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CIRCUITS, WAVES, AND PIPES-A BIOPHYSICAL PERSPECTIVE FOR IMPARTING CONCEPTS IN CARDIOVASCULAR AND PULMONARY PHYSIOLOGY TO UNDERGRADUATE MEDICAL STUDENTS

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ABSTRACT Imparting concepts in flow dynamics of cardiovascular and respiratory physiology can be intimidating to the beginners in medical schools. We have tried to simplify the approach in effective transfer of these concepts using biophysical concepts and principles pertaining to functioning of these systems which help in easy understanding of the two vital systems in human body. Beginners in medical education find it easy to relate to these concepts.

KEYWORDS : circuits and pipes, biophysical concepts, haemodynamics, cardiovascular, respiratory

INTRODUCTION

Nature offers great symmetry and basic physical laws that determine the pressure, wave characteristics, and flow in elastic pipes. These laws can be used to gain insight into the functions of the cardiovascular system.

Conventional teaching in concepts of haemodynamics fails to establish a proper link between elementary physical laws and the intricacies of the cardiovascular system. Concepts such as Windkessel models, elastic waves, and flow dynamics in elastic tubes should be introduced in first-year physiology lectures to make it easier for beginner students to draw parallels between various mechanical and electrical analogies. This will help them understand concepts, such as arterial waveforms and ventilatory curves.

Once these physiological principles are understood through these physical laws, understanding the pathological processes and basis of pharmacological interventions in various cardiovascular and pulmonary diseases is also simplified. In this article, various biophysical concepts that are important to understand and gain an intuitive understanding of the functioning of the cardiovascular system are discussed.

Biophysical modelling of the lung

To understand the biophysics of spontaneous ventilation, consider a mechanical analogy with an open pipe and elastic balloon connected to one end of the pipe. If one starts pumping/pushing air at a constant force into the aforementioned system, the balloon starts expanding. As the elastic balloon progressively expands, the rate of influx of air into the balloon progressively decreases as the balloon offers significant resistance to the incoming mass of air in the form of an internal recoil force or tension. 1 Thus, the pressure difference, which is the driving air flow, decreases exponentially, and a time occurs when the gradient becomes zero and there is no further flow into the system. At this stage, the applied pressure counteracts the collapsing tendency or tension in the balloon to prevent deflation or the discharge of air from the elastic structure.

As inspiration begins, the initial rise in the air current is dependent on the value of the airway resistance. However, as the first bit of air reaches the alveoli and stretches their elastic wall, a radial passive recoil force is generated, and the pressure in the alveoli begins to rise. The rising tension force impedes the further admission of the air volume and slows the rise of air current in the circuit.

When inspiration ceases, the external force that was maintaining the alveoli in a distended/expanded state falls to zero, allowing the alveoli to discharge. The rate of outflow during expiration depends on the collapsing tendency or the recoil force of the alveoli and the expiratory resistance of the airways. In pathological states such as obstructive diseases there is an anatomical or functional obstruction, which leads to a decreased discharge velocity and incomplete emptying of the air, leading to hyperinflation and it's subsequent delirious consequences.2

Obstructive and restrictive lung diseases

Modelling the lung in the biophysical realm can help us understand the physiology and pathology of common lung diseases. Pulmonary diseases can be classified as obstructive and restrictive. In the following, we will incorporate our knowledge of mechanical analogies to understand the physical basis of lung pathologies.

Restrictive disease is characterized by a decreased compliance/increased stiffness of the alveoli, which allows the production of a greater recoil force at lower tidal volumes, thus leading to a greater and steeper increase in inspiratory pressures, providing impedance to the further entry of air. This leads to an increase in the pressure required to pump the same volume of air as a normal lung. The decreased compliance of the system leads to a decrease in the time constant of the system and allows for the rapid discharge of the air volume once inspiration ends.

