

Blood Flow Modelling to Improve Cardiovascular Diagnostics: a Preliminary Review of 1-D Modelling

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Abstract

Cardiovascular diseases issue an enormous threat to the health and general wellbeing of the population, therefore multidisciplinary knowledge of the cardiovascular system and its mechanics has become a necessity. Due to the lack of wide scale experimental studies, the limitations associated with it and the immense advances in computational technology, in recent years, numerical modelling of the cardiovascular system has gained popularity as a viable alternative. One-dimensional models as compared to higher dimensional models provide a feasible and efficient means to study the dynamics of pulse wave propagation in order to increase the comprehension of circulatory physiology. The aim of this paper is to provide an overall review of the types, solution methods, treatment of boundary conditions, applications and advantages as well as disadvantages of one-dimensional models of the human arterial network.

Keywords: Arterial network; Blood flow modelling; Computational fluid dynamics; Numerical modelling; One-dimensional model.

1. Introduction

As per the World Health Organization, cardiovascular diseases are the leading cause of death globally. The numbers speak for themselves; Hypertension is now identified as the most important single cause of mortality worldwide [1]. By 2025 it is predicted that 1.56 billion people will have hypertension [2]. 30% of all deaths annually are due to cardiovascular diseases, the actual number of deaths lies somewhere in between 17 to 18 million. This percentage is expected to increase in the future due to the lifestyle of the modern age. Therefore, the research in cardiovascular systems in multiple fields is necessary; consequently, it is a very active field of research in today's world.

An important research area in this field is the propagation of the pulse wave in the arterial network and how this propagation is affected by various factors ranging from the tapering of arteries to the reflections caused to the flow due to branching of the arteries. However, in-vivo and in-vitro studies to examine these factors are difficult, time consuming and costly to carry out. Therefore, numerical modelling offers an alternative to experimental studies as a non-invasive and feasible tool.

Over the last few decades, one-dimensional mathematical modelling has gained popularity as a very useful tool to understand some of the effects these factors have on the propagation of blood. [3]–[26]. In the one-dimensional model, only the axial direction of the vessels is simulated. In most studies [4], [5], [7], [12], [15], [26]–[28], large arteries of the systemic circulation are solely simulated as the assumptions of blood propagation models are more applicable to large arteries. Additionally, the material properties and the geometric complexity of the smaller vessels are difficult to measure; hence, large arteries are simulated and truncated through various methods.

This paper aims to provide a condensed and general review of one-dimensional modelling of the arterial network by summarizing

existing research. A brief description of the cardiovascular system and how it can be represented as a one-dimensional model will be provided. This paper also examines the various existing one-dimensional models, their differences, the methods with which their governing equations are discretized and solved as well as the treatment of boundary conditions in these models.

2. Brief Historical Review

Although not a prerequisite to understand the underlying physics of blood propagation, the knowledge of its historical background does provide insight into how the concept of blood propagation has evolved over a period that spans from the Common Era to the present day.

The first known writing which corrected some of the wrong understandings of blood propagation of the time came from a Roman Physician, Galen. (129-210 AD) He found that the arteries contained blood, rather than air which was the common belief at the time. Additionally, he believed that there were two systems at work in the body, a venous system that provides nutrition and the arterial system that was the source of heat for the body [29], [30]. Like Greek physician Erasistratus (304-250 BC), Galen falsely believed the blood originated in the Liver and was consumed by the organs instead of circulating periodically. He also put forth the inaccurate idea that the heart has two chambers.

Most of these beliefs were amazingly unquestioned for the next 1500 years. The first physician to actually oppose these beliefs was Ibn An-Nafis (1210-1288 AD) who was also responsible for the first ever description of pulmonary circulation [31]. Leonardo Da Vinci (1452-1519 AD) took great interest in human anatomy and produced numerous drawings of the visible anatomical features. In drawings of the heart, he used his knowledge of engineering to understand how the heart functions and correctly described the closure mechanism of the Aortic valve. Not long after, Andre-

as Vesalius (1514-1564) wrote one of the most influential books on human anatomy, "De Humani Corporis Fabrica" (On the Fabric of the Human Body) which questioned Galen's universally accepted views.

Gradually, Galen's views started becoming less popular until William Harvey (1578-1657) completely deconstructed the incorrect views of blood circulation in his book "De motu Cordis et Sanguinis in Animalibus" (Movement of the heart and blood in Animals, an anatomical essay) [32]. Harvey deduced that the blood flows from the heart to the rest of the body and instead of the organs consuming the blood; the blood goes back to the heart. He put forth the idea that the pulsatile behavior of blood flow through the arterial network was due to the contraction of the heart. He also realized that the variations of the pulsation in an unhealthy body was due to the anomalies in the function of the heart and blood vessels which directly leads to the establishment of the effect wave reflection has on the arterial pulse. Harvey was limited by the technology of his time, which did not allow him to conclusively discover capillaries. Nevertheless, he still deduced the existence of capillaries until Marcello Malpighi (1628-1694), Jacob van Swammerdam (1637-1680) and Anthony van Leeuwenhoek (1632-1723) finally discovered them. They used a microscope to describe the capillary networks that connect the arterioles and venules and explained the shape of the red blood cells [30]. Actual measurement of pressure in the circulation system started with experiments of Reverend Stephen Hales (1677-1761) who is often credited with the first ever blood pressure measurement. Hales compared the arterial system and the heart to a medieval fire cart. In this comparison, the air-filled chamber is analogous to the compliance of the arteries and the fire hose nozzle represents the peripheral resistance. This idea came to be known as the Windkessel effect, illustrated in Fig. 1.

