

A 0D representation of the arterial network incorporating the heart

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Abstract. Since Otto Frank first introduced the Windkessel (0D) model in 1899 to reproduce the arterial network, various versions were constructed which differ through their number of elements and their disposal. The purpose of this paper is to couple the left heart model given by M. Danielsen and J.T. Ottesen to a classic three-element 0D representation of the arterial network and compare the results with the output of a 13-elements arterial model coupled with the heart. This is done in order to prove that no great benefit is gained when increasing the number of elements of the Windkessel model if the input satisfies normal physiological conditions. After confirming that the model works properly, a simulation is performed with parameters specific for a hypertensive person. The output is assessed against results specific for a normal person and the differences are further analysed.

1. Introduction

A breakthrough in understanding the blood motion came in 1899 from Otto Frank [1]. One of his approaches was to treat the elements of the arterial system as compartments. This method received the name of Windkessel model and it has developed constantly since its introduction. The advantage of this method is simplicity, offering a rough estimate of the average arterial pressure per each compartment. As its implementation is straightforward, it is often used in modelling the most complex element of the cardiovascular system, namely, the heart. In this paper, the model used by M. Danielsen and J.T. Ottesen [2] is implemented together with a three-element Windkessel (RCR) model of the arterial system. As shown by Kind et al. [3], the RCR model manages to capture with high accuracy the main features of the cardiovascular system, offering a good match with in vivo measurements. Once these two models are constructed, they are coupled and the output is inspected with respect to the results obtained by M. Danielsen and J.T. Ottesen [2], where the heart is attached to a 13-elements Windkessel model of the arterial system. Both sets of data are compared and the benefits of the two types of coupling highlighted. Moreover, due to the high flexibility of the model, various physiological / pathological conditions can be captured. Here, the cardiac cycle of a normal individual is compared with the one of a hypertensive person (with less elastic arteries) in order to emphasize the amount of work done by the human heart in the two cases.



2. Methodology

2.1. Heart Model

A visualization of the whole heart is offered in figure 1 [4] together with the name for each of its main components. This specific nomenclature is used throughout the paper.

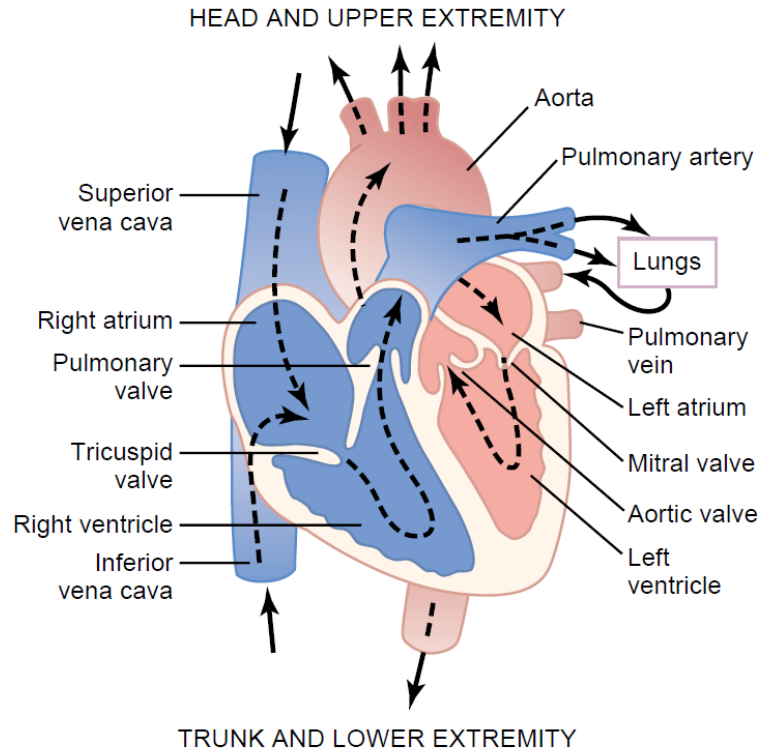


Figure 1. Structure of the human heart, the blood flow through the heart chambers and their specific valves. Reproduced from figure 9-1 of [4, pp 104].