These biophysical alterations lead to a decrease in the tidal volume and an increase in the alveolar discharge rate during expiration, producing characteristic increases in the forced expiratory volume in the first second (FEV1) and FEV1/forced expiratory vital capacity. Figure 1

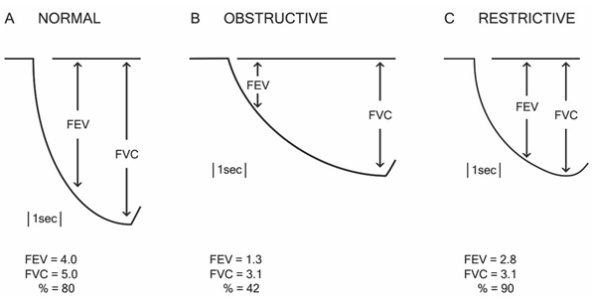

Figure 1. Restrictive vs Obstructive Disease. Restrictive diseases are characterised by increased discharge velocity of air volume during expiration, leading to a rise in FEV1/FVC ratio. On the other hand, obstructive diseases are characterised by slower emptying of alveolar units during expiration, leading to a fall in FEV1/FVC ratio.

In contrast, obstructive diseases are characterized by an increased airway resistance, particularly during expiration (dynamic airway collapse). This leads to an increased work of breathing during inspiration to overcome the increased airway resistance. During expiration, the time constant of the circuit increases, leading to a decreased outflow rate and

slower discharge velocity. Therefore, owing to the structural or functional airway narrowing, the alveoli do not discharge rapidly, and the next inspiratory cycle begins without the complete emptying of the alveoli. This leads to breath stacking, which is a characteristic feature of obstructive diseases, such as asthma and chronic obstructive pulmonary disease (COPD). The incomplete emptying of the alveoli and dynamic hyperinflation leads to detrimental effects on the mechanical and ventilatory functions of the lung.

With the progressive air volume being stored in the alveoli, the positive end expiratory pressure (PEEP) rises, leading to an increasing ventilation-perfusion mismatch. The increased alveolar volume leads to the compression of the surrounding pulmonary capillaries, thus creating a region of high ventilation and low perfusion.

The rising auto-PEEP and progressive breath stacking lead to lung hyperinflation and a fall in the mechanical advantage of the inspiratory muscles, decreasing their capacity to perform positive work.3

Furthermore, the already fatigued respiratory muscles must counteract an increased elastic load due to alveolar overdistension.

The increased respiratory muscle load and lowered respiratory muscle work capacity lead to a fall in the tidal volume, reducing the alveolar ventilation and thus creating a low ventilation-perfusion lung unit. These changes lead to a rise in the ventilation/perfusion ratio (V/Q) scatter and reduce the efficiency of the gaseous exchange in obstructive lung diseases.

Modelling the cardiovascular system as a parallel RC circuit

To understand a parallel RC circuit, we use a mechanical analogy in which the elastic chamber has two diametrically opposite openings; to one of the openings, we will connect a pipe, and the air/fluid mass will be fed from the opposite opening. As we push air, it initially attempts to expand the elastic chamber rather than exit through the pipe. The expansion generates an elastic recoil force and increases the intraluminal pressure. With progressive inflation, the tension in the wall of the chamber will increase and redirect the current through the resistive pipe.

Thus, we can imagine this setup as a parallel RC circuit to which a pulsed current is applied. As the current begins to flow, it preferentially moves to the capacitive limb because it offers negligible resistance. However, with the first bit of charge, the capacitor develops some voltage and a repulsive electric field, which provides some opposition to the further flow of current into the capacitive limb. The current is split between the resistor and capacitor according to the instantaneous impedance of each limb.4 Figure 2

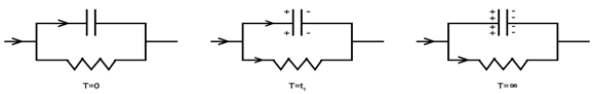

Figure 2—A parallel RC Circuit slows the rise of current through the resistor and ensures a gradual and steady increase in resistive current. As the capacitor gets charged, current is diverted to the resistor. This is analogous to the buffering action of the aorta, which slows the rise of current through the resistor, till enough upstream pressure is generated to overcome the arteriolar resistance.

