A lumped parameter mathematical model to analyze the effects of tachycardia and bradycardia on the cardiovascular system: A LUMPED PARAMETER MATHEMATICAL MODEL

Article *in* International Journal of Numerical Modelling Electronic Networks Devices and Fields · July 2014

DOI: 10.1002/jnm.2010

CITATIONS
34

READS
1,172

5 authors, including:

Mohsen Abdi
Iran University of Science and Technology
14 PUBLICATIONS **113** CITATIONS
SEE PROFILE

Mahdi Navidbakhsh
Iran University of Science and Technology
143 PUBLICATIONS **2,423** CITATIONS
SEE PROFILE

Pirzad Gila
Baqiyatallah University of Medical Sciences
41 PUBLICATIONS **272** CITATIONS
SEE PROFILE

Kamran Hassani
Islamic Azad University Tehran Science and Research Branch
68 PUBLICATIONS **574** CITATIONS
SEE PROFILE

INTERNATIONAL JOURNAL OF NUMERICAL MODELLING: ELECTRONIC NETWORKS, DEVICES AND FIELDS
Int. J. Numer. Model. (2014)
Published online in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/jnm.2010

A lumped parameter mathematical model to analyze the effects of tachycardia and bradycardia on the cardiovascular system

Mohsen Abdi[1,2], Alireza Karimi[1,2,*,†], Mahdi Navidbakhsh[1,2], Gila Pirzad Jahromi[3] and Kamran Hassani[4]

[1]*School of Mechanical Engineering, Iran University of Science and Technology, Tehran 16846, Iran*
[2]*Tissue Engineering and Biological Systems Research Lab, School of Mechanical Engineering, Iran University of Science and Technology, Tehran 16846, Iran*
[3]*Neuroscience Research Center of Baqiyatallah University of Medical Sciences, Tehran 1956837173, Iran*
[4]*Department of Biomechanics, Science and Research Branch, Islamic Azad University, Tehran 755/4515, Iran*

ABSTRACT

In this study, the cardiovascular system and heart hemodynamic performance are modeled using lumped method (electrical analogy) to evaluate the effects of abnormal heartbeats on the cardiovascular system performance. Lumped method (voltage–current relations of an electrical circuit) is able to simulate the cardiovascular (CV) system behavior in various physiological conditions. CV system consists of 42 compartments, including artery, vein, capillary set, and heart chambers. Each blood circulatory subsystem (compartment) is modeled using electrical elements, such as resistor, capacitor, and inductor. In this study, by utilizing lumped model, CV system is simulated in MATLAB software (SIMULINK environment). There are two major types of irregular heart rates. In tachycardia, the heartbeats are too quick: over 100 beats per minute. In bradycardia, the heart beat is too slow: less than 60 beats per minute. Healthy blood circulation and heart performance are modeled (heartbeat: 75 beat/minute), and the results such as left atrium outflow-time graph and pressure-time diagram of aorta artery and pulmonary circulation are obtained. The present results are found to be in agreement with numerical and experimental studies. Then, by increasing and decreasing the heartbeat, the abnormality (150 and 50 beat/minute representing tachycardia and bradycardia, respectively) is simulated. The results show that the tachycardia leads to a significant reduction of capillary blood flow into less than 100 ml/s, while it exceeds 100 ml/s when heart has normal function. The results of the present study have clinical implications for detailed diagnosis of CV diseases when experimental studies have limitation. Copyright © 2014 John Wiley & Sons, Ltd.

Received 18 October 2013; Revised 14 April 2014; Accepted 29 May 2014

KEY WORDS: abnormal heartbeats; compartment; lumped method; cardiovascular system; tachycardia and bradycardia

1. INTRODUCTION

The cardiovascular (CV) diseases are responsible for most of the mortalities and morbidities in industrialized nations [1–4]. The circulatory system serves as a transporting vehicle for the nutrients, oxygen, carbon dioxide, hormones, blood cells, and waste to and from the cells [5]. The majority of studies regarding the physiological systems are conducted based on experiments on animals [6–8]. The clinical results found for animals' CV system are bounded values and can be only used for an approximate estimation and comparative assessment of the cognate parameters in human [9–11]. Recent development in mathematical methods brings about a less complicated computational approach to model the physiological functions of the CV system (such as lumped method) [12].

*Correspondence to: Alireza Karimi, School of Mechanical Engineering, Iran University of Science and Technology, Tehran 16846, Iran.
†E-mail: alirezakarimi@mecheng.iust.ac.ir

The circulatory system of a human body can be considered as a complex hydraulic network where pulsatile pump simulates the heart [13, 14]. Different behavior can be observed at various locations of the closed loop [15, 16]. The 0D models (named equivalent electrical circuit models) regard as uniform the distribution of essential variables (blood pressure, volume, and flow rate) in each isolated compartment (artery, vein, etc.) of the simulation [17].

The variability of the parameters can be approximated by setting up the so-called multi-compartments models, each compartment of which is regarded to be homogenous and expressed by a lumped parameter model [18]. The accurate measurement of the arterial pressure and flow is known to have great diagnostic and prognostic value [19], which inspired many attempts to simulate the circulatory system. The major focus of study for a perfect description of the CV system is undoubtedly the Navier–Stokes equations [20, 21]; however, its practical implementation for the whole body due to enormous complexities within its formulation seems impossible [22]. There have been many efforts to combine the fluid equations with the Windkessel model (WM) so as to attain both precise circulatory representation and reduction of the inherent complexities [22]. The WM is extensively adopted in applications linking blood pressure, flow, and heart load [23]. Most studies focused on parts of the circulatory system and in particular either the systemic or pulmonary arterial tree [24, 25]. The model suggested in these studies expands on the equations to cover the whole CV system [26] where it is proposed an analogous variation of WM layout without attempting to solve the resulting systems of equations. The importance of this closed-loop approach is that it offers a functional insight in the complicated CV system in terms of directly providing estimates of the blood pressures, flows, and volumes in various parts of the CV system [17, 27].

Arrhythmias (in this paper bradycardia and tachycardia) are a disturbed rhythm of human heartbeat. Tachycardia occurs when the heart beat is too fast, generally more than 100 beats per minute. Some kinds of tachycardia are without difficulty treated and not serious to consider, but others can be life-threatening. Tachycardia can be a normal reaction to physical activity but can also be a symptom of a medical problem. Bradycardia happens when heartbeats are too slow, generally less than 60 beats per minute. When heartbeats are so slow, it cannot pump enough blood to meet human body's needs and would lead to life-threatening problem.