The modelling of the left heart follows the procedure described in [2]. The basic idea consists in varying the elastance (E_{lv}) of the left ventricle in time. The time dependency is given by:

$$E_{lv}(t) = E_{min,lv}(1-\phi(t)) + E_{max,lv}\phi(t), \quad (1)$$

where

$$\phi(t) = \begin{cases} 0 & \text{for } 0 \leq t < t_s \\ a_\phi \cdot \sin\left(\frac{\pi(t-t_s)}{t_c}\right) - b_\phi \cdot \sin\left(\frac{2\pi(t-t_s)}{t_c}\right) & \text{for } t_s \leq t < t_c + t_s \\ 0 & \text{for } t_c + t_s \leq t \leq t_h \end{cases}$$

In equation (1), $E_{min,lv}$ and $E_{max,lv}$ are the minimal and maximal values of the left ventricle elastance which occur during the diastole and systole, respectively. In the definition of ϕ , t_s represents the time when the systole starts, which corresponds with the closing of the mitral valve. The constants a_ϕ and b_ϕ are some specific parameters of the model and t_c represents the time at which the elastance becomes constant. In [5] a linear relation is offered between the t_c and the heart period t_h as described by:

$$t_c = \kappa_0 + \kappa_1 \cdot t_h, \quad (2)$$

where the κ_0 and κ_1 are constants.

The numerical values of all parameters used for modelling the elastance are given in table 1. This term was created through the analogy of a capacitor as a spring and it is also used for related quantities in some other energy domains, including fluid dynamics. The generated plot of the elastance (the ratio of pressure over volume) versus time is offered in figure 2. The periodic elastance model is defined by the cardiac contractile properties of the left ventricle. This is a characteristic of the heart and the parameters vary from person to person. Their values are taken to be equal with the ones from [2] in order to guarantee a good reproduction. However, they can be changed, depending on the individual's heart conditions.

Table 1. Constants used in the definition of the left ventricle elastance. The values are taken from [2].

Parameter	Value	Unit
a_ϕ	0.9	[-]
b_ϕ	0.25	[-]
κ_0	0.29	[-]
κ_1	0.2	[-]
$E_{\max,lv}$	2.49	mmHg/ml
$E_{\min,lv}$	0.049	mmHg/ml
t_s	0.05	s
t_h	0.8	s

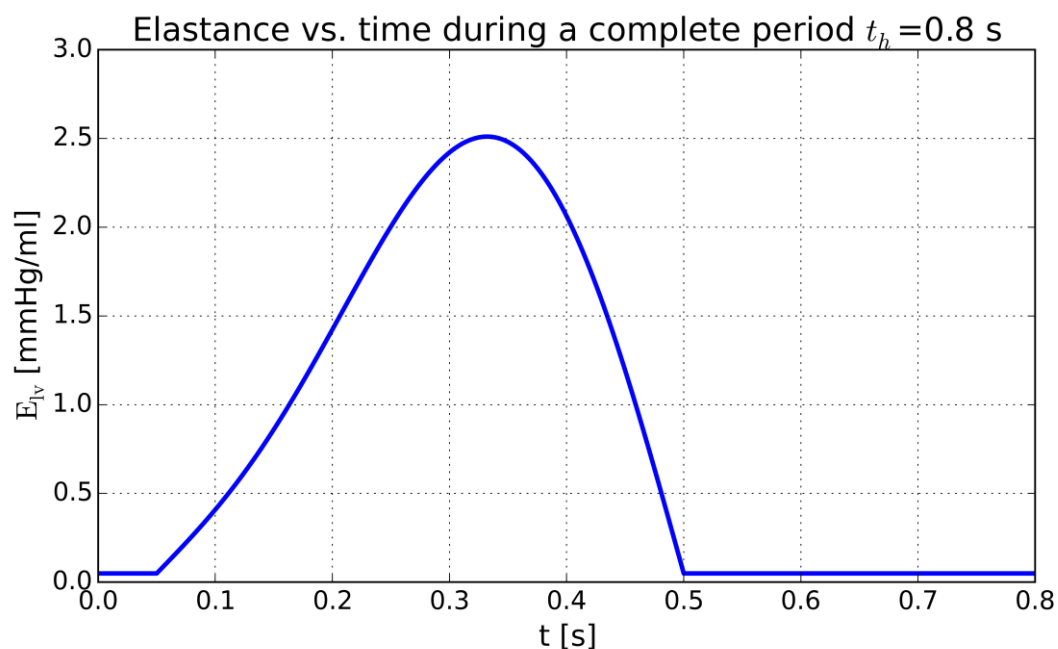


Figure 2. Elastance of the left ventricle as a function of time. Reproduced based on figure 6.4 from [2, pp 145].

