index, since the corresponding trends were observed for T_{oes} in 50% and 13% of the sweat onsets (respectively; Fig. 1).

Van Beaumont and Bullard (1965) have demonstrated the influence of T_{skl} on the initial $\dot{m}_{sw}$ during cycling. However, the present low correlations between T_{skl} and the transient $\dot{m}_{sw}$ ($r=0.16$) did not support such a facilitation. Since T_{skl} also modifies established local sweating (Nadel et al. 1971; Elizondo 1973; Ogawa and Asayama 1986), it was envisaged that T_{skl} may have contributed to the topographic variance of steady-state $\dot{m}_{sw}$ between subjects. However, only 2.5% of the distribution could be explained by T_{skl} variance alone ($P>0.05$); perhaps attributable to the narrow range of T_{skl} (35.8–36.6° C). This dissociation of T_{skl} and $\dot{m}_{sw}$ has been observed in resting (Park and Tamura 1992) and exercising subjects (Bothorel et al. 1991), and has been attributed to nonthermal sweat drive accompanying exercise (Bothorel et al. 1991). Furthermore, it has been reported that $\dot{m}_{sw}$ from the skin overlying exercising muscle is not modified by local influences resulting from exercise (Bothorel et al. 1991), and steady-state $\dot{m}_{sw}$ topography is not modified by arm versus leg exercise (Ayling 1986). Thus, the present steady-state $\dot{m}_{sw}$ topographies were not attributable to T_{skl}, exercise mode, or exercise per se.

The present regional $\dot{m}_{sw}$ data are considered broadly to reflect the steady-state sweat topography which exists in asymptomatic, habitually active men during cycling in the heat. These data extend previous observations, since a moderately severe thermal load was imposed upon the subjects in this study, requiring near maximal efforts to complete each HST. The resultant topography of relatively high steady-state sweating (Fig. 3), is broadly in agreement with previous observations obtained at lower absolute $\dot{m}_{sw}$, in that individuals

tend to display their own pattern of sweating (Weiner 1945; Höfler 1968). The notion of Weiner (1945), that steady-state sweating is generally greater at central than peripheral regions, was only supported to the extent that the forehead and possibly the scapula had comparatively high $\dot{m}_{sw}$, particularly in relation to the consistently low $\dot{m}_{sw}$ of the lower limbs.

While the cause of the present steady-state $\dot{m}_{sw}$ topography remains uncertain, the distribution of active sweat glands is a major determinant of within-subject differences (Sato and Dobson 1970; Roberts et al. 1970). However, since the neural drive for sweating is, in itself, insufficient to determine the density of active eccrine glands, and since various regions respond differently to sudorific stimuli (Randall 1946), it would not be expected that the steady-state $\dot{m}_{sw}$ topography could be explained simply on the basis of inter-regional estimates of active glands. Furthermore, it has been suggested that the distribution of sweating may be modified by changes in gland function accompanying postural changes (Ferres 1960; Park and Tamura 1992), individual gland responses to neural input (Randall 1946; Sato and Dobson 1970), and heat acclimation (Höfler 1968; Schvartz et al. 1979).

In conclusion, there existed large interindividual variations in the magnitude and distribution of onset and steady-state $\dot{m}_{sw}$ during cycling in a hot environment. The recruitment of sweating was not caudal-to-rostral, and occurred at equivalent core temperature thresholds across regions. The forehead and scapula regions consistently had higher $\dot{m}_{sw}$, both transiently and at steady-state levels, especially in relation to the chest, upper arm and lower limb. However, the data tended neither to support Hertzman's (1957) observation that $\dot{m}_{sw}$ declines laterally, nor did it support the observation that regional $\dot{m}_{sw}$ approach uniformity with increasing heat stress (Hertzman 1957).

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biodynamics

Whole-body hyperhydration in endurance-trained males determined using radionuclide dilution

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ABSTRACT

MAW, G. J., MACKENZIE, I. L., COMER, D. A. M., and TAYLOR, N. A. S. Whole-body hyperhydration in endurance-trained males determined using radionuclide dilution. *Med. Sci. Sports Exerc.*, Vol. 28, No. 8, pp. 1038–1044, 1996. Despite evidence of hypervolemia following endurance training, there is little information regarding corresponding extravascular fluid volumes. Quantification of such volumes relies upon radionuclide dilution methods, previously hampered by the loss of plasma albumin. It was our purpose to measure human body-fluid distribution in eight endurance-trained males, using a simultaneous radionuclide dilution technique, incorporating radioiodinated serum fibronogen (RISF). Fluid distribution was measured on three occasions, using 2 μCi of RISF, 8 μCi of ^{51}Cr-labeled erythrocytes, and 20 μCi of $Na^{82}Br$ and 450 μCi of $^{3}H_2O$; to measure PV, erythrocyte (RCV), extracellular (ECFV), and total-body water (TBW) volumes, respectively. Respective volume means, standard deviations, and coefficients of variation were: 46.6 (±4.9; 8.44%), 33.3 (±2.9; 3.89%), 258.1 (±12.1; 4.93%), and 654.2 (±13.4; 3.24%) ml · kg^{-1}. The incorporation of RISF provided a reliable modification to previous methods, and revealed a body-fluid expansion in endurance-trained males. It was concluded that such subjects were hyperhydrated, possessing proportionately expanded fluid volumes throughout both intravascular and extravascular spaces. This was attributed to training history and accompanying reductions in adiposity.

BLOOD VOLUME, ERYTHROCYTE VOLUME, EXTRACELLULAR FLUID VOLUME, INTRACELLULAR FLUID VOLUME, PLASMA VOLUME, TOTAL BODY WATER

A commonly reported, though not universally observed (27,33), adaptation accompanying habitual physical exercise is an expansion of blood volume (BV). This is typically reflected by a supranormal plasma volume (PV) (25,28), with trained male runners, for example, having BV of up to 107 ml · kg^{-1}, including 66 ml · kg^{-1} of plasma, compared with BV of between 75 and 85 ml · kg^{-1} in untrained males (10,19).

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The mechanisms of exercise-induced hypervolemia are presently unclear, although both hydrostatic and osmotic causes have been suggested (2,6). Brotherhood et al. (2) suggested that BV was expanded hydrostatically in order to fill the enlarged volume of the trained heart and circulation. Harrison et al. (14) proposed that plasma expansion was stimulated by a post-exercise intravasation of protein, which osmotically drew water from the extravascular compartment. Thus, PV expands in response to physical training, with the possible consequence of reduced mixed-venous hematocrit ($Hct_{\bar{v}}$) and hemoglobin concentrations (25,28).

Depressed $Hct_{\bar{v}}$ and hemoglobin concentrations are not consistent consequences of physical training. For example, when investigating exercise-induced anemia, Magnusson et al. (23) found no difference between the hemoglobin concentrations of long-distance runners and inactive males. When measured directly, hemoglobin mass or erythrocyte volume (RCV) has been found to be enlarged following endurance training, or in comparison with nonathletic counterparts (2,19). Dill et al. (10) reported a mean RCV of 41.4 ml · kg^{-1} in 12 middle-distance runners compared with 35.2 ml · kg^{-1} in a similar, more sedentary, population. Such a trend has been attributed to erythropoietin release following localized hypoxia during physical exertion (2,28). The hypoxia may result from reduced blood flow, from increased oxygen demand, or from the initial hemodilution associated with exercise training. Regardless of the process, it appears that prolonged habitual physical training may provoke an expansion of BV that, while initially confined to the plasma phase, is later reflected by a proportionate expansion of RCV as well.

The proportionate expansion of PV and RCV is not surprising in light of the freedom-of-water movement around the body. Acute reductions in PV during dehydration, for example, have been shown to rapidly deplete

TABLE 1. Physical characteristics of subjects.

	Age (yr)	Height (cm)	Mass (kg)	$\Sigma_{8skinfolds}$ (mm)	$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)
S1	24.6	165.7	72.62	80.7	53.6
S2	24.5	180.2	64.55	60.0	84.9
S3	23.2	183.8	85.81	66.8	69.1
S4	24.3	184.3	88.67	83.0	68.7
S5	21.0	176.4	78.64	89.3	59.4
S6	27.8	183.9	90.07	95.5	53.6
S7	33.8	174.5	73.60	64.3	74.6
S8	28.8	185.6	80.85	44.5	64.0
Mean	26.0	179.3	79.35	73.0	66.0
σ	4.0	6.8	8.81	17.0	10.7

S# = subject identity code, σ = standard deviation, $\Sigma_{8skinfolds}$ = the sum of biceps, triceps, subscapular, mid-axillary, suprailiac, abdominal, thigh, and calf skinfold thicknesses; and $\dot{V}O_{2max}$ = predicted maximal aerobic power.

RCV due to the osmotic transfer of water across the cellular membrane (8). Similarly, it would be surprising if chronic hypervolemia were maintained at the expense of depleted extravascular fluids. However, there is presently a paucity of information regarding the volume and distribution of extravascular fluids in endurance-trained individuals, so that speculation regarding such fluid balance often lacks empirical support.

Measurements of whole-body fluid distribution, which could shed light on this balance, have previously been hampered by methodological limitations including time constraints, subject safety, and internal validity. For example, the initial method of simultaneously measuring total-body water (TBW), extracellular fluid, plasma, and RCV required 36 h to complete, and the withdrawal of 200 ml of blood (24). A later modification allowed similar volume measurements within 3 h (31), but was limited by the use of radioiodinated serum albumin (RISA) to measure PV. RISA dilution has consistently been shown to exaggerate PV in comparison with the dilution of larger tracers due to the constant exchange of albumin between the plasma and interstitial fluids (1,22). In contrast, radiolabeled fibrinogen (RISF) is slow to leave the vascular space (1,9), and, hence, in 1980 the International Committee for Standardization in Haematology (ICSH) (18) suggested that the dilution of RISF may provide a more accurate measure of PV than presently afforded by the use of RISA. It was, therefore, the purpose of this study to develop an accurate and convenient method for measuring the overall distribution of body fluid using a method of simultaneous radionuclide dilution incorporating RISF and to subsequently determine the distribution of body fluids in endurance-trained males.

METHODS

Body-fluid distribution was measured in eight adult males (Table 1), using the simultaneous dilution of four radionuclides. Subjects were asymptomatic, trained endurance athletes, five of whom had achieved National- or State-level representation in their chosen sport. Subjects were fully informed of the experimental procedures, which were approved by the University of Wollongong Human Experimentation Ethics Committee, and subsequently provided informed consent. Body-fluid distribution was assessed on three occasions, each separated by a minimum of 28 d, to permit the decline of residual radioactivity.

Subjects arrived at the laboratory 1 h prior to assessment, in a rested state, following a 12-h overnight fast. Following a standard breakfast (38 $kJ \cdot kg^{-1}$ of body mass plus 5 $ml \cdot kg^{-1}$ of water), 20 ml of blood were collected, without stasis, from an antecubital vein. 10 ml were used in the preparation of a radiochromate injection and 10 ml were stored as a background reference. A urine sample was collected, from which a sample was stored as a background reference. Subjects were first seated at rest for 30 min to stabilize circulation. Our clinical procedures for such measurements typically use a seated posture, and since postural changes affect fluid distribution, we kept subjects seated during both the preparatory and the data collection phases of each trial.

The radiochromate injection, used to measure RCV, was prepared in accordance with ICSH (18) recommendations. Packed erythrocytes from 10 ml of blood were incubated for 20 min at 37°C with 8 μCi of sodium-radiochromate (Amersham Australia. $Na^{51}Cr$), washed three times, and resuspended to an approximate volume of 10 ml in isotonic saline. Four radionuclide injections were then administered into a second antecubital vein, via a Teflon cannula, within a 30-s period. Two μCi of radioiodinated human serum fibrinogen (Amersham Australia, ^{125}I Human Fibrinogen; RISF), the ^{51}Cr-labeled autologous erythrocytes, 20 μCi of sodium-radiobromide (Australian Radioisotopes, $Na^{82}Br$), and 500 μCi of tritiated water (Amersham Australia, ^3H_2O) were injected sequentially to enable measurement of PV, RCV, extracellular fluid (ECFV), and TBW volumes, respectively. The mid-time of the fibrinogen injection was considered as the commencement of assessment (t_0). The exact quantity of each injection was determined gravimetrically, and small quantities were used to prepare the respective radionuclide standards. The cannula was immediately flushed with sufficient heparinized saline to render it suitable for subsequent blood sampling (21).

Blood samples (10 ml) were collected, without stasis, at t_{30}, t_{60}, and t_{180}, and treated with ethylenediamine tetra-acetic acid (EDTA). Collection of each sample was preceded by removal from the cannula of 5 ml of supernatant, and followed by a 10-ml flush of heparinized saline. A urine void was collected and measured at t_{180}. Blood samples, including the background reference, were centrifuged for 40 min at 1700 × g to separate erythrocytes, and the volume of erythrocytes adjusted to account for 2% trapped plasma (3). On seven occasions, a 3-ml aliquot of whole blood was removed prior to blood separation to assist in the calculation of ECFV. Aliquots (3

ml) of plasma and erythrocytes from each sample were placed into glass vials and refrigerated at 4°C pending γ-radiation counting; a 3-ml aliquot of urine was similarly stored. Erythrocyte aliquots were hemolyzed prior to storage, using a trace of powdered saponin (Sigma Chemical Company, S-1252). Further 0.5-ml aliquots of plasma and urine were placed into glass vials in preparation for β-radiation counting. The preparation involved vigorously mixing each aliquot with 0.05 ml of 1 M · l^{-1} hydrochloric acid to solubilize all solid tissues, followed by 9 ml of liquid scintillation cocktail (Packard Instruments, Emulsifier-Safe). The combination of the acid and the cocktail effected clear solutions with homogeneous radiation quench, regardless of the original fluid.

Radiobromine activity was counted on the day of assessment using a calibrated well-type γ scintillation counter (Abbott Laboratories, Auto-LOGIC). Counting of all other radionuclide samples was delayed for 14 d, pending the decay of ^{82}Br, since its activity interfered with the detection of ^{125}I and ^{51}Cr. Spears et al. (31) previously suggested allowing between 5 and 7 d to elapse prior to the measurement of ^{125}I and ^{51}Cr in order to permit the decay of ^{82}Br; however, in the present study, considerable ^{82}Br was still detectable in both plasma and erythrocytes 10 d after their collection, making it impossible to differentiate the respective activities. It was therefore necessary to allow ^{82}Br to decay for 14 d prior to the measurement of ^{125}I and ^{51}Cr, at which time only 0.14% of the original ^{82}Br remained, due to its half-life of 36 h (16). ^{3}H activity was counted using a liquid scintillation counter (LKB Wallac, 1219 Rackbeta), while ^{125}I and ^{51}Cr were counted using the γ scintillation counter. Aliquots were counted twice for ^{82}Br due to its short physical half-life; and three times for ^{3}H, ^{125}I, and ^{51}Cr, for a minimum aggregate of 10,000 counts each. For each radionuclide, the sequence of vials was reversed between counts. Radionuclide concentrations were then averaged and expressed as counts · min · ml^{-1}. After radiation counting, plasma aliquots were assessed in triplicate for protein concentration using a refractometer (Otago, 93032). Plasma protein concentration was considered as the mean of the three refractometer readings.

Three-ml dose standards were prepared for each radionuclide from dilution samples. For ^{51}Cr, this was achieved by combining 0.5 ml of the radiochromated erythrocyte preparation with distilled water to produce a total sample volume of 250 ml. Tritium, ^{82}Br, and ^{125}I standards were similarly prepared, diluting approximately 0.1, 0.1, and 0.2 ml of the respective preparations with distilled water to provide dilution volumes of 500, 500, and 250 ml, respectively. A 3-ml distilled water sample was also stored as a background reference. These standards were subsequently used to determine the exact injected doses of the four radionuclides in the calculation of respective compartmental fluid volumes.

Compartmental fluid volumes were determined using equations described by Chien and Gregersen (4). Thus, in determining TBW, it was necessary to correct plasma ^{3}H concentration ([^{3}H]) for the presence of protein and ^{125}I, and for ^{3}H loss in urine. Similarly, ^{82}Br concentration ([^{82}Br]) was corrected for the presence of protein, and for ^{82}Br loss in erythrocytes and urine; ^{51}Cr concentration ([^{51}Cr]) was corrected for ^{51}Cr loss in urine; and ^{125}I concentration ([^{125}I]) was corrected for the gradual loss of ^{125}I from the vascular space. This latter extravasation was accounted for using semilogarithmic extrapolation of the ^{125}I elution curve to estimate the theoretical [^{125}I] at t_0 (18).

$$TBW = d\left(\frac{(S_H \times S_d \times S_v) - U_v(U_H - iU_I)}{P_H - iP_I}\right)$$

where d = protein displacement factor (see below); S_H = [^{3}H] of the ^{3}H standard; S_d = dilution of the ^{3}H standard; S_V = volume of the $^{3}H_2O$ injection; U_V = volume of urine collected at t_{180}; U_H = [^{3}H] in the urine collected at t_{180}; i = ratio of ^{125}I detected in the ^{3}H and ^{125}I energy ranges; U_I = [^{125}I] in the urine collected at t_{180}; P_H = [^{3}H] in the t_{180} plasma aliquot; P_I = [^{125}I] in the t_{180} plasma aliquot.

$$ECFV = \frac{d}{r}\left(\frac{(S_B \times S_d \times S_V) - U_V(U_B - cU_C) - RCV(E_B - cE_C)}{P_B} - PV\right) \div PV$$

where d = protein displacement factor (see below); r = Gibbs-Donnan ratio (taken as 1.02; 4); S_B = [^{82}Br] of the ^{82}Br standard; S_d = dilution of the ^{82}Br standard; S_V = volume of the Na^{82}Br injection; U_V = volume of urine collected at t_{180}; U_B = [^{82}Br] in the urine collected at t_{180}; c = the ratio of ^{51}Cr detected in the ^{82}Br and ^{51}Cr energy ranges; U_C = [^{51}Cr] in the urine collected at t_{180}; RCV = erythrocyte volume; E_B = [^{82}Br] in the t_{180} erythrocyte aliquot; E_C = [^{51}Cr] in the t_{180} erythrocyte aliquot; P_B = [^{82}Br] in the t_{180} plasma aliquot; PV = plasma volume.

$$RCV = \frac{(S_c \times S_d \times S_V) - 0.25(U_C \times U_V)}{E_C}$$

where S_C = [^{51}Cr] of the ^{51}Cr standard; S_d = dilution of the ^{51}Cr standard; S_V = volume of the Na^{51}Cr injection; 0.25 accounts for the first 45 min of the t_{180} urine collection; U_C = [^{51}Cr] in the urine collected at t_{180}; U_V = volume of urine collected at t_{180}; E_C = mean of [^{51}Cr] in the t_{30} and t_{60} erythrocyte aliquots.

$$PV = \frac{S_I \times S_d \times S_V}{P_{t0}}$$

where S_I = [^{125}I] of the ^{125}I standard; S_d = dilution of the ^{125}I standard; S_V = volume of the RISF injection; P_{t0} = theoretical [^{125}I] in plasma at t_0.

BLE 2. Body-fluid volumes in endurance-trained males, obtained from triplicate determinations, using the simultaneous dilution of radioiodinated serum fibrinogen, ^{51}Cr-labeled ythrocytes, $Na^{82}Br$, and $^{3}H_2O$.

	TBW	ICW	ECFV	IFV	BV	PV	RCV
Absolute volume (ml)							
S1	47,985	30,440	17,760	14,700	5,255	3,060	2,190
S2	43,085	25,135	18,200	14,600	6,180	3,600	2,580
S3	56,375	35,240	21,380	17,870	6,420	3,510	2,910
S4	57,130	34,240	23,185	19,045	6,810	4,140	2,670
S5	50,850	22,985	21,155	17,030	6,740	4,125	2,610
S6	56,390	34,660	21,995	18,200	6,770	3,795	2,970
S7	48,700	29,735	19,200	15,815	5,895	3,390	2,505
S8	54,065	33,840	20,485	16,720	6,350	3,765	2,580
Mean	51,822	30,784	20,420	16,747	6,302	3,673	2,627
σ	5,022	4,631	1,893	1,621	529	365	242
Control	48,000	27,200	21,010	17,800	5,200	3,000	2,200
Relative volume (ml · kg^{-1})							
Mean	654.2	399.4	258.1	211.5	80.1	46.6	33.3
σ	14.3	15.6	13.0	8.1	7.6	5.2	3.1
Control	600.0	340.0	262.6	222.5	65.0	37.5	27.5
Reliability							
υ (%)	3.24	5.82	4.93	5.46	6.00	8.44	3.89
P	0.327	0.234	0.118	0.499	0.799	0.803	0.633

S# = subject identity code, TBW = total body water, ICW = intracellular water volume, ECFV = extracellular fluid volume, IFV = interstitial fluid volume, BV = blood volume, PV = plasma volume, RCV = erythrocyte volume, Control = reference control values for sedentary 80-kg man (17), σ = standard deviation, Reliability = analysis of variance comparing repeat assessments on each subject, P = probability that differences among the three measurements were due to chance alone, and υ = coefficient of variation (averaged across subjects).

The protein displacement factor (d) was calculated as:

$$d = \frac{100 - (0.073 \times [PP])}{100}$$

where [PP] = plasma protein concentration.

On seven occasions, when a whole-blood aliquot was preserved, the extent of ^{82}Br loss into erythrocytes was determined both directly from measuring [^{82}Br] in erythrocytes and from calculating erythrocyte-[^{82}Br] from the whole-blood concentration adjusted for $Hct_{\bar{v}}$. Erythrocyte-[^{82}Br] was then calculated as:

$$E_B = P_B + \frac{(W_B - cW_C) - P_B}{Hct_{\bar{v}}}$$

where P_B = [^{82}Br] in the t_{180} plasma aliquot; W_B = [^{82}Br] in the t_{180} whole-blood aliquot; c = ratio of ^{51}Cr detected in the ^{51}Cr and ^{82}Br energy ranges; W_C = [^{51}Cr] in the t_{180} whole-blood aliquot; $Hct_{\bar{v}}$ = hematocrit of the t_{180} blood sample.

Further calculations were made to determine intracellular water volume (ICW = TBW − ECW, where ECW was ECFV-corrected for the total volume of plasma solutes (4), interstitial fluid volume (IFV = ECFV − PV), and BV (BV = RCV + PV).

Compartmental fluid volumes were then considered for each subject as the mean of three assessments; retest reliability was considered as the mean of each subject's coefficient of variation for each fluid volume. The three assessments were compared using analyses of variance, while comparisons of two related measures were made using paired t-tests. *Alpha* was set at the 5% level, and specific differences were then examined using Tukey's test of Wholly Significant Difference ($Tukey_{WSD}$). Data are reported as means with standard error of the means, unless otherwise stated as standard deviations (σ).

RESULTS

Body-fluid volumes did not differ significantly within subjects between repeat assessments, with the mean intrasubject coefficients of variation generally being less than or equal to 6% (Table 2). The exception was for PV, with a coefficient of variation of 8.44%, for which considerable physiological variation was possible during the 56-d course of the study. The coefficients of variation embraced both physiological and methodological variation, and hence, for all volumes, variation due to methodological error could be considered to be less than the reported coefficient. The compartmental fluid volumes for TBW, ECFV, RCV, and PV averaged 51,822 (±5022), 20,420 (±1893), 2627 (±242), and 3673 (±365) ml, respectively (means ± σ, Table 2). Hence, the respective ICW, IFV, and BV were 30,784 (±4630), 16,747 (±1621), and 6302 (±529) ml (means ± σ, Table 2).

Measurement variability was probably unaffected by the presence of residual radiation as, at the time of reassessment, less than 0.01% of the previous ^{3}H, ^{125}I, and ^{51}Cr doses remained in the blood; no residual ^{82}Br was detected, reflecting the 36-h half-life of the ^{82}Br nuclide (16). The clearance of residual radiation confirmed the report of Fortney et al. (12) that the multiple radionuclide method could safely be used for sequential determinations of human body-fluid distribution, with the total radiation dose imposed by three assessments

amounting to approximately one-fifteenth of the annual dose permitted for an industrial worker (16).

The measurement of ECFV was dependent on the method of calculating intracellular losses of ^{82}Br in the seven assessments when erythrocyte-[^{82}Br] was measured both directly from erythrocytes and calculated from the plasma and whole-blood [^{82}Br]. The apparent erythrocyte-[^{82}Br] was higher in the latter measure (P = 0.001). The corresponding ratio between erythrocyte-[^{82}Br] and that in plasma was 0.804 (±0.019), compared with 0.703 (±0.011) when erythrocyte and plasma concentrations were both measured directly. Consequently, ECFV was 532 (±54) ml smaller when erythrocyte-[^{82}Br] was derived from plasma and whole-blood concentrations compared with the volume determined directly from plasma and erythrocytes (P = 0.001).

Analysis of urine voided showed that 1.25% (±0.08%) of the ^{82}Br dose was excreted during the 3-h assessments. Similarly, an average of 0.85% (±0.10%), 15.60% (±0.97%), and 0.44% (±0.07%) of respective ^{3}H, ^{125}I, and ^{51}Cr doses was also excreted during the course of assessment.

DISCUSSION

The distribution of body fluid was measured in eight endurance-trained males. Previous studies have examined the body-fluid distribution of normal or bed-ridden populations (11,12,31), or the intravascular fluid volumes of athletes (10,19,23). However, no studies were found documenting the distribution of both intra and extravascular fluids in endurance-trained subjects.

The body-fluid volumes of the present sample were all slightly enlarged compared with previously reported reference values (17,18; see Table 2), but were considered normal in light of the subjects' athletic history and body composition (26,30). The subjects were all well-trained, participating in endurance exercise daily, and, on the basis of their estimated maximal aerobic power, were among the top 10% of age-matched Australian males (13). Similarly, based on their skinfold measurements, subjects were among the leanest 5% of aged-matched Australian males (13).

Low adiposity is known to be accompanied by a large volume of TBW relative to body mass due to the higher concentration of water in lean tissue than in fat (26). Hence, in comparison with less lean populations, the present subjects possessed enlarged TBW, resulting in similarly expanded ICW and ECFV. Fluid volumes were still comparable to values at the upper end of the reference data, determined using similar dilution methods on more sedentary male populations (12,31), confirming the validity of the present simultaneous dilution method. For example, Dyrbye and Kragelund (11) reported values of 609, 352, and 265 ml · kg^{-1} for TBW, ICW, and ECFV, respectively.

Despite the present absolute volumes being enlarged, the relative contributions of both the ICW and ECFV to TBW remained similar to previously reported values. ICW and ECFV accounted for 61% and 39% of TBW, respectively, compared with respective values ranging from 57% to 63% and from 37% to 43% in more sedentary populations (11,17,31). Similarly, the relationships between TBW and the intravascular fluid volumes were essentially maintained, with BV and PV accounting for 12.3% and 7.1% of TBW compared with previously reported values ranging from 11.2% to 12.4% and 6.9% to 7.7%, respectively (11,17,31).

