

Cardiovascular System Modeling Using Modified Nodal Analysis and Trapezoidal Rule

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Abstract— the heart and circulatory system (also called the cardiovascular system) make up the network that delivers blood to the body's tissues. With each heartbeat, blood is sent throughout our bodies, carrying oxygen and nutrients to all of our cells. Numerical techniques for the simulation of the human cardiovascular system can provide valuable information to physicians, surgeons and pharmaceutical companies. This paper presents a simple cardiovascular model in MATLAB using a circuit simulation approach. The model leverages a fluid dynamic-electrical analogy, which provides equivalent circuits for all major parts of the circulatory system. The numerical results of the blood pressure, volume and flow are obtained at different parts of the body, and there has been satisfactory agreement between the model and experimental results.

Keywords—Cardiovascular system, lumped element model, analog electrical circuit, modified nodal analysis, MATLAB, trapezoidal rule

I. INTRODUCTION

Heart disease and stroke are two of the three leading causes of death in Canada. Every 7 minutes in Canada, someone dies from heart disease or stroke. In 2008 cardiovascular disease accounted for 29% of all deaths in Canada (69,703 deaths – or more than 69,500). Heart disease and stroke costs the Canadian economy more than \$20.9 billion every year in physician services, hospital costs, lost wages and decreased productivity [1].

Having a good model of human cardiovascular system can be useful for a deeper understanding of the complex process occurring in the heart and blood vessels under normal and pathological conditions. Through this model development process, scientists and physicians would be able to gain and verify new knowledge in cardiovascular system quantitatively. These models can help clinicians to initiate new techniques for diagnosis and treatment. Implementing these models with computers also facilitates a cost-effective way for medical education, clinical training, as well as medical device development.

II. THE CARDIAC CYCLE AND ANATOMY

The transport of blood is accomplished through two circuits within the cardiovascular system: the systemic and pulmonary circuit. Each circuit begins and ends at the heart. The heart has four chambers that are enclosed by thick, muscular walls. The bottom part of the heart is divided into two chambers called the **right** and **left ventricles**, which pump blood out of the heart. The upper part of the heart is made up of the other two

chambers, called the **right** and **left atria**, which receive the blood entering the heart.

There are two pairs of valves in the heart to ensure unidirectional blood flow. Two valves separate the atria and the ventricles. They are called **tricuspid valve**, which separates the right atrium from the right ventricle, and the **mitral valve**, which separates the left atrium and the left ventricle. The other two valves separate the ventricles and the large blood vessels that carry blood leaving the heart. These valves are called the **pulmonic valve**, which separates the right ventricle from the **pulmonary artery** leading to the lungs, and the **aortic valve**, which separates the left ventricle from the **aorta**, the body's largest blood vessel [2].

The contraction and relaxation of the heart muscles in the heart chamber causes the mitral and aortic valve to open and close due to pressure differences. The five phases of cardiac cycle are as follows [3]:

1. **Isovolumic ventricular contraction**, when all are closed. Pressure is being built up in the ventricles until the pressure rises sufficiently above the aortic and pulmonary pressures to open the aortic and pulmonic valves.
2. **Ventricular ejection**. It occurs when the mitral and tricuspid valves are closed, and the aortic and pulmonic valves are open. Blood flows out of the ventricles into the aorta and pulmonary artery.
3. **Isovolumic ventricular relaxation**, when all the valves are closed. Pressures in the ventricles decrease until they are so low that the mitral and pulmonic valves open.
4. **Passive ventricular filling**. It occurs when the mitral and tricuspid valves are closed. Blood flows into the ventricles.
5. **Atrial contraction**, when atria contract and pump blood, the cycle then repeats itself.

The first two phases are called systole and the last three phases are called diastole.

III. CARDIOVASCULAR SYSTEM MODELING WITH WINDKESSEL MODEL

The **Windkessel Model** was developed by the German physiologist Otto Frank in the late 1800's. The model describes the flow of the blood through the heart and the

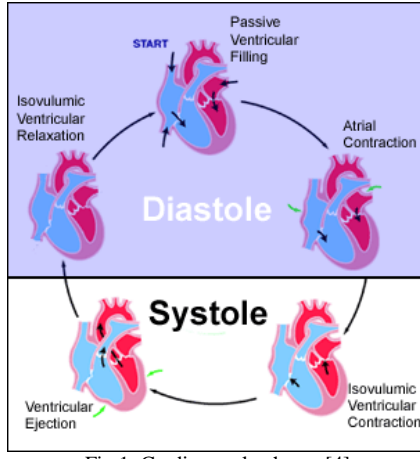


Fig 1: Cardiac cycle phases [4]

arteries as the flow of fluid through the water pump and pipes. It relates the blood pressure and flow in the system, and characterizes the resistance of the blood vessel, the arterial compliance, and the inertia of blood flow [5].

