Conceptual model of arterial tree based on solitons by compartments

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Abstract— Models define a simplification of reality, which help to understand function. The arterial system has been modeled in many ways: lumped models, tube models and anatomically based distributed models. In this work, arterial segments were modeled as thin nonlinear elastic tubes filled with an incompressible fluid, whose governing dynamics were denoted by the Korteweg and DeVries equation. In order characterize the pressure pulse propagation, a discrete multisegmented conduit was proposed. Arterial wall mechanical parameters were obtained from existing literature and assigned to each individual segment. The numerical model was developed starting in the aortic arch, and ending at the femoral artery.

The main idea of this article was to perform a computational simulation of pressure wave propagation, considered as a solitons combination, along several segments of the arterial tree.

I. INTRODUCTION

Historically, a qualitative interpretation of changes in the texture and strength of the arterial pulse is associated with a change in health and disease. In this sense, atherosclerosis constitutes the essential link between the risk factor and clinical cardiovascular disease as myocardial infarction. stroke or arteriopathy of the lower limbs [1]. The beating heart pumps blood pressure and flow pulsations that propagate as waves through the arterial tree. These waves are reflected at transitions of the arterial geometry and elasticity [2]. Associated abrupt changes in geometry and elastic properties induce abnormal wave reflections which modify the characteristics of arterial wave propagation and even affect cardiac dynamics [3]. Under physiological conditions, the pulsatile burden is lower in central than in peripheral arteries, thus protecting the heart against an excess load [4].For this reason, the study of wave contour and speed of the arterial blood pressure (ABP) results highly relevant in the arterial wall mechanics.

The modeling of arterial wave propagation extends our knowledge about the functioning of the cardiovascular system and provides means to diagnose disorders and predict the outcome of medical interventions [2]. Models define a simplification of reality, which help to understand their function. The arterial tree has been described in many ways: lumped models, tube models and anatomically based distributed models [5]. The cardiovascular system can be

This work was supported by National Technological University (NTU) – PID 2220.

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seen as a complex double chamber pump, which ejects blood into vessels organized into vascular compartments forming a closed circulation loop. This point of view is useful for building models of the whole system as interconnection of simpler subsystem models [6].

In the systemic circulation large vessels are approximated by tubes with thin-elastic walls, while the blood filling the vessel is considered as a continuum, incompressible fluid [7]. The propagation of finite amplitude waves in a fluid-filled elastic or viscoelastic tube has been examined in several works [8], [9], [10] [11], [12]. Depending on the balance of nonlinearity, dispersion and dissipation, the Korteweg and DeVries equation (KdV), Burgers or KdV-Burgers equations are obtained as the evolution equation.

In [10] and [11] it has been proved that if the blood viscosity is neglected, a segment can be modeled as a thin nonlinear elastic tube filled with an incompressible fluid, whose governing dynamics is denoted by the KdV equation (KdVE). Despite this, few numerical simulations of the obtained models equations have been developed in order to study wave behavior in a system composed of arterial compartments. The ABP representation by means of solitons has been introduced by [10]. Additionally, an interesting reduced model, consisting of a nonlinear superposition of two or three-solitons for the solution of the KdV equation (KdVE) has been used to describe these propagation phenomena [13].

This new approach, which provides a soliton combination as a solution, already captures some of the known pulse pressure phenomena such as steepening (increase in the amplitude) and peeking (decrease in the width). Solitons can be defined as nonlinear dispersive waves that travel without presenting structural changes. Additionally, an interaction between solitary waves only influences their relative phase, preserving its original properties [14].

The main idea of this article is to perform a computational simulation of pressure wave propagation, considered as a solitons combination, throughout several segments of the arterial tree. To this end, each segment was modeled as a KdVE where vascular dimensions and elastic constants were obtained from the existing literature.

II. MATERIALS AND METHODS

A. Model Equations

In the model proposed in [6] several assumptions were made. Firstly, large arteries were considered as an elastic tube and the fluid was supposed to be incompressible with a Poiseuille flow. Secondly, blood viscosity was neglected and only contemplated in the outflow boundary condition. As a result, a quasi 1D model of the Navier Stokes equation in the boundary layer of a thin nonlinear elastic tube was proposed as follows:

$$A_T + Q_Z = 0, \tag{1}$$

$$Q_T + \left(\frac{\alpha Q^2}{A}\right)_Z + \frac{A}{\rho} P_Z = 0.$$
 (2)