Intravascular volumes were also larger, in absolute terms, than those considered normal for more sedentary populations, with the BV averaging 80 ml · kg^{-1}; 47 ml · kg^{-1} for plasma, and 33 ml · kg^{-1} for erythrocytes. In comparison, the ICSH (18) reported normal BV, measured using the simultaneous dilution of RISA and radiochromated erythrocytes (^{51}Cr), to be between 65 and 75 ml · kg^{-1}. Of this BV, plasma accounted for 40 ml · kg^{-1} and erythrocytes for 25–35 ml · kg^{-1}. Similarly, Sawka et al. (27) found that the blood volume of 22 healthy young males was 69 ml · kg^{-1}, including 43 ml · kg^{-1} of plasma and 26 ml · kg^{-1} of erythrocytes. We suggest that the normal BV determined using RISF may be slightly smaller than these values, as PV measured using RISA may be an overestimate of the true circulating plasma, due to its relatively greater leakage from the vascular space. Consequently, it has been shown that PV is enlarged by approximately 6% when measured using RISA dilution compared with that measured using the dilution of RISF (1,22).

The present intravascular fluid volumes were comparable to values obtained from trained subjects. For example, BV between 79 and 85 ml · kg^{-1} have been reported for trained cyclists and runners (23,30), while even larger volumes are found in highly trained athletes (93–104 ml · kg^{-1}) with maximal aerobic power around 75 ml · kg^{-1} · min^{-1} (2,10,19). The relationship between BV and maximal aerobic power has previously been identified (15), and it was considered that the present mild hypervolemia was the result of training history of the subjects rather than methodological error. It was therefore concluded that the radionuclide dilution method employed in the current investigation provided a reliable means of determining the distribution of intravascular and extravascular fluid volumes.

In the present study, IFV was not decreased compared with normal values (17), and Hct_c was normal (23). It is therefore likely that hypervolemia was a reflection of whole-body hyperhydration (5), including a proportionate expansion of all body-fluid compartments. Proportionately expanded PV and RCV have previously been reported following prolonged periods of exercise training (19,28), while depressed Hct_c is primarily evident during the early stages of training, before RCV has responded

(6,25). Increased RCV has been attributed to the stimulation of erythropoietin production (2,28), although, this does not always increase following exercise (20). In light of the present data, expansion of the RCV may also be explained by homeostatic balancing of the body-fluid compartments, since it is unlikely that whole-body hyperhydration would be maintained without a proportionate increase in RCV.

The increased volume of TBW, necessary to support such a whole-body hyperhydration, may be accounted for by increased water retention due to increased plasma renin, aldosterone, and vasopressin activity following exercise (7). In particular, increased sodium retention, due to increased renal sensitivity to aldosterone, may be an important factor in exercise-induced water retention (5). In addition, part of this hyperhydration may be associated with reduced adiposity. Dill et al. (10) suggested that at least one-third of exercise-induced hypervolemia could be explained by reduced adiposity accompanying habitual exercise. In fact, the present data indicate that differences in adiposity between the current trained subjects and more sedentary reference males may actually account for more than one-third of the apparent hyperhydration. For example, PV adjusted for adiposity (estimated from TBW) (26) equated to approximately 52 $ml \cdot kg^{-1}$ of fatfree mass compared with a volume of 50 $ml \cdot kg^{-1}$ from normal values (18; based on body fat content of 20%). It is therefore suggested that, when considering the chronic status of body-fluid volumes, consideration must be given to the relative adiposity of the subject while recognizing the inaccuracies of estimating body fat content.

Methodological Considerations

The present method for measuring body-fluid volumes, using the simultaneous dilution of ^3H_2O, $Na^{82}Br$, RISF, and ^{51}Cr-labeled erythrocytes, produced results that were not only considered normal for the appropriate sample, but were also reliable between repeated assessments. Seven subjects underwent three repeated assessments, spaced at 28-d intervals, and the resultant coefficients of variation between repeated assessments was less than or equal to 6% for measured and derived fluid volumes (Table 2). The exception was PV, which produced a coefficient of variation of 8.4%, which may be attributed to physiological changes between repeat assessments, as well as to methodological error. Considerable physiological variation is possible in PV, and other fluid volumes, due to changes in environmental conditions and health status (12). Such variations may well have occurred during the 56-d course of the present study. Spears et al. (31) reported a variation in plasma volume of just 1.2% during a 7-d period, and variations of 0.5%, 1.3%, and 3.9% in TBW, ECFV, and RCV, respectively. In addition, in the present study, the accuracy of RISF and ^{51}Cr dilution was found to be ±2.50% and ±2.82%, respectively, when measuring *in vitro* volumes of between 2 and 7 l. Hence, it was considered that, in the present study, the variation in volume measurements due to methodological error was probably less than the calculated coefficients of variation, and may well have approached 3% for all compartmental volumes.

The accuracy of ECFV measurement was further influenced by the method of its calculation, specifically the method of accounting for the loss of ^{82}Br into erythrocytes. The discrepancy between directly measured erythrocyte-[^{82}Br] and that calculated from plasma and whole blood was difficult to explain. Had the direct measure been influenced by the presence of ^{82}Br in trapped plasma, it would probably have exceeded the indirect [^{82}Br]. In practice, the reverse was true, making the directly determined ECFV smaller than that derived from indirect calculations. In the former case, the ratio between directly measured erythrocyte-[^{82}Br] and plasma-[^{82}Br] was similar to values previously reported during the calculation of extracellular fluid volume (32). Hence, it was considered that erythrocyte-[^{82}Br] should be measured directly from packed cells, rather than calculated as a function of whole blood concentration, during the measurement of the extracellular space. This procedure is recommended when adopting similar methods to determine ECFV.

CONCLUSION

It has been shown that the simultaneous dilution of ^3H_2O, $Na^{82}Br$, RISF, and ^{51}Cr-labeled erythrocytes provides a reliable method for measuring body-fluid distribution in humans. Each of the respective dilution volumes required several corrections, but, having done so, retest variance was generally around 6%. The compartmental fluid volumes of endurance-trained males were generally larger than normally observed in sedentary adult males, revealing a whole-body hyperhydration, but with normal between-compartment fluid distribution. While hypervolemia has previously been observed in trained subjects, this study has shown that such hyperhydration is also found in all other fluid compartments.

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An evaluation of the role of skin temperature during heat adaptation

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This project sought to evaluate the importance of skin temperature during heat acclimation, using an isothermal-strain model. Two groups of seven matched males, participated (1 h per day, 10 days) in one of two conditions: (i) temperate physical training (TEMP: 22.4±0.7 °C, relative humidity (r.h.) 41.0±0.9%); or (ii) combined physical training and heat acclimation (HEAT: 38.2±0.7 °C, r.h. 39.7±1.3%). Isothermal strain was induced in both groups by rapidly elevating rectal temperature by 1 °C (cycling), then holding it constant by manipulating external work. Subjects completed two three-phase heat stress tests (39.8±0.1 °C, r.h. 38.6±1.2), consisting of 20 min rest, then 20 min cycling at each of 30% and 45% of peak power, before and after each regimen. While there was a difference of 4.2 °C in mean skin temperature between treatments, both regimens elicited a similar peripheral sudomotor increase, indicating a core temperature dependent adaptation. However, based on significant pre- vs. post-acclimation decreases in average auditory canal temperature (0.4±0.1 °C), average forehead skin blood flow (26%), average perceived exertion (11%), and a 5% increase in average forehead sweat rate (0.1±0.04 mg cm^{-2} min^{-1}), the HEAT regimen elicited a more complete acclimation. While elevation in core temperature is critical to acclimation, it also appears necessary to expose subjects to an external thermal stress. This observation has not been previously demonstrated under conditions of isothermal strain, and verifies the importance of skin temperature elevation in the acclimation process.

Keywords acclimation, adaptation, core temperature, heat stress, physical training, sweating.

Thermal adaptation can be induced in response to natural climatic changes (acclimatization: Hellon *et al.* 1956), to artificial heat exposure (acclimation: Nadel *et al.* 1974, Roberts *et al.* 1977) and to endurance exercise producing significant elevations in body core temperature (T_c: Gisolfi & Robinson 1969, Henane & Valatx 1973). Typically, adaptations result in decreased cardiac frequency (f_c), T_c and mean skin temperature ($\bar{T}_{sk}$), and increased sweat rate ($\dot{m}_{sw}$: Nadel *et al.* 1974, Roberts *et al.* 1977). While it is known that the body can adapt to repeated thermal stress of both endogenous (metabolic) and exogenous (environmental) origins, we do not have a full understanding of differences between acclimation states induced by these different sources of heat stress, or indeed, whether these adaptations are physiologically similar. While this question has been of interest to physiologists for some time, the focus of this paper was not simply a comparison of heat acclimation techniques, but rather an investigation of the mechanisms resulting in heat adaptation. Of particular interest was the role of skin temperature elevation in the acclimation process.

A useful model by which to evaluate the role of skin temperature afferents in the acclimation process is found within the comparison of exercise and heat-induced thermal adaptation. Differences in resulting adaptation characteristics may help elucidate the role of cutaneous thermal afferents. However, in attempting to evaluate differences between acclimation states accompanying thermal stress imposed by these two sources, a variety of methods have been employed by which the separate stimuli, and their resultant physiological strain, may be evaluated. Some groups

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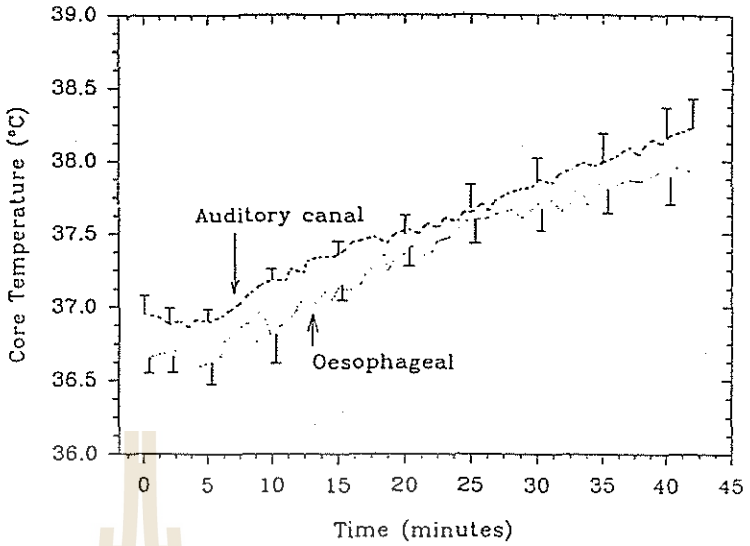

Figure 1 A comparison of zero-gradient auditory canal temperature with oesophageal temperature while subjects ($n = 6$) performed steady-state cycle ergometry at 36 °C (r.h. 50%; modified from Cotter *et al.* 1995).

temperate or hot conditions, and while remaining seated on the ergometer, cycled intermittently to hold T_{re} constant. During acclimation, T_{re} was measured using a thermistor (Yellow Springs Instrument Co., Inc., YSI probe no. 401, Ohio), positioned 12 cm beyond the anal sphincter. Independent trials showed that, while T_{re} lags behind other T_c indices, it did not result in differences in thermal strain being imposed upon the two experimental groups. Thus, both groups experienced a constant and equivalent elevation in T_{re}, but different T_A and $\bar{T}_{sk}$ between conditions.

Pre- and post-acclimation HSTs consisted of three 20-min phases (T_A 39.8±0.1 °C, r.h. 38.6±1.2%). Between-test differences enabled an evaluation of the role of skin temperature within the acclimation process. The presence of an acclimation effect was determined from a significant reduction in T_c during the second HST. Within each HST, subjects rested for 20 min (seated), they cycled at each of two different relative intensities: 30% of peak power (20 min), and 45% of peak power (20 min). $\dot{V}_{O_2peak}$ was determined before and after acclimation (Quinton Instrument Company, QPlex I, USA).

During the HSTs, T_c was determined from auditory canal (aural) temperature (T_{ac}) using zero-gradient aural thermometry (after Keating & Sloan 1975). This technique involves independent monitoring of the temperature of the auditory canal and an outer ear heating pad. The temperature of the latter was controlled to match the temperature of the auditory canal, isolating it from environmental artefacts. This technique has been shown to remove the auditory canal temperature gradient, thus allowing T_{ac} to faithfully track tympanic temperature (Moore & Newbower 1978). The method has been validated within our laboratory against oesophageal (Fig. 1), tympanic and rectal temperatures (Cotter *et al.* 1995): during cycling under similar environmental conditions, T_{ac} is typically 0.21 °C above oesophageal ($r = 0.940$), 0.19 °C greater than tympanic ($r = 0.974$) and 0.21 °C less than rectal temperature ($r = 0.965$).

Skin temperatures were measured at 0.2 Hz from eight sites (Grant Instruments Ltd., 1206 Series Squirrel UK), using thermistors (Yellow Springs Instruments, EU mini thermistors, USA) attached with a single layer of waterproof tape: forehead, right scapula, left upper chest, right upper arm, left lower arm, left hand, right anterior thigh and left calf. $\bar{T}_{sk}$ was derived using the ISO-9886 eight-site equation (International Standards Organization 1992). All thermistors were calibrated against a certified reference thermometer (Dobbie Instruments, Dobros total immersion, Australia).

Skin blood flow (SkBF) was sampled at 1 Hz using laser-Doppler velocimetry (Vasamedics Inc., TSI Laserflo BPM^2, USA: 780 nm wavelength). Zero SkBF was determined using an opaque, white surface, to which all SkBFs were referenced. Since this technique measures blood flow only within a small tissue volume (1.0–1.5 mm radius hemisphere), and since it quantifies relative rather than absolute blood flows, data are reported as voltage outputs directly from the monitor, rather than being converted to blood flow units. SkBF was measured at six sites, using a single probe. During each HST, forearm SkBF was continuously measured during the first 15 min of each 20-min HST phase. To facilitate this, subjects adopted a cycling posture with the upper body in a semi-prone position, with forearms supported on the handle bars. This posture enabled precise between-trial control of the vertical distance between each subject's forearms and heart, minimizing

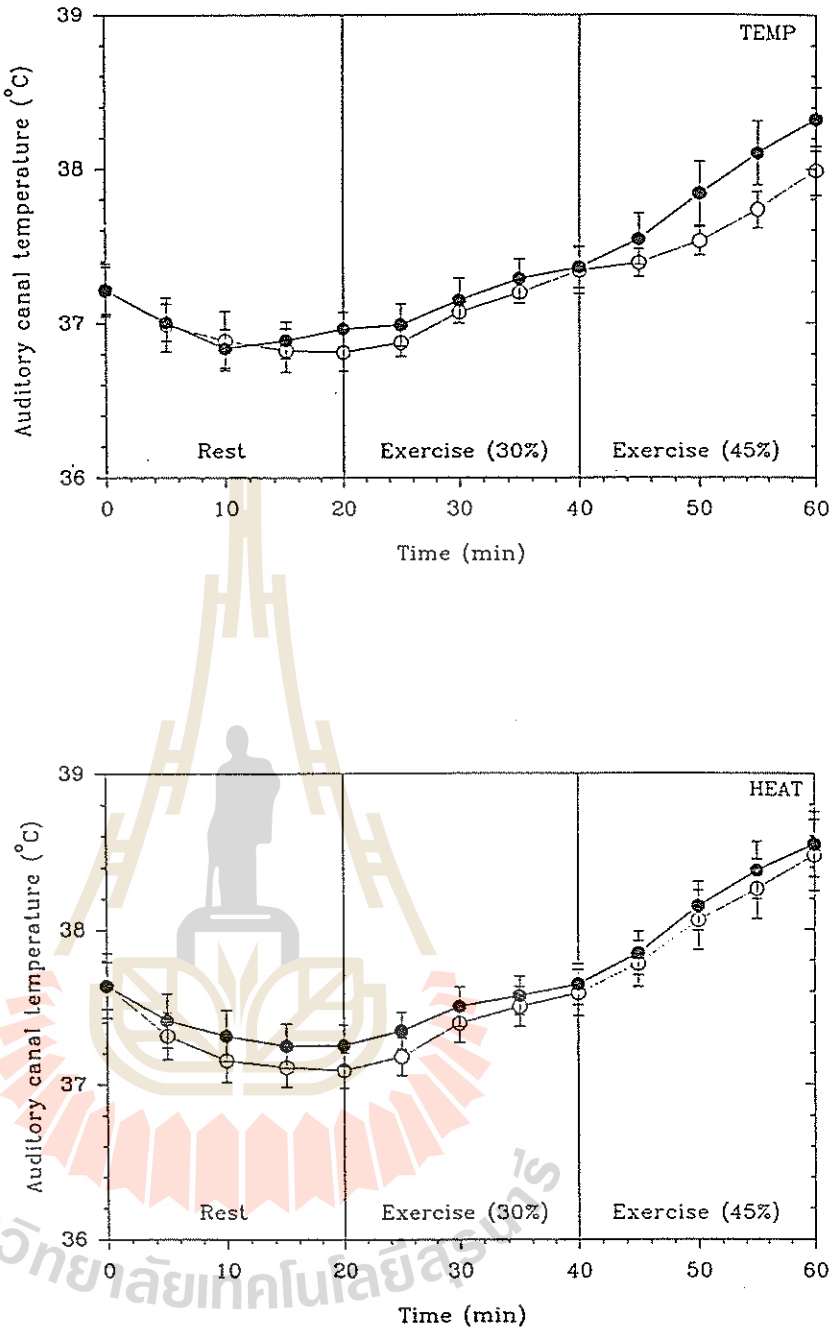

Figure 2 Auditory canal temperature, before and after 10-day heat acclimation regimens, during a three-phase heat stress test (39.8 °C, r.h. 38.6 %). Temperatures are normalized to the basal temperature recorded before the first heat stress test. Data are means with standard errors of the means, and correspond to subjects exposed to either a temperate temperature exercise adaptation (22.4 °C: ●, pre-adaptation TEMP; ○, post-adaptation TEMP), or a combined heat and exercise adaptation (38.2 °C: ●, pre-adaptation HEAT; ○, post-adaptation HEAT).

average times to reach the 1 °C target elevation in T_{re} were 29.5±2.7 (SD) min (TEMP) and 29.2±2.0 min (HEAT; $P > 0.05$). Once elevated, the average T_{re} was 38.0 °C (±0.03) for both groups ($P > 0.05$). $\bar{T}_{sk}$ averaged 33.2 °C (±0.07) for the TEMP group and 37.4 °C (±0.24) for the HEAT group over the 10 days ($P < 0.05$). However, there were no significant differences between the $\bar{T}_{sk}$ recorded for days 1 and 10 of acclimation within either group ($P > 0.05$). These data confirm that the isothermal technique resulted in equivalent elevations in T_{re}, in the presence of significant $\bar{T}_{sk}$ differences. f_c showed significant decreases in both groups throughout acclimation, averaging 133.8 beats min^{-1} (±1.1) and 143.0 beats min^{-1} (±2.4) for the TEMP and HEAT groups respectively across the 10 days ($P < 0.05$). Thus, while the TEMP subjects worked significantly harder, they did so while maintaining a significantly lower f_c. This observation highlights the difficulty of equating strain on the basis of f_c. Both groups experienced non-significant increases in $\dot{V}_{O_2peak}$ over the 10-day adaptation period: 5.62±8.57 (SD) mL kg^{-1} min^{-1} and 4.38±2.44 (SD) mL kg^{-1} min^{-1} respectively ($P > 0.05$). Body mass did not change significantly

Table 2 Core temperature (T_{ae}), mean skin temperature ($\bar{T}_{sk}$) at sweat onset (°C), and sweat gains (mg min^{-1} cm^{-2} °C^{-1}) for the forehead and forearm in two groups of heat acclimated subjects

	Forehead			Forearm		
Treatment/HST	T_{ae} onset	Gain	$\bar{T}_{sk}$ onset	T_{ae} onset	Gain	$\bar{T}_{sk}$ onset
TEMP						
1	37.30	2.52	36.37	37.30	1.92	36.14
	(0.31)	(0.38)	(0.23)	(0.32)	(0.46)	(0.25)
2	36.98	3.19	36.76	37.02	2.71	36.67
	(0.12)	(0.59)	(0.37)	(0.38)	(0.54)	(0.38)
HEAT						
1	37.47	2.83	36.93	37.63	1.95	36.82
	(0.30)	(0.84)	(0.36)	(0.30)	(0.37)	(0.34)
2	36.92	3.14	36.64	37.01	2.08	36.37
	(0.12)	(0.71)	(0.11)	(0.16)	(0.36)	(0.14)

Abbreviations: HST, heat stress test; TEMP, temperate physical training group (22.4 ± 0.7 °C, r.h. 41.0 ± 0.9 %); HEAT, combined heat and physical training group (38.2 ± 0.7 °C, r.h. = 39.7 ± 1.3 %). Data are means with standard errors of the means.

Table 3 Heat adaptation reflected by differences in physiological responses (change scores: test2 − test1) to a three-phase heat stress test conducted before and after two acclimation protocols (temperate exercise (22.4 °C; TEMP), or combined heat and exercise (38.2 °C; HEAT))

		Change score		
Variable	Treatment	Phase 1	Phase 2	Phase 3
T_{ae} (°C)	TEMP	0.06 (0.11)	−0.08 (0.08)	−0.29 (0.10)
	HEAT	−0.16 (0.12)	−0.10 (0.07)	−0.09 (0.05)
Forearm SkBF (volts)	TEMP	−0.12 (0.08)	0.12 (0.08)	0.23 (0.19)
	HEAT	−0.02 (0.09)	−0.02 (0.04)	0.06 (0.10)
$\bar{T}_{sk}$ (°C)	TEMP	0.27 (0.09)*	0.30 (0.07)*	0.18 (0.10)*
	HEAT	−0.39 (0.16)	−0.61 (0.12)	−0.76 (0.11)
f_c (beats min^{-1})	TEMP	−6.11 (1.02)	−7.79 (1.64)	−11.07 (1.21)
	HEAT	−8.85 (1.81)	−5.82 (1.54)	−11.19 (1.78)
RPE	TEMP	—	0.04 (0.26)*	−0.06 (0.28)*
	HEAT	—	−1.82 (0.29)	−1.20 (0.18)
Forehead $\dot{m}_{sw}$ (mg cm^{-2} min^{-1})	TEMP	−0.19 (0.06)*	−0.37 (0.07)*	−0.03 (0.09)*
	HEAT	−0.04 (0.02)	−0.05 (0.07)	0.29 (0.11)
Forearm $\dot{m}_{sw}$ (mg cm^{-2} min^{-1})	TEMP	−0.03 (0.04)*	0.11 (0.07)	0.34 (0.07)
	HEAT	−0.02 (0.02)	0.16 (0.07)	0.41 (0.08)

Abbreviations: T_{ae}, auditory canal temperature; SkBF, skin blood flow; $\bar{T}_{sk}$, mean skin temperature; f_c, cardiac frequency; RPE, rating of perceived exertion; $\dot{m}_{sw}$, sweat rate; *, significant difference between conditions (within each test phase); —, data not available. Data are means with standard error of the means in parentheses.

significant ($P > 0.05$; Table 3). f_c decreased in all HST phases for both the TEMP and HEAT conditions. However, these changes were equivalent between groups for each of these periods ($P > 0.05$), yet the whole-body RPE change score differences were significant ($P < 0.05$; Table 3).

Post-adaptation sweat changes appeared more pronounced at the forearm, with the forehead generally displaying a diminished sweat response during HST2. This latter change was more obvious for the TEMP subjects (Table 3) which, in the light of equivalent mass losses between treatments (TEMP: HST1 = 0.89 ± 0.16 kg, HST2 = 0.76 ± 0.12 kg; HEAT: HST1 = 0.99 ± 0.16 kg, HST2 = 0.94 ± 0.32 kg; $P > 0.05$ for all comparisons), was indicative of a possible peripheral redistribution of sweat secretion. Accordingly, both treatments resulted in equivalent elevations in forearm sweating during the two exercise phases ($P > 0.05$).

DISCUSSION

The purpose of this study was to investigate the role of skin temperature in the acclimation process. This was achieved by maintaining a constant central thermal

load (T_{re}) while exposing subjects to different skin temperatures across two acclimation regimens. Thermal adaptation was defined on the basis of reduced physiological strain, as reflected by changes in one or more variables, following an acclimation protocol. In the current investigation, the criterion variable upon which acclimation status was assessed was T_{ac}, with a significantly lower T_{ac} at the same relative exercise intensity indicating an acclimation effect. As a result, both the HEAT and TEMP acclimation groups experienced this trend, with only the HEAT group experiencing a significant reduction in T_{ac}.

Several factors may account for this observation. First, differences in the pre-experimental acclimation state may have existed between the groups. While this possibility was minimized by conducting trials during winter, and matching subjects for aerobic power, the TEMP subjects had a significantly lower f_c during HST1. This may imply a superior acclimation state before commencing the project. Second, even though 10 days of acclimation have been shown to be adequate to produce acclimation effects (Nadel *et al.* 1974), the duration of the treatment may not have induced complete acclimation. Third, differences in the total volume of exercise performed by the two subject groups could have resulted in differences in the adaptation stimulus. This possibility is not considered to have been important, since the work volume was dictated by changes in T_{re}, which were equivalent between treatments. Given that the key stimulus for heat adaptation is T_c elevation, and that exercise, in the absence of a T_c elevation, is not an adequate stimulus for heat adaptation (Avellini *et al.* 1982, Hessemer *et al.* 1986), we believe that the additional work performed by the TEMP subjects did not impact upon heat acclimation. Therefore, it is considered that both groups were exposed to equivalent central body thermal loads, and centrally-mediated stimuli for adaptation. Finally, and possibly the most appropriate explanation for the between-group differences in acclimation, it is probable that differences between the two treatments resulted in superior acclimation within the HEAT treated subjects. This is not an unique observation, however, it has not been previously demonstrated under conditions of isothermal strain. For example, Shvartz *et al.* (1973), in a comparison of three acclimation protocols in different thermal environments, found that exercise in a hot dry environment produced a superior acclimation effect than did exercise in a temperate environment.

The major differences between the present acclimation regimens were the T_A, and therefore the skin temperature of the subjects, and the total volume of intermittent exercise undertaken during acclimation, with subjects in the TEMP group performing a greater work volume to hold a constant isothermal strain. $\bar{T}_{sk}$ is a function of the balance between radiative, evaporative, convective and conductive exchanges, and across a wide range of T_A (5–30 °C), it remains largely independent of T_c (Nielsen & Nielsen 1965). During adaptation, the $\bar{T}_{sk}$ of the HEAT subjects significantly exceeded that of the TEMP subjects. Given the present regulation of wind speed, relative humidity and pedal cadence, this difference may be primarily ascribed to T_A. Following acclimation, the HEAT subjects experienced a significantly lower $\bar{T}_{sk}$ during all phases of HST2. Similar lowering of $\bar{T}_{sk}$ following heat acclimation has been reported previously (Shvartz *et al.* 1973, 1979), yet $\bar{T}_{sk}$ increased significantly following the TEMP training. For the HEAT treated subjects, this $\bar{T}_{sk}$ change resulted in a more favourable core:periphery thermal gradient, facilitating heat dissipation. The opposite trend was apparent for the TEMP subjects, where T_{ac} remained stable and $\bar{T}_{sk}$ increased. This latter occurrence is attributed to acclimation-induced changes in SkBF and $\dot{m}_{sw}$. Changes in SkBF were generally not significant following TEMP adaptation, although a reduction in SkBF was observed for the upper back, and a higher SkBF was noted at the forehead, chest and thigh during phase two of HST2. Forearm $\dot{m}_{sw}$ increased during HST2 for the TEMP group, however, this change was not as great as the overall decrease in forehead $\dot{m}_{sw}$. Thus, overall evaporative cooling may have decreased, contributing to the increase in $\bar{T}_{sk}$.