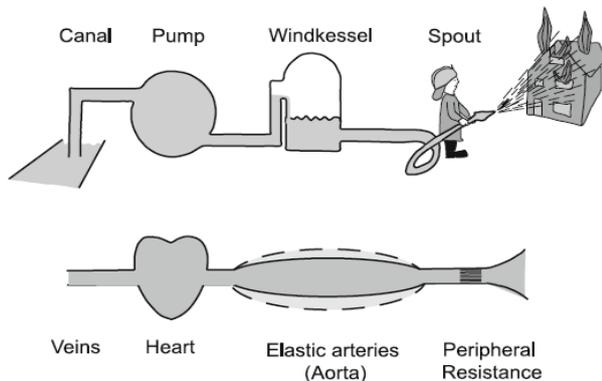


Fig. 1: The Windkessel concept. Large arteries are analogous to the air chamber (act as the Windkessel) [139]

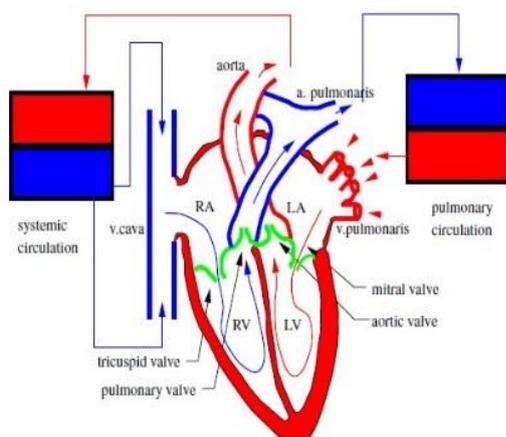


Fig. 2: Schematic representation of the heart and the circulatory system [44]

Through his experiments, Hales showed that that the greatest resistance to blood flow came from arterioles and capillaries [33]. A major contribution was made by Leonhart Euler (1707-1783), who derived the general equations of mass and momentum conservation of an inviscid fluid. Euler was the first one to apply these equations to blood flow but failed to find a solution to these equations because he did not identify the wave like nature of blood flow [34]. Euler's close friend, Daniel Bernoulli (1700-1782), although known for being a prominent mathematician, used his multidisciplinary knowledge to come up with the Bernoulli equation. This equation directly led to the conversion of pressure to potential and kinetic energy. [35] The first person to describe the wave like nature of blood flow scientifically was Thomas Young. (1773-1829) Young is often described as "The last man who knew everything" [36]. He contributed immensely to the study of elasticity, with the modulus of elasticity (Young's modulus) being a direct consequence of his work. His work in hemodynamics led him to propose a relationship between propagation velocity of the arterial pulse and elastic properties of the vessel walls [37]. Moens [38] and Korteweg [39] later formally formulated an equation that related propagation velocity and Young's modulus. The Moens Korteweg equation combined with the one-dimensional Navier-Stokes equations, which resemble the equations derived by Euler, form a mathematical model of pulse wave propagation. Bernhard Riemann (1826-1866) introduced a method in 1860, [40] that was vital in solving this mathematical model. The method is known as the method of characteristics, which finds characteristics, or curves along which the partial differential equation is reduced to an ordinary differential equation.

Rereferring back to Euler's model, the non-linear equations he proposed become analogous to electrical transmission line equations once they are linearized. As a result, several studies were conducted in the frequency domain [41], [42]. Noordergraaf and Westerhof [43] constructed the model for the large systemic human arteries, which, since the advent of computers have been improved upon by various researchers such as Avolio [3], Olufsen et al [5], Sherwin et al [7], and Stergiopulos [4] among others.

3. The Cardiovascular System

At its core, the cardiovascular system consists of a cardiac pump (the heart) and circulatory network (blood vessels). This system is responsible for the transport of blood among the several organs of the mammalian body. This convective transport of blood has two main purposes; the first is to allow for the diffusive transport of oxygen and other nutrients to the tissues and the second is to remove carbon dioxide and harmful waste products from the tissues. These waste products are an end result of cell metabolism. Due to the large diffusional resistance, this interchange of nutrients would not be possible without the convective transport [44]. The cardiovascular system or the circulatory system can be further divided into two systems; namely the systemic circulation and pulmonary circulation, with the heart being at the helm of both these systems. The systemic circulation starts at the left ventricle and ends at the right atrium, while the pulmonary circulation originates at the right ventricle and ends at the left atrium. To fully comprehend these systems, it is first necessary to understand the basic functioning of the heart.

3.1. The Heart

The heart, essentially, consists of two synchronized pumps that are parallel to each other (the left and the right heart). Both of them comprise of two chambers each; the upper ones are called the atria (right and left atrium) while the lower chambers are called the ventricles (right and left ventricle). At the end of each chamber, there is a valve. These valves regulate blood flow and ensure that the blood only flows in one direction [45]. All four chambers and their corresponding valves can be seen in Fig. 2.

