

A POSSIBLE APPLICATION OF THE MECHANICS OF FLUIDS AND THE NONLINEAR DYNAMICS OF BEHAVIOR OF THE HEART

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ABSTRACT

In this work, we use some results from mechanics of fluids and the thermodynamics of finite time to calculate, the efficiency of the heart, considering the heart like a mechanics pump. We also study the model of the heartbeat. We find results of the efficiency to heart far away from the real values, i.e. the model to heart like a mechanics pump is too oversimplified, the above is because the heart is a complex system. However using the nonlinear dynamics considering the time delays inherent of the system, we obtain two fixed points which sustained oscillations, and these could be the Diastole and Systole, these parameters are associated with blood flow in human body called pressure arterial and are very important in the cardio-vascular problems. We have to point out that our work is from point view academic, and pretend to the students taking in account that the theory can be immediately applied to many real systems, but also has very limitations.

INTRODUCTION

Mathematically modeling physical phenomena, such as a rigid body dynamics of the deformation of an elastic material or the propagation of an electromagnetic wave in the atmosphere, our best scientific knowledge about the laws of physics. So for the first case we used Newton's laws, the continuum mechanics and Maxwell's laws.

These laws are the result of centuries of experimentation, observation and inspiration of the scientists involved in the creation of knowledge of nature.

In biology and life sciences in general, the interaction between the observed phenomena and their mathematical description, are still in the early stages of development and apart from the Hardy-Weinberg equilibrium. The philosophy is to develop mathematical models that can describe in a qualitative manner observed biological processes.

There is a lot of work in the literature of complex systems, which consider its efficiency analysis and even related to probable heart heartbeat analyzed as time series, others show dynamic and thermodynamic models. Our the study subject is the heart, so we give a brief description about its operation.

The heart is one of the most important organs of the human body and it has as principal task, maintain circulation the blood, so the function of the heart is to pump blood. The blood carries oxygen (O_2) from the lungs to the various tissues of the body and it carries carbon dioxide (CO_2) from these tissues back to the lungs. Since the circulation forms a closed loop, its description can begin anywhere. We will begin on the left side of the heart. The left heart side receives blood rich in O_2 and pumps this blood into the systemic arteries. These form a tree of progressively smaller vessels that supply fully oxygenated (and hence bright red) blood to all of the organs and tissues of the body. From the smallest of the systemic arteries, blood flows into the systemic capillaries, which are roughly the diameter of a single red

blood cell. It is in capillaries that the actual exchange of O_2 and CO_2 takes place. The blood that leaves the systemic capillaries carries less O_2 and more CO_2 than the blood that entered. (The loss of O_2 causes a change in the color so that the blood is now more bluish than before.)

Leaving the systemic capillaries, the blood enters *systemic veins* through which it flows in vessels of progressively increasing size toward the right side of the heart.

The right heart pumps blood into the *pulmonary arteries* which form a tree that distributes the blood to the tissues of the lung. The smallest branches of this tree give rise to the *pulmonary capillaries* where CO_2 leaves the blood stream and O_2 enters from the air space of the lungs. Leaving the pulmonary capillaries, the oxygenate blood is collected in the pulmonary veins through which it flows back to the left heart. This complete the circulation, F. C. Hoppensteadt [1].

BLOOD FLOW

The circulatory blood system

If we consider for a moment a simplified concept of the circulatory blood system in man, we can imagine that we have a pump delivering blood to a complicates networks of pipes, which has innumerable connections. To develop an appropriate mathematical model of this system and its behavior is an almost impossible task. Thus, in order to make any progress, we attempt to model parts of system separately. Here we concentrate on a small section of this circuit, say in the region of the aorta as shown in Fig. 1. Indeed we shall consider the relative straight section between A and B. One can imagine that the blood flow in this section behaves in much the same way a water in a cylindrical tube. This, however, is a gross oversimplification of the situation. To see this, let us consider some salient facts regarding blood flow. First of all unlike water, blood does not have constant

viscosity and this vary with the velocity. Thus blood may be claimed to be non-Newtonian; indeed the properties of blood change rapidly if removed from the system and so it is extremely difficult to perform experiments on it under laboratory conditions .

