A New Minimum Order Lumped-Parameter Model of Circulatory System for Patients with Suffered Left and Right-Sided Heart Failure

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ABSTRACT

In this study a new minimum lumped electrical model of total circulatory system through numerical solutions to approximate the response of the human circulatory system was presented. Among five existent ventricular elastances as activity function of heart pumps, a more accurate model was chosen as well as the operation of baroreflex system to accommodate variations in heart rate and systemic vascular resistance of a native human circulatory system. The model with extracting the hemodynamic reaction of a patient with heart failure has the ability to study completely the influence of left heart failure on the right ventricle performance. The model can be used to analyze the variations of pressure-volume loop curve for normal hearts and dilated and hypertrophic heart failure patients at rest. The simulation results, in comparison with the clinical data, showed that the model could efficiently simulate the progressed states of heart failure with an acceptable accuracy and among all studied states of disease, hypertrophic LS and RSHF state caused to make critical conditions for the simulated patient.

KEYWORDS

Circulatory system model, lumped, heart failure (*HF*)

1. INTRODUCTION

Heart failure (HF) is a serious condition in which the heart's pumping action is in trouble. While in the early stages, HF may not have any symptoms, in later stages, the patient may have severe symptoms such as: systolic heart failure (SHF) and diastolic heart failure (DHF). In SHF the weakened heart is unable to pump adequate amounts of blood during its contraction. The heart muscles don't contract with enough force, so less oxygen-rich blood is pumped throughout the lungs and body. In DHF the heart may contracts normally, but the ventricles do not relax properly or are stiff therefore less blood enters the heart during normal filling. Symptoms may be identical to SHF or it might be a precursor to SHF. In these situations the chambers of the heart respond by stretching to hold more blood for pumping through the body or by becoming thicker and stiffer. This helps to keep the blood moving for a short while, but then the heart muscle walls weaken and are not able to pump as strongly which lead to damaged heart muscle walls and disorder in pumping and filling. HF has been defined and classified in a number of different ways by physicians and medical institutions. It can be classified by the side of the heart it affects: left sided heart failure (LSHF) and right-sided heart failure (RSHF). In LSHF, the left ventricle (LV) cannot adequately pump oxygen-rich blood from the heart to the rest of the body and in RSHF, the right ventricle (RV) is not pumping the blood to the lungs adequately, which tends to cause fluid build-up in the veins and swelling (oedema) in the legs and ankles. RSHF usually occurs as a direct result of LSHF. Modeling of HF will help to better understand the variations of the abnormal hemodynamic parameters of the ventricles in patients with systolic and diastolic dysfunctions and therefore to diagnose the disease.

Between the presented models of left heart [1]-[3], lumped-parameter models schematically using an electrical equivalent. These models can simulate the output and hemodynamic parameters of normal subjects and HF patients, such as aortic pressure (AOP) and aortic

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flow (AF) effectively. Some researchers developed complete circulatory system models including the systemic and pulmonary circulation [4]-[9] while other researchers studied the simplified models such as windkessel or westkessel models combined with the left heart [4]-[5], [10]-[13]. Wu *et al* [4] presented the total cardiovascular model with muscle pumps and used a simplified model of left heart circulation system (*LHCS*) in order to control rotary pumps for patients with *LSHF*. Ferreira *et al* [10] used a 4th order *LHCS* model to obtain the hemodynamic parameters of normal hearts and patients with systolic dysfunction of *LV*. Chen [11] added influence of central baroreflex control system (*BCS*) to the model (the same circulation model used in [10]) for heart rate (*HR*) and mean arterial pressure (*MAP*) regulation. In this model, the output parameters of heart were obtained for normal subjects and patients with *LSHF* at rest and several exercise conditions. Shi *et al* [6] presented a numerical model for investigation of the human cardiovascular system, where the function of the impaired *LV* is augmented by the pulsatile ventricular assist device.