The fluid mechanics domain is often converted to the electrical domain because network analysis in electricity is highly developed. Traditional for cardiovascular simulation, various properties of the left ventricle are modelled using lumped parameters. The inertia of blood is taken into account by including an inductance, while the viscous properties of blood are modelled using resistances. The equivalent electrical circuit for the heart is offered in figure 3. The valves are assimilated by diodes, while the elastance is equivalent to a variable capacitor due to its dependence on time.

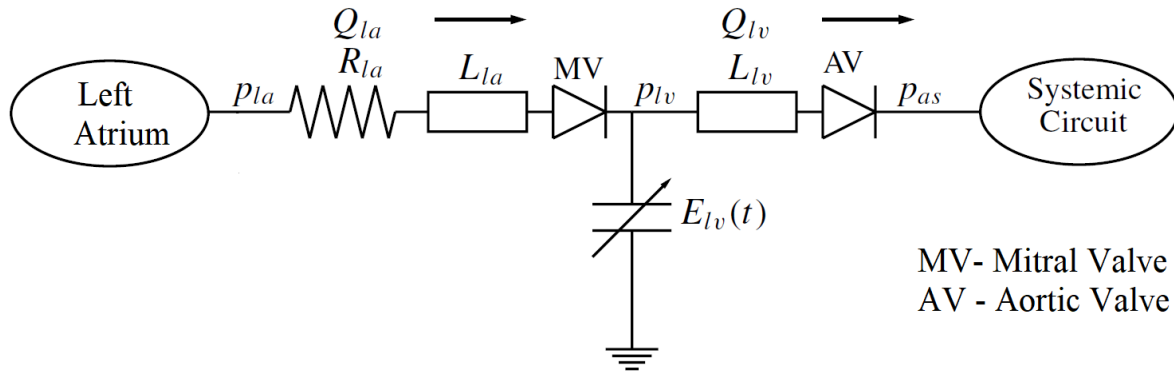


Figure 3. The 0D model (Windkessel) of the left heart. Reproduced with slight changes from figure 6.3 of [2, pp 141].

The motion of the blood through the left heart is described by equations (3) to (12).

Systolic equations

$$Q_{la} = 0 \quad (3)$$

$$p_{lv} = E_{lv}(V_{lv} - V_{d,lv}) \quad (4)$$

$$\frac{dV_{lv}}{dt} = Q_{la} - Q_{lv} \quad (5)$$

Aortic valve closed:

$$Q_{lv} = 0. \quad (6)$$

Aortic valve opened:

$$\frac{dQ_{lv}}{dt} = \frac{1}{L_{lv}}(p_{lv} - p_{as}) \quad (7)$$

Diastolic equations

$$Q_{lv} = 0 \quad (8)$$

$$p_{lv} = E_{lv}(V_{lv} - V_{d,lv}) \quad (9)$$

$$p_{la} = E_{min,lv}(V_{max,lv} - V_{d,lv}) \quad (10)$$

Mitral valve closed:

$$Q_{la} = 0. \quad (11)$$

Mitral valve opened:

$$\frac{dQ_{la}}{dt} = \frac{1}{L_{la}}(p_{la} - p_{lv}) - \frac{R_{la}}{L_{la}}Q_{la} \quad (12)$$

In the two sets of equations, the Q is the blood flow rate, V the volume, p the pressure, L the inductance and R the resistance. As before, the subscripts lv and la stand for left ventricle and left atrium, respectively. The aortic pressure is denoted by p_{as} and it is dependent on the model used for the systemic circulation. Here, the heart is coupled with the classical three-element Windkessel (RCR) arterial model.

The boundary conditions needed to solve the differential equations are the maximum left ventricle volume – $V_{\max,lv}$, the left ventricle volume at 0 pressure – $V_{d,lv}$ and the pressure at time 0 – $p_{lv}(0)$, which are specified in table 2 together with other constants. The numerical values are taken from [2], but they can be modified in order to be specific for a certain individual.

Table 2. Constant terms required to solve the equations (3) to (12).

