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Full Length Research Paper

The use of a continuity equation of fluid mechanics to reduce the abnormality of the cardiovascular system: A control mechanics of the human heart

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The paper is aimed at presenting the differential equations for the cardiovascular system with the help of continuity equation of fluid mechanics to reduce the abnormality of the rate of blood flow and variation of blood volume in different parts of the system. The equations are used to explain the Frank-Starling mechanism, which plays an important role in the maintenance of the stability of the distribution of blood in the system. This is a reasonable approach based on mathematical considerations as well as being further motivated by the observations that many physiologists cite optimization as a potential influence in the evolution of biological systems. We present a model as an application in the provision of a basis for developing information on steady state relations and also to study the nature of the controller and key controlling influences. The model further provides an approach for the study of complex physiological control mechanisms of the cardiovascular system and possible pathways of interaction between the cardiovascular and respiratory control systems. The study also provides an easy way for students of both physics and mathematical sciences, with no previous knowledge of human physiology, to understand the basic systems in cardiovascular concept.

Key words: Continuity equations, fluid mechanics, cardio-vascular system.

INTRODUCTION

In this paper, the concept of fluid mechanics are shown to be useful for the understanding of cardiovascular mechanics, exemplifying the fact that physics can play a fundamental role in the investigation of phenomena in human physiology. To properly understand the application of fluid mechanics (Falkovic, 2011) to human cardiovascular system (Hugh et al., 2005), it is necessary to make the assumption that the cardiovascular system is a closed system and also to describe the cardiovascular system itself (Dwivedi and Dwivedi, 2007). The cardiovascular system is a closed tabular system in which the blood is propelled by a muscular heart via two circuits: the pulmonary and systemic that consist of arterial, capillary and venous components (Dwivedi and Dwivedi, 2007; Mohammadali et al., 2009). It is a system that keeps life pumping through the body with its complex pathways of veins, arteries and capillaries (Mohammadali et al., 2009; West, 2008). The cardiovascular system consists of the heart, blood vessels and the circulatory system (Taylor et al., 1997). On the other hand, the heart is a muscular organ that provides a continuous blood circulation through the cardiac cycle. The heart is divided into four chambers: the two upper chambers called the left and right auricles and the two lower chambers called the left and right ventricles. There is a thick wall of muscles separating the right side and the left side of the heart called the septum. The heart consists of two large veins, the superior/anterior and the inferior/posterior vanae cavae, which bring red deoxygenated blood from

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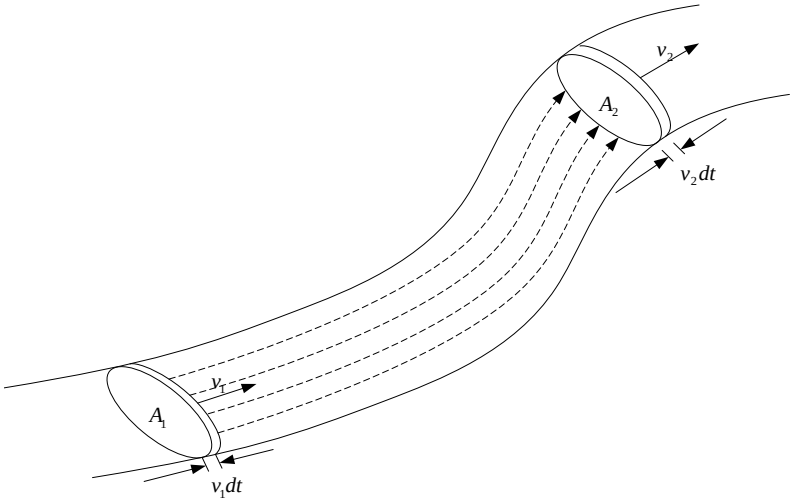

Figure 1. Diagrammatic representation of a flow tube with changing cross-sectional area. If the fluid is incompressible, the product Av has the same value at all points along the tube.

the various parts of the body with the exception of the lungs. The superior is located near the top of the heart while the inferior is located just beneath the superior (Taylor et al., 1997). The main function of the heart is therefore to pump blood round the tissue through systolic and diastolic processes triggered by the spontaneous discharge (Mathias and Andrew, 2011). These can be explained through electrical and mechanical activities of the heart (Massey and Ward-Smith, 2005; Falkovic, 2011). These processes are therefore interesting not only for physics students, but also for students of the biological and medical sciences, because they illustrate the possibility of formulating theories in these areas; hence the justification for the present study.

AN OVERVIEW OF CONTINUITY EQUATION

One may need to know the idea behind the continuity equation of fluid mechanics before discussing it with respect to cardiovascular system. The mass of a moving fluid doesn't change as it flows. This leads to an important quantitative relationship called the continuity equation. Zemasky (2005) considers a portion of flow tube between two stationary cross sections with areas A1 and A2 (Figure 1). The fluid speeds at these sections are v_1 and v_2, respectively. No fluid flows in or out across the sides of the tube because the fluid velocity is tangent to the wall of every point on the wall. During a small time interval dt, the fluid at A1 moves a distance v_1dt, so a cylinder of fluid with height v_1dt and volume $dv_1 = A_1v_1dt$ flows into the tube across A1. During this same interval, a cylinder of volume $dv_2 = A_2v_2dt$ flows out of the tube across A2. Let's first consider the case of an incompressible fluid so that the density ρ has the same value at all points. The mass dm_1 flowing into the tube across A1 in time dt is $dm_1 = \rho A_1v_1dt$. Similarly, the mass dm_2 that flows out across A2 in the same time is $dm_2 = \rho A_2v_2dt$. In steady flow the total mass in the tube is constant, so $dm_1 = dm_2$ and $\rho A_1v_1dt = \rho A_2v_2dt$ or, continuity equation, incompressible fluid:

$$A_1v_1 = A_2v_2 \quad (1.1)$$

The product Av is the volume flow rate dv/dt, the rate at which volume crosses a section of the tube; *volume flow rate*

$$\frac{dv}{dt} = Av \quad (1.2)$$

The mass flow rate is the mass flow per unit time through a cross section. This is equal to the density (ρ) times the volume of flow rate (dv/dt)