Authors in the previous works [4], [6]-[8], [10]-[13] have not studied the influence of the *LSHF* on performance of the *RV* (and vice versa) completely. In this paper, a minimum total circulatory model which has the ability to extract the hemodynamic parameters of a normal heart and suffered *HF* patients at rest was presented.

2. CIRCULATORY MODEL

An advantage of representing the model of circulatory system as a global circuit is that Kirchhoff's laws which for node currents and loop voltages can be applied The model used in this paper can be analogously represented by a minimum 7th order electric circuit [4]-[7] and is shown schematically in Fig. 1. [15].

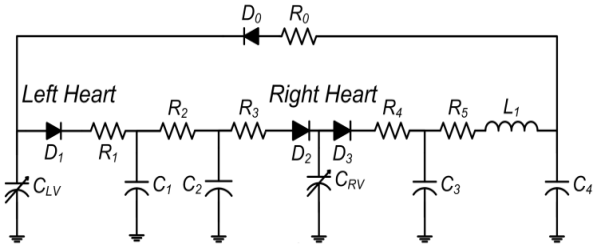

Figure 2

Figure 1: The electric analog of circulatory system model [15]

The annotations and numerical values for each element which correspond approximately to an adult male human weighing 70kg at rest are listed in the Appendix. In this model resistors represent the viscous property of blood flow in vessels, while inductors model the inertia property of blood and capacitors embody the elastic property or compliance of the vessels wall. For mimicing the properties of one-directional valves, ideal diodes are used in series with on resistances and the main pumps of the heart are described as time-varying capacitors.

One way to model the behaviour of the ventricles is using reciprocal of their compliance. The below equation determines the relationship of left ventricular pressure (*LVP*) with the change in ventricular volume, and is defined as elastance function:

$$E_L(t) = \frac{LVP(t)}{LVV(t) - V_0} = \frac{1}{C_{LV}(t)} \quad (1)$$

where *LVV(t)* (ml) and *LVP(t)* (mmHg) represent the variations of *LV* volume and pressure in each cycle respectively and V_0 is a reference volume of *LV*. The description of $E_R(t)$ for *RV* has been defined as similar as expression for *LV*. In order to model *LV* contractility, several analytical expressions for *E(t)* were developed and tested through simulation of the models [3]-[4], [10], [14], [6], [8]. Five of these expressions in this research were selected considering their simplicity and accuracy to mimic the normal ventricular elastance [10]-[11], [5], [14]. With comparative study of the functions finally it was resulted in double hill function was a proper choice as the main model of *LV* capacitor. Using this function [10] from analytical point of view, simulation process cannot go to instability and the response of the system has been gotten faster. The main form of the function is:

$$E(t) = (E_{\max} - E_{\min}).E_n(t_n) + E_{\min} \quad (1)$$

in which $E_n(t_n)$ is expressed as:

$$E_n(t_n) = 1.55\left[\frac{\left(\frac{t_n}{0.7}\right)^{1.9}}{1+\left(\frac{t_n}{0.7}\right)^{1.9}}\right]\left[\frac{1}{1+\left(\frac{t_n}{0.7}\right)^{21.9}}\right] \quad ,0 \le t \le t_c$$

$$t_n = \frac{t}{T_{sys}}, T_{sys} = 0.2 + 0.15t_c, t_c = \frac{60}{HR} \quad (3)$$

where *HR* represents heart rate. Notice that in (2), *E(t)* is the re-scaled form of $E_n(t_n)$. In (3) E_{min} (mmHg/ml) corresponds to the minimum elastance value and E_{max} (mmHg/ml) models contraction power or the active value of the ventricle reached in a cardiac cycle. In a normal heart at rest in which *HR* was set in 75beats per minute (bpm), T_{sys} was calculated to be 0.32sec, the parameters for *LV* were set to E_{min}=0.07, E_{max}=2.7, for *RV* E_{min}=0.08, E_{max}=1mmHg/ml, total blood volume that *LV* and *RV* able to keep in their chamber were assumed 250 and 275ml respectively.

