

VASCULAR FLUID VOLUMES DURING POSTURAL, THERMAL AND EXERCISE STRESS: METHODOLOGICAL COMPARISONS.

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INTRODUCTION

Direct blood volume measurement, in clinical settings, involves the dilution of radiolabelled erythrocytes and albumin, to measure red cell (RCV) and plasma (PV) volumes. Evan's blue dye dilution is also used to quantify PV (1). However, neither PV technique permits reliable serial measurement, since both label albumin, which readily leaves the vascular space (2). Consequently, acute PV changes are often quantified indirectly, using mixed-venous haematocrit (Hct_v) and haemoglobin ([Hb]) changes (3). Chronic PV changes may be tracked by referencing these changes to a directly measured, control PV (1). To avoid problems associated with albumin loss, we use radioiodinated serum fibrinogen (RISF) to measure PV (4). In this paper, we compare RCV and PV changes measured using RISF, with values derived from Hct_v and [Hb] variations, during postural, thermal and exercise stresses.

MATERIALS and METHODS

Eight males (26.0 ± 3.8 y; 79.4 ± 8.3 kg) were tested in 3 resting postures (30 min each), and during cycling (50% maximal, temperate work rate) under 3 ambient conditions (balanced order). (i) Temperate (ambient temperature (T_a) 22.0 ± 1.0°C, relative humidity (rh) 52.0 ± 6.0%): seated, supine and standing postures, in a balanced order, and separated by, and finishing with, 30 min seated rest; 50 min cycling. (ii) Hot (T_a 36.2 ± 0.7°C, rh 44.0 ± 3.0%): 30 min seated; 50 min cycling. (iii) Cool (T_a 14.4 ± 1.6°C, rh 74.0 ± 9.0%): 30 min seated; 50 min cycling. In conditions (ii) and (iii), subjects were returned to temperate conditions between rest and exercise exposures, while maintaining a constant posture.

Ten-ml blood samples were collected at 10-min intervals (cycling), or at 15 and 30 min (all other exposures). Vascular fluid volumes were measured using radiochromated erythrocytes (8 μCi Na⁵¹Cr) and RISF (2 μCi ¹²⁵I) dilution (4), with blood volume (BV) the sum of RCV and PV. Whole-body haematocrit (Hct_w) was taken as the ratio of RCV to BV. Relative vascular volume changes (ΔBV, ΔRCV, and ΔPV) were calculated, and compared with indirectly determined volume changes using Hct_v and [Hb] (Coulter Electronics, S-Plus IV; after 3). Volume differences were compared using ANOVA (Tukey's_{WSD} *post hoc*). Data are reported as means with standard errors of the means.

RESULTS

Basal seated RCV, PV and BV averaged 2627 (± 80), 3673 (± 121) and 6348 (± 184) ml respectively. The corresponding Hct_w was 0.418 (± 0.007), compared to Hct_v of 0.446 (± 0.003): basal f -ratio ($Hct_w:Hct_v$) was 0.939 (± 0.015). During supine rest, BV expanded ($+89 \pm 82$ ml), primarily due to a PV increase ($+52 \pm 70$ ml), while both BV and PV contracted during upright rest (-406 ± 89 ml and -233 ± 64 ml). The relative changes in BV, RCV and PV were not significantly different between techniques (Table 1), and the f -ratio remained consistent across postures ($p=0.09$).

Table 1: Red cell (ΔRCV), plasma (ΔPV) and blood volume changes (ΔBV).

Task	Method	Condition	ΔRCV	ΔPV	ΔBV
supine	A	temperate	+1.8%	+1.1%	+1.5%
	B		-0.1%	+3.7%	+2.0%
seated	A	temperate	+2.1%	-1.5%	0.0%
	B		+0.1%	-0.9%	-1.0%
standing	A	temperate	-5.7%	-6.0%	-6.0%
	B		-0.9%	-7.4%	-4.5%
seated	A	hot	+0.7%	+3.4%	+2.2%
	B		-0.1%	+2.5%	+1.2%
seated	A	temperate	+1.1%	-1.8%	-0.6%
	B		0.0%	-2.1%	-1.2%
seated	A	cool	-3.5%	-6.2%	-5.0%
	B		-0.6%	-7.3%	-4.3%

A = radionuclide dilution; B = Hct_v and [Hb].

The seated heat exposure, increased BV and PV ($+124 \pm 150$ and $+108 \pm 123$ ml), in contrast to decreases observed in the cool (-302 ± 76 and -205 ± 60 ml). The between-method vascular volume changes (Table 1) were again non-significant, and the f -ratio remained constant across air temperatures ($p=0.55$).

Exercise produced similar BV and RCV changes between methods, regardless of T_a or duration ($p=0.48$ and $p=0.82$). Both BV and RCV decreased during the first 20 min ($-534 (\pm 169)$: hot), $-136 (\pm 98)$: temperate) and $-297 (\pm 66)$: cool) ml, for BV; -114 ± 62 , -76 ± 55 and -75 ± 37 ml respectively for RCV). ΔBV averaged -7.7% , -1.9% and -5.0% , while ΔRCV averaged -3.3% , -2.5% and -2.7% , respectively. The corresponding indirect ΔBV s were -7.3% , -2.4% and -3.6% , while ΔRCV s averaged -0.6% , -0.4% and $+0.1\%$. In contrast, indirect ΔPV s significantly exceeded directly measured ΔPV s during the initial 10 min in all environments

($p=0.02$; Figure 1A). PV decreased $-356 (\pm 128$: hot), $-110 (\pm 46$: temperate) and $-243 (\pm 42$: cool) ml during this time, equating to losses of -9.4% , -3.0% and -7.3% . The indirect ΔPV s were in the same direction, but were larger: -11.8% , -6.3% and -9.0% , respectively. This methodological discrepancy coincided with an f -ratio change, which, when averaged across air temperatures, decreased to $0.916 (\pm 0.019)$ after 10 min ($p=0.01$), then recovered towards baseline (Figure 1B).