Equation (1.1) shows that the volume flow rate has the same value at all points along any tube. When the cross

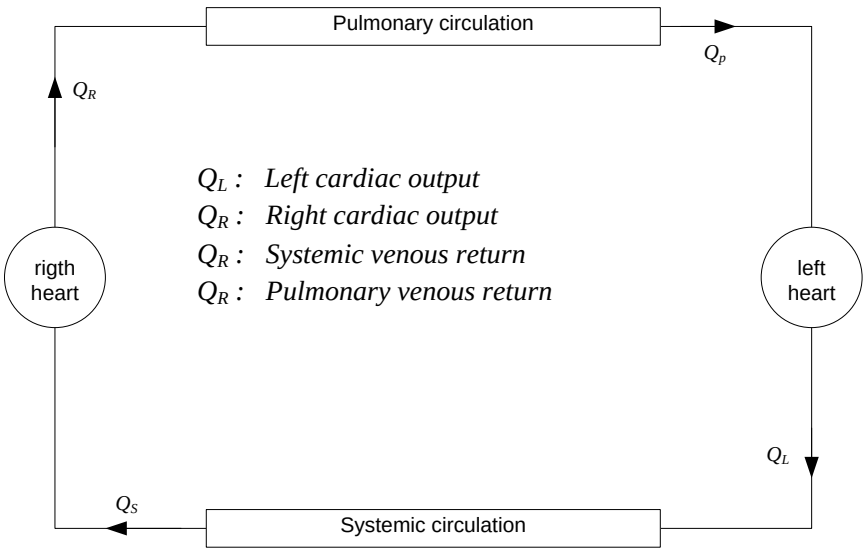

Figure 2. The cardiovascular system.

section of a flow tube decreases, the speed increases, and vice versa.

We can now generalize Equation (1.1) for the case in which the fluid is not incompressible. If ρ_1 and ρ_2 are densities at sections 1 and 2, then, the continuity equation, compressible fluid:

$$\rho_1 A_1 v_1 = \rho_2 A_2 v_2 \quad (1.3)$$

Cardiovascular system

Cardiovascular system consists of a double pump, the heart, and two distinct circulatory systems, i.e., systemic and pulmonary. The heart is divided into two parts, which are called the left and right hearts. Each has two chambers, the atrium and the ventricle, which periodically contract and relax. The relaxation and contraction are synchronized such that when the atrium is contracting, the ventricle is relaxing and vice-versa. The atrium receives and stores blood during the ventricular contraction, and blood flows from the atrium to the ventricle pumped by each heart per unit time is called the cardiac output (Williams and Ganong, 2005). The flow of blood through the system circulations depends on the contraction of the left ventricle, whereas the right heart drives blood through the pulmonary circulation, where blood is oxygenated and CO_2 is disposed. A system of valve ensures that blood flows in the direction as shown in Figure 2.

In Figure 2, Q_L is the cardiac output, Q_R right cardiac output, Q_S systemic nervous return and Q_P pulmonary venous return respectively.

The cardiovascular system is regulated by both internal and external factors. The effectiveness of the regulation is manifested by the remarkable stability of the system. External factors including nervous activity and chemical substance called hormones, can affect the cardiac performance. There is an internal control mechanism, called the Frank–Starling mechanism that plays an essential role in the maintenance of the balance between the right and the left ventricular outputs and in the distribution of blood between the systemic and pulmonary circulation. A mathematical analysis of this control mechanism is essential for the thorough understanding of cardiovascular mechanics.

MATERIALS AND METHODS

Differential equation for the cardiovascular system

Cardiovascular mechanics is based on the continuity equation and on the momentum equations for blood flow throughout the system. Because the cardiovascular system is very complex, idealizations and approximations are necessary. However, there are numerous mathematical models, which differ in the way the momentum equations are taken into account. In simple models, the momentum equation are taken into account by phenomenological equations that relate the pressure, volume, and the flow of blood in different parts of the system, and by parameters that depends on the blood viscosity and on the geometry and elastic properties of the vascular beds. In more complex models, Navier – Stokes equations are applied to the blood flow and numerically integrated for the assumed boundary conditions (t and t + T). In this paper, only the continuity equation was considered. This is because it is sufficient to explain a control mechanism of blood flow in cardiovascular system.

Let $V_R^i(t)$ be the instantaneous volume of blood contained in

the right heart at time t . The continuity equation can be written as

$$d\left[V_R^i\left(t\right)\right]/\ dt = Q_S^i\left(t\right) - Q_R^i\left(t\right) \quad (1.4)$$

Where $Q_R^i\left(t\right)$ is the systemic or right venous return, that is, the instantaneous rate of blood flow (in liters per minute) from the systemic circulation into the right atrium, and $Q_R^i\left(t\right)$ is the instantaneous right cardiac output. The superscript i denotes instantaneous values.
Also, the instantaneous volume of blood in the systemic circulation, in the left heart, and in the pulmonary circulation satisfies

$$dV_S^i\left(t\right)/\ dt = Q_L^i - Q_S^i\left(t\right) \quad (1.5)$$

$$\frac{dV_L^i\left(t\right)}{dt} = Q_P^i\left(t\right) - Q_L^i\left(t\right) \quad (1.6)$$

$$\frac{dV_P^i\left(t\right)}{dt} = Q_R^i\left(t\right) - Q_P^i\left(t\right) \quad (1.7)$$

Where: $Q_L^i\left(t\right)$ and $Q_P^i\left(t\right)$ are the instantaneous left cardiac output and pulmonary venous return respectively. From (1.4) - (1.7), it can be deduced that:

$$d\left(V_L^i + V_R^i + V_S^i + V_P^i\right)dt = 0 = \frac{dV}{dt}, \quad (1.8)$$

which, expresses the conservation of total blood volume in the cardiovascular system.
In steady state, the instantaneous left cardiac out is a periodic function of time and can be written as

$$Q_L^i\left(t\right) = Q_L + f\left(t\right) \quad (1.9)$$

Where: Q_L is the average value of the left cardiac output, given by:

$$Q_L = \left(\frac{1}{T}\right)\int_t^{t+T} Q_L^i\left(t\right)dt \quad (1.10)$$

which, is always constant in time and its value is one after integration. The periodic function $f\left(t\right)$ has an average value equal to zero; T is the cardiac period.

