

Individualization of the parameters of the 3-elements Windkessel model using carotid pulse signal

Marek Żyliński^a, Wiktor Niewiadomski^{b,c}, Anna Strasz^{b,c}, Anna Gąsiorowska^{b,d}, Martin Berka^a, Marcel Młyńczak^a, Gerard Cybulski^{a,b*}

^aInstitute of Metrology and Biomedical Engineering, Faculty of Mechatronics, Warsaw University of Technology, Warsaw, Poland

^bDepartment of Applied Physiology, Mossakowski Medical Research Centre, Polish Academy of Sciences, Warsaw, Poland

^cDepartment of Experimental and Clinical Physiology, Medical University of Warsaw, Warsaw, Poland.

^dLaboratory of Preclinical Studies in Neurodegenerative Diseases, Department of Neurophysiology, Nencki Institute, Warsaw, Poland.

ABSTRACT

The haemodynamics of the arterial system can be described by the three-elements Windkessel model. As it is a lumped model, it does not account for pulse wave propagation phenomena: pulse wave velocity, reflection, and pulse pressure profile changes during propagation. The Modelflow© method uses this model to calculate stroke volume and total peripheral resistance (TPR) from pulse pressure obtained from finger; the reliability of this method is questioned.

The model parameters are: aortic input impedance (Z_0), TPR, and arterial compliance (C_w). They were obtained from studies of human aorta preparation. Individual adjustment is performed based on the subject's age and gender. As C_w is also affected by diseases, this may lead to inaccuracies.

Moreover, the Modelflow© method transforms the pulse pressure recording from the finger (Finapres©) into a remarkably different pulse pressure in the aorta using a predetermined transfer function — another source of error.

In the present study, we indicate a way to include in the Windkessel model information obtained by adding carotid pulse recording to the finger pressure measurement. This information allows individualization of the values of C_w and Z_0 . It also seems reasonable to utilize carotid pulse, which better reflects aortic pressure, to individualize the transfer function.

Despite its simplicity, the Windkessel model describes essential phenomena in the arterial system remarkably well; therefore, it seems worthwhile to check whether individualization of its parameters would increase the reliability of results obtained with this model.

Keywords: Windkessel model, carotid pulse, individualization, Modelflow

1. INTRODUCTION

The relationship between arterial blood flow and arterial blood pressure is complex due to features of the arterial tree's anatomy, nonlinear properties of arterial vessels' walls and blood, pulse wave propagation phenomena, and active regulation of vessel diameter. Accordingly complicated mathematical functions should be applied to obtain an adequate quantitative description of the arterial pressure-flow relationship. Too simple a model will be inaccurate, too complex – impractical, as it will require the identification of a multitude of parameters. Practical models should keep a balance between necessary complexity and the ability to obtain the values of model's parameters.

Arterial pressure can easily be measured continuously and non-invasively; continuous and non-invasive measurement of arterial flow requires trained personnel and movement of patient is prohibited. The Windkessel model allows one to calculate arterial flow from arterial pressure.

The three-elements Windkessel model

Frank [1] uses a two-elements Windkessel model to describe the arterial system. This model consists of two lumped elements, total peripheral resistance (TPR) and arterial compliance (Cw). It fairly accurately describes phenomena that take place during diastole, i.e. when the aortic valve is closed, there is no outflow from the heart, and arterial pressure decreases exponentially. This model corresponds to an electric circuit of a capacitor discharging through a resistor.

The two-element model fails to describe arterial flow during systole [2, 3]. To overcome this deficiency, aortic input impedance (Zo) has been added, yielding a three-elements Windkessel model (Fig. 1).

Wesseling et al. [4] proposed to use this model to calculate stroke volume using radial pressure. They compared the model's mean flow with thermodilution cardiac output estimation. The pooled mean difference was 7% and the SD 22%; after calibration the model difference decreased to 2%, the SD 8%. Tam et al. assessed this method positively when correction with another technique was applied [5], but other authors found the model a poor predictor of cardiac output [6, 7].

