

# Effects of prolonged bed rest on the cardiopulmonary response to postural changes in humans.

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## Abstract

In order to gain a better understanding of the cardio-pulmonary impairment induced by prolonged bed rest, we measured steady-state oxygen uptake ( $\dot{V}O_2$ ) and carbon dioxide output ( $\dot{V}CO_2$ ), by Grønlund's algorithm on a breath-by-breath basis, cardiac output ( $Q'$ ), by open circuit acetylene washout, heart rate (HR), by electrocardiography, and mean arterial pressure (MAP), by fingertip plethysmography, on 18 male subjects ( $33.1 \text{ years} \pm 0.9$ ;  $71.1 \text{ kg} \pm 1.1$ ;  $1.75 \text{ m} \pm 0.01$ ) before (BB) and after (AB) 90 days of head down tilt bed rest, in the following conditions : upright and supine posture, both at rest and at the 50 W exercise. The stroke volume (SV) was calculated as the ratio of  $Q'$  to HR. The total peripheral resistance (TPR) was calculated as the ratio of MAP to  $Q'$ .  $\dot{V}O_2$  was lower ( $p < 0.01$ ) supine than upright ( $0.43 \text{ l/min}$  vs  $0.52 \text{ l/min}$  at rest;  $1.06 \text{ l/min}$  vs  $1.27 \text{ l/min}$  at 50W) in BB. In AB,  $\dot{V}O_2$  was  $0.81 \text{ l/min}$  at rest and  $1.61 \text{ l/min}$  at 50W upright, whereas supine it was  $0.74 \text{ l/min}$  at rest and  $1.54 \text{ l/min}$  at 50W. All the values in AB were significantly higher than the corresponding values in BB.  $\dot{V}CO_2$  followed the same patterns as  $\dot{V}O_2$ , so that the gas exchange ratio was the same in all conditions. In BB,  $Q'$  was the same supine and upright. In AB,  $Q'$  was significantly higher supine than upright at rest ( $6.11 \text{ l/min}$  vs  $5.40 \text{ l/min}$ ). All  $Q'$  values observed in AB were significantly higher than the corresponding values in BB (rest:  $5.40$  vs  $4.50$  upright;  $6.11$  vs  $5.05$  supine. 50 W exercise  $8.26$  vs  $6.99$  upright and  $9.18$  vs  $7.38$  supine). SV was higher supine than upright ( $71.2 \text{ ml}$  vs  $51.2 \text{ ml}$  BB,  $78.6$  vs  $55.4 \text{ ml}$  AB at rest;  $76.9 \text{ ml}$  vs  $67.2 \text{ ml}$  BB,  $87.5 \text{ ml}$  vs  $70.8 \text{ ml}$  AB at 50W). HR was lower supine ( $70.3 \text{ bpm}$  at rest,  $95.2$  at 50W) than upright ( $87.8 \text{ bpm}$  at rest,  $104.7$  at 50 W) in BB, and was ( $77.3 \text{ bpm}$  at rest,  $103.9$  at 50W) and ( $97.7 \text{ bpm}$  at rest,  $116.8$  at 50 W) in AB. SV was the same in AB as in BB, whereas HR was higher in AB than in BB. MAP was lower supine than upright in AB ( $94.3 \text{ mmHg}$  vs  $105.4 \text{ mmHg}$  at rest;  $101.6 \text{ mmHg}$  vs  $115.4 \text{ mmHg}$  at 50W). In supine posture, MAP was lower in AB than in BB, both at rest and at exercise. TPR was lower supine than upright in AB ( $17.8 \text{ mmHg} \cdot \text{min/l}$  vs  $21.6 \text{ mmHg} \cdot \text{min/l}$  at rest;  $12.8 \text{ mmHg} \cdot \text{min/l}$  vs  $16 \text{ mmHg} \cdot \text{min/l}$  at 50W). In supine posture, TPR was lower in AB than in BB, both at rest and at exercise. These results suggest that the cardio-pulmonary system adjusts to reduced blood volume and increased venous compliance essentially by increasing HR, perhaps modulated by increase in sympathetic output tone. The HR increase is larger than the drop in SV, so that  $Q$  is increased in AB. This is coherent with the higher  $\dot{V}O_2$  levels in AB than in BB at the same power. The  $\dot{V}O_2$  increase could be mainly due to an impairment of the motor control system after 90 days of bed rest.