Blood enters the right atrium (RA) via the Venae Cavae. This is the point of entry of the blood into the heart. The blood that enters the right atrium is deoxygenated blood, this is the blood that has transported its oxygen to the organs and is returning to the heart with the carbon dioxide it removed from these organs. From the right atrium, the blood goes into the right ventricle (RV) and from this point on, the pulmonary circulation begins. The objective of the pulmonary circulation is to transport deoxygenated blood to the lung tissues, where a gaseous exchange takes place; the blood picks up oxygen and the lungs remove the carbon dioxide from the blood. This gaseous exchange is known as respiration. Once the blood is oxygenated, it is transported to the left atrium (LA) via the pulmonary veins. The oxygenated blood is now ready to be transported from the left atrium to the rest of the body thus concluding the pulmonary circulation.

The blood flows from the left atrium to the left ventricle (LV) and at this point, the systemic circulation is put into motion. The blood flows from the left ventricle into the aorta and the rest of the body. The objective of the systemic circulation is to transport oxygen rich blood to all the organs and tissues. When the blood reaches an organ, it exchanges oxygen with carbon dioxide. Once the gaseous exchange with all the organs is complete, the deoxygenated blood goes back in the right atrium and the whole process starts again. This concludes one cardiac cycle or one cycle of systemic and pulmonary circulation.

Fig. 3 is a diagrammatic representation of the route taken by the blood in one cardiac cycle.

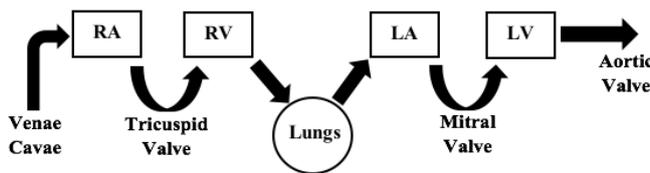


Fig. 3: Block diagram illustration of the route taken by the blood

and diastole. When the blood is in the left atrium, the heart muscles contract causing the blood to flow from the left atrium to the left ventricle. Once the blood flows into the left ventricle, the valve between the left atrium and ventricle, the mitral valve, closes so that none of the blood flows back into the left atrium. Consequently, the pressure in the left ventricle increases until a certain pressure level called the aortic pressure is reached. At this pressure, the valve between the left ventricle and ascending aorta, the aortic valve, opens and a large amount of blood is ejected into the aorta at a high flow rate. This is known as the systolic phase or simply systole.

When the pressure in the left ventricle falls below the aortic pressure, the aortic valve shuts and remains shut until the next cardiac cycle. This is known as the diastolic phase or simply diastole. During diastole, the heart muscles relax and the blood moves from the atrium into the ventricular cavity.

The systole and diastole alternate and repeat periodically. This is what produces the “lub” “dub” sounds. The lub being systole and dub being diastole. It is the periodic repetition of a high outflow and no outflow which in turn, leads to flow and pressure pulsations in the arteries [46]. The Systolic and Diastolic pressure are the measurements taken in a routine blood pressure checkup. This cardiac cycle is better presented with the aid of Fig. 4.

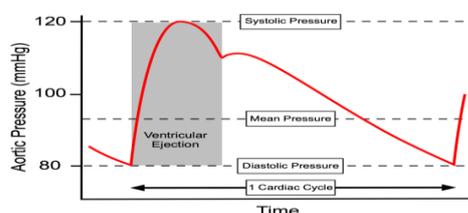


Fig. 4: A cardiac cycle representing the Systolic, Diastolic and Mean Pressures in the Aorta [49]

Diastole takes approximately twice the time as systole. In a healthy human being, the cardiac cycle lasts about 0.8s, which equates to 75 beats per minute. In each stroke, the left ventricle ejects approximately 70ml of blood into the ascending aorta [47]. This is known as the stroke volume (SV).

Based on these values, we can define two important terms now; the heart rate (HR), which is the number of times the heart beats per minute and the cardiac output (CO), which is the amount of blood pumped by the heart per minute. We have already established that the heart rate is 75 beats/min which yields a cardiac output of 5 l/min ($CO=SV \times HR$) [47].

3.2. The Venous System

The organization of systemic and pulmonary circulation is very similar, in that they both have an arterial part (arteries) and a venous part (veins). The arteries usually take oxygenated blood from the heart to the tissues while the veins take deoxygenated blood away from the tissues and into the heart.

Branches and networks of vessels constitute and aid the cardiovascular system. The size of these vessels and their geometrical properties vary [48]. In order to understand how the nutrient exchange between the blood and the organ/tissue takes place, it is necessary to understand the hierarchy of these blood vessels. These vessels can be divided into the arterial system, capillary system and venous system. Table 1 summarizes the functions of the different types of systems and the vessels in them.

Table 1: Different Vessels and their functions

System	Vessels	Function
Arterial system	Arteries	Distribute blood throughout body and maintain blood pressure between heart-beats
	Arterioles	Transport blood to capillary beds
Capillary system	Capillaries	Diffuse oxygen and nutrients to cells
	Venules	Collect deoxygenated blood from capillaries
Venous system	Veins	Return deoxygenated blood to the heart

For the exchange of nutrients and oxygen to occur, the blood first goes from arteries to arterioles and from there on into a network of capillaries that cover all organs/tissues. The arterioles act as control valves between the arteries and capillaries. The purpose of these control valves is to regulate the amount of blood flowing into the capillaries in response to the needs of a certain organ [46]. The permeability of the capillary walls is enough to let small molecules diffuse across. Through the walls of the capillaries the oxygen, hormones and nutrients from the blood are diffused to the interstitial fluid of the cells of the organ while removing carbon dioxide and other waste products of cell metabolism. The blood that now contains the waste products and carbon dioxide is collected by the venules from the capillaries and is transported back to the heart via larger veins. The larger veins take the deoxygenated blood to the heart and the pulmonary circulation begins. Fig. 5 shows how the exchange of nutrients takes place with a certain tissue.