Due to the close correspondence between the cardiovascular system and electrical circuits, it is convenient to model the whole system using lumped parameter method. The analogy between cardiovascular and electrical parameters can be summarized as in the following table:

Cardiovascular	Unit	Electrical	Unit
blood volume (V)	mL	electric charge (Q)	C
flow rate (F)	mL/s	current (I)	A
pressure (P)	mmHg	potential (V)	V

Table 1: Analogy between cardiovascular and electrical parameters [6]

A. Vessel Resistance as Electrical Resistance

Blood vessels' resistance depends on the blood viscosity and the vessel diameter, can be modeled as resistors. The blood vessel resists the blood flow by creating a pressure gradient between its end points as a resistor resists the current flow. Assuming a linear relation between the pressure difference ΔP and the flow F , from Ohm's law, the vessel resistance can be expressed as:

$$R_c = \frac{\Delta P}{F} \quad (1)$$

Poiseuille's law
for fluids

Ohm's law
for electric circuits

Fig 2: Analogy between vessel and electrical resistance

B. Vessel Compliance as Capacitance

Due to elastic deformation, blood vessels can also accumulate and release blood as a capacitor stores and release electric charges due to the potential difference at its ends. From the current-voltage relation of a capacitor, the pressure-flow relation in terms of vessel compliance is:

$$F = C_c \frac{dP}{dt} \quad (2)$$

C. Blood Inertia as Inductance

Since blood is inert, when it flows through a long vessel, its mass resists the tendency to move due to the pressure difference, just as an inductor prevents the current to change instantaneously. The pressure-flow relation of the blood inertia can be expressed as:

$$P = L_c \frac{dF}{dt} \quad (3)$$

D. Heart Valve as Diode

A heart valve allows the blood flow in one direction and blocks the flow in the other direction, opposing a small resistance R_c to the flow whenever the input pressure is higher than the output pressure within a heart chamber or an artery. It is common to use an ideal diode to model the behaviour of a valve as follows:

$$F = \begin{cases} 0 & \text{if } \Delta P < 0 \\ \frac{P}{R_c} & \text{if } \Delta P \geq 0 \end{cases} \quad (4)$$

E. Change in Volume over Time

The Kirchhoff's voltage and current laws are applicable everywhere in the lumped parameter model. It leads to the following additional relation for mass conservation under steady-state flow:

$$\frac{dV}{dt} = F_{in} - F_{out} \quad (5)$$

This is analogous to the conservation of charge.

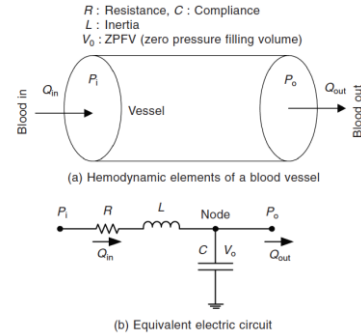


Fig 3: The Windkessel building block of the cardiovascular model [7]

The analogy between cardiovascular and electrical behaviour and their units are summarized as follows:

Cardiovascular	Relation	Unit
vessel resistance	$R_c = \Delta P / F$	mmHg • s / mL
vessel compliance	$C_c \frac{dP}{dt} = F$	mL / mmHg
blood inertia	$L_c \frac{dF}{dt} = P$	mmHg • mL / s ²
heart valve	$F = \begin{cases} 0 & \text{if } \Delta P < 0 \\ \frac{P}{R_c} & \text{if } \Delta P \geq 0 \end{cases}$	mL / s
Electrical	Relation	Unit
resistance	$R_e = \Delta V / I$	Ω
capacitance	$C_e \frac{dV}{dt} = I$	F
inductance	$L_e \frac{dI}{dt} = V$	H
diode	$I = \begin{cases} 0 & \text{if } \Delta V < 0 \\ \frac{V}{R_e} & \text{if } \Delta V \geq 0 \end{cases}$	A

Table 2: Analogy between cardiovascular and electrical behaviour [5]

IV. BASIS OF THE MODEL

The model for simulating the cardiovascular system is based on the mathematical model published by Ursino in 1998[8]. It includes an elastance variable description of the left and right heart, and two closed loops representing the systemic (splanchnic and extrasplanchnic) and pulmonary circulations. Besides the four heart chambers, the entire system is also divided into eight compartments, as shown in the Fig. 5a. These compartments are the pulmonary arteries, veins and peripheral, systemic arteries and peripheral, splanchnic venous and peripheral, and extrasplanchnic venous and peripheral. Each compartment includes a vessel resistance and compliance. For the sake of simplicity, the inertial effects of blood are only included in the systemic arteries, where blood acceleration is significant. The model also includes four diodes for simulating the behaviour of four heart valves.