This intuitive understanding of parallel RC circuits can be compared to the functioning of the cardiovascular system, wherein the aorta and total peripheral resistance (arterioles) are functionally parallel to each other. Upon the injection of blood volume into the system, some current is stored in the aorta and causes the elastic aortic walls to expand and develop a passive recoil force, thereby allowing the blood to experience a circumferential passive squeeze, enabling an increase in pressure and redirecting the current towards the arterioles.

The pumping force of the heart at one end and the resistance at the other end maintain the aorta in a distended state with significant tension in its walls. As soon as systole ends, the aorta begins discharging its current. The rate is determined by the time constant or product of the resistance and capacitance[5].

This model helps elucidate the influence of total peripheral resistance on the blood pressure. If the system is equilibrated at a certain input current and a particular resistance and we suddenly increase the resistance, then the resistive current will abruptly decrease because the same pressure will not allow the same current to be pumped across the new, greater resistance. Although the resistive current has decreased, the total input current remains the same; thus, some current is now split between the resistive and capacitive elements until equilibrium is reached again, allowing the entire current to flow through the total peripheral resistance, albeit at a greater aortic pressure.

The consequence of changing the vascular resistance is a transient change in the capacitive current, which causes a parallel change in the aortic pressure. In the cardiovascular system, the same current rate is not always achieved upon increasing the resistance, as the increase in capacitive current is insufficient to sufficiently increase the aortic pressure to cause the same outflow as before. This mechanism helps reduce blood flow through the arterioles, as per the body's requirements.

A seemingly fit analogy can be found if we consider a pipe connected to a fluid source; if the opening of the pipe is partially occluded, the exiting fluid velocity increases due to the reduction in the area, but the increase in velocity might not be proportional to the decrease in the area (equation of continuity) because the mass or volume of the fluid exiting per minute can be reduced, owing to the increased resistance of the outflow orifice. If sufficient fluid accumulates upstream, it will cause a sufficient increase in pressure to maintain the same volumetric flow rate at a higher value of resistance, and the increase in velocity can now be estimated by applying the continuity equation[6].

The parallel RC model allows for a certain degree of delay in the rise and fall of current flowing through the resistor. The capacitive current at the beginning allows for the splitting of the input current and hence ensures a slow increase in the current through the resistor. When the source is turned off, the capacitor discharges and ensures that the resistive current decreases slowly to zero, with some delay.

The qualitative understanding obtained from this description aids in the development of an intuitive sense of capacitive and resistive currents and how the functional arrangement of the arterial tree can be compared to various electrical and mechanical models.

In reality, this model is used at low frequencies of cardiac contraction, where the effect of the pulse wave transmission can be neglected and the increase and decrease in pressure across the arterial tree are assumed to occur simultaneously. This model lacks the effects of pulse wave transmission, such as reflection, transmission, and the pulse wave velocity[7].

Vascular compliance and energy optimization

To optimize cardiac energy, the increase in the pressure of

ventricular ejection should be slow and gradual. Compliant arteries store significant blood volume without drastic changes in the vascular pressure. This allows perfusion to be maintained during the entire cardiac cycle[8].

In the case of rigid vessels with almost negligible compliance, the circuit becomes purely resistive, which leads to a sharp and sudden increase in the pressure upon the admission of the blood volume. This arrangement forces the heart to pump against a greater systolic pressure and causes the complete cessation of flow during diastole. Therefore, the efficiency of the cardiac pump is significantly reduced.

In contrast, a system of compliant vessels is adapted to store greater degrees of capacitive current while ensuring a gradual and smooth increase in the systolic pressure. The stored volume is released when the vessel recoils during diastole, which allows for an increased diastolic perfusion, thereby increasing the cardiac efficiency.

