Keeping the BEAT 🫀❤️!

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Human Heart: Pacemaker

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Abstract

The system we are choosing to study is the human heart and a pacemaker. We decided to study this topic because we wanted the challenge of studying a system we are unfamiliar with. The human heart is interesting to us because there are many variables to be studied, and several different interventions that are possible. A pacemaker seems like a good device to add to the system because we can easily see how it controls the rhythm of the heart. This will be a good control feedback system for us to model. We are planning to investigate a condition that can be treated with a pacemaker, create a model of the heart-pacemaker system and produce an animation showing how the beat is corrected once treatment begins. The main focus of our model will be the heartbeat rhythm and impulse given by the pacemaker.

Research

The human heart operates using a complex sequence of electrical impulses that form from the sinoatrial node (SA). The purpose of this node is to act as the body's natural pacemaker and hold the rhythm for the entire cardiac cycle by forming impulses that travel through the system. The impulses travel across the atria, atrioventricular (AV) node, and ventricles. The impulses then trigger the contraction that's purpose is to pump blood throughout the body. An average adult experiences between 60 to 100 beats per minute (BPM) which is sufficient to maintain stable blood pressure for the vital organs. When the electrical conduction system malfunctions it could cause the heart to beat too slowly, too quickly, or irregularly. The abnormalities in the rhythm represent a control problem within the cardiovascular system and can be treated using a pacemaker. The pacemaker serves as an external controller that regulates the timing of each heartbeat. Understanding the causes for the diseases and how a pacemaker can

help alleviate the problems it forms is essential for modeling the heart-pacemaker system as a feedback controlled dynamic system.

Several cardiovascular diseases disturb the normal conduction of electrical pulses. The Sinus Node Dysfunction (SND) is when the SA node is unable to produce a sufficient rate of impulses. Because the SA node is the pacemaker of the heart, any dysfunction will lead to a slowed down or irregular heartbeat. Some symptoms of SND are fatigue, dizziness, and fainting because their heart cannot maintain appropriate cardiac output. This condition is also one of the most common causes of Bradycardia. Another major issue is the heart block, which is caused when the electrical conduction between the atria and the ventricles is slowed down or stopped at the AV node. In severe heart block cases, the ventricles beat as low as 30 to 40 beats per minute. This is not enough beats per minute for the human body to function properly. Pacemakers are frequently used to overcome these problems by generating impulses that synchronize the atrial and ventricular contractions. Chronotopic incompetence is also an important condition to note. In this disorder, when the individual is exercising or stressed the heart is unable to increase its rate of impulses. This failure leads to fatigue and poor performance while exercising. Pacemakers with rate-responsive features fix this problem by automatically increasing the frequency of the pace during physical activity.

One of the more severe heart conditions is Bradycardia. Bradycardia is when someone has a resting heart rate below 60 BPM and this becomes dangerous because the slow pace prevents the heart from delivering sufficient blood to the body. When the heart beats too slowly each contraction becomes too weak and unstable to keep a steady blood pressure which leads to a decrease in oxygen transported to the brain and vital organs. Some symptoms are fatigue, dizziness, shortness of breath, confusion or memory loss, chest discomfort, and fainting.

Bradycardia can be caused by SA node dysfunction, AV node problems such as heart block, low thyroid function, congenital structural abnormalities, infection of cardiac tissue, chemical or electrolyte imbalances, or damage resulting from aging or surgery. Even though there are a plethora of causes, they all cause the heart rate to slow, which can be solved by a pacemaker.

Pacemakers work by constantly monitoring the intrinsic cardiac activity and giving an electrical pulse when the heart fails to beat within the specified time interval. The programmable parameters determine how the pacemaker will interact and help with the heart. Pacing pulse timing is important because it controls when an electrical impulse is delivered. If the heart rate falls below 60 BPM, the pacemaker will give a pulse that helps jumpstart a heartbeat and restores it to the proper timing. Pulse amplitude is the strength of the electrical signal and is an important control input that must be high enough to depolarize the cardiac tissue. If it's too high it will waste the battery power of the pacemaker and irritate the myocardium. Pulse width is the time duration of the electrical pulse and it affects how well the heart tissue will respond. Wider pulses increase the likelihood of successful capture in damaged tissue where, on the other hand, short pulses conserve energy. In dual-chamber pacemakers, AV delay is programmed to mimic the natural delay between atrial and ventricular contraction which helps to maintain the normal synchronization between chambers. With proper control parameter inputs, pacemakers can regulate heart rhythm and restore normal blood flow.

Control Inputs

To model the behavior of the cardiovascular system and its interactions with the pacemaker, we represent the heart rate using a simplified first-order ODE. In this, the rate of change of the heart depends on the difference between the current heart rate and the intrinsic rate

of the patient, which is usually low in Bradycardia patients. This pacemaker provides the external input that drives the system toward the ideal heart rate. The proposed model is a closed loop system which includes an ODE in the form dx/dt + a(x(t) - HR) = u(t), where x(t) represents the current heart rate, HR is the patient's natural intrinsic heart rate, and u(t) represents the pacemaker's electrical input. The pacemaker uses a proportional control law u(t) = Kp(x(t) - r), where r is the reference heart rate, set to 60 BPM, and Kp determines how strongly the pacemaker responds to the deviations from this target. This model allows us to analyze and simulate how pacing pulses influence the heart's dynamics, especially in Bradycardia patients whose intrinsic heart rate is below 60 BPM, usually between 30 to 50 BPM.