The compartments are subscripted as follows:

i	in	o	out
l	left	r	right
la	left atrium	ra	right atrium
lv	left ventricle	rv	right ventricle
sa	systemic arteries	pa	pulmonary arteries
pp	pulmonary peripheral	p	pulmonary veins
sp	systemic peripheral	sv	splanchnic venous
ep	extrasplanchnic peripheral	sp	systemic peripheral

Table 3: Subscripts of the variables

All parameter values in the model are based on a typical individual with a 70-kg body weight, which were taken from Ursino's paper [8]. They can also be adjusted to accommodate the physiological specifications for individual patients.

A. State Equations Governing the Ventricular Blood Flow

Other than the nodal equations derived earlier from the RLC analogues, the ventricular volumes also need to be solved to fully describe the system behaviour. The volumes depend on the opening of the atrioventricular valves and are computed by applying conservation of mass:

$$\frac{dV_{lv}}{dt} = F_{i,l} - F_{o,l} \quad (6)$$

$$F_{i,l} = \begin{cases} 0 & \text{if } P_{la} < P_{lv} \\ \frac{P_{la} - P_{lv}}{R_{la}} & \text{if } P_{la} \geq P_{lv} \end{cases}$$

$$F_{o,l} = \begin{cases} 0 & \text{if } P_{max,lv} < P_{sa} \\ \frac{P_{max,lv} - P_{sa}}{R_{lv}} & \text{if } P_{max,lv} \geq P_{sa} \end{cases}$$

$$\frac{dV_{rv}}{dt} = F_{i,r} - F_{o,r} \quad (7)$$

$$F_{i,r} = \begin{cases} 0 & \text{if } P_{ra} < P_{rv} \\ \frac{P_{ra} - P_{rv}}{R_{ra}} & \text{if } P_{ra} \geq P_{rv} \end{cases}$$

$$F_{o,r} = \begin{cases} 0 & \text{if } P_{max,rv} < P_{pa} \\ \frac{P_{max,rv} - P_{pa}}{R_{rv}} & \text{if } P_{max,rv} \geq P_{pa} \end{cases}$$

where $P_{max,rv}$ and $P_{max,lv}$ are the right and left ventricular pressures under isometric conditions and the pressures and resistance in these ventricles are given by

$$R_{lv} = k_{r,lv} P_{max,lv} \quad (8.a)$$

$$P_{lv} = P_{sa} \quad (8.b)$$

$$P_{max,lv}(t) = E_n(t)E_{max,lv}(V_{lv} - V_{u,lv}) + [1 - E_n(t)]P_{0,lv}(\exp(k_{E,lv}V_{lv}) - 1) \quad (8.c)$$

$$R_{rv} = k_{r,rv} P_{max,rv} \quad (9.a)$$

$$P_{rv} = P_{pa} \quad (9.b)$$

$$P_{max,rv}(t) = E_n(t)E_{max,rv}(V_{rv} - V_{u,rv}) + [1 - E_n(t)]P_{0,rv}(\exp(k_{E,rv}V_{rv}) - 1) \quad (9.c)$$

B. Ventricular Elastance Function

The normalized **ventricular elastance function** $E_n(t)$ or **ventricular activation function** ($\phi(t)$ as in Ursino's paper) controls P_{max} 's in equations (8.c) and (9.c), it describes the contractility of a heart. It equals to 1 at maximum contraction and 0 at complete relaxation. Instead of using the squared half-sine wave proposed by Piene [9], a more complex expression is used here, which yielded more accurate results with better shapes by comparing with real physiological data [10]:

$$t_n = \frac{t}{0.2 + 0.1555 T_{cycle}} \quad (10)$$

$$E_n(t_n) = 1.553174 \left[\frac{(t_n/0.7)^{1.9}}{1 + (t_n/0.7)^{1.9}} \right] \left[\frac{1}{1 + (t_n/1.173474)^{21.9}} \right]$$

where t_n is the normalized time, $E_n(t_n)$ is the normalized ventricular elastance function, and T_{cycle} is the duration of one period of cardiac cycle. The heart rate is assumed to be 75 beats per minute, or $T_{cycle} = 0.8$ sec.

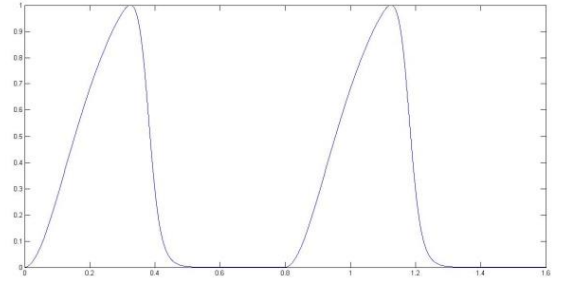


Fig 4: Normalized ventricular elastance function

V. SOLVING THE SYSTEM WITH MATLAB

To obtain the closed form solutions to the nodal equations relating the pressures and volumes of the system is non-trivial, and it often involves complicated exponential expressions. However, we are only interested in the numerical results which can be conveniently obtained by modified nodal analysis and trapezoidal rule.