In a nutshell, this paper includes the following steps:

- The simplified block diagram of CV system is developed in new electrical circuit.
- The operation of a new circulatory system is simulated.
- The pressure and flow distribution of different parts of CV system (such as left atrium outflow, aorta artery flow, left ventricle pressure wave propagation, aorta artery pressure waveform, pulmonary circulation pressure, pulmonary circulation flow, capillary set flow, right ventricle inflow from right atrium, and left atrium outflow) are assessed under normal and pathological (heart tachycardia and bradycardia) condition.
- The results are compared with the relevant experimental and clinical observation for verification.

2. MATERIALS AND METHODS

The human CV system components consist of a pump, the heart, and an extensive system of tubes, the blood vessels that make up a closed circular system. The vessels that leave the heart are called arteries, and those that return blood to the heart are called veins. Pumping of the heart generates an arterial blood pressure of about 100 mmHg, while venous blood returns to the heart at a pressure of about 5–10 mmHg.

The equivalent electrical circuit of circulatory system is based upon [27, 28] and depicted in Figure 1(a). In this electrical analogy, electric potential and current are corresponded to the average pressure and flow rate, respectively. A special vessel (or cluster of vessels) is characterized by means of its impedance, which is represented by a suitable combination of resistors, capacitors, and inductors. The resistors are utilized to simulate viscous dissipation, while the capacitors account for vessel compliance, the ability to accumulate and release blood due to elastic deformation. Finally, the inductors are used to simulate inertia terms. Calculation of these parameters is carried out according to previous studies [17, 27–30]. Regions of the vascular system can then be modeled and linked in a circuit network (42 compartments).

DOI: 10.1002/jnm

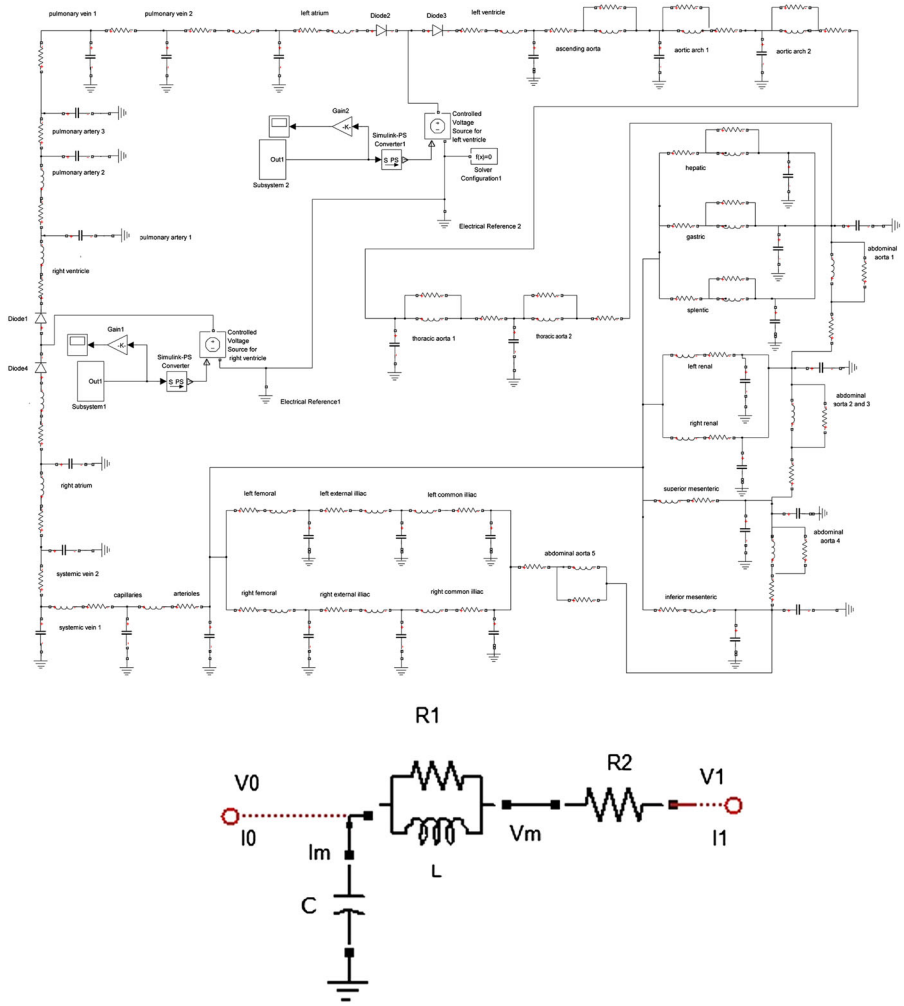

Figure 1. (a) Equivalent electronically circuit of the human cardiovascular system (42 compartment subsystems). (b). Typical electrical compartment and corresponding block diagram.

Considering electrical compartment at Figure 1(b), we can now write an equation for the pressure drop across the first resistor and capacitor as shown in Equation 1.

$$\begin{aligned} V_0 &= \frac{1}{C}\int (I_0 - I_1)dt + V_{C_0} \\ V_m &= V_1 + R_2 I_1 \\ I_1 &= \frac{1}{L}\int (V_m - V_0)dt + \frac{(V_m - V_0)}{R_2} \end{aligned} \tag{1}$$

In this model, the parameters are roughly determined. The diastolic filling and the systolic period are assumed to last for 0.5 and 0.3 s, respectively, for a heart period total of 0.8 s, which corresponds to 60/0.8 = 75 beats/min.

In the case of tachycardia and bradycardia (abnormal heart beats), the simulation is performed by changing heart beats and recalculating of electric lumped parameter such as resistance, inductance, and compliance amount [28]. By inserting these values into the computer simulation, the pressure and flow (similar to voltage and current) wave propagations at different parts of CV system are achieved.