Term	Value	Unit
$V_{\max,lv}$	127	ml
$p_{lv}(0)$	80	mmHg
$V_{d,lv}$	10	ml
$\widetilde{V}_{lv,b}$	2.0	ml
R_{la}	0.000089	mmHg·s/ml
L_{lv}	0.000416	mmHg·s ² /ml
L_{la}	0.00005	mmHg·s ² /ml

For the mitral valve, the closing and opening depends only on the difference in pressure between the left atrium and the left ventricle. If the pressure in the former one is higher the valve opens and remains opened till the difference in pressures becomes negative ($p_{la} - p_{lv} < 0$). The same mechanism works also for the opening of the aortic valve. However, the closing for this valve is more complicated due to the fact that the valve allows for some blood flow to return to the heart. The maximum volume allowed back in the left ventricle is noted $\widetilde{V}_{lv,b}$ and its value is given in table 2. This quantity needs to be increased when modelling various pathological cases (bicuspid aortic valve, calcified valve, etc.).

2.2. Arterial Model

The arterial model attached to the heart is a simple three-element Windkessel model (RCR), which is outstanding through its simplicity and ability to capture the main features of the arterial network. The circuit is shown in figure 4, where R_{0s} is the aortic resistance, C_a is the arterial compliance and R is the peripheral resistance. $P_a(t)$ stands for the aortic pressure, $P_p(t)$ denotes the pressure across the compliance while $Q_a(t)$ is the aortic flow rate. Their equivalence in electrical terms would be voltage for pressure and current for the flow rate.

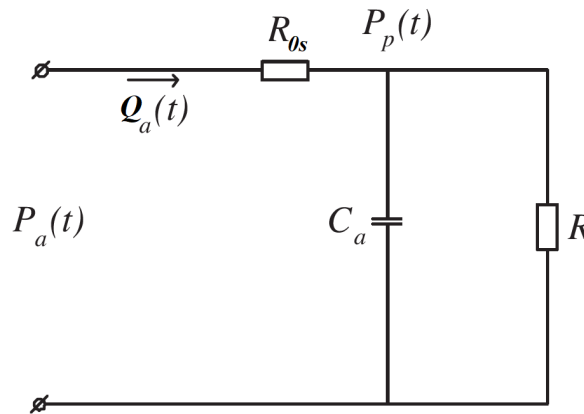


Figure 4: The three-element Windkessel model (RCR). Reproduced with slight modification from figure 1 of [3, pp 1533].

The pressure and the flow are functions of time, while the parameters R_{0s} , R and C_a are constant. These parameters are obtained through experiments and vary from person to person. Based on the network theory, the coupled equations which characterize the circuit are easily derived as described in [3] and given by:

$$\begin{cases} \frac{dP_p(t)}{dt} = -\frac{1}{RC_a} P_p(t) + \frac{1}{C_a} Q_a(t) \\ P_a(t) = P_p(t) + Q_a(t)R_{0s} \end{cases} \quad (13)$$

These equations are in differential form and they are solved numerically using the explicit Euler time integration. The coupling between the heart and the arterial model consists in a constant exchange of information between the two models. The flow exiting the left ventricle is used as input boundary condition for the Windkessel model while the pressure in the aorta is used as outflow condition for the heart. Figure 5 shows schematically the coupling between the two models, offering an overview on the inputs and how the output of each model is assessed.