It is observed that during transient phenomena, all the physiological variables in Equations (1.4) - (1.7) are not exactly periodic functions of time, and consequently, their average values in a given cardiac cycle are not necessarily equal to the corresponding values in another cycle. Thus, during transient phenomena, the average values of physiological variables depend on time.

For transient phenomena, during which the physiological quantities have averages that are slowly varying functions of time, Equations (1.4) - (1.7) yield:

$$\frac{dV_R}{dt} \cong Q_S - Q_R \quad (1.11)$$

$$\frac{dV_S}{dt} \cong Q_L - Q_S \quad (1.12)$$

$$\frac{dV_L}{dt} \cong Q_P - Q_L \quad (1.13)$$

$$\frac{dV_P}{dt} \cong Q_R - Q_P \quad (1.14)$$

In this approximation, the form of the equations for the average quantities is almost the same as that for the instantaneous ones. In the steady state, the time derivation on the left-hand side of Equations (1.11) - (1.14) are equal to zero, and hence, all rates of blood flow on the right-hand side have the same value.

It is important to note that there must exists a control mechanism that maintains for long time a balance between the left and right cardiac output, because without such a mechanism the distribution of blood in the system would be unstable. For example, if the right cardiac output remained larger than the left one for a long time, an abnormal accumulation of blood in the pulmonary circulation would occur, whereas the systemic circulation would gradually be emptied of blood. The control mechanism that prevents this problem and maintains the stability of the blood distribution is known as the Frank–Starling mechanism.

The ventricular function curve

It is noted that in 1914, Starling used a canine heart – lung preparation to demonstrate that increasing the stretch on the ventricle of the mammalian heart during relaxation increases the pressure developed during contraction, as depicted by Frank in 1895 where he used a ventricle of the frog heart to demonstrate a similar relationship. Starling found experimentally that there is a relation between cardiac output and right atrial filling pressure. The latter variable determines the degree of filling of the ventricle and may be regarded as a measure of the average blood volume in the heart. The experimental data obtained by Starling shows that the cardiac output first increases and then decreases when the right atrial filling pressure rises (William, 2005). The second part of the relation is called "*the descending limb of the starling curve*" and has been controversial. Because the right atrial filling pressure is a measure of the average volume of blood contained in the heart, Starling's experimental results can be expressed by saying that cardiac outputs are functions of the average volume of blood contained in the respective hearts. The relations $Q_L = Q_L\left(v_L\right)$ and $Q_R = Q_R\left(v_R\right)$ are called *ventricular functions* or *cardiac output*.

When subjects stand in the upright position, the ventricle clearly operates on the ascending limb of its function curve in a relatively steep region, and consequently, fluid administration can markedly

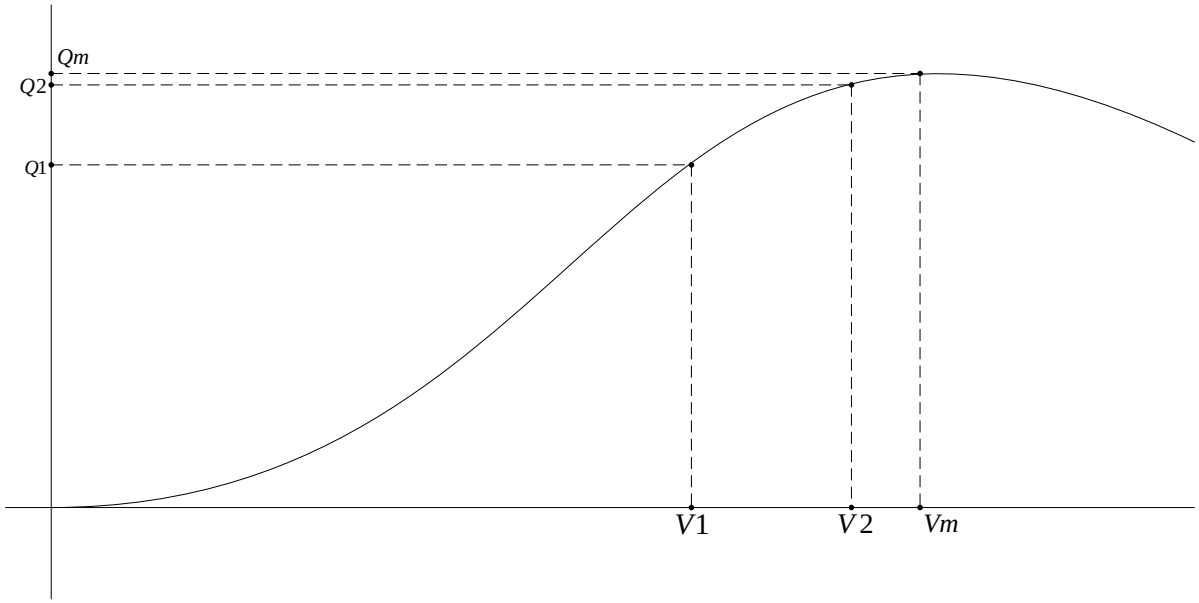

Figure 3. Ventricular function Q denotes cardiac output and v is blood volume in the heart.

enhance pump function. In contrast, in the supine position, in which the subject lies float on hither back, the ventricle operates closer to the maximum of its function curve. Attempts to increase the filling volume lead to an increase in filling pressure, but only to modest improvement in ventricular performance (Uehara and Sakane, 2002).

RESULTS AND DISCUSSION

The experimental observations show that the ventricular function is non-linear as illustrated in Figure 3, in which Q represents Q_L or Q_R, and v represents v_L or v_R. For simplicity, only one function is shown in Figure 4, but it should be noted that the two ventricular functions are not exactly the same (Uehara and Sakane, 2002).