A modification of sudomotor function accompanied adaptation in both the TEMP and HEAT conditions. A general increase in sweating following heat acclimation is well established (Henane & Valatx 1973, Shvartz *et al.* 1979). However, in the present investigation, these modifications were not consistent across the forehead and forearm sites. During rest and phase two of HST2, forehead $\dot{m}_{sw}$ was equivalent to pre-acclimation values in the HEAT group, which, in the presence of lower normalized T_{ac} indicates a more sensitive sweat response. During phase three of HST2, the HEAT group produced significantly greater $\dot{m}_{sw}$ than pre-acclimation levels, while normalized T_{ac} remained lower, reinforcing this trend. It is probable that normalized T_{ac} was held lower by the combination of a greater core:periphery thermal gradient and a greater $\dot{m}_{sw}$, which facilitated heat loss, but with some physiological cost. In contrast, the first two phases of HST2 showed a lower forehead $\dot{m}_{sw}$ for the TEMP group, indicating reduced sudomotor activity in the presence of a decreased normalized T_{ac} between HSTs.

Conversely, forearm $\dot{m}_{sw}$ increased during the final phase of the post-acclimation HST in both acclimation

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ORIGINAL ARTICLE

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Sweat distribution before and after repeated heat exposure

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Abstract We investigated the impact of short-term, moderate humidity heat acclimation upon sweat distribution. Eight males completed six daily heat exposures [cycling: ambient temperature 39.5 (0.2)°C, relative humidity 59.2 (0.8)%], during which auditory canal temperature (T_{ac}) was maintained 1.4°C above pre-exposure levels for 70 min by manipulating the work rate. On days 1 and 6, T_{ac} and local sweat rates ($\dot{m}_{sw}$: eight sites) were monitored. The pre-exposure, resting T_{ac} and the T_{ac} sweat threshold decreased from day 1 to day 6 [36.83 (0.05)°C vs 36.62 (0.05)°C, and 36.90 (0.05)°C vs 36.75 (0.05)°C, respectively; both $P < 0.05$]. However, the sweat-onset time, sweat sensitivity ($\Delta\dot{m}_{sw}/\Delta T_{ac}$) and established $\dot{m}_{sw}$ were unaltered ($P > 0.05$). There was also no evidence of a post-acclimation redistribution in established $\dot{m}_{sw}$ between the eight skin regions, though both the sweat sensitivity and established $\dot{m}_{sw}$ for the forehead and hand were significantly greater than at the remaining sites ($P < 0.05$). It is concluded that the 5-day heat acclimation regimen provided only a minimal stimulus for sudomotor adaptation.

Key words Heat exposure · Physical exercise · Acclimation · Sweating

Introduction

Humans have an exceptional capacity to tolerate dry-heat stress, facilitated by a well-developed sweating (sudomotor) mechanism. Sudomotor function may be enhanced through regular exercise (training), or by repeated exposure to an external heat stress, of natural (acclimatisation) or artificial (acclimation) origin. Such enhancement is typified by a reduction in both the core temperature (T_c) threshold for, and the time to, sweating onset (Nadel et al. 1974; Shvartz et al. 1979; Nielsen et al. 1993), an increased sweat sensitivity to changes in T_c (Henane et al. 1977; Libert et al. 1983), and an elevated steady-state sweat rate (Fox et al. 1964; Shvartz et al. 1979; Candas et al. 1983).

Prior to heat acclimation, peripheral (limb) sweat rates ($\dot{m}_{sw}$) are lower than, or similar to, the $\dot{m}_{sw}$ for the rest of the body (Weiner 1945; Kuno 1956; Hertzman et al. 1953; Park and Tamura 1992). However, some groups report that heat acclimation induces a differential elevation in local sweating, such that the post-acclimation limb $\dot{m}_{sw}$ is increased more than central body $\dot{m}_{sw}$ (Fox et al. 1964; Shvartz et al. 1979; Regan et al. 1996). Unfortunately, the methods employed in these studies were not well suited to investigating differential sweat responses. We are aware of only one investigation in which an acclimation-induced sweat redistribution was suitably examined (Höfler 1968), and this report supported a post-acclimation sweat redistribution towards the limbs.

Therefore, while heat acclimation generally improves sudomotor function, there is little direct evidence underlying the notion that it induces a sweat redistribution. Moreover, while most sudomotor changes that accompany heat acclimation appear to take at least 7 days to develop (Armstrong and Maresh 1991), there is considerable evidence that effects of moderate heat acclimation may be induced by a short-term regimen, involving exercise in the heat (Shvartz et al. 1972, 1973a, Gonzalez et al. 1974; Hessemer et al. 1986). Accordingly, we investigated short-term (5 day), moderate humidity heat acclimation, during which T_c was elevated 1.4°C for 70 min per day. During standard heat-stress tests (days 1 and 6), local $\dot{m}_{sw}$ was sampled simultaneously at eight sites, to assess sudomotor adaptation, and a possible redistribution of sweat secretion towards the limbs.

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Methods

Eight healthy, habitually active, but otherwise unacclimated males participated after providing informed consent [values are means (SD): age = 25.6 (7.1) years; mass = 74.6 (8.1) kg; and height = 178.6 (6.2) cm. All methods were approved by the Human Research Ethics Committee.

Subjects (wearing shorts and shoes) completed active, heat acclimation on 6 days consecutively. Subjects cycled intermittently (Quinton Excalibur ergometer, Quinton Instrument Company, USA) in a climate chamber [dry bulb temperature (T_{db}) = 39.5 (0.2)°C; relative humidity = 59.2 (0.8)%; air velocity < 0.1 m · s^{-1}; black globe temperature within 1.5°C of dry bulb temperature], to maintain auditory canal temperature (T_{ac}) 1.4°C above its pre-exposure value. Days 1 [T_{db} = 39.6 (0.1)°C] and 6 [T_{db} = 39.5 (0.1)°C] were standardised heat-stress test (HST) days, used to assess thermoregulatory effector function. Accordingly, HSTs were conducted at the same time of day, and subjects were asked to attain a state of euhydration (consume 20 ml · kg^{-1} of water 1–2 h before reporting to the laboratory), and to abstain from strenuous exercise, alcohol (20 h) and caffeine (2 h).

Following preparation, subjects entered the heated chamber and were seated on the cycle, adopting a posture with the forearms and hands supported on a padded frame mounted on the handlebars. This posture was maintained for the duration of each HST. An initial 10 min of seated, non-exercising data were collected, after which cycling commenced. The work rate was set initially to 50% [185.3 (25.6) W] of the work rate obtained at each subject's peak aerobic power. A 70-min period began when T_{ac} had risen 1.2°C, and work rate was periodically modified to maintain an elevation of 1.4°C. Pedal cadence was selected by each subject, and was held constant within subjects. No fluid replacement was provided during HSTs.

The non-HST sessions (days 2–5) differed from the HST sessions in the following respects: (1) cycling posture was not rigidly controlled; (2) fluid replenishment was provided (1200 ml · h^{-1}); (3) there was no initial period of seated rest; and (4) the initial work rate was increased by 2% per day.

T_{ac} was measured using an insulated, moulded earplug and aural thermistor (Edale Instruments, UK). While T_{ac} is influenced by environmental conditions, this artefact is minimised by insulating the thermistor and when T_{db} is close to T_c. These conditions were satisfied, and, under this state, T_{ac} and tympanic temperature faithfully track oesophageal temperature (Cotter et al. 1995). Thus, dampening effects sometimes seen with T_{ac} are negated within the current experimental design, in which subjects acted as their own controls, and in which the environmental conditions were equivalent between HSTs. Accordingly, while sweat-onset thresholds and sensitivities may not be identical to those obtained using oesophageal temperature, under the current design they provided reliable within-subject assessments of sudomotor function.

Body mass changes provided an estimate of total sweat activity (uncorrected for metabolic or respiratory losses; A and D electronic balance, Model No. fw-150k, USA). Local $\dot{m}_{sw}$ were measured simultaneously using ventilated sweat capsules [3.16 (0.05) cm^2; Multi-Site Sweat Monitor, Clinical Engineering Solutions, Australia], attached to the dorsal foot, lateral leg, lateral thigh, dorsal hand, ventral forearm, anterior arm, rostral scapular region (right side) and the forehead. To avoid pressure-induced hidrosis, sweat capsules were constructed with 4.5-mm flanged perimeters, to which was applied a skin adhesive (Collodion, Mavidon Medical Products, USA), sealing the capsules to the skin. The air ventilating each capsule was initially passed over a saturated solution of lithium chloride (0.4–0.6 l · min^{-1}) of a constant, known temperature. The relative humidities (capacitance hygrometers) and temperatures (thermistors) of post-capsular air were recorded at 1 Hz (Metrabyte DAS 1602, USA) and saved on computer. All hygrometers were calibrated using saturated solution standards of lithium chloride, sodium iodide, potassium chloride, sodium chloride and potassium diphosphate. This system and the relevant computations are described elsewhere (Turner and Gass 1993; Taylor et al. 1997).

Sweat onset (threshold) was defined as the point from which $\dot{m}_{sw}$ was maintained above baseline for at least 5 min. Since different skin regions commenced sweating at various times within HSTs, thresholds were determined from the raw data for each of the eight sites. The $\dot{m}_{sw}$ sensitivity (gain) was calculated as the increase in $\dot{m}_{sw}$ during the period 3–7 min after exercise onset, relative to the corresponding change in T_{ac} ($\Delta\dot{m}_{sw}/\Delta T_{ac}$). This period was selected since it corresponded with a rapid and essentially linear elevation in $\dot{m}_{sw}$, which was associated with the onset and early elevation of T_c. The mean $\dot{m}_{sw}$ for the period 12–20 min after exercise onset was deemed to represent the established $\dot{m}_{sw}$ and was intended to provide an approximation of the steady-state $\dot{m}_{sw}$. This was the last period during which $\dot{m}_{sw}$ was minimally influenced by alterations in work rate (to maintain T_{ac} elevation) or the loss of data. While some regions had not fully attained the peak $\dot{m}_{sw}$ for all subjects during this period, $\dot{m}_{sw}$ values had established a consistent inter-regional relationship, and were approaching steady-state levels.

Skin temperature was measured adjacent to each sweat capsule and at the medial chest, using surface thermistors (Yellow Springs Instruments, EU mini thermistor, USA), which were attached with a single layer of waterproof tape. Mean skin temperature ($\bar{T}_{sk}$) was determined using the ISO-9886 eight-site equation (International Standards Organisation 1992). All thermistors were calibrated (± 0.05°C) against a certified reference mercury thermometer (Dobros total immersion thermometer, Dobbie Instruments, Australia). Temperatures were recorded at 0.2 Hz using a portable data logger (Grant Instruments, 1206 Series Squirrel, UK).

Cardiac frequency (f_c) was monitored from ventricular depolarisation, logged at 0.2 Hz (Polar Electro Sports Tester, model PE3000, Finland). Thermal sensation and discomfort votes were recorded using a 13-point scale (1 = unbearably cold, to 13 = unbearably hot) and a 5-point scale (1 = comfortable, to 5 = extremely uncomfortable) respectively. Affect state and perceived exertion were measured using an 11-point scale (−5 = feeling very bad, to +5 = feeling very good; after Rejeski 1985), and the 15-point Borg scale (6 = very very light, to 20 = very very hard; after Borg 1962), respectively.

Inter-regional differences in sweat responses were analysed using ANOVA for repeated measures, and isolated using Tukey's HSD procedure (α = 0.05). The influence of acclimation status (day 1 versus day 6) was examined using the one-sample Student's *t*-test, for which the Bonferroni correction was applied to each of four families of dependent variables: (1) temperature data; (2) sweat data; (3) psychophysical measures; and (4) external work performed. Thus, probability values, reported for differences between days 1 and 6, denote the "family-wise probability value" (family-wise error rate = α = 0.05), and not the individual probability value for the variable concerned.

Results

The regional $\dot{m}_{sw}$ responses for one subject are illustrated for one trial (day 6), and serve to illustrate several recurrent features of the sudomotor response (Fig. 1). Sweat-onset thresholds occurred at variable times between skin regions within an HST, although the commencement of exercise generally provided an adequate non-thermal stimulus for all regions that had not previously begun sweating. This non-thermal drive was strong, though transient, as reflected in the early bursting and overshoot in $\dot{m}_{sw}$ (e.g. Fig. 1, min 10). Thus, only when T_{ac} began to rise did sweat elevation become well established. While the sudomotor response displayed variable inter-regional sensitivities, the transition towards steady-state levels occurred approximately simultaneously across skin regions.

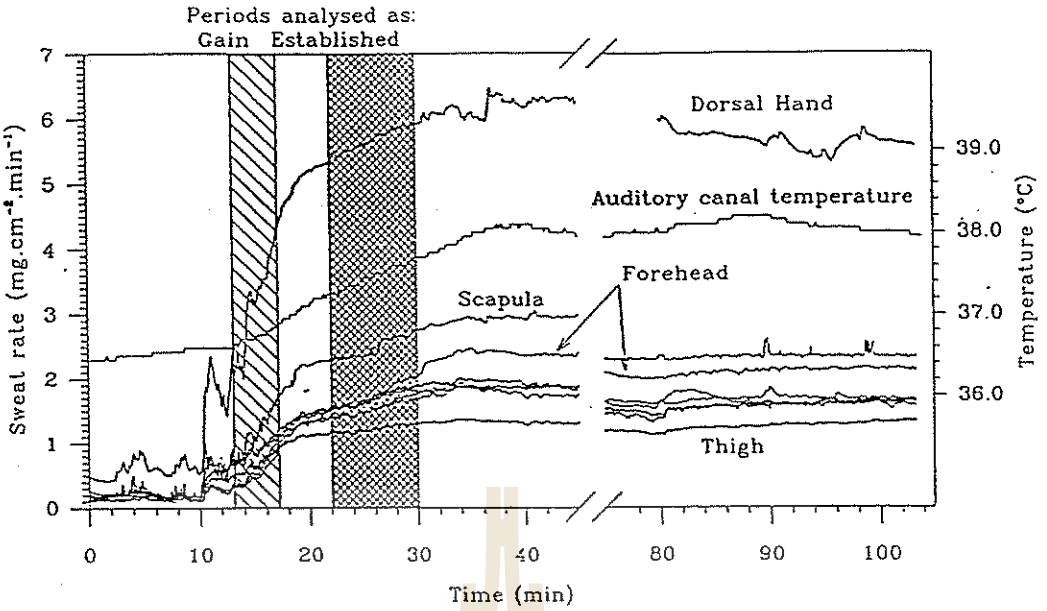

Fig. 1 Sample sweating patterns, recorded at eight skin regions, and auditory canal temperatures for one subject (no. 6) during an exercise heat stress trial (day 6: ambient temperature 39.5°C, relative humidity 59.2%), in which auditory canal temperature was elevated and maintained 1.4°C above the pre-exposure value for a 70-min period, using intermittent cycling. Four sweat traces are not labelled (foot, leg, forearm and arm) due to extensive overlap. The *shaded bands* indicate periods used for calculating sweat sensitivity and established sweat rates

Table 1 Physiological and psychophysical indices of thermal strain before and after a 5-day exercise and heat acclimation [ambient temperature 39.5 (0.2)°C; relative humidity 59.2 (0.8)%], in which subjects maintained auditory canal temperature at 1.4°C above the resting value for 70 min, using intermittent cycling. Data are means with standard error of the means. Sweat data are the means of eight sites. Sweat sensitivity is for the 4-min period beginning 3 min after exercise onset. Cardiac frequency was averaged from 5 to 95 min. Data for psychophysical variables are for 12 min after exercise onset. n = 8, except for mass loss (n = 6) and cardiac frequency (n = 7)

Variable	Day 1	Day 6
Total work performed (kJ)	441.7 (30.1)	444.4 (39.2)
Sweat threshold (°C)	36.90 (0.05)	36.75 (0.05)*
Sweat threshold (s)	523.5 (35.9)	494.2 (50.8)
Sweat sensitivity ($mg \cdot cm^{-2} \cdot min^{-1} \cdot °C^{-1}$)	3.5 (0.3)	3.2 (0.4)
Mass loss ($mg \cdot cm^{-2} \cdot min^{-1}$)	2.2 (0.5)	2.1 (0.3)
Cardiac frequency ($beats \cdot min^{-1}$)	136.8 (6.2)	128.4 (5.4)*
Perceived exertion (6–20)	11.9 (0.5)	12.1 (0.5)
Thermal sensation (1–13)	9.4 (0.2)	9.3 (0.2)
Thermal discomfort (1–5)	2.0 (0.2)	1.6 (0.2)
Affect (−3 to +3)	0.4 (0.5)	0.0 (0.4)

* Significant difference between day 1 and day 6

Heat acclimation response (day 6 vs day 1)

The nature of the HST, with no fluid replacement on days 1 and 6, resulted in greater heat strain during the HST days than was encountered during the intervening acclimation days. For example, the total external work required to maintain the same T_{ac} elevation on day 5 was markedly greater than that on day 6 [329.1 (15.4) kJ vs 246.4 (23.5) kJ; $P < 0.05$]. However, the total amount of external work performed to maintain the target T_{ac} elevation over the 70-min heat-stress period was not significantly greater on day 6 than on day 1 ($P > 0.05$; Table 1), indicating that neither the confounding influence of a training effect, nor heat acclimation due to the experimental protocol, had a significant impact upon heat balance.

The pre-exposure resting T_{ac} was significantly lower on day 6, relative to that on day 1: 36.83 (0.05)°C versus 36.62 (0.05)°C ($P < 0.05$). The overall T_{ac} sweat thresholds (averaged across sites) were significantly lower on day 6 ($P < 0.05$), although the overall sweat-onset time was equivalent (Table 1, Fig. 2A). The $\bar{T}_{sk}$ did not decrease from day 1 to day 6, either at the start of the HST exposure [35.0 (0.2)°C and 35.2 (0.1)°C; $P > 0.05$], or when averaged across the trial duration [37.1 (0.1)°C and 37.1 (0.1)°C; $P > 0.05$].

The heat acclimation regimen did not significantly alter the sweat sensitivities, or the established $\dot{m}_{sw}$, either within skin regions, or when averaged across regions (Fig. 2B,C, Table 1; $P > 0.05$). Accordingly, whole-body sweat loss was unaffected by the 5-day acclimation regimen (mass change: Table 1; $P > 0.05$). Similarly, the psychophysical responses were equivalent between HSTs (Table 1; $P > 0.05$), indicating that effort sense, thermal sensation and discomfort, and their impact on affect state in the heat, were unaltered by short-term heat acclimation. f_c was significantly reduced on day 6 (Table 1; $P < 0.05$).

Acclimation-induced sweat redistribution

There was no evidence of a post-acclimation redistribution of sweating between skin regions (Fig. 2B,C).

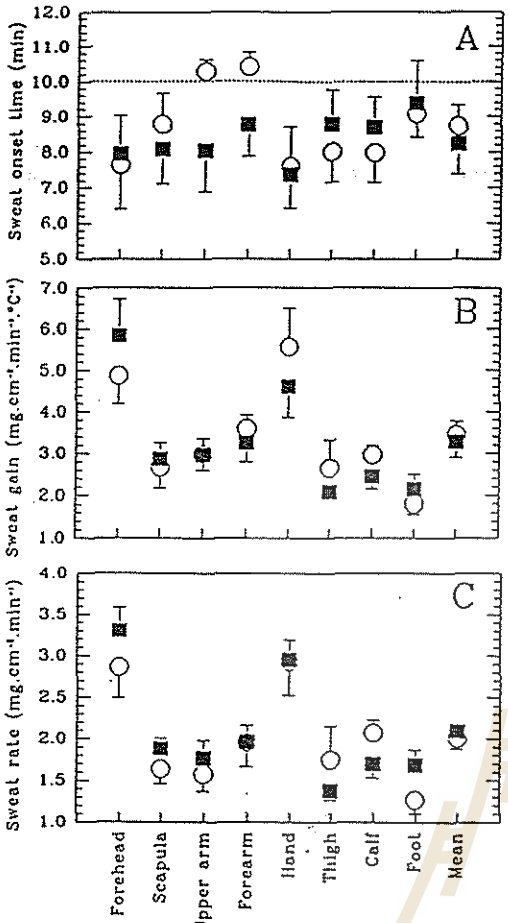

Fig. 2 The mean (SE) sweat thresholds (A), sweat sensitivities (B), and established sweat rates (C), for each of eight skin regions, on days 1 (○) and 6 (■) of an active heat-acclimation regimen [ambient temperature 39.5 (0.2)°C; relative humidity 59.2 (0.8)%; n = 8]. The *dashed line* in Fig. 1A denotes the commencement of exercise. The data labelled "*mean*" were derived by averaging across the eight sites. Only the mean core temperature sweat-onset threshold was lower on day 6 than on day 1 ($P < 0.05$)

Changes in the established $\dot{m}_{sw}$ from days 1 to 6 (expressed as a percentage of the value on day 1) revealed that six of the eight regions experienced an elevation in their established $\dot{m}_{sw}$: forehead [127.6 (19.3)%], scapula [126.0 (15.5)%, n = 7], arm [122.6 (19.2)%], forearm [106.3 (7.3)%], hand [110.4 (12.5)%, thigh [94.0 (11.3)%, n = 7], leg [81.7 (5.3%] and foot [134.4 (11.2)%, n = 6; $P > 0.05$ for all regions].

Sweat distribution patterns during exercise in the heat

Since the inter-regional sweat onset, sensitivity and established sweating were not appreciably influenced by the acclimation regimen (Fig. 2A–C), statistical analyses of inter-regional differences were performed using data pooled from days 1 and 6. These analyses permitted an overall assessment of sweat distribution during exercise in the heat. Sweat-onset thresholds were equivalent across the eight skin regions ($P > 0.05$). However, the forehead and hand exhibited higher sweat sensitivities, with the forehead sensitivity being greater than for all regions except the hand, which, in turn, was higher than for all other regions except the forearm ($P < 0.05$). The correlation between the sweat sensitivity and established $\dot{m}_{sw}$ distributions was 0.89 ($P < 0.05$). Accordingly, the established $\dot{m}_{sw}$ values for the forehead [3.22 (0.32) mg · cm^{-2} · min^{-1}; mean (SE)] and hand [3.07 (0.31) mg · cm^{-2} · min^{-1}] were greater than those for all other regions: forearm [2.04 (0.29)], leg [1.91 (0.19)], scapula [1.76 (0.16)], arm [1.63 (0.21)], thigh [1.57 (0.24)] and foot [1.48 (0.18); $P < 0.05$].

Discussion

The heat load imposed throughout any given heat acclimation session was greater both in magnitude and duration than that which we have previously shown able to elicit heat-adaptation effects (Regan et al. 1996). However, the number of repeated heat exposures was reduced in the current project. From those indices which may be used to evaluate acclimation (Table 1, Fig. 2), only the T_{ac} sweat threshold and f_c showed significant changes following the 5-day protocol. Furthermore, there was no evidence of a redistribution of sweat secretion towards peripheral skin regions. Thus, it is concluded that, while short-term heat acclimation may cause adaptation trends in some physiological variables (Armstrong and Maresh 1991), the current acclimation regimen elicited only central sudomotor changes (reduced sweat threshold), with no evidence of peripheral sudomotor adaptation (elevated sensitivity or steady-state $\dot{m}_{sw}$).

Previous groups have reported that short-term heat acclimation induces sudomotor adaptation (Shvartz et al. 1972, 1973a, b; Gonzalez et al. 1974; Hessemer et al. 1986), while others report a tendency for more prolific sweat secretion to occur on the limbs relative to the torso or head following more extended acclimation regimens (Fox et al. 1964; Höfler 1968; Shvartz et al. 1979; Regan et al. 1996). Two explanations may account for the current disparity with the latter observation.

First, the present subjects may have already possessed some degree of exercise-induced acclimation, by virtue of their habitual physical activity level. Physical training induces glandular or neuro-glandular sweat adaptations (Yamazaki et al. 1994), as typified by an elevated sweat sensitivity, whereas heat acclimation tends to induce adaptation of a central or neural origin (e.g. reduced sweat threshold: Nadel et al. 1974; Libert et al. 1983; Sugenoya et al. 1986). The present data revealed a reduced T_{ac} sweat threshold with no change in sweat sensitivity (Table 1, Fig. 2). Thus, the exercise habits of our subjects may have caused sufficient localised training effects on glands, so that the impact of the short-term heat acclimation was minimised.

Second, while the acclimation regimen produced some evidence of physiological adaptation, the protocol

ıay have been too brief to permit more complete ıdomotor changes and a sweat redistribution. While ɜductions in resting T_{ac} and sweat threshold were obɜrved (Table 1), a redistribution of sweat secretion ɣould not be anticipated in the absence of more general udomotor changes. Furthermore, improved peripheral udomotor function is among the more delayed of the ɔhysiological adaptations, requiring 8–14 days to obtain bout 95% of the final acclimation status (Armstrong nd Maresh 1991). Therefore, our current data indicate hat a sweat redistribution does not occur following hort-term heat acclimation, at least in physically conlitioned individuals. While both the brevity of the aclimation regimen and the cardiorespiratory training tatus of our subjects may have contributed to this obervation, we cannot discount the possibility that a weat redistribution does not occur during active, noderate humidity, heat acclimation.

The pooling of inter-regional sweat onsets, sensitivies and established $\dot{m}_{sw}$ from days 1 and 6 provided a neans for verifying previous observations of sweat disribution during exercise in the heat (Cotter et al. 1995). The typical pattern of sweat onset observed during the HSTs was characterised by a relatively short latency with a strong inter-regional uniformity, both before and after heat acclimation (Fig. 2A), i.e. sweating commenced at approximately the same time at all eight sites. This pattern of sweat onset is consistent with previous observations for heat stress whilst in the upright position and exercising (Nadel et al. 1971; Cotter et al. 1995), and was attributed to the combined influence of exogenous and endogenous heat sources, while also being posture dependent (Ogawa 1984). However, this sweat-onset pattern differs markedly from that obtained during both supine rest and mild heat stress. In the former case, a caudal-to-rostral pattern of sweat onset has been demonstrated (Randall and Hertzman 1953; Hertzman 1957; Park and Tamura 1992).

During mild heat stress, steady-state $\dot{m}_{sw}$ has been reported to be most prominent at the forehead, and considerably lower on the arms (Hertzman et al. 1953), hands (Weiner 1945) or chest (Park and Tamura 1992). In the current study, both before and after heat acclimation, the sweat sensitivities and established sweat rates were remarkably uniform between regions, with the exception of notably higher values from the forehead and hand (Fig. 2C). With the exception of Kuno (1956), the high sudomotor output of the dorsal hand surface has not been previously reported. Both the high forehead and hand $\dot{m}_{sw}$, and the $\dot{m}_{sw}$ equivalence at the remaining six skin sites are consistent with observations of sweat-gland density, which show a relatively uniform density across most body regions except for higher densities on the hand, and especially the forehead (Montagna and Parakkal 1974; Samueloff 1987). We have recently reported sweat distributions (11 sites) in subjects during continuous cycling in the heat (Cotter et al. 1995). In these experiments, there was a tendency for the forehead and scapula to sweat more profusely than other regions, with the remaining nine regions producing equivalent sweat responses. Differences between the $\dot{m}_{sw}$ of the scapula (Cotter et al. 1995) and the hand (current observations) probably reflect technical improvements designed to minimise pressure-induced hidrosis at all capsule sites, which may have existed previously. This trend is supported by evidence that the scapula sweat-gland density is not higher than that on the hand (Montagna and Parakkal 1974; Samueloff 1987). It is therefore considered that the present data may more closely reflect the actual inter-regional sweat distribution pattern seen during moderately strenuous cycling in humid heat, where the established $\dot{m}_{sw}$ distributions vary in accord with differences in sweat-gland density, with the forehead and hand showing the greatest $\dot{m}_{sw}$ values.