DOI: 10.1002/jnm

We can derive the units of C, L, and R:

$$C_{\text{units}} = \text{cm}^4\text{*s}^2/\text{g}$$
$$L_{\text{units}} = \text{g}/\text{cm}^4$$
$$R_{\text{units}} = \text{g}/\ (\text{cm}^4\text{*s})$$

Note that the viscosity μ of normal blood is $\mu = 0.035$ poise (g/cm*s) and normal blood density is $\rho = 1.05\ \text{g/cm}^3$. Pressure unit in this circuit is $\text{g/cm} * \text{s}^2$ (is not N/m^2), but multiplying the pressure in mmHg unit by number 1332, the unit change to $\text{g/cm} * \text{s}^2$.

3. RESULTS

3.1. Flow and pressure wave propagation in different parts of cardiovascular system under normal condition (healthy heart)

The flow-time graph of pulmonary circulation is shown in Figure 2(a), where the waveform varies between 0 and 260 ml/s (electric current). The waveform starts from 0 mm/s, and the peak is in 260 ml/s. The flow-time graph of capillary set is shown in Figure 2(b). This graph shows that capillary set flow varies between 58 and 105 ml/s (diastole-systole). Figure 2(c) shows that right ventricle inflow varies between 0 and 225 ml/s (at heart cycle).

3.2. Flow and pressure wave propagation in different parts of cardiovascular system under pathological condition (tachycardia)

The maximum amount of aortic pulse wave flow and left atrium outflow in Figure 3(a) is 950 and 230 ml/s, respectively. Figure 3(b) shows maximum pressure (during systole) in left ventricle and aorta artery, which presents a sinusoidal pattern. Figure 3(c) depicts pressure patterns in pulmonary circulation where the waveform varies between 7.5 and 32.5 mmHg. Figure 3(d–f) indicates flow graph of pulmonary circulation, capillary set, and right atrium as a function of time is tachycardia.

3.3. Flow and pressure wave propagation in different parts of cardiovascular system under pathological condition (bradycardia)

The maximum amount of aortic pulse wave flow and left atrium outflow in Figure 4(a) is 600 and 200 ml/s, respectively. Figure 4(b) shows maximum pressure (during systole) in left ventricle and aorta artery, which presents a sinusoidal pattern. Figure 4(c) depicts pressure patterns in pulmonary circulation where the waveform varies between 4.9 and 32 mmHg. Figure 4(d–f) indicates flow graph of pulmonary circulation, capillary set, and right atrium as a function of time when bradycardia occurs.

4. DISCUSSION

4.1. Under normal condition

Under normal condition, each time period takes 0.8 s. The average heart rate for adult humans is 75 beats per minute in a normal undisturbed mode. A person's blood pressure is usually expressed in terms of the systolic pressure over diastolic pressure (for left ventricle) and is measured in millimeters of mercury (mmHg), for example, 140/50 mmHg. Blood pressure usually ranges between 32 for the top or maximum number (systolic) and 5 for the bottom or minimum number (diastolic) for the right ventricle [27, 28]. The left and right ventricular pulse pressures are considered as entrance condition.

The maximum amount of aortic pulse wave flow and left atrium outflow in Figure 5(a) is 800 and 300 ml/s, respectively (healthy heart). To validate our model, we compare it (Figure 5(a)) with Reference [17] as shown in Figure 5(b). However, in the reference, minor differences naturally should be found. This difference could be the result of the type which the problem is solved and the formulae that are also applied to the solution. In both diagrams, there are similarities. For example, both have

DOI: 10.1002/jnm

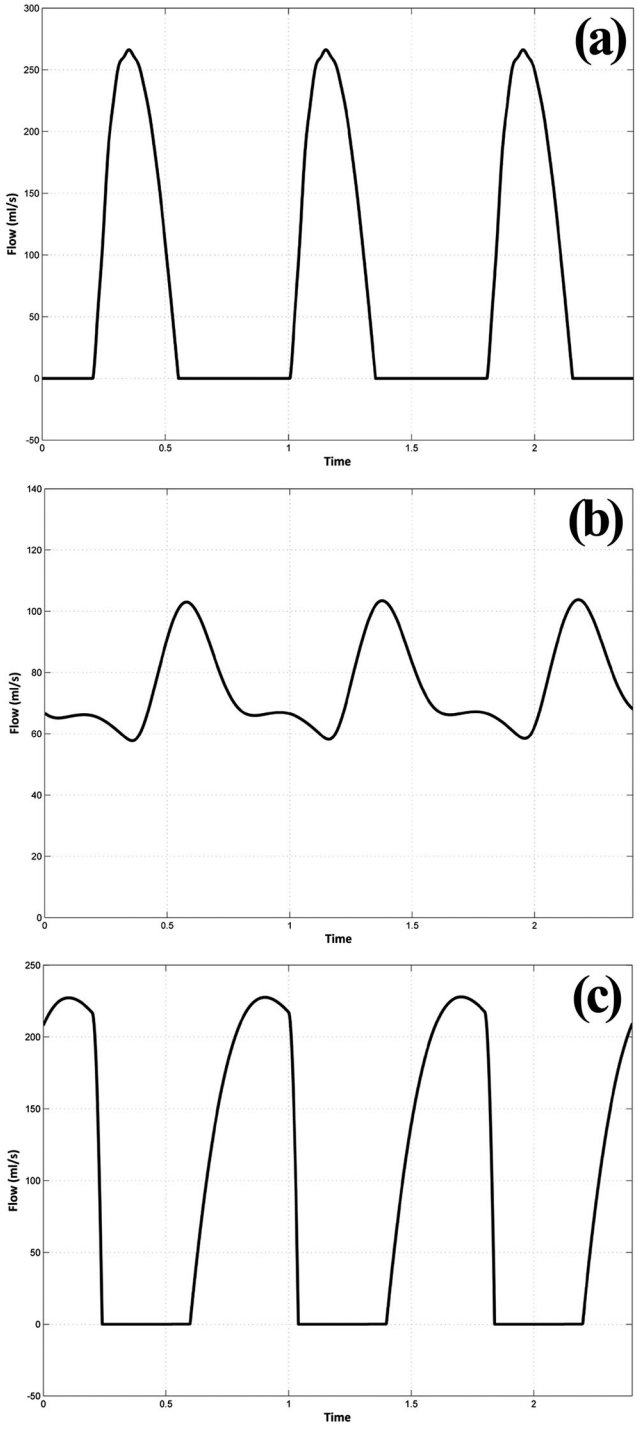

Figure 2. Flow-time graph of pulmonary circulation under normal condition (a). Flow versus time graph of capillary set under healthy condition (b). Inflow-time graph of right ventricle that is supplied from right atrium under normal heart hemodynamic performance (c).

two peaks, although in both of them, basically there are clearly some differences in pattern. Pressure-time diagram of aorta artery and pulmonary circulation that is inserted in the clinical and numerical references [14, 17, 31] (Figure 5(d) and(e)) is the other guideline that we could use for the comparison with Figure 5(c and f), respectively.

