Cardiac Output Control in Artificial Heart

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***Abstract*— Artificial hearts are increasingly needed with the prevalence of heart failures around the world. By applying our biosystems control knowledge, we developed an artificial heart model that can be used to regulate cardiac output when paired with a mechanical artificial heart. In building our cardiac model, we based our ODEs and relationships on electrical circuit dynamics. The final form of our model is based on a 3 module windkessel model. We were then able to provide time and s domain transformations of our model to create successful block diagrams mapped and analyzed through Simulink. Using inputs of blood pressure differences and output of volume blood flow, we were able to achieve a regulated system where the flow was regulated back to the target cardiac output of 5 ml/min. Error output was also regulated to zero. The system response to pressure difference mimicked in situ patterns. Through Bode Plot analysis, we determined that our system was strictly stable. As such, we have developed a suitable cardiac output control system that can be applied to save patient lives and to increase quality of life via artificial heart implantation.**

I. Background Physiology

The purpose of an artificial heart is to restore blood circulation for patients with diseased or damaged ventricular tissues. The artificial heart replaces the ventricles with hydraulic pumps and is controlled by a portable machine connected outside of the body that can be held in a small backpack or bedside table [10]. In a total artificial heart, or TAH, there are 4 valves, just like in a healthy heart, that connects the ventricles to the atria on both the left and right sides. Another type of artificial heart that this report is focused on is a left ventricle assist which moves blood from the left ventricle into the aorta to take over the heart's workload [4]. When modelling an artificial heart there are four phases to take into consideration: isovolumetric relaxation (diastole), filling phase, isovolumetric contraction (systole), and ejection phase. The mechanics of the heart that the artificial heart has to also preserve is the homeostasis of blood pressure. The heart adapts when homeostasis is disturbed, when the blood pressure rises above or falls below the normal range, which also changes the heart rate. If blood pressures rise above the target range then the baroreceptors are stimulated which leads to the cardioacceleratory center to be inhibited, the cardioinhibitory center stimulated, and the vasomotor inhibited as well. These effects lead to vasodilation in the blood vessels and a decrease in cardiac output. When the blood pressure falls below the target range the opposite occurs; the baroreceptors are inhibited, which leads to stimulation of the vasomotor center, inhibited cardioinhibitory center, and stimulated cardioacceleratory center [5]. These effects lead to vasoconstriction and an increase in cardiac output to maintain homeostasis. An artificial heart needs to have a control mechanism that accounts for both of the stated changes in order to produce a consistent cardiac output of 5 L/min which is the average in a healthy adult. The difficulties with controlling an artificial heart come from the inability to measure many parameters in a healthy human. Typically only pressure and volume in the heart chamber can be measured [4].

A. Model Basis

For the purposes of our biosystem control, we needed a suitable model system to develop our artificial heart's ODEs and subsequent block diagram through. We used the WindKessel Model. This model has been widely used in biosystem mapping as it is analogous to an electrical circuit. As such, the same Ohm's Laws and Kirchoff's Laws that drive electrical circuit functionality, can also be applied to the Windkessel Model, wherein blood pressure is analogous to voltage, blood flow to current, heart/vessel constrictions to resistance, and atrial/vessel compliance to capacitance. This electrical correspondence for cardiac modelling also has precedence in situ since heart pumping is based on electrical conduction systems. [1]

Blood flowing from the arteries into the aortic valve encounters resistance. Assuming a cylindrical vessel model, with the pressure difference between its two ends with linear dependence, an Ohm's Law equivalent can be applied here. Rp, or the resulting resistance, is equal to pressure divided by the blood volume flow (*See Table 1).* Additionally, Cardiac muscles change the volume and pressure in the vessel. The blood flow into such an elastic chamber/vessel corresponds to the rate of change of blood volume, which is related to a change of pressure P inside the chamber. With the assumption of a linear relationship, we can apply an analogous capacitance equation (*See Table 1).*