For analysing the presented model (Fig. 1), *MATLAB* was used. In transient phase of simulation, the model after 5 to 6 heart cycles of calculation reached periodic solution. The P-V loop curve of the ventricles for a normal heart is shown in Fig. 2 and the simulated values

of the heart parameters are listed in table 1. As shown in Fig. 2, the peak of the right ventricular pressure (*RVP*) was calculated to be 32.5mmHg which was nearly in accordance with 30mmHg in a normal human. Left and right atrial pressure changed between 5 to 12.55mmHg and 4.7 to 7.17mmHg respectively. According to the results of the table 1, with applying double hill function, the ejection fraction (*EF*) of both ventricles was set between 50-75% similar to the *EF* of the ventricles of a native human. The peak of the *RVP* was calculated to be 32.5mmHg which was nearly in accordance with 30mmHg in a normal human. Left and right atrial pressure changed between 5 to 12.55mmHg and 4.7 to 7.17mmHg respectively.

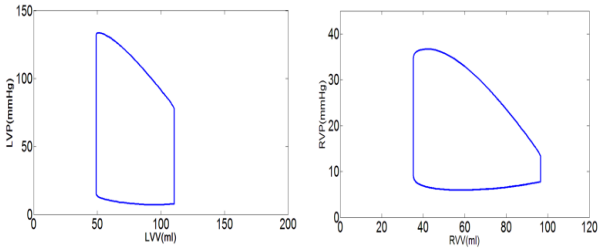

Figure 2: Pressure-volume (P-V) relationship curve of left and right ventricle for a normal heart at rest

TABLE 1: SIMULATED OUTPUT PARAMETERS OF A NORMAL HEART AT REST

Hemodynamic (unit)	Normal heart(Rest)
AOP_{max}/AOP_{min} (mmHg)	135/84
MAP (mmHg)	101
EDV[1]/ESV[2] of LV (ml)	106.5/49.5
Stroke Volume(SV) (ml)	57.5
EDV/ESV of RV (ml)	88.5/31
CO[3] (lit/min)	4.7309
EF of LV (%)	54
EF of RV (%)	64.97

A. HF in the early and progressed stages

Hypertrophic and dilated *HF* is the progressed diseases of the *DHF* and *SHF*. In hypertrophic *HF*, the heart muscles become enlarged, which results in a thickening of the ventricles wall and causes their shrinkage. People with hypertrophic *HF* are also at risk of cardiac sudden death; because the heart muscles cannot get enough oxygen to meet their needs and heart cells begin to die. Dilated *HF* is a state in which the ventricles of heart are relaxed further from normal situation; therefore, an amount of blood remains in the ventricles during each cycle. Dilated *HF* is a state in which the ventricles of heart are relaxed further from normal situation; therefore, an amount of blood remains in the ventricles during each cycle. Table 2 lists the changed parameters of the new model in dilated and hypertrophic *HF* accordance with the clinical data used in [11]. In this table *SVR* represents the systemic vascular resistance between aorta and right atrium which denoted with R_2 in the main model (Fig. 1). Fig. 3 shows the simulation results of the P-V loop curves of a normal heart in comparison with the patients who suffered from dilated *LS* and *RSHF*.

As shown in Fig. 3(a) the systolic and diastolic pressures of aorta in dilated *LSHF* with *EF* of 16.51% than the systolic state increased but also the stroke volume of the ventricle decreased slightly while In P-V loop of *RV* the shape of curve didn't change significantly and only the peak of *RVP* reached to about 35mmH g. In dilated *RSHF* the parameters *ESV*, *EDV* of *RV* with decreasing of *RV* contraction increased (*EF*=34.31%) but also in P-V loop of *LV* the curve was set between the curves of normal heart and systolic *RSHF*. In this state *CO* decreased to 4.3309lit/min. If the simulated patient suffered from both states of dilated *HF*, in P-V loop curve of *LV*(Fig. 3(c)) with decreasing the parameters *ESV* and *EDV*, *EF* of the ventricle reached to the minimum value of 15.38% but in P-V loop curve of *RV*, *ESV* and *EDV* increased significantly (*EF*=23.21%) which in led to decrement of *CO* to 2.6372lit/min.