Relationship between the Frank–Starling mechanism and differential equations of the cardiovascular system

The Frank–Starling mechanism or Starling's law of the heart states that the stroke volume of the heart increases in response to an increase in volume of blood filling the heart (the end diastolic volume). The increase volume of blood stretches the ventricular wall, causing cardiac muscle to contract more forcefully (the Frank–Starling mechanism). The stroke volume may also increase as a result of greater contractility of the cardiac muscle during exercise, independent of the end-diastolic volume. The Frank–Starling mechanism appears to make its greatest contribution to increasing stroke volume at lower work rates, and contractility has its greatest influence at higher rates (Costanzo, 2007). This allows the cardiac output to be synchronized with the venous return, arterial blood supply and humeral length without depending upon external regulation to make alterations. In addition, the Frank-Starling mechanism plays an essential role in the maintenance of the balance between the right and the left ventricular outputs and in the distribution of blood between the systemic and pulmonary circulation (Uehara and Sakane, 2002; Mathias and Andrew, 2011).

Before using Equations (1.11) - (1.14) to explain the control mechanism, it is interesting to consider one quantitative explanation that can be found in physiology textbooks (McGeon, 1996). It was reported that the most important intrinsic mechanism involved in the control of cardiac output is usually referred to as Starling's law of the heart, or the Frank–Starling mechanism, after the two physiologist who first described it. Starling's law helps explain two important features of cardiac function, namely that cardiac output equals venous return and that the average outputs from the two ventricles are equal. If venous return suddenly rises above ventricular output, blood will accumulate in the ventricle, increasing the end diastolic volume. Starling's law predicts that this will lead to an increase in both stroke volume and cardiac output. Until a new state is reached in which cardiac output equals venous return again. Because the output from one ventricle is responsible for the venous return to the other side of the heart in the intact circulation, this mechanism will also ensure that the cardiac output from the two ventricles remains equal. For example, if the cardiac output from the left ventricle increases, this will increase

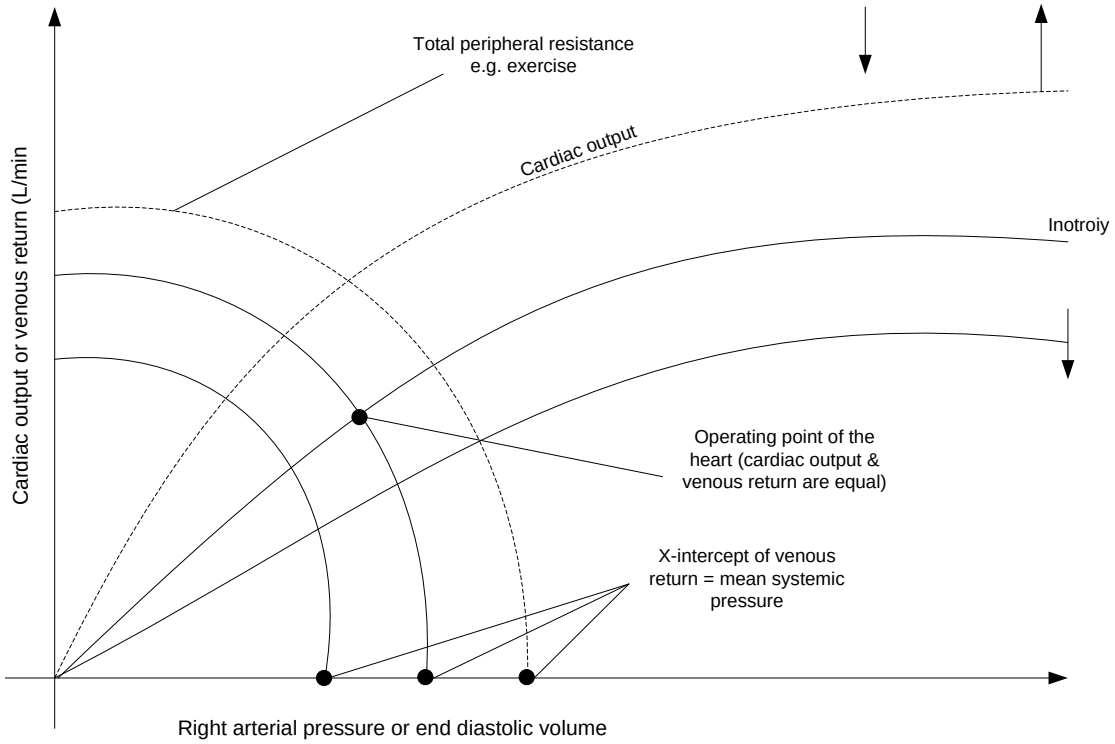

Figure 4. The Frank–Starling law of the heart. The three curves illustrate that shift along the same line indicate a change in preload, while shifts from one line to another indicate a change in after load or contractility.

right venous return and right ventricular output will rise as a consequence.

The above quantitative explanation can be substantiated by a discussion based on Equations(1.11) - (1.14), and on the hypothesis that both hearts work on the ascending part of their ventricular function curves. Equations (1.11) - (1.14) yield

$$\frac{dv_L}{dt} = -Q_L\left(v_L\right) + Q_R\left(v_R\right) - \frac{dv_P}{dt}, \tag{1.15}$$

$$\frac{dv_R}{dt} = Q_L\left(v_L\right) - Q_R\left(v_R\right) - \frac{dv_S}{dt}. \tag{1.16}$$

The blood volume distribution in the cardiovascular system depends on body posture relative to the gravitational field. For example, the average blood volumes in the pulmonary circulation and in the heart in the supine position are larger than in the upright position, because gravity induces redistribution of blood volume in the system (Grodins, 2001). Therefore, in a movement from sitting to supine position, there is a transient behaviour of the system during which the blood is redistributed between the pulmonary and systemic circulation according to Equations (1.15) and(1.16).