A. Diode Stamp

There are four heart valves and are represented by ideal diodes D_{lv} , D_{rv} , D_{la} and D_{lv} . Each diode is connected in series with their associated resistor, namely R_{lv} , R_{rv} , R_{la} and R_{ra} . R_{lv} and R_{rv} are time-varying and dependent on $P_{max,rv}$ and $P_{max,lv}$, while R_{la} and R_{lv} are constant and independent of time.

A separate matrix G_d with the same size of nodal matrix G is created to contain the diode stamps. The diode stamp is somewhat similar to the resistor stamp, except the resistance values have to be updated by solving the equations (8.a) to (9.c) at each time interval. Also a check on the sign of the pressure difference over each valve should always be performed to decide whether the diode resistance should be stamped in G_d . If the pressure difference is negative, the diode will simply be treated as an open circuit and the resistance will not be stamped.

The matrix G_d is always reset to 0 at the start of each time interval, and then it will be added with the nodal matrix G after the valve pressure sign checks.

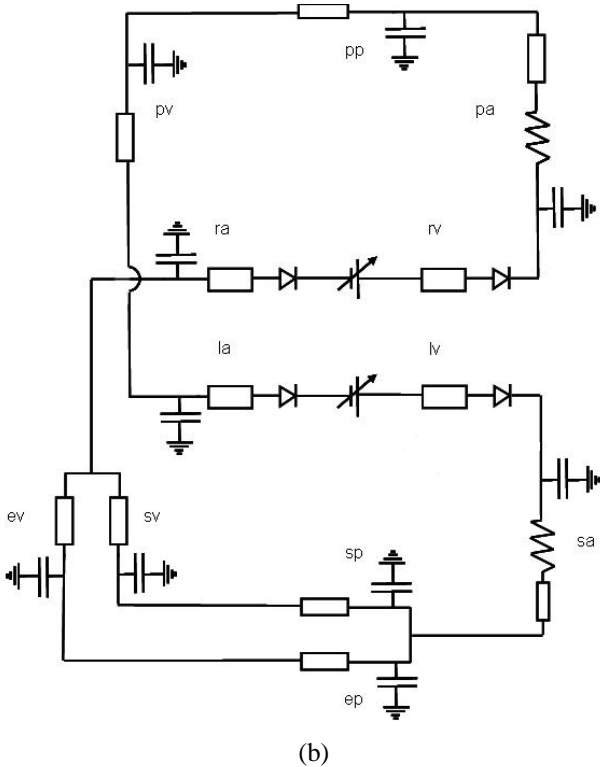
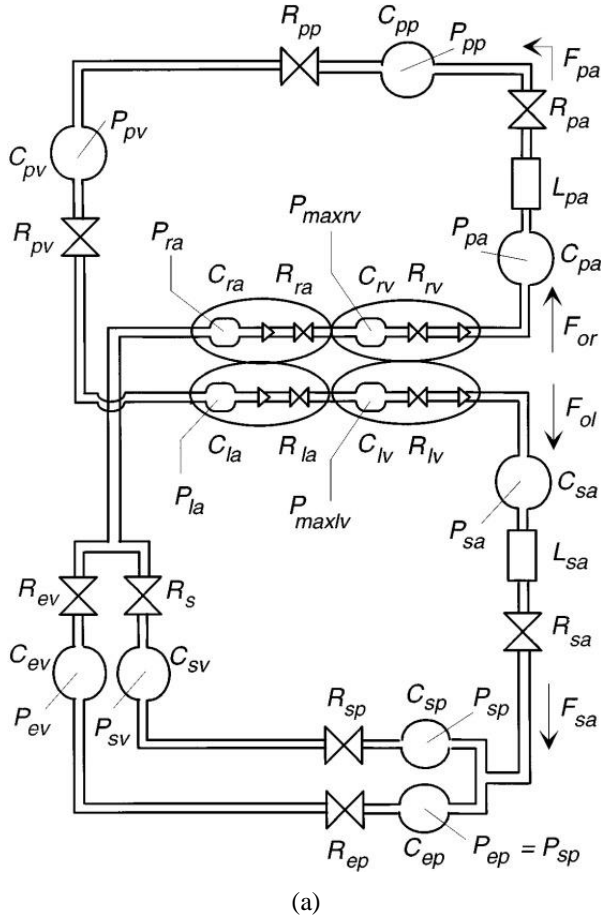


Fig 5: (a) Hydraulic analog of the cardiovascular system. (b) lumped parameter model of the cardiovascular system.

B. Application of the Trapezoidal Rule

There are four pressure sources $P_{\max,lv}$, $P_{\max,rv}$, P_{lv} and P_{rv} can be distinguished in the heart. The pressures are controlled by the baroreflex regulatory system, which is a highly complex function of the sinus nerve. $P_{\max,lv}$ and $P_{\max,rv}$ can be treated as time-varying independent voltage sources defined by equations (8.b) and (9.b), while P_{lv} and P_{rv} can be treated as unity gain voltage controlled voltage sources with the controlling voltages between the nodes P_{sa} and ground, nodes P_{pa} and ground respectively.

