PAPER • OPEN ACCESS

Arterial pulse pressure amplification described by means of a nonlinear wave model: characterization of human aging

To cite this article: M Alfonso *et al* 2016 *J. Phys.: Conf. Ser.* **705** 012029

Arterial pulse pressure amplification described by means of a nonlinear wave model: characterization of human aging

M Alfonso[1], **L Cymberknop**[1], **R Armentano**[1, 2,] **F Pessana**[1, 2,] **S Wray**[1], **W Legnani**[1]

[1] Buenos Aires Regional Faculty and from School of Advanced Studies in Engineering Sciences, National Technological University, Buenos Aires, Argentina
[2] Faculty of Engineering, Exact and Natural Sciences, Favaloro University, Buenos Aires, Argentina.

Abstract. The representation of blood pressure pulse as a combination of solitons captures many of the phenomena observed during its propagation along the systemic circulation. The aim of this work is to analyze the applicability of a compartmental model for propagation regarding the pressure pulse amplification associated with arterial aging. The model was applied to blood pressure waveforms that were synthesized using solitons, and then validated by waveforms obtained from individuals from differentiated age groups. Morphological changes were verified in the blood pressure waveform as a consequence of the aging process (i.e. due to the increase in arterial stiffness). These changes are the result of both a nonlinear interaction and the phenomena present in the propagation of nonlinear mechanic waves.

1. Introduction

Pulse Pressure Amplification (PPA) is conventionally understood in clinical practice as an increase in the entire amplitude of Pulse Pressure (PP) as it travels distally. By definition, it is better seen as a distortion than an amplification, translated into an alteration of the waveform morphology. Aging is the main non-modifiable factor associated with a decreased PPA. Additionally, PPA changes in subjects with traditional cardiovascular risk factors [1]. After reaching the age of 60, a substantial decrease in diastolic pressure and an increase in systolic pressure as a consequence of a progressive increase of arterial stiffness (AS) are observed [2]. This is the result of a greater load and demand of oxygen in the ventricle, together with an arterial degenerative process caused by the breaking of arterial wall elastin fibers [3]. This elasticity loss is responsible for an increment of the propagation velocity of Blood Pressure (BP) pulse waves along the arterial conduits. Furthermore, BP values measured in peripheral sites (PBP), i.e. brachial or femoral arteries, overestimate the value of the central blood pressure (CBP), measured in the aorta. The amplification of PBP and CBP, is greater at low propagation velocities and decreases with aging [1].

BP representation through nonlinear waves called "solitons" was introduced initially by Yomosa et al [4]. These waves propagate with no changes in their structure and interact with each other while maintaining their original properties. The nonlinear representation of the overlap of two or three solitons for BP waves is described in detail by Laleg et al [5]; mathematically, this behavior is described using the Korteweg and DeVries equation (KdV). This representation of pressure pulse waves as a combination of solitons, captures many of the phenomena observed in BP propagation,

---

[1]   Manuel Alfonso. malfonso@frba.utn.edu.ar.

such as peaking (increase in amplitude), steepening (decrease in width) and the variation of wave propagation velocity.

The aim of this work is to analyze the applicability of a compartmental model for BP propagation previously used by our group [6], regarding PPA changes associated with arterial aging. Firstly, PPA phenomena will be described based on a synthesized soliton combination. Secondly, the effects of increasing vascular stiffness on the PPA will be tested on the model. Finally, the system will be validated with data from individuals categorized in age groups.

2. Materials and Methods

Our group has previously proposed the use of a compartmental model of the arterial tree [6] from which simulations of nonlinear wave propagation can be performed throughout a path of different arterial segments. Each of the segments is considered as a thin-walled elastic tube, where BP pulse wave (P) behavior is described by the partial differential equation obtained by Crepeau et al [7]:

$$P_Z + d_0 P_T + d_1 P\, P_T + d_2 P_{TTT} = 0, \tag{1}$$

where Z and T are the variables of space and time respectively, P_Z and P_T are the space and time partial derivatives, and:

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

$$c_0 = \sqrt{\frac{E h_0}{2\rho R_0}}, \tag{3}$$

where c_0 is the Moens-Korteweg propagation velocity in an elastic tube, ignoring nonlinear effects; E is the elastic modulus; h_0 is the initial thickness; R_0 is the initial radius and ρ_w is the density of the arterial wall, respectively. Finally, ρ is the blood density and α is a nonlinearity coefficient. The numerical evolution was performed by means of a pseudo-spectral high order approximation method developed by Cox et al [8], whose properties were previously evaluated by our group [9].