DOI: 10.1002/jnm

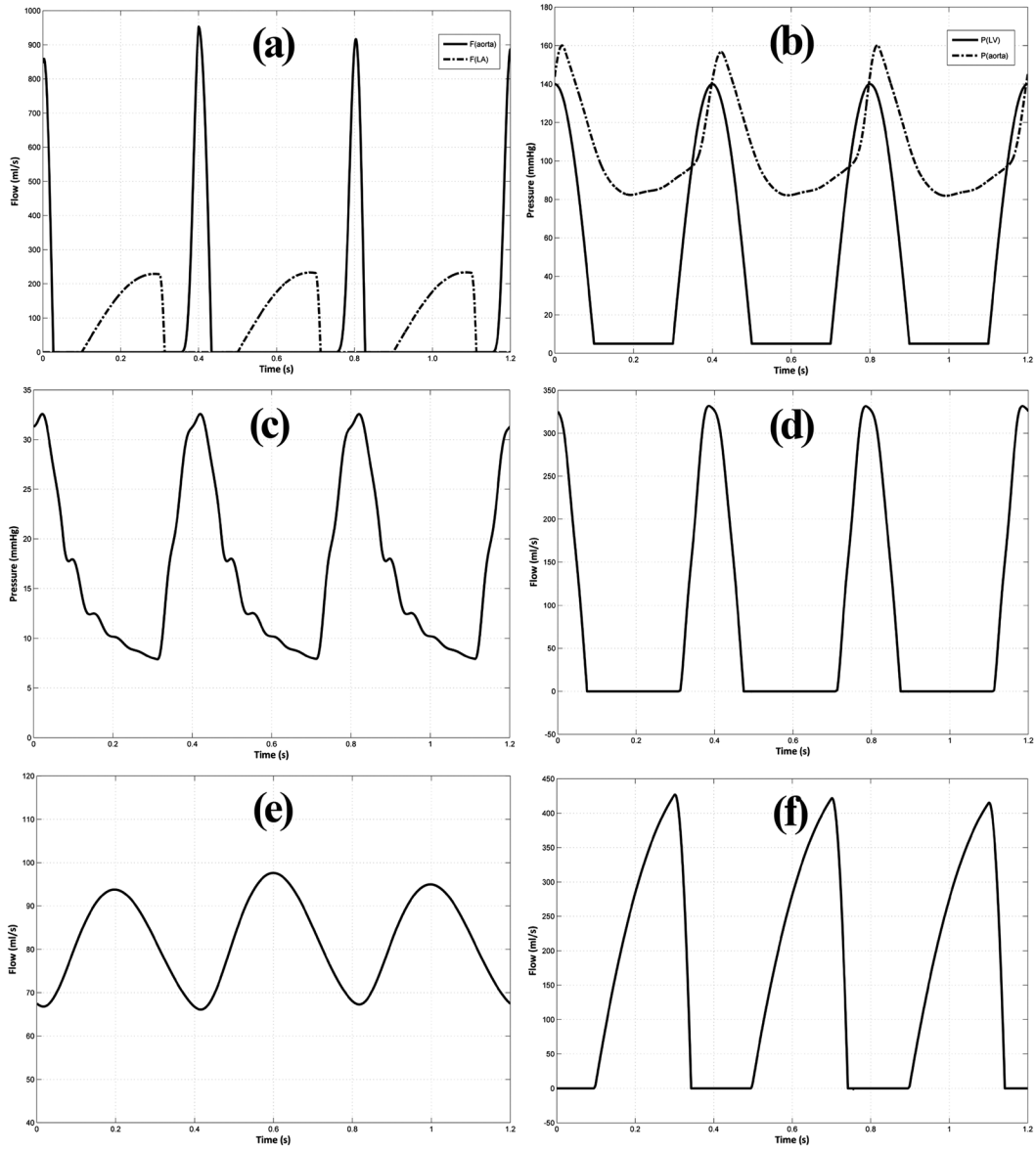

Figure 3. Flow wave propagation along the aorta and also through the left atrium with respect to time under abnormal circumstance (tachycardia) (a). Left ventricle and aorta artery pressure pulse as a function of time (b). Pressure over cardiac cycles of pulmonary circulation (c). Flow-time graph of pulmonary circulation (d). Flow in capillary set as a function of time (e). Right ventricle inflow perfuse from right atrium over cardiac cycles (f).

4.2. Under pathological condition (tachycardia)

In this section, by frequency changing and increased heart rate to 150 beat per minute, tachycardia is simulated. We compare pressure and flow-time graph of different organs under both conditions to further assess the possible effects of the tachycardia on CV function. As it is shown, maximum flow rate through the aortic artery exceeding 900 ml/s when tachycardia (Figure 3(a)) occurs, while results depict 800 ml/s of aorta artery flow in the name of a healthy heart (Figure 5(a)). In addition, aortic artery maximum flow takes place in a shorter amount of time (tachycardia) with respect to normal condition.

