Computer simulation of haemodynamic parameters changes with left ventricle assist device and mechanical ventilation

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Abstract

Left Ventricular Assist Device is used for recovery in patients with heart failure and is supposed to increase total cardiac output, systemic arterial pressure and to decrease left atrial pressure. Aim of our computer simulation was to assess the influence of Left Ventricular Assist Device (LVAD) on chosen haemodynamic parameters in the presence of ventilatory support. The software package used for this simulation reproduces, in stationary conditions, the heart and the circulatory system in terms of pressure and volume relationships. Different circulatory sections (left and right heart, systemic and pulmonary arterial circulation, systemic and pulmonary venous circulation) are described by lumped parameter models. Mechanical properties of each section are modelled by RLC elements. The model chosen for the representation of the Starling’s law of the heart for each ventricle is based on the variable elastance model. The LVAD model is inserted between the left atrium and the aorta. The contractility of the heart and systemic arterial resistance were adjusted to model pathological states. Our simulation showed that positive thoracic pressure generated by mechanical ventilation of the lungs dramatically changes left atrial and pulmonary arterial pressures and should be considered when assessing LVAD effectiveness. Pathological changes of systemic arterial resistance may have a considerable effect on these parameters, especially when LVAD is applied simultaneously with mechanical ventilation. Cardiac output, systemic arterial and right atrial pressures are less affected by changes of thoracic pressure in cases of heart pathology. © 2000 Elsevier Science Ltd. All rights reserved.

Keywords: Computer modelling; Left ventricular assist device; Mechanical ventilation; Haemodynamics

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1. Introduction

The aim of cardiopulmonary system is to deliver oxygen to the tissues. In addition to use as a bridge to transplant, the LVAD may be temporary used in patients affected by severe heart failure and drugs resistance with the goal of recovering the failing heart and prevent multiorgan failure. One of the main targets of this kind of mechanical heart assistance is the reduction of the external work of the natural ventricle and left atrial pressure \((P_{la})\) and the increase of total cardiac output \((CO)\) and systemic arterial pressure \((P_{as})\). The systemic arterial pressure and flow are indexes of circulation. The left atrial pressure reflects the preload of the heart. The application of mechanical ventilatory support during the LVAD assistance can affect the values of basic haemodynamic parameters: \(CO\), \(P_{as}\) and also right atrial pressure \((P_{ra})\), left atrial pressure, pulmonary arterial pressure \((P_{ap})\) and systemic venous pressure \((P_{vs})\).

Positive pressure ventilation has an adverse effect on cardiovascular system. Usually positive pressure in alveoli diminishes lung perfusion, venous return and cardiac output in turn, as during the respiratory cycle this positive pressure is transferred into thoracic space. The haemodynamic effects of mechanical ventilation are mainly dependent on a single variable — mean value of thoracic pressure \((P_t)\) generated during ventilatory cycle [1–6]. The mode of mechanical ventilation has a secondary importance and may not be considered in this aspect. The aim of our computer simulation was to trace the influence of LVAD on above mentioned haemodynamic parameters, in the presence of ventilatory support to the patient’s lungs.

2. Materials and methods

Our work was based on Suga and Sagawa’s [7] studies of the cardiac work cycle on the Pressure–Volume \((P–V)\) plane. In this plane the pressure–volume loops described by the heart are bounded by the End Systolic Pressure Volume Relationship (ESPVR) line and the Diastolic \(P–V\) relation curve. The loop together with these pressure–volume relationships gives information concerning haemodynamic and energetic variables (Fig. 1). The haemodynamic parameters were computed using a computer simulator of cardiovascular system (CARDIOSIM*) [8]. This software package runs on PC using Windows95™–98™ capabilities. Different modules, using different languages and application software, compose it. Microsoft® Visual Basic® 5.0 language is used for graphical presentation and for data storing. Borland® C++® 4.0 language is used for calculation and automatic control routine. EXCEL® worksheet is used to analyse and store ‘on line’ or ‘off line’ data obtained by simulation. CARDIOSIM* is able to reproduce, in stationary conditions, the effect of the heart and the circulatory system in terms of pressure and volume relationships. It is composed by using several modules including mechanical heart assist devices (LVAD, Right Ventricle Assist Device, Biventricular Assist Device and Intraaortic Balloon Pump) which can be put separately according to the experiment to be performed.