1.1. Simulation using compartmental modelling

The considered arterial path for BP propagation started at the aortic arch and continued with the subclavian, axillary and brachial artery segments, reaching the radial artery [6]. For the analysis of the model behavior, a synthesized BP wave was defined by combining two solitons, consistent with the normal BP values for an average adult (120/80 mmHg for systolic/diastolic pressure, and a heart period between 0.7 and 0.8 seconds). The resulting wave can be expressed as follows [7]:

$$P(Z,T) = 12\frac{d2}{d1}\frac{a_1^2 f_1 + a_2^2 f_2 + 2\left(\frac{a_1 - a_2}{a_1 + a_2}\right)^2 (a_2^2 f_2 f_1^2 + a_1^2 f_1 f_2^2)}{\left(1 + f_1 + f_2 + \left(\frac{a_1 - a_2}{a_1 + a_2}\right)^2 f_1 f_2\right)^2} \tag{4}$$

$$f_j(Z,T) = \exp-\left(a_j\left(T - S_j - Z(do + a_j^2 d_2)\right)\right), \tag{5}$$

where a_j and s_j are the amplitude and position of each soliton. The AS values were adjusted in order to observe the variations in BP, in terms of peaking and steepening phenomena. As established in the previous section, PPA appears to be affected by vascular aging, showing a smaller increase in the systolic peak of the PBP with respect to the CBP systolic peak. In this sense, it is worth mentioning that the diastolic pressure has been considered as a constant value along the arterial system.

1.2. Validation

The model was validated using BP waveforms that belong to two well-differentiated age groups. Radial artery BP was measured, using the tonometry technique (Millar Inc., Houston, Texas, USA), in 8 subjects (ages 20 to 29 years [n=4] and ages 50 to 69 [n=4]) who had no prior cardiovascular risk factors. The waveforms were obtained in basal position and calibrated using sphygmomanometric

measurements. CBP was determined by means of a transfer function using a previously validated algorithm (SphygmoCor, Atcor Medical, Illinois, USA).

For all individuals (young and adults), CBP wave was numerically propagated through the mentioned arterial path. The original AS values were iteratively adjusted, in order to obtain the best fit of the observed PPA, with a maximum error of 1 mmHg. This allowed analizing the ability of the model to reproduce the PPA variations generated in the BP pulse wave propagation as a result of aging. Furthermore, in order to compare the morphology between simulated and measured PBP waveforms, normal mean square error (NMSE) was calculated. Finally, data were expressed as mean ± standard deviation. An unpaired Student-t test was adopted, where a null hypothesis probability of $p<0.05$ was considered as statistically different.