$P = F \cdot R_p$	*Cardiac Resistance*	 *Resistance*	$V = I \cdot R_t$ *(Ohm's Law)*
$C_c \cdot \frac{dP}{dt} = F$	*Cardiac Compliance*	*El*  *ıce*	$C_c \cdot \frac{dV}{dt} = I$
$F_1 = F_2 + F_3$	*Blood Flow*	*Current Flow*	$I_1 = I_2 + I_3$ *(Kirchhoff's Law)*

Table 1. Electrical Circuit Equations Analogous to Cardiac Dynamics

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With the defined relationships, we can apply Kirchoff's laws to form the entire cardiac circuit further. To model the conservation of blood mass, the sum of currents, or blood flows, entering any junction is equal to the sum of currents leaving that junction. The sum of all the voltages, or blood pressures, around a loop is equal to zero. *(See Table 1)*

These relationships and equations culminate into the ODEs discussed in a later section that define the Windkessel model that relates the cardiac and hemodynamics. For our purposes, we chose the 3-Module Windkessel Model which was formulated by the Swiss physiologist Ph. Broemser together with O. Franke and it was published in an article in 14 Proceedings of the 58th European Study Group Mathematics with Industry 1930 [2]. This model builds on the above ODEs and introduces an extra resistor Ra, which represents the resistance encountered by blood as it enters the aortic valve. The corresponding electrical circuit is shown in figure 1.

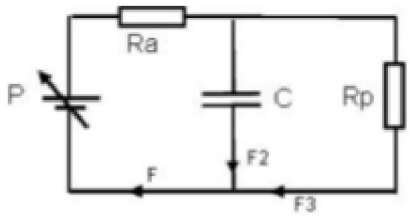

Figure 1: Windkessel Model

For the input of the system, we used a pressure difference between ventricular and atrial pressures. This changes throughout a single heartbeat as displayed in the figure below. This difference is recorded by the fluid filled catheter measuring components of the mechanical heart.

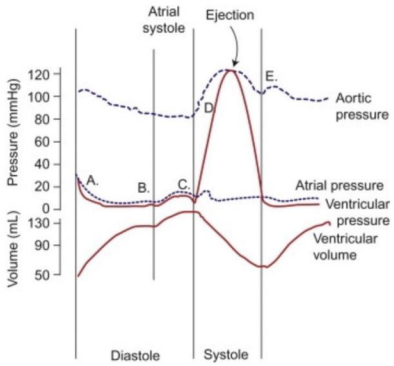

Figure 2: Cardiac Cycle

II. Limited Aspects of Control System

A. Assumptions and Limitations

The boundary for where the system begins is the central circulatory system, which impacts the peripheral circulatory system and the body through the transportation of oxygen and nutrients. Beyond this is the imaginary universe. We were specifically concerned with the heart, but our artificial heart does take in blood from nearby and expels it, thus it impacts the entire circulatory system and responds to any changes in the circulatory system as well. For our control system, we assume the vessels are modeled in cylindrical shape. For the flow, we assume they are uninterrupted, and undergo perfect laminar flow. This obviously is a drawback as blood does not typically undergo such perfect conditions in our bodies, nor are our vessels perfectly cylindrical shapes. Moreover, due to this laminar flow, we assume linear conditions for resistance, but non linear conditions are present in situ. Additionally, for our equations and models to work, we assume the non zero pressure difference is great enough to open our aortic valve for filling our artificial heart. For compliance purposes, we assume a linear elastic model of the cardiac tissues. Though the heart exhibits linear relationships at time in situ, this is not always the case and thus could be a limiting factor.

Additionally, we simplify the pressure difference to be a single pulse per heartbeat as seen in figure 2 between points D and E. We also assume no initial conditions for the flow and pressure as seen later in the Laplace transform equations.

A. System Boundaries

As stated before artificial hearts are controlled by machinery outside of the body. The connection between the control system of the machinery and the control system that we have developed for an artificial heart creates a boundary between the system and the universe.