2.1. Circulatory system model

The configuration used here (block diagram Fig. 2 and legend in Table 1) consists of six
connected sections: left and right hearts, systemic arterial and venous sections and as well as pulmonary arterial and venous sections. The general relationships inside the circulatory network are mainly referred to Guyton and Sagawa’s models. The atrial section (left and right) is a single constant compliance ($C_{la}$ and $C_{ra}$) and its value is taken from Guyton [9]. The model chosen for the representation of the ventricle (left and right), previously described elsewhere [8], is based on a variable elastance model modified according to Sagawa’s studies [7] (for the ejection). For the filling of the ventricle we used the End Diastolic Pressure–Volume Relationship (EDPVR) proposed by Gilbert and Glantz [10]. They are adequate for the reproduction of the Starling’s law of the heart. The heart contraction and ejection phase is described by the following equation:

$$p(t) = E(t) \cdot [v(t) - V_0] \cdot f[v(t), \dot{v}(t), \dot{v}_{max}]$$

where $p(t)$ and $v(t)$ are the instantaneous ventricular pressure and volume, $E(t)$ is the isovolumetric time-varying elastance, $V_0$ the ventricular volume at zero pressure (defined as the intercept of the end systolic pressure–volume relationship with volume axis) during systole, and $f[v(t), \dot{v}(t), \dot{v}_{max}]$ a corrective function for the isovolumetric time-varying elastance taking into account the ejection rate, the maximal ejection rate and the ejection volume. Changes in contractility are induced by changes in $E(t)$.

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 back through is proportional to the

![Fig. 1. Cardiac loop, end systolic pressure–volume relationship (ESPVR) and end diastolic pressure–volume relationship (EDPVR) in the $P$–$V$ plane. $P_{es}$ is end systolic pressure, $V_{es}$ is end systolic volume, $V_{ed}$ is end diastolic volume, $V_0$ is ventricular volume at zero pressure.](image-url)
pressure drop, and there is no flow when it is closed. Mechanical properties of each section are
modelled by RLC elements, as in an electrical analogue circuit. The representation of the
systemic arterial section (and pulmonary arterial section) is realised by means of modified
windkessel [11] including peripheral (variable) resistor $R_{as}$ ($R_{ap}$). These resistors can be adjusted
automatically to keep the afterload constant. In the RLC model the inductance represents the
inertia of blood. The representation of the systemic venous section is realised by means of
simple windkessel and contains a venous capacitor ($C_{vs}$) and a systemic venous resistance ($R_{vs}$).
The value of $R_{vs}$ is automatically adjusted according to the relationship proposed by Guyton
($K/P_{vs}$) [9], where the value of the constant ($K$) is assumed to be, according to Guyton, 0.16
mm Hg cm$^{-3}$ s. The pulmonary arterial tree is represented by the modified windkessel.
According to Guyton’s studies, for the pulmonary venous section we used a single compliance
($C_{vp}$) because the pulmonary venous resistance is negligible.

2.2. LVAD model

The pneumatic Left Ventricular Assist Device (LVAD) has been modeled describing

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![Fig. 2. The structure of the computer simulator of cardiovascular system CARDIOSIM®. The symbols describing the elements of the simulator are in Table 1.](image-url)
separately the ejection and the filling. The ejection is described by the air outflow from a high-pressure tank connected to a pressure source \( (P_d) \) towards a lower-pressure tank, which is the ventricle itself. The filling is achieved by the air outflow from a higher pressure tank (the ventricle itself) to a lower pressure tank connected to the vacuum source \( (P_v) \) [8,12]. This is a simple model to describe the physical phenomenon underlying the filling and ejection phases in the artificial ventricle, but is enough for our purpose. LVAD is inserted (in parallel) between the left atrium and the aorta (Fig. 2) and, during our simulation, it is synchronized with the onset of the natural ventricle contraction.