3. Results
Figure 1 shows the changes in PPA for a synthetic simulated pressure wave in relation to the variations of AS. As it can be observed, the BP wave morphology (proximal to the heart) is affected by the peaking and steepening phenomena when arriving to the periphery.
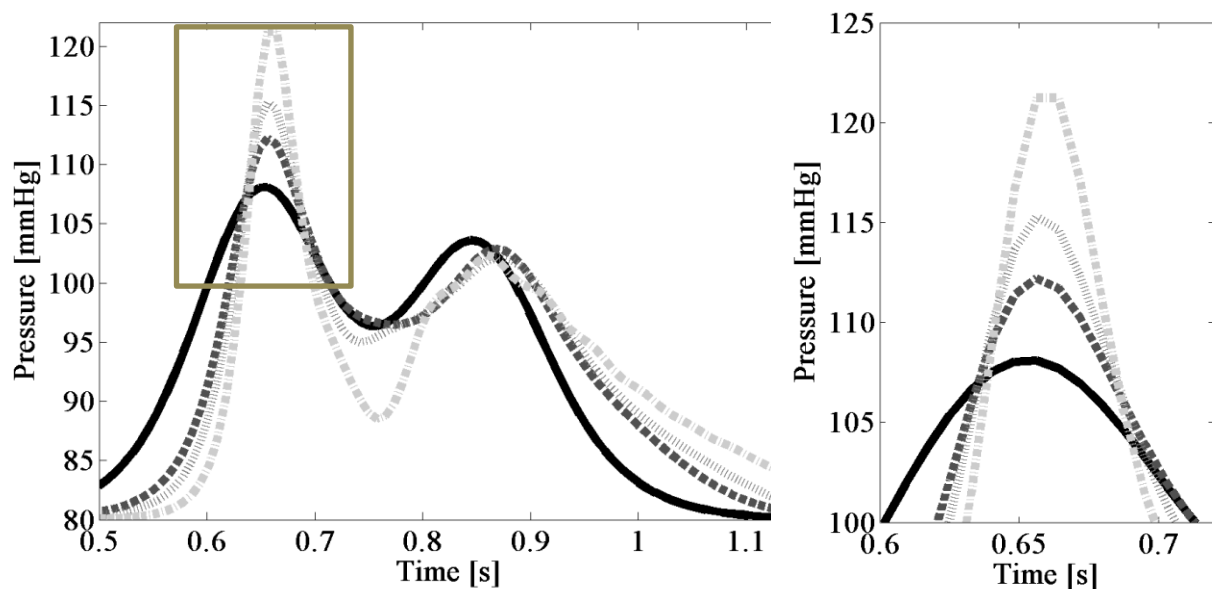

Figure 1. Changes in the synthesized blood pressure (BP) as a result of the application of the model and the increase in the stiffness modulus. Central BP (—), radial BP with initial stiffness (‑ • ‑), radial BP with stiffness increased by 50% (• • •), radial BP with stiffness increased by 100% (‑ ‑ ‑).|

Measured values of systolic CBP and PBP and calculated PPA are shown in Table 1. AS_{it}/AS constitutes the relation between the original AS [10] and the adjusted AS obtained as a consequence of the iterations (AS_{it}). The calculated normalized mean square error (NMSE) between the measured and the obtained waveforms is shown in the last column.

Table 1. Calculated parameters: Systolic CBP (SCBP), Systolic PBP (SPBP), pulse pressure amplification (PPA), increment in [10] AS

Individual	SCBP	SPBP	PPA	ASit/AS	NMSE
Young_01	98.64	117.53	18.89	1.00	0.77
Young_02	105.62	126.7	21.08	1.625	0.80
Young_03	108.5	122.16	13.66	1.625	0.84
Young_04	86.64	109.42	22.78	1.063	0.66
Mean±SD	99.85±9.73	118.95±7.38	19.10±3.96	1.33±0.34	0.77±0.08
Adult_01	135.5	144.33	8.83	3.50	0.90
Adult_02	137.9	147.63	9.73	3.00	0.64
Adult_03	122.44	130.7	8.26	3.00	0.90
Adult_04	143.94	151.79	7.85	4.50	0.95
Mean±SD	134.95±9.06*	143.61±9.13*	8.67±0.81*	3.50±0.71*	0.84±0.14

