A BLOOD VOLUME MODEL FOR THE SIMULATED SHORT TERM RESPONSE TO HEAD-UP-TILT

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MOTIVATION

For a human assuming upright posture (orthostasis), blood tends to shift toward the feet. This reduces the heart filling pressure, cardiac output (CO), and blood pressure (BP), which reflexes attempt to restore to its normal value of approximately 100 mmHg [1]. Our earlier study of short term BP regulation predicted non-physiological magnitudes and time scales for the BP transients [2]. We attribute this to problems with the model of blood volume distribution. Here, we identify key volume related parameters, and formulate a simulation that predicts them, in the absence of reflex control effects.

BACKGROUND

Physiology

The mean circulatory filling pressure (MCFP) is a measure of blood volume. Blood volume, as a function of MCFP gives the whole body compliance, which is almost linear [3], see Table 1.

The blood is divided between arterial and venous, and stressed and unstressed (45 ml/kg [4]) volumes. Also, the tissues compliances differ: skeletal muscle, which is 38% of the body mass, has a compliance of 0.48 ml/mmHg/kg tissue mass, while the visceral values are respectively 10% and 20-80 [3,5]. Thus, the relative position of the heart and the viscera helps determine BP change with posture.

On standing, the relocated blood is accommodated by both elastic and creep responses of the vessels, and also by filtration of blood plasma into the extravascular space. Therefore time scale affects volume transfer estimates, which vary dramatically, see Table 1. The lower end of this range is relevant to short term response, and beat-to-beat data immediately following orthostasis should be considered. Such data show that BP falls by approximately 20mmHg in the 8 s following standing, and then recovers due to reflexes [6]. However, the initial BP drop is largely a mechanical response.

Simulations

One-dimensional approximations to the momentum and continuity equations, together with a tube law, describe flow in the blood vessels [7]. Wave propagation is the dominant process, and the equations are best solved by computational fluid dynamics (CFD) methods designed for such systems: the MacCormack method [7], and the split coefficient matrix (SCM) method [2]. For time step Δt, spatial grid increment Δx, flow speed U, and wave speed c, these methods are all stable provided

\[ \Delta t = \Delta x / (|U| + c) \]  (1)

The network is truncated, and the vascular beds are approximated by lumped terminal loads, see Figure 1, [1,7]. Vessel boundary conditions rely on the method of characteristics.

METHODS

The integrator is a first order SCM method with a second order volume correction [2]. At vessel boundaries, volume is conserved, losses are due to lumped resistances, and the method of characteristics is used. Valves do not leak when shut, but may offer resistance when open. The heart pumps by a time varying (rectified sinusoid) elastance, and heart parameters are identical to the left ventricular ones used by Peterson et al [7]. The current model has no reflex control.

The arterial vessel and bed pressures are linear functions of area or volume, while, with r interpreted as relative area or volume, the pressure law used for all veins and venous bed capacitors is:

\[ \Phi(r) = r - 1 + 0.02(r^3 - r^{1.5}) \]  (2)

This weak nonlinearity, together individual stiffness constants, matches the compliance data. The network is shown in Figure 2. It has distributed vessels, and vascular beds of the type shown in Figure 1.

**Figure 1: Lumped terminal load**
RESULTS AND DISCUSSION

<table>
<thead>
<tr>
<th>MODEL</th>
<th>TARGET</th>
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<tbody>
<tr>
<td>Physiological Operating Point</td>
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<tr>
<td>MCFP</td>
<td>mmHg</td>
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<tr>
<td>Blood Vol.</td>
<td>ml</td>
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<tr>
<td>Venous Vol.</td>
<td>% total</td>
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<tr>
<td>Compliance</td>
<td>ml/mmHg/kg</td>
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<tr>
<td>Compliance Nonlinearity</td>
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<td>MCFP, 4mmHg</td>
<td>ml/mmHg/kg</td>
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<tr>
<td>MCFP, 12mmHg</td>
<td>ml/mmHg/kg</td>
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<td>Supine</td>
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<tr>
<td>Upright</td>
<td>ml</td>
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<td>Transfer</td>
<td>ml</td>
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Table 1: Model predictions compared with physiological data [3,4,6]

Table 1 shows that a three bed model can result in physiological values for the parameters selected. More detailed results are shown in Figure 3, where the three panels illustrate the responses of mean arterial pressure (MAP), mean right atrial pressure (MRAP), and CO to head-up-tilt after 20 s of supine rest. In each panel, results are shown for three values of the bed I venous stiffness constant: 11.4, 22.7, and 45.4 kPa. The magnitude of the BP transients on head-up-tilt are controlled by the relative positions of the heart and bed G, and by the change in thoracic pressure. However, the time scale for the changes is determined by the RC constant for the transfer of blood through the peripheral resistor (R_p) into the venous capacitor (C_v) of the bed I. R_p is determined by the regional blood flow. Therefore, the venous stiffness constant is varied. The magnitudes of the changes in BP are insensitive to this value, but the time scale changes markedly. To complete the blood transfer in 8 s requires a large stiffness constant that results in a small blood volume transfer consistent with the lowest reported value that we have encountered.

REFERENCES