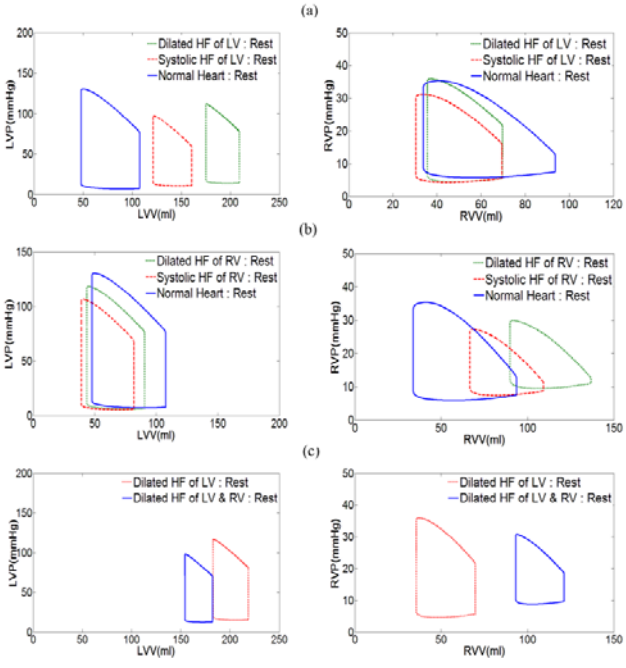

Figure 3: comparison of P-V loop curve in the early (systolic) and progressed stage (dilated): (a) left heart failure (b) right heart failure and (c) left and right heart failure

[1] End Diastolic Volume (EDV)
[2] End Systolic Volume (ESV)
[3] Cardiac Output (CO)

Fig. 4 shows the simulation results for the hypertrophic state. In Fig. 4(a) the systolic and diastolic pressures of aorta compared to the diastolic state decreased further and expected that there may not be a small change in *EF* of *LV*, but in this state *EF* from value of 54.9% in diastolic state decreased to 45.59% and caused to decrement of *CO* to about 2.8143lit/min. In these conditions the peak of *RVP* changed slightly and with decreasing *EDV*, *EF* of the ventricle decreased to 48.94%. In hypertrophic *RSHF* (Fig. 4(b)), the parameters *ESV* and *EDV* of *LV* in comparison with hypertrophic *LSHF* didn't change significantly but *MAP* decreased further and reached to 65mmHg (*CO*=2.88lit/min). In hypertrophic *LS* and *RSHF* as shown in Fig. 4(c), ESV and *EDV* of *LV* (*SV*=16.4ml) and *MAP* decreased more than hypertrophic *LSHF* and led to decrement of *SV* of *RV* from value of 57.5ml in a normal heart to about 18ml but *EF* of *LV* and *RV* was calculated to be 42.59 and 45.94% respectively near *EF* of hypertrophic *LSHF*.