A stiffer segment also means that blood is rapidly shunted from one segment to another, thus causing an increase in the pulse wave velocity. The increased pulse wave velocity leads to the early arrival of the reflected wave from the periphery, causing an increase in the afterload faced by the left ventricle. Reflection, Resonance and Pulse Wave Velocity—Distributed arterial models

We will now imagine a distributed arterial model, which analyses the characteristics of each arterial segment as an independent oscillator, no different than a rope which can be broken down into a series of infinite coupled oscillators.

Unlike, the lumped models such as the two element windkessel model, this model helps to understand phenomenon such as reflection, resonance and signal/pulse wave velocity.

Reflection in the arterial system occurs, when blood transitions from one arterial segment with a certain characteristic stiffness/impedance to another segment with different characteristic stiffness/impedance.

This is because of the difference in the admittance of each vascular segment to incoming wave of pressure energy.

Pressure disturbances or signals reflected from such sites of impedance mismatch allow, signalling downstream information from the peripheral sites to the heart and modifying the vascular load on the heart.

Resonance In The Arterial System

The forward and reflected pressure and flow waves interfere with each other to produce net waves. At certain frequencies, however, the crests of the incident and reflected waves perfectly line up, creating a standing wave pattern, and then the system reaches resonance.

At these resonant frequencies, the pressure and current nodes and antinodes form at sites throughout the arterial tree. The ratio of the pressure and flow harmonics at the mouth of the aorta provides the input impedance or overall resistance to the phasic ejection of blood encountered by the heart.

An infinitely long arterial tree system demonstrates only the characteristic impedance, which is a property of the compliance and the inertance of the arterial tree itself[9]. However, in reality, the source(heart) sees the characteristic impedance for a very short duration, until the traveling signals encounter a site of impedance mismatch or reflection. As some of the pressure and current waves are reflected back from these sites, they alter the load at the source, and the heart now encounters an input impedance that considers the global effect of reflection and the load mismatch on the heart.

Pulse wave velocity

The arterial tree can be considered to consist of an infinite number of coupled oscillators that allow for the propagation and transmission of a pressure-volume wave, shunting blood from one segment to another to create space for the newly ejected blood volume. The pulse signal is generated by the linear superposition of various harmonic frequencies and signals the distal sites of the cardiac systole/ejection of blood volume[10].

As soon as the blood is pumped in, the proximal part expands and develops tension, allowing for an increase in pressure and ensuring an adequate pressure signal to overcome the inertia of the pre-existing blood volume. This leads to a shunting phenomenon, and the blood rapidly moves from one segment to another, liberating the arterial tree of the previously occupied blood volume.

The rate at which this push signal or pressure-volume wave propagates is known as the pulse-wave velocity. Understanding the rate at which an elastic wave propagates provides insight into the stiffness and elasticity of the medium through which the wave propagates. Similarly, pulse wave velocity helps us to understand the mechanical properties of arterial trees.

As blood is pumped into the most proximal aortic segment, it takes finite time and volume to stretch the elastic vessel wall sufficiently to generate a sufficient elastic recoil force and increase the local pressure to generate the push signal for mobilization of the adjacent segment. If the vessel is less distensible, the desired push signal can be generated rapidly with less delay, and the pre-existing column of blood can be quickly mobilized to liberate space for the incoming blood volume[11].

Therefore, pulse wave velocity can be used to assess the stiffness or rigidity of an arterial segment. The signal velocity should not be confused with the blood-flow velocity. Flow velocity is the velocity at which the blood mass moves from one segment to another while receiving the push signal. Subsequently, the pulse wave velocity determines the speed at which the push signal rises in a segment and the rate at which the fluid mass reacts to this push signal.

The consequence of the finite time taken by the pulse wave to travel and reach a distal site is that it enables the formation of an oscillating pressure gradient between the proximal and distal sites, which exerts an oscillatory force on the blood column, leading to its acceleration and deceleration.