Conclusion

Following 5 days of combined cycling and moderately humid heat acclimation, there was a significant reduction in the T_{ac} sweat threshold, but there were no other indications of sudomotor adaptation, or evidence of a redistribution of the sweat secretion pattern. While short-term heat acclimation may elicit an adaptation within some physiological variables, it had only minimal impact on sudomotor function. It is therefore concluded that a 5-day heat acclimation regimen, where T_{ac} is maintained 1.4°C above the pre-exposure level for 70 min, provides an inadequate adaptation stimulus for sweating within moderately trained subjects.

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ORIGINAL ARTICLE

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Effects of artificially-induced anaemia on sudomotor and cutaneous blood flow responses to heat stress

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Abstract The influence of artificially induced anaemia on thermal strain was evaluated in trained males. Heat stress trials (38.6°C, water vapour pressure 2.74 kPa) performed at the same absolute work rates [20 min of seated rest, 20 min of cycling at 30% peak aerobic power ($\dot{V}O_{2peak}$), and 20 min cycling at 45% $\dot{V}O_{2peak}$] were completed before (HST1) and 3–5 days after 3 units of whole-blood were withdrawn (HST2). Mild anaemia did not elevate thermal strain between trials, with auditory canal temperatures terminating at 38.5°C [(0.16), HST1] and 38.6°C [(0.13), HST2; $P > 0.05$]. Given that blood withdrawal reduced aerobic power by 16%, this observation deviates from the close association often observed between core temperature and relative exercise intensity. During HST2, the absolute and integrated forearm sweat rate ($\dot{m}_{sw}$) exceeded control levels during exercise ($P < 0.05$), while a suppression of forehead $\dot{m}_{sw}$ occurred ($P < 0.05$). These observations are consistent with a possible peripheral redistribution of sweat secretion. It was concluded that this level of artificially induced anaemia did not impact upon heat strain during a 60-min heat stress test.

Key words Anaemia · Exercise · Heat stress · Skin blood flow · Sweating

Introduction

Anaemia occurs across the health spectrum. At rest, acute anaemia, and the resultant reduction in oxygen transport, is compensated for by elevations in cardiac output and blood flow redistribution, resulting in increased oxygen extraction (Woodson et al. 1978). However, these mechanisms do not compensate satisfactorily for reduced oxygen carriage during submaximal or maximal exercise (Celsing et al. 1986; Woodson et al. 1978), and peak aerobic power ($\dot{V}O_{2peak}$) and endurance are diminished by acute anaemia (Kanstrup and Ekblom 1984; Osborne et al. 1994).

During incremental exercise in the heat, there is an attenuation of the cutaneous vasodilatory response to rising body core temperature (T_c) once the higher work loads are reached. This reflects a preferential redistribution of blood to the exercising muscles (Brenglemann et al. 1977) and is incompatible with thermal homeostasis during endurance exercise. Consequently, heat dissipation no longer matches heat production, and the body stores heat. One may predict that anaemic subjects would experience a proportionately greater demand for blood at the active muscles, since a reduction in red cell numbers and haemoglobin concentration ([Hb]) reduces oxygen transport. Thus, anaemia would act to exacerbate the heat-induced skin blood flow ($\dot{Q}_{skin}$) attenuation that occurs during exercise. Acute anaemia should therefore not only reduce $\dot{V}O_{2peak}$ and endurance, but may also compromise thermal tolerance. Since the authors were unaware of research that has evaluated this possibility, we used artificially induced (blood drawn) anaemia to test this hypothesis.

Methods

Six endurance-trained males participated in heat stress trials (HST) before (control: HST1) and after being rendered mildly anaemic (HST2: 25–36 days later). This relative anaemia was induced by the removal of 3 units (1.35 l) of whole blood over the course of 5 days with a 24-h period separating each unit withdrawn. Since we were not interested in true clinical anaemia, but acute mild anaemia, in which significant changes in red cell numbers, [Hb] and haematocrit could be induced without eliciting overt symptoms of anaemia, the removal of 3 units of whole blood was deemed adequate. Each HST was performed at the same time of day, with the latter conducted 3–5 days after the last phlebotomy, at which time it was assumed

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subjects were isovolaemic. All procedures were approved by the Human Research Ethics Committee, and subjects provided informed consent.

Three-phase HSTs were used, each consisting of 20 min of seated rest, 20 min of cycling at 30% peak power [126.5 (14.1) W (SD); Monark friction-braked ergometer, Sweden], and 20 min of cycling at 45% peak power [188.5 (20.3) W] at an ambient temperature of 38.6°C [(0.7)°C (SD), water vapour pressure of a 2.74 kPa, black globe temperature within 1.5°C of dry bulb, and wind velocity of $<0.1\ m \cdot s^{-1}$]. Aerobic power was determined using a cycle ramp protocol ($36\ W \cdot min^{-1}$; Quinton Excalibur and Q-Plex I, Quinton Instrument Company, USA), and facilitated the determination of work rates for HST1. The arterial oxygen saturations (S_aO_2) obtained during this protocol were used to approximate arterial oxygen concentrations for the HSTs [$c_aO_2 = ([Hb] \cdot S_aO_2 \cdot 1.31) + 0.3\ ml \cdot l^{-1}$; Ohmeda 3700e pulse oxymeter, USA]. In HST1 (control), work rates averaged 30.3 (0.5)% (SD) and 45.1 (0.3)% $\dot{V}O_{2peak}$. As we were interested in evaluating the influence of mild anaemia upon the ability to perform a standardised work task, the same absolute work rates were used for HST2. The removal of 3 units of whole blood reduced $\dot{V}O_{2peak}$ (Table 1; $P < 0.05$). Therefore, the mean relative exercise intensities increased to 33.2 (1.2)% ($P < 0.05$ HST1 versus HST2), and 49.6 (1.5)% of $\dot{V}O_{2peak}$ ($P < 0.05$).

The sweat rate ($\dot{m}_{sw}$) was measured at the forehead and forearm using a capacitance hygrometry sweat capsule system (Sweat Monitor, Clinical Engineering Solutions, Australia: Turner and Gass 1993), and from changes in body mass (uncorrected for metabolic and respiratory mass changes). In the former instance, air was pumped ($0.4\ l \cdot min^{-1}$) through a sealed flask containing a saturated salt solution (lithium chloride) and onto the skin below each sweat capsule ($2.19\ cm^2$). The exhaust air from each capsule was passed into sealed chambers containing humidity and temperature sensors. The humidity sensors were calibrated at 38°C by introducing relative humidity standards obtained over saturated solutions of lithium chloride, sodium iodide, sodium chloride and potassium phosphate. Sweat rate was derived from changes in the relative humidity and temperature of the air entering and leaving each sweat capsule. Thus:

$$\dot{m}_{sw} = [(rh_{ex} * P_{H_2O_a} * \dot{m}/100 * T_{cap} * k) - (rh * P_{H_2O_a} * \dot{m}/100 * T_a * k)]/A,$$

where: $\dot{m}_{sw}$ = mass flow of water off the skin ($g \cdot cm^{-2} \cdot min^{-1}$), rh and rh_{ex} = relative humidity of air entering and leaving the capsule (%), $P_{H_2O_a}$ = partial pressure of water vapour of air entering the capsule, if 100% saturated (mmHg), $\dot{m}$ = airflow through the capsule rotameter ($l \cdot min^{-1}$), k = 3.464 (water vapour gas constant in $mmHg \cdot l \cdot g^{-1} \cdot K^{-1}$), T_a and T_{cap} = temperature of air entering and leaving the capsule (K), and A = area of skin under the capsule ($2.19\ cm^2$).

The median $\dot{m}_{sw}$ for the first 2 min of each HST was taken as the baseline (zero) $\dot{m}_{sw}$ for each site. Sweat thresholds were determined from raw data (sampled at 1 Hz), as points where $\dot{m}_{sw}$ first increased above the median $\dot{m}_{sw}$ without returning to baseline within the next 5 min. This was defined as continuous sweating. During the initial period of such sweating, which displays an approximately linear relationship with T_c change, forehead and forearm $\dot{m}_{sw}$ sensitivities (gains: $\Delta\dot{m}_{sw}/\Delta T_c$) were computed. Since sudomotor stimuli were held approximately constant between trials (T_a, duration, absolute exercise intensity), changes in sudomotor function were also evaluated by integrating $\dot{m}_{sw}$ with respect to time.

Core temperature was determined from auditory canal (aural) temperature (T_{ac}; zero-gradient thermometry: after Keatinge and Sloan 1975). Separate thermistors monitored T_{ac} and that of an insulated outer ear heating pad. A servo-heating unit maintained the ear pad at T_{ac}, isolating it from environmental artefacts, and removing the temperature gradient within the auditory canal (Moore and Newbower 1978). Under these conditions, T_{ac} tracks tympanic temperature, and is typically 0.21°C above oesophageal ($r = 0.940$), 0.19°C above tympanic ($r = 0.974$), and 0.21°C less than rectal temperatures ($r = 0.965$). Furthermore, the dynamic response of this zero-gradient T_{ac}, while minimally damped, closely tracks changes in oesophageal and tympanic temperatures (Cotter et al. 1995).

$\dot{Q}_{skin}$ was measured using laser-Doppler velocimetry (TSI Laserflo BPM^2, Vasamedics, USA: 780 nm wavelength). Since this technique relies upon the assumption that site-specific $\dot{Q}_{skin}$ represents segmental $\dot{Q}_{skin}$, we previously isolated discrete skin regions in which local $\dot{Q}_{skin}$ best reflected segmental $\dot{Q}_{skin}$ (Cotter et al. 1993). From 225 upper-body sites (head, chest, back, arm, forearm and hand), assessed under thermoneutral conditions, the following sites were selected: forehead (right temple), chest (fourth and fifth intercostal space, lateral to the sternum), back (upper lateral scapular border), arm (medial biceps, midway between the antecubital fossa and the axilla), and forearm (ventral surface, midpoint of the radius). In each case, the correlation with segmental $\dot{Q}_{skin}$ exceeded 0.80. Data were also collected from the stationary thigh (ventral surface, midpoint of the femur). Zero $\dot{Q}_{skin}$ was determined using an opaque, white surface to which all $\dot{Q}_{skin}$ measurements were referenced. During each HST, $\dot{Q}_{skin}$ was measured continuously from the forearm during the first 15 min of each phase of the HST. During the last 5 min of each phase, $\dot{Q}_{skin}$ was measured at each of the five other sites, for 20 s in each minute. Sites were clearly marked with circular adhesive rings, permitting the probe to be returned to each site with precision within each trial. Between trials, the probe markers were relocated after Cotter et al. (1993). Since this laser-Doppler technique measures $\dot{Q}_{skin}$ only within a small volume of tissue (radius 1.0–1.5 mm), and since it quantifies relative blood flow changes rather than absolute flows, data are reported as voltage outputs, and not in absolute blood flow units.

The analog outputs from the $\dot{Q}_{skin}$ monitor, sweat monitor and auditory canal thermometer were passed, via an analog-to-digital converter (PPIO-A18, Computer Boards, USA), to a mains-isolated laptop computer (Notebook 386SX, Total Peripherals,

Table 1 Characteristics of the subjects (f_{cpeak} peak cardiac frequency, $\dot{V}O_{2peak}$ peak aerobic power, *SD* standard deviation)

Subject	Age (years)	Height (cm)	Mass (kg)	Peak power (W)		f_{cpeak} ($beats \cdot min^{-1}$)		$\dot{V}O_{2peak}$ ($l \cdot min^{-1}$)	
				control	anaemia	control	anaemia	control	anaemia
S1	28	180	67.8	420	395	190	195	5.48	4.48
S2	22	171	76.8	375	330	185	203	4.88	4.38
S3	27	187	79.0	430	390	170	169	6.06	4.66
S4	27	169	72.2	490	430	178	183	6.13	5.45
S5	22	178	71.8	360	330	179	187	4.85	4.18
S6	23	185	83.2	436	410	191	199	6.06	4.96
Mean	24.8	178.3	75.1	418.5	380.8	182.2	189.3	5.58	4.69*
SD	(2.8)	(7.3)	(5.6)	(46.6)	(41.8)	(8.04)	(12.4)	(0.59)	(0.46)

* differences between control and mild anaemic states significant at the $P < 0.05$ level

Australia). Data were sampled at 1 Hz. Cardiac frequency (f_c) was monitored continuously from ventricular depolarisation at 0.2 Hz (Polar Electro Sport Tester, model PE3000, Finland) and downloaded to computer.

Skin temperatures were monitored using thermistors attached to eight sites with a single layer of waterproof tape (EU mini thermistors, Yellow Springs Instruments, USA): forehead, right scapula, left upper chest, right arm, left forearm, left hand, right anterior thigh, and left calf. Data were recorded at 0.2 Hz using a portable data logger (1206 Series Squirrel, Grant Instruments, UK), and used to derive mean skin temperature ($\bar{T}_{sk}$), using the ISO-9886 eight-site standard (International Organisation for Standardization 1992). All thermistors (including the auditory canal thermistor) were calibrated in a stirred water bath (Grant Instruments) against a certified reference thermometer (Dobros total immersion, Dobbie Instruments, Australia).

Subjective reports of thermal sensation and physical exertion were recorded at 5-min intervals. Familiarisation with each of the rating scales preceded HST1, with subjects receiving standardised written instructions and a single question for each index. Thermal sensations, registered throughout the HSTs, were rated using a scale from 1 (extremely cold) to 13 (extremely hot). Perceived exertion ratings (RPE) were obtained during exercise using the 15-point Borg Scale (Borg 1962), with ratings obtained for the whole body, legs and chest.

On arrival, subjects were asked to empty their bladder, and were then measured for mass (A&D, model no. fw-150k, USA). The auditory canal thermistor was inserted and allowed to equilibrate. A venous blood sample was drawn to evaluate their haematological status prior to each exposure (Coulter Counter, S-Plus IV, Coulter Electronics, USA). On leaving the chamber, subjects were towelled dry, and a final body mass was recorded.

This study was based on a 2 × 3 factorial design, with subjects fully crossed for factor one (venous haematocrit: control and mildly anaemic), and factor two (HST exercise intensity: rest, cycling at 30% and 45% $\dot{V}O_{2peak}$). Data were averaged over 5-min intervals (±20 s), and analysed using multivariate analysis of variance with Tukey's honestly significant difference post hoc analysis. Paired t-tests were also conducted on data collapsed across time, within each of the three 20-min phases of the HST, and on integrated sweat data. The level of significance was set at the $P < 0.05$ level. Data are reported as means with standard errors, unless otherwise stated.

Results

The three phlebotomies induced significant reductions in venous haematocrit, [Hb] and red cell count (Table 2; $P < 0.05$), but mean (SD) red cell volumes [85.0 (1.7) versus 85.0 (2.0) fl: control versus anaemia], mean red cell [Hb] [354.8 (6.5) versus 350.0 (5.6) $g \cdot l^{-1}$], and white cell counts [5.0 (1.0) versus 5.8 (2.8) $\times 10^9 \cdot l^{-1}$; $P > 0.05$] were unaffected by the manipulation. Subjects did not attain the haematological status associated with clinical anaemia. Notwithstanding, these changes resulted in an average reduction in $\dot{V}O_{2peak}$ of 15.9% (Table 1; $P < 0.05$), and, when averaged across work loads, c_aO_2 was reduced from 184.7 $ml \cdot l^{-1}$ (HST1) to 160.8 $ml \cdot l^{-1}$ (HST2; $P < 0.05$), verifying that the intervention had significantly altered arterial oxygen transport.

Subjects commenced HST2 with a significantly greater pre-exposure T_{ac} (time = 0, Fig. 1a; $P < 0.05$), but at no point beyond time zero were the between-condition T_{ac} differences significant ($P > 0.05$). When averaged within HSTs, T_{ac} was 37.5°C [(0.064): HST1] and 37.5°C [(0.065): HST2; $P > 0.05$]. One subject (S2) terminated the anaemic trial at 55 min due to fatigue with his f_c reaching 90% f_{cpeak}. Subjects completed HSTs with a final T_{ac} of 38.5°C [(0.16); HST1] and 38.6°C [(0.13); HST2; $P > 0.05$], at respective termination times of 60.0 min and 58.5 min [(2.04); SD]. f_c did not differ between HSTs at rest ($P > 0.05$), but was significantly elevated in the anaemic state during both exercise phases, averaging 6 beats $\cdot$ min^{-1} greater than observed during HST1 (Fig. 1b; $P < 0.05$). A recalibration of the climate chamber thermistor inadvertently resulted in the T_a averaging 0.9°C higher during the anaemic trials: 39.1°C versus 38.2°C. Consequently, $\bar{T}_{sk}$ was higher during HST2 ($P < 0.05$), averaging 36.5°C [(0.23): HST2] and 37.3°C [(0.02): HST2]. Since the changes in $\bar{T}_{sk}$ and T_a were about equal, then convective and conductive heat flows would have been approximately equal between HSTs, while radiative gains may have been elevated fractionally in HST2. It was considered that this technical error, while perhaps elevating thermal strain in HST2, had minimal physiological influence.

Forearm $\dot{Q}_{skin}$ was monitored continuously throughout the HSTs (Fig. 2a), except for the last 5 min of each 20-min phase, where data from the forehead, chest, back, arm and thigh were recorded (Fig. 2b). In the anaemic state, the resting forearm $\dot{Q}_{skin}$ exceeded that

Table 2 Summary of haematological changes following the withdrawal of 3 units (1350 ml) of whole blood. (*RBC* Red blood cell count, *[Hb]* haemoglobin concentration, *Hct*, mixed-venous haematocrit)

Subject	RBC $\times 10^{12} \cdot l^{-1}$		[Hb] $(g \cdot l^{-1})$		$Hct_{\bar{v}}$ (%)	
	control	anaemia	control	anaemia	control	anaemia
S1	4.79	4.18	148	127	40.9	36.5
S2	4.78	4.33	146	134	41.7	37.8
S3	4.55	3.84	132	112	38.1	32.1
S4	4.35	4.13	131	124	37.4	36.1
S5	4.78	4.36	142	130	39.4	36.2
S6	4.98	4.07	153	123	42.5	35.3
Mean	4.71*	4.33	142.0*	125.0	40.0*	35.7
SD	(0.22)	(0.19)	(8.9)	(9.4)	(2.0)	(1.9)

* Difference between control and mild anaemic states significant at the $P < 0.05$ level

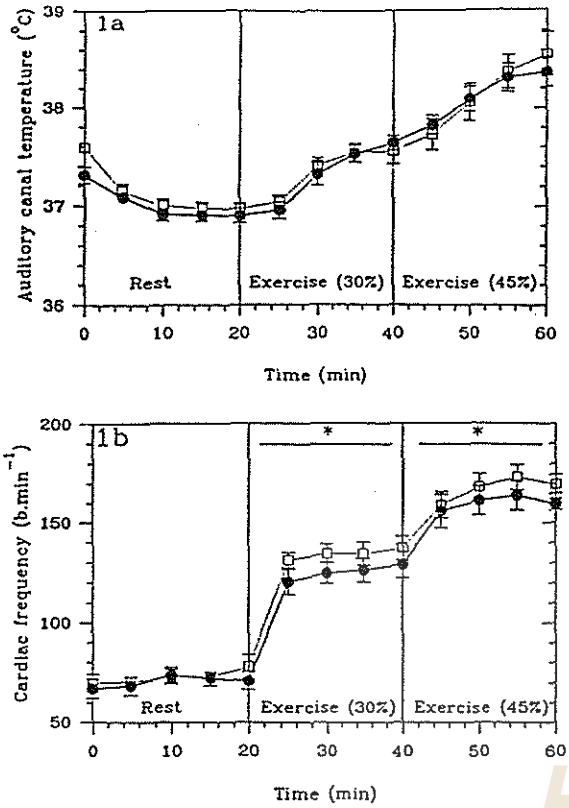


Fig. 1 Auditory canal temperature (a) and cardiac frequency (b) during a three-phase exercise and heat stress test at an ambient temperature of 38.6°C (water vapour pressure 2.74 kPa), with subjects in both control (●) and artificially induced mild anaemic states (□). Data are means with standard errors of the means. An *asterisk* indicates that differences between the control and anaemic states were significant across the corresponding test phase

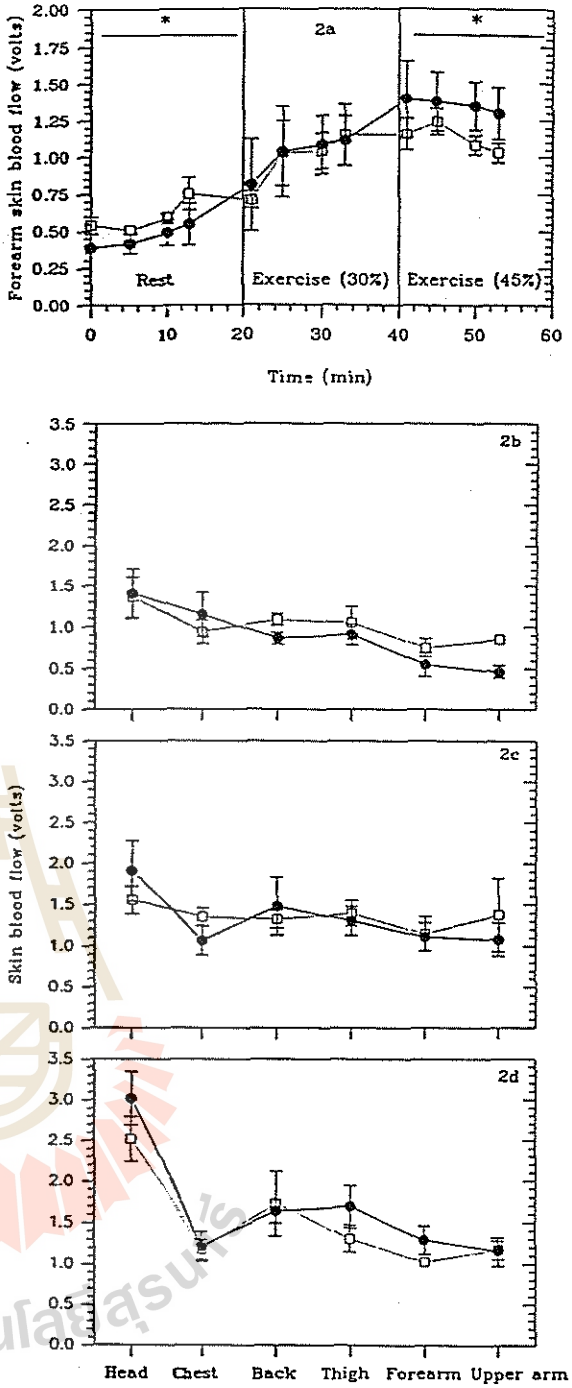
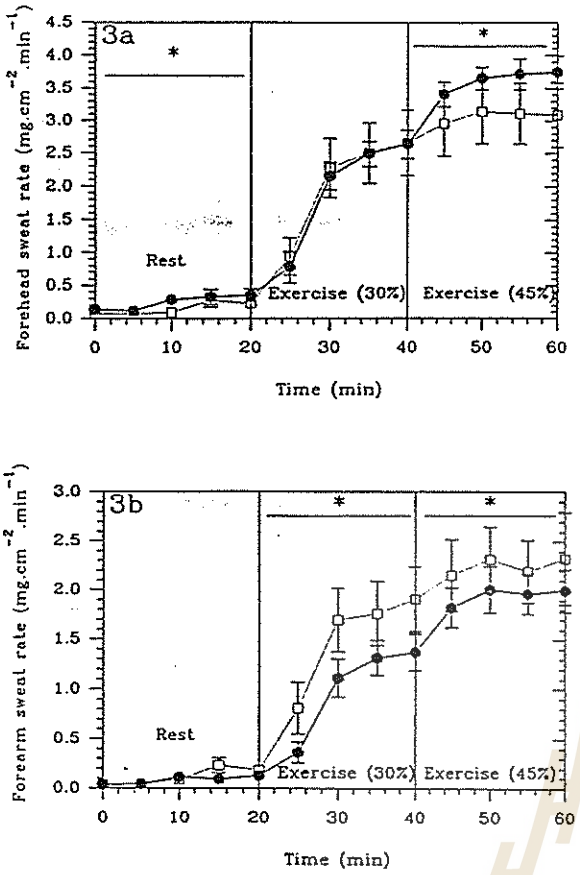


Fig. 2 Skin blood flow, measured at the forearm (a) during the first 15 min of each of three phases of a combined exercise and heat stress test (38.6°C; water vapour pressure 2.74 kPa), and at six sites during the last 5 min of each test phase (b, c and d, respectively). At these times, forearm data were taken from the last 10-s period of each exercise phase, immediately before measures were recorded at the other five sites: control (●); mildly anaemic (□). Data are means with standard errors of the means. An *asterisk* indicates that differences between the control and anaemic states were significant across the corresponding test phase

observed for HST1 ($P < 0.05$). However, during exercise, forearm $\dot{Q}_{skin}$ were equivalent during the first exercise phase (Fig. 2a; $P > 0.05$), but were lower in HST2 for the last 20-min period. In both HSTs, the rise in forearm $\dot{Q}_{skin}$ over time, was attenuated in both HSTs, with this attenuation being more pronounced in HST2 ($P < 0.05$). The forehead displayed a consistently greater $\dot{Q}_{skin}$ than the other sites, with the same general $\dot{Q}_{skin}$ distribution between regions being observed across both time and experimental conditions. All local sites exhibited a progressive elevation in $\dot{Q}_{skin}$ with time in both conditions (Fig. 2b). This was most apparent at the forehead, with $\dot{Q}_{skin}$ increasing twofold in the control state, but only by a factor of 1.8 during mild anaemia.

Total body $\dot{m}_{sw}$ did not vary between trials: mass losses were 0.91 kg [(0.12); HST1] and 0.92 kg [(0.12); HST2; $P > 0.05$]. During HST1, there were no differences between the forehead and forearm sweat onset times or T_{ac} thresholds ($P > 0.05$), but the forehead $\dot{m}_{sw}$ sensitivity exceeded that of the forearm ($P < 0.05$). In HST2, forehead sweat onset times, T_{ac} thresholds, initial $\dot{m}_{sw}$ sensitivities and $\dot{m}_{sw}$ matched that observed within the control condition during rest and the first exercise stage ($P > 0.05$; Table 3 and Fig. 3a). However, a marked suppression of forehead $\dot{m}_{sw}$, relative to HST1, occurred across the second exercise period, after subjects were rendered mildly anaemic ($P < 0.05$). Conversely,



ig. 3 Forehead (a) and forearm sweat rates (b) during a three-phase ercise and heat stress test at an ambient temperature of 38.6°C vater vapour pressure 2.74 kPa), with subjects in both the control ●) and artificially induced, mildly anaemic conditions (□). Data are eans with standard errors of the means. An *asterisk* indicates that ifferences between the control and anaemic states were significant ross the corresponding test phase

forearm $\dot{m}_{sw}$ during HST2 exceeded control $\dot{m}_{sw}$ during both exercise stages ($P < 0.05$; Fig. 3b). The forearm $\dot{m}_{sw}$ sensitivity increased by 24% after anaemia was induced (Table 3; $P < 0.05$), while sweat onset times and T_{ac} thresholds remained stable between trials (Table 3; $P > 0.05$). Consequently, when analysed across the total HST, the average forearm $\dot{m}_{sw}$ was significantly higher during HST2: 1.21 (0.20) versus 0.97 (0.12) mg · cm^{-2} · min^{-1} ($P < 0.05$). Furthermore, the integrated forearm $\dot{m}_{sw}$ was significantly greater in HST2 than in HST1 (91.6 versus 70.8 mg · cm^{-2}; $P < 0.05$), while forehead $\dot{m}_{sw}$ revealed a non-significant reduction (124.5 versus 135.2 mg · cm^{-2}; $P > 0.05$).