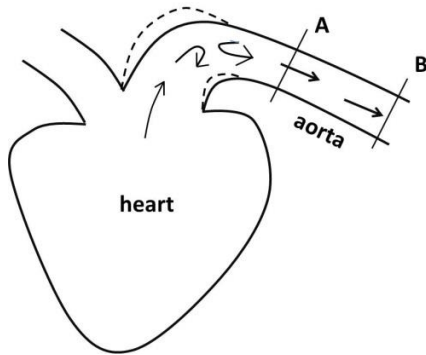


Fig. 1 Schematic description of an aorta

If we now consider the type of flow in an artery, it is apparent that because the heart delivers blood in short bursts during contraction into systole, the flow is pulsatile and not uniform. Furthermore, we do not know the velocity profile of the flow entering A in Fig. 1 and consequently the velocity profile at B is also unknown. This observation is of fundamental importance in the mathematical description of blood flow. On the other hand, the hydrodynamic problem of considering the change of an initial velocity profile a Newtonian fluid in a rigid pipe is fairly well understood and is based on the fundamental theory of Poiseuille (1846). One should remark here that Poiseuille whose contributions to hydrodynamics are well known to engineers and mathematicians, was in fact a physician and his interest was precisely the problem we are considering here, namely, the study of the blood flow.

Let us now focus on the arteries themselves. We know them to be elastic and a typical cross section may change significantly with time due to pulsating nature of the flow of blood. Thus once again it may be unreasonable to treat the arteries as rigid tubes. Nevertheless we find it necessary to assume this as first approximation.

In Fig. 1 consider the flow of blood delivered into an aorta. The blood is pumped in an asymmetrical fashion and there large cross-channel components of velocity in the arch region and consequently large thoracic surgery on animals. However, away from the arch itself, say in section A-B, the cross-channel components of velocity are considerably reduced and the flow is almost entirely longitudinal but, of course, still pulsatile. In the arc region it is found thoracic surgery that the arc pliant and yields easily to the cross-channel pressure gradients. Thus it is reasonable to assume changes and the “general give” radially of all cross sections of the aorta cause changes in pressure to be dampened, especially the radial components. We radial velocity components may be neglected. This assumption is known to physiologists as the Windkessel effect assumption, an idea introduced by the German physiologist Otto Frank, D. S. Jones [2].

Mechanics of Fluids

We define a fluid as a substance which must continue to change shape as long as there is a shear stress, however small, present. By contrast a solid undergoes a definite displacement

(or breaks completely) when subjected to a shear stress. In fluid mechanics study different types of fluids such as compressible, incompressible, Newtonian and non-Newtonian. In the previous section studied that blood flow can be considered as a non-Newtonian fluid, as this does not satisfy the conditions to be studied as incompressible and Newtonian fluid. A. Bejan [3] was obtained an expression to calculate the efficiency for a piston and cylinder apparatus for extracting mechanical power from the flow of a fluid between two pressure reservoirs, given as

$$\eta_{max} = \frac{1}{2} \left(1 - \frac{P_2}{P_1} \right) \quad (1)$$

where η_{max} is the maximum efficiency, P_1 is the reservoir pressure, P_2 is the reservoir pressure with $P_1 > P_2$. The Fig.11-28 of A. Vander et al [4] shows that the initial pressure reaches a value more than 110 mmHg before going through the aorta.

Now as we saw in the physiology of the left side of the heart, we can assume in a first approximation that this can be represented as proposed in [3], this is shown in Fig.2.

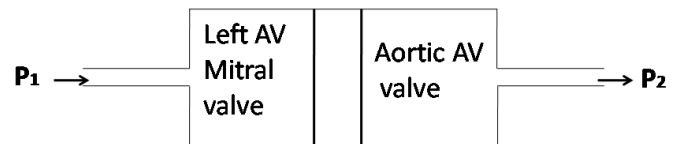


Fig. 2 Piston and cylinder apparatus for extracting mechanical power from the flow of a fluid between two pressure reservoirs.

On the other hand, the maximum pressure supported by a vein is 140 mmHg, also the normal arterial pressure in person without hypertension is 120 mmHg, moreover the biological systems have good efficiency, then we can suppose that the heart’s efficiency is around 30%, i.e. $\eta_{max} = 0.3$, so from Eq. (1) we obtain that $P_1 = 300\text{mmHg}$, with $P_2 = 120\text{ mmHg}$, these result cannot be real. Therefore our approximation of the heart like mechanics pump is far from the reality. And we have to propose a model that takes in account more details of the blood flow.

