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 dilutionis 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 quantifiedindirectly,using mixed-venoushaematocrit (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 $\pm$3.8 y; 79.4 $\pm$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 $\pm$ 1.0°C, relative humidity (rh) 52.0 $\pm$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 $\pm$0.7°C, rh 44.0 $\pm$3.0%): 30 **min** seated; 50 mincycling. (iii) Cool (T_a 14.4 $\pm$1.6°C, rh 74.0 $\pm$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 $Na^{51}Cr$) and RISF (2 μCi ^{125}I) 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 (ABV, ARCV, and APV) 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 (f 184) **ml** respectively. The corresponding Hct_w was 0.418 (±0.007), compared to Hct_v of 0.446 (f0.003): basal *f*-ratio (Hct_w:Hct_v) was 0.939 (f0.015). During supine rest, **BV** expanded (+89 ±82 **ml),** primarily due to a **PV** increase (+52 f70 **ml),** while both **BV** and **PV** contracted during upright rest (-406 f89 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 **(ARCV),** plasma **(APV)** and blood volume changes **(ABV).**

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 ± 150 and + 108 ± 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 (±169: hot), -136 (±98: temperate) and -297 (±66: cool) ml, for **BV;** -114 ±62, -76 f55 and -75 f37 **ml** respectively for **RCV). ABV** averaged -7.7%, -1.9% and **-5.0%,** while **ARCV** averaged -3.3%, -2.5% and -2.7%, respectively. The corresponding indirect ΔBVs were -7.3%, -2.4% and **-3.6%,** while ΔRCVs averaged -0.6%, -0.4% and +0.1%. **In** contrast, indirect **APVs** significantly exceeded directly measured **APVs** during the initial 10 **min** in all environments

($p=0.02$; Figure 1A). PV decreased -356 (±128: hot), -110 (±46: temperate) and -243 (±42: cool) **ml** during this time, equating to losses of -9.4%, -3.0% and -7.3%. The indirect APVs 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 (±0.019) after 10 min ($p=0.01$), then recovered towards baseline (Figure 1B).

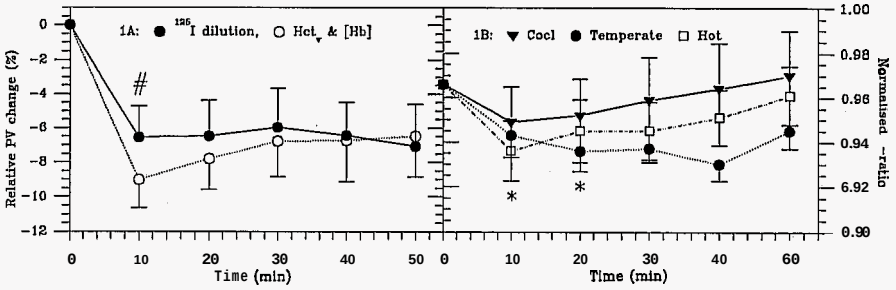

Figure 1: Changes in plasma volume (PV: 1A), averaged across hot, temperate and cool **conditions (radionuclide dilution versus mixed-venous haematocrit (Hct_v) and haemoglobin concentration ([Hb])), and they-ratio (1B) when cycling in hot (36°C), temperate (22°C) and cool (14°C) conditions (normalised 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 venoconstriction), 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 (−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_v and Hct_w (f-ratio; Figure 1B). This disparity leads to uncertainty regarding the validity of indirect APV 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_v, such that acute Hct_v 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_v and [Hb] changes do not reflect true APV at cycling onset, they do provide valid indices of fluid shifts during acute postural or thermal stress, and during prolonged cycling.

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