* **P**<0.05 was considered as statistically significant

Figure 2 and Figure 3 show the measured CBP, PBP and the propagation of CPB with the corresponding adjusted AS_{inc} in two of the considered subjects. In Figure. 2, pulse propagation of CBP through the application of the model for a young individual is shown. It can be observed that the systolic peaks in comparison to the corresponding of PBP are equal. In Figure. 3, the CBP pulse of an adult individual was also propagated. For all cases, the corresponding PPA of the adult group has resulted smaller than in the young group, while the AS_{inc} became higher (Table 1).
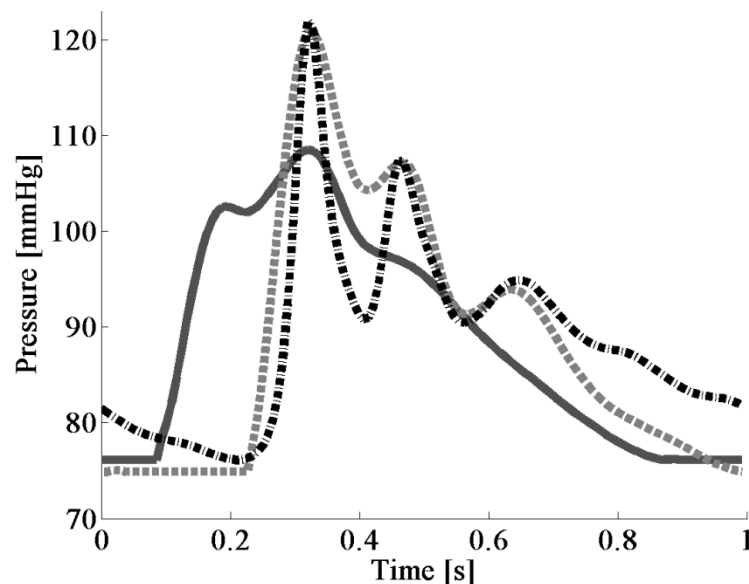

Figure 2. Young individual. Central blood pressure (BP) waveform (——). Radial BP measured for each subject (- - -). Result of the application of the model with adjusted AS_{inc}(- • -).
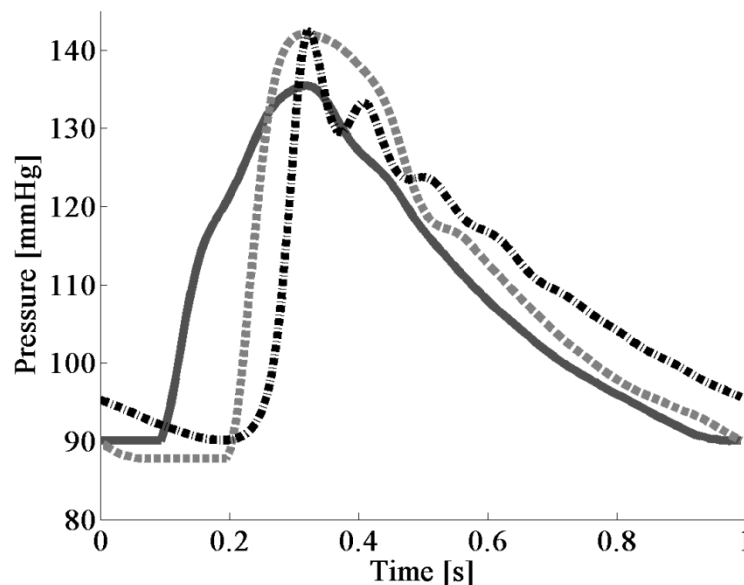
Figure 3. Adult individual. Central blood pressure (BP) waveform (——). Radial BP measured for each subject (- - -). Result of the application of the model with adjusted AS_{inc}(- • -).

4. Discussion

In this study, the compartmental model proposed in [6] –in which pressure waves were considered as a combination of solitary waves– was utilized in order to evaluate the effects of aging on BP waves, more precisely in PPA phenomena. BP waveforms were considered from its origin (CBP) as nonlinear waves, and then propagated through a defined vascular path. For our knowledge, a model based on the propagation of this type of waves for the description of the alteration of the vascular wall as a result of aging, has not been previously reported in the existing literature.

Arterial stiffening, radius dilatation and increased wall thickness are some of the well-known changes that characterize the aging of the cardiovascular system. The values shown in Table 1, in which the PPA becomes lower and AS increases as a result of aging are in accordance with previous results [11,12]. It can be observed that the AS values corresponding to an adult subject are doubled in relation with young individuals. Furthermore, in the last column, the goodness of fit provided by the NMSE is between 70-80 percent, which constitutes an acceptable waveform representation for the developed model.