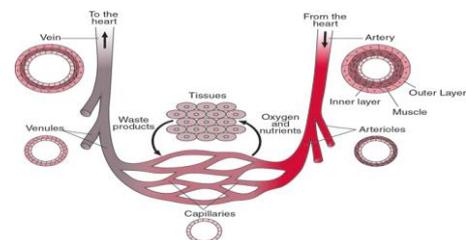


Fig. 5: The exchange of nutrients between the capillaries and the tissues. The artery brings oxygen and nutrient rich blood and to the capillaries. The vein takes waste products from the capillaries and transports them back to the heart [49]

It is abundantly clear that all the vessels need to be well adapted in order to carry out their function. This is the reason why the vessels in the arterial, capillary and venous system have varying geometrical and mechanical properties. The variation in these properties can be seen in Fig. 6.

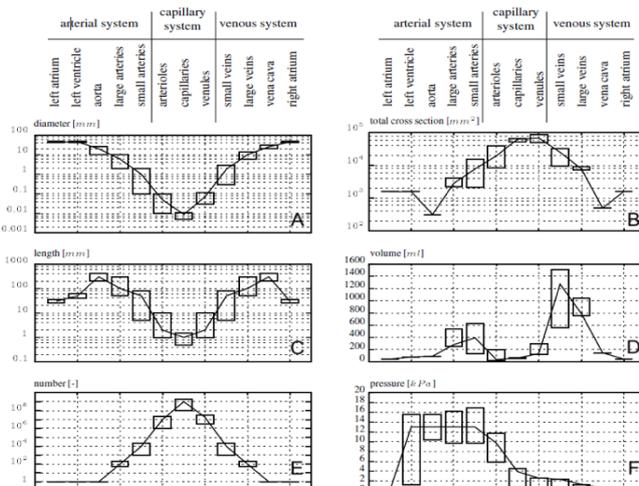


Fig. 6: Rough estimates of the diameter, length and number of vessels, their total cross-section and volume and the pressure in the vascular system [44]

From figure 6, it can be seen that the vessels of the capillary system have the smallest diameter (A) and the greatest cross-sectional areas (B) to help in diffusing the nutrients and oxygen to the surrounding tissue. The large number of vessels in the capillary system (E) is because these vessels need to be distributed in a structured way so that they can cover the tissue evenly by minimizing their volume and maximizing their surface, again to facilitate the diffusion of oxygen and nutrients [50].

As mentioned before, the oxygenated blood ejected by the heart is transported to the organs via the arterial system. Naturally, the pressure in the systemic arteries is much higher as compared to the rest of the veins and capillaries. The higher pressure in larger arteries can be seen in the figure. (F) Lastly from the figure (D) we can see that the large veins have the highest blood volume. This implies that veins, in addition to transporting deoxygenated blood back to the heart, also act as a reservoir.

3.3. Systemic Arteries

Systemic arteries are the ones responsible for delivering oxygenated blood to all the organs. Larger arteries, smaller arteries and arterioles constitute the systemic arteries by forming a network of branching vessels. This network can be seen in Fig. 7.

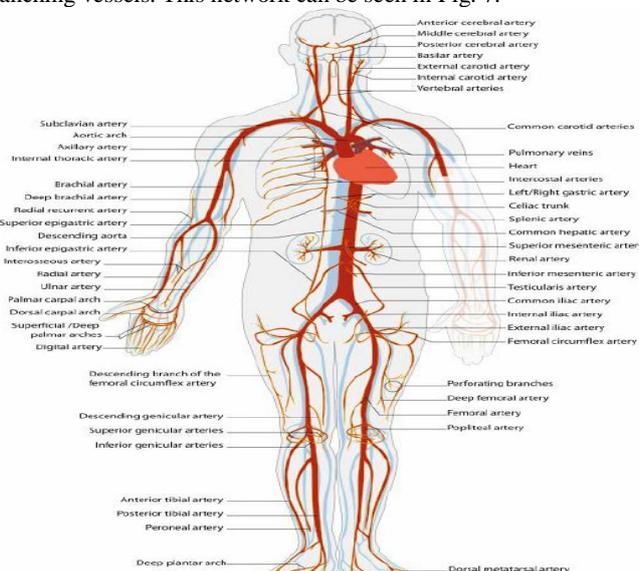


Fig. 7: The systemic arteries. [48]

Arterial walls consist of three layers:

The innermost layer is known as the Tunica Intima. This layer is the one in contact with the blood and it consists of an elastic membrane and smooth endothelial cells. The circular cavity in which the blood flows is called the lumen.

The middle layer is called the Tunica Media. This layer consists of smooth muscle and elastic fibres

The strong outer covering is known as the Tunica Adventitia. This layer consists of connective tissues, collagen and elastic fibres.

The complex composition of arterial walls make its elastic properties non-linear [51] Due to the varying amounts of elastic fibres and muscles, arteries are able to expand and contract to accommodate the pulsation of blood propagation. The behavior of the arteries is not purely elastic, in fact, arteries exhibit some viscoelastic behavior [46]. However, the effects of viscoelasticity under physiological conditions are small [52].

