INFLUENCE OF GRAVITY-INDUCED VENOUS RETURN CHANGES: STROKE VOLUME DURING APNEA AND EXERCISE

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When an exercise workload increase is combined with breath-hold, a bradycardia occurs after 10 to 15 s of regular increase in heart rate (HR), in response to exercise. In parallel, mean arterial blood pressure (MAP) increases until the end of breath-hold. The understanding of this physiological response is of interest for sporting activities when breathing is restricted, such as competitive swimming and free diving. The effect of venous return is an important factor in the HR/MAP reactions which was not studied previously. The purpose of this study was to determine how changes in venous return, during gravity changes of parabolic flight, affect stroke volume (SV) during exercise with breath-hold as compared to constant 1 g condition.

Methods:
The experiments consisted of three treatments conducted at 1 g on 13 subjects and during parabolic flight (ESA, campaign 41) on 6 subjects. The treatments were: 1) free-breathing/30W cycle ergometer (loP), 2) breath-hold/200W cycle ergometer (BH-hiP) and 3) free-breathing/200W cycle ergometer (hiP). Each treatment was repeated 5 times on each subject and followed a standardized protocol of: 80 s pedalling at 30 W, treatment, and then 80 s of recovery without pedalling. Breath-hold was performed after a deep inspiration. SV, MAP, and HR were measured beat to beat with the Task Force (R) Monitor (CNSystems, Austria).

Results and discussion:
The time courses for changes in MAP and HR during BH-hiP under 1 g are consistent with previous findings: an initial decrease of MAP for approximately 10 s is followed by an increase until the end of the treatment, while HR initially increases for 10 to 15 s then decreases until the end of treatment. A general influence of the parabola (1 g – hyper-g – 0 g – hyper-g 1 g) was seen on all three parameters (MAP, HR, SV) in all three treatments.

The effect of breath-hold on HR and MAP was identical for 1 g and during changes of gravity. In 1 g SV decreased in hiP for approx. 10 ml, whereas for BH-hiP a slightly increased during the treatment (5 ml). In the parabola the expected decrease during hyper-g was found (5 to 10 ml). After the change to 0 g SV in hiP and loP increased (30 ml) with a peak after 10 s, whereas in BH-hiP SV demonstrated a less pronounced and delayed increase during the treatment and 0 g (20 ml).

One explanation of the change in SV during breath-hold could be related to changes in the intra-pleural pressure. Holding the inspiratory position in 1 g creates a significant under-pressure in the thorax. In 0 g this inspiratory position creates less significant under-pressure. The reduced pressure gradient in combination with the absence of respiratory pump and the absence of gravity may explain the different effect of breath-hold on SV. These results confirm the significant role of respiratory activity onto SV.

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