A complete analysis of transient cardiovascular phenomenon would require the derivation of additional differential equations to describe the response of the pulmonary and systemic circulation to the perturbation of the system. In Equations (1.15) and (1.16) the circulation response is represented by the time derivatives, $\frac{dv_P}{dt}$ and $\frac{dv_S}{dt}$. These quantities depends on the blood viscosity and on the geometry and elastic properties of the vascular beds, which are constituted by arteries and veins (McGeon, 1996). The number of required equations depends on the number of components of the pulmonary and systemic systems.

If we assume that the vascular beds are rigid, Equations (1.15) and (1.16) can be reduced to:

$$\frac{dv_L}{dt} = -Q_L\left(v_L\right) + Q_R\left(v_R\right), \tag{1.17}$$

$$\frac{dv_R}{dt} = Q_L\left(v_L\right) + Q_R\left(v_R\right). \tag{1.18}$$

These equations contain only physiological quantities

related to the heart, the active element of the system, and are useful in explaining the role of both hearts in the control mechanism. Mathematically, Equations (1.17) and (1.18) represent the core of the Frank – Starling control mechanism. The average cardiac outputs are implicit time functions with time derivatives given by:

$$\frac{dQ_L}{dt} = \left(\frac{dQ_L}{dv_L}\right)\left(\frac{dv_L}{dt}\right) = \left(\frac{dQ_L}{sv_L}\right)\left[Q_R\left(v_R\right) - Q_L\left(v_L\right)\right] \tag{1.19}$$

$$\frac{dQ_R}{dt} = \left(\frac{dQ_R}{dv_R}\right)\left(\frac{dv_R}{dt}\right) = \left(\frac{dQ_R}{sv_R}\right)\left[Q_L\left(v_L\right) - Q_R\left(v_R\right)\right] \tag{1.20}$$

Equations (1.19) and (1.20) yield:

$$\frac{d\left(Q_L - Q_R\right)}{dt} = -\left(Q_L - Q_R\right)\left[\frac{dQ_L}{dv_L} + \frac{dQ_R}{dv_R}\right] = -\left(Q_L - Q_R\right)\varphi/T, \tag{1.21}$$

Where:

$$\left(Q_L - Q_R\right) = T\left[\frac{dQ_L}{dv_L} + \frac{dQ_R}{dv_R}\right] \tag{1.22}$$

If, due to a perturbation of the system, the left cardiac output becomes larger thank the right one, both outputs will vary in time and the difference between them will vary according to Equation (1.21). If both hearts work on the ascending part of the ventricular function curve, the derivatives $\frac{dQ_L}{dv_L}$ and $\frac{dQ_R}{dv_R}$ will be positive and, in this case, Equation (1.21) shows that the difference between left and right cardiac output will decrease in time until the steady state is again restored. The positive value of the φ expresses the fact that the Frank – Starling mechanism is effective in restoring the steady state of the cardiovascular system. Furthermore, for positive values, the larger the value of φ, the faster the steady state is again attained. Therefore, φ, measures the effectiveness of the Frank – Starling mechanism.

If both hearts work on the descending limb of the ventricular function curve, the derivatives $\frac{dQ_L}{dv_L}$ and $\frac{dQ_R}{dv_R}$ will be negative and consequently, dv_L , φ, will also be negative. In this case, Equation (1.21) shows that the difference between left and right cardiac output will increase with time and the Frank–Starling mechanism would be completely exhausted as a control mechanism, a fact expressed by the negative value of φ. Therefore, both hearts cannot work on the descending limb of the ventricular function curve.

In the linear approximation, in which the time variations of $\frac{dQ_L}{dv_L}$ and $\frac{dQ_R}{dv_R}$ are neglected, Equation (1.21) has the solution:

$$Q_L\left(t\right) - Q_R\left(t\right) = \left[Q_L\left(0\right) - Q_R\left(0\right)\right]\exp\left[-\left(\varphi/T\right)t\right],$$

Which, shows that for $\varphi > 0$, the transient duration is inversely proportional to φ.

The above conclusions concerning the Frank–Starling mechanism were deduced without assuming a particular form for the ventricular function. This generality is important because the ventricular function (and φ) varies from person to person, and from moment to moment, depending on the individual's physical conditions. In spite of these variations, the control mechanism is able to maintain the stability of the system.

If we assume, for simplicity, that the left and right ventricular functions have the same mathematical form, we can write $Q_L\left(v_L\right) = Q\left(v_L\right)$ and $Q_R\left(v_R\right) = Q\left(v_R\right)$. Thus, for a steady state, Equation (1.22) reduces to:

$$\varphi\left(v\right) = 2T\left(\frac{dQ}{dv}\right) \tag{1.24}$$

Because of the variability of φ, its numerical value is used to compare the cardiovascular system of different individuals, or of the same person under different physical and health conditions as an example, it is interesting to compare the congestive failing heart with

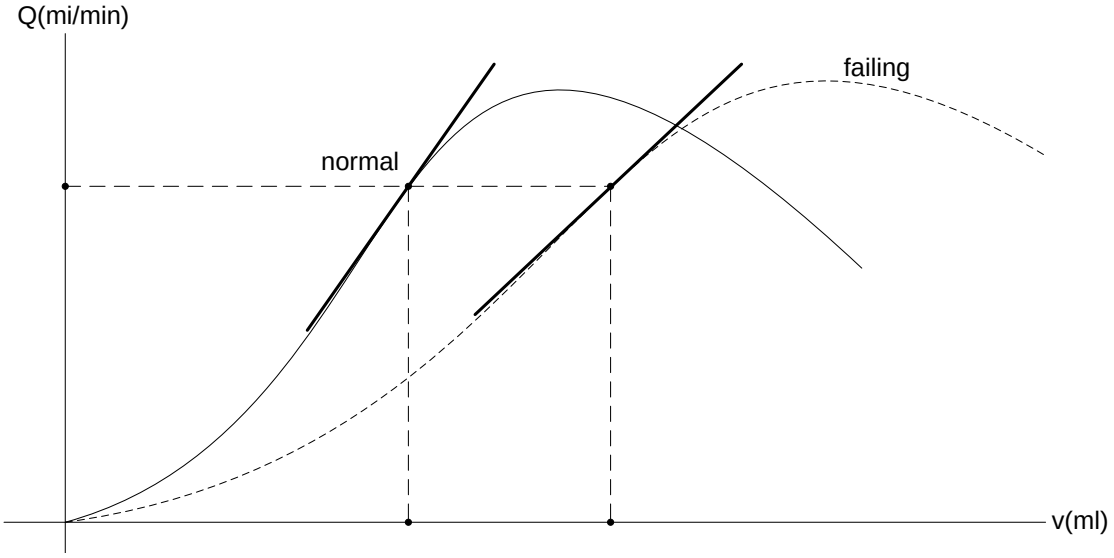

Figure 5. Ventricular function curve for the normal and failing heart.

