NONINVASIVE BEAT-TO-BEAT STROKE VOLUME COMPUTATION DURING ACUTE HYDROSTATIC PRESSURE CHANGES IN PARABOLIC FLIGHT

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ABSTRACT

A three-element model of the cardiovascular system was used to monitor stroke volume (SV) changes during parabolic flight. Aortic blood flow was estimated from continuous arterial finger pressure and SV computed by integrating simulated aortic flow during each systole. SV was significantly higher in microgravity (μ G) compared to 1G whereas in hypergravity (hG), SV was significantly lower. Exponential SV transients were observed after the transitions to and from μ G and the succeeding or preceeding hG phases. These SV transients present different time constants, which reflect two different mechanisms of cardiovascular adaptation to sudden gravitational changes.

These results show that beat-to-beat computation of SV provides noninvasive information on circulatory adaptation to acute hydrostatic pressure changes.

1. INTRODUCTION

We applied a noninvasive technique to estimate SV in order to study the mechanisms involved in SV changes induced by sudden alterations in vertical acceleration (G_z). Parabolic flight causes these sudden G_z changes, exposing subjects to short periods of μG (~20s), preceded and followed by short periods of hG (<10s).

The suppression of all hydrostatic pressure gradients during μG results in blood being shifted to the upper parts of the body and in an increased venous return, inducing a rise in stroke volume. In contrast, during hG, blood is pulled to the lower limbs, venous return is impaired and SV is reduced.

2. METHODS

2.1 Protocol and data

Measurements were performed on five young healthy male subjects (mean \pm SD: age 31.8 \pm 7.4 yr, weight 72.2 \pm 4.5 kg, height 179.4 \pm 4.3 cm) during ESA's 29th parabolic flight campaign.

Data presented here was acquired during the last 5 parabolas out of 15 overall recordings for each subject. During these last 5 parabola the subject was passively standing on a platform making a 60° angle with the aeroplane's floor and breathing at a paced rhythm of 0.25Hz (15 breath/min).

Continuous, noninvasive finger pressure (Finapres) and G_z were recorded at a sampling frequency of 1 kHz. Intermittent measures of mean, diastolic and systolic brachial blood pressure were performed between parabolas for calibration of the continuous finger pressure readings.

2.2 Model

Aortic blood flow was computed from continuous arterial pressure by using a nonlinear, time varying, three-element model of the cardiovascular system [1]. Elements of the model represent the characteristic impedance of the aorta, arterial compliance and systemic vascular resistance (Fig. 1). Values for the parameter elements were computed from published pressure-area relationship [2].

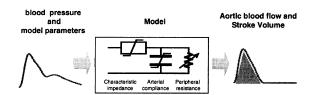


Fig. 1: Three-element SV computation model.

Beat-to-beat SV was computed by integrating simulated aortic flow during each systole (Fig. 2). SV data was normalized by the individual mean SV value in 1g preceding the parabola.

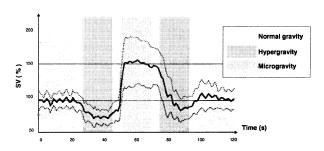


Fig. 2. Computed SV (mean \pm SD, normalized by individual 1g values) for all parabola of one subject.

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2.4 Exponential time constant

In order estimate the time constants of SV changes induced by the sudden variations of G_z , two exponential functions (Eq. 1-2) were fitted to the SV transients (ΔSV) induced by G_z changes:

$$\Delta SV = K \cdot \left(1 - \exp\left(-\frac{t}{T_1}\right) \right) \tag{1}$$

corresponding to the transition from hG to µG, and:

$$\Delta SV = K \cdot \left(\exp \left(-\frac{t}{T_2} \right) \right) \tag{2}$$

for the transition from μG into hg.

In these equations t is time, T_1 and T_2 are the time constants associated with SV transient, and K is a constant (Fig. 3).

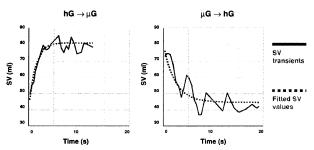


Fig. 3. Exponential function fitting to SV transients.

2.5 Statistics

Unbalanced analysis of variance with the number of parabola and subject as factors of variability and Bonferroni adjustment for multiple comparisons with significance level at P < 0.05 (*) was performed.

3. RESULTS

Exponential SV transients were observed during the parabolic flight manoeuvre. At the beginning of μG , SV increased and reached a stationary state during the remaining μG phase.

The SV response to the increase in G_z level after the parabola was observed to be delayed by approximately 5 heartbeats. SV was significantly higher in μG compared to 1G whereas SV in hG was significantly lower (Table 1).

Computation of T_1 and T_2 showed that for all subjects SV transients were significantly shorter in transitions leading to μG than in transitions from μG to hG (Table 2).

Table 1: SV before, during and after the parabola compared to 1G.

G _z level	SV (%)
1G	100
HG (before parabola)	- 78.85 *
μG	+ 170.60 *
HG (after parabola)	+ 101.17

Table 2: Time constants T₁ and T₂ fitted to the SV transients.

Time Constant	Mean ± SE (s)
T_1	2.97 ± 0.50
T ₂	7.77 ± 0.44 *

4. DISCUSSION

Differences between T_1 and T_2 suggest that distinct regulation mechanisms are responsible for the adaptation of SV to sudden G_z transitions. After the onset of μG the increasing pulmonary arterial pressure might slow the blood transfer to the pulmonary circulation, whereas the reduction in SV during hG might reflect the unload of the pulmonary vascular system.

These results show that beat-to-beat computation of SV, based on a three-element model of the cardiovascular system, provides noninvasive information on circulatory adaptation to acute hydrostatic pressure changes.

5. REFERENCES

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