As it is plotted, aortic pressure exceeding 160 mmHg when tachycardia occurred (Figure 3(b)), while healthy heart diagram show less than 150 mmHg of maximum amount of aortic pressure (Figure 5(c)). Thus, faster resting heart rate has been shown to be associated with a higher risk of developing hypertension and a greater incidence of CV morbidity and mortality. Arterial pulse analysis

DOI: 10.1002/jnm

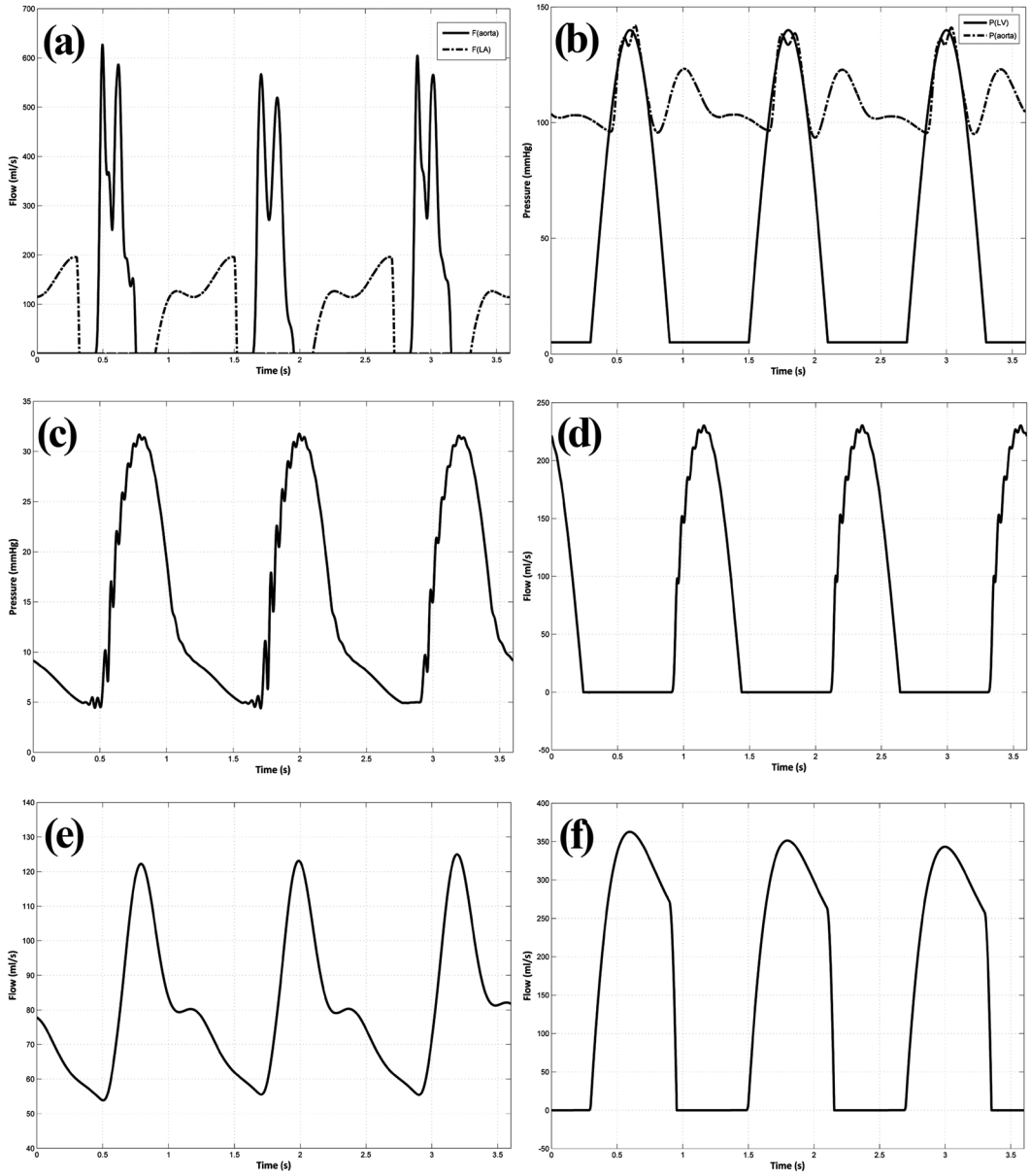

Figure 4. Flow wave propagation along the aorta and also through the left atrium respect to time under abnormal circumstance (bradycardia) (a). Left ventricle and aorta artery pressure pulse as a function of time (b). Pressure over cardiac cycles of pulmonary circulation (c). Flow-time graph of pulmonary circulation (d). Flow in capillary set as a function of time (e). Right ventricle inflow perfuse from right atrium over cardiac cycles (f).

was noted to provide useful diagnostic tool. Maximal and minimal values of pulmonary arterial pressure (Figure 3(c)) do not substantially change (for tachycardia) from normal hearts (Figure 5(f)), but the shape of the curve is slightly different. Pulmonary arterial pressure is generated by the right ventricle ejecting blood into the pulmonary circulation, which acts as a resistance to the output from the right ventricle.

Pulmonary blood flow in the normal heart is about 260 ml/s (Figure 2(a)), while the blood flow reaches 300 ml/s when tachycardia occurs (Figure 3(d)). Tachycardia-induced heart causes capillary blood flow to become less than 100 ml/s (Figure 3(e)), while exceeding 100 ml/s when heart has normal function (Figure 2(b)). In this case, owing to an increased heart rate, capillary flow has less opportunity to reach maximum amount. Right ventricular inflow volume in induced tachycardia heart increased to the amount of 400 ml/s (Figure 3(f)), while healthy heart shows the flow rate less than 250 ml/s (Figure 2(c)).