To summarise, the distributed model of arterial haemodynamics considers the individual capacitance of each segment along with the inertia of blood in these segments. In this system, we consider the transmission of a pressure volume wave through the arterial tree and effects, such as the pulse wave velocity, impedance mismatch, reflection, and formation of standing waves, at resonant frequencies.

Elastic tube--negotiator for flow limitation:

In an elastic tube, the flow generated is not only dependent on the pressure gradient but also on the elasticity of the vessel wall. Several theories have been proposed to explain the flow limitation in elastic tubes, such as the waterfall principle, dynamic compression, and vessel wall oscillations. Herein, we will examine these concepts in detail.

Waterfall Principle:

Contrary to an intuitive understanding, as provided by Ohm's law, an increase in the pressure gradient (either by increasing

the upstream pressure or lowering the downstream pressure) will lead to an increase in flow. However, this is not the case for elastic tubes or vessels. There is a point after which the decrease in pressure downstream does not lead to a further increase in the flow rate. This is because any downstream disturbance in pressure needs to travel via the medium to the mouth of the pipe. When the rate of fluid flow becomes equal to the rate at which the signal travels upstream, downstream perturbations will not reach upstream to influence/ modulate the flow, thus limiting any further increase in the flow. The situation can be compared to a man walking on a travelator in the direction opposite to the travelator's movement. As soon as the velocity of the walking man and travelator become equal, albeit in opposite directions, the man appears to be stationary with respect to an external observer. In a similar way, when the fluid velocity matches the signal velocity, in opposing directions, the relative velocity of the pressure disturbance becomes zero, and the signal fails to further influence/ moderate the flow.

Flow Limitation due to Vessel Wall Oscillations--

If the elastic wall is considered an underdamped oscillator, the volumetric flow rate will depend on the time of a single oscillation and the amplitude of the oscillation. A stage occurs when the amplitude becomes maximum and the opposite vessel walls approximate, which leads to flow limitation, as the amplitude of the oscillation cannot increase any further.

Dynamic airway collapse:

As air flows in the respiratory bronchioles or conduit pipes, some of its pressure energy is lost in the form of viscous losses. In the case of an obstruction, these viscous losses are enhanced, which leads to a decrease in the transmural pressure, especially during forced expiration, and leads to collapse of the proximal airways.

At such sites of local constrictions, there is an interconversion of the pressure energy to kinetic energy according to the Bernoulli's theorem, and this further leads to a decrease in the pressure and lowers the transmural pressure, allowing the pleural pressure to gain over the intraluminal pressure and facilitating the further collapse of such airways/elastic conduits[12]. The collapse of airways, either due to increased viscous losses or floppy airways, leads to an obliteration of the airway lumen, thus creating a choke point, which limits further increases in the flow rate. Any further rise in the fluid current, will exaggerate viscous losses(Ohm's Law) leading to further obliteration of the airways and causing a proportionate rise in the resistance, thus preventing any further rise in fluid current.

DISCUSSION

Biophysics is at the core of understanding cardiopulmonary physiology. In this article, we incorporated various mechanical and electrical models to develop a physical approach to physiology for undergraduate students.

We modelled the cardiovascular system as a parallel RC model for certain frequencies and as a general distributed or transmission line model, which allowed us to understand phenomena such as the pulse wave transmission, velocity, and signal reflection. Further, we delved into the workings of the lung and compared it to an RC series circuit and alterations in the compliance or resistance of lung elements, leading to the findings that are clinically seen in these conditions.

The abovementioned analogies and concepts were discussed with first-year undergraduate medical students as part of regular physiology lectures. The mechanical and electrical models simulating the cardiopulmonary system helped bridge the gap between the basic sciences and physiological principles. This approach received a good response to the traditional physiology teaching methodology. Hence, these basic models can serve as physical tools to elaborate on the physiology and pathology of the cardiac and pulmonary circuits.

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