The block diagram for the closed loop system can be seen in figure 1.

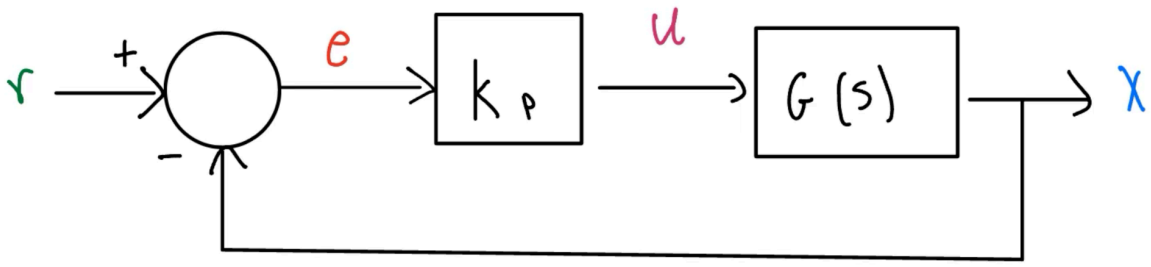

Figure 1: Closed Loop Block Diagram

By using a laplace transform X(s) can be determined from the ODE and the proportional control law.

$$\frac{dx}{dt} + \alpha(x(t) - HR) = (u(t))$$
$$u(t) = Kp\,(x(t) - r)$$

$$\frac{dx}{dt} + \alpha(x(t) - HR) = Kp\,(x(t) - r)$$
$$\frac{dx}{dt} + \alpha x(t) - Kp\,x(t) = \alpha HR - Kpr)$$

Now a Laplace transform can be applied:

$$sX(s) + \alpha X(s) - Kp\,X(s) = (\alpha HR - Kpr)/s$$
$$X(s) = (\alpha HR - Kpr)/(s^2 + s(\alpha - Kp))$$

The Transfer function is determined:

$$G(s) = \frac{X(s)}{U(s)}$$
$$G(s) = \frac{1}{s+\alpha}$$

Limiting Cases

In the case that the heart of the patient is beating faster than expected such that it is greater than 60 BPM our model would provide a negative controller input to try and bring the heart rate back down to 60 BPM. This is not necessary because a human heart beating between 60 BPM and 100 BPM is in the healthy range. In order to avoid this problem the ODE for the controller could include saturation that makes the controller input zero if the value of Kp (x(t) - r) is negative.

In the case that the heart is beating slower than expected, lower than 30 BPM, the value of u(t) will be very large for this simple linear ODE and proportional control law. This can cause impossible or dangerous rates with huge overshoot and again negative rates from the controller. Again, this problem could be avoided by saturating the control that defines a maximum value of u(t).

In conclusion, the simple ODE and control law could be edited to more safely monitor the patient's heart rate with saturation of the control law. An example of appropriate saturation is shown below.

$$u(t) = sat(Kp\,(x(t) - r), 0, u_{max})$$

$$\text{v} = Kp\,(x(t) - r)$$

If $v \leq 0$ the control input is defined as $u(t) = 0$

If $0 < v < u_{max}$ the control input is defined as $u(t) = Kp\,(x(t) - r)$

If $v \geq u_{max}$ the control input is defined as $u(t) = u_{max}$

State Space Model

To analyze how a pacemaker interacts with the heart, we created a simplified state-space model that captures cardiac electrical activation of a pacemaker and the resulting arterial pressure response. Pacemakers function by sensing electrical depolarizations in cardiac tissue and delivering pacing pulses when the intrinsic rhythm is too slow or irregular. Although dual-chamber pacemakers stimulate both the atrium and ventricle, our model focuses on the ventricular response, since this chamber generates the arterial pressure needed to maintain blood flow.

The system has two states:

$x_1(t)$: ventricular electrical activation

$x_2(t)$: arterial blood pressure deviation from baseline

The system output is $y(t) = x_2(t)$, which indicates how much the arterial pressure rises after each heartbeat.

The input to the system is $u(t)$ = pacemaker stimulus, representing the electrical pulse delivered by the pacemaker when needed.

Ventricular electrical activation decays rapidly if no stimulation occurs. A pacemaker pulse provides an immediate increase in activation. This behavior is modeled by the first equation:

$$\dot{x}_1 = -\frac{1}{\tau_e}x_1(t) + k_e u(t)$$

Here, $\tau_e = 0.1s$ is the fast electrical time constant, and $k_e = 1$ scales the effect of the pacing pulse.