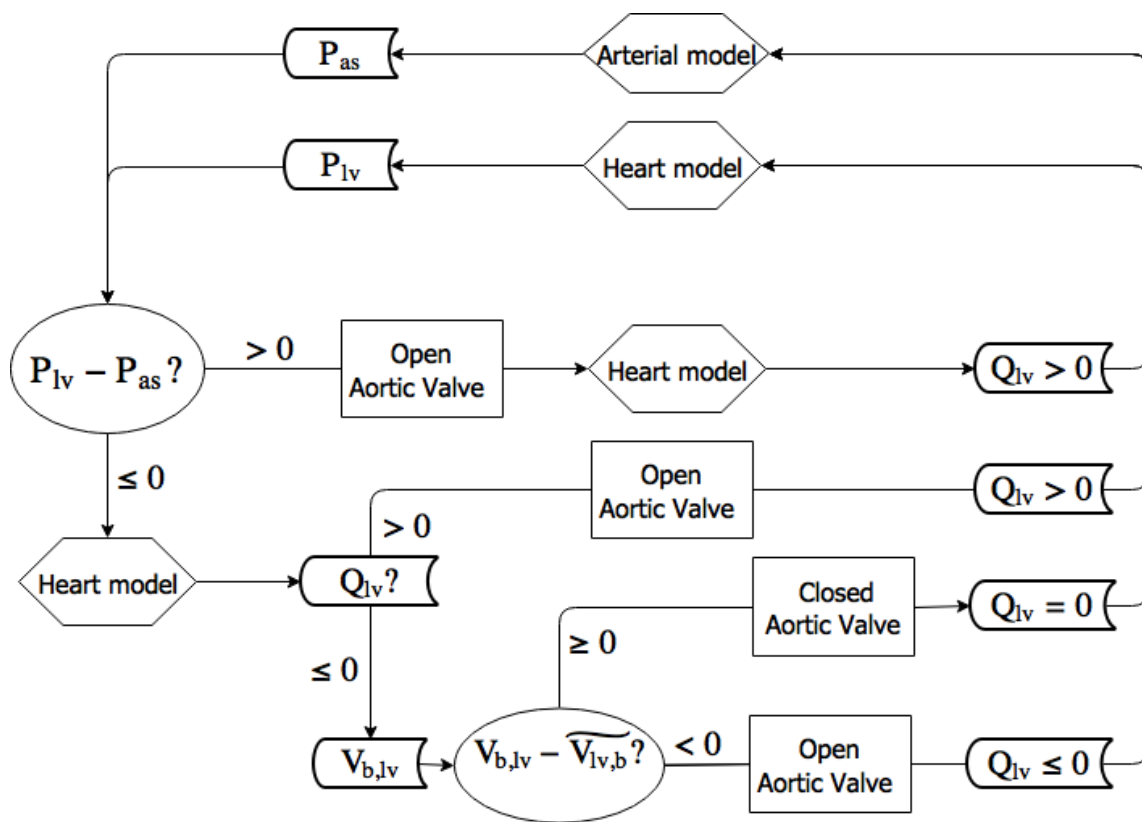


Figure 5. Schematic representation of the coupling between the heart and the aortic model.

3. Results and discussion

3.1. Verifying the model

The two specific plots which characterizes the cardiovascular system are the aortic pressure versus time and the cardiac cycle. The former one describes the arterial system, while the latter one offers an image of how much work the heart is performing in each cycle. Firstly, the constructed model of the heart coupled with the RCR model is verified against literature data. The data used for verifying the results is acquired from the plots of [2] using WebPlotDigitizer [6]. As the lumped description of the circulation in [2] consists of a 13-elements model, the magnitude of the Windkessel model components taken initially from [3] needs to be slightly changed in order to have a better match between the two circuits. The best correlation is found for $R_{0s} = 0.0568 \text{ mmHg}\cdot\text{s}\cdot\text{ml}^{-1}$, $C_a = 1.72 \text{ ml}\cdot\text{mmHg}^{-1}$ and $R = 1.075 \text{ mmHg}\cdot\text{s}\cdot\text{ml}^{-1}$.

In figure 6, it can be seen clearly the good correlation between the generated results and the reference data. The area enclosed by the first plot of figure 6 determines the total work done by the left ventricle in one cycle. This is computed to be 1.1 J of work for the implemented simulation. This value agrees with the estimation from [7], where is calculated that during one cardiac cycle the left ventricle performs on average 1 J of work.

The generated results resemble accurately the reference data from [2] in all the stages of the cardiac cycle. An interesting feature of this model is the ability to allow for some amount of blood to flow back in the ventricle before the aortic valve is closed. This causes the plot to have a bump in its left upper part. Initially, the ventricular volume reaches its minimum of 52.8 ml, but because of the flow coming back into the heart, the volume slightly increases to 54.8 ml, when the aortic valve closes. This limit of 2 ml chosen as the maximum volume allowed to flow back in the ventricle was taken from [2], but it can be changed depending on the pathology of the implemented left heart. For the other graph, the resembling is acceptable taken into account that a more complex arterial model is used in [2]. The dicrotic notch (a secondary upstroke in the descending port of a pulse training corresponding to the transient increase in aortic pressure upon closure of the aortic valve) at the end of the systole (0.4 s) caused by the wave reflection pressure cannot be predicted by the simple RCR model. This is an important feature, as the pressure in the first part of the diastole (from 0.4s) is underestimated by the model used. However, this is a region of low pressure, where precision is less critical. In the middle of the systole, where an accurate prediction is important due to the high pressure in the region, the model behaves very well, the pressure being slightly overestimated.