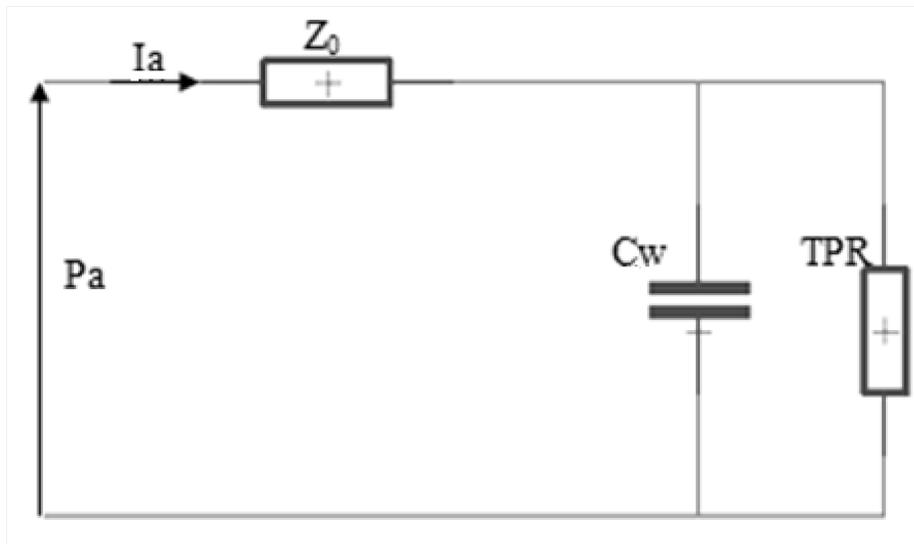


Figure 1: Three-element Windkessel model, I_a – aortic blood flow, P_a – aortic pressure, Z_0 – aortic input impedance, C_w – arterial compliance, TPR – total peripheral resistance.

2. THE RELATION BETWEEN PULSE WAVE VELOCITY (PWV) AND ARTERIAL COMPLIANCE (CW)

It has been shown that pulse pressure velocity (PWV) can provide information allowing calculation of the parameters of the three-elements model [8].

Compliance quantifies vascular ability to store blood and mechanical energy. Arterial compliance is defined as the ratio of arterial volume increase to aortic blood pressure increase:

$$C_w = \frac{\Delta V_w}{\Delta P_a} \quad (1)$$

Arterial compliance is related to pulse wave velocity (PWV) by the formula:

$$C_w = \frac{V_w}{PWV^2 * \rho} \quad (2)$$

where V_w – volume of arterial tree, ρ – blood density.

Arterial volume is calculated by the formula:

$$V_w = Q_o * L_w \quad (3)$$

where Q_o – the cross-sectional area of the aortic arch, which can be measured directly or calculated using the empirical formula [9]:

$$Q_o = A^{0.388 * BSA^{0.5}} \quad (4)$$

where A [years] – subject's age, BSA [m^2] – subject's body surface area.

BSA is calculated with the DuBois formula:

$$BSA = 0.20247 * H^{0.725} * W^{0.425} \quad (5)$$

where H [m] – height, W [kg] – body mass.

L_w is the length of the cylinder with constant cross-sectional area best matching the storage capability of all elastic arteries together and can be calculated with the empirical formula [6]:

$$L_w = 0.3 H \quad (6)$$

The blood ejected from the heart travels along the aorta as pulse wave. This means that ejected blood travels over a relatively short distance and then stops; however, it imparts its energy to an adjacent portion of blood which also travels over a relatively short distance. This sequence of events causes the pulse wave to propagate to the end of the aorta. It is energetically more advantageous to accumulate blood in a given segment of the aorta by stretching its walls than to rapidly put in motion the whole aortal volume of blood. The more distensible walls of the aorta, the more blood will be stored and the longer the recoil will be, as a consequence slowing propagation of the pulse wave.