Arterial pressure responds to ventricular activation and then slowly returns toward baseline. This is modeled by:

$$\dot{x}_2 = -\frac{1}{\tau_p}x_2(t) + k_p x_1(t)$$

The pressure time constant $\tau_p = 1\ s$ is much slower than τ_e, reflecting the duration of the mechanical pressure waveform. The gain $k_p = 10$ determines how strongly electrical activation affects pressure.

Together, these equations represent the sequence of the pacemaker pulse causing electrical activation which leads to contraction then a pressure increase and then relaxation.

Define the state vector as $x(t) = \left[x_1(t),\ x_2(t)\right]^T$

The state-space equations are:

$$\dot{x}(t) = Ax(t) + Bu(t)$$
$$y(t) = Cx(t) + Du(t)$$

where:

$$A = \begin{bmatrix} -\frac{1}{\tau_e} & 0 \\ k_p & -\frac{1}{\tau_p} \end{bmatrix}$$

$$B = \begin{bmatrix} k_e \\ 0 \end{bmatrix}$$

$$C = [1\ 0]$$
$$D = [0]$$

Step Response

The system response was modelled in MATLAB to create a visualization of the system response to a step input over time. In reality, the patient's heart rate would not be starting from zero bpm, however the response shape, rise time, and steady state error would be very similar.

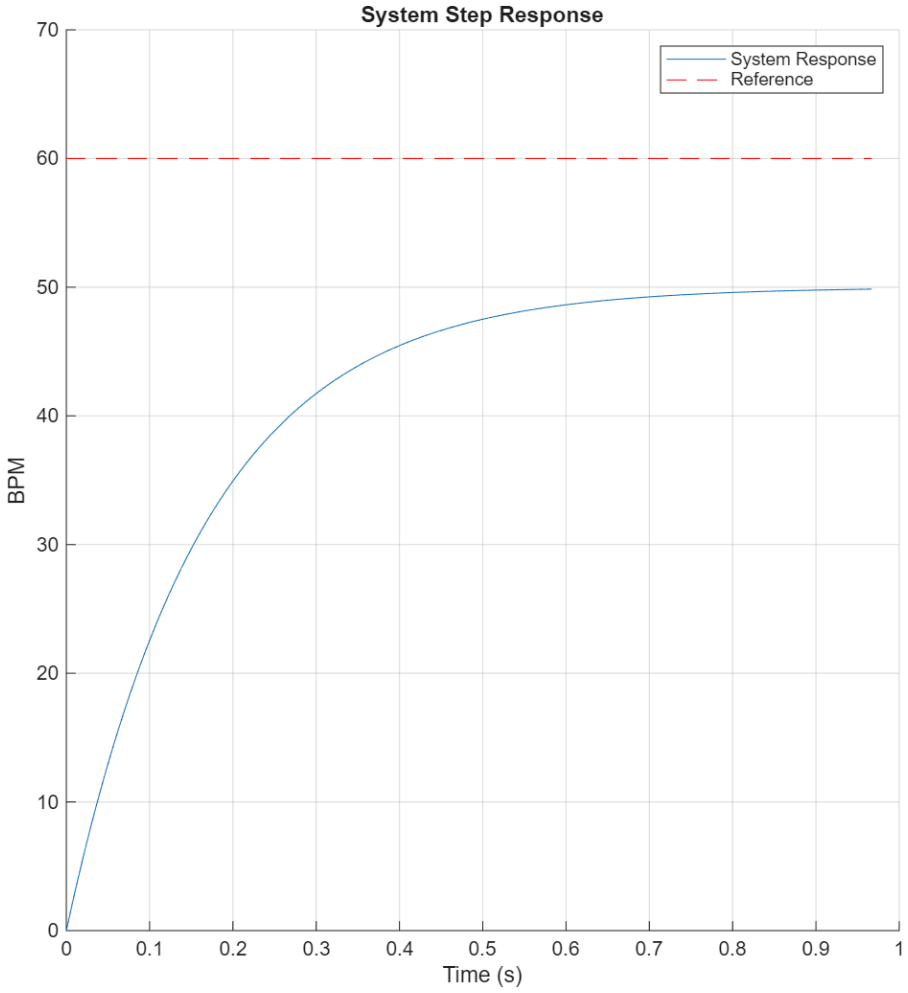

Figure 2: step response graph

As seen in figure 2, a steady state error is present in this system due to the use of only a proportional controller. This could be dangerous in an application such as the pacemaker-heart system, where the system needs to react quickly and accurately. In reality, a pacemaker would need to be designed with a PD or PID controller.

Sources Cited

- **https://www.ncbi.nlm.nih.gov/books/NBK544253/**
- **https://www.mayoclinic.org/diseases-conditions/bradycardia/symptoms-causes/syc-20355474**
- **https://www.heart.org/en/health-topics/arrhythmia/about-arrhythmia/bradycardia--slow-heart-rate**
- **https://pmc.ncbi.nlm.nih.gov/articles/PMC5466412/**
- **https://oneheartcardiology.com.au/service/pacemaker-implantation/**