DOI: 10.1002/jnm

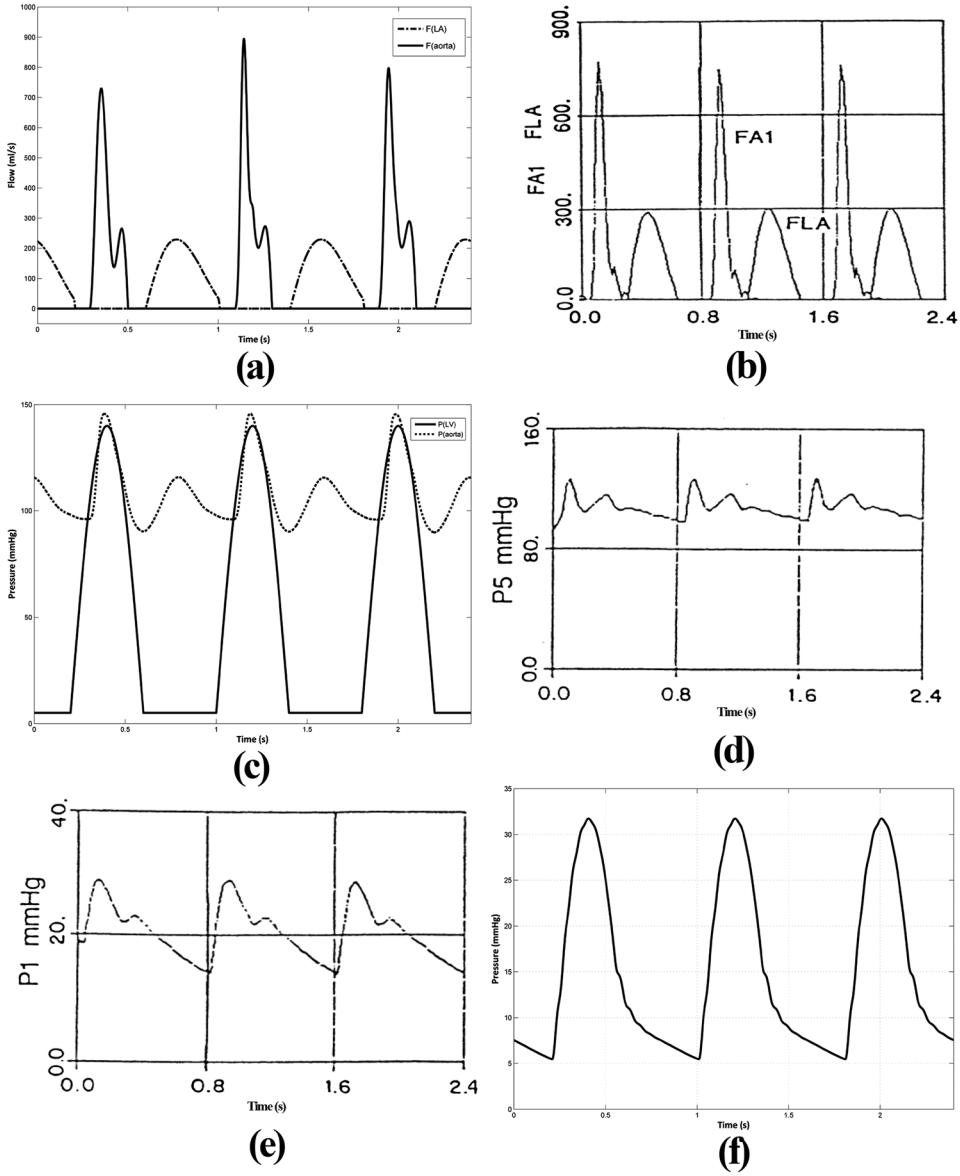

Figure 5. Flow wave propagation along the aorta artery as a function of time and also outflow wave propagation of left atrium as a function of time in a healthy young adult (a). Aorta artery flow wave propagation as a function of time and also outflow-time graph of left atrium as a function of time under normal condition based upon reference (b). Pressure over cardiac cycles of left ventricle and aorta artery in healthy young adult (c). Aorta artery pressure as a function of time under normal condition based upon literature (d). Pressure-time graph of pulmonary circulation (e). Pressure-time graph of pulmonary circulation (f).

4.3. Under pathological condition (bradycardia)

In this section of this study, by frequency changing and decreased heart rate to 50 beat per minute, bradycardia is simulated. Similar to tachycardia part, we compare pressure and flow-time graph of different organs under both conditions (bradycardia-induced heart and healthy heart) to further assess the possible effects of the bradycardia on CV function.

It is depicted that the maximum flow rate through the aortic artery is decreased to 600 ml/s when bradycardia occurs (Figure 4(a)), while results show 800 ml/s of aorta artery flow in the name of a healthy heart (Figure 5(a)). As it is plotted, aortic pressure decreased to 140 mmHg when bradycardia

DOI: 10.1002/jnm

occurs (Figure 4(b)), while healthy heart diagram shows less than 150 mmHg of maximum amount of aortic pressure (Figure 5(c)). In this case, maximum amount of aortic pressure is equal to maximal value of left ventricle pressure approximately (Figure 4(b)). Maximal and minimal values of pulmonary arterial pressure do not substantially change (for bradycardia same as tachycardia) from normal hearts (Figure 5(f)), but the shape of the curve is slightly different (Figure 4(c)). Pulmonary blood flow in the normal heart is about 260 ml/s (Figure 2(a)), while the blood flow decreased to 200 ml/s when bradycardia occurs (Figure 4(d)). Bradycardia-induced heart causes capillary blood flow to become more than 120 ml/s (Figure 4(e)), while it reaches to 100 ml/s when heart has normal function (Figure 2(b)). In this case, because of a decreased heart rate, capillary flow has more opportunity to reach maximum amount. Right ventricular inflow volume in induced bradycardia heart increased to the amount of 350 ml/s (Figure 4(f)), while healthy heart shows the flow rate less than 250 ml/s (Figure 2(c)).

The incorporated explanation of the whole arterial system by means of a lumped system can adequately describe pressure-flow relations. If we can extract some initial values such as length, radius, and elastic modulus for every vessel by experimental works, then we can obtain electrical elements such as resistor, capacitor, and inductor. By inserting these element to electrical circuit and by using lumped method (equivalent electronically circuit), the pressure and flow propagation of every vessel or set of vessels can be assessed, which is impossible by experimental studies. This method can be helpful to diagnose human heartbeat problems in specific region such as capillary set when there are no signs at other vessels in some patient.

4.4. Limitations

In our outlook, a more accurate but clearly more complex model of the systemic arteries can be constructed by considering the bifurcating network of branched arteries and describing each element in that network by its appropriate lumped model. An observable bug of this research is the lack of experimental confirmation, because of the lack of detailed information on the geometry and elasticity of the arterial network and on the boundary conditions. In this study, atriums are modeled using constant capacitance while can be simulated by variable parameters such as ventricles in the future studies. Moreover, realistic mechanical properties of arteries and veins using uniaxial [32–39] or puncture [40] tests using low strain rates [41–47] would help us to strengthen the results of numerical models.

5. CONCLUSIONS

The equivalent electrical circuit (lumped parameter models) that represents the main components of the system (vasculature compartments and the heart with its valves) is suitable for the study of global distribution of the pressure, flow rate, and blood volume, for specific physiological conditions. This study had aim to model the pathological disorder in CV system using equivalent electrical system modeling. In fact, this is one the main advantage of lumped method (computational modeling) that we can acquire pressure and flow pattern with respect to time, which is very hard to capture by experimental methods. In addition, by numerical approach such as lumped model incremental rate of parameters (e.g., pressure and flow) in pathological condition such as stenosis, aneurysm, and abnormal heart beat can be obtained, which is impossible by laboratory and experimental methods.

CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflicts of interests.

ACKNOWLEDGMENT

The authors acknowledge the help and support of their colleagues at Iran University of Science and Technology.

DOI: 10.1002/jnm

REFERENCES

1. Karimi A, Navidbakhsh M, Shojaei A, Hassani K, Faghihi S. Study of plaque vulnerability in coronary artery using Mooney–Rivlin model: a combination of finite element and experimental method. *Biomed Eng: Appl, Basis Commun* 2013; **26**:145–152. doi:10.4015/S1016237214500136.
2. Karimi A, Navidbakhsh M, Alizadeh M, Shojaei A. A comparative study on the mechanical properties of the umbilical vein and umbilical artery under uniaxial loading. *Artery Res* 2014; **8**:51–56.
3. Karimi A, Navidbakhsh M, Rezaii T, Hassani K. Measurement of the circumferential mechanical properties of the umbilical vein – experimental and numerical analysis. *Comput Methods Biomech Biomed Engin* 2014. doi:10.1080/10255842.2014.910513.
4. Karimi A, Navidbakhsh M, Razaghi R, Haghpanahi M. A computational fluid-structure interaction model for plaque vulnerability assessment in atherosclerotic human coronary arteries. *J Appl Phys* 2014; **115**:144702–144711.
5. Guyton AC. Textbook of Medical Physiology (11). Elsevier Saunders: Philadelphia, 2006.
6. Wu XM. Modeling and simulation of cardiovascular circulation system: status and prospect. *Science* 1994; **2**:112–116.
7. Karimi A, Navidbakhsh M, Yousefi H, Motevalli Haghi A, Adnani Sadati SJ. Experimental and numerical study on the mechanical behavior of rat brain tissue. *Perfusion* 2014. Published online 11 February 2014, DOI: 10.1177/0267659114522088.
8. Karimi A, Navidbakhsh M, Beigzadeh B, Faghihi S. Hyperelastic mechanical behavior of rat brain infected by Plasmodium berghei ANKA-experimental testing and constitutive modeling. *Int J Damage Mech* 2013. Published online 27 November 2013, DOI: 10.1177/1056789513514072
9. Karimi A, Navidbakhsh M, Shojaei A, Faghihi S. Measurement of the uniaxial mechanical properties of healthy and atherosclerotic human coronary arteries. *Mater Sci Eng C* 2013; **33**:2550–2554. doi:10.1016/j.msec.2013.02.016.
10. Karimi A, Navidbakhsh M, Faghihi S. A comparative study on plaque vulnerability using constitutive equations. *Perfusion* 2014; **29**:179–154. doi:10.1177/0267659113502835.
11. Karimi A, Navidbakhsh M, Faghihi S, Shojaei A, Hassani K. A finite element investigation on plaque vulnerability in realistic healthy and atherosclerotic human coronary arteries. *Proc Inst Mech Eng H* 2013; **227**:148–161. doi:10.1177/0954411912461239.
12. Cellier FE, Kofman E. Continuous System Simulation. Springer-Science: New York, 2006.
13. Olufsen MS, Nadim A. On deriving lumped models for blood flow and pressure in the systemic arteries. *Math Biosci Eng* 2004; **1**:61–80.
14. de Canete JF, Saz-Orozco P, Moreno-Boza D, Duran-Venegas E. Object-oriented modeling and simulation of the closed loop cardiovascular system by using SIMSCAPE. *Comput Biol Med* 2013; **43**:323–333.
15. Rideout VC, Snyder MF. Computer simulation study of the cardiovascular and related physiological systems. *Math Comput Simul* 1974; **16**:26–34.
16. Rupnik M, Runovc F, Sket D, Kordas M. Cardiovascular physiology: simulation of steady state and transient phenomena by using the equivalent electronic circuit. *Comput Methods Programs Biomed* 2002; **67**:1–12.
17. Rideout VC. Mathematical and Computer Modeling of Physiological Systems. Prentice Hall: New York, 1991.
18. Abdolrazaghi M, Navidbakhsh M, Hassani K. Mathematical modelling of intra-aortic balloon pump. *Comput Methods Biomech Biomed Engin* 2010; **13**:567–576.
19. O'Rourke MF, Adji A. Noninvasive studies of central aortic pressure. *Curr Hypertens Rep* 2012; **14**:8–20.
20. Melbin J, Noordergraaf A. Pressure gradient related to energy conversion in the aorta. *Circ Res* 1983; **52**:143–150.
21. Halabian M, Karimi A, Barati E, Navidbakhsh M. Numerical evaluation of stenosis location effects on hemodynamics and shear stress through curved artery. *J Biomater Tissue Eng* 2014, in press.
22. Quarteroni A, Engquist B, Schmid W. Modeling the Cardiovascular System: A Mathematical Challenge Inmathematics Unlimited-2001 and Beyond. Springer: Berlin, 2000.
23. Lankhaar JW, Westerhof N, Faes TJC, Marques KMJ, Marcus JT, Postmus PE. Quantification of right ventricular afterload in patients with and without pulmonary hypertension. *Am J Physiol Heart Circ Physiol* 2006; **291**:1731–1737.
24. Fogliardi R, Di Donfrancesco M, Burattini R. Comparison of linear and nonlinear formulations of the three-element Windkessel model. *Am J Physiol Heart Circ Physiol* 1996; **271**:2661–2668.
25. Laskey WK, Kussmaul WG, Noordergraaf A. Valvular and systemic arterial hemodynamics in aortic valve stenosis. *Circulation* 1995; **92**:1473–1478.
26. Keener J, Sneyd J. Mathematical Physiology, Interdisciplinary Applied Mathematics. Springer Verlag: Berlin, 1998.
27. Hassani K, Navidbakhsh M, Rostami M. Simulation of the cardiovascular system using equivalent electronic system. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub* 2006; **150**:105–112.
28. Abdi M, Karimi A, Navidbakhsh M, Hassani K, Faghihi S. Modeling of cerebral aneurysm using equivalent electrical circuit (lumped model). *Perfusion* 2013; **29**:143–153. doi:10.1177/0267659113498617.
29. Hassani K, Navidbakhsh M, Rostami M. Modeling of the aorta artery aneurysms and renal artery stenosis using cardiovascular electronic system. *Biomed Eng Online* 2007; **9**:22.
30. Abdi M, Karimi A, Navidbakhsh M, Rahmati M, Hassani K. Modeling of coronary artery balloon-angioplasty using equivalent electrical circuit. *Biomed Eng: Appl, Basis Commun* 2014; **26**:145–152.
31. Guyton AC. Text Book of Physiology. W.B. Saunders: Philadelphia, 1996.
32. Karimi A, Navidbakhsh M, Alizadeh M, Razaghi R. A comparative study on the elastic modulus of polyvinyl alcohol sponge using different stress-strain definitions. *Biomed Tech (Berl)* 2014. doi:10.1515/bmt-2013-0110.
33. Karimi A, Navidbakhsh M, Faghihi S. Measurement of the mechanical failure of PVA sponge using biaxial puncture test. *J Biomater Tissue Eng* 2014; **4**(1):46–50.
34. Karimi A, Navidbakhsh M. An experimental study on the mechanical properties of rat brain tissue using different stress-strain definitions. *J Mater Sci - Mater Med* 2014. doi:10.1007/s10856-014-5198-0.
35. Karimi A, Faturechi R, Navidbakhsh M, Hashemi A. A nonlinear hyperelastic behavior to identify the mechanical properties of rat skin under unaxial loading. *J Mech Med Biol* 2014; **14**:1450075–1450089.
36. Karimi A, Navidbakhsh M, Yousefi H, Alizadeh M. An experimental study on the elastic modulus of gelatin hydrogel using different stress-strain definitions. *J Thermoplast Compos Mater* 2014. doi:10.1177/0892705714533377.
37. Karimi A, Navidbakhsh M, Faghihi S. Fabrication and mechanical characterization of polyvinyl alcohol sponge for tissue engineering applications. *Perfusion* 2014; **29**(3):232–238.
38. Karimi A, Navidbakhsh M, Motevalli Haghi A, Faghihi S. Measurement of the uniaxial mechanical properties of rat brains infected by Plasmodium berghei ANKA. *Proc I Mech H* 2013; **227**(5):609–614.
39. Karimi A, Navidbakhsh M, Beigzadeh B. A visco-hyperelastic constitutive approach for modeling polyvinyl