the normal one. In the former, the cardiac muscle is weaker than the normal one. Research has shown that the ventricular function curve of the failing heart is depressed in comparison to the normal heart curve, and the failing heart works on a relatively flat part of the ventricular function curve. Because, of cardiovascular adaptive abilities, it is possible for cardiac output to be nearly normal, even though the cardiac muscle is severally diseased. However, to maintain normal cardiac output, the heart must dilate.

These observations can be expressed mathematically by saying that $\frac{dQ}{dt}$ for the failing heart is smaller than the corresponding value for the normal heart, and consequently, φ, for the failing heart is small compared to the normal value. Figure 5 shows the ventricular function curve of the failing heart in comparison to the normal curve.

In Equations (1.15) and(1.16), the derivatives $\frac{dv_P}{dt}$ and $\frac{dv_S}{dt}$ represent the response for the pulmonary and systemic circulation to a perturbation of the system and depend on the geometry and elastic properties of the vascular beds and on the blood viscosity. Equations (1.17) and (1.18) were derived by assuming that $\frac{dv_P}{dt} = \frac{dv_S}{dt} = 0$, which neglect the elastic properties of the vascular beds (Grodins, 1999).

The first mathematical description of the complete cardiovascular system was published by Grodins in 1966 where it was reported that the cardiovascular system manifests mechanical self-regulation. His model includes the elastic properties of vascular beds, and satisfies the conditions that $\frac{dQ_L}{dv_L}$ and $\frac{dQ_R}{dv_R}$ must be positive.

The mechanical self-regulation of the cardiovascular system is based on the fact that the left and right hearts are connected in series, and both hearts work on the ascending part of their ventricular function curve.

Blood volume re-distribution

Since the heart operates like two pumps connected in series through the pulmonary and systemic circulations (Figure 2), its optimum for redistribution of blood volume in the system is described below (Figures 6 to 10): Consider a situation in which the subject moves from sitting to supine position. The cardiovascular system will show a transient behaviour during which blood will be redistributed between the systemic and pulmonary circulation (Dwivedi and Dwivedi, 2007; West, 2008). As noted, a complete analysis of the transient behaviour requires the derivation of additional differential equations. Although, Equations (1.15) and (1.16) do not constitute a complete set, interesting results can be derived from them as follows:

$$\frac{d\left(v_L - v_R\right)}{dt} = -2Q_L + 2Q_R - \frac{d\left(v_P - v_S\right)}{dt}, \quad (1.25)$$

If we integrate Equation(1.25), we have:

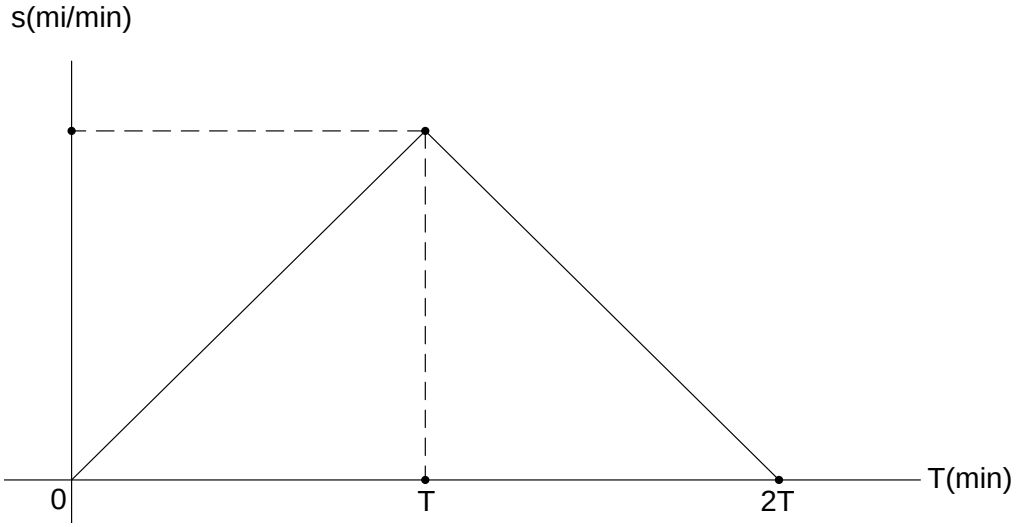

Figure 6. The time function s (t). T is the cardiac period.

$$\int_{t_I}^{t_F}\left(Q_R - Q_L\right)dt = \frac{1}{2}\left[v_P\left(t_F\right) - v_P\left(t_I\right) - v_S\left(t_F\right) + v_S\left(t_I\right)\right]$$

$$= \frac{1}{2}\left[\Delta v_P - \Delta v_S\right] \tag{1.26}$$

Where: t_I and t_F denotes the initial and the final instants of blood volume redistribution respectively (Figures 6 -10). For a movement from sitting to supine position, the integral in Equation (1.26) is positive because the average blood volume in the pulmonary (systemic) circulation in the supine position is larger (smaller) than in the sitting position. Therefore, during the redistribution of blood (Figure 8) between the pulmonary and systemic circulation, the function $Q_R\left(t\right)$ must necessarily be different from $Q_L\left(t\right)$, $Q_R\left(t\right)$ and $Q_L\left(t\right)$ depend on how the subject move between the initial and final position, but the integral $\int_{t_I}^{t_F}\left(Q_R - Q_L\right)dt$ depends only on variation of the volume of blood in the pulmonary and systemic circulation, as shown by Equation (1.26). The average value of the difference between $Q_R\left(t\right)$ and $Q_L\left(t\right)$, during a transient of duration $\Delta = t_F - t_I$ is given by:

$$\left[Q_R - Q_L\right]_{av}\Delta = \frac{1}{2}\left[\Delta v_P - \Delta v_S\right] \tag{1.27}$$