The structure of the systemic arteries changes gradually as we move from larger arteries to the smaller arterioles. According to Wheatler et al, [53] as the arteries become smaller, the elastic tissues decrease, while the smooth muscles become more prominent.

Due to this, the arteries become stiffer (their Young's modulus increases) as the distance from the heart increases. These changes in stiffness regulate blood flow and are an important aspect to be considered while modelling blood flow. This change in the material properties is taken into consideration via the constitutive equation, which will be seen later.

3.4. The Blood

Blood is a fluid in which various nutrients, cells, waste materials and hormones are dissolved. All these are exchanged with various tissues in the body as the blood flows through the venous system, as discussed earlier. The fluid part of blood is plasma. Blood has been consistently recognized as a non-Newtonian fluid because, although, plasma is Newtonian [54], red blood cells are suspended in plasma making blood non-Newtonian. This implies that the viscosity is dependent on shear rate [33], [55]. However, for flow in larger arteries (systemic circulation), the diameters of large arteries is large compared to the suspended particles in blood. Additionally, the viscosity is independent of the shear rates because the shear rates are high in larger vessels [46]. This makes the non-Newtonian behavior of blood inconsequential in larger vessels. Hence, blood is treated as a Newtonian fluid in numerical modelling of systemic arteries.

According to Pedley, [56] blood can be approximated as a homogeneous and incompressible fluid as 90% of plasma is water and the nutrients and cells in the plasma are much smaller than the diameter of the arteries. Usually, the density of blood is taken as $\rho = 1055 \text{ Kg/m}^3$ while the viscosity is $\mu = 4.0 \text{ mPas}$.

4. One-Dimensional Model

4.1. What is the One-Dimensional Model and how does it compare with other Dimensional Models?

The research into the cardiovascular system has come a long way over the years. Even with state of the art technology and computational methods, there are significant questions that still need answering.

As it was seen earlier, in the cardiovascular system the blood vessels have a structure that resembles that of a tree, the vessels start off with larger vessels (arteries) and branch into smaller vessels (arterioles, capillaries and venules). While this branching occurs, not only does the size of the vessels change but other properties change as well. For instance, the diameters of the vessels decrease progressively while the area of the lumen along with the stiffness of the vessels increases downstream. The varying properties and the bifurcating nature of the arterial network, has an effect on the heart loading and coronary perfusion [57]. This in turn effects the

aortic blood pressure waveform as well as the relationship of this waveform to peripheral blood pressure waveforms. These effects are yet to be fully investigated [58].

The accuracy and aims of the research are the prerequisite to selecting which kind of model should be used to simulate propagation of pulse wave in the arterial network (i.e. whether to use a zero-dimensional, one-dimensional, two-dimensional or three-dimensional model).

A table has been devised (Table 2) to show the various models. In this table the variable properties are pressure, volume and flow.

Table 2: General comparison of modelling techniques for cardiovascular dynamics studies

Model	Assumed distribution of variable properties	Types of governing equations	Applications of model
0-D	Uniform	<ul style="list-style-type: none"> 2 ODE's (Conservation of mass and momentum) Algebraic equilibrium equation (relates volume to Pressure) 	Suitable for inspection of overall pressure distribution, flow and volume of blood. Can, at times, provide boundary conditions for three-dimensional model
1-D	Non-Uniform	<ul style="list-style-type: none"> 2 PDE's (Conservation of mass and momentum) Equilibrium Equations 	Represents wave reflection/transmission effect which allows for better boundary conditions for three-dimensional models
2-D	Non-Uniform	<ul style="list-style-type: none"> 2 PDE's (Conservation of mass and momentum) Equilibrium Equations 	Represents radial variation of velocity in an axisymmetric tube allows for even improved boundary conditions for three-dimensional models but to a certain limit off applicability
3-D	Non-Uniform	<ul style="list-style-type: none"> 2 PDE's (Conservation of mass and momentum) Equilibrium Equations 	Compute complex flow patterns in any small region of the system

Zero-dimensional models are suitable if the general distribution of the pressure, flow and volume of blood needs to be inspected. However, for a more specific research, higher models are used where these flow variables are assumed to be non-uniform. Another important difference between zero-dimensional and higher models is that zero-dimensional models do not include the nonlinear convective acceleration term whereas other models do [57]. On the other hand, two-dimensional and three-dimensional models give more details about the distribution of variable properties in a small segment of the vascular system. For instance, these models can be used to reveal the meticulous pressure and flow distribution in a specific section of a certain vessel. In order to simulate the entire systemic circulation, which is extremely vast, modelling the entire arterial tree using two-dimensional and three-dimensional models is impractical. Additionally, the exact geometrical and material properties of the entire arterial tree are still unknown. Two-dimensional and three-dimensional modelling are not only costly but the computational times of simulations with these models for a few arteries takes far too long [59]–[62]. When using a one-dimensional model in comparison to a three-dimensional model, the computation cost reduces by at least 1000 times [63]. According to Alastruey et al., [28] a simulation of one cardiac cycle for approximately 100 segments takes less than a minute. A detailed comparison of one-dimensional and three-dimensional formulations can be found in [63].

The various models used to simulate the cardiovascular system are shown in Fig. 9.