The differential equations () relate to ventricular volume and flow rate can be written in the forward Euler form as:

$$V_{lv}(t_{n+1}) = V_{lv}(t_n) + \Delta t (F_{i,l} - F_{o,l}) \quad (11)$$

$$V_{rv}(t_{n+1}) = V_{rv}(t_n) + \Delta t (F_{i,r} - F_{o,r}) \quad (12)$$

By writing the nodal equations in the form of trapezoidal rule, we have the following expression:

$$(M + \frac{2}{\Delta t} C) x_{n+1} = -(M - \frac{2}{\Delta t} C) x_n + b(t_{n+1}) + b(t_n) \quad (13)$$

where $M = G + G_d$, G is the nodal matrix containing the resistor and inductor stamps. And $b(t)$ is a vector containing all the independent voltage sources:

$$b(t) = [0 \ 0 \ \dots \ 0 \ P_{\max,lv}(t) \ P_{\max,rv}(t) \ 0 \ 0 \ 0 \ 0]^T \quad (14)$$

The first 15 zeros correspond to the total number of nodes in the circuit, while the last 4 zeros correspond to the total number of dependent voltage sources and inductors since neither of them modifies the $b(t)$ vector.

VI. SIMULATION RESULTS

Side by side comparisons between the simulated results and the physiological data are conducted for cardiovascular system model validation. The model matches well with the reality in terms of both the amplitude and waveform shape. Notice that some initial conditions must be provided to generate non-trivial results. One choice can be setting $V_{lv} = V_{rv} = 120$ mL and the other parameters equal to zero at $t = 0$ sec, but any values within reasonable range can be used. Some instability will be experienced initially, but the system will reach steady state after a period of time.

Two diagrams particularly useful for clinical diagnosis are the Wiggers diagram and Pressure-Volume Loop for the left heart [11].

A. Wiggers Diagram

The Wiggers diagram is a graphical representation of all of the events associated with beat-to-beat activity of the heart as a function of time. The parameters measured and compared include the aortic, ventricular, atrial pressures, and the ventricular volume.

The pressure and the volume curves representing the left ventricle (blue), left atrium (red), and aorta (green) appear to resemble well with physiologically analogous curves. From Fig. 6a, the peak systolic aortic pressure reaches 120 mmHg and falls to its minimum diastolic value at around 70 mmHg. The maximum and minimum of the aortic pressure are within a healthy range for a person to have. The amplitude of the left ventricular pressure is also within the physiological range.

The left aortic pressure curve is slightly different from a real physiological pressure graph of the left ventricle. The slope of rise of the aortic pressure during ventricular systole is not as steep as it should be, because due to simplicity, the

model assumes the atria to be non-elastic which is not physiologically true.

In Fig. 6b, the simulated left ventricular volume graph shows the left ventricle fills to a maximum about 60 mL of blood before ejecting blood at systole. The total stroke volume of left ventricle, the amount of blood ejected by the heart into systemic circulation, is the total difference in volume of the heart from the end of diastole to the end of systole. In the normal patient case, the stroke volume is 70 mL, so the simulated result is almost within the physiological range.