The performed simulations showed that the main aspects of PPA have been emulated. It is noteworthy that, unlike the traditional approach (where the effects of PPA are described through the wave reflection phenomenon [2]), this study analyses the evolution of nonlinear waves traveling from the aortic arch, and whose interaction determines the morphology of the peripheral wave.

The morphological dependence on AS can be easily described by means of soliton theory, where waves with different amplitudes travel at different speeds, due to an amplitude-velocity relationship. In this sense, at low speeds (i.e. AS in young individuals with no vascular disease), the different solitary waves that shape the CBP are separated when reaching the periphery, where the peaking and steepening phenomena are evidenced. Increased AS, caused by aging, diminishes this separation and consequently a smaller PPA is accused.

5. Conclusion

In the present work, a nonlinear wave model was used to evaluate human aging. AS adjustments were performed to recreate morphological changes and obtained PPA variations are in accordance with previous studies. Additionally, soliton theory was used to explain changes experimented by CBP, from

a totally different approach. As a result, this model allows aging simulation and can be useful for explaining the clinical implication of PPA.

The analysis of BP morphological variations in terms of soliton wave propagation is very promising and deserves further studies.

6. References

[1]   Avolio A P, Bortel L M V, Boutouyrie P, Cockcroft J R, McEniery C M, Protogerou A D, Roman M J, Safar M E, Segers P and Smulyan H (2009). Role of Pulse Pressure Amplification in Arterial Hypertension Experts' Opinion and Review of the Data *Hypertension* **54** 375–83

[2]   Nichols W, O'Rourke M and Vlachopoulos C (2011). *McDonald's Blood Flow in Arteries, Sixth Edition: Theoretical, Experimental and Clinical Principles* (London: CRC Press)

[3]   Armentano R, Megnien J L, Simon A, Bellenfant F, Barra J and Levenson J (1995). Effects of hypertension on viscoelasticity of carotid and femoral arteries in humans *Hypertension* **26** 48–54

[4]   Yomosa S (1987). Solitary Waves in Large Blood Vessels *J. Phys. Soc. Jpn. - J PHYS SOC JPN* **56** 506–20

[5]   Laleg T-M, Crépeau E and Sorine M (2007). Separation of arterial pressure into a nonlinear superposition of solitary waves and a windkessel flow *Biomed. Signal Process. Control* **2** 163–70

[6]   Alfonso M R, Cymberknop L J, Legnani W, Pessana F and Armentano R L (2014). Conceptual model of arterial tree based on solitons by compartments *Conf. Proc. Annu. Int. Conf. IEEE Eng. Med. Biol. Soc. IEEE Eng. Med. Biol. Soc. Annu. Conf.* **2014** 3224–7

[7]   Crepeau E and Sorine M (2005). Identifiability of a reduced model of pulsatile flow in an arterial compartment *44th IEEE Conference on Decision and Control, 2005 and 2005 European Control Conference. CDC-ECC '05* 44th IEEE Conference on Decision and Control, 2005 and 2005 European Control Conference. CDC-ECC '05 pp 891–6

[8]   Cox S M and Matthews P C (2002). Exponential time differencing for stiff systems *J. Comput. Phys.* **176** 430–55

[9]   Manuel R Alfonso W E L (2011). A Numerical Study For Improving Time Step Methods In Psudospectral Schemes Applied To The Korteweg And De Vries Equation *Mecánica Comput.* **XXX** 2763–75

[10] Avolio A P (1980). Multi-branched model of the human arterial system *Med. Biol. Eng. Comput.* **18** 709–18

[11] Black J and Hastings G (1998). *Handbook of Biomaterial Properties* (Boston, MA: Springer US)

[12] Langewouters G J, Wesseling K H and Goedhard W J (1985). The pressure dependent dynamic elasticity of 35 thoracic and 16 abdominal human aortas in vitro described by a five component model *J. Biomech.* **18** 613–20