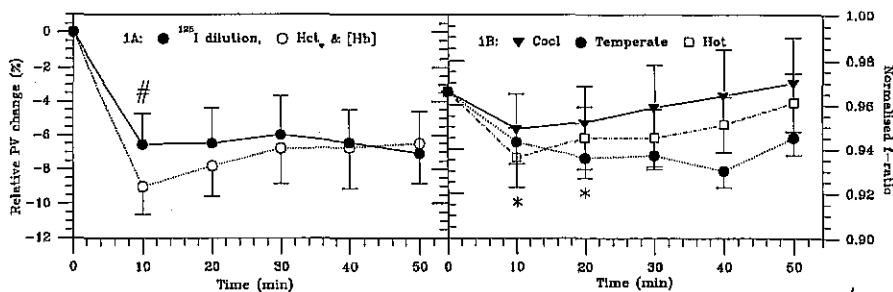


Figure 1: Changes in plasma volume (PV: 1A), averaged across hot, temperate and cool conditions (radionuclide dilution versus mixed-venous haematocrit (Hct_v) and haemoglobin concentration ([Hb])), and the f -ratio (1B) when cycling in hot (36°C), temperate (22°C) and cool (14°C) conditions (normalised to pre-exercise level: # = significant difference between methods, * = different from time zero).

DISCUSSION

The current observations confirm the dependence of vascular volumes on posture, T_a and exercise state. Vascular volume changes with posture were considered to reflect hydrostatic pressure modification in dependent vascular beds. Changes in Hct_v and [Hb] closely tracked directly measured variations in PV and BV, indicating the indirect method provides a valid means for determining vascular fluid shifts under these conditions.

Vascular volumes decreased during cool stress and increased in the heat. These changes were dominated by plasma shifts, and were probably related to changes in capillary hydrostatic pressure, which increases during cold stress (cutaneous vasoconstriction), and decreases in the heat, when venodilation enables an influx of interstitial fluid (5). Irrespective of transcapillary fluid exchange, the resultant Hct_v and [Hb] changes provided valid determinations of vascular fluid changes during mild thermal stress at rest.

Regardless of T_a , cycling apparently increased capillary filtration, drawing plasma across capillary membranes. At exercise onset, Hct_v changes exaggerated this

filtration, producing a greater apparent reduction in PV than observed using RISF (Figure 1A). This discrepancy could be attributed to the Coulter counter method, which, in the presence of appreciable cell dehydration, has been shown to underestimate red cell haemoglobin concentration, relative to that obtained from spun samples (6). While direct [Hb] determination and centrifugation would eliminate this possibility, we contend that the reduction in cell water content required to invalidate this method ($\sim 40\%$; 6), would not have occurred within the first 10 min, across the three environments. Indeed, RCV decrements during the first 20 min were less than 3%. Therefore, we ascribe this methodological variance to concurrent changes in the relationship between Hct_s and Hct_w (f -ratio; Figure 1B). This disparity leads to uncertainty regarding the validity of indirect ΔPV measurement during the initial period of cycling, regardless of air temperature. It is possible that cycling transiently changed blood flow dynamics, with axial erythrocyte flow increasing more than plasma flow, artificially increasing Hct_s , such that acute Hct_s changes no longer reflected Hct_w . As such differences were not apparent during postural and resting thermal stress, this trend was possibly exercise-dependent. Thus, while Hct_s and [Hb] changes do not reflect true ΔPV at cycling onset, they do provide valid indices of fluid shifts during acute postural or thermal stress, and during prolonged cycling.

REFERENCES

1. Greenleaf, J.E., Convertino, V.A., and Mangseth, G.R. 1979, Plasma volume during stress in man: osmolality and red cell volume. *Journal of Applied Physiology*, **47**, 1031-1038.
2. Bent-Hansen, L. 1989, Initial plasma disappearance and distribution volume of [^{131}I]albumin and [^{125}I] fibrinogen in man. *Acta Physiologica Scandinavica*, **136**, 455-461.
3. Dill, D.B., and Costill, D.L. 1974, Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. *Journal of Applied Physiology*, **37**, 247-248.
4. Maw, G.J., Mackenzie, I.L., Comer, D.A.M., and Taylor, N.A.S. 1994, Whole body hyperhydration in endurance-trained males determined using radionuclide dilution. *Medicine and Science in Sports and Exercise*, **28**(8), in press.
5. Harrison, M.H. 1985, Effects of thermal stress and exercise on blood volume in humans. *Physiological Reviews*, **65**, 149-209.
6. Mohandas, N., Clark, M.R., Kissinger, S., Bayer, C., and Shobet, S.B. 1980, Inaccuracies associated with the automated measurement of mean cell hemoglobin concentration in dehydrated cells. *Blood*, **56**, 125-128.