III. Performance Goals and Constraints

The overall goal of the artificial heart is to respond to changes in the circulatory system to maintain a stable cardiac output or volumetric blood flow. Therefore, our artificial heart needs to be able to regulate cardiac output like a natural heart by pumping a healthy amount of blood per minute. The normal cardiac output of a healthy adult is 5-6 Liters per minute [2]. This translates to a target value of 91.67 mL/s for our control system.

We are constrained by the limitations of simulink and the integrated complexity of the circulatory system in creating a model that is able to adapt and regulate all aspects of the circulatory system. Therefore, we had to simplify inputs to be pressure and the output to be flow when in a natural heart, many control systems are in place that regulate heart rate, flow, and pressure. For simplification, we have focused on the regulation of cardiac output, or volumetric blood flow.

IV. Math

The primary equations utilized to develop the artificial heart model are:

Pressure Difference (Input):

$$P_{diff} = P_v - P_A \text{ (ie Ventricular - Atrial Pressure)}$$

Windkessel Model:

$$(1 + \frac{R_a}{R_p}F) + R_a C_c \frac{dF}{dt} = \frac{P}{R_p} + C_c \frac{dP}{dt}$$

$R_a =$ *aortic valve resistance* $= 0.001 \frac{N \cdot sec}{cm^5}$

$R_p =$ *resistance* $= 0.016 \frac{N \cdot sec}{cm^5}$

$C_c =$ *compliance* $= 967.58 \frac{cm^5}{N}$

Final System of equations:

The first equation states that the derivative of volume is volumetric blood flow or cardiac output. The second equation is derived from the windkessel model.

System of Equations:

$$\frac{dV}{dt} = F(t)$$

$$\frac{dF}{dt} = \frac{1}{R_a R_c}[(\frac{P(t)}{R_p} + C_c \frac{dP(t)}{dt}) - (1 + \frac{R_a}{R_p})F(t)]$$

V. Transfer Functions

Transfer functions were developed from the combination of the ODE's with flow, F(s) being the output and pressure difference P(s) being the input. The control system used R(s) was a PI controller since controlled artificial heart outputs like pressure difference are contaminated by noise. Additionally, derivative control is useful for high frequency response but the heart pumps at a comparatively slow frequency so derivative control is not needed [6]. See A1 for derivation of H(s).

Transfer Functions:

$$H(s) = \frac{F(s)}{P(s)} = \frac{1 + C_c R_p s}{R_p(1+sR_a) + R_a}$$

$$R(s) = P + I\frac{1}{s} + D\frac{N}{1+N\frac{1}{s}}$$

P = 0.0000001

I = 0.0000001

D = 0

VI. Block Diagram

The simplest block diagram model is shown in figure 3 using a biosystem with a transfer function H(s) based on the windkessel model and a control system R(s) that provides proportional and integral control.

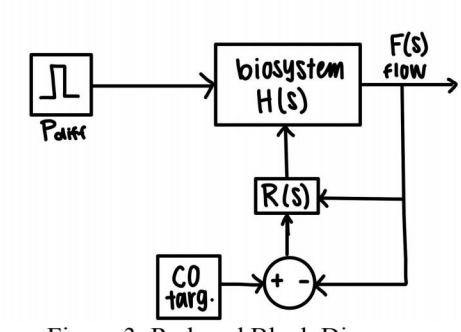

Figure 3: Reduced Block Diagram

VII. Simulink Model

The input signal of pressure difference is the difference between atria pressure and ventricular pressure. The artificial heart measures atrial and ventricular (artificial) pressure using a fluid filled catheter and outputs a desired flow or cardiac output. This model converts the pressure difference input into flow by using the Windkessel model. The flow is then compared to the target flow or the ideal cardiac output, and the error is combined with the flow output and is sent through the controller and Windkessel model to produce a new, corrected, flow output. The volume was found by taking the integral of the flow. All values were in units of N, cm, s. The pressure input was a pulse of 122 mmHg for 0.6s to simulate a single heartbeat. The frequency of pulsation was related to heart rate. This was simulated to be 1 beat per second since a normal heart rate is 60-100 bpm.