where Z and T are the spatial and time variables respectively, R(T,Z) is the vessel radius, A(T,Z) is the cross-sectional area of the vessel, Q(T,Z) is the blood flow, P(T,Z) is the blood pressure, ρ is blood density and α is the momentum-flux correction coefficient. The subscript T or Z indicates time or spatial derivative. Furthermore, the motion of the wall satisfies:

$$\left(\frac{\rho_w h_0 R_0}{A_0}\right) A_{TT} = (P - P_e) - \frac{h_0}{R_0} \sigma$$
 (3)

where ρ_w is the wall density, P_e is the pressure outside the tube, h_0 the wall thickness, R_0 the mean radius and σ the extending stress in tangential direction.

This system is completed by modeling the local compliance of the vessels, a state equation:

$$\sigma = E \frac{\Delta A}{2A_0}.$$
 (4)

where $\Delta A = A - A_0$, with A_0 the cross sectional area at rest, and *E* the coefficient of elasticity.

Applying a singular perturbation technique to the above equations, ABP can be represented as a KdVE as follows [6], [14]:

$$P_Z + d_0 P_T + d_1 P P_T + d_2 P_{TTT} = 0, (5)$$

where the equation coefficients are defined by:

$$d_0 = \frac{1}{c_0}, d_1 = -\left(\alpha + \frac{1}{2}\right) \frac{1}{\rho c_0^3}, d_2 = -\frac{\rho_W h_0 R_0}{2\rho c_0^3} \tag{6}$$

and the constant $c_0 = \sqrt{\frac{Eh_0}{2\rho R_0}}$ determines the typical Moens-Korteweg velocity of a wave propagating in an elastic tube, when all nonlinear terms are neglected.

Equation (5) admits a multi-soliton solution [13]. Particularly the 2-soliton analytical solution can be expressed as:

$$P(Z,T) = 12 \frac{d2}{d1} \frac{a_1^2 f_1 + a_2^2 f_2 + 2\left(\frac{a_1 - a_2}{a_1 + a_2}\right)^2 (a_2^2 f_2 f_1^2 + a_1^2 f_1 f_2^2)}{(1 + f_1 + f_2 + \left(\frac{a_1 - a_2}{a_1 + a_2}\right)^2 f_1 f_2)^2}$$

where $f_j(\mathbf{Z}, \mathbf{T}) = \exp(-a_j \left(T - S_j - Z \left(do + a_j^2 d2\right)\right))$

B. Numerical Scheme

Computer simulations of solitons propagation and their interaction result in an elevated computational cost. Therefore, spectral and pseudospectral numerical schemes are recommended in the numerical integration of the KdVE [15]. In this sense, appropriate methods for this numerical integration were discussed in [16], where the exponential

time differencing (ETD) method with a 4th order Runge-Kutta (ETDRK4) emerged as the best option [17], [18].

The ETD method proposed by [17] is defined as follows. If the equation (5) is written as

$$u_t + L(u) + N(u) = 0,$$
 (6)

where L is a linear operator and N is a nonlinear operator, with periodic boundary conditions. Applying the Fourier transform to (6), and then multiplying by e^{Lt}

$$\hat{u}_t e^{Lt} + L\hat{u}e^{Lt} + N(\hat{u})e^{Lt} = 0.$$
(7)

If a change of variables is performed:

$$v = \hat{\mathbf{u}}e^{Lt}, v_t = \hat{\mathbf{u}}_t e^{Lt} + L\hat{\mathbf{u}}e^{Lt}$$
(8)

replacing (8) in (7)

$$v_t + N(\hat{\mathbf{u}})e^{Lt} = 0. \tag{9}$$

Discretizing (9) in time and performing the integration over a single time step (Δt), the following expression is obtained

$$u_{n+1} = e^{L\Delta t} u_n + \int_0^{\Delta t} e^{-L\tau} N(u(t_n + \tau), t_n + \tau) d\tau \quad (10)$$

where Δt is the time step for the numerical integration. The expression (10) is exact, and the various order ETD schemes arise from the manner in which this integral is solved. In the present work, in order to have the best approximation [16]the integral in (10) was approximated by a fourth order Runge Kutta as stated in [16].

III. NUMERICAL EXPERIMENTS

In order to characterize the pressure pulse propagation, a discrete multi-segmented conduit was proposed. Arterial wall mechanical parameters were obtained from existing b) literature [19] and assigned to each individual segment. The numerical model was developed starting at the aortic arch, and ending at the femoral artery (Table I). Gravitatory effects were neglected assuming supine position. Aortic pressure waveform was described using a two-soliton solution as initial condition. For a continuity solution, the output state of each simulated segment was used as the initial condition of the next. Additionally, the influence of the pressure of bifurcations was considered as a small loss of pressure at the end of each conduit [20].

