

Simulation of a Human Circulatory System

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Abstract. This article describes a mathematically based human circulatory model. The model consists of lumped elements made of venous, arterial, peripheral, pulmonary vein and artery segments. A heart model is simulated using 4 chambers (left and right atriums and ventricles). The heart pump mechanism is operated by a simple piston based models for each of the chambers. The simulation consists of 19 (states) first order differential equations, and simulated with Matlab and Simulink. The simulation computes volume, flow rate and pressures in each segment.

Keywords. Simulation, Bond Graph, Human Circulatory system, Heart

Introduction

The human circulatory system is a complex process that provides body temperature control, oxygen and nutrient transport to various parts of the body and organs. The circulatory system is made of several components which rely on feedback mechanisms to control the volume, flow of blood and pressure during varying operating loads such as drug administration, physical and mental changes of a patient. A mathematical circulatory model would provide insight into circulatory behavior during varying operating loads, help design implantable biomechanical structures, medicine effects, control artificial hearts and other circulatory assist devices. The use of a circulatory model in virtual simulation would be invaluable to provide a level of realism in simulated surgical procedures. In this paper, we describe a simple circulatory model which provides comparable results with physiological values for a nominal human.

The use of bond graph modeling techniques provides a method of taking a simple block model representation and converting it into a set of first order differential equations that can be rapidly prototyped into a simulation. The use of bond graph methods provides deep insights into the dynamic mechanism of the system during the derivation process and the interaction between the various model elements and states.

1. Model

An initial simulation block diagram is converted into a bond graph diagram [1] using resistive, inertance and compliance elements as shown in Figure 1. The simulation model consists of lumped segments of an aorta, arterial, peripheral, venous and vena

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cava (green). The pulmonary system consists of lumped segments the pulmonary vein, pulmonary artery and a lung model (blue). A heart model consists of 4 chambers similar to the human heart left and right atria and ventricular chambers (red). The flow, volume and pressures in the heart are controlled by four passive valves which represent the aortic, pulmonary, mitral, tricuspid valves (yellow). Additional two valves are used to provide increased control over the model during off nominal performance. The simulation computes pressure and flow rate of blood in each of the segments.