Overall, the match between the generated results and the reference data from [2] is very good. Even if the implemented simulation uses only a three-element Windkessel model for the circulation with respect to the 13-elements model applied in [2], it produces a fast and qualitative output which reproduces the main features of the cardiac cycle.

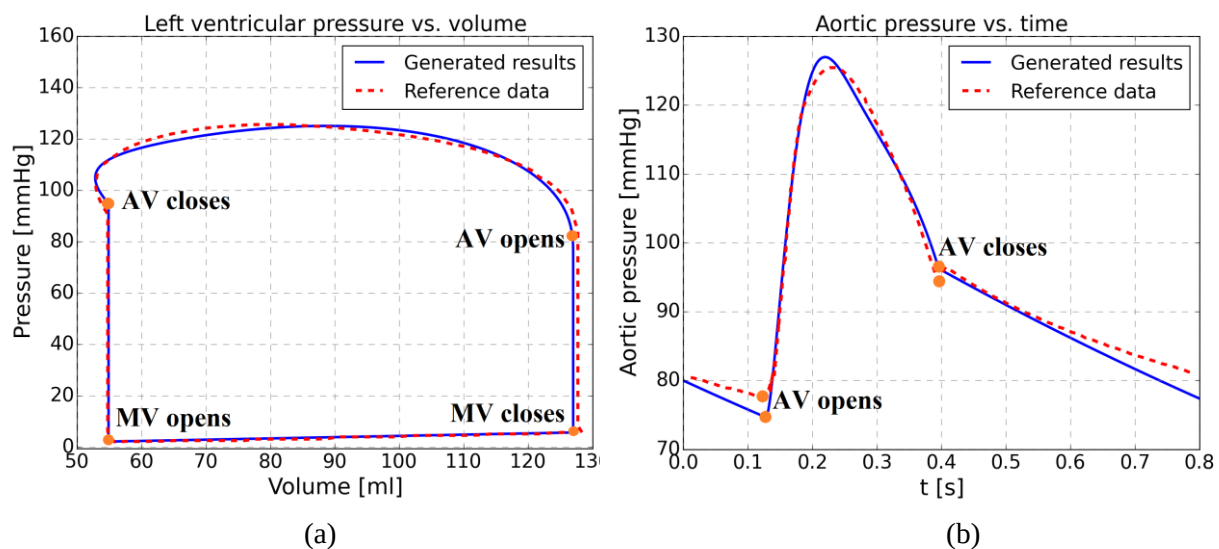


Figure 6. The left ventricular pressure versus volume (a). The aortic pressure in one cardiac cycle of 0.8 s (b). The AV and MV stand for the aortic and mitral valve, respectively.

The reference data is taken from [2].

3.2. Hypertensive case

In order to test the model for the pathological cases, the parameters of the arterial system (RCR) are modified to be specific for a hypertensive person. The specific values are taken from [3] for both the normal and hypertensive case and they are offered in table 3.

Table 3. The parameters of the RCR model of the arterial system for a normal and hypertensive person [3].

Case	Term	Value	Unit
Normal	R_{0s}	0.033	$\text{mmHg}\cdot\text{s}\cdot\text{ml}^{-1}$
	C_a	1.5	$\text{ml}\cdot\text{mmHg}^{-1}$
	R	0.95	$\text{mmHg}\cdot\text{s}\cdot\text{ml}^{-1}$
Hypertensive	R_{0s}	0.05	$\text{mmHg}\cdot\text{s}\cdot\text{ml}^{-1}$
	C_a	0.7	$\text{ml}\cdot\text{mmHg}^{-1}$
	R	1.4	$\text{mmHg}\cdot\text{s}\cdot\text{ml}^{-1}$

In figure 7 the graphs describing the heart motion and the arterial pressure in time are offered. In the first plot, the four most important points of the cardiac cycle are highlighted, namely the moments when the two valves of the left ventricle close and open.