Due to wall thickness, vessel radius and elasticity variations, maximum values of velocity and pressure are expected to be modified as can be observed in arterial tree. Another phenomenon to be evaluated is the separation of the two initial solitons due to the difference in their velocities.

TABLE I. SEGMENTS

Segment	Length L (cm)	Radius R (cm)	Wall Thickness h (cm)	E x10 ⁶ dyn/cm ²
Aortic Arch	3.9	1.07	0.127	4
Thoracic Aorta	5.2	1.00	0.120	4
Thoracic Aorta	5.2	0.95	0.116	4
Thoracic Aorta	5.2	0.95	0.116	4

Segment	Length L (cm)	Radius R (cm)	Wall Thickness h (cm)	E x10 ⁶ dyn/cm ²
Abdominal Aorta	5.3	0.87	0.108	4
Abdominal Aorta	5.3	0.57	0.080	4
Abdominal Aorta	5.3	0.57	0.080	4
Common Iliac	5.8	0.52	0.076	4
External Iliac	8.3	0.29	0.055	4
External Iliac	6.1	0.27	0.053	4
Femoral Artery	12.7	0.24	0.050	8
	1	1	Va	lues taken from

IV. RESULTS

The two-soliton analytical solution used as the initial condition for the numerical modeling can be observed in Figure 1 resembling a typical pressure waveform at the aortic arch.



Figure 1. Pressure initial condition for simulations (Aortic Arch, contiuous line). Final state of simulation (femoral artery, dotted line).

The simulated evolution of the pressure wave trough the discrete multi-segmented conduit structure (almost 70 cm of total length) can be seen in Figure 2. The observed waveforms resemble the behavior shown in [3], obtained from experimental data.

As expected, peaking and steepening phenomena during the wave propagation were visualized (Figure 1, dotted line). Additionally, the separation of the solitary waves was verified. Three main propagation sections can be also differentiated. The first one from thoracic to abdominal aorta (approx. 25cm), the second, from abdominal aorta to iliac artery (approx. 40cm) and the last one, from iliac to femoral arteries. For each section, the relationship between the vessel radius and the wall thickness remained almost constant. Finally, the foot to foot difference between simulated aortic and femoral waves was assessed in 0.12s for the entire conduit of 68.3cm. The obtained mean pulse wave velocity corresponds to 5.69m/s, which is in agreement with physiological ranges [19].



Figure 2. Pressure waveform traveling from the heart to femoral artery

V. DISCUSSION

In the present work, ABP propagation trough the arterial tree was numerically simulated by a nonlinear dynamical system. For this purpose, a quasi 1D model of the Navier Stokes equation for the boundary layer of a thin nonlinear tube of elastic wall was implemented. A discrete multi-segmented conduit was proposed, where the obtained KdVE was numerically integrated for each segment with specific values of wall thickness, elasticity and vessel radius. Obtained results showed a waveform behavior (peaking and steepening phenomena) that can be compared to experimental data [3]. In this sense, the assumption that the pressure wave may be represented as a combination of solitons constitutes an interesting approach. Taking into account that the cardiac muscle exhibits low mechanical efficiency values [21], and considering that the cardiovascular system constitutes a highly complex structure, the presence of solitons waves seems to be a natural choice for the propagation phenomena.

The main property of the developed model is that it allows the evaluation of regime transitions (changes in structure) between contiguous segments, which cannot be obtained by means of the analytical solution alone. Moreover, complex multi-soliton solutions can be implemented [13] in a simple manner. Furthermore, structural changes in arterial wall mechanics as a consequence of aging or the presence of a vascular disease (such as atherosclerosis) could be analyzed.

VI. CONCLUSION

In the present study, a conceptual model of arterial tree based on solitons by compartments was proposed, where ABP propagation phenomenon was quantitatively reproduced.

Further studies are needed in order to improve the complexity of the model where other physiological and structural parameters could be considered.

REFERENCES

 J. L. Megnien, A. Simon, M. Lemariey, M. C. Plainfossé, and J. Levenson, "Hypertension promotes coronary calcium deposit in asymptomatic men," Hypertension, vol. 27, no. 4, pp. 949-954, Apr. 1996.

