XVII IMEKO World Congress
Metrology in the 3rd Millennium
June 22–27, 2003, Dubrovnik, Croatia

IN VIVO AND NUMERICAL SIMULATION STUDIES ON HAEMODYNAMIC PARAMETERS AND CARDIOPULMONARY INTERACTION

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Abstract – In the paper the results of clinical and simulation studies on main haemodynamic parameters were compared. Cardiac output, arterial, atrial and venous pressures were measured in vascular patients during spontaneous breathing and artificial ventilation. Computer simulator was used to model a human cardiovascular system and calculate values of haemodynamic parameters in steady states. Changing intrathoracic pressure, from negative to positive values simulated cardiopulmonary interaction during spontaneous breathing and controlled ventilation. The results of the studies presented in the paper proved that numerical simulation is a very suitable and reliable tool to predict trends of haemodynamic parameters changes. Also the potential influence of mechanical ventilatory support on these parameters may by analysed before application of less or more positive airway pressure. It is an important contribution to the safety and efficacy of ventilatory support in these patients in which high intrathoracic pressure may cause a dramatic drop of cardiac output, venous return and arterial pressure.

Keywords: Haemodynamic, Mechanical Ventilation, Computer Simulation

1. INTRODUCTION

In patients undergoing surgical operation the main haemodynamic parameters like cardiac output (CO), systemic arterial pressure (Pas), pulmonary arterial pressure (Pap), central venous pressure (CVP) and left atrial pressure (Pla) are important diagnostic and sometimes also therapeutic parameters. They should be measured continuously and monitored on-line in order to check the state of a patient. During surgery some patients require mechanical ventilation of the lungs, which may result in a positive intrathoracic pressure high enough to change dramatically the values of measured haemodynamic parameters, especially in cases of cardiovascular pathology. The aim of this study was to use computer simulation to assess this problem and to predict the changes of haemodynamic parameters which would be caused by the proposed method of mechanical ventilation (MV) of the lungs.

2. METHODS

In vivo measurements were performed on seven vascular patients during aortic grafting. Patients were monitored by Hewlett Packard (1175A – Merlin) monitoring system. Arterial pressure (Pas) was acquired from left radial artery with 20 G needle. Pressures (Pap and CVP) were measured using Swan Ganz catheters and cardiac output (CO) was measured by a thermodilution method.

Computer simulator CARDIOSIM® [1] was used to model a human cardiovascular system, to simulate changes of mentioned above parameters and calculate their values in steady states. The cardiovascular system (Fig. 1) was divided into seven parts: left and right hearts, pulmonary and systemic

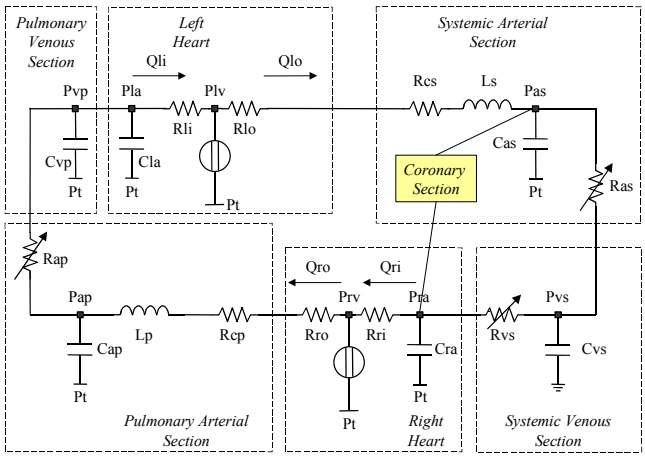

Figure 1. Electric analog of the cardiovascular system.

arterial sections, pulmonary and systemic venous sections and a coronary section. The Starling's law of the heart was

reproduced by a variable elastance model modified according to Suga and Sagawa's studies [2]. The heart contraction and an ejection phase was described by the equation :

$$p(t) = E(t) \cdot [v(t) - Vo] \cdot f[v(t), \dot{v}(t), \dot{v}_{max}] \qquad (1)$$

where *p(t)* - instantaneous ventricular pressure, *v(t)* - instantaneous ventricular volume, *E(t)* - isovolumetric time-varying elastance, *Vo* - ventricular volume at zero pressure [defined as the intercept of the End Systolic Pressure Volume Relationships (ESPVR) with the volume axis] and *f[……]* - corrective function for the isovolumetric time-varying elastance, taking into account the ejection rate, the maximal ejection rate and the ejected volume. The filling of the ventricle was described by the End Diastolic Pressure-Volume Relationships proposed by Gilbert and Glantz [3]:

$$p(t) = C \cdot e^{k \cdot v(t)} + D \cdot e^{-j \cdot v(t)} + E \qquad (2)$$

where the coefficients *C, D, E, k, j* represent the ventricular properties during the filling. The first component $(C \cdot e^{kv(t)})$ affects the stiffness of the ventricle. The second $(D \cdot e^{-jv(t)})$ influences the diastolic suction. The ventricular rest volume *Vo* is affected by both components.