Mild anaemia resulted in significantly greater effort sense (whole-body RPE) within both the exercise periods ($P < 0.05$). This trend was similarly reflected in the fractionated RPE, with both the chest and leg RPE being significantly greater during HST2 ($P < 0.05$). However, thermal sensation was only modestly influenced by blood removal, such that during the second exercise phase, subjects reported a greater thermal sensation when rendered mildly anaemic ($P < 0.05$).

Discussion

Acute mild anaemia produced significant reductions in aerobic power, yet it did not result in greater thermal strain, relative to the control state, during combined exercise and heat stress. Subjects did, however, experience a relatively greater attenuation of the $\dot{Q}_{skin}$ response accompanying an elevation in T_c, and an apparent redistribution of sweat output.

Implicit within this study is the assumption that blood volume had returned to control levels before

able 3 Forearm and forehead sweat onset times, auditory canal emperature thresholds, and response sensitivities during a three-hase exercise and heat stress test at an ambient temperature of 38.6°C (water vapour pressure 2.74 kPa), with subjects in both control and artificially induced, mild anaemic states. (*Subj* Subject identification number, *SEM* standard error of the mean)

Condition	Subj	Onset time arm (s)	Threshold: arm (°C)	Sensitivity: arm (mg·cm^{-1}·min^{-1}·°C^{-1})	Onset time head (s)	Threshold: head (°C)	Sensitivity: head (mg·cm^{-1}·min^{-1}·°C^{-1})
Control	S1	1476	37.0	3.31	1486	37.0	4.74
	S2	738	37.1	0.99	296	37.1	2.22
	S3	1461	36.6	0.87	1459	36.7	2.05
	S4	1223	36.8	1.97	1245	36.8	3.12
	S5	517	37.0	1.85	512	37.0	3.07
	S6	545	37.0	2.38	535	37.0	4.37
	Mean	993	36.9	*,**1.90	922	36.9	**3.26
	SEM	(182)	(0.1)	(0.37)	(218)	(0.1)	(0.45)
Anaemia	S1	1313	37.1	4.26	1376	37.1	4.77
	S2	643	37.2	1.97	640	37.1	6.24
	S3	1492	37.0	1.19	1536	37.1	1.12
	S4	1189	36.9	2.73	1334	36.9	4.90
	S5	600	37.1	2.67	604	37.1	4.11
	S6	352	36.8	2.16	348	36.7	2.84
	Mean	932	37.0	*2.50	973	37.0	4.00
	SEM	(188)	(0.1)	(0.42)	(204)	(0.1)	(0.73)

* Difference between control and mild anaemic states significant at the $P < 0.05$ level
** Difference between forearm and forehead significant at the $P < 0.05$ level

HST2, and that the current results reflected the effects of acute anaemia and not hypovolaemia. While blood volume measures were not taken within this investigation, there is ample evidence to support the assumption that hypovolaemia was indeed transient. In normal healthy subjects, acute hypovolaemia is rapidly countered via atrial stretch receptors, baroreceptors and osmoreceptors (Gauer et al. 1970). Furthermore, following similar experimental perturbations, Celsing et al. (1986) and Ekblom et al. (1976) have verified a rapid return to isovolaemia. Ekblom et al. (1976) withdrew 800 ml of whole blood, observing that blood volume returned to control levels 2 days after phlebotomy. In the present study, 450 ml of whole blood was removed at each phlebotomy, with withdrawals at 24-h intervals. HST2 was then delayed 3–5 days after the third phlebotomy. While hypovolaemia cannot be totally eliminated, it was considered that the present subjects were in a state of isovolaemic mild anaemia.

At no point during the HSTs were there between-condition T_{ac} differences (Fig. 1a), indicating that thermal strain was not elevated in the anaemic state. This result was not anticipated. Blood removal produced a 16% reduction in $\dot{V}O_{2peak}$ (Table 1), resulting in anaemic exercise, conducted at a constant absolute work rate, being performed at significantly greater relative exercise intensities. On a simple first principles basis, one would predict that equal work loads would result in approximately equal heat production, and approximately equal elevations in T_{ac} between trials on the same sample. However, it has been shown that the rise in T_c is closely coupled to the relative exercise intensity (Saltin and Hermansen 1966). Considering these points, it was anticipated that T_{ac} would rise faster during HST2.

An uncoupling of the relationship between T_c and exercise intensity has also been reported by Greenleaf et al. (1969) and Rowell et al. (1982). These groups artificially lowered $\dot{V}O_{2peak}$ (11–32% and 27%, respectively), then exercised subjects at the same absolute work load. The rectal temperature response to exercise was equivalent to that in the control state. Thus, it appears that the relationship between T_c and relative work load is not as tightly coupled as the data of Saltin and Hermansen (1966) suggest, existing primarily to explain exercise-related differences in T_c between different subject groups. The exercise-induced changes to heat loss mechanisms within-subjects, that accompany reductions in arterial oxygen carriage, such as changes in blood flow distribution, may act to uncouple this relationship by altering the mechanisms, and the effectiveness of the mechanisms through which thermal homeostasis is achieved.

A major means for moving heat from the core to the periphery is via an elevation in $\dot{Q}_{skin}$. During the resting phase, forearm $\dot{Q}_{skin}$ was significantly elevated in the anaemic state (Fig. 2a). This may simply be attributed to the influence of a higher pre-exposure T_{ac} (time 0: Fig. 1a). However, a significantly lower forearm $\dot{Q}_{skin}$ was observed during the last 20 min of anaemic exercise (Fig. 2a). As hypothesised, it appears that the attenuation of the cutaneous vasodilatory response of the forearm to a rising T_c (Brenglemann et al. 1977; Patterson et al. 1994) becomes more exaggerated in anaemic subjects. This trend could reflect a relatively greater work rate in the anaemic trials, although Patterson et al. (1994) have shown that $\dot{Q}_{skin}$ attenuation is independent of exercise intensity. Instead, it is suggested that the greater $\dot{Q}_{skin}$ attenuation observed in the anaemic state may be due to an elevation in the demand for blood by the active muscles, resulting in a baroreceptor-mediated suppression of active cutaneous vasodilation (Kenney et al. 1994). The stimulus for such a change would most probably have been a reduced oxygen carrying capacity of arterial blood following blood withdrawal.

Consistent with these forearm $\dot{Q}_{skin}$ observations are the changes in the $\dot{Q}_{skin}$ of the thigh (Fig. 2b–d). During HST2, thigh $\dot{Q}_{skin}$ was 15% greater at rest, 7.7% greater during the first exercise period, and 22.5% less during the final 20 min of exercise than observed in HST1. Similar $\dot{Q}_{skin}$ attenuation patterns were observed for the forehead, with respective reductions for the two exercise stages being 18.4% and 16.6%, but not for the chest, back and arm. However, such attenuations in $\dot{Q}_{skin}$ did not impair the ability of the subjects to regulate T_{ac}, and subjects appeared to tolerate the HST under both conditions equally well, perhaps by a greater reliance upon evaporative heat loss. Since body mass changes at the conclusion of the HSTs were equivalent, total body $\dot{m}_{sw}$ was similar between trials. Consequently, subjects could only modify evaporative heat losses via a redistribution of sweat between the body surfaces, resulting in a more uniform skin wetness. Accordingly, the forehead exhibited a lower $\dot{m}_{sw}$, while forearm $\dot{m}_{sw}$ was elevated in HST2 (Fig. 3a, b). While these observations are consistent with sweat redistribution, they are not conclusive since $\dot{m}_{sw}$ was recorded at only two sites.

The decline in forehead $\dot{m}_{sw}$ that was observed during the anaemic trials was accompanied by a continued rise in T_{ac}. This response was not attributed to either the attainment of peak $\dot{m}_{sw}$, or to a local suppression of sweat secretion (hidromeiosis), since a greater sweat secretion was achieved in HST1. Since T_{ac}, and the sweat thresholds and sensitivities were equivalent between conditions, the lower forehead $\dot{m}_{sw}$ may not be ascribed to differences in central sudomotor drive, or glandular responses to that drive. A lower skin temperature is known to suppress local sweating (Benzinger 1970). However, $\bar{T}_{sk}$ was actually higher in the anaemic trials. The lower forehead sweat production could be associated with a reduction in forehead $\dot{Q}_{skin}$, which was consistently lower during the anaemic trials (Fig. 2c, d). Alternatively, there may have been a reduced glandular secretion to the central drive during the final 20 min of exercise in HST2. Finally, the possibility exists that there is an active redistribution of sweat to more distal regions. While our experimental design does not permit a thorough evaluation of such a hypothesis, the simultaneous elevation in forearm $\dot{m}_{sw}$ is consistent with this

explanation. This implies a reduced central sudomotor drive to the forehead, with a concomitant rise in sudomotor drive to the forearm. Such a mechanism presupposes a separate and independent regulation of sudomotor function at these sites. However, we are unaware of any direct evidence for such independent regulation.

Conclusions

Rendering endurance-trained males mildly anaemic reduced aerobic power, but did not result in an elevation in thermal strain during exercise in the heat. While the exercise intensities were relatively low, in combination with the environmental conditions, our subjects found these conditions to be very stressful. However, it remains uncertain to what extent anaemia may have influenced thermal tolerance under a more protracted and more intense exercise exposure. Subjects experienced a greater attenuation of their $\dot{Q}_{skin}$ response to a rising T_{ac}, reflecting a greater muscle blood flow, and resulting in the redistribution of blood flow away from the skin, which, during more extended exercise, would possibly result in greater heat storage.

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Human sudomotor responses to heating and cooling upper-body skin surfaces: cutaneous thermal sensitivity

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ABSTRACT

The influence of local skin temperature (T_{skl}) on the control of local and whole-body sweating was evaluated in eight healthy males. A water-perfusion garment (37 °C) and a climatic chamber (36.45 ± 0.78 °C; [±SD]; relative humidity 60.3 ± 1.6%) were used to raise and clamp skin and core temperatures. Warm and cool stimuli were applied to four upper-body skin regions (face, arm, forearm, hand) using perfusion patches (249.0 ± 0.2 cm^2). Heating elevated, while cooling suppressed sweat rate ($\dot{m}_{sw}$) locally, and at other skin surfaces. However, the tendency for T_{skl} manipulations to induce localized sweat responses was no more powerful than it was at stimulating sweating in non-treated regions ($P > 0.05$). Accordingly, neither thermal stimulus produced significantly greater local sudomotor influences than were elicited contralaterally ($P > 0.05$). No statistical support was found for the notion of inter-regional differences in upper-body cutaneous thermal sensitivity for sudomotor control, and, regardless of the stimulation site, whole-body sudomotor responses to localized thermal treatments were equivalent ($P > 0.05$).

Keywords body temperature regulation, central sudomotor drive, skin temperature, sweating, thermal sensitivity.

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Terrestrial endotherms have evolved behavioural and autonomic mechanisms to regulate body-core temperature (T_c). Stimuli eliciting autonomic responses emanate from the body core (Hammel 1968, Hellon 1983, Jessen *et al.* 1990) and from cutaneous thermoreceptors (Crawshaw *et al.* 1990). The relative importance of these central and peripheral inputs has generally been identified. Proppe *et al.* (1976) demonstrated that an independent elevation of T_c in baboons produced a 10-fold greater elevation in right iliac blood flow, than did an independent elevation in skin temperature. In humans, a similar central:peripheral thermal sensitivity ratio appears to dictate the control of skin blood flow (Wenger *et al.* 1975) and sweat rate ($\dot{m}_{sw}$, Nadel *et al.* 1971). However, our understanding of the relative sensitivities of specific thermosensitive sites within either the core or cutaneous regions in humans, is less well defined. For example, relatively little is known concerning the thermal sensitivities of various skin regions, and many investigators consider the skin as a single source of afferent input. However, inter-regional differences in cutaneous thermosensitivity have been identified in some animal species (Hales & Hutchinson 1971, Necker 1977), and, to some extent, in humans (Nadel *et al.* 1973, Werner & Heising 1990). Nevertheless, the relative contributions made by thermosensitive skin sites to the control of human sudomotor function is still relatively undefined. Accordingly, we investigated the differential effects of heating and cooling discrete, upper-body skin regions on the control of human eccrine sweating.

When ambient conditions change, the skin thermoreceptors provide the first thermoregulatory input, giving rise to thermal sensations (Hammel 1968). As the distribution of thermal spots is not uniform (Hardy & Opel 1938), and thermoreceptor firing rates vary across a range of skin temperatures (Hensel 1981), inter-regional differences in the contributions of skin receptors to thermoregulation might be expected. Accordingly, thermal sensation to locally applied stimuli has been shown to be non-uniform (Stevens *et al.* 1970, Crawshaw *et al.* 1975). Furthermore, local skin temperature (T_{skl}) changes may independently modify sudomotor and vasomotor responses (Nadel *et al.* 1973,

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Taylor *et al.* 1984, Bothorel *et al.* 1991), both locally and within other skin regions, even when these other regions are at an elevated temperature (McCaffrey *et al.* 1979). However, evidence for the impact of T_{skl} on autonomic responses is not unequivocal (Benzinger 1970, Wyss *et al.* 1974), and a case exists to investigate possible inter-regional differences in cutaneous thermosensitivity.

Two groups have previously investigated inter-regional differences in the role of T_{skl} in the control of sweating. The earliest work came from an American group (Nadel *et al.* 1973, Crawshaw *et al.* 1975). Nadel *et al.* (1973) irradiated various skin surfaces (except the thigh; $n = 2$), and evaluated thermal sensitivity according to the extent to which these regions elicited sweat responses at the thigh. Localized heating initiated sweating, with the face reportedly showing the greatest thermal sensitivity. Crawshaw *et al.* (1975) applied a water-cooled thermode to five body surfaces, studying the inhibitory effect of local cooling on sweating in the untreated thigh ($n = 3$). Forehead cooling was reported to produce the greatest inhibition of sweating. As these groups rapidly applied thermal stimuli, both sets of observations are limited to the role of T_{skl} transients on sweat regulation. Moreover, as the treated surface areas and the changes in T_{skl} were not equal between treatment sites, and since neither T_c nor mean skin temperature ($\bar{T}_{sk}$) was clamped (controlled), these data should be viewed with some circumspection.

The second (French) group used sealed limb chambers and a climate chamber to independently control steady-state T_{skl} of the arms, legs and the head–torso (Libert *et al.* 1984; $n = 5$). Differences in regional thermosensitivity were gauged from right arm sweating. While it remains uncertain to what extent the miniature chambers affected the microclimate, T_{skl} changes at the head–torso showed the greatest influence upon sweating, highlighting the significance of T_{skl} on thermoregulation. However, the value of the inter-regional comparisons of thermal sensitivity was somewhat restricted, as the areas of treated skin were unequal, and the T_c, $\bar{T}_{sk}$ and the temperatures of the non-treated skin surfaces were not controlled.

In the present investigation, we raised and clamped both core and skin temperatures, then evaluated cutaneous thermal sensitivity by equivalently manipulating the T_{skl} of four upper-body skin surfaces, each of the same surface area (≈250 cm^2). We used this unique circumstance to assess the thermal sensitivities of four upper-body skin regions, independently of either T_c and $\bar{T}_{sk}$ influences, and to test the hypothesis that the stimulation of some upper-body sites would evoke more pronounced sudomotor effects than others, with an apparent hierarchical thermal sensitivity between treated skin regions.

METHODS

Eight physically x active males (age 25.6 ± 7.1 years, mass 74.6 ± 8.1 kg, height 178.6 ± 6.2 cm, mean ± SD) participated in this project, which involved a 6-day heat acclimatization period, followed by 1 day of testing, during which the thermal sensitivity of four upper-body skin regions was evaluated. All procedures were approved by the Human Research Ethics Committee (University of Wollongong), and subjects provided informed consent.

Pre-experimental heat acclimatization was undertaken to optimize sudomotor responsiveness, and to minimise possible between-subject $\dot{m}_{sw}$ differences associated with varying heat acclimatization and physical training status. The acclimatization regimen involved cycling in an air temperature (T_a) of 39.5 °C (±0.2) and 59.2% (±0.8) relative humidity for 6 consecutive days. The initial work rate (194.2 ± 26.7 W) was set to rapidly elevate the auditory canal temperature (T_{ac}) by 1.4 °C (21.8 ± 7.2 min). Thereafter, the work rate was adjusted to hold T_{ac} constant for a further 70 min (after Regan *et al.* 1996), producing a total heat exposure of 91.8 min (±7.2). During days 2–5, fluid replacement was enforced at regular intervals, to a total of 1200 mL. Following sessions one and six, which were used to evaluate the affects of short-term heat acclimatization (Cotter *et al.* 1997), subjects consumed 75% of mass loss before leaving the laboratory.

One to two days after heat-acclimatization, thermal sensitivity experiments commenced, with all trials occurring at the same time of the day. Subjects were studied resting (supine) on a wide-mesh, wire bed, at an T_a of 36.5 °C (±0.8; relative humidity 60.3% (±1.6)). The ambient conditions, combined with the use of a whole-body water-perfusion suit (water temperature 37 °C; Paul Webb Associates, Yellow Springs, USA), were used to elevate and then to clamp T_c and $\bar{T}_{sk}$. This method of controlling skin temperature was chosen as it enabled the exposure of maximal skin surface area to the ambient conditions, while minimizing the establishment of local microclimates at the skin surface. This combination of air and water temperatures was used to ensure that both the T_c and $\bar{T}_{sk}$ were elevated above the sweat threshold, resulting in sweat secretion at each of eight recording sites. While we sought to ensure that sweating was clearly established, thereby allowing local cooling to have an inhibitory effect, it was also important that the basal $\dot{m}_{sw}$ was not so high that heating failed to elicit further increments in $\dot{m}_{sw}$. Finally, the air and perfusion-suit water temperatures were selected to establish a uniform skin temperature distribution across the skin surface. We have recently shown this method to be capable of clamping, and sustaining a stable thermal load for up to 3 h, as reflected by T_c, $\bar{T}_{sk}$ and

the mean body temperature stability (Cotter *et al.* 1995). In the current project, such clamping resulted in an average T_c (oesophageal, auditory canal, rectal) of 36.9 °C (±0.1) and $\bar{T}_{sk}$ of 36.2 °C (±0.1), which were maintained over the 3-h experimental period.

The water-perfusion garment consisted of 140 meters of tubing (ID = 1.58 mm, OD = 3.0 mm) which covered the body in separate jacket and trousers sections. The jacket consisted of 60 1-m tubes covering the torso, and 15 1-m tubes for each arm, while the trousers had 25 1-m tubes for each leg. Each tube was set in parallel to ensure uniform flow. This was verified using video filming during coloured-water perfusion. Every 4 cm, the tubes were clipped alternately to adjacent tubes, resulting in a diamond-shaped skin coverage (8 cm × 2 cm). To ensure testing was performed with minimal influence from extraneous factors, subjects were asked to avoid exercise 24 h prior to thermal sensitivity trials, other than the final heat acclimatization trial. Subjects also abstained from alcohol 24 h prior, caffeinated drinks 4 h prior, and food 2 h prior to trials. To negate possible residual dehydration influences from the previous 6 days, subjects followed an enforced drinking schedule, consuming 1 L of water before retiring on the night before testing, and another litre 2 h prior to testing.

Independent warm and cool stimuli were applied to discrete skin regions (face, right arm, right forearm, right hand), using perfusion patches with water controlled at pre-determined temperatures. These patches were positioned in contact with the skin, beneath the main suit, and consisted of tubes (ID = 1.58 mm, OD = 3.0 mm) running in parallel, 8–12 mm apart. The face patch varied from this arrangement, and was made up of two sections: forehead and cheek/chin sections. The latter was positioned under the chin and on each cheek. The arm and forearm patches were positioned on the dorsal skin surfaces. The hand patch was placed on the dorsal surface of the hand, and the ventral and dorsal surfaces of the fingers. Patches had a mean effective stimulation area of 249.0 cm^2 (±0.2).

Three 38-L water baths (Types VFP and ZD, Grant Instruments, UK) supplied water for the perfusion garment and patches. One bath was used to supply the whole suit, and those patches not being used to manipulate T_{skl}. The second and third baths were used to manipulate T_{skl}, and incorporated refrigeration units (Type CK2, Grant Refrigeration Systems, Grant Instruments, UK) to control water temperature. The temperature of baths two and three varied between treatment regions, and was determined by the water temperature required to apply an equivalent skin temperature stimulus to each site: approximately ±3 °C.

Following subject preparation and the attainment of thermal stability, the thermal sensitivity trials commenced. Each trial started with a 10-min baseline period, during which the perfusion suit and all patches were supplied with water at 37 °C. Every 20 min thereafter, a local (single patch) temperature manipulation began. These manipulations consisted of a 7-min treatment (heating or cooling), with a 13-min control period separating regional treatments. At the end of a manipulation, the target patch was briefly (60 s) flushed with warm water (after a cooling trial) or cool water (after a heating trial), so that the time taken for the T_{skl} to return to control levels was reduced. During the 13-min control period, 37 °C water perfused the patch, allowing T_{skl} to return to its pre-treatment temperature. With this protocol, subjects experienced eight 20-min test phases during a thermal sensitivity experiment, with a total trial duration of 176.4 min (±3.4). The first two T_{skl} manipulations were heating treatments, and the subsequent two manipulations were cooling of these two sites. With this exception, both the order of regions treated and the treatment temperatures, were balanced between subjects.

Sweat rate was measured continuously at eight sites (1 Hz), using two four-channel sweat systems (capacitance hygrometry; Sweat Monitor, Clinical Engineering Solutions, Australia: Turner & Gass 1993). Air was pumped through a sealed flask, containing a saturated salt solution (lithium chloride: 0.2 L min^{-1}), through eight sweat capsules (3.16 ± 0.05 cm^2), and then over humidity and temperature sensors. The sweat rate was derived from changes in the relative humidity and temperature of the air (Taylor *et al.* 1997). Sweat capsules were positioned at each of the manipulated skin regions (forehead, right arm, right forearm, dorsal right hand), at the corresponding regions on the contralateral arm, and on the left, mid-anterior leg. The left leg was used as a sudomotor reference site, as it was a lower body location and was not on the same side of the body receiving the thermal stimuli. Local, adjacent, contralateral and ipsilateral sudomotor affects could all be evaluated with this configuration of perfusion patches and sweat capsules.

Three indices of T_c were used to obtain a simple mean core temperature ($\bar{T}_c$) index: oesophageal temperature (T_{es}), auditory canal temperature (T_{ac}) and rectal temperature (T_{re}). An oesophageal thermistor (Edale Instruments Ltd, UK) was inserted transnasally (after Mekjavic & Rempel 1990). Auditory canal temperature was measured using an ear-moulded plug and thermistor (Edale Instruments Ltd, UK). Oesophageal and auditory canal temperatures were sampled at 0.2 Hz (Squirrel data logger, 1200 Series, Grant Instruments, UK). Rectal temperature was measured using a thermistor (YSI probe 401, Yellow Springs Instrument Company, USA) inserted 12 cm beyond the anal sphincter, and recorded from a tele-thermometer

(YSI model 46, Yellow Springs Instrument Company, USA), prior to, and at the conclusion of each T_{skl} manipulation.

Skin temperatures were measured adjacent to each sweat capsule (YSI probe 409B, Yellow Springs Instrument Company, USA), and also from the right upper chest, right upper scapular, right mid-anterior thigh, right medial-anterior calf and right dorsal aspect of the foot. Eight of these sites were used to obtain an eight-site $\bar{T}_{sk}$ (International Organization for Standardization, 1992). The $\bar{T}_{sk}$ was calculated by incorporating only the non-manipulated skin regions, thus providing an assessment of the thermal load applied to the remainder of the skin. That is, when the right forearm was treated, the left forearm T_{sk} was used in the derivation of $\bar{T}_{sk}$. When the face was treated, this region was omitted from the $\bar{T}_{sk}$ computation. Mean body temperature ($\bar{T}_b$) was taken as: $0.8\bar{T}_c + 0.2\bar{T}_{sk}$. Five thermistors were also used to track the change in temperature of the patch–skin interface (YSI Type EU, Yellow Springs Instrument Company, USA), at each site of T_{skl} manipulation. Two thermistors were positioned near the water inlet, two near the water outlet, and the fifth was positioned in the middle of the patch. All skin temperatures were recorded at 0.2 Hz using a data logger (Squirrel data logger, 1200 Series, Grant Instruments, UK), and subsequently downloaded to a computer. Cardiac frequency (f_c) was obtained from ventricular depolarization, recorded at 0.2 Hz (PE4000, Polar Electro SportTester, Finland), and similarly downloaded.

This study was based upon a repeated-measures factorial design, with four levels of factor one (skin region manipulated: face, arm, forearm, hand) and two levels of factor two (local thermal stimulation: heating and cooling). Subjects were fully crossed for both factors, with data analysed using Multivariate Analysis of Variance (MANOVA) and paired *t*-tests (methodological assessments), and repeated-measures MANOVA with contrast analyses (for comparisons of sudomotor responses across treatments). Following a significant overall *F*, Tukey's honestly significant difference *post-hoc* analysis (corrected for the number of repeated measures contrasts) was used to isolate sources of significant differences, resulting from the corresponding univariate analyses, within a thermal treatment. *Alpha* was set at the 0.05 level for all analyses.

RESULTS

The plateau (steady state) T_{skl} at each of the four experimental sites was significantly elevated during heating (3.04 ± 0.07 °C) and reduced during cooling (3.02 ± 0.10 °C), relative to the control, pre-manipulation period ($P < 0.05$). When averaged across the entire 7-min treatment period, including the transient T_{skl} changes, the mean thermal stimulus was 2.36 °C (±0.07, heating) and –2.12 °C (±0.10, cooling; $P < 0.05$; Table 1), with the

Table 1 Body temperatures in supine males prior to (control), and during local skin temperature manipulations (treatment).