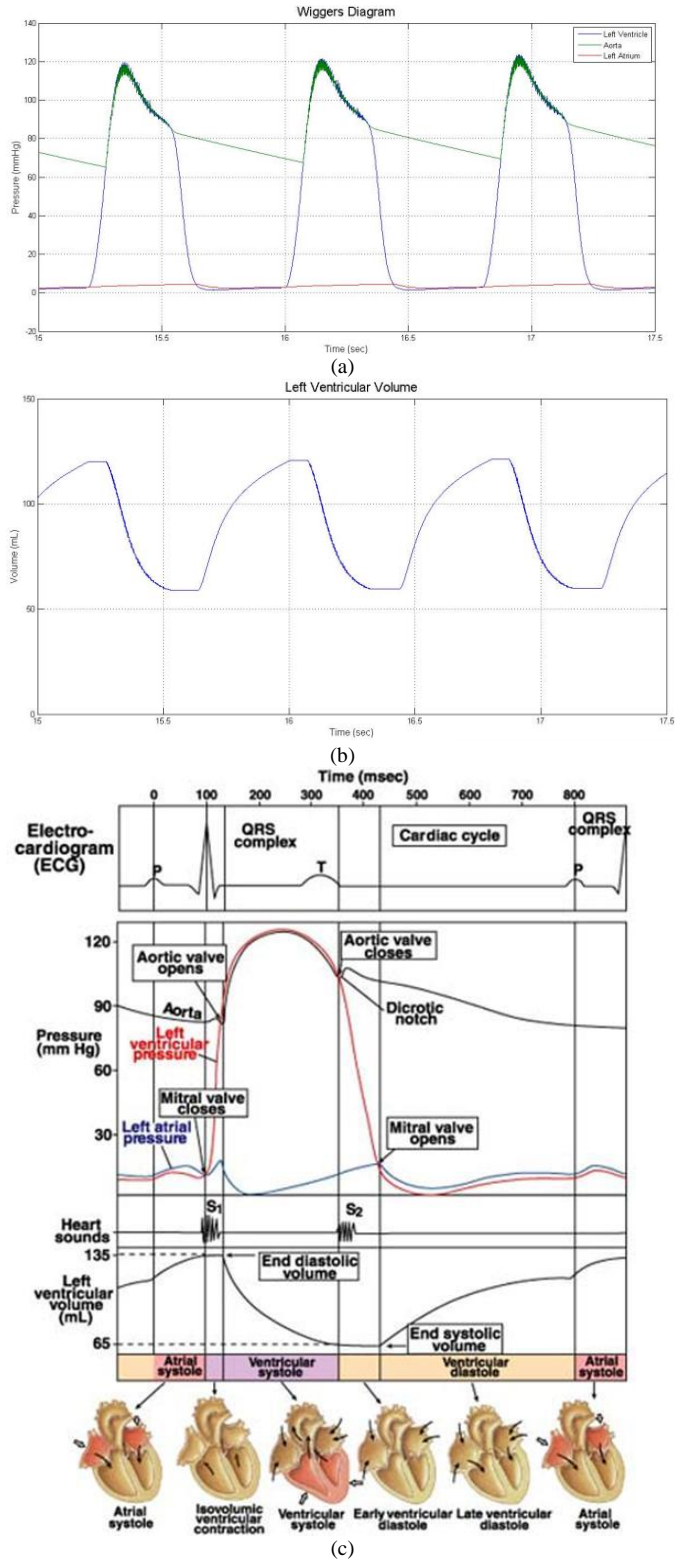


Fig 6: Comparison between (a), (b) simulated pressure and volume curves vs (c) corresponding physiological data for the left heart [12]

B. Aortic Blood Flow Rate

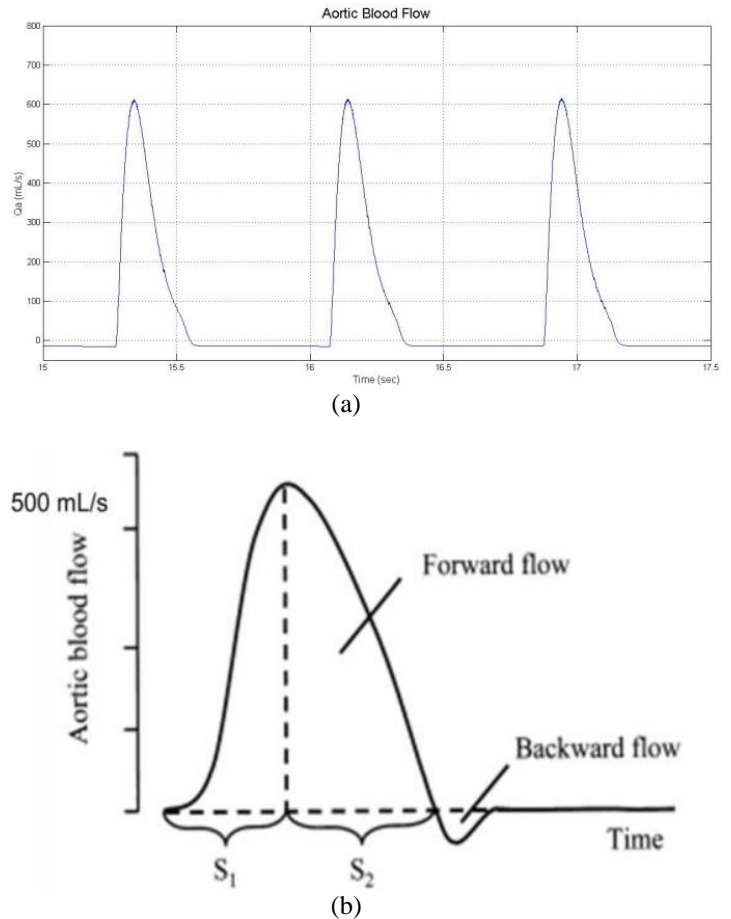


Fig 7: Comparison between (a) simulated aortic flow rate curve vs (b) corresponding physiological data for the left heart [13]

The peak value of the simulated aortic blood flow rate is about 600 mL/s, which is very much in the physiological range. However, it rises and falls a bit too fast compare with the physiological curve because the model assume the aortic valve to be an ideal diode, which opens and closes immediately when the sign of the pressure difference over the valve changes.

C. Pressure-Volume Diagram

The pressure-volume diagram, or PV loop describes the relationship between left ventricular pressure and left ventricular volume during the cardiac cycle. It is widely used in basic research and preclinical testing, to characterize the intact heart's performance under various situations (effect of drugs, disease etc.). Each loop is formed in an anti-clockwise direction.