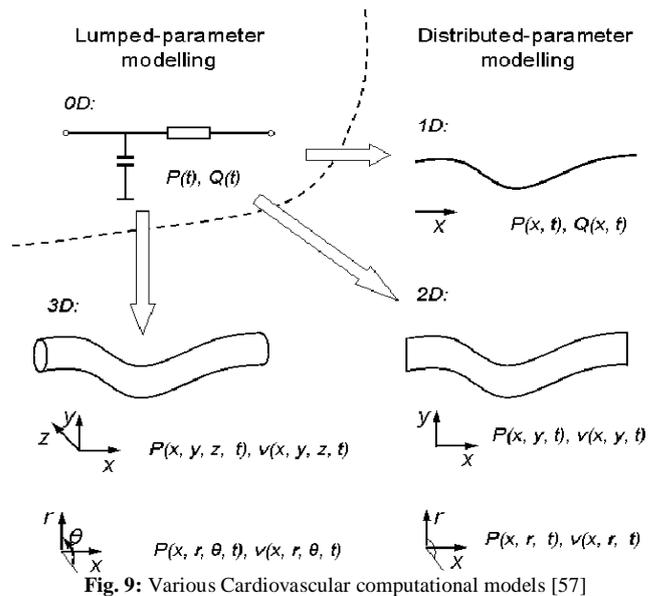


Fig. 9: Various Cardiovascular computational models [57]

Canic and Kim [64] investigated the characteristics of the governing equations of blood propagation. They demonstrated that the wavelengths of the pressure and flow waves from the heart are larger than the vessel diameters. Hence it is rational to consider the flow quasi one-dimensional [43], [65]–[67]. Therefore, a one-dimensional model is sufficient to model the entire arterial tree, as it can easily represent the changes in the variable properties along the entire tree while being economically feasible at the same time.

4.2. Types of One-Dimensional Models

A number of one-dimensional models for pulse wave propagation in the arterial network have been developed previously for various applications. [20], [21], [26], [50], [65], [68]–[80]. The equations governing these models are alike, with differences arising in the types of methods used to solve these equations, the boundary conditions applied to these models, whether non-linear effects are considered and if they are, which non-linear effects are considered [57].

Modelling pulse wave propagation in an artery using a one-dimensional model is simulated as a fluid-structure interaction problem between the flow of blood and the displacement of the arterial wall. The axisymmetric form of the one-dimensional incompressible continuity and Navier-Stokes equations governs the propagation of blood in a vessel (Equations 1 & 2) while the motion of the arterial wall is governed by the equations of equilibrium. (Equation 3) The derivation of these equations can be found here [46].

$$\frac{\partial A}{\partial t} + \frac{\partial q}{\partial x} = 0 \tag{1}$$

$$\frac{\partial q}{\partial t} + \frac{\partial}{\partial x} \left(\frac{q^2}{A} \right) + \frac{A}{\rho} \frac{\partial p}{\partial x} = - \frac{2\pi\nu R q}{\delta A} \tag{2}$$

where, x is the position along the vessel, $A(x, t)$ is the cross-sectional area of the vessel, $q(x, t)$ is the flow, $p(x, t)$ is the pressure, ν is the kinematic viscosity, δ is the thickness of the boundary layer and $R(x)$ is the radius of the vessel.

Although intricate models exist [75], the much simpler linear or non-linear constitutive equations are most commonly used to describe the pressure/cross-sectional area relationship [7], [20], [21], [26], [50], [65], [68]–[74], [76]–[81].

$$p = p_{ext} + \beta(\sqrt{A} - \sqrt{A_0}) \quad (3)$$

where,

$$\beta = \frac{\sqrt{\pi} h E}{A_0(1 - \sigma)^2} \quad (4)$$

and h is the vessel wall thickness, p_{ext} is the nominal (diastolic) pressure, A_0 is the cross-sectional area at nominal pressure ($p = p_{ext}$) and σ is the Poisson's ratio.

Details of other pressure-area relations used in previous research can be found here [12].

Other variations include the study of vessel tapering [7], [21], [71], [72], [77], [78] as well as vessel collapse [70], [71], [80]. The methods to include effects of blood viscosity also bring about slight variations in the model. Majority of authors assume Poiseuille flow (over a given cross section, the fully developed flow has a parabolic velocity profile) [22], [78], [82], [83]. Variations of these approximations can be found here [4], [66], [67]. There are some other models which simulate with the assumption that the blood is non-Newtonian, again introducing a slight variation in the formulation [84], [85].

4.3. What Can One-Dimensional Models Capture?

Unlike higher order models, one-dimensional models cannot simulate intricate details of blood flow such as flow separation or vortex formation. However, in-vivo measurements [5], [26], [86]–[93], in-vitro experiments [28], [94]–[98] as well as three-dimensionally modelled numerical data [12], [63] demonstrate that one-dimensional models can successfully capture the main features of pressure, flow and area waveforms in the systemic circulation's arterial network.

4.4. Where is the One-Dimensional Model Used?

One-dimensional models have been used for blood propagation in arterial segments [65], [69], [76] as well as entire arterial networks [5], [26]–[28], [33], [73], [74] as well as blood flow in the pulmonary circulation [99]. A variety of models have been used to study diseased vessels such as ones with stenosis or bypass grafts [21], [75], [77]–[79], [86], [100]. Other studies have looked into animal arterial networks [71], [101], [102]. Wave Intensity Analysis (WIA), developed by Parker and Jones [76], helps in understanding forward and backward pulse propagation and is a direct application of one-dimensional models. WIA has been applied to the left ventricle [103], [104], systemic arteries [105], coronary vessels [106] as well as pulmonary arteries [107], [108] to study pulse wave propagation. One-dimensional models, as pointed out earlier, are also used as boundary conditions for three-dimensional models [59], [72], [109], [110].