2.3. Mechanical ventilation model

Any mode of artificial ventilation of the lungs (except Continuous Positive Airway Pressure — CPAP) generates cyclic changes of intrathoracic pressure. As our aim was to assess the

Table 1
The parameters used in CARDIOSIM® computer simulator

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Value</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Circulatory network</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left (right) heart</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left input (output) valve resistance</td>
<td>( R_{li} (R_{ro}) )</td>
<td>0.01 (0.01)</td>
<td>( \text{mm Hg cm}^{-3} \text{s} )</td>
</tr>
<tr>
<td>Right input (output) valve resistance</td>
<td>( R_{ri} (R_{ro}) )</td>
<td>0.01 (0.01)</td>
<td>( \text{mm Hg cm}^{-3} \text{s} )</td>
</tr>
<tr>
<td>Left (right) atrial compliance</td>
<td>( C_{la} (C_{ra}) )</td>
<td>5 (5)</td>
<td>( \text{cm}^3 \text{ mm Hg}^{-1} )</td>
</tr>
<tr>
<td>Left (right) atrial pressure</td>
<td>( P_{la} (P_{ra}) )</td>
<td></td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td>Left (right) ventricular pressure</td>
<td>( P_{lv} (P_{rv}) )</td>
<td></td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td>Left input (output) flow</td>
<td>( Q_{li} (Q_{lo}) )</td>
<td></td>
<td>( \text{l min}^{-1} )</td>
</tr>
<tr>
<td>Right input (output) flow</td>
<td>( Q_{ri} (Q_{ro}) )</td>
<td></td>
<td>( \text{l min}^{-1} )</td>
</tr>
<tr>
<td><strong>Systemic (pulmonary) arterial section</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systemic (pulmonary) characteristic resistance</td>
<td>( R_{cs} (R_{cp}) )</td>
<td>0.014 (4.5e^{-3})</td>
<td>( \text{mm Hg cm}^{-3} \text{s} )</td>
</tr>
<tr>
<td>Systemic (pulmonary) inertia</td>
<td>( L_{s} (L_{p}) )</td>
<td>( 1e^{-5} (1e^{-5}) )</td>
<td>( \text{mm Hg cm}^{-3} \text{s}^2 )</td>
</tr>
<tr>
<td>Systemic (pulmonary) arterial compliance</td>
<td>( C_{as} (C_{ap}) )</td>
<td>1.8 (4.8)</td>
<td>( \text{cm}^3 \text{ mm Hg}^{-1} )</td>
</tr>
<tr>
<td>Systemic (pulmonary) arterial resistance</td>
<td>( R_{as} (R_{ap}) )</td>
<td>0.81 (0.11)</td>
<td>( \text{mm Hg cm}^{-3} \text{s} )</td>
</tr>
<tr>
<td>Systemic (pulmonary) arterial pressure</td>
<td>( P_{as} (P_{ap}) )</td>
<td></td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td><strong>Systemic (pulmonary) venous section</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systemic venous resistance</td>
<td>( R_{vs} )</td>
<td>0.11</td>
<td>( \text{mm Hg cm}^{-3} \text{s} )</td>
</tr>
<tr>
<td>Systemic (pulmonary) venous compliance</td>
<td>( C_{vs} (C_{vp}) )</td>
<td>82.5 (5)</td>
<td>( \text{cm}^3 \text{ mm Hg}^{-1} )</td>
</tr>
<tr>
<td>Systemic (pulmonary) venous pressure</td>
<td>( P_{vs} (P_{vp}) )</td>
<td></td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td>Thoracic pressure</td>
<td>( P_t )</td>
<td></td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td><strong>LVAD</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Input (output) valve resistance</td>
<td>( R_{vi} (R_{vo}) )</td>
<td>0.15 (0.15)</td>
<td>( \text{mm Hg cm}^{-3} \text{s} )</td>
</tr>
<tr>
<td>Air compliance</td>
<td>( C_{air} )</td>
<td></td>
<td>( \text{cm}^3 \text{ mm Hg}^{-1} )</td>
</tr>
<tr>
<td>Ventricular pressure</td>
<td>( P_{pv} )</td>
<td></td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td>Ventricular air pressure</td>
<td>( P_{air} )</td>
<td></td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td>Driving pressure</td>
<td>( P_d )</td>
<td>250</td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td>Vacuum pressure</td>
<td>( P_v )</td>
<td>(-10)</td>
<td>( \text{mm Hg} )</td>
</tr>
<tr>
<td>Input (output) flow</td>
<td>( Q_{pi} (Q_{po}) )</td>
<td></td>
<td>( \text{l min}^{-1} )</td>
</tr>
</tbody>
</table>
influence of positive pressure ventilation on haemodynamic cardiovascular variables in steady state conditions we assumed that mean thoracic pressure ($P_t$) is a parameter responsible for the cardiovascular variables changes. This assumption was well proved by experimental and clinical studies [3]. Mean thoracic pressure is defined as:

$$P_t = \frac{1}{T} \cdot \int_0^T p(t) \, dt$$

(2)

where $T$ is a ventilatory cycle time and $p(t)$ is an instantaneous thoracic pressure. Thus, the simulation of mechanical ventilation was performed simply by changing the level of mean thoracic pressure ($P_t$) in the range $-2 \ldots 5$ mm Hg. The use of mean thoracic pressure instead of cyclic intrathoracic pressure during simulation has also a practical reason. There are so many different modes of mechanical ventilation available nowadays that the consideration of cyclically changing thoracic pressure would require the introduction of too many independent variables describing these pressure changes, like — minimum and maximum pressure amplitude, slope of its increase or decrease, inspiratory and expiratory times ratio, ventilatory frequency and its time shift in relation to systolic (or diastolic) heart action.

2.4. Experimental method

To study the influence of LVAD on haemodynamic parameters in the presence of mechanical ventilatory support we have performed the experiments in three steps:

- In the first step we compared the effect of elevated airway pressure on our simulation results with in vivo data available in the literature. For this purpose, we used data from a paper [6] reporting values of cardiac output and pulmonary vascular resistance as a function of airway pressure. These data were obtained from dogs. To make the comparison we compared in vivo and in vitro Relative Cardiac Output (RCO) for each intrathoracic pressure. RCO was defined as:

$$\text{RCO}_{(P_t=k)} = \frac{\text{CO}_{(for \ P_t=k)} - \text{CO}_{(for \ P_t=3)}}{\text{CO}_{(for \ P_t=3)}} \cdot 100 \quad (k = -2, 0, 2, 3)$$

(3)

- In the second step a pathological condition of the left ventricle was reproduced by left ventricular contractility $C_a = 1.5 \text{ cm}^3 \text{ mm Hg}^{-1}$ (slope of ESPVR) and the ventricular volume at zero pressure $V_0 = 15 \text{ cm}^3$, like in ischemic heart disease. Peripheral resistance $R_{as}$ was changed to model physiological ($R_{as} = 0.81 \text{ mm Hg cm}^{-3} \text{ s}$) and pathological ($R_{as} = 0.45; 1.52 \text{ mm Hg cm}^{-3} \text{ s}$) conditions. The influence of mechanical ventilation support was introduced by changing levels of thoracic pressure ($P_t$) from $-2$ till 5 mm Hg [13]. The haemodynamic parameters were computed and presented as a function of thoracic pressure and peripheral resistance.

- In the third step haemodynamic variables were also computed as function of $P_t$ and $R_{as}$ but with the simultaneous presence of LVAD.

Heart rate was set to $H_R = 80 \text{ bts min}^{-1}$. The values of right ventricular contractility and
rest volume at zero pressure were respectively Ca = 1.3 cm³ mm Hg⁻¹ and V₀ = 5 cm³. The values of other parameters are reported in Table 1.

3. Results and discussion

The effects of mechanical ventilation of the lungs on haemodynamics have been studied experimentally and clinically. The results of these studies [1–6] have evidenced how it affects mainly preload, stroke volume (SV) and cardiac output. The mechanism underlying these effects is not completely clear and to some extent controversial. Certainly, variable by which it exerts its action is mean thoracic pressure. By the point of view of our simulation what we tried to verify was the net effect of this variable on haemodynamics. Data in the literature show clearly that positive intrathoracic pressure decreases preload, SV and cardiac output. In general, our simulation confirmed these findings. As it was said in Section 2, we could compare relative cardiac output computed by Eq. (3) using our simulation data and in vivo data found in literature [6].

Fig. 3 shows the results of this comparison. The model parameters were set according to Table 1 and Pᵗ and Rᵃᵖ were changed according to the table, comparing simulation and in vivo data, included in Fig. 3. In vivo data were measured in dogs. In both studies, RCO changes with elevation of airway pressure are similar. It is interesting to evidence the similarity of their trend even if they were obtained in different circulatory conditions (our simulation uses human circulatory parameters, while in vivo data were obtained from dogs [6]). This is in fact the use of relative cardiac output to allow their comparison.