		Control				Treatment			
Manipulation	Site	T_{skl}	$\bar{T}_{sk}$	$\bar{T}_c$	$\bar{T}_b$	T_{skl}	$\bar{T}_{sk}$	$\bar{T}_c$	$\bar{T}_b$
Heating	Face	36.70	36.20	36.85	36.75	39.05†	36.20	36.90	36.80
		(0.05)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)
	Arm	36.80	36.15	36.85	36.70	38.95†	36.20	36.90	36.75
		(0.05)	(0.15)	(0.05)	(0.05)	(0.10)	(0.15)	(0.05)	(0.05)
	Forearm	36.80	36.30	36.90	36.80	39.25†	36.30	36.90	36.80
		(0.20)	(0.15)	(0.05)	(0.10)	(0.10)	(0.10)	(0.05)	(0.10)
	Hand	36.65	36.20	36.85	36.75	39.10†	36.30	36.90	36.75
		(0.10)	(0.10)	(0.05)	(0.05)	(0.10)	(0.10)	(0.05)	(0.05)
Cooling	Face	36.70	36.20	36.90	36.80	34.40†	36.20	36.95	36.75
		(0.05)	(0.10)	(0.05)	(0.10)	(0.15)	(0.15)	(0.10)	(0.10)
	Arm	36.75	36.30	36.95	36.85	34.85†	36.25	36.95	36.80
		(0.05)	(0.05)	(0.05)	(0.05)	(0.15)	(0.10)	(0.05)	(0.05)
	Forearm	36.85	36.25	36.95	36.80	34.60†	36.25	36.95	36.80
		(0.15)	(0.10)	(0.05)	(0.05)	(0.15)	(0.10)	(0.05)	(0.10)
	Hand	36.65	36.25	36.90	36.80	36.65†	36.25	36.95	36.80
		(0.10)	(0.10)	(0.05)	(0.05)	(0.20)	(0.10)	(0.05)	(0.05)

Control values are means of the 4-min period prior to temperature manipulation. Treatment values are means of the 7-min temperature manipulation period. Values are means with standard errors of the mean in parenthesis. Abbreviations: T_{skl} = local skin temperature; $\bar{T}_c$ = the simple mean of oesophageal, auditory canal and rectal temperatures; $\bar{T}_{sk}$ = mean skin temperature; $\bar{T}_b$ = mean body temperature ($0.8\ \bar{T}_c + 0.2\ \bar{T}_{sk}$). † = differences between control and treatment are significant at the 0.05 level.

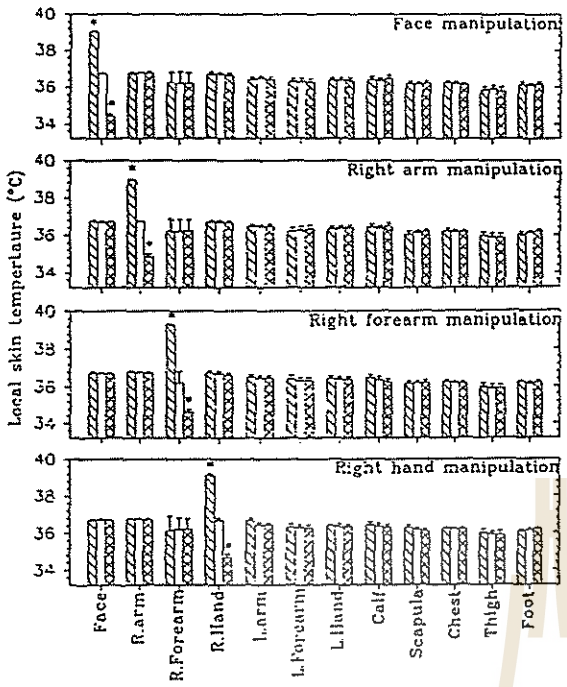

Figure 1 Regional skin temperatures during local heating and cooling of four skin regions. Data are means with standard errors of the mean. * indicates that the treatment (heating or cooling) was significantly different ($P < 0.05$) from the control skin temperature.

difference between treatment sites being non-significant ($P > 0.05$). That is, the current procedures consistently and equivalently modified T_{skl} across the four treatment sites. During T_{skl} manipulations, skin temperature at all the untreated sites remained relatively unchanged ($P > 0.05$; Fig. 1), confirming that the T_{skl} manipulations were localized to the treated site, and did not influence T_{skl} of regions either immediately adjacent or contralateral to the treated segment, during any manipulation period. While measures were not taken across every body segment, it is reasonable to assume, on the basis of these data, that T_{sk} remained stable across all non-treated body regions.

While $\bar{T}_{sk}$ was marginally influenced by local heating and cooling, it was not significantly altered during T_{skl} manipulations ($P > 0.05$; Table 1). This was verified from comparisons between the first and last minutes of each treatment period ($P > 0.05$). Therefore, the thermoafferent drive from the non-treated skin regions was deemed to have been clamped during T_{skl} manipulations, for all T_{skl} manipulations, during both cooling and heating. Thus, the only skin temperature modification induced by this experiment was a local effect, restricted to the site of the T_{skl} manipulation.

There was a constant temperature offset between the three core temperature indices, with T_{ac} and T_{re} being higher than T_{es}. However, these T_c indices remained unchanged during either localized cooling or heating ($P > 0.05$; Table 1). These combined observations demonstrate that T_{skl} manipulations did not affect either T_c or $\bar{T}_{sk}$, but remained isolated to the site where the manipulation took place. Resultant alterations in sweat output could therefore be said to be independent of either T_c or $\bar{T}_{sk}$ changes. Finally, neither $\bar{T}_b$ (Table 1) nor f_c were significantly affected by heating or cooling local skin surfaces, or by the site of local skin manipulation ($P > 0.05$).

Sweat rates at all eight recording sites exhibited the usual pulsatile variations. Due to the large variability of the sweat response, both between sites and subjects, data were analysed as $\dot{m}_{sw}$ changes, where data were integrated over the 7-min T_{skl} manipulation, and expressed as changes in sweat output relative to the similarly derived pre-manipulation $\dot{m}_{sw}$ (control: 4-min integration). While $\dot{m}_{sw}$s were recorded in mg cm^{-2} min^{-1}, for simplicity, sudomotor output is expressed in mass units (mg). When summed up across the eight measurement sites, and averaged across the four treated regions, the resultant $\dot{m}_{sw}$ change provided an overall assessment of the effects of local heating and cooling on total-body sweating, independent of both measurement and treatment sites. Heating induced an overall sweat elevation of 7.05 mg (±2.27), while cooling suppressed sweating by −2.40 mg (±1.04). That is, at a steady-state $\dot{T}_c$ of 36.9 °C and a steady-state $\dot{T}_{sk}$ of 36.2 °C, when warm and cool thermal stimuli of ≈3 °C were applied to the skin, the warm stimulus had approximately a three times more powerful affect upon whole-body sudomotor function.

Figure 2 summarizes the effects of local heating/cooling on $\dot{m}_{sw}$ at the site of the T_{skl} treatment. Regardless of water temperature used in the treatment, there was no evidence for either localized heating or cooling effects on $\dot{m}_{sw}$ ($P > 0.05$). That is, while heating tended to elevate, and cooling suppress, localized sweating, these differences were no greater than the sweating responses exhibited at other, non-treated skin regions at the time of the T_{skl} treatment. For example, face heating (far left bar within each group, Fig. 2a) elicited approximately equivalent sweat elevations across each of the eight measurement sites. The apparently greater sweat responses of the left and right hands were not significant, because of inherent $\dot{m}_{sw}$ variability at these sites ($P > 0.05$).

Because $\dot{m}_{sw}$ was recorded from three sites on the left upper limb (arm, forearm and hand), concurrently with each of the right arm treatments, data were available to assess the effects of treatments on the contralateral limb sweating responses. Neither stimulus produced significantly greater local sudomotor influences than were elicited contralaterally ($P > 0.05$). Figure 2 reveals that, in general, when any upper limb

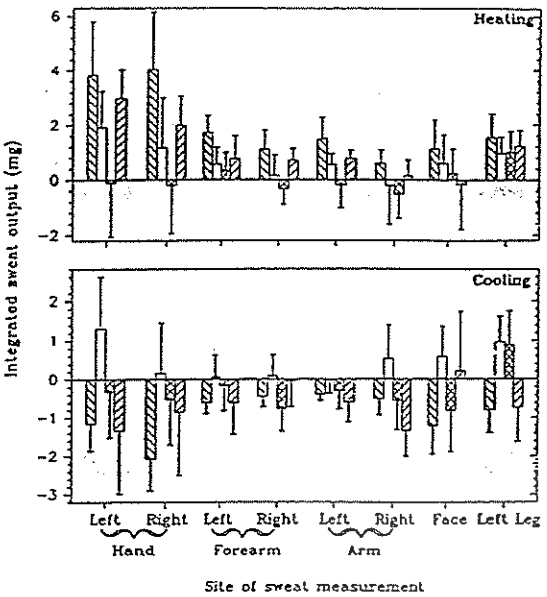

Figure 2 Changes in sweat output for each of eight sites (clustered bars), induced during heating and cooling of four skin regions (individual bars). Sweat outputs were derived by integrating sweat rates during a manipulation and subtracting the control sweat rate. Data are expressed as mass loss means, with standard errors of the mean.

segment was treated, quantitatively similar sweat responses were elicited on the corresponding contralateral limb segment.

The major aim of this investigation was to determine whether one of the four upper-body skin regions exerted a more powerful influence (greater sensitivity) over sweat secretion. Using the sweat output change data described above, differences in thermal sensitivity between the manipulated skin regions were examined using a repeated-measures, multivariate design. The within-subjects repeated measures were the eight sites for recording $\dot{m}_{sw}$. A significant main effect for treatment site would indicate that a between-site thermal sensitivity difference existed, and would be identifiable by the presence of a significant difference in the $\dot{m}_{sw}$ change (collapsed across measurement sites), produced by treating any of the four local skin regions. The resultant thermal sensitivity data are summarized in Fig. 3. The simple rank order (descending) for regional skin thermal sensitivity during local heating was the face, hand, arm and forearm, with forearm heating failing to elicit a sudomotor response above basal levels ($P > 0.05$). While these simple comparisons showed that, during local heating, the face was 1.9 times more sensitive than the hand, 2.9 times more sensitive than the arm, and 16.2 times more sensitive than the forearm (Fig. 3), none of these between-site differences were significant ($P > 0.05$). This point is emphasized because previous investigations have reported inter-regional differences in thermosensitivity purely on the basis of raw data comparisons, and without subsequent statistical analysis.

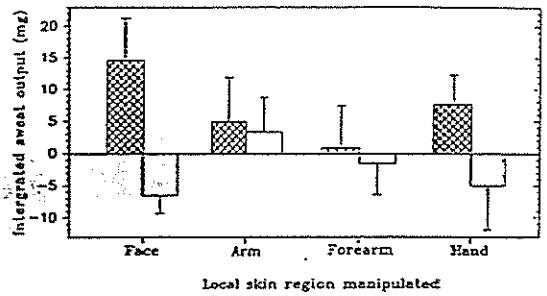

Figure 3 Whole-body sweat output (mg), summed across eight measurement sites, during localized heating and cooling of four skin regions. Sweat output was calculated by integrating sweat rates during a manipulation and subtracting the control sweat rate. Data are means with standard errors of the mean.

Cooling discrete skin surfaces resulted in a similar thermal sensitivity rank order (descending): face, hand, forearm and arm (Fig. 3). The raw data again revealed an apparently greater facial thermosensitivity: 1.3 times more sensitive than the hand, and 4.4 times more sensitive than the forearm (Fig. 3). Again, these apparent relative sensitivity differences were not significant ($P > 0.05$).

DISCUSSION

The current experimental manipulations effectively and consistently modified the thermal impulse at each treatment site. These manipulations were restricted to the treated site, and did not influence T_{skl} of body segments either immediately adjacent, or contralateral to that site. It was therefore assumed that the only skin temperature modification induced by the current experiment was a local effect. Furthermore, both T_c and $\bar{T}_{sk}$ were effectively clamped across the full duration of each trial. From these data, it may be concluded that the T_{skl} changes remained isolated to the site where the manipulations took place. We are unaware of other research which has been able to effectively isolate such thermal stimuli, while clamping other body temperatures.

This unique circumstance permitted an assessment of the thermal sensitivities of four upper-body skin regions, independently of either T_c and $\bar{T}_{sk}$ influences. On the basis of a simple comparison of the raw data, it is possible to infer that an apparent between-site, thermal sensitivity hierarchy existed, with the face demonstrating the greatest thermosensitivity, and the forearm being relatively insensitive (Fig. 3). Such a conclusion has previously been put forward on the basis of similar comparisons (Nadel *et al.* 1971,

Crawshaw *et al.* 1975). However, the methodological limitations of these studies (noted above), combined with the relatively small sample sizes, and the absence of statistical analyses may limit such interpretations.

In the current investigation, where both T_c and $\bar{T}_{sk}$ clamping was verified, and where equivalent warm and cool stimuli were applied to matched skin areas, statistical analyses did not support the differences in thermosensitivity of the forehead, right arm, right forearm and right hand. The present design was uniquely suited to reveal significant thermosensitivity differences, should they exist. However, the absence of significant differences, when considered with the repeated observation of a greater facial thermosensitivity, does not mean that this region is not more sensitive. It may simply mean that we were unable to statistically demonstrate a sensitivity difference.

Three additional features were present within the data. First, in accordance with the literature (Nadel *et al.* 1973, Werner & Heising 1990, Bothorel *et al.* 1991), local heating induced an overall sweat elevation, while cooling suppressed sweating (Fig. 2). Second, the tendency of the T_{skl} manipulations to induce a localized sweat response, was no more powerful than it was at producing a similar sweat response within any other, non-treated skin region (Fig. 2). Third, neither heating nor cooling the hand, forearm or arm produced a significantly more powerful local influence on sweating than could be induced contralaterally (Fig. 2).

The absence of a T_{skl} effect on local $\dot{m}_{sw}$ was an unexpected, but not an uncommon observation. Numerous groups have reported that, during T_{sk} perturbations, local $\dot{m}_{sw}$ will experience a greater change than at non-treated regions (e.g. MacIntyre *et al.* 1968, Ogawa 1970, Nadel *et al.* 1971). This local $\dot{m}_{sw}$ response was attributed to a thermal potentiation of efferent flow at the neuroglandular junction (Ogawa 1970, Nadel *et al.* 1971). While the present design does not permit an evaluation of the mechanisms underlying this hypothesis, the current data are not consistent with such observations, with alterations in sweat output occurring in all regions simultaneously, with each region being affected by about the same extent, regardless of the skin region being treated. Similar trends have been observed by Heising & Werner (1987), Werner & Heising (1990) and Bothorel *et al.* (1991). These groups variously concluded that T_{skl} minimally affects local $\dot{m}_{sw}$. However, it is possible, because the sweat capsules were ventilated with air at ambient temperature, that T_{sk} directly under the capsules located at each of the four treatment sites, was not equivalent to the T_{sk} that existed elsewhere below the treatment patches. That is, the impact of the T_{skl} change may not have been adequately applied below the sweat capsule located at the treatment site, and this small area of skin (3.16 cm^2) may not have experienced the full treatment effect. As T_{skl} within each sweat capsule was not measured, this possibility cannot be excluded.

Given the contralateral and ipsilateral potentiation of sudomotor function (Fig. 2), the present data imply that sudomotor changes, accompanying T_{skl} manipulations, have their neural origins within the central nervous system. For the T_{skl} change to affect both contralateral and ipsilateral sweating, some interaction between thermal afferents must occur, either at the spinal cord or the hypothalamus. Bothorel *et al.* (1991) suggested an interaction may occur between afferent and efferent signals at the spinal level, possibly affecting $\dot{m}_{sw}$ just at the level of interaction. Alternatively, the changes in T_{skl} may be integrated at the hypothalamus, resulting in a modification of central sweat drive, and simultaneously affecting $\dot{m}_{sw}$ at all skin regions (Bothorel *et al.* 1991). Regardless of the mechanisms, the results of the current project indicate that changes in the sudomotor response to T_{skl} alterations are possibly the result of an altered central sudomotor drive.

It is concluded that, when equal skin areas are either heated or cooled, such stimuli produce significant changes in sudomotor function, which appeared to be equally reflected both locally and contralaterally. Furthermore, the cutaneous thermoreceptor-induced, sudomotor responses to such localized treatments are equivalent, regardless of the site of thermal stimulation. While consistent with previous observations of a greater facial thermosensitivity, neither the current nor previous data statistically support the notion of inter-regional differences in cutaneous thermal sensitivity for sudomotor control.

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Human body-fluid distribution during exercise in hot, temperate and cool environments

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ABSTRACT

Using a simultaneous-dilution technique, we investigated body-fluid volume changes during exercise in seven males, during 50 min of cycling (50% maximal work rate) in hot (36.2 °C), temperate (22.0 °C) and cool conditions (14.4 °C). Total body water (TBW), extracellular fluid (ECFV), plasma (PV) and erythrocyte volumes (RCV) were measured, while blood volume (BV), interstitial fluid volume (IFV), extracellular water (ECW) and intracellular water volumes (ICW) were derived. During the initial 10 min of cycling, BV decreased in all environments ($P = 0.01$), primarily because of a PV reduction ($P = 0.01$), while IFV, ECFV and ICW were not significantly changed. By 30 min, BV recovered in the temperate and cool conditions, despite mass losses of 563 and 520 mL (respectively), but remained depleted in the hot condition ($P = 0.01$). The 50-min volume changes revealed that, throughout exercise, body-fluid losses appeared to be drawn primarily from the extracellular space, regardless of air temperature. In the hot condition, the PV change represented 63% of the TBW loss, with the ICW contributing 23%. It was concluded that, during cycling, progressive dehydration mainly affected the extracellular space, with the intravascular and intracellular spaces being defended in less stressful conditions.

Keywords blood volume, body fluids, exercise, extracellular fluid, heat stress, interstitial fluid, intracellular fluid, plasma volume.

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During exercise, both the volume and distribution of body fluids are challenged by thermoregulatory, hydrostatic and osmotic perturbations. Increased metabolic heat production elevates sweating, induces a peripheral redistribution of blood (Savard *et al.* 1988), and leads to between-compartment, body-fluid shifts (Harrison 1985). These factors result in the blood volume (BV) becoming unstable, and eventually decreasing during cycle exercise in the heat (Maw *et al.* 1996a,b), even in adequately hydrated subjects. This BV decrease is partially associated with increased capillary hydrostatic pressure, which elevates capillary filtration into the interstitium (Lundvall *et al.* 1972), and is also attributable to an elevated intramuscular tonicity (Sejersted *et al.* 1986, Björnberg 1990). Thus, under these conditions, the plasma volume (PV) tends to decrease, and this change has even been observed during exercise in cool environments (Harrison 1985).

During prolonged exercise, fluid losses from the plasma result in plasma hypertonicity, relative to the red blood cells. The red cell volume (RCV) decreases following a fluid efflux into the plasma space, and is apparent across a range of environmental conditions (Åstrand & Saltin 1964, Costill *et al.* 1974). However, this change has not been universally supported, with some studies reporting no change during exercise (Myhre & Robinson 1969). This inconsistency may be because of differences in measurement techniques between investigations, or even because of differences in the mode of exercise. For instance, Maw *et al.* (1996a,b) found that changes in mixed-venous haematocrit and haemoglobin concentration do not permit the reliable tracking of PV changes during the first 10 min of exercise. As the most commonly used method of evaluating PV change is based upon alterations to those indices (Dill & Costill 1974), it remains relatively uncertain how the intravascular volumes respond to combined exercise and thermal stress.

It is similarly undetermined how the redistribution of intravascular fluid during cycling affects the volume

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and distribution of extravascular fluid. Nose *et al.* (1988) suggested that plasma left the circulation, being taken up to defend extravascular cell volume, and to fuel sweat secretion. Similarly, Costill *et al.* (1976) showed that the intracellular compartment appeared to be defended, relative to the extracellular space, during the early stages of exercise-induced dehydration. Both studies determined fluid volumes using the chloride method. While this technique is regularly used, it may be influenced by changes in muscle membrane potential during exercise, possibly causing a change in chloride concentration independently of fluid movement (Sjøgaard & Saltin 1982). Therefore, the pattern of whole-body-fluid movement in exercising humans, remains somewhat unclear, especially under thermal stress conditions, where we are heavily reliant upon data collected primarily from the vascular compartment. As the PV represents less than 10% of total body water (TBW), a clear picture of whole-body-fluid movement during combined exercise and thermal stress is somewhat difficult to derive from these data. The current investigation addressed this restriction, employing simultaneous radionuclide dilution to measure fluid distribution within the major compartments, during exercise. This unique method has resulted in the provision of new data for body-fluid distribution during cycling in hot, temperate and cool conditions.

METHODS

Body-fluid distribution was measured in seven healthy, physically active males, during 50-min cycling [subject characteristics: 26.2 year (±4.0), 178.5 cm (±6.5), 78.02 kg (±8.61), sum of eight skin folds 71.6 mm (±16.5), mean aerobic power 65.6 mL $kg^{-1}min^{-1}$ (±10.7): means with standard deviations]. Exercise was performed on a cycle ergometer (Monark, 868) in hot [36.2 °C (±0.7), relative humidity (rh) 44% (±3)], temperate [22.0 °C (±1.0), 52% rh (± 6)], and cool conditions [14.4 °C (± 1.6), 74% rh (± 9)], while wearing a swimsuit and running shoes. Air movement was < 0.5 m s^{-1}, and black-globe temperature was always within ± 0.5 °C of air temperature. Subjects were tested at the same time for each condition, with exposure order balanced between subjects, and each trial separated by 28 days. The residual radiation before each reassessment was < 0.01% of the previous dose. During this 28-day period, subjects resumed normal dietary and exercise patterns. Subjects provided informed consent for procedures approved by the Human Research Ethics Committee (University of Wollongong).

Subjects presented in a rested state, following a 12-h overnight fast, consumed a standardized food and water intake (38 kJ kg^{-1} and 5 mL kg^{-1}). No other dietary restrictions were applied. Subjects were seated in a temperate environment (22.0 °C) for 30 min to stabilize metabolism. An antecubital vein was catheterized, and four radionuclides were injected: 450 μCi of tritiated water (^3H_2O, Amersham Australia); 20 μCi of sodium radiobromide ($Na^{82}Br$, Australian Radioisotopes); 2 μCi of radioiodinated serum fibrinogen (^{125}I Human Fibrinogen, Amersham, Australia); and 8 μCi of radiochromated autologous erythrocytes ($Na^{51}Cr$, Amersham, Australia). Using the simultaneous dilution of these radionuclides, TBW, extracellular fluid (ECFV), PV and RCV, respectively, were measured (after Maw *et al.* 1996a,b). Subjects remained seated for 270 min following these infusions, to allow radionuclide equilibration within the relevant compartments.

Subjects were transferred between environments in a wheelchair, to minimize postural disturbance Maw *et al.* (1995), and immediately commenced cycling. Work rates were 50% of the peak work rate achieved during a ramp cycle forcing function, determined 3 days before the first trial. The same absolute work rate was used in each condition. Blood samples (10 mL), collected using the indwelling catheter, were taken just before the exercise, at 10-min intervals during the exercise, and immediately after the exercise, followed by a 10-mL flush of heparinized saline. Body mass was determined at 10-min intervals, and a urine void was collected before and after the exercise.

Plasma ^3H was analysed using liquid scintillation (LKB Wallac, 1219 Rackbeta), while plasma ^{82}Br and ^{125}I, and erythrocyte ^{82}Br and ^{51}Cr were determined using γ-scintillation (Abbott Laboratories, Auto-LOG-IC). Radionuclides were also measured in the urine samples. Samples were counted in triplicate, for a minimum aggregate of 10000 counts for each radionuclide. Total body water was calculated by comparing the plasma ^3H concentration with the exact ^3H dose, corrected for the presence of plasma protein (Otago refractometer Model 93032) and ^{125}I, and for ^3H urinary, sweat and respiratory losses. Extracellular fluid volume was determined from plasma ^{82}Br concentration, corrected for the presence of protein, for ^{82}Br erythrocyte, urinary and sweat losses, and for the Gibbs–Donnan electrolyte ratio (1.02). Plasma volume was derived by comparing the measured plasma ^{125}I concentration with that predicted for the corresponding time using semi-logarithmic extrapolation of the ^{125}I elution curve. Finally, RCV was calculated from erythrocyte ^{51}Cr concentration, corrected for ^{51}Cr urinary loss. See Maw *et al.* (1996a,b) for comprehensive details of these procedures.

Intracellular water volume (ICW) was taken as the difference between TBW and extracellular water volume (ECW), while the latter ECW was calculated from ECFV adjusted for the presence of all plasma solutes.

Interstitial fluid volume (IFV) was considered as the difference between ECFV and PV. Blood volume was then the sum of PV and RCV.

Body-core temperature (T_c), skin temperatures, and cardiac frequency (f_c) were recorded at 5-s intervals throughout exercise. Core temperature was recorded using a zero-gradient auditory (aural) canal thermistor (T_{ac}), secured with a cotton wad, and covered by an insulated servo-heated headset (Keatinge & Sloan 1975). This technique involved independent monitoring of auditory canal temperature and that of an outer-ear heating pad. The temperature of the latter was controlled to track auditory canal temperature, which, after equilibration, acts to isolate T_{ac} from environmental and skin temperature influences. This technique removes the auditory canal thermal gradient, permitting T_{ac} to faithfully track tympanic temperature (Moore & Newbower 1978), and has been validated within the present laboratory against oesophageal, tympanic and rectal temperatures (Cotter *et al.* 1995). During cycling, under similar environmental conditions, T_{ac} is typically 0.19 °C above oesophageal ($r = 0.940$), 0.21 °C greater than tympanic ($r = 0.974$) and 0.21 °C less than rectal temperature ($r = 0.965$).

Skin temperatures were recorded at 0.2 Hz from eight sites (Grant Instruments Ltd, 1206 Series Squirrel, UK), using surface thermistors (Edale Instruments, Cambridge, UK), secured with a single covering of waterproof tape. Mean skin temperature ($\overline{T}_{sk}$) was subsequently calculated from the area-weighted mean of these temperatures (International Organization for Standardization 1992). Cardiac frequency was recorded from ventricular depolarization (Polar Electro Sport-Tester, PE3000, Finland), validated using 12-lead electrocardiography.

Whole-body sweat loss was determined from mass changes (standing; A & D Instruments, FW-150K, Germany), corrected for the exchange of oxygen and carbon dioxide, absorption of sweat into clothing, urinary and respiratory losses, and blood sampling, but was not subdivided into its evaporative and non-evaporative fractions. Mass determinations immediately followed blood sampling, and took < 1 min to perform. It was considered that such a postural disturbance had minimal impact upon the seated body-fluid distribution. Sweat was wiped from both the scale and the subject prior to weighing, and the subject's swimsuit and shoes were weighed before and after exercise, to determine sweat absorption. Subjects were not rehydrated within trials, as our aim was not to equate TBW between exposures, but to evaluate the impact of these exposures on TBW, and then to determine the origins of fluid lost from the total system.

Respiratory water loss ($\dot{M}_{E\,H_2O}$) was determined from the temperature, water content (Hygrodynamics, 15–3080E, USA) and volume (Vacumetrics, Air Flow Meter 17150, USA) of expired gases, collected using a pre-warmed respiratory face mask, low-resistance tubing and non-diffusing collection bag (Hans Rudolph, Series 7910 face mask and non-diffusing collection bag, U.S.A.) after 5, 15 and 45 min of exercise. Thus:

$$\dot{M}_{E\,H_2O} = M_{E\,H_2O} \times \dot{V}_{E_{ATPS}}\ g \cdot min^{-1}$$

where $M_{E\,H_2O}$ = mass of water per litre expired gas ($g\ L^{-1}$; Weast *et al.* 1989); $\dot{V}_{E_{ATPS}}$ = expiratory flow ($L\ min^{-1}$ ATPS).

Auditory canal temperature, $\overline{T}_{sk}$ and f_c were averaged at 10-min intervals (± 30 s). To negate the effects of normal physiological variation on pre-exposure fluid volumes, over the 56-day course of the study, all fluid volumes were normalized to each subject's mean pre-experimental value, determined before each trial. Data were then analysed using factorial analysis of variance, to determine differences related to environment and time, with alpha set at the 0.05 level. Subsequent *post-hoc* analyses were performed using Tukey's test of Wholly Significant Difference. Data are reported as means with standard errors of the mean.