alcohol sponge. *Tissue Cell* 2014; **46**(1):97–102.
40. Faturechi R, Karimi A, Hashemi A, Navidbakhsh M. Mechanical characterization of Peritoneum/Fascia under uniaxial loading. *J Biomater Tissue Eng* 2014; **4**(3):189–193.
41. Karimi A, Navidbakhsh M. Mechanical properties of polyvinyl alcohol sponge under different strain rates. *Int J Mater Res* 2014; **105**:404–408.
42. Faghihi S, Gheysour M, Karimi A, Salarian R. Fabrication and mechanical characterization of graphene oxide-reinforced poly (acrylic acid)/gelatin composite hydrogels. *J Appl Phys* 2014; **115**(8):083513.
43. Karimi A, Navidbakhsh M. Measurement of the nonlinear mechanical properties of PVA sponge under longitudinal and circumferential loading. *J Appl Polym Sci* 2013; **131**(10):40257.
44. Karimi A, Navidbakhsh M, Haghpanahi M. Constitutive model for numerical analysis of polyvinyl alcohol sponge under different strain rates. *J Thermoplast Compos Mater* 2014. doi:10.1177/0892705713520176.
45. Faghihi S, Karimi A, Jamadi M, Imani R, Salarian R. Graphene oxide/poly(acrylic acid)/gelatin nanocomposite hydrogel: experimental and numerical validation of hyperelastic model. *Mater Sci Eng C* 2014; **38**:299–305.
46. Karimi A, Navidbakhsh M, Razaghi R. An experimental-finite element analysis on the kinetic energy absorption capacity of polyvinyl alcohol sponge. *Mater Sci Eng C* 2014; **39**:253–258.
47. Karimi A, Navidbakhsh M. Mechanical properties of PVA material for tissue engineering applications. *Mater Technol: Adv Perform Mater* 2014; **29**(2):90–100.

AUTHORS' BIOGRAPHIES

Mohsen Abdi received the MSc degree in biomedical engineering in 2012 from the Iran University of Science and Technology, Tehran, Iran. His primary area of interest is the electro-mechanical modeling of the cardiovascular systems, system biology, and advanced mathematics in bioengineering.

Alireza Karimi was born in Tehran/Iran on March 22, 1988. He received the MSc degree in biomedical engineering/biomechanics from the Iran University of Science and Technology, Tehran, Iran in 2012. He currently holds a PhD position in mechanical engineering/biomechanics at the Iran University of Science and Technology. So far, he published more than 50 peer-reviewed articles in well-known scientific journals. Nowadays, he is working on the mechanical properties of soft biological tissues using new continuum mechanics approach.

Mahdi Navidbakhsh was born in Tehran, Iran, in 1965. He received the BS degree from the Sharif University of Technology in 1989, and the MSc and PhD degree in 1993 and 1996, respectively, from the INPL (ENSEM), Nancy, France. Since 1991, he has been at the Department of Mechanical Engineering of the Iran University of Science and Technology, Tehran, Iran as an associate professor. He is involved in bio fluidic and mechanical characterization techniques for biological systems.

DOI: 10.1002/jnm

Gila Pirzad Jahromi was born in Tehran, Iran, in 1961. She received the BS and MSc degree from the Tehran University of Medical Sciences and the Baqiyatallah University of Medical Sciences, respectively. She also received her PhD degree in the field of human physiology from the Tehran University of Medical Sciences. She is currently working as an assistant professor at the Baqiyatallah University of Medical Sciences. Her research interests are including primary cell culture, cell therapy, ischemia injuries in brain, lung diseases, and immunocytochemistry.

Kamran Hassani received his MSc and PhD degrees from Iran University of Science and Technology and Amirkabir University of Technology in biomedical engineering in Tehran. He is now with the Department of Biomechanics of Science and Research branch of Islamic Azad University since last 6 years. He is interested in cardiovascular system modeling and study of the abnormities using lumped models or FSI/CFD simulations.

DOI: 10.1002/jnm