NON- LINEAR DYNAMICS

As is known, the heart muscle is an autonomous system and has an intermittent dynamic, i.e. its operation is periodic. Then we use Non-linear Dynamics considering the time delays inherent to the heart to study the model heart’s dynamics proposed by E. C. Zeeman [5].

Times Delays

In real life situations when the value of a variable is modified the effect in the dynamic response of the system is not observed immediately. A certain time must elapse until the system begins to respond or "feel" the effect of the changes made. Suppose we modify the concentration of a reactor feed. Our experience, and common sense tells us that time passes until the variables that characterize the dynamic behavior of the reactor (eg concentration) begin to modify its value relative to their pre-change. These systems are known as dynamical systems. Delayed systems appear naturally in Medicine, Biology and Engineering. These systems have been

studied since before the last century. Studies in Medicine and Biology begin with Ross' epidemiology models (1911) and others in the early twentieth century, which were studied by Lotka, Volterra and Kostitzin, N. McDonald [6]. A distinctive feature of these systems is that their rate of evolution is described by differential equations that include information about the history of the system. The effects of delays are of great interest, since their presence may include complex behavior (oscillations, instability, bad system performance). R. Páez-Hernández et al [7] studied the effect time delays produced in a mathematical model for the stretch reflex regulatory pathway. A. Rojas-Pacheco et al [8] studied time-delay effects on dynamics of a two-actor conflict model.

Fixed points and linearisation system with delays

Consider a dynamic system which has a single variable with time delays ξ ,

$$\frac{dx}{dt} = f(x, y_\xi) \quad (2)$$

$$\frac{dy}{dt} = g(x_\xi, y) \quad (3)$$

where subscript ξ is a time delay variable. Following step to step to H. S. Strogatz [9] to obtain a linear system,

$$\dot{u} = f(x^*, y^* + v_\xi), \quad \dot{v} = g(x^* + u_\xi, y^* + v), \quad (4)$$

where u and v represent a small perturbation of the system and (x^*, y^*) is a fixed point, now we do a Taylor's series expansion to Eq. (2) and we consider negligible the terms of two on ward, and evaluate in the steady-state and we obtain

$$\dot{u} = \left. \frac{\partial f}{\partial x} \right|_{(x^*, y^*)} u + \left. \frac{\partial f}{\partial y} \right|_{(x^*, y^*)} v_\xi \quad (5)$$

$$\dot{v} = \left. \frac{\partial g}{\partial x} \right|_{(x^*, y^*)} u_\xi + \left. \frac{\partial g}{\partial y} \right|_{(x^*, y^*)} v. \quad (6)$$

Now we assume that u and v are of the form

$$u = A_1 e^{\lambda t} \quad (7)$$

$$u_\xi = A_1 e^{\lambda t} e^{-\lambda \xi} \quad (8)$$

$$v = A_2 e^{\lambda t} \quad (9)$$

$$v_\xi = A_2 e^{\lambda t} e^{-\lambda \xi} \quad (10)$$

where λ is a complex number, A_1 and A_2 are constant.

Substituting Eqs. (7)-(10) into Eqs. (5) and (6) leads to the following set of homogeneous linear system for A_1 and A_2 :

$$(f_x - \lambda)A_1 + f_{y_\xi} e^{-\lambda \xi} A_2 = 0 \quad (11)$$

$$f_{x_\xi} e^{-\lambda \xi} A_1 + (g_y - \lambda)A_2 = 0. \quad (12)$$

This system of equations has non-trivial solutions only if the determinant of the matrix of coefficients equals zero, i.e.

$$(f_x - \lambda)(g_y - \lambda) - f_{x_\xi} g_{y_\xi} e^{-2\lambda \xi} = 0. \quad (13)$$

This equation is also called the transcendental characteristic equation, and can be written as

$$H(z) + K(z)e^{-z\xi} = 0, \quad (14)$$

with z an eigenvalue, and $H(z)$ and $K(z)$ are polynomials of second and zero order, respectively.