For constant thoracic pressure (Pᵗ) the application of LVAD shifts blood from systemic to
pulmonary circulation. Generally, it increases CO and $P_{as}$, significantly decreases $P_{la}$ and $P_{ap}$ but slightly changes $P_{vs}$ and $P_{ra}$. As a matter of fact, the effects of LVAD on haemodynamics depend on several factors including ventricular interdependence [14], the state of the right circulation and right ventricular contractility that, in turn, can be affected by LVAD. This complex interaction including, last but not least, the control strategy of the assistance, can produce different and opposite effects on haemodynamics [15–20].

We tried to verify the results of our simulation comparing our data with the average trend of haemodynamic data found in the literature [17]. Accordingly, we assumed that, after the onset of LVAD assistance, pulmonary vascular resistance decreases (about 40%), systemic arterial resistance decreases (about 30%) and right ventricular elastance remains unchanged (about 1.5 cm$^3$ mm Hg$^{-1}$ [15]). The results of this comparison are reported in Fig. 4. It shows clinical [17] and simulation data on the left and right side, respectively. Data have been normalised and the table, in the same Fig. 4, shows their values.

In conclusion, the haemodynamics effect of the assistance and its deviation from expected results, depends on the specific circulatory conditions. What is important to evidence, to use our results to predict LVAD influence on main haemodynamic parameters, is that the trend of haemodynamic variables is the same in our simulation and in clinical data.

The net effect of simultaneously applied LVAD and mechanical ventilation on cardiovascular variables can only be assessed by means of simulation, as there is lack of clinical data available nowadays concerning heart and lung assistance.

The results of our simulation are reproduced in Figs. 5–7.

![Fig. 4. Comparison between haemodynamics data obtained in vivo [17] and by computer simulation. Data are normalized and their values are reported in the table.](image-url)
Fig. 5 reproduces, in the $P–V$ plane, different cardiac cycles stored during computer simulation. Lower (upper) panel shows a pathological condition (continuous lines A) of the left (right) ventricle and its evolution after LVAD activation (dashed lines B). During the simulation the value of systemic peripheral resistance was 0.45 mm Hg cm$^{-3}$ s and the value of the mean thoracic pressure was 2 mm Hg. LVAD was synchronized with the onset of the natural ventricle contraction, the driving and vacuum pressures were respectively $P_d = 250$ mm Hg and $P_v = -10$ mm Hg. It is possible to observe from this figure:

- A change of the stroke volume, especially for left ventricle (when LVAD was applied). In the right ventricle the SV increased and consequently increased the total cardiac output (LVAD flow + natural flow).
- Changes of end-systolic ($V_{es}$) and end-diastolic volume ($V_{ed}$) for left (and right) ventricle with consequent changes of afterload and preload.
- In the left ventricle cardiac cycle end systolic pressure $P_{es}$ increased (from $P_{es A}$ to $P_{es B}$) and as a result mean systolic arterial pressure $P_{as}$ increased as well.
- In the right cardiac cycle end-systolic volume ($V_{es}$) and pressure decreased and as a result mean arterial pulmonary pressure $P_{ap}$ decreased as well. This decrease in pulmonary artery pressure is caused by the reductions in left ventricular filling pressure during LVAD assistance.

From haemodynamic data presented in the table we calculated the stroke volume index (SVI) using the following formula:

$$SVI = \frac{[CO/(BSA \cdot HR)]}{1000}$$

Eq. (4) was calculated, assuming 1.7 m$^2$ for body surface area (BSA), for CO in pathological condition and after the onset of LVAD assistance. SVI was found to be 27.2 and 36.9 (ml/beat/m$^2$) respectively.

Haemodynamics data obtained for different $P_t$ and $R_{as}$ values are reported in Figs. 6 and 7. Our simulation shows that, in pathological condition of the left ventricle, positive thoracic pressure changes all haemodynamic parameter values. An increase of $P_t$ causes the reduction of CO, $P_{as}$, $P_{la}$ and $P_{ap}$ along with the increase of $P_{vs}$. The values of $P_{ra}$ only slightly change with $P_t$ level. This effect is independent of LVAD presence. These changes are also independent of $R_{as}$ values.