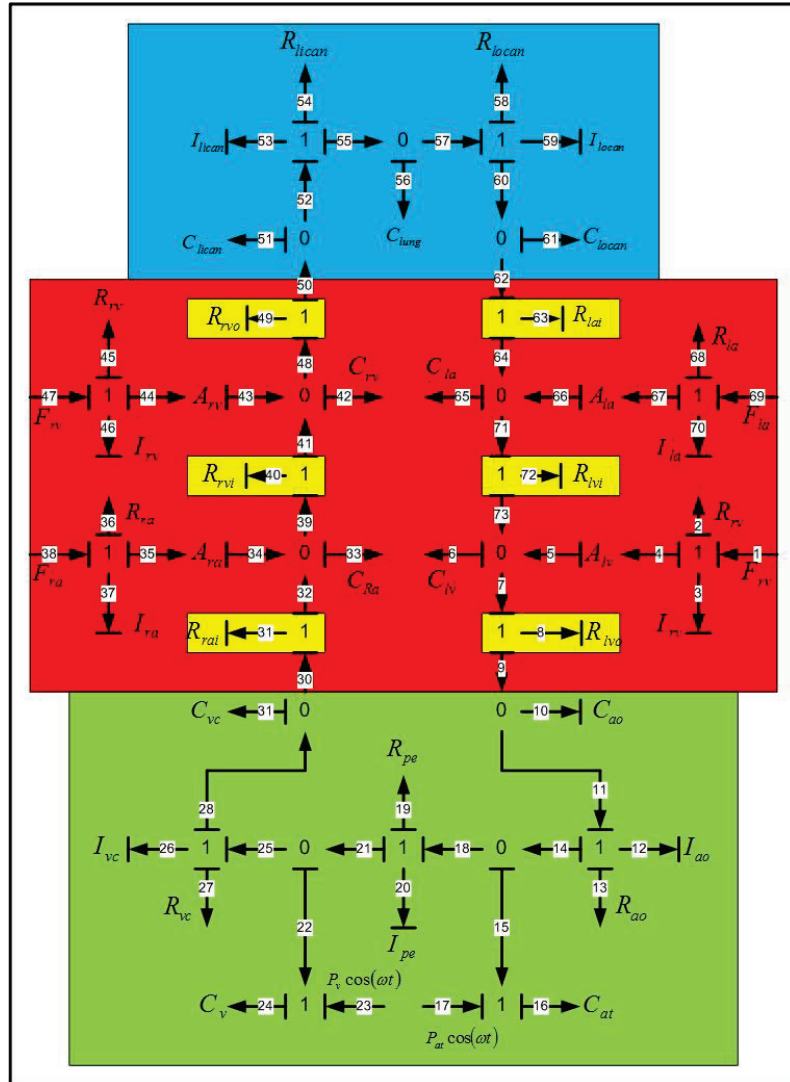


Figure 1. Simulation Bond Graph

The bond graph identified 19 energy variables (state variables) and generates 19 first order differential equations. The circulatory model is a closed loop model. The

heart pump model provides the forcing function to generate the flow and pressures to move the blood within the system.

1.1. Heart Pump Model

The circulatory model has four inputs or driving functions. The inputs are the forces exerted by the contraction of the heart muscles during diastolic and systolic regimes of the heart beat cycle. These are approximated by a piston model with inertia and resistance elements modeling mass and friction respectively.

The four chambers (atria and ventricular) have separate force generating piston models, hence each chamber behavior is uncoupled with each other. The forcing function currently generated by the motion of the piston is a square waveform making up the systolic and diastolic phases of the beat cycle as shown in Figure 2. The synchronization and timing of each piston is created by setting the diastolic and systolic fraction of the beat cycle. During systole the ventricles contract and during diastole the atrium contracts. During contraction the piston moves forward and the mechanical energy of the piston is transformed into hydraulic pressure in their respective heart chambers as defined by a transformer bond simulated by elements 4-5, 35-34, 44-43 and 67-66 in Figure 1 for the four heart chambers. The relationship between the energy transfer is given by the surface area of the piston.

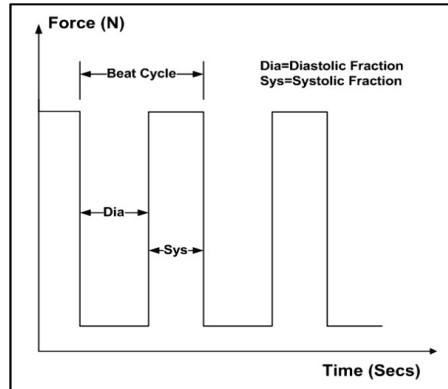


Figure 2. Heart Pump Waveform

The forcing function is simulated through a maximum (F_{max}) and minimum (F_{min}) force through an applied gain (K_{gain}) as shown in Eq. (1):

$$F_{piston} = K_{gain} * F_{max} + F_{min} \quad (1)$$

The $K_{gain} = K_{dia}$ during diastole phase which is calculated by Eq. (2):

$$K_{dia} = rem\left(t, \frac{60}{b}\right) < f_{dia} \quad (2)$$

The $K_{gain} = K_{sys}$ during systole phase is calculated by Eq. (3):

$$K_{sys} = \left[rem\left(t, \frac{60}{b}\right) > f_{dia} \right] \& \left[rem\left(t, \frac{60}{b}\right) < (f_{dia} + f_{sys}) \right] \quad (3)$$

Where f_{dia} and f_{sys} are the diastolic and systolic fractions of the beat, t is time in seconds, b is the number of heart beats per minute and $rem(x,y)$ is the fractional portion.