- [2] F. N. van de Vosse and N. Stergiopulos, "Pulse Wave Propagation in the Arterial Tree," Annual Review of Fluid Mechanics, vol. 43, no. 1, pp. 467–499, 2011.
- [3] F. Liang, S. Takagi, R. Himeno, and H. Liu, "Multi-scale modeling of the human cardiovascular system with applications to aortic valvular and arterial stenoses," Med Biol Eng Comput, vol. 47, no. 7, pp. 743– 755, Jul. 2009.
- [4] A. Benetos, F. Thomas, L. Joly, J. Blacher, B. Pannier, C. Labat, P. Salvi, H. Smulyan, and M. E. Safar, "Pulse pressure amplification a mechanical biomarker of cardiovascular risk," J. Am. Coll. Cardiol., vol. 55, no. 10, pp. 1032–1037, Mar. 2010.
- [5] N. Westerhof, J.-W. Lankhaar, and B. E. Westerhof, "The arterial Windkessel," Med Biol Eng Comput, vol. 47, no. 2, pp. 131–141, Feb. 2009.
- [6] E. Crepeau and M. Sorine, "Identifiability of a reduced model of pulsatile flow in an arterial compartment," in 44th IEEE Conference on Decision and Control, 2005 and 2005 European Control Conference. CDC-ECC '05, 2005, pp. 891–896.
- [7] R.C. Cascaval, "Variable Coefficient KdV Equations and Waves in Elastic Tubes", Evolution Equations. CRC Press, Vol 234, 57-69, 2003.
- [8] L. Formaggia, F. Nobile, A. Quarteroni, A. Veneziani. "Multiscale modelling of the circulatory system: a preliminary analysis". Comput Visual Sci 2:75-83, 1999.
- [9] M. Zamir. "The Physics of Pulsatile Flow". New York: Springer Verlag, 2000.
- [10] S. Yomosa, "Solitary Waves in Large Blood Vessels," Journal of The Physical Society of Japan - J PHYS SOC JPN, vol. 56, no. 2, pp. 506– 520, 1987.
- [11] H. Demiray, "Nonlinear waves in a viscous fluid contained in a viscoelastic tube," Z. angew. Math. Phys., vol. 52, no. 6, pp. 899–912, Nov. 2001.
- [12] J. C. Misra and M. K. Patra, "A study of solitary waves in a tapered aorta by using the theory of solitons," Computers & Mathematics with Applications, vol. 54, no. 2, pp. 242–254, Jul. 2007.
- [13] T.-M. Laleg, E. Crépeau, and M. Sorine, "Separation of arterial pressure into a nonlinear superposition of solitary waves and a windkessel flow," Biomedical Signal Processing and Control, vol. 2, no. 3, pp. 163–170, Jul. 2007
- [14] H. Demiray, "Head-on collision of solitary waves in fluid-filled elastic tubes," Applied Mathematics Letters, vol. 18, no. 8, pp. 941–950, Aug. 2005.
- [15] P. G. Drazin and R. S. Johnson, Solitons: An Introduction, 2nd ed. Cambridge University Press, 1989.
- [16] M. R. Alfonso and W. E. Legnani, "A Numerical Study for Improving Time Step Methods in Pseudospectral Schemes Applied to the Korteweg and De Vries Equation", Mecánica Computacional, vol. XXX, pp. 2763–2775, 2011.
- [17] S. M. Cox and P. C. Matthews, "Exponential time differencing for stiff systems," J. Comput. Phys., vol. 176, no. 2, pp. 430–455, Mar. 2002.
- [18] A.-K. Kassam and L. N. Trefethen, "Fourth-Order Time-Stepping for Stiff PDEs," SIAM Journal on Scientific Computing, vol. 26, p. 1214, 2005.
- [19] A. P. Avolio, "Multi-branched model of the human arterial system," Med. Biol. Eng. Comput., vol. 18, no. 6, pp. 709–718, Nov. 1980.
- [20] W. Duan, Y. Shi, X. Hong, K. Lü, and J. Zhao, "The reflection of soliton at multi-arterial bifurcations and the effect of the arterial inhomogeneity," Physics Letters A, vol. 295, no. 2–3, pp. 133–138, Mar. 2002.
- [21] G. H. ten Velden, G. Elzinga, and N. Westerhof, "Left ventricular energetics. Heat loss and temperature distribution of canine myocardium," Circulation Research, vol. 50, no. 1, pp. 63–73, Jan. 1982.