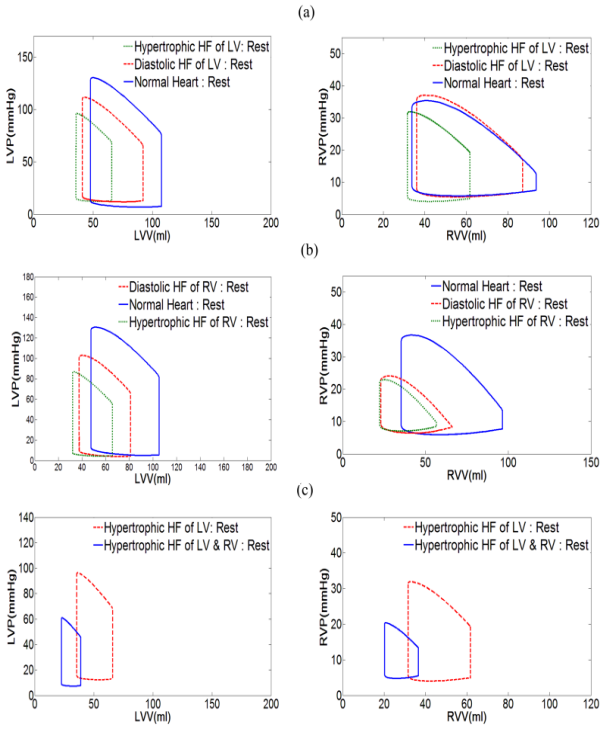

Figure 4: comparison of P-V loop curve in the early (diastolic) and progressed stage (hypertrophic): (a) left heart failure (b) right heart failure and (c) left and right heart failure

3. DISCUSSION AND CONCLUSION

Authors in the previous works [4]-[7], [10]-[11] have used the mathematical equations in state space to simulate their lumped models but in our study, the *simscape* library of the *MATLAB* was used for analysing the model. In this method, step of the simulation process and number of consecutive zero crossing relative tolerance for control of solver diagnostic were selected automatically. Using this method due to the complexity of the left and right capacitor and the valves of the model (Fig. 1), stability of state equations of system from analytical point of view is guaranteed. In order to simulate efficiently, proper choice of a solver was essential. Ode 15s (stiff/NDF) solver as a BDF method with relative tolerance of 1E-3 and number of consecutive zero crossing allowed of 10000 was used for simulating the model. In this study we presented a new lumped model of circulatory system with the minimum electrical order between the all presented cardiovascular models [4-8] which can be used for simulating the hemodynamic parameters of a normal heart and patients with severed HF at rest.

In order to evaluate performance of the ventricles effectively in simulating the P-V loop curve, the capability of five elastances *double hill*, *sinusoidal*, *modified exponential*, *gaussian* and *trapezoidal* for choosing a best one, instead of the time-varying capacitors of the primary model, was extracted. In sinusoidal and modified exponential functions the end systolic pressure of *RV* was calculated to be about 35.9mmHg which because of comparison with value of 30mmHg in a native human and also for their minimum computed *CO*, these functions weren't acceptable. Trapezoidal and gaussian functions from the curve schema point of view couldn't be acceptable and finally the double hill function with calculated reference volume of 15ml was a proper choice as the ventricles pump and has the ability that is used instead of the right and left ventricular pump.

Table 2: Changed parameters of the ventricles of the main model in severe heart failure at rest

Parameter(unit) at Rest	Normal heart	Dilated HF of LV	Dilated HF of RV	Dilated HF of LV & RV	Hypertrophic HF of LV	Hypertrophic HF of RV	Hypertrophic HF of LV & RV
E_{min} of LV/RV (mmHg/ml)	0.07/0.08	0.07/0.08	0.07/0.08	0.07/0.08	0.2/0.08	0.07/0.15	0.2/0.15
E_{max} of LV/RV (mmHg/ml)	2.7/1	0.64/1	2.7/0.33	0.64/0.33	2.7/1	2.7/1	2.7/1
SVR (mmHg.sec/ml)	1.29	1.83	1.29	1.83	1.83	1.5	2.04
HR (bpm)	75	86	86	86	86	86	86
Total volume of the LV/RV (ml)	250/275	345/275	250/379	345/379	155/275	250/170.5	155/170.5
R_5 (mmHg.sec/ml)	0.2	0.27	0.2	0.27	0.27	0.25	0.37

In [4], [7]-[8], [10]-[11] several authors has used lumped-parameter model and studied the influence of *HF* in decrement of *CO*, pressure of aorta and variations of P-V loop during progression of the disease. Shi *et al* [6] in their model have decreased the left ventricular contraction (E_{max}) and showed that *MAP* reached to 53mmHg. But results of this study shows that in the critical conditions of progression the disease, in diastolic and systolic dysfunction synchronously, *MAP* decreased to 58.8mmHg and only in hypertrophic *HF* of both ventricles was computed to be 50.33mmHg with *CO* of 1.5766lit/min. Chen [11] added the *BCS* to a 4th order lumped model and studied the changed parameters of left heart circulatory system in normal and *HF* conditions. In this research, we showed that without adding an influence of central baroreflex control system to the model, the output parameters of the model has been simulated as well as the similar work in [11] and also by using this method of analysis due to the improvement of the response of the simulated model, the influence of *LSHF* on to the *RV* operation and vice versa was completely studied.