The connection of the ventricle to the circulatory network was realised by means of a valve (Rlo, Rli, Rro and Rri), which was assumed to be ideal: i.e. when it was open the flow through it was proportional to the pressure drop and there was no flow when it was closed. For the atrial sections (left and right) we used a single constant compliance (Cla and Cra). Its value was taken from Guyton [4].

Mechanical properties of each section were modelled by RLC elements. The systemic (and pulmonary) arterial section was represented by means of a modified windkessel Rcs, Ls and Cas (Rcp, Lp and Cap) including a variable peripheral resistor Ras (and Rap). The representation of the systemic venous section was very simple (a windkessel) and contained a venous capacitor (Cvs) and a systemic resistance (Rvs). Resistance Rvs can be automatically adjusted according to the relationship proposed by Guyton [4]: Rvs = K/Pvs, where K is the constant and is assumed to be 0.16 $mmHg^2 \cdot cm^{-3} \cdot s$ and Pvs is a venous systemic pressure. The pulmonary venous section was represented by means of a single compliance (Cvp) because, according to Guyton [4], the pulmonary venous resistance was negligible.

As far as lung ventilation influence on cardiovascular system is concerned, it can be easily modelled in CARDIOSIM© by adjusting the mean value of intrathoracic pressure (Pt). It is possible to define the mean thoracic pressure using:

$$Pt = \frac{1}{Ti + Te} \cdot \left(\int_0^{Ti} pi(t) \cdot dt + \int_{Ti}^{Ti+Te} pe(t) \cdot dt \right) \qquad (3)$$

where *pi(t)* - intrathoracic pressure change during the inspiratory period, *pe(t)* - intrathoracic pressure change during the expiratory period, *Ti* and *Te* - inspiratory and expiratory time, respectively.

Using Pt expressed by (3) it 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 MV of any type on the cardiovascular system.

Three of the patients under study were sedated and mechanically ventilated. Mechanical ventilation was simulated by a positive mean intrathoracic pressure of +3 mmHg set in CARDIOSIM® [5].

3. RESULTS

In Figs. 2, 3 and 4 the results of *in-vivo* measurements of CO, CVP, Pas, and Pap were shown and compared with values of these parameters obtained during computer simulation. CVP measured during *in-vivo* studies was compared with simulated mean right atrial pressure, as CVP is not available in CARDIOSIM® (see Fig. 1). Only in four patients CVP measurements were done properly. The data concerned spontaneously breathing patients.

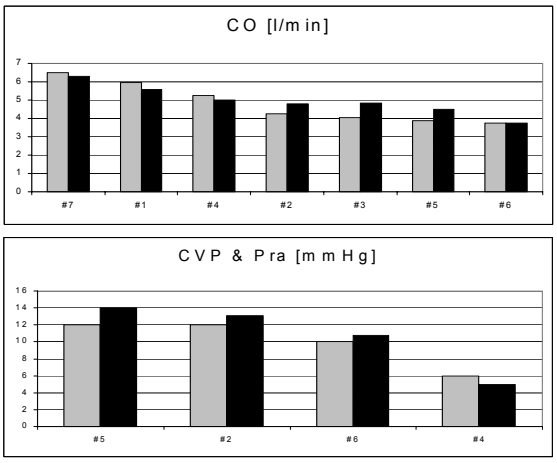

Figure 2. Comparison of the measurements of CO and CVP obtained during *in-vivo* (grey bars) and simulation (black bars) studies. Data concerned spontaneously breathing patients.

In Figs. 5 and 6 the results of *in-vivo* measurements of CO, mean, systolic and diastolic Pas were shown and compared with values of these parameters obtained during computer simulation. They were done on three patients breathing spontaneously (Pt=-2 mmHg) and then mechanically ventilated (Pt=+3 mmHg).

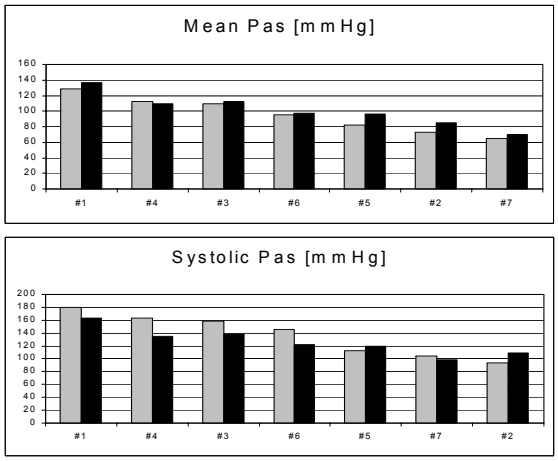

Figure 3. Comparison of the measurements of mean and systolic Pas obtained during *in-vivo* (grey bars) and simulation (black bars) studies. Data concerned spontaneously breathing patients.