4.5. Solution Methods

In order to solve the system of non-linear hyperbolic equations that govern the one-dimensional propagation of the pulse wave, several analytical and numerical methods have been used. Method of characteristics was used by Olufsen [46], Schaaf [66], Parker and Jones [76], Bodley [69], Wang and Parker [73], Wang et al. [74] and Steeter et al. [65]. In the method of characteristics, the governing equations (continuity and momentum) are transformed from partial differential equations to ordinary differential equations along the direction of certain curves called characteristic curves (or lines). These characteristic curves correspond to the two characteristic variables (Riemann invariants). Once they have been transformed, these ordinary differential equations can be easily solved.

Finite difference methods (FDM) have also been used to discretize and solve the governing equations [4], [26], [70], [75], [80]–[82], [111]. In FDM, the derivatives of the governing equations form the basis of the discretization. Each of these derivatives is substituted with an approximate difference formula. Details of the finite difference method can be found in [112]–[115]. Within the finite difference method, the Lax-Wendroff scheme [5], [70], [80], [116]–[118] and MacCormack scheme [9], [80], [94], [119], [120] have been used to discretize the governing equations.

Recently, Finite volume methods (FVM) have also been used to discretize and solve the governing equations [121], [122]. In FVM, the integrals of the governing equations form the basis of the discretization. The governing equations are first discretized into finite volumes after which they are solved in each of these finite volumes. For details about the finite volume method see [123]–[125]. Within the finite volume methods, the Godunov scheme is the most popularly used [71], [101] scheme to discretize the governing equations.

Finite element methods (FEM) have also been a popular method to discretize the governing equations. In FEM, a piecewise representation of the solution in terms of specified basis functions forms the basis of the discretization. The domain where the computations is carried out is broken down into smaller domains or finite elements, see [126]–[128] for details on the finite element method. Comparison of the three methods is difficult, primarily due to the many variations of all three methods. FVM and FDM provide discrete solutions, while FEM provides a continuous solution. Generally, it is believed that FVM and FDM are easier to implement when compared to FEM. Within FEM, the Galerkin scheme [7], [8], [12], [28], [77], [78], [89], [90], [129], discontinuous Galerkin scheme [15], [20], [21], [100], Taylor-Galerkin scheme [6], [21], Galerkin Least Squares [22] and Yoshida projection scheme [72] have been used amongst others to discretize and solve the governing equations.

Other solution methods include hybrids of one-dimensional models with Womersley flow (classic linear analytical solution) [3], [43], [130], [131]. These methods do not take into account the effects of non-linearities. [21], [69], [83]. Details of these methods are beyond the scope this review but can be found here, [7], [132].

4.6. Boundary conditions

When modelling pulse wave propagation using a one-dimensional model in an arterial network or an artery (for simplicity), three boundary conditions have to be imposed, one at the proximal end, one at the distal end and one at the bifurcating end [35]. The one on the proximal end is a rather simple boundary condition where either pressure or flow (derived from experimental data or literature) can be specified. The one on the distal end, however, needs deliberation.

In various arterial tree one-dimensional models [3], [21], [22], [25], the aorta has been used as the point for the initial boundary condition. A pressure wave is either imposed as the initial condition [21] or a derived function of flow rate is specified [41], [78], [133], [134]. This makes the formulation simpler as the values are prescribed instead of modelling the aortic valve. Additionally, reverse flow into the left ventricle is not considered.

Vessel branching is another boundary condition that needs consideration. A number of researchers have applied conservation of flow (i.e. the flow in the parent vessel must be equal to the sum of flows in the daughter vessels) and continuity of pressure (the pressure in the parent vessel is equal to the pressures in each of the daughter vessels) [50], [78], [100]. Some researchers, applied wave reflection coefficients to estimate changes in flow and pressure [26] [73].

Flow characteristics in the smaller vessels is not the same as that in the larger arteries as was pointed out earlier. The fluid properties as well as the material properties change which effects the

characteristics of flow. This, in addition to the arterial tree's branching structure means there is a necessity to truncate the model. Moreover, modelling each vessel from large arteries all the way to the capillaries is simply not feasible. This need for truncation of the one-dimensional model gives rise to boundary conditions at the distal ends of the vessels. Anything beyond the truncation point (i.e. smaller vessels) needs to be consolidated.

Like the inlet condition, various researchers have prescribed a combination of pressure and flow rate [65], [69]–[71], [76], [80], [100] for the distal boundary condition.

In an attempt to introduce boundary conditions that represent physiological downstream conditions, some researchers used purely resistive loads [3], [20], [21], [43], [66], [73], [77]. However, there is no method available yet, that allows for the calculation of the values used for these loads. It has also been recognized that this method does not take into consideration the compliance of the vessels [82]. This results in a reflection coefficient, which depends on frequency (inversely proportional to any given harmonic of the pressure change).