In diastolic and systolic *HF* of *LV* occurred synchronously, the minimum and maximum pressure of blood in respiratory circulatory system (Fig. 5(a)) because of the decrement of stiffness power of the ventricle, increased from 10-32.5mmHg to about 26.8-38.8mmHg which may lead to disease of edema of the lungs. Also in Fig. 5(b) the left atrial pressure (*LAP*) increased to about 23-27mmHg while right atrial pressure (*RAP*) was set about 5mmHg. These results implicate the control necessity of the atrial pressures especially pressure of left atrium to prevent from atrial diseases in patients with both states of *LSHF*. Between the all studied states of disease, hypertrophic *LS* and *RSHF* state caused to make critical conditions for the simulated patient. In this state, *CO* and peak of *RVP* reached to the minimum value of 1.5766lit/min and 20mmHg respectively.

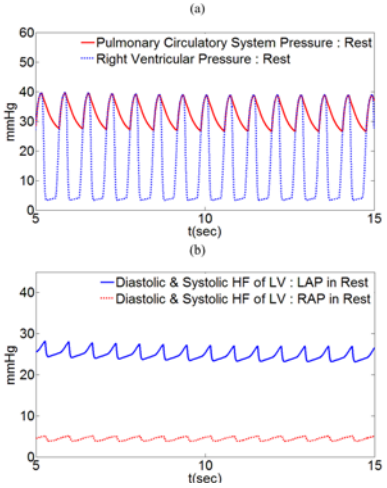

Figure 5: Simulation results of pressure curves in diastolic and systolic heart failure synchronously (a) pulmonary circulatory system and right ventricle and (b) left and right atrium

4. Future Work

With the total circulatory system (*TCS*) with the minimum order electrically, modelling of diseases which engender after *HF* can be considered completely. Study of exercise activity in the suffered *HF* patients using the *TSC* models, adding the left ventricular assist devices (*LVADs*) models to the new model for therapy of this disease and study pump performance on the variations of the heart parameters, are the current topics that the authors are studying.

5. Acknowledgments

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6. APPENDIX

PARAMETERS OF THE MODEL WITH ITS VALUES

C_{LV}: capacitance of left ventricle(time-varying), $C_1 = 0.75 ml/mmHg$: capacitance of aorta, $C_2 = 25 ml/mmHg$: capacitance of systemic vein and right atrium, C_{RV}: capacitance of right ventricle(time-varying), $C_3 = 1.5 ml/mmHg$: capacitance of pulmonary artery, $C_4 = 5 ml/mmHg$: capacitance of pulmonary vein and left atrium; $R_0 = 0.004 mmHg.sec/ml$ resistance of pulmonary vein and left atrium(resistance of mitral valve), $R_1 = 0.004 mmHg.sec/ml$: resistance of aortic valve, $R_2 = 1.29 mmHg.sec/ml$: systemic vascular resistance, $R_3 = 0.004 mmHg.sec/ml$: resistance of systemic vein and right atrium(resistance of tricuspid), $R_4 = 0.002 mmHg.sec/ml$: resistance of pulmonary great artery(resistance of pulmonary valve), $R_5 = 0.2 mmHg.sec/ml$: resistance of pulmonary vascular; D_0: mitral valve, D_1: aortic valve, D_2: tricuspid valve, D_3: pulmonary valve; $L_1 = 0.002 mmHg.sec^2/ml$: inductance of blood in pulmonary circulation.

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