The ratio of pulse pressure (PP) to pulsatile blood flow (I_p) is called aortic input impedance (Z_o) and can be approximated by the formula:

$$Z_o = \frac{PP}{I_p} \quad (7)$$

where PP – pulse pressure, I_p – pulsatile flow.

Aortic input impedance is related to pulse wave velocity (PWV) by the formula [8]:

$$Z_o = \frac{PWV * \rho}{Q_o} \quad (8)$$

Individualization of the Windkessel model parameters using carotid pulse pressure

The Windkessel model gives rise to the following equation:

$$C_w Z_o \frac{dI_a}{dt} + I_a \left(1 + \frac{Z_o}{TPR} \right) = C_w \frac{dP_a}{dt} + \frac{1}{TPR} P_a \quad (9)$$

where I_a [cm³/sec] – aortic flow, P_a – aortic pressure.

Individualization of the model parameters may be based on pulse wave propagation velocity. Pulse wave propagation velocity is calculated by dividing distance between measurement points by propagation time. Using formulas (2) and (8), this equation can be rewritten in such a way as to show the role of PWV:

$$\frac{V_w}{PWV \cdot Q_0} \frac{dI_a}{dt} + I_a \left(1 + \frac{PWV \cdot \rho}{Q_0 \cdot TPR} \right) = \frac{V_w}{PWV^2 \cdot \rho} \frac{dP_a}{dt} + \frac{1}{TPR} P_a \quad (10)$$

V_w , ρ , and Q_0 can be treated as constant individual parameters.

If the parameters C_w and Z_0 could be treated as constant, then a single measurement of PWV would be required. However, it is well known that they depend on blood pressure. As PWV also depends on blood pressure, the possibility of measuring PWV continuously should be considered. This would allow one to update C_w and Z_0 each heart cycle.

In equation (10), only the TPR value is not related to PWV. It is produced by blood viscosity and depends on the geometry of the vascular bed. For steady, laminar flow of Newtonian liquid through a rigid tube, the Hagen-Poiseuille law applies, and vessel resistance is given by:

$$TPR = \frac{8\eta l}{\pi r^4} \quad (11)$$

where l – length of vessel; η – blood viscosity; r – radius of vessel.

This formula reveals the dominant and key role of vessel radius for regulation of vessel resistance. However, it is nearly useless in practice as blood is a non-Newtonian fluid, arterial flow is pulsatile, blood vessels are elastic tubes connected in serial and parallel fashion and vessel radius is continuously changed by metabolic, nervous and hormonal factors.

Therefore, despite formally being a parameter, due to the impossibility of being calculated, TPR has to be estimated from measurements.

For instance, Wesseling *et al.* [4] proposed to calculate TPR for every heart cycle as the ratio of mean blood pressure to cardiac output:

$$TPR = \frac{\text{mean blood pressure}}{\text{mean blood flow}} \quad (12)$$

Mean blood pressure was obtained directly from signal; mean blood flow is taken from the model calculation from the last heart cycle. For a start value, set: mean blood pressure = 100 mmHg, mean blood flow = 3 l/min; TPR will stabilize after some heart cycles.

Assuming $I_a = 0$, equation (10) reduces to one, which describes the two-elements Windkessel model. This assumption is physiologically sound, as axial aortic flow ceases to 0 during diastole. In this case, the product of C_w and TPR is the time constant of the capacitor discharge equation:

$$\frac{dP_a}{dt} = - \frac{1}{C_w \cdot TPR} P_a = - \frac{PWV^2 \cdot \rho}{V_w \cdot TPR} P_a \quad (13)$$

Below, the solution of this equation is given by:

$$P_a(t) = P_{start} * e^{-\frac{t}{\tau}} = P_{start} * e^{-\frac{t \cdot PWV^2 \cdot \rho}{V_w \cdot TPR}} \quad (14)$$

where P_{start} – pressure at beginning of exponential decay of arterial pressure, t – time elapsed since this moment