The solutions to this equation are not obvious because has an infinite number of roots [6]. One way to overcome this situation is to consider the fact a common effect of time delays to destabilize stable fixed points or to stabilize unstable fixed points by sustained oscillations. If we assume that $(z = i\omega)$, and substitute in (14), we obtain a complex variable equation.

$$P(\omega) + iQ(\omega) = e^{-i\omega\xi} \quad (15)$$

where $P(\omega)$ and $Q(\omega)$ are second and first order polynomials, respectively. We observe that the right hand side of this equation represents the unitary circle whereas the left hand side describes a parabola. The intersection of these two curves could represent a change in the stability of the system. The analysis of intersection between the parabola and the unitary circle leads to the following classification:

- If the parabola does not intersect the unitary circle, and the system is stable to $\xi = 0$, then the system is stable independent of delay.
- If the system is stable for $\xi = 0$ and the parabola intersects the unit circle, then the system can be affected by delays.

Non-Linear Dynamic Model of Heart

A mathematical model that describes the behaviour of the heartbeat was developed in [5], where it was suggested that such a model contain three basic features:

- a stable equilibrium state representing diastole;
- the threshold for triggering the electrochemical wave causing the heart to go into systole; and
- the return of the heart into the diastolic state.

The resulting model is given by

$$\dot{x} = -\frac{1}{\epsilon}(x^3 - Tx + y) \quad (16)$$

$$\dot{y} = x + x_d \quad (17)$$

where $x(t)$ represents the length of the muscle fiber, $y(t)$ is a variable related to electrochemical activity; the parameter ϵ is a small positive constant associated with the fast eigenvalue of the system, x_d is a scalar quantity representing a typical length of muscle fiber in the diastolic state, and T represents tension in the muscle fiber. Now we use the result for linearization systems, for the case $\xi = 0$,

$$f(x, y) = -\frac{1}{\epsilon}(x^3 - Tx + y) \quad (18)$$

and

$$g(x, y) = x + x_d \quad (19)$$

this yields,

$$f_x|_{(x^*, y^*)} = \frac{T}{\epsilon} \quad (20)$$

$$f_y|_{(x^*, y^*)} = -\frac{1}{\epsilon} \quad (21)$$

$$g_x|_{(x^*, y^*)} = 1 \quad (22)$$

$$g_y|_{(x^*, y^*)} = 0 \quad (23)$$

now substituting Eqs.(20)-(23) into Eq. (13), we obtain the eigenvalues $\lambda_1 = 3.62$ and $\lambda_2 = 1.38$ for $T = 1$, $\epsilon = 0.2$ and $x_d = 0$. Therefore, the origin is unstable since both eigenvalues are real and positive. In Fig. 3 we show the phase portrait of Eqs. (2) and (3), with the same values for the parameters, the cubic line (red curve) represents the steady state of Eq.(18), A and B may be represent the systole and diastole points.

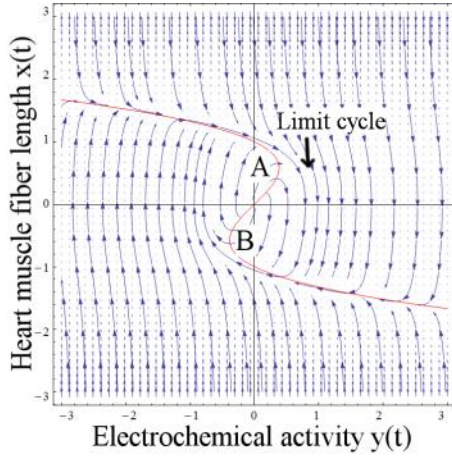


Fig.3 Phase portrait of the heartbeat model.

Dynamic effects of time delays

Consider again the systems of delay differential equations given by Eqs. (2) and (3), but now $\xi = \frac{\pi}{2}$. They can rewritten as

$$\begin{aligned} \frac{dx}{dt} &= f(x, y_{\pi/2}) \\ \frac{dy}{dt} &= g(x_{\pi/2}, y) \end{aligned}$$

with f and g as defined in Eqs. (18) and (19). Following section 2.1, the time course of small perturbations from the steady state is determined; we can write Eq. (13) as

$$(f_x - \lambda)(g_y - \lambda) - f_y g_x e^{-\lambda\pi} = 0. \quad (24)$$

The stability analysis of a dynamic system involving time delays can be quite complicated due to the fact that, in general, the characteristic equation has an infinite number of solutions. On the other hand, it is known that a common effect of time delays is to destabilize formerly stable steady states by inducing sustained oscillations. To test whether this happens, assume that λ is imaginary ($\lambda = i\omega$) and substitute into the characteristic equation to obtain

$$(-E\omega^2 + F) + iD\omega = e^{(-i\omega\pi)}, \quad (25)$$

wit

$$E = \frac{1}{f_y g_x}, \quad F = \frac{f_x g_y}{f_y g_x} \quad \text{and} \quad D = \frac{f_x + g_y}{f_y g_x}.$$

It follows from Eqs. (20)-(23) that $f_x > 0$, $f_y < 0$, $g_x > 0$ and $g_y = 0$. This further implies that $E < 0$, $F=0$, and $D > 0$.