The duration of the transient depends on how quickly or slowly the subject moves from one position to another. The faster the movement, the smaller the value of Δ. Hence, Δ cannot equal zero because as $\Delta \to 0$, $\left[Q_R - Q_L\right]_{av} \to \infty$, which is physiologically impossible. Hence, Δ must satisfy the condition $\Delta \geq \Delta_{\min} > 0$, where $\Delta_{\min}$ is the minimum transient duration, which occurs when $\left[Q_R - Q_L\right]_{av}$ is a maximum. It is interesting to note that this result was derived from the fact that in Equation(1.26), the integral depends only on the distribution of blood corresponding to the initial and final steady states, that is, it does not depend on how the subject moves from one position to another. Thus, for a movement from sitting to supine position, there is a minimum transient duration that depends on the characteristic of the heart vascular beds, as well as on blood viscosity, so that it is a measure of the $\Delta_{\min}$ effectiveness of the cardiovascular system, considered as a whole, in restoring the steady state that was perturbed by the movement of the subject. Additional equations are necessary for a theoretical estimate of $\Delta_{\min}$, but it can be experimentally determined. The arterial blood pressure shows a transient behaviour when the subject moves from sitting to supine position, so that $\Delta_{\min}$ can be experimentally determined by observing this transient behaviour for subject movements at different speeds (Figures 6 to 10).

Because the physics of the cardiovascular system is

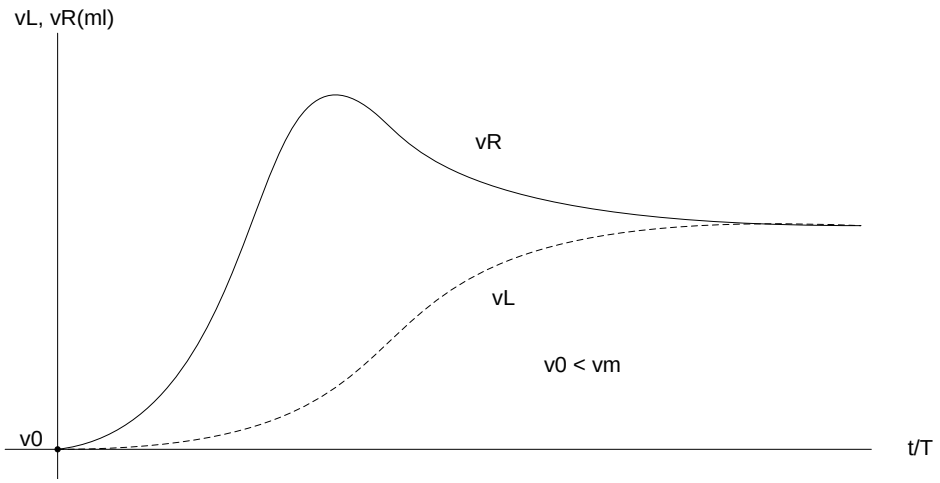

Figure 7. Blood volumes in the left and right heart, v_L and v_R, as time functions, initial volume v_0 smaller than the volume v_m corresponding to the maximum of the ventricular function.

not discussed in physics textbooks, all the idealizations and approximations are clearly stated in this paper, so that it will be useful for teaching one way of doing physics. Due to its great complexity and idealized representation of the cardiovascular system is necessary for deriving differential equations for the blood flow. The idealized representation illustrated in Figure 2 is very simple, but it is sufficient for the derivation of blood flow in the system. It would be interesting to compare the idealized representation in Figure 2 with the real anatomy of the cardiovascular system to stressing the drastic simplification used in our treatment.

The reason for taking the average of Equations (1.4) – (1.7) is to simplify the mathematical discussion, which is much simpler in terms of average quantities. For example, in the steady state, the time derivatives of the instantaneous blood volumes in the left and right heart, $\frac{dv_L^i}{dt}$ and $\frac{dv_R^i}{dt}$, are functions of time, whereas for the average blood we have:

$$\frac{dv_L}{dt} = \frac{dv_R}{dt} = 0$$

The averaging process, which in this case was accomplished by approximation, represents a great simplification of the mathematical description.

Equations (1.17) and (1.18) were derived from Equations (1.15) and (1.16) assuming that $\frac{dv_S}{dt} = \frac{dv_P}{dt} = 0$. This assumption described the limiting case in which the elastic properties of the vascular beds are neglected. The study of "Limiting cases" was considered to be one of the most useful and educational things we can do with any equation. In our treatment, the study of the limiting case made possible an analytical discussion of the Frank–Starling mechanism in which it was not necessary to assume a particular form for the ventricular functions, a generality that is important considering that the ventricular function varies from person to person, and from moment to moment.

It is also noted that the output from one ventricle is responsible for the venous return to the other side of the heart (Mc. Geon's 1996). This statement is expressed mathematically by Equations (1.12) and (1.14), which more precisely show that the venous return $Q_S\left(Q_P\right)$ is related not only to cardiac output $Q_L\left(Q_R\right)$, but also to the time derivative of the volume of blood in the systemic (pulmonary) circulation $\frac{dv_S}{dt}\left(\frac{dv_P}{dt}\right)$. In the steady state, and also in the limit in which vascular beds are assumed to be infinitely rigid, these times derivative are equal to zero, and consequently, $Q_L = Q_S$ and $Q_P = Q_R$. If the systemic venous return Q_S suddenly rises above the right cardiac output Q_R, blood will accumulate in the heart, as can be seen from Equation (1.11) with respect to the relationship between cardiac output and blood volume in the heart, this increase of blood volume in the right heart will lead to an increase in the right cardiac

