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 (µG) compared to 1G whereas in hypergravity (hG), SV was significantly lower. Exponential SV transients were observed after the transitions to and from µG and the succeeding or preceding 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 (Gz). Parabolic flight causes these sudden Gz 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 ± SD: age 31.8 ± 7.4 yr, weight 72.2 ± 4.5 kg, height 179.4 ± 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 Gz 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].
2.4 Exponential time constant

In order to 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 ($\Delta SV$) induced by $G_z$ changes:

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

(1)

corresponding to the transition from hG to µG, and:

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

(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).

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

<table>
<thead>
<tr>
<th>$G_z$ level</th>
<th>SV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1G</td>
<td>100</td>
</tr>
<tr>
<td>HG (before parabola)</td>
<td>-78.85 *</td>
</tr>
<tr>
<td>µG</td>
<td>+170.60 *</td>
</tr>
<tr>
<td>HG (after parabola)</td>
<td>+101.17</td>
</tr>
</tbody>
</table>

Table 2: Time constants $T_1$ and $T_2$ fitted to the SV transients.

<table>
<thead>
<tr>
<th>Time Constant</th>
<th>Mean ± SE (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_1$</td>
<td>2.97 ± 0.50</td>
</tr>
<tr>
<td>$T_2$</td>
<td>7.77 ± 0.44 *</td>
</tr>
</tbody>
</table>

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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