RESULTS

Pre-exposure T_{ac} averaged 37.0 °C (±0.2), $\overline{T}_{sk}$ was 30.9 °C (± 0.3), and the mean resting f_c averaged 75 beats min^{-1} (± 8). In the temperate and hot trials, T_{ac} initially fell from baseline, before rising by 1.3 °C (± 0.2) and 1.4 °C (± 0.2; Fig. 1; $P = 0.01$), respectively, after about 10 min. However, T_{ac} was independent of air temperature, and did not differ between conditions over time ($P = 0.34$). In contrast, $\overline{T}_{sk}$ was consistently higher during exercise in the hot condition, than in the other two conditions ($P = 0.01$), and was also higher in the temperate than in the cool trials (Fig. 1; $P = 0.01$), reflecting the combined effects of air temperature and cutaneous vascular responses. Cardiac frequency was also higher in the hot condition than in either of the other conditions (Fig. 1; $P = 0.01$), but was equivalent between the temperate and cool environments ($P = 0.11$). This f_c elevation was an almost constant displacement, such that across trials, f_c was 13–14 beats min^{-1} greater than observed in either the temperate or the cool states ($P = 0.01$).

Acute body-fluid responses to cycling (0–10 min)

Baseline TBW, ECFV, PV and RCV averaged 50975 mL(±1763), 20316 mL(±632), 3488 mL(±136) and 2698 mL(±152), respectively, with the corresponding ICW, IFV and BV being 30853 mL(±1267), 16828 mL(±519), and 6186 mL(±258). These volumes, with the exception of ECFV, were slightly higher than

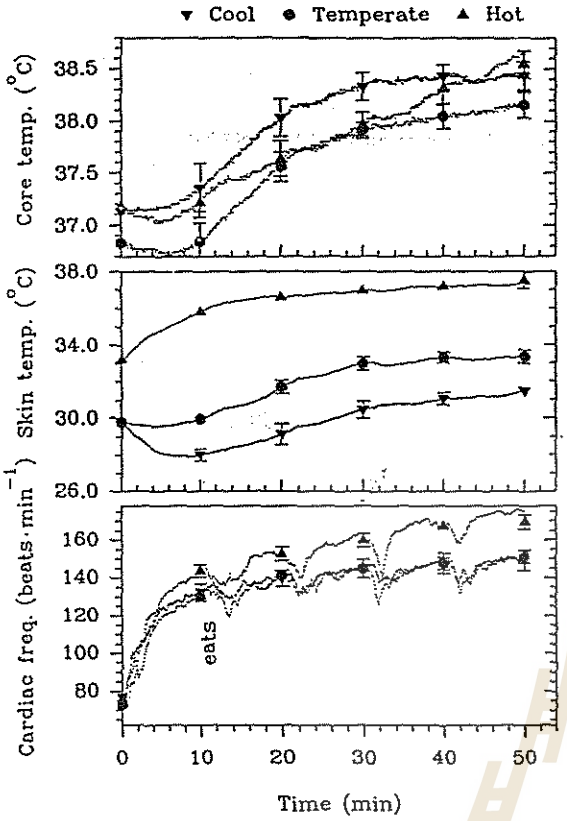

Figure 1 Auditory canal and mean skin temperatures, and cardiac frequency during 50 min of cycling at 50% of maximal work rate in hot (36 °C), temperate (22 °C) and cool (14 °C) environments. Data are means ± SEM.

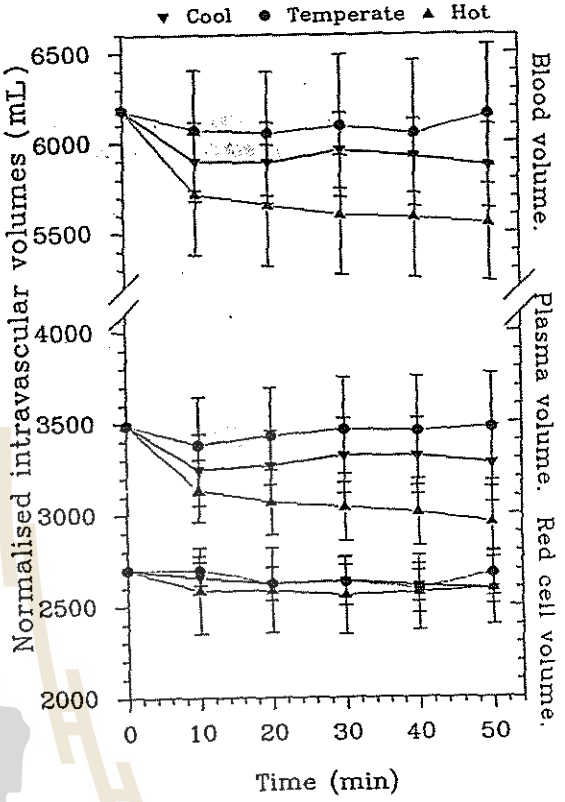

Figure 2 Blood, plasma and erythrocyte volumes during 50 min of cycling at 50% of maximal work rate in hot (36 °C), temperate (22 °C) and cool (14 °C) conditions. Volumes were normalized to pre-experimental levels, and are presented as means ± SEM.

reference standards for healthy adult males (International Committee for Standardization in Haematology 1980), ranging from 8 (TBW) to 17% (PV) greater than the reference standards. However, such volume expansions were considered appropriate for relatively lean, endurance-trained males (Sjöstrand 1962), and have been previously detailed (Maw *et al.* 1996a,b).

At the commencement of cycling, BV decreased in all environments ($P = 0.01$), with the decrease being greater in both the hot (470 ± 192 mL) and the cool exposures (287 ± 60 mL) than in the temperate condition (114 ± 86 mL; $P = 0.01$; Fig. 2). These changes were largely accounted for by plasma shifts, with PV contracting in all conditions (Fig. 2; $P = 0.01$), but more so in the hot and cool exposures (356 ± 128 and 243 ± 42 mL, respectively) than in the temperate condition (110 ± 46 mL; $P = 0.01$). Red cell volume made only a minor contribution to BV losses, with differences from pre-exposure levels not reaching significance until 20 min (Fig. 2), by which time RCV had decreased by 114 (± 62), 76 (± 55) and 75 (±37) mL in the hot, temperate and cool conditions, respectively ($P = 0.01$ within conditions, and $P = 0.87$ between conditions).

Interstitial fluid, ECFV and ICW were not significantly affected during the initial 10 min of exercise, regardless of the environment (Fig. 3; $P = 0.54$, $F = 0.73$ and $P = 0.35$, respectively). Similarly, TBW was unaltered and sweat losses were minimal, although mass decreased in all conditions: 46 mL (±18; hot), 49 mL (±23; temperate) and 79 mL (±46; cool; $P = 0.78$). The difference between the TBW and mass changes were within the measurement error of the former technique. The apparent contradiction between the hot and cool sweat losses was not significant.

Body-fluid responses to extended cycling (10–50 min)

By 30 min in the temperate and cool conditions, BV had recovered to basal levels, where it remained for the duration of exercise, but BV stayed depleted in the hot condition (Fig. 2; $P = 0.01$). In this exposure, BV progressively decreased with time, reaching a 635-mL reduction after 50 min of exercise. Red cell volume was significantly depleted from 20 min onwards in each environment (Fig. 2; $P = 0.01$), losing a total of 103 (±101), 20 (±40) and 106 (±52) mL during 50 min in the hot, temperate and cool states. These between-

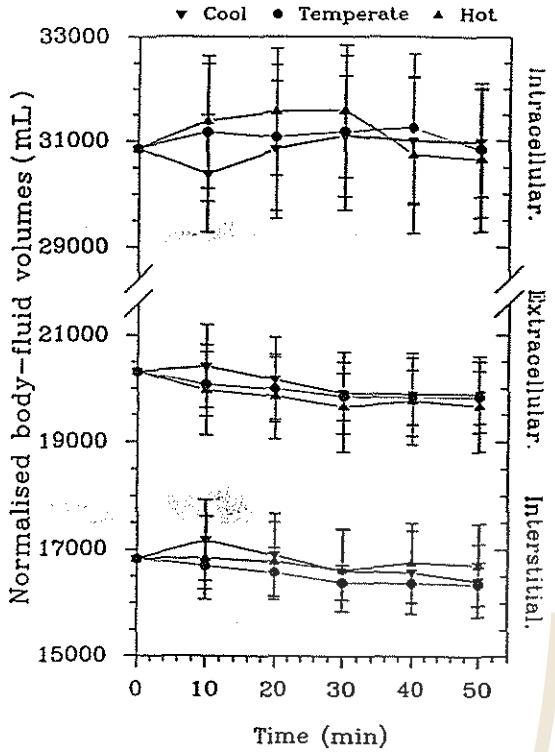

Figure 3 Intracellular water, extracellular fluid and interstitial fluid volumes during 50 min of cycling at 50% of maximal work rate in hot (36 °C), temperate (22 °C) and cool (14 °C) environments. Volumes were normalized to pre-experimental levels, and are presented as means ± SEM.

condition differences were not significant ($P = 0.87$). Thus, in each condition, PV was the major determinant of the BV. Following acute reductions, PV recovered by 30 min in the temperate and cool trials, but slowly decreased throughout the heat exposure (Fig. 2; $P = 0.01$).

Beyond 10 min, IFV and ECFV gradually decreased during all trials (both $P = 0.01$), with changes being independent of air temperature (Fig. 3). No consistent changes were detected in ICW throughout exercise (Fig. 3; $P = 0.35$), although this probably reflected the relative sensitivity of ICW measurement, as much as it did the stability of the intracellular compartment. For example, the biggest change in ECFV, a decrease of 654 mL after 50 min exercising in the hot condition, equated to less than 2.5% of ICW, and may have been close to the ICW measurement error.

To summarize these trends from a more general perspective, body-fluid changes, across the entire exposure, were also derived for each compartment, by comparing the pre-trial and the 50-min fluid volumes. These data are summarized in Fig. 4. While these data do not convey information about the dynamics of such changes, they do provide an appreciation of the longer-term affects of exercise under thermal stress.

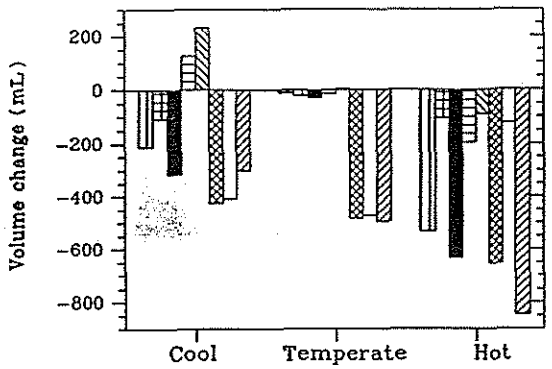

Figure 4 Absolute changes in body-fluid volumes following 50 min of cycling at 50% of maximal work rate in hot (36 °C), temperate (22 °C) and cool (14 °C) environments. Volumes include: plasma volume (PV); red cell volume (RCV); blood volume (BV); intracellular water (ICW); extravascular intracellular water (ICW-RCV); extracellular fluid volume (ECFV); interstitial fluid volume (IFV); and total body water (TBW). For simplicity, volume changes are presented only as means. (▥ PV, ▤ RCV, ■ BV, ▤ ICW, ▧ ICW–RCV, ▨ ECFV, □ IFV, ▨ TBW.)

In all instances, mass losses accounted for less than 2% of the initial TBW. These losses were fractionated into sweat secretion [861 mL (± 100; hot), 563 mL (±61; temperate), 520 mL (±70; cool)], respiratory water loss [60 mL (± 3; hot), 59 mL (±2; temperate), 55 mL (±3; cool)], and urinary losses [13 mL (±9; heat), 51 mL (±18; temperate), 83 mL (±33; cool)]. In the heat condition, these deviations were always significantly different from fluid losses sustained in the cool condition ($P = 0.01$).

DISCUSSION

Acute body-fluid responses to cycling (0–10 min)

During the first 10 min of exercise within each environment (the acute response), there was a rapid decrease in BV. This trend may be explained on the basis of both an increased intravascular hydrostatic pressure and an elevated intramuscular osmotic force. Capillary hydrostatic pressure increases during cycling (Björnberg 1990), forcing an extravasation of fluid through the capillary walls. This can occur at the capillary endothelium via pores or clefts. A simultaneous increase in intramuscular tonicity, perhaps accompanying an increase in metabolites within the muscle, would facilitate fluid movement into the active muscle tissues (Sejersted *et al.* 1986, Björnberg 1990). Hence, an acute BV reduction was observed across conditions, with this change primarily attributable to changes within the PV, rather than the RCV space.

Although resting heat stress can expand BV, as a result of rapid and generalized cutaneous venodilation

(Harrison 1985), cycling in the hot condition significantly reduced BV to a greater extent than observed in the temperate condition (Fig. 2). Regardless of the environmental temperature, the fluid leaving the capillaries should consist of plasma and its subcellular constituents, as capillary membranes are impermeable to intravascular cells. Thus, the acute decrease in BV was brought about by a plasma efflux (Fig. 2). However, it is possible that the fluid volume leaving the plasma probably exceeded the measured net PV decrease, as a result of the concurrent influx of fluid from other sources. For example, while plasma is primarily lost at the arterial ends of capillaries, and returned at their venous boundaries, Lundvall *et al.* (1972) suggested that, during exercise, fluid is also drawn into the vascular space from inactive tissues, while simultaneously filtering into the more active tissues.

Assuming that cutaneous venodilation had occurred in the hot trials, and in the absence of colloid osmotic pressure measurements from either the vascular and interstitial spaces, then a BV contraction may result from a more powerful and extensive vasodilation, which is known to occur in both muscle and cutaneous beds during exercise in the hot state (Rowell *et al.* 1969). Under such circumstances, changes in the hydrostatic pressure favours a plasma efflux, and both the PV and BV were reduced (Fig. 2).

It is not possible, from the present data, to precisely determine the destination of the fluid lost from the vascular space during the initial 10 min of cycling, as IFV, ECFV and ICW were apparently unchanged, regardless of the environment. However, Sjøgaard & Saltin (1982) have shown that intramuscular water content increases during short-duration cycling, with the majority of the increase occurring in the extracellular compartment. Thus, it is probable that, in this study, the plasma filtrate was drawn into the extracellular compartment of active muscles, in response to an increase in that compartment's osmotic potential.

Body-fluid responses to extended cycling (10–50 min)

As cycling progressed in the temperate and cool environments, the PV, and hence BV, progressively returned towards pre-exposure levels (Fig. 2), even though TBW had declined about 1%. This restoration was probably associated with parallel increments in interstitial hydrostatic and capillary osmotic pressures, reversing the acute plasma loss (Jacobson & Kjellmer 1964). Prior to exercise, and during the acute cycling phase, the interstitial hydrostatic pressure facilitates plasma efflux. However, the initial PV depletion would increase interstitial hydrostatic pressure, and limit continued PV losses (Aukland & Nicolaysen 1981). At the same time, an increasing plasma tonicity creates an osmotic gradient favouring fluid influx from both the interstitium and the red blood cells (Nose *et al.* 1991). The net result of these factors was a trend towards PV restoration in both the cool and temperate conditions.

The fluid shift from the erythrocytes to the plasma was only significant beyond 20 min, varying between 2.7 and 4.2% (at 20 min) across the three exposures (Fig. 2). Similarly, Diaz *et al.* (1979) and Åstrand & Saltin (1964) found that RCV decreased during cycling and cross-country skiing. This reduction may also be attributed to a decrease in circulating cell numbers, or the volume of individual cells. Exercise-induced haemolysis will reduce red cell counts, but this is unlikely within the first 20 min of cycling at 50% peak aerobic power, especially given the return to pre-exposure RCV observed within the temperate state (Fig. 2). Furthermore, Laub *et al.* (1993) suggested that erythrocyte numbers may actually increase early during exercise, because of splenic discharge. If some degree of splenic emptying is assumed, then the current RCV depletion must have resulted from cellular dehydration. However, such decreases are not universally corroborated. For example, Myhre & Robinson (1969) and Wilkerson *et al.* (1977) found no change in RCV during prolonged exercise, despite significant decreases in PV. While it is difficult to reconcile these conflicting reports, it is evident these changes, even when statistically significant, are quite small in relation to the PV change (Fig. 2).

In the hot state, the pre-exposure BV was not re-established, but continued to decline. It is suggested this gradual loss was because of less pronounced changes in capillary osmotic and interstitial hydrostatic pressures during the hot exposure. Senay *et al.* (1980) provided evidence for altered capillary osmotic pressure during cycling in the heat, demonstrating that total circulating albumin decreased as a result of increased capillary filtration. Thus, an elevated capillary tonicity would have been less apparent during the current hot trials. At the same time, progressive dehydration would facilitate a more rapid clearance of fluid entering the interstitium, minimizing the interstitial hydrostatic pressure. Such a clearance was manifest within progressive reductions of the IFV and ECFV in each environment (Fig. 3).

While the discussion above has focused upon the dynamics of fluid-volume changes, a more general appreciation of the longer-term affects of exercise under thermal stress is obtained from Fig. 4. For instance, it is apparent that, within the temperate condition, TBW changes were almost entirely attributable to losses from the IFV, with neither the intravascular nor the intracellular fluid spaces experiencing physiologically significant changes. Therefore, the acute reduction in PV, seen across all conditions, was not apparent in the temperate trials within this analysis, and it is assumed

that the PV restoration occurred at the expense of the IFV. Across the cool trials, the extravascular intracellular compartment was the only volume space to be augmented, *albeit* by <1% of the pre-exposure volume, with both the plasma and interstitial spaces decreasing.

During exercise in the hot condition, all fluid compartments declined, with TBW loss primarily associated with a 532-mL fluid loss from the PV (Fig. 4). Using this initial versus final volume comparison, the PV change represented 63% of the TBW loss, with the ICW contributing 23%. It may be assumed this fluid passed into the interstitium, helping replace that lost through sweating, buffering the IFV decrease to be only 14% of the TBW decline. Consequently, the extracellular space accounted for 77% of the total fluid loss. Prior to exercise, ECFV represented only 40% of the TBW, while ICW provided the remaining 60%. Thus, intracellular fluid was defended under each of the three test conditions. While such trends have been previously reported (Costill *et al.* 1976, Durkot *et al.* 1986, Nose *et al.* 1988), it must be emphasized that, as a result of the somewhat short-duration exercise, the modest elevation in T_c, and the relatively small dehydration effects, the extravascular body-fluid changes represent relatively small fractions of both the TBW and the extravascular spaces. Therefore, while Fig. 4 provides a useful summary of these changes, some are close to, or within, the resolution of the measurement techniques, and require verification under more stressful experimental conditions.

To our knowledge, these trials represent the first simultaneous measurement of the major body-fluid compartments during cycling across cool–hot environments. Three principle observations were made. First, during the initial 10 min of cycling, BV was decreased across the three conditions, primarily because of PV reduction. Second, in support of previous research, it was found that the intravascular volume appeared to be defended to some extent in all but the hot environment, where it was progressively depleted. Such defence would minimize cardiovascular strain. Third, body-fluid losses appeared to be drawn primarily from the extracellular reserves, regardless of air temperature. Consequently, it was concluded that, during cycling, progressive dehydration mainly affected the extracellular space, with the intravascular and intracellular spaces being defended in the two less stressful conditions.

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Can skin temperature manipulation, with minimal core temperature change, influence plasma volume in resting humans?

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Abstract We investigated body-fluid distribution in resting humans, during short-term, whole-body skin temperature modification, in which core temperature changes (ΔT_c) were minimal. Seven males participated in hot (36.2°C (*s.d.* 0.7), 44% relative humidity (rh; *s.d.* 3)), temperate (22.0°C (*s.d.* 1.0), 52% rh (*s.d.* 6)), and cool trials (14.4°C (*s.d.* 1.6), 74% rh (*s.d.* 9)), while seated at rest. Total body water (TBW), extracellular fluid (ECF), erythrocyte (RCV) and plasma volumes (PV) were measured using a simultaneous radionuclide dilution technique. In the cold, PV contracted by 205 ml (±60) by the end of exposure ($p = 0.04$), while in the heat, PV expanded 108 ml (±123; $p = 0.02$). Both RCV and TBW remained stable, regardless of the environment. Despite fluid movement across the vascular wall, ECF, interstitial and intracellular volumes were relatively unaffected by skin temperature. It was concluded that, at rest, and with minimal ΔT_c, the intravascular fluid volume was dependent on prevailing environmental conditions, and its impact on local skin temperature and venomotor tone.

Key words Blood volume · Body fluids · Extracellular fluid · Heat · Plasma volume · Rest · Total body water

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Introduction

When air temperature approximates body-core temperature (T_c), dry heat exchange is negligible, and body fluids facilitate evaporative cooling. The resultant water loss and vascular responses, particularly during exercise at an elevated T_c, affect body fluids and their distribution (Nose et al. 1988; Maw et al. 1998). Postural changes (Maw et al. 1995) and significant T_c reductions also lead to plasma volume (PV) adjustments (Vogelaere et al. 1992). Furthermore, when commencing from thermoneutral rest, it has been established that generalised skin heating and cooling, which also induce large parallel T_c changes, result in haemoconcentration (Harrison et al. 1983). However, since most research has focussed upon the PV which occupies only 7–10% of the total fluid volume, little is known of the extravascular volumes during resting treatments. Furthermore, we are unaware how sensitive PV changes are to skin temperature manipulations, without concurrent and comparable T_c modifications. Accordingly, we herein report fluid volumes in hot and cool states, in which T_c changes were minimal, fractionating total body water into its tissue compartments.

Methods

Seven physically-active males (26.2 yr (*s.d.* 4.0); height 178.5 cm (*s.d.* 6.5); mass 78.0 kg (*s.d.* 8.6); sum of seven skinfolds 65.6 cm (*s.d.* 16.5)) were studied under three conditions: hot (36.2°C (*s.d.* 0.7), 44% relative humidity (rh; *s.d.* 3)), temperate (22.0°C (*s.d.* 1.0), 52% rh (*s.d.* 6)), and cool (14.4°C (*s.d.* 1.6), 74% rh (*s.d.* 9)). Air movement was < 0.5 m · s^{-1}, and black-globe temperature was within 0.5°C of air temperature. Trials were separated by 28 days, and presented in an approximately balanced order between subjects, who provided informed consent to procedures approved by the University's Human Research Ethics Committee.

In each trial, body-fluid compartments were quantified using a simultaneous radionuclide dilution method (Maw et al. 1996). Radioiodinated human serum fibrinogen (RISF; ^{125}I human fibrinogen, Amersham, Australia), radiochromated autologous erythrocytes ($Na^{51}Cr$ Amersham, Australia), radiobromide ($Na^{82}Br$,

Australian Radioisotopes, Australia) and tritiated water (^3H_2O, Amersham, Australia) were used to quantify PV, red cell volume (RCV), extracellular fluid volume (ECF) and total body water (TBW) respectively.

Subjects arrived in a rested, fasting state. Urine and venous blood samples were collected for reference and radiochromate labelling. A cannula was inserted (antecubital vein), through which to administer the radionuclides and to draw blood samples. A controlled breakfast (38 kJ · kg^{-1} body mass, plus 5 ml · kg^{-1} of water) was consumed, and subjects assumed a seated posture, which was maintained for 180 min prior to exposure. Two microcuries (μCi) of RISF, 8 μCi of sodium radiochromated erythrocytes, 20 μCi of $Na^{82}Br$, and 500 μCi of ^3H_2O were injected within 30 s (Maw et al. 1996). The cannula was flushed with 15 ml of saline, and 5 ml of heparinised saline. Ten-millilitre blood samples were collected after 30, 60 and 180 min (urine at 180 min), to determine pre-exposure fluid volumes (Maw et al. 1996). The final 30-min period of this 180 min, was spent within a temperate (control) environment. Wearing a swimsuit and shoes, subjects were then moved from the control to the treatment environment, using a wheelchair to maintain posture. From min 180–210, subjects remained seated (string-backed chair) in one of the treatment conditions. During these exposures, 10-ml blood samples were collected at 15 and 30 min, with a urine sample collected after exposure.

Compartmental fluid volumes were determined using equations described by Chien and Gregersen (1962). Plasma ^3H was analysed using liquid scintillation (1219 Rackbeta, LKB Wallac, Finland), while plasma ^{82}Br and ^{125}I, and erythrocyte ^{82}Br and ^{51}Cr were determined using γ-scintillation (Auto-LOGIC, Abbott Laboratories), with samples counted in triplicate. Total body water was calculated by comparing the plasma ^3H concentration with the ^3H dose, corrected for plasma protein and ^{125}I, and for ^3H urinary, sweat and respiratory losses. Extracellular fluid volume was determined from plasma ^{82}Br concentration, corrected for protein, erythrocyte, urinary and sweat losses, and for the Gibbs-Donnan electrolyte ratio (1.02). The mid-time of the fibrinogen injection taken as the commencement of assessment (t_0), and the ^{125}I concentrations obtained 15, 30 and 60 min after infusion were used to derive an elution curve, from which to determine: (a) the theoretical ^{125}I plasma concentration at t_0; and (b) the PV that would exist had the experimental manipulation not occurred (Harrison and Edwards, 1976). All ^{125}I concentrations were corrected for the gradual loss of ^{125}I. Experimental PV was derived by comparing the plasma ^{125}I concentration with that predicted for the corresponding time. Finally, RCV was calculated from erythrocyte ^{51}Cr concentration, corrected for ^{51}Cr urinary loss. See Maw et al. (1996) for procedural details. Intracellular water volume (ICW) was taken as the difference between TBW and extracellular water volume, with the latter calculated from ECF adjusted for all plasma solutes. Interstitial fluid volume (ISF) was the difference between ECF and PV, and blood volume (BV) the sum of PV and RCV. Body-fluid volumes were normalised to their initial values, to negate physiological variations over the 56-d experimental period.

$$PV = S_I * S_d * S_V / T_{I0}$$

where: S_I = ^{125}I concentration of the ^{125}I standard;
S_d = dilution of the ^{125}I standard;
S_V = volume of the RISF injection; and
P_{I0} = theoretical ^{125}I concentration in plasma at t_0.

Body-core temperature (zero-gradient auditory canal thermistor (T_{ac})), skin temperatures, and cardiac frequency (f_c) were recorded at 5-s intervals. Zero-gradient thermometry minimises the auditory canal thermal gradient and air temperature artefact, permitting tympanic and oesophageal temperature tracking. Skin temperatures were recorded from eight sites (1206 Series Squirrel, Grant Instruments Ltd., U.K.), using surface thermistors (EU thermistors, Edale Instruments, U.K.), with mean skin temperature ($\bar{T}_{sk}$) calculated using an area-weighted mean. Thermistors were calibrated against a certified mercury-in-glass thermometer. Mean body temperature ($\bar{T}_b$) was derived as: $0.8 * T_{ac} + 0.2 * \bar{T}_{sk}$ (temperate); $0.65 * \bar{T}_{ac} + 0.35\bar{T}_{sk}$ (cool); and $0.9 * \bar{T}_{ac} + 0.1\bar{T}_{sk}$ (hot). Cardiac frequency was recorded from ventricular depolarisation (PE3000, Polar Electro SportTester, Finland). Body mass was measured before and after each exposure (Fw-150 k, A&D, Germany), and corrected for metabolic, urinary and respiratory losses, sweat absorption into clothing, and blood sampling.

Data were analysed using one-way analysis of variance, and are presented as means with standard errors of the means, unless stated otherwise.