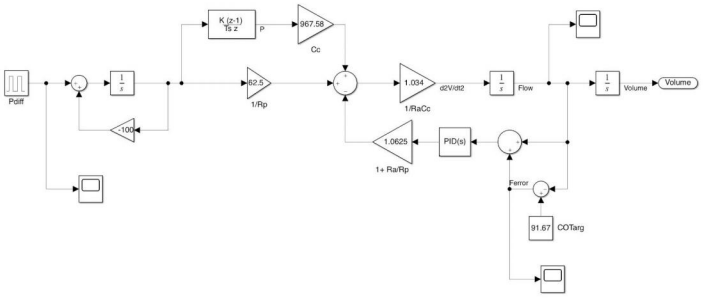

Figure 4: S Domain Block Diagram

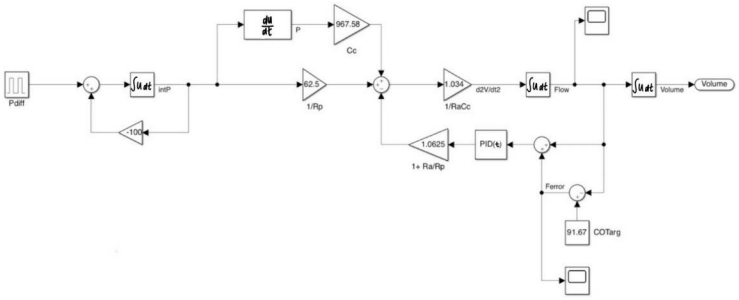

Figure 5: Time Domain Block Diagram

VIII. Simulation Results

The results of the simulation show that the volume flow rate was able to be regulated by this artificial heart model, to produce a constant cardiac output of 91.67 mL/s which is the same as the target cardiac output. This is confirmed by examining the error output, which is regulated to zero. Additionally, the speed at which the system is able to respond to the pressure difference is also successful and mimics the same pattern as the natural heart.

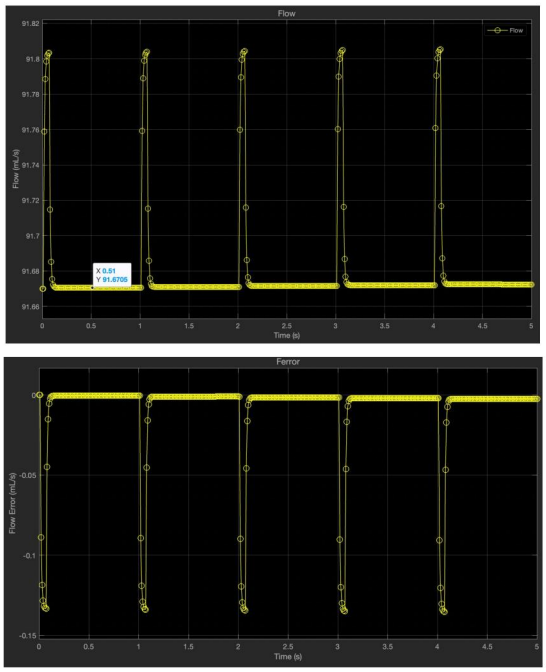

Figure 6: Flow and Error Results

IX. Sensitivity Analysis

From the transfer function, stability of the control system can be calculated. The system had one zero and one pole, -0.0646 and -1062.5 $\frac{cm^5}{N \cdot s}$ respectively. The negative pole value shows that the system is strictly stable. This is also evident in the bode plot. The magnitude of the system is controlled between 80 and 0 dB and the phase angle does not reach a phase shift less than 180 degrees.