For different values of the peripheral resistance ($R_{as} = 0.45; 0.81; 1.52$ mm Hg cm$^{-3}$ s), the rates of CO, $P_{as}$, $P_{la}$, $P_{vs}$ and $P_{ap}$ (Fig. 6) changes as a function of $P_t$ are similar in cases when ventilatory and LVAD support are simultaneously applied or not. On the other hand another effect can be observed. The presence of LVAD dramatically changes the values of $P_{la}$ (Fig. 6) and $P_{ap}$ (Fig. 7) and increases the total cardiac output (CO) and the systemic arterial pressure ($P_{as}$) (Fig. 6).

Our simulation showed that the effects of spontaneous breathing ($P_t$ changes around $-2$ mm Hg on an average) on cardiovascular variables (CO, $P_{as}$, $P_{la}$, $P_{vs}$ and $P_{ap}$) are opposite to the effects of mechanical ventilation on these variables. Negative intrathoracic pressure usually increases CO by increasing venous return to the right atrium while positive alveolar pressure generated during mechanical ventilation is transmitted into thorax and impedes venous return. Relatively small changes of CO, $P_{as}$ and $P_{ra}$ as a function of $P_t$ (Figs. 6 and 7) could be
Fig. 5. Two different cardiac cycles for left (lower panel) and right (upper panel) ventricles are shown. The curves were obtained during simulation of a pathological condition (continuous lines A) and in the presence of LVAD (dashed lines B). The pathological condition of the left ventricle is characterised by \( C_a = 1.5 \text{ cm}^3 \text{ mm Hg}^{-1} \) and \( V_0 = 15 \text{ cm}^3 \).
Fig. 6. Static characteristic CO, $P_{as}$ and $P_{la}$ versus $P_t$ (from -2 to 5 mm Hg) for pathological ($Ca = 1.5 \text{ cm}^3 \text{ mm Hg}^{-1}$, $V_0 = 15 \text{ cm}^3$) and assisted (with LVAD) conditions of the left ventricle. In both conditions systemic arterial resistance was changed ($R_{as} = 0.45; 0.81; 1.52 \text{ mm Hg cm}^{-3} \text{ s}$). When LVAD was applied CO was total CO (pump flow ($Q_{po}$) + natural flow)
Fig. 7. Static characteristic $P_{sv}$, $P_{ap}$ and $P_{ra}$ versus $P_t$ (from $-2$ to $5$ mm Hg) for pathological ($C_a = 1.5 \text{ cm}^3 \text{ mm Hg}^{-1}$, $V_0 = 15 \text{ cm}^3$) and assisted (LVAD) conditions of the left ventricle. In both conditions systemic arterial resistance was changed ($R_{as} = 0.45; 0.81; 1.52 \text{ mm Hg cm}^{-3} \text{ s}$).
explained by assumed pathological conditions of the left ventricle \((Ca = 1.5 \text{ cm}^3 \text{ mm Hg}^{-1}, V_0 = 15 \text{ cm}^3)\) during simulation. This fact made CO and then \(P_{as}\) and \(P_{ra}\) much less sensitive to thoracic pressure changes than we could have expected in a case of physiological conditions for the left ventricle [21].

4. Conclusion

The results of computer simulations enables to formulate the following statements:

- Some cardiovascular parameters \((P_{la}\) and \(P_{ap}\)) are very sensitive to changes of positive pressure in the thorax, when mechanical ventilatory support and cardiovascular (LVAD) support are simultaneously applied.
- Changes of systemic arterial resistance have a negligible effect on the rate of cardiovascular parameter changes as a function of thoracic pressure changes.
- Changes of \(R_{as}\) may have a dramatic effect on such cardiovascular parameters as \(P_{la}\) and \(P_{ap}\), especially when thoracic pressure is high and LVAD is applied simultaneously.
- LVAD produces a shift of \(P–V\) loop for both ventricles, which modifies end-systolic and end-diastolic volumes.

The results obtained in this work are strongly connected to the choice of the control strategy used for the LVAD (fixed or variable HR, different synchronisation) as they can produce, according to the data presented in the literature, different effects on haemodynamics.

Our model of cardiovascular system has some limitations concerning the representation of impedance properties of human cardiovascular system. As other authors dealing with this problem [22,23], we chose lumped and constant parameters as resistances and capacitances to simulate mechanical properties of different parts or sections of the cardiovascular system. We are aware of the fact that such simplification may limit the results of our simulation as it does not allow studying phenomena connected to waveform transmission. However, it can be accepted as our main concern in this study was to access the trends and interaction between mechanical ventilation and simultaneous LVAD application in terms of pressures and volume distribution.

References


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