Results

In the temperate environment, T_{ac} averaged 37.22°C (±0.3), with $\bar{T}_{sk}$ being 31.4°C (±0.7), and f_c 61 b min^{-1} (±10). While $\bar{T}_{sk}$ followed air temperature, reaching 35.8°C (±0.1) and 28.1°C (±0.4) at the end of the hot and cool trials respectively (p = 0.001), T_{ac} changed paradoxically. During the first 20 min, T_{ac} fell 0.4°C in the heat, but increased 0.6°C in the cool, with respective average T_{ac}s of 36.84°C (±0.11) and 37.80°C (±0.16; p = 0.001). Terminal $\bar{T}_b$ was: 35.56°C (±0.07: temperate), 36.68°C (±0.09: hot) and 34.43°C (±0.23; cool; p = 0.01). Cardiac frequency increased in both the hot (7 b · min^{-1}) and cool trials (4 b · min^{-1}; p = 0.001). Thus, this protocol induced a significant, *albeit* mild, thermal strain in resting subjects, but it did so with minimal T_{ac} change, and without driving T_{ac} in the direction of the thermal stimulus.

Total body water remained constant across exposures (p = 0.810), with fluid losses of 32 (±12: hot), 16 (±6: temperate) and 59 ml (±17: cool; p = 0.133). The maximum urinary and evaporative fluid loss (118 ml) accounted for less than 0.3% of TBW, and was within the error of TBW measurement. Interstitial, extra- and intracellular fluid volumes were similarly relatively unaffected by air and skin temperature changes (p = 0.641, 0.417 and 0.589; Fig. 1).

During the initial 15 min in the cool, BV decreased by 166 ml (±63: cool; p = 0.051; Fig. 1), and continued so over the next 15 min (−302 ml (±76); p = 0.055). This was primarily attributable to PV adjustments, contracting by 144 ml (±53) during the first 15 min, and progressing to −205 ml (±60) by the end of the cool exposure (p = 0.040). In the heat, BV increased by 142 ml at 15 min, with PV expansions of 165 ml (±108; 15 min) and 108 ml (±123; 30 min; p = 0.020; Fig. 1). However, RCV remained relatively constant, regardless of air and skin temperatures (p = 0.447).

Discussion

The current data demonstrate that, when seated at rest with minimal T_{ac} change, intravascular volume may be modified by air temperature, and its affect upon skin temperature. It has been established that both profound skin heating and cooling, associated with large parallel T_c changes, produce haemoconcentration (Harrison et al. 1983). Nevertheless, BV shifts accompanying these skin treatments have not been established in the presence

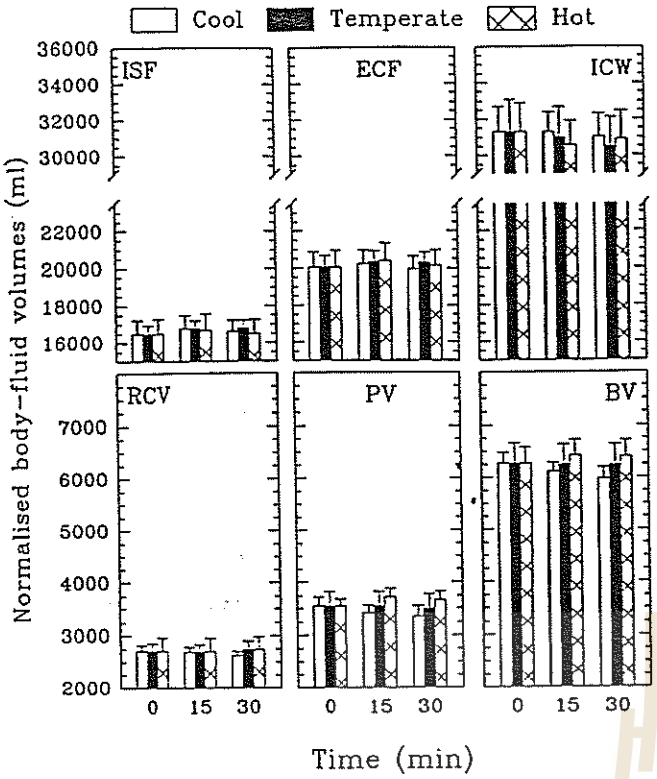

Fig. 1 Interstitial fluid (ISF), extracellular fluid (ECF), intracellular water (ICW), erythrocyte (RCV), plasma (PV) and blood (BV) volumes in cool, temperate and hot trials

of small, paradoxical T_c changes. Furthermore, in contrast to previous observations, the current data show that haemodilution, and not haemoconcentration, accompanies such heating. Using warm-water immersion (33°C), we have recently observed a similar haemodilution relative to the pre-immersed state, with T_c remaining fixed (Regan et al. 1997).

With these short-term skin treatments, cutaneous venomotor tone is the primary determinant of blood volume (Harrison 1985). Thus, in the skin cooling state, venoconstriction would increase capillary hydrostatic pressure, producing a nett filtration of plasma into the interstitium. The stability of RCV probably reflected a constant plasma tonicity, which Vogelaere et al. (1992) had shown to remain stable during prolonged cold stress, despite a significant PV reduction. The destination of this plasma is difficult to determine, as ISF, ECF and ICW remained relatively unchanged. Cold-induced diuresis was not apparent, and TBW was unaltered, so it is assumed this volume remained within the body. However, since a BV reduction of 302 ml equates with $<2\%$ of ISF and ECF, and $<1\%$ of ICW, such a change was within the volume measurement error (Maw et al. 1996). Given that non-significant increases in ICW occurred at both sampling points, while the ECF decreased, it may perhaps be inferred that the plasma filtrate moved through the interstitium to the extravascular cellular compartment, in response to altered venomotor tone.

During skin heating, fluid was again exchanged between the intravascular and extravascular spaces, although this time originating from both the intracellular and interstitial spaces, and probably in response to venodilation. Rapid venodilation would reduce capillary hydrostatic pressure, and induce an influx of interstitial fluid. However, such a shift was not reflected in ISF, ECF or ICW changes. It is unlikely that whole-body hydration was affected, due to the relatively short exposure. Indeed, evaporative water losses did not differ from those observed in the two cooler environments. It is therefore suggested that the present heat stress caused an iso-osmotic fluid shift into the blood, which expanded the PV, but did not affect either plasma tonicity or RCV.

It is concluded that short-term thermal exposures (30 min), in seated resting humans, can induce intravascular fluid shifts, even when T_c changes are ≤ 0.6°C. That is, plasma fluxes show considerable sensitivity to skin temperature manipulation. Since T_{ac} remained lower (heat) and higher (cool) than the control T_{ac}, it may be further concluded that such fluid shifts were primarily mediated by the affect of air temperature upon the skin, and its local affect on venomotor tone, rather than through hypothalamic control of cutaneous blood flow. In fact, the paradoxical changes in T_{ac} themselves may also be attributed to such cutaneous blood flow changes. While these fluid movements undoubtably involved both the intra- and extravascular compartments, they were sufficiently small so that they only appeared as significant changes within the smaller vascular space.

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COMPARISON OF THERMOREGULATORY RESPONSES OF THAI AND JAPANESE STUDENTS IN A HOT ENVIRONMENT

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INTRODUCTION

Many researchers have studied about the artificial heat acclimatization on Caucasian subjects in temperature climatic zones (1,2,3). But less study was conducted in natural acclimatization especially in non-Caucasian populations. The largest number of investigation of comparative nature have involved Negroids of African and Caucasions of European ancestery (4). Strydom and Wyndham (5) have done a study on Australian Aborigines and Sahara Arabs, while Ohara K et al. (6) studied on native Japanese and Caucasian. Duncan and Horvath (7) studied on tropical Asian races such as Malay, Indian, and Chinese. Although Thai and Japanese people are the same Asians, but Thailand is in the tropical zone whose temperature in the whole year is about 28-38°C and a relative humidity of 73-82.7%. Whereas Japan has four seasons with a whole year temperature is about 13.8°C and the relative humidity 71.9 % .However, in southern part of Japan such as Fukuoka city has the temperature about 6.4 -27.6°C and the relative humidity is 64 -76%. Less study about the thermoregulatory responses in ethnics differences in Asian populations. The purpose of this study was to investigate the thermoregulatory responses between Thai and Japanese people during heat exposure.

MATERIALS AND METHODS

Subjects

Eight young male Thai (TS) and eight young male Japanese (JS) students volunteered to participate in this experiment. All subjects were totally informed with regard to experimental risk and gave their written informed consent. They are allowed to do their daily activities freely. No food or water was ingested from at least 2 h before arrival at the laboratory until the end of the experiment. The physical characteristics of the subjects were shown in the Table 1.

Table 1. Physical characteristics of Thai and Japanese students.

Parameters	Thai students	Japanese students
Age (yr)	19.88±0.64	22.13±2.34
Height (cm)	172.25±4.40	165.77±5.72
Body weight (Kg)	58.4±4.57	60.19±9.99
BSA (m^2)	1.69±0.07	1.90±0.52
BSA /weight(cm^2/kg)	289.37±13.82	314±42.32
% Body fat	14.24±3.44	16.59±6.42

The data are mean±sd

Procedures

The experiments were set for the Thai students at a thermoregulation laboratory in Suranaree University of Technology, Nakhon Ratchasima, Thailand, while for the Japanese students at the thermoregulation laboratory at Kyushu Institute of Ergonomic Design, Fukuoka, Japan. We tried to set all of the parameters in the same condition in both two places. On the experimental day, the subjects wore the short pants without shirt and entered the climatic room having an ambient temperature of 30°C and the relative humidity of 70%. Then they rested by sitting on a chair for 10 minutes, and immersed their legs in a 42 ° C water-bath (the level of the water is up to knee) for 60 minutes. During the experiment, the rectal temperature (Tre) was monitored continuously by the thermistor probe, which was inserted 12 cm. above the anal sphincter. The skin temperature was measured with thermistors attached at seven sites (forehead, forearm, hand, trunk, thigh, leg, and foot) with surgical tape throughout the experiment. The mean skin temperature (Tsk) was calculated by using Hardy and DuBois's equation (8). The local sweat rate was collected at the forearm and the back sites by using an attached the sweat capsule, while the sweat was obtained by using a piece of filter paper having an area of 12.4 cm^2 at 20, 40 and 60 min, respectively. The heat-activated sweat gland was measured by iodine technique (9) at 20, 40, and 60 min, respectively. The tympanic temperature was also measured every 20 minutes. Thermal sensation and thermal comfort were measured every 20 minutes during heat exposure (10). The total sweat loss was calculated before and after the experiments from the amount of the sweat clothes (short pants) and body weight loss. Respiratory water loss was not considered. Water losses were not replaced until all procedures were completed. The skinfold thickness was measured using a caliper at the sites of triceps and subscapular. Body density was calculated from the skinfold thickness based on Nagamine and Suzuki (11), while Brozek's method(12) was used for calculating the percentage of body fat.

Statistics

All data are reported as mean± sd. One way analysis of variance was performed to assess one between-subject factor for the thermoregulation variables over the time course of the 60 min heat test. The differences were considered statistically significant when $p< 0.05$

RESULTS AND DISCUSSIONS

The physical characteristics of Thai and Japanese students were not significantly different as shown in Table 1. The delta rectal temperature of the TS group was significant higher than that of the JS group at the end of heat exposure. The mean skin temperature of the TS group was significant higher ($p<0.01$) than that of the JS group throughout the experiment as shown in Fig. 1.

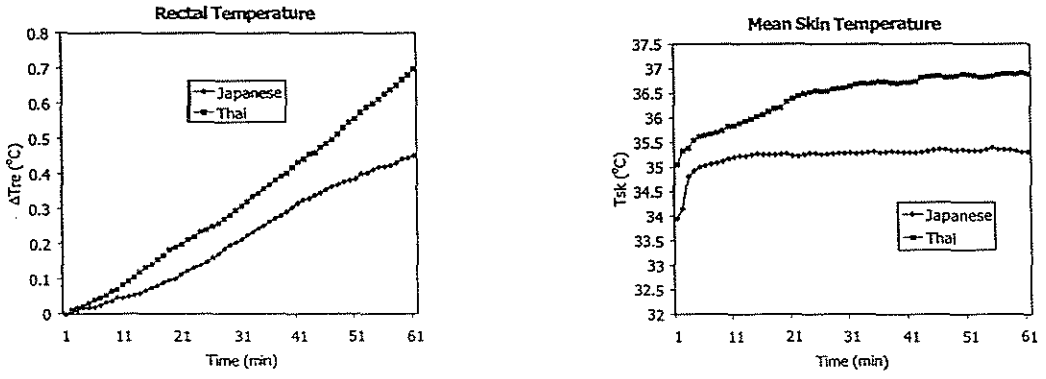

Figure 1. The delta rectal temperature and the mean skin temperature of Thai and Japanese students groups.

Table 2. The local sweat rate, the heat-activated sweat gland (HASG), and the sweat gland output (SGO) between the Thai students group (TS group) and the Japanese students group (JS group). The data are mean±sd.

Parameters	TS Group			JS Group		
	20min	40min	60min	20min	40min	60min
Local sweat rate ($mg.12.4cm^{-2}.20min^{-1}$)						
Forearm site	89.6±71.3	188.3±37.6	194.0±45.9	67.5±62.4	150.5±73.1	126.5±69.9*
Back site	125.2±51.4	194.3±35.7	212.0±36.8	98.3±75.6	157.5±66.6	165.4±81.3
HASG ($glands.cm^{-2}$)						
Forearm site	43.9±12.3	47.3±7.9	51.0±13.9	128.6±50.8**	143.0±45.0***	31.9±44.7**
Back site	40.1±14.0	42.5±11.8	37.5±11.9	87.5±31.8**	86.6±25.6 ***	81.3±27.3***
SGO ($\mu g.HASG^{-1}.min^{-1}$)						
Forearm site	8.6±7.4	16.4±4.3	16.0±5.1	3.5±4.9	4.8±3.2***	4.4±3.0***
Back site	14.0±6.5	19.7±6.5	24.8±9.0	5.5±4.2 ***	8.1±4.6 ***	9.3±6.0***

Significant differences between groups *p<0.05, **p<0.01, ***p<0.001

The total body weight loss of the TS group was increased (425.00±83.71 g) significantly ($p<0.05$) than that of the JS group (308.63±82.19 g). The local sweat rate of the TS group at the forearm site was also significantly higher than that of the JS group only at 60 min during heat exposure. Whereas this of the TS group at back site was not significantly different, but has a tendency of increasing more than that of the JS group. The heat-activated sweat gland of the TS group both forearm and back sites were significantly lower ($p<0.01$,$p<0.001$) than those of the JS group at 20,40, 60 min, respectively. The sweat gland output (SGO) of TS group at forearm and back site were significantly higher ($p<0.001$) than that of the JS group as shown in Table 2.

Matsumoto and Sugenoya, 1998 (13) found that the local sweat rate and the SGO at the forearm of the Thai subjects were lower than those of the Japanese subjects during immersion of their legs in a 43 °C water-bath with the ambient temperature was 26.6°C. These data were in contrast with our present experiment because first, the environmental condition and the time to exposure the heat stress were not the same. Our study was set at higher ambient temperature and the longer duration of experiment than that of the Matsumoto's experiment. Second, from the observation during the experiment, it seem that the sweat gland output of the Japanese students has a smaller size than that of the Thai students as we found that the SGO of Japanese group was significantly lower than that of Thai group. This was supported by the studies reported by Landing et al., 1968 (14), Sato and Sato, 1983 (15), and Falk et al,1992 (16).

In conclusions, the total body sweat loss in TS group was increased significantly than that of the JS group. Whereas the SGO of JS group has a smaller size than that of TS group. The mean skin temperature of TS group was higher than that of JS group. Eventhough Thai and Japanese are the same Asians, but the geographic of these countries are different. These may induces the thermoregulatory responses between these countries are different.

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2. นส.ศิริวรรณ โลกระโทก

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2. นายปณิก อวิรุทธการ

3. นส.ศมสรส มิ่งคำ

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2. นส.ณัฏฐา บูรณะเศรณี

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1. นายแพทย์นริศ วารณะวัฒน์
2. Miss Minako Takada

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1. นายบุญศักดิ์ หล่อพิพัฒน์

บริษัท ยูแฟม คลีนิค จำกัด

1. นส.นวลศรี กิตติเจริญฤกษ์

บริษัท หริกุล กรุ๊ป จำกัด

1. นายอนุชา การถาวร

****

ขอขอบคุณหน่วยงานที่สนับสนุนการจัดประชุมเชิงปฏิบัติการ
เรื่อง สมดุลอุณหภูมิกาย : บทบาทและการตอบสนองของระบบไหลเวียนโลหิตและระบบหายใจ
ณ ห้องประชุม 1 อาคารวิชาการ มหาวิทยาลัยเทคโนโลยีสุรนารี
23 - 25 เมษายน 2546

ทบวงมหาวิทยาลัย

MARATHON บริษัท มาราธอน (ประเทศไทย) จำกัด

ufam บริษัท ยูแฟม คลีนิค จำกัด

Harikul บริษัท หริกุล กรุ๊ป จำกัด

บริษัทยูแฟม คลีนิค จำกัด

"เทคโนโลยีเหนือราคา มุ่งพัฒนาสินค้าและบริการ"

ยูแฟม เป็นบริษัทชั้นนำด้านผลิตภัณฑ์ทางกายภาพบำบัด, กิจกรรมบำบัด, กายอุปกรณ์ และวิทยาศาสตร์การกีฬา

อาทิ เช่น : ลิฟท์เคลื่อนย้ายผู้ป่วย (Ceiling Hoist)

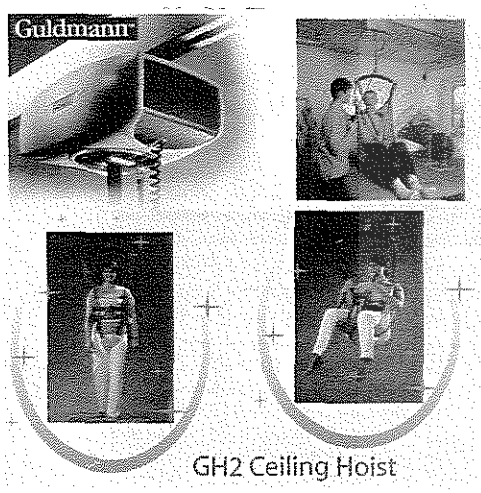
: เครื่องรักษาทางกายภาพบำบัดด้วยคลื่นอัลตราซาวด์ ผลิตภัณฑ์คุณภาพของ Chattanooga

: ห้องกระตุ้นพัฒนาการเด็กและระบบประสาท (snoezelen)

: เฝือกพลาสติก ผลิตภัณฑ์คุณภาพ ของ Orfit

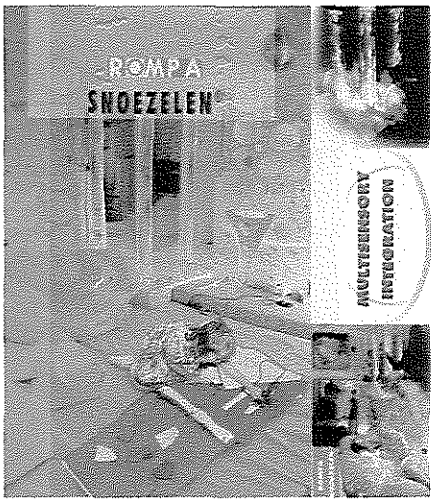
: อุปกรณ์ช่วยพยุงร่างกายและรองเท้าสุขภาพ (Orthopedice supports)

: ขาเทียมคอมพิวเตอร์, ขาเทียมกีฬา, ผลิตภัณฑ์คุณภาพของ Endolite

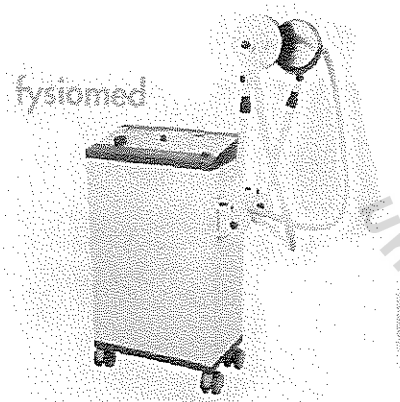


GH2 Ceiling Hoist

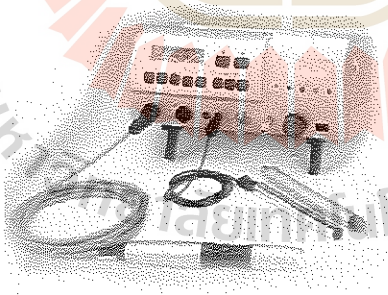
microwave diathermy model 25/p



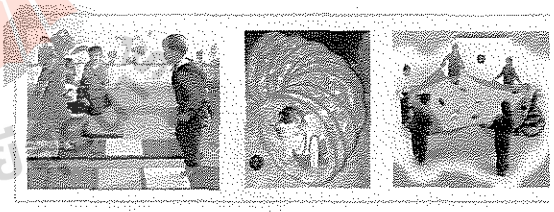
Multisensory Integration Room

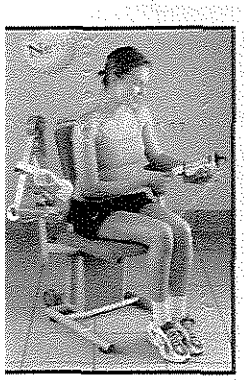


shortwave diathermy

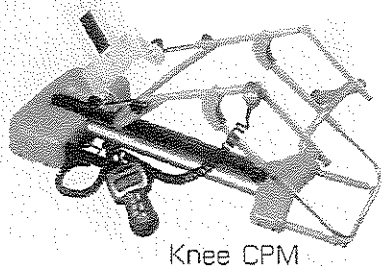


physiolaser olympic

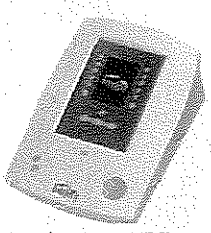




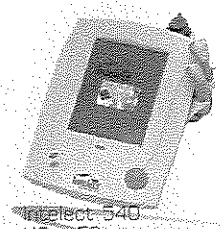
shoulder CPM

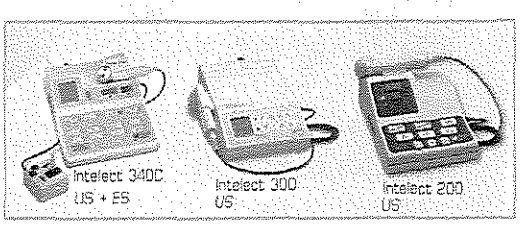


Knee CPM



Intelect 440 (ES)





ufam CLINIQUE บริษัท ยูแฟม คลีนิค จำกัด 1296/69-70 ถ.ประชาราษฎร์ บางซื่อ กทม. 10800 Tel. 02-911-2801-6 Fax. 02-911-4786

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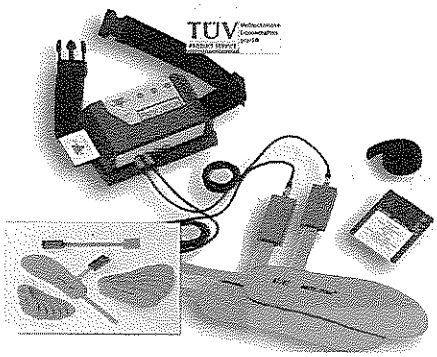
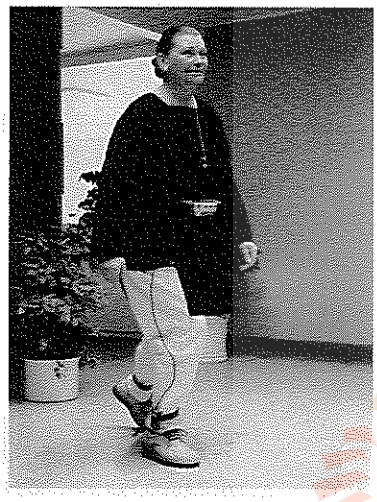
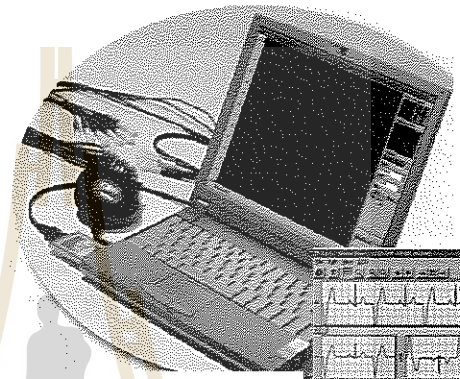
เครื่อง ISOKINETIC "CON-TREX MJ"



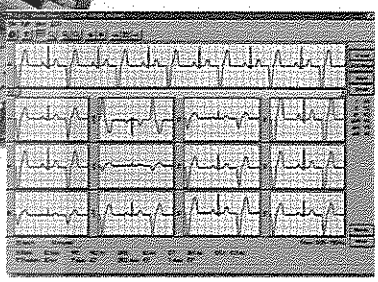


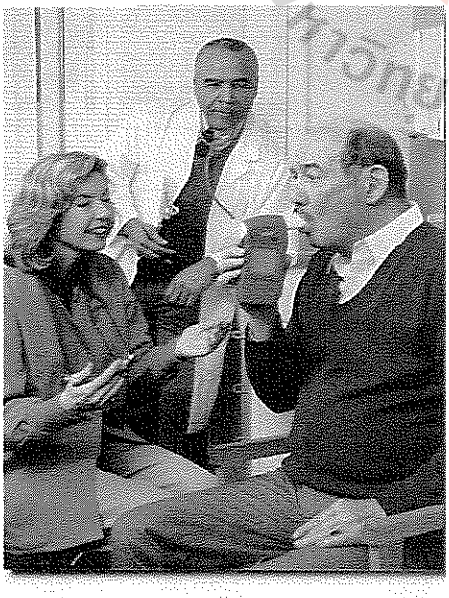
Load Distribution Measurement in Satic and Dynamic

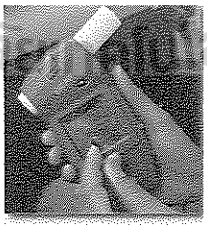
Parotec system

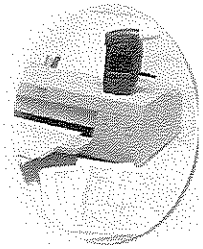


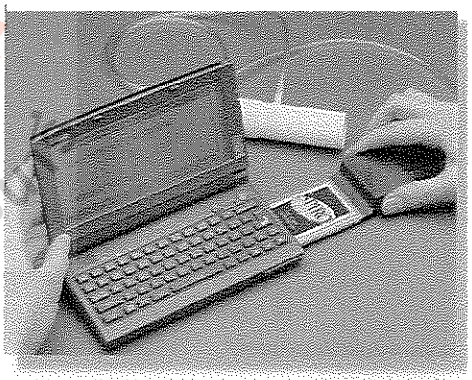
EKG card

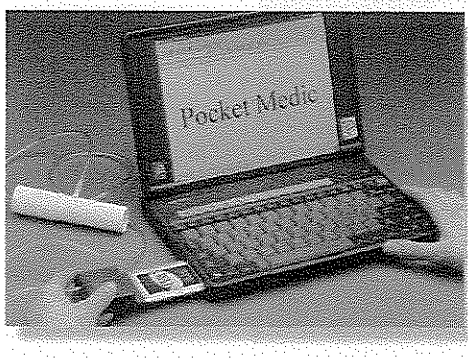












Handheld Spirometer model sensair

Spirometer and pulse oximeter model spiro