

INFLUENCE OF BLOOD VOLUME ON BAROREFLEX CONTROL OF FOREARM VASCULAR RESISTANCE

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INTRODUCTION

During pooling of blood in the legs, arterial blood pressure is maintained by cardio-acceleration and constriction of splanchnic and cutaneous vascular beds. Cardioacceleration and the gain of the baroreflex control of forearm vascular resistance are both attenuated in physically fit individuals (1,2). The increased blood volume (BV) accompanying training has been suggested as a causal mechanism (2). This study investigated that hypothesis by measuring the gain of the cardiopulmonary baroreflex control of FVR during progressive lower body negative pressure (LBNP) before and after expansion of the BV by infusion.

METHOD

Six healthy, moderately active, male student volunteers (aged 20-28 years) were subjected to venous pooling by progressive LBNP (0 to -201 mmHg). All experiments were carried out in an environmental chamber at 30°C and low humidity and with the subject in a post-absorptive state. Subjects were asked to refrain from vigorous prior exercise on the day of each experiment.

Central venous pressure (CVP) was approximated by the measurement of peripheral venous pressure (PVP) in the antecubital vein of the dependent right arm, using the method of Gauer and Sieker (2,3). Arterial measures were taken with the subject supine. Forearm blood flow (FBF) was measured by venous occlusion plethysmography, using a Whitney mercury-in-Silastic strain gauge on the left forearm with the hand circulation occluded at the wrist. Heart rate was calculated from an electrocardiograph. Arterial blood pressures were determined by the oscillometric technique. Mean arterial pressure (MAP) was calculated as diastolic + $\frac{1}{3}$ pulse pressure. FVR was calculated by dividing MAP (mmHg) by FBF ($\text{ml} \cdot \text{min}^{-1} \cdot 100 \text{ml}^{-1}$); these values were expressed as resistance units (U). The gain of the baroreflex control of FVR was defined as the coefficient of regression of FVR on PVP.

BV was then expanded by approximately 10% by the drip infusion of 8g per kg bodyweight of 5% serum albumin in Ringer's solution and the measurements were repeated.

The main effects of blood volume and LBNP levels were analysed by two-way ANOVA with post-hoc analysis of specific differences by contrasts using the SYSTAT program. Regression slopes were tested by a one-tailed paired t-test. The 0.05 level of significance was accepted.

RESULTS

There was no significant effect of either LBNP or its interaction with BV on any of the arterial measures of blood pressure or heart rate. After BV expansion, there were no changes in heart rate or diastolic blood pressure, but there were significant increases in systolic (7 ± 2 mmHg) and pulse pressures (7 ± 2 mmHg) (mean \pm SE). FBF was progressively reduced during LBNP from 3.8 ± 0.7 at

ambient pressure to $2.1 \pm 0.6 \text{ ml}\cdot\text{min}^{-1}\cdot 100\text{ml}^{-1}$ at -20mmHg , but, after BV expansion, was higher at all levels of LBNP by $1.0 \pm 0.5 \text{ ml}\cdot\text{min}^{-1}\cdot 100\text{ml}^{-1}$ ($p < 0.05$). A corresponding progressive increase with LBNP occurred in FVR from 24 ± 4 to 63 ± 23 units. During hypervolemia, this dropped to 20 ± 3 to 37 ± 10 units ($p < 0.05$).

PVP, like FBF, was progressively reduced during LBNP, from 7.2 ± 0.9 to $2.0 \pm 0.6 \text{ mmHg}$ and after BV expansion was likewise higher at all levels of LBNP by $1.4 \pm 0.6 \text{ mmHg}$ ($p < 0.05$).

The coefficient of regression of mean FVR on PVP, representing the gain of the baroreflex control of FVR, was reduced from -7.0 to $-2.9 \text{ U}\cdot\text{mmHg}^{-1}$ by hypervolemia ($p < 0.05$) (Fig. 1). Five of the six subjects followed the same pattern, but one had an increase in regression coefficient from -0.6 (control) to -2.2 during hypervolemia. On a previous control test his coefficient was -4.7 .

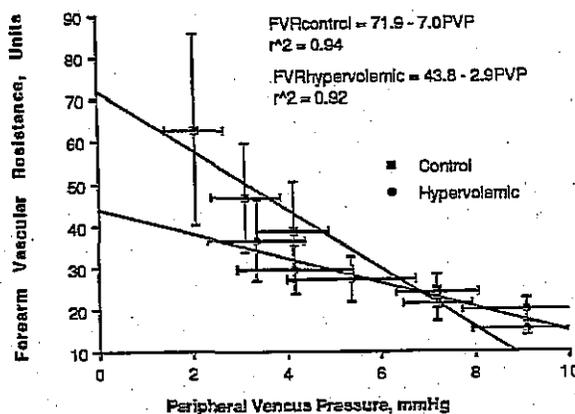


Figure 1. Hypervolemic attenuation of baroreflex control of FVR ($n=6$).

CONCLUSIONS

After BV expansion, PVP and FBF increased significantly with no change in sensitivity to LBNP. FVR was reduced significantly during acute hypervolemia, although the lack of interaction between BV and LBNP effects ($p = 0.815$) suggested no significant change in its sensitivity to LBNP. The coefficient of linear regression of FVR on PVP, the gain of the baroreflex, was significantly reduced by acute hypervolemia, consistent with the hypothesis that the attenuation of the baroreflex seen in fit subjects may be the result of increased blood volume, but a short-term, rather than a long-term effect.

REFERENCES

1. Raven, P.B., Rohm-Young, D. and Blomqvist, C.G. Physical fitness and cardiovascular response to lower body negative pressure. *J. Appl. Physiol.* **56**: 138-144, 1984.
2. Mack, G.W., Shi, X., Nose, H., Tripathi, A. and Nadel, E. Diminished baroreflex control of forearm vascular resistance in physically fit humans. *J. Appl. Physiol.* **63**(1): 105-110, 1987.
3. Gauer, O.H. and Sieker, H.D. The continuous recording of central venous pressure changes from an arm vein. *Circ. Res.* **4**: 74-78, 1956.