In Fig.7a, four complete cycles are plotted and superimposed over each other. The end-systolic volume is at 60 mL, the end-diastolic volume is at 120 mL, and the maximum ventricular pressure is about 120 mmHg. These amplitudes are all within the physiological range. Notice that during ventricular filling phase, the ventricle is at rest and there is very little pressure increase.

D. Pressure and Volume Plots for the Right Heart

Although the pressure and volume diagrams for the right heart are not as important as the Wiggers diagram, the shapes of their waveforms are similar to each other.

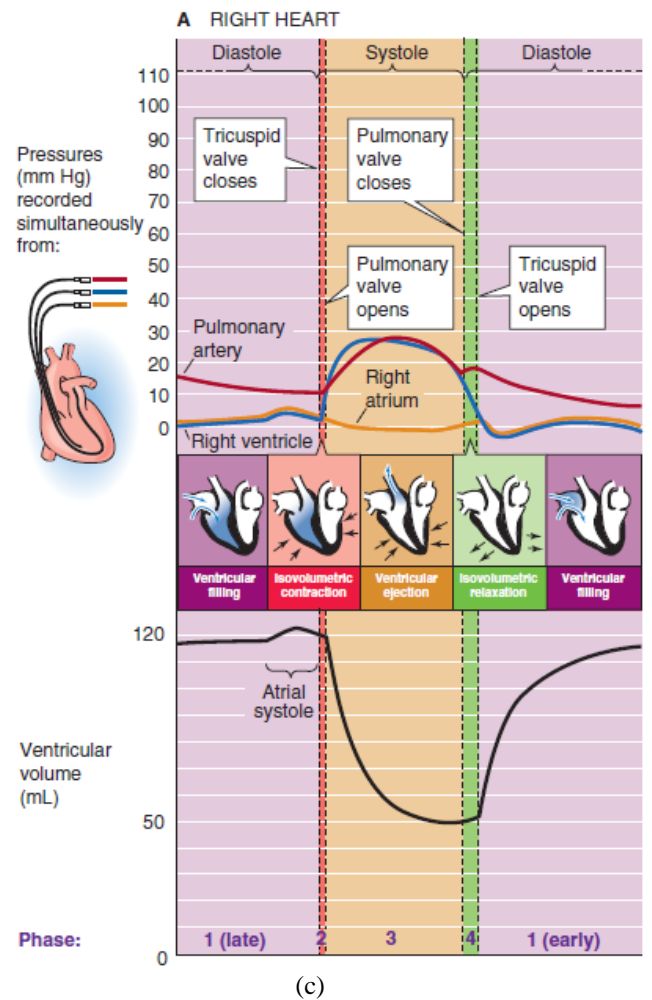
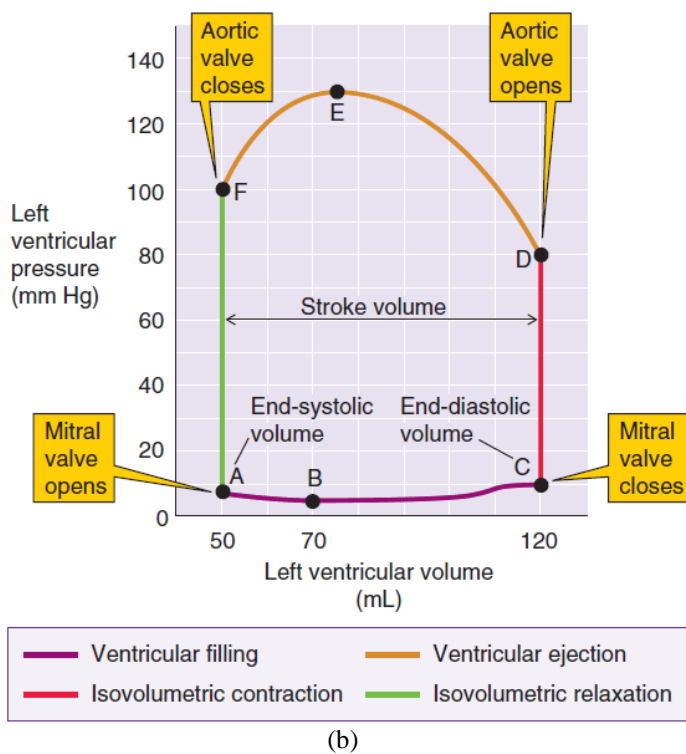
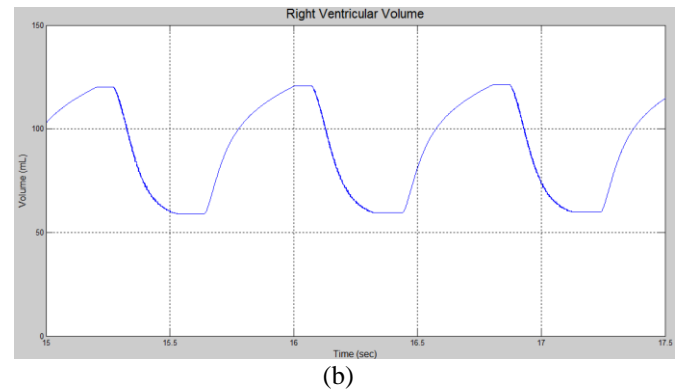
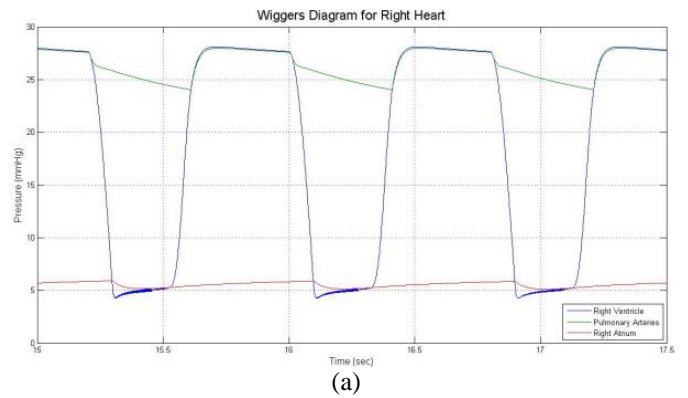
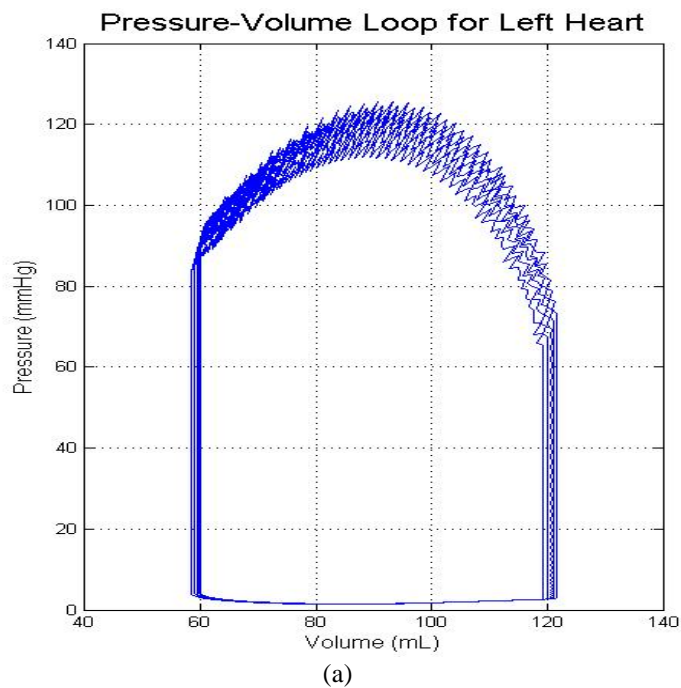