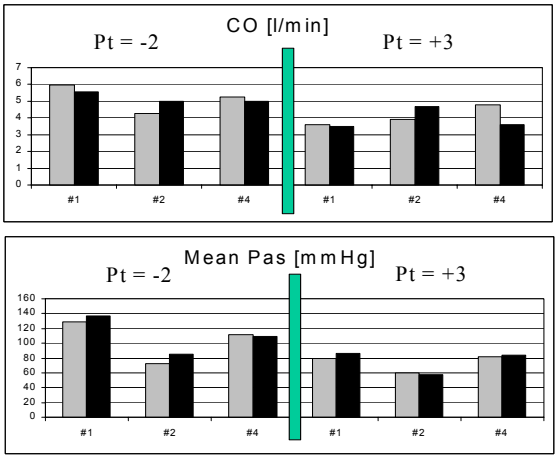

Figure 5. The influence of mechanical ventilation (Pt=+3 mmHg) on CO and mean Pas shown as comparison of *in-vivo* measurements (grey bars) and numerical simulation (black bars), concerning three patients. During spontaneous breathing Pt=-2 mmHg.

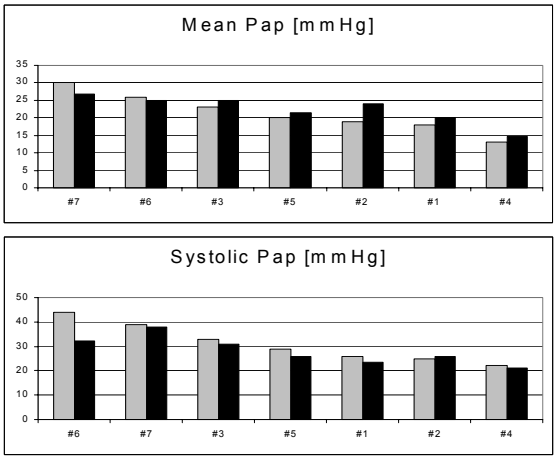

Figure 4. Comparison of the measurements of mean and systolic Pap obtained during *in-vivo* (grey bars) and simulation (black bars) studies. Data concerned spontaneously breathing patients.

4. DISCUSSION

As can be seen from Figs. 2 ÷ 6 the difference between values obtained *in vivo* study and by numerical simulation varied, depending on the haemodynamic parameter and a patient under consideration. These differences were mainly due to the fact that not all parameters necessary to model the cardiovascular system were available from *in vivo* studies. Also the literature on this subject does not provide sufficient data, that could be successfully used in numerical simulation. As far as ventilated patients are concerned the trends of main cardiovascular parameters changes (Figs. 5 and 6) are very similar in *in vivo* study and obtained by CARDIOSIM®.

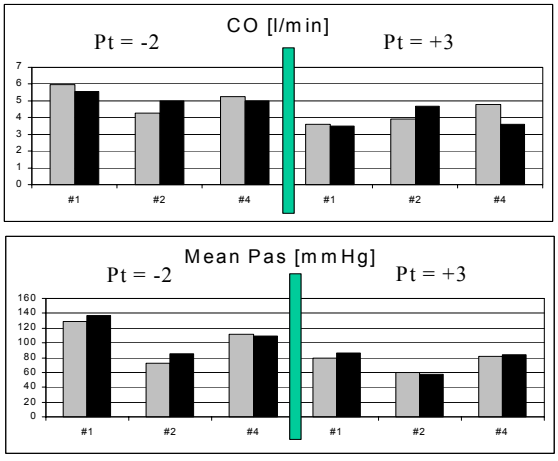

Figure 6. The influence of mechanical ventilation (Pt=+3 mmHg) on systolic and diastolic Pas shown as comparison of *in-vivo* measurements (grey bars) and numerical simulation (black bars), concerning three patients.
During spontaneous breathing Pt=-2 mmHg.

When mean intrathoracic pressure (Pt) increases from –2 mmHg to +3 mmHg the same changes can be observed in all

patients: CO, mean, systolic and diastolic Pas decreases. Thus up to now CARDIOSIM® is much more useful in predicting a trend of the changes of a particular parameter than to foresee its absolute values. Nevertheless the results presented in this paper proved that numerical simulation can be a very valuable tool to assess the changes of main haemodynamic parameters (CO, Pas) before applying the less and/or more intensive (Pt = +3 mmHg) methods of ventilatory support of the lungs. It should be added that structure of CARDIOSIM® enables to insert mechanical ventricular support devices (like left ventricular assist devices, bi-ventricular assist device and intra-aortic balloon pump) into the model. It gives the possibility to assess independently the influence of mechanical support of respiratory and circulatory systems on haemodynamical and energy-related cardiovascular variables [6,7,8].

5. CONCLUSIONS

In conclusion, the preliminary results of numerical simulation, when compared with *in vivo* studies, are promising. They show that numerical simulation is reliable and may be very helpful in predicting the influence of mechanical ventilation on haemodynamics. This fact is especially important for patients with cardiovascular pathology, when non-physiological, positive intrathoracic pressure may dramatically diminish venous return, cardiac output and systemic arterial pressure.

ACKNOWLEDGMENT

This work was done in the frame of grant 7T11E00420 from KBN (Committee for Scientific Research), Poland.

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