Eur J Appl Physiol (2000) 81: 159–162

SHORT COMMUNICATION

Graeme J. Maw · Ian L. Mackenzie · Nigel A.S. Taylor

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

Accepted: 12 July 1999

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

G.J. Maw
Department of Biomedical Science, University of Wollongong, Northfields Avenue, Wollongong, NSW 2522

I.L. Mackenzie
Haematology Department,
Illawarra Regional Hospital,
Wollongong, NSW 2500, Australia

N.A.S. Taylor (✉)
Department of Biomedical Science,
University of Wollongong,
Northfields Avenue,
Wollongong NSW 2522, Australia
e-mail: nigel_taylor@uow.edu.au
Tel.: 61-2-4221-3881; Fax: 61-2-4221-4096

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

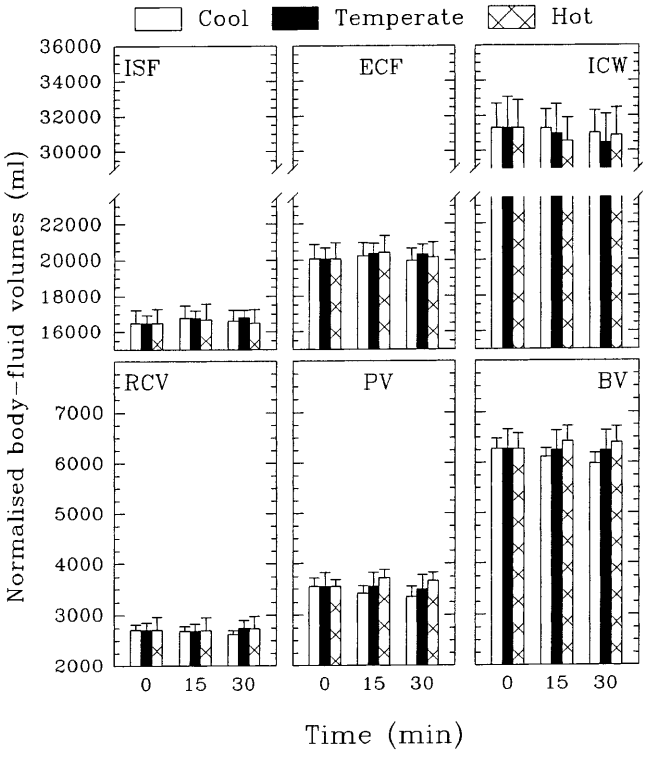

Fig. 1 Interstitial fluid (ISF), extracellular fluid (ECF), intracellular water (ICW), erythrocyte (RCV), plasma (PV) and blood (BV) volumes in cool, temperate and hot trials

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.

Acknowledgments This research was supported by grants from the Australian Research Council, and the Australian Institute of Nuclear Science and Engineering.

References

Bazett HC, Sunderman FW, Doupe J, Scott JC (1940) Climatic effects on the volume and composition of blood in man. Am J Physiol 129:69–83

Chien S, Gregersen MI (1962) Determination of body fluid volume. In Nastuk WL (ed), Physical Techniques in Biological Research, IV, Special Methods. Academic Press, New York, pp 1–105

Harrison MH (1985) Effects of thermal stress and exercise on blood volume in humans. Physiol Rev 65:149–209

Harrison MH, Edwards RJ (1976) Measurement of change in plasma volume during heat exposure and exercise. Aviat Space Environ Med 47:1038–1045

Harrison MH, Edwards RJ, Cochrane LA, Graveney MJ (1983) Blood volume and protein responses to skin heating and cooling in resting subjects. J Appl Physiol 54:515–523

Maw GJ, Mackenzie IL, Taylor NAS (1995) Redistribution of body fluids during postural manipulations. Acta Physiol Scand 155:157–163

Maw GJ, Mackenzie IL, Comer DAM, Taylor NAS (1996) Whole-body hyperhydration in endurance-trained males determined using radionuclide dilution. Med Sci Sports Exerc 28:1038–1044

Maw GJ, Mackenzie IL, Taylor NAS (1998) Body-fluid distribution during exercise in hot and cool environments. Acta Physiol Scand 163:297–304

Nose H, Mack GW, Shi X, Nadel ER (1988) Shift in body fluid compartments after dehydration in humans. J Appl Physiol 65:318–324

Regan JM, Patterson MJ, Hyde DE, Mittleman KD, Taylor NAS (1997) Comparison of human body-fluid responses during rest and exercise in warm and cold water. XXXIII International Congress of Physiological Sciences. St. Petersburg, Russia. p 164

Vogelaere P, Savourey G, Deklunder G, Lecroart J, Brasseur M, Bekaert S, Bittel J (1992) Reversal of cold induced haemoconcentration. Eur J Appl Physiol 64:244–249