1.2. Pressure-Volume Relationship

The lumped circulatory elements are modeled individually by a linear Pressure-Volume relationship. The input to the relationship is the volume of blood in the segment, its compliance and the pressure offset and the output is the pressure simulated in the segment. Eq. (4) shows linear pressure and volume relationship.

$$Pressure = \frac{Blood\ Volume}{Segment\ Compliance} + Pressure\ Offset. \quad (4)$$

1.3. Valves

The circulatory model contains 4 functional one way valves. These valves are the tricuspid, pulmonary, mitral and aortic valves, each of which are modeled as resistive elements given by the bonds 40, 49, 72 and 8 respectively as in Figure 1. The closure and opening of the valves are controlled passively by the pressure gradient over the valve given by Eq. (5)

$$R_i = \begin{cases} R_i & \text{for } dP > 0 \\ R_i * 10^8 & \text{for } dP < 0 \end{cases} \quad (5)$$

The valves are modeled closed when the pressure difference across the valve is negative. During a negative gradient across the valve the resistance of the valve is increased by a factor of 10 to the power of 8 to restrict flow, hence simulating a closed valve. The increased resistance does not however completely close the valve to flow. A small amount of flow across the valve however still exists. This flow magnitude is extremely small as compared to the nominal flow of blood when the valve is open and therefore negligible.

There are two additional valves simulated, namely the R_{rai} and R_{lai} representing the right atrium inlet and the left atrium inlet identified by bonds 31 and 63 respectively in Figure 1. The purpose of these valves are to simulate resistance in the vena cava and pulmonary vein. For nominal cases these resistance are set to very low values thus not interfering with the flow or pressures.

2. Results

The simulation was run with Matlab and Simulink. Due to the stiffness of the system and the 4 valves the simulation runs in non real-time. The simulation is solved using Adams method (ode113) for 60 - 300 seconds with variable-step and relative tolerance set to 1E-03. The simulation runs on a Core i7 CPU Q720, 1.60Hz with 6GB of ram laptop computer. The current simulation run time on average is 0.0034 Simulation seconds per Real-time seconds.

2.1. Model Parameterization and Simulation Results

The model parameters are tuned to provide nominal physiological results at steady state. The tuning process is through setting initial volumes and flows in each segment and guess values for compliance, inertance and resistance. Initial parameter values are calculated roughly based on physiological segments average length, diameter and thickness [3, 4]. The parameters are then tweaked based on previous simulation results until satisfactory nominal physiological conditions at steady state are obtained.

The tuning process is extensive and iterative. Also due to the slow simulation performance in Matlab the results for runs can take several hours to attain steady state conditions to provide adequate convergence.

The heart pump parameters are tuned similarly to provide expected systolic and diastolic pressures and volumes for nominal physiology. Figures 3 - 6 shows the pressures of various elements with the model during nominal simulation.

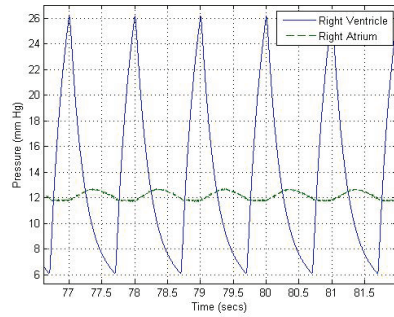


Figure 3: Right heart pressures

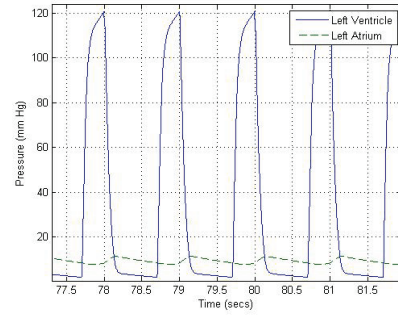


Figure 4: Left heart pressure

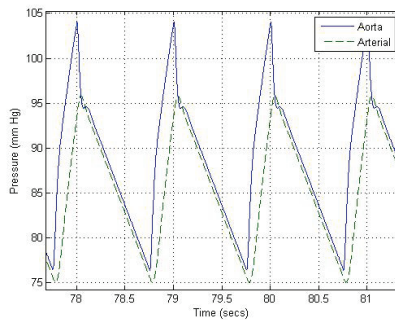


Figure 5: Arterial pressures

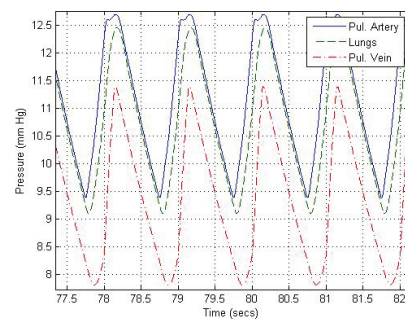


Figure 6: Pulmonary pressures

The simulation output states variables are used to calculate the physiological parameters of pressure, flow and volume in all the various segments. The Table 1 shows the various segments maximum and minimum pressures which are compared with values of a human cardiovascular system [3].

