SIMULTANEOUS APPLICATION OF HEMOPUMP AND MECHANICAL VENTILATION: COMPUTER SIMULATION

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Abstract

The aim of this work is to predict the influence of mechanical ventilation on haemodynamic variables in different circulatory conditions when Hemopump is applied. To study this interaction we used a computer simulator of human cardiovascular system in which lumped parameters models are used to reproduce the circulatory phenomena in terms of pressure and volume relationships. Variable elastance models reproduce the Starling’s law of the heart, for each ventricle. In the study the left ventricular elastance assumed two different values. Systemic arterial resistance was changed during simulation. The influence of mechanical ventilation was introduced by positive mean thoracic pressure. Positive thoracic pressure, changes of peripheral resistance and different ventricular elastance values have a significant influence on haemodynamic variables.

Key Words
Assist Device, Mechanical Ventilation, Numerical Simulation

1. Introduction

Ventilation and ventilatory maneuvers can have profound cardiovascular effects, which can be either beneficial or detrimental. In particular left ventricular performance can be significantly influenced by changes in intrathoracic pressure. Sustained increases in intrathoracic pressure unload the left ventricle, but since venous return decreases, increased intrathoracic pressure is associated with a decreased cardiac output and systemic arterial pressure. Mechanical ventilation of the lungs may also have different effects on aortic flow and coronary blood flow. The aim of our study was to predict, by numerical simulation, the effect of simultaneous application of Hemopump and mechanical ventilation on cardiac output (CO), mean systemic (Pas) and pulmonary (Pap) arterial pressure, left (Pla) and right (Pra) mean atrial pressure. In the study left ventricular elastance (Elv) assumed two values and systemic arterial resistance (Ras) assumed different values. Computer simulation methods can overcome the difficulty of limited measurement techniques and financial and ethical problems connected with animal experiments. Of course, with the guidance of simulation results, the number of clinical or animal experiments can be substantially reduced.

2. Materials and methods

For our experiments we used a modular numerical model (CARDIOSIM®) [1] that can be assembled in different way. We used for the cardiovascular network a simple model in which it was possible to change the mean intrathoracic pressure (Pt).

2.1 Cardiovascular numerical model

The cardiovascular system is divided into seven parts: left and right hearts, pulmonary and systemic arterial sections, pulmonary and systemic venous sections and coronary section (Fig.1). A variable elastance model modified according to Suga and Sagawa’s studies [2] reproduces the Starling’s law of the heart. The heart contraction and ejection phase is described by the End Systolic Pressure-Volume Relationships (ESPVR). End Diastolic Pressure-Volume Relationships (EDPVR) proposed by Gilbert and Glantz [3] describes the filling of the ventricle. The connection of the ventricle to the circulatory network is realised by means of a valve, which is assumed to be ideal: i.e. when it is open the flow through is proportional to the pressure drop and there is no flow when it is closed. For the atrial sections (left and right) we used a single constant compliance (Cla and Cra). Its value is taken from Guyton [4]. Mechanical properties of each section are modeled by RLC elements. Systemic (and pulmonary) arterial section is represented by means of a modified windkessel including a variable peripheral resistor Ras (Rap). The representation of systemic venous section is very simple (a windkessel) and contains a venous capacitor (Cvs) and a systemic resistance (Rvs). Pulmonary venous section is represented by means of a
single compliance (Cvp) because, according to Guyton [4], the pulmonary venous resistance is negligible.

When the device is activated, the pump impellers rotate, and blood is drawn through the inlet cannula into the pump and then discharged into the aorta. The electrical analog of the Hemopump is represented in Fig. 2 [5]. In this system the pressure at the inlet (Pin = pressure in left ventricle), the pressure at the outlet (Pout = pressure in aorta) and the flow change with time during cardiac cycle. Inertial property of the blood is represented by the inertia H. The constant flow source (Qn) depends of the rotation speed of the pump [5]. It is possible to choose seven different rotation speeds.

### 2.3 Mechanical ventilation model

As our aim was to study the influence of positive pressure ventilation on cardiac output, coronary flow and myocardial oxygen consumption in steady state conditions, we assumed that mean thoracic pressure (Pt) is a parameter responsible for the cardiovascular variable changes. This assumption was confirmed by experimental and clinical works [6]. Mean thoracic pressure is defined in the following way:

\[
Pt = \frac{I}{T} \int_{0}^{T} p_{t}(t)\,dt
\]  

(1)

where T is a ventilatory cycle time and pt(t) is an instantaneous thoracic pressure. Using Pt expressed by (1) it is possible to take into account both the actual amplitude of the intrathoracic pressure and the time period when this pressure is applied. In this hypothesis Pt is a good index of the influence of mechanical ventilation of any time on cardiovascular system [7].