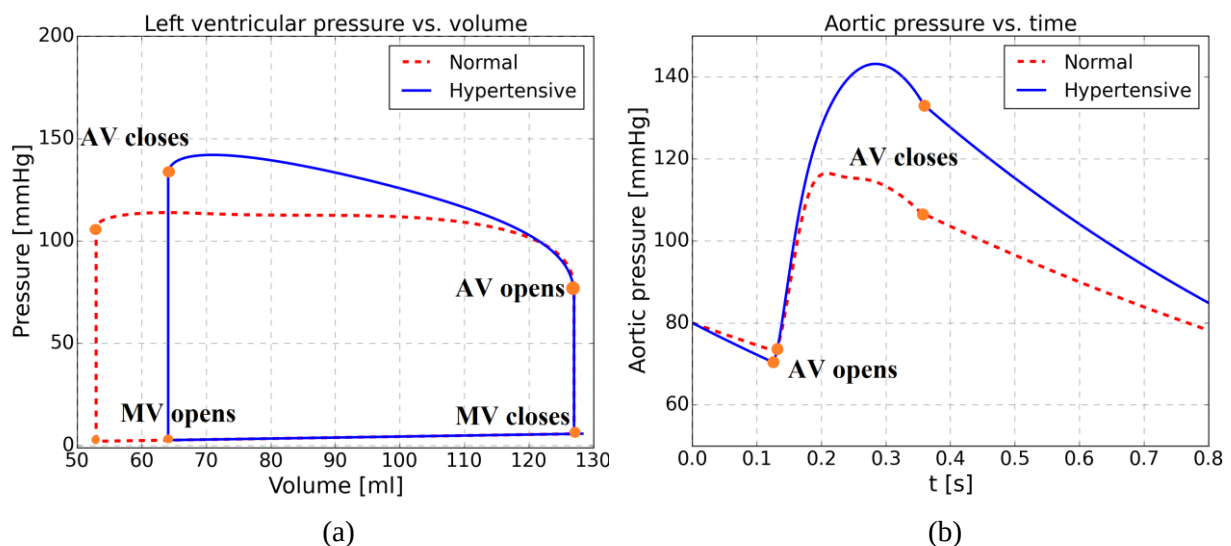


Figure 7. The left ventricular pressure versus volume (a). The aortic pressures in one cardiac cycle (b). The AV and MV stand for the aortic and mitral valve, respectively.

The model of the human heart was considered to have the same parameters for both the hypertensive and normal conditions. This is a valid assumption especially before the left ventricle starts to overdevelop. Keeping the parameters constant is the reason why the aortic valve opens and the mitral valve closes at the same pressure and volume for both cases. For the other two important points on the cardiac cycle, the difference between the normal and the hypertensive simulation is significant. As seen in both graphs of figure 7, due to higher pressure in the aorta for the hypertensive case, the aorta valve closes before the heart is able to deliver the required quantity of blood in the arterial system. The aortic valve closes after the heart pumped into the arterial system just 64.2 ml of blood with respect to 74.6 ml delivered by the heart of a healthy person. Even if the difference in the quantity of the pumped blood is significant lower (by 14%), the work done by the heart of a hypertensive person (1.01 J) is only 3% less than for a healthy individual (1.04 J). This means, that due to hypertension, the heart becomes less efficient and it is unable to pump the necessary quantity of blood to the body without increasing the pulse. For this specific model, an increase of 16% (from 75 to 87 beats per minute) will be required in order to keep the same quantity of blood pumped into the body. This causes over time left ventricular hypertrophy which needs to be taken into account in the heart model. However, this is beyond the scope of this paper, where the differences are inspected before the anatomy of the heart will adapt to the hypertensive conditions.

From an engineering point of view, the heart is analogous to a mechanical pump. For both of them the interaction between fluid flow and valve dynamics plays an essential role in the overall behaviour of the system.

4. Conclusions

The purpose of this research was to build and test a simple Windkessel model of the cardiovascular system in various conditions. The model of the heart was taken from literature, while the arterial system was taken into account as a RCR circuit. The coupling between the two systems allows for aortic valve regurgitation, feature which makes the model more flexible. The results match almost perfectly, the verification data confirming that the model works properly. With the model built, a comparison was conducted between a healthy and a hypertensive individual. For the latter one, an important decrease in heart's efficiency was observed, the heart consuming 15.7 J/l of blood pumped, while for a normal person this value does not exceed 13.9 J/l. As both people need the same amount of blood, in the hypertensive case, the heart needs to beat 16% faster in order to compensate for the lower amount pumped in one cardiac cycle. Overall, the performed simulation worked properly, proving its ability to capture the main features of the cardiovascular system in both normal and pathological conditions. Further work is in progress to improve the model of the arterial system in order to acquire the response of the baroreceptors.

5. References

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