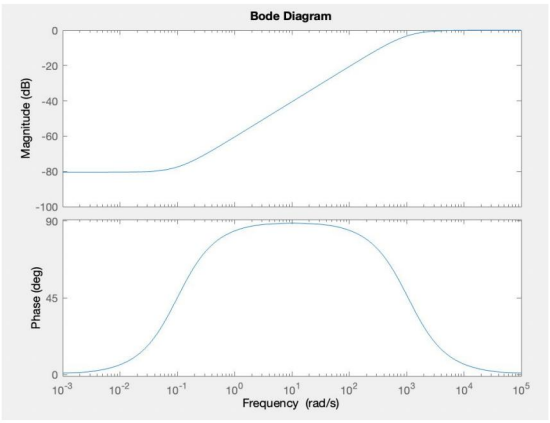

Figure 7: Bode Plot

X. Errors Present in Simulation

Errors present in our simulation could be due to the simplification of the model. For example, our input of the control system is the pressure difference between the ventricle and atria. This input is able to affect flow of blood through the heart by instigating the amount of force the pump is pumping into the working components of the heart and thus circulating around the body. In this model, we simplify the input pressure difference into a pulse related to the standard pressure difference during the cardiac cycle, during heart ejection. This value was estimated to be constant at 120 mmHg as seen in the image below. This pressure lasts approximately 0.06s for the estimated time of ejection (half of the QRS complex time). In reality, this pressure difference can vary between heart beats and heart rate is not constant. This pressure would also change depending on the blood flow but our model simplifies these two variables by neglecting their influence and assuming that pressure is independent of flow.

XI. Application to Clinical Syndrome

A clinical syndrome that corresponds to the modified version of our system is tachycardia. Our model is controlling the flow by inputting the pressure difference in the ventricles and atria. This difference is run through the system by pulses which are in relation to the tempo of a heartbeat. Because of this relationship we increase the pulse rate when we can symbolize tachycardia through our control system of an artificial heart. To model tachycardia we increased the frequency of the pressure difference to 2 pulses per second which corresponds to a fast heart rate of 120 bpm. The results of this simulation again show the control system is successful at responding to the pressure difference and outputting a consistent cardiac output (flow) of 91.67 mL/s.

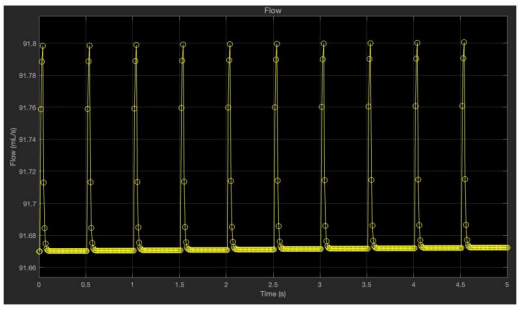

Figure 8: Tachycardia Flow Results

XII. Discussion

The disadvantages of this simulation is that it relies on assumptions to simplify the cardiac system and heart function. Some limitations come from the Windkessel model ODE that we are basing our control system off of. Our model does not fully encompass the complexity of the heart therefore, there is some room for error. Another limitation is that the model does not account for the inertia of blood nor the elasticity of the vessels as blood is being pumped through them [4].

APPENDIX

$$\left(1+\frac{R_a}{R_p}\right)F + R_a C_c \frac{dF}{dt} = \frac{P}{R_p} + C_c \frac{dP}{dt}$$

laplace transform

$$\left(1+\frac{R_a}{R_p}\right)F(s) + s R_a C_c F(s) - \cancel{R_a C_c F_0} = \frac{P(s)}{R_p} + C_c P(s) s + \cancel{C_c P_0}$$

$$\left(1+\frac{R_a}{R_p}\right)F(s) + s R_a C_c F(s) = \frac{P(s)}{R_p} + C_c P(s) s$$

$$F(s)\left(\left(1+\frac{R_a}{R_p}\right) + s R_a C_c\right) = P(s)\left(\frac{1}{R_p} + C_c s\right)$$

$$H(s) = \frac{F(s)}{P(s)} = \boxed{\frac{1 + C_c R_p s}{R_p(1 + s R_a C_c) + R_a}}$$

A1: Work for Deriving Transfer Function

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