Another way to represent the distal end of vessels is to derive a model based on the impedance at the terminal end of a vessel. This is done by using a three-element Windkessel model [4], [22], [26], [75], [78], [82] as the boundary condition. The Windkessel model characterizes the compliance as well as the resistance of the vessels by using an electric analog model. This model immensely improves the distal representation. However, this model cannot capture wave propagation effects [50] which causes undulations in the input impedance. A solution to this problem is to directly prescribe the wave reflection coefficient at the terminals [21], [73], [79] or to use the structured tree outlet boundary condition [50] in which the impedance at the terminal is estimated by the linear form of the Navier-Stokes equations. In the structured tree outlet boundary condition model, the small arteries are joined to the distal ends of the large arteries and modelled as binary asymmetric structured trees. Similar to large arteries, the equations that govern blood propagation in small arteries can be derived from the axisymmetric form of Navier-Stokes equations. However viscous effects are more prominent in small arteries as compared to inertial effects hence the Navier-Stokes equations can be linearized by neglecting the non-linear terms [5]. Once the equations are derived [5] they predict the flow $Q(x, \omega)$ and pressure $P(x, \omega)$ in the frequency domain. A no-slip boundary condition is used to link the equations together. A convolution integral of the impedance and flow rate computes the pressure at each outlet providing a physiological outflow condition for the large arteries. This boundary condition can be used for one-dimensional, two-dimensional and three-dimensional models [135]–[137] and captures wave propagation effects well, however applying this type of boundary condition to a non-linear model is tedious [12] and the computation is costly especially for 3D models. [138]

4.7. Arterial Segments

For one-dimensional modelling, the arterial network is broken down into smaller arterial sections that are connected to each other. The number of arterial segments used in one-dimensional modeling has increased in recent years from 29 to over 4 million [3], [12], [17], [25], [26], [46], [84]. Inherently, the greater the amount of arterial sections a network is broken down into, the more information required for the input parameters.

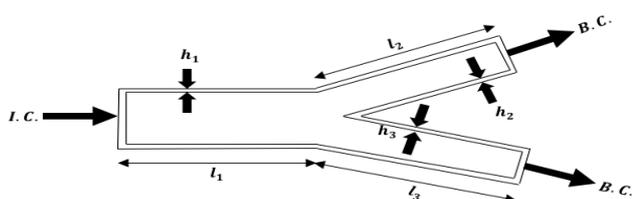


Fig. 10: A bifurcating artery

For instance, if a simple bifurcating artery is broken down into three segments, that is, the parent vessel and the two daughter vessels it bifurcates into (Fig. 10), parameters are required for each of these segments. The parameters include (but are not limited to) the lengths of each of these vessels (L_1, L_2, L_3), their inlet and outlet radii, the vessel thickness' (h_1, h_2, h_3), the stiffness of each of these vessels, the inlet boundary condition (*I.C.*) for the parent vessel and the outlet boundary condition (*B.C.*) for the daughter vessels.

If a multi-branched model is being made for the arterial network, it will have multiple bifurcations and with each generation of bifurcation, the input parameters needed increases. Moreover, these input parameters vary from person to person, making patient-specific multi branched modelling extremely tedious. A method is needed to minimize the arterial segments the network is broken down into, so that lesser input parameters are needed for patient-specific modelling [1]. Olufsen's structured tree boundary condition [46] provides a viable option for this purpose and can be taken further to incorporate patient specific models.

5. Conclusion

This paper examines available research on one-dimensional models of the cardiovascular system regarding solution methods, boundary conditions and applications. Each of the one-dimensional models described in this paper have their own applicability and advantages. Although, one-dimensional model simulations are not as exhaustive as higher-dimensional models, which include elaborate details, one-dimensional models do capture the main features of pulse wave propagation. Their computational cost effectiveness allows them to simulate large segments and at times, entire arterial networks which is not feasible using higher dimensional models, allowing one-dimensional models to be used for a wide range of applications.

As with most numerically simulated models, there is a lack of real time data that can be used to fully validate the numerical model. Indeed, determination of model input parameters from clinical data is a very challenging inverse problem. Additionally, several assumptions are made, that, although are justified, still cause the numerical model to deviate slightly from real world phenomenon. However, an important consideration is that in the present era, given the requirements of data assimilation and the nature of clinical applications, the computations not only have to be fast and iterative but they also have to be real time computations. For this purpose alone, one-dimensional models are ideal, as they are well balanced between computation cost and complexity as compared to higher dimensional models. Thus, making one-dimensional modelling very attractive for various patient-specific as well as other clinically relevant applications.

Various solution methods have been discussed that discretise and solve the governing equations. FDM and FVM are generally considered easier to implement as compared to other methods. However, both of these methods provide discrete solutions while FEM provides continuous solutions. FEM solution is also in terms of basis function, which allows more details about the solution. Although, the choice of the basis functions is of utmost importance, it must be emphasized though that a comparison between solution methods cannot be easily drawn due to the multiple variants of each of these methods. Choosing a suitable solution method depends on the complexity of the study being undertaken as well as the purpose of the study.

Conservation of flow and continuity of pressure serve as good approximations to account for the loss of energy at the bifurcation points especially because loss coefficients cannot be estimated analytically in a one-dimensional model. For outflow conditions, the most commonly used boundary condition is the three-element Windkessel model but it lacks the ability to capture wave propagation effects. Additionally, the method of estimation/choice of Windkessel parameters is not obvious. The structured tree outflow

condition includes wave propagation effects and allows pressure and flow to be measured in large as well as small arteries since flow in the small arteries is modelled using linearized Navier-Stokes equations.

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