Table 1. Pressures in various segments of simulation and published ranges.

| Segment | Min Pressure (mm Hg) | Max Pressure (mm Hg) | Published Range [3] (mm Hg) |
|------------------|----------------------|----------------------|-----------------------------|
| Left Atrium | 8 | 11.5 | 0-10 |
| Left ventricle | 2 | 120 | 0-120 |
| Aorta | 76.5 | 104 | 80-120 |
| Arterial | 75 | 96 | 80-100 |
| Venous | 12.25 | 12.25 | 0-5 |
| Vena Cava | 11.75 | 12.75 | 0-5 |
| Right Atrium | 11.8 | 12.7 | 0-18 |
| Right Ventricle | 6 | 26 | 0-25 |
| Pulmonary Artery | 9.4 | 12.7 | 10-22 |
| Lungs | 9.0 | 12.5 | 10-22 |
| Pulmonary Vein | 7.8 | 11.4 | 0-10 |

The total blood volume in the system is 4.5 Liters. The average volume of blood distribution through the system segments at steady state nominal conditions are shown in Table 2. These volumes are compared with published results [3]. The average computed flow rate of the blood in the system is 4.4 L/min.

Table 2. Blood volume distribution

| Segment | Volume (cm ³) | Percentage (%) | Percentage Grouped (%) | Ref[3] (%) |
|------------------|---------------------------|----------------|------------------------|------------|
| Left Atrium | 30 | 0.7 | 9.8 | 12 |
| Left ventricle | 100 | 2.2 | | |
| Right Atrium | 5 | 0.1 | | |
| Right Ventricle | 108 | 2.4 | | |
| Aorta | 200 | 4.4 | 80.2 | 74 |
| Arterial | 75 | 1.7 | | |
| Venous | 2960 | 66.2 | | |
| Vena Cava | 550 | 12.3 | | |
| Pulmonary Artery | 120 | 2.8 | 10 | 14 |
| Lungs | 200 | 4.4 | | |
| Pulmonary Vein | 120 | 2.8 | | |
| Total | 4468 | 100 | 100 | 100 |

2.2. Sensitivity

In order to gain an understanding of the dynamics and influence of the various model parameters a sensitivity analysis is conducted. The information attained from the analysis is used to provide control of the model parameters at run-time to simulate various effects.

When increasing the compliance of a segment the simulation causes the blood volume in the segment to increase during steady state. Similarly increasing resistance causes an increase of pressure in the segment and also increases the velocity of the blood flow through the segment. subsequently an increase in Inertance of a segment causes the flow of blood in the segment to decrease.

Increases in valve resistance prevents flow of blood through the segment when the resistance is very large. However low resistance allows blood to flow through the segment which can be used to simulate prolapsed valve behavior.

The increase in the maximum force of the forcing function increase the ejection pressures of the heart chambers. Subsequently reducing the minimum forces on the piston reduces the internal pressure in the chamber. These changes can be used to regulate the passive valves of the heart to flow blood between the chambers.

The pressure-volume relationship in the segments can be changed to provide non-linear functions. These non-linear functions can provide better pressure-volume relationships. However the computation time are increased due to the nonlinearity and dependences with other states variables in the system equations [5].

3. Future Work

Current work is to create a real-time C++ version of the simulation for use in a surgical simulation to provide physiological feedback from surgical trauma, medication, loss of blood and administration of anesthesia. Initial implementations are showing real-time results are achievable and the current slow speeds attained from Matlab are possibly due to the licensing checks and that Matlab is an interpreted language.

An addition of the lung model provides a method of modeling O₂ and CO₂ exchange during respiration. A respiration model provides breathing with a simple gas exchange model can provide O₂ and CO₂ results in the blood.

Introduction of drug delivery and medicine during simulated surgery can provide physiological changes though changes to various model parameters.

4. Acknowledgements

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