### 2.4 Experimental method

To study the influence of Hemopump and mechanical ventilatory support (MVS) on haemodynamic parameters the experiments were performed in two steps. In the first, a pathological state of the left ventricle was represented by the left ventricular elastance Elv=0.66 mmHg·cm⁻³ and the ventricular volume at zero pressure V₀Lᵥ=15 cm³, like in the ischemic heart disease. Ras was changed to simulate physiological (Ras=0.81 mmHg·cm⁻³·s) and pathological conditions (Ras=0.45; 1.52 mmHg·cm⁻³·s). The influence of mechanical ventilation was introduced by positive mean thoracic pressures (Pt changed from −2 to +5 mmHg) [8]. The computed variables CO, Pas, Pap, Pla and Pra were presented as a function of the Pt and Ras.

In the second step, the haemodynamic variables were also computed as function of Pt and Ras but during simultaneous assistance of MVS and Hemopump.
In both sets of experiments heart rate was set to HR=80 bts·min⁻¹ and total blood volume was 5.3 l. The values of the right ventricular elastance and the rest volume were \( E_{RV}=0.77 \text{ mmHg·cm}^{-3} \) and \( V_{0RV}=5 \text{ cm}^3 \), respectively. During the simulations, the Hemopump always runs at constant rotation speed. Consequently we studied the net effects of the pump operated at a certain constant speed. During the study the speed of the pump was fixed to 20000 revolutions min⁻¹.

3. Results and discussion

The results obtained as a function of Ras and Pt are presented in Figures 3, 4 and 5. According to the simulation in the pathological state of the left ventricle positive Pt values changes all the haemodynamic parameter values.

![Figure 3](image3.jpg)

Figure 3. Cardiac output (CO) surfaces in left ventricular pathological (dark surface) and assisted (light surface) conditions. When the Hemopump was applied CO was the total output composed of the left ventricle and Hemopump flows.

An increase of intrathoracic pressure causes a reduction of CO [Fig.3], Pas, Pap [Fig.4] and Pla [Fig.5]. The values of Pra [Fig.5] slightly change with Pt variations. Regardless of values of the peripheral resistance (Ras=0.45; 0.81; 1.52 mmHg·cm⁻³·s), the variations of CO Pas, Pla and Pra as a function of Pt, are similar in the cases when the ventilatory and Hemopump assistance are simultaneously applied or not.

The presence of Hemopump dramatically changes the values of Pla and Pap, increases the total cardiac output (composed of the left ventricle and Hemopump flows) and the systemic arterial pressure. Figure 3 shows that cardiac output is evidently affected by the presence of the assistance (as could be expected). The positive intrathoracic pressure produces small effects on CO in both cases (assisted and not assisted). The cardiac output sensitivity is higher at lower values of peripheral resistance (Ras) and for values of Pt higher than 2 mmHg.

![Figure 4](image4.jpg)

Figure 4. Systemic (Pas) and pulmonary (Pap) arterial pressure surfaces in left ventricular pathological (dark surface) and assisted (light surface) conditions.

A similar trend can be observed in a case of mean systemic arterial pressure [Fig. 4]. Mean pulmonary arterial pressure is increased by the presence of Hemopump and almost linearly falls with the Pt augmentation. The left atrial pressure is decreased by the Hemopump presence and is sensitive to intrathoracic pressure variations. The right atrial pressure [Fig.5] is slightly affected by the assistance and its sensitivity to Pt is minimum around 2 mmHg.

The simulation has shown that the effects of the spontaneous breathing (Pt changing around –2 mmHg on average) on the cardiovascular variables (CO, Pas, Pla and Pap) are opposite to the effects of mechanical ventilation on these variables. The negative Pt value usually increases cardiac output by increasing the venous return to the right atrium, while the positive alveolar pressure generated during mechanical ventilation, is transmitted into the thorax and impedes the venous return. Many other predictions from the model are also in good
agreement with the reported results of clinical and animal experiments [9,10].

The direct effects of the Hemopump on the haemodynamic variables of the circulatory system include an increase in mean aortic pressure, an increase in stroke volume, a decrease in left ventricular volume, a decrease in blood flow pumped out by the left ventricle, a decrease in the area of the pressure-volume (P-V) loop of the left ventricle during a cardiac cycle, and a decrease in left atrial pressure. The increase in Pas increases the coronary blood flow and this effect produces an increase in the oxygen supply to the heart.

The decrease in the left ventricle volume and the amount of blood pumped out by the left ventricle imply a reduction in the left ventricle workload. On the other hand, the reduction of the left P-V loop area produces a decrease in the heart oxygen consumption [2]. The result is that the Hemopump can assist the failing heart by increasing the oxygen supply while decreasing the oxygen consumption.

References