The left-hand side of Eq. (25) determines the lower branch of a horizontal parabola in the complex plane. This parabola opens to the to the right and its vertex is located in the point (0,0). On the other hand, the right-hand side of Eq. (25)

determines a unitary circle in the complex plane. The points where these curves cross correspond to values of ω and π at which sustained oscillations appear due to a destabilization of the steady state, or vice versa. Let ρ and σ real variables along the real and the imaginary axes of the complex plane, respectively. In terms of these variables, the equation for the parabola can be written as

$$\rho = -\frac{E}{D^2} \sigma^2, \quad (26)$$

While the equation for the circle is

$$\rho^2 + \sigma^2 = 1. \quad (27)$$

To find the points where both curves cross, solve for σ in Eq. (26) and substitute into Eq. (27) to obtain

$$\rho^2 - \frac{D^2}{E} \rho = 0. \quad (28)$$

The solutions to this last equation give the real coordinates of the crossing points. The corresponding imaginary coordinates can then calculated as $\sigma = -\sqrt{1 - \rho^2}$. The solutions of Eq. (28) are

$$\rho_1 = \frac{L}{2} + \frac{1}{2} \sqrt{L^2 - 4K}, \quad (29)$$

$$\rho_2 = \frac{L}{2} - \frac{1}{2} \sqrt{L^2 - 4K}, \quad (30)$$

with $L = D^2/E$ and $K = -1$. From its definition and the fact that E is negative and D is positive, L is negative. Notice that ρ_1 and ρ_2 have common points. Therefore, the parabola of Eq. (26) crosses the unitary circle in the points $(-0.19, \pm 0.98)$, this indicating that there a two points which induce oscillations, i.e., those points can be destabilize the system. In Fig. 4 shows these points.

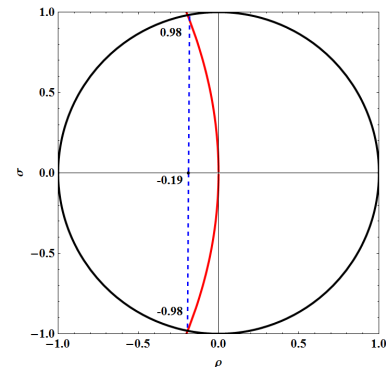


Fig. 4 Plot shows the intersections between unit circle and the parabola given in Eqs.(26) and Eqs.(27).

COMMENTS

From the result obtained with mechanics of fluids we can assert, that our approximation of the heart like mechanics pump is far from the reality. We will looking another model that takes in account more details of the blood flow, and probably we can get an expression, that gives values more realist. Finally we have to remark that Non-linear dynamics is an useful and powerful tool to tackle system with time delays, because it was possible to get two points which destabilizes the system, and assume that the intersection of these points could be associated with both arterial pressure the called Systole and Diastole, important parameters of pressure in blood flow.

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NOMENCLATURE

Symbol	Quantity	SI Unit
x	length of muscle fiber	(m)
x_d	typical length of muscle fiber in the diastolic state	(m)
y	variable related to electrochemical activity	(J)
δx	small disturbances from the corresponding fixed point values	(m)
δy	small disturbances from the corresponding fixed point values	(m)
λ_1	eigenvalue 1	(Hz)
λ_2	eigenvalue 2	(Hz)
ξ	time delay	(s)
ω	frequency	(Hz)
P_1	constant high pressure reservoir	(Pa)
P_2	constant low pressure reservoir	(Pa)
T	tension in the muscle fiber	(N)
\vec{u}_1	eigenvector corresponding to eigenvalue λ_1	(m)
\vec{u}_2	eigenvector corresponding to eigenvalue λ_2	(m)

Dimensionless Quantity

ϵ	is a small positive constant
η_{max}	efficiency
A	systole
B	diastole
E	constant
F	constant
D	constant
i	imaginary unit
A_1	arbitrary constant 1
A_2	arbitrary constant 2
B_1	arbitrary constant 1
B_2	arbitrary constant 2

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