Fig 7: Comparison between (a) simulated pressure-volume loop vs (b) corresponding physiological data for the left heart [11]

From Fig. 8a, the maximum pressures for the right ventricle and pulmonary arteries are at about 28 mmHg. This is much lower than the left ventricular and aortic pressure, because the path of the pulmonary circulation is much shorter than the systemic circulation and less resistance is encountered, thus lower pressure will be sufficient.

Fig 8: Comparison between (a), (b) simulated pressure and volume curves vs (c) corresponding physiological data for the right heart [11]

VII. CONCLUSIONS AND DISCUSSIONS

In this project, a human cardiovascular system has been modeled using lumped elements and solved by trapezoidal rule. Major hemodynamic characteristics including blood pressure, volume and flow rate in the heart chambers and aorta have been compared with the physiological data from various sources to validate the model. Although the model is simple and light-weighted, it has low computational cost, and more importantly, it is able to provide the information on the critical hemodynamic parameters with good accuracy. All data compared are within reasonable physiological range.

However, it is also important to point out some limitations with the model, which can be improved and even re-designed in the future. Firstly, the model only assumes elastic ventricles, but atria to be inelastic due to simplification. This could affect the accuracy of the atrial pressure and flow rate simulations. An atrial elastance function can be adopted in the future. Secondly, the four heart valves can be modeled with more real diodes instead of the ideal diodes. It is physiologically impossible for the heart valves to open and close instantaneously, it takes some time for a heart valve to open from partially to completely. This will require a new stamping scheme for the diodes. Thirdly, more segments such as head and limbs can be included in the model to achieve higher degree of accuracy and capture more details of hemodynamics. For example, blood pressure and flow can be measured at any arbitrary distance from the heart instead of restricting the measurement at a few fixed locations. This will also require the per unit distance data of vessel resistance, compliance and blood inertia for different parts of the body.

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