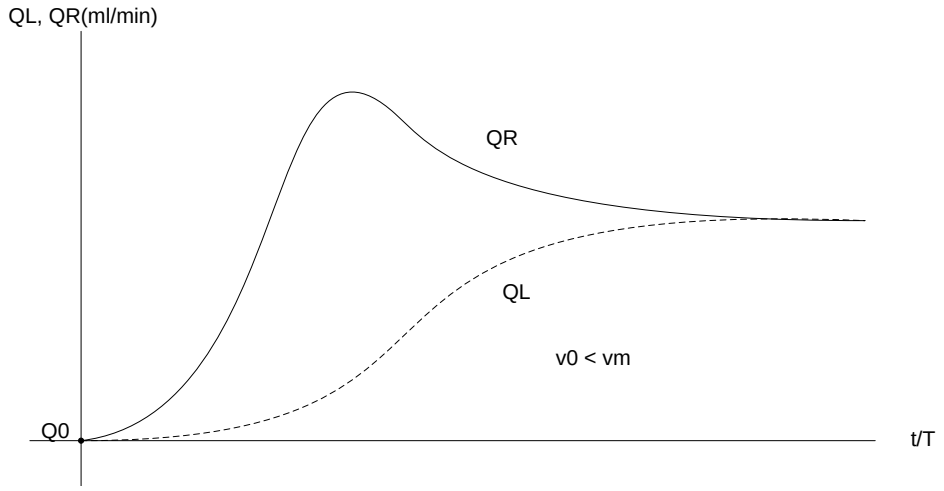

Figure 8. Left and right cardiac outputs Q_L and Q_R , as time functions; $v_0 < v_m$ (initial heart operating on the ascending part of the ventricular function curve).

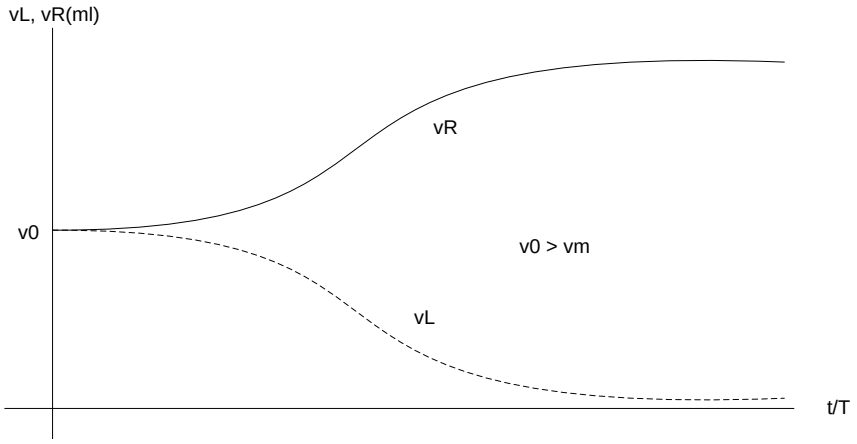

Figure 9. Blood volumes in the left and right heart, v_L and v_R , as time functions, initial volume v_0 larger than the volume v_m corresponding to the maximum of the ventricular function.

output until a new steady state is reached in which the cardiac output Q_R equals the nervous return Q_S again.

The experimental observation that there is a relation expressed by the ventricular function between cardiac output and the volume of blood in the heart is essential for explaining the auto-regulation of the system. Because there is as yet no theory that derives the ventricular function from first principles, this function was included in Equations (1.15) and (1.16) as expressing experimental data. The inclusion of an empirical relation illustrates the formation of a phenomenon of logical theory that contains elements based on experimental observation that are waiting for a more fundamental explanation (Uehara and Sakane, 2002).

Conclusions and recommendations

The present work describes the application of the continuity equation to the rate of blood flow and variation of the volume of blood in different parts of the cardiovascular system in conjunction with physiological observations has provided a set of differential equations that are useful for clarifying the essential points of the Frank–Starling mechanism. The basis hypothesis for

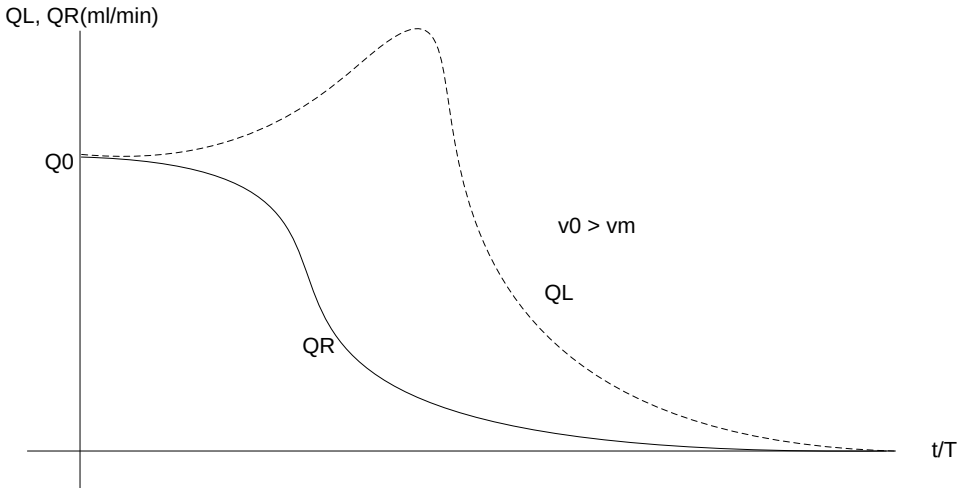

Figure 10. Left and right cardiac outputs Q_L and Q_R , as time functions; $v_0 < v_m$ (initial heart operating on the ascending part of the ventricular function curve)

explaining this mechanism is that the heart operates like two pumps connected in series through pulmonary and systemic circulation (Figure 2). There is a relationship expressed by the ventricular function between cardiac output and blood volume contained in the heart. Both hearts work on the ascending part of their respective ventricular function curves; and the total blood volume in the cardiovascular system is constant. There is need for extension of this work in order to derive more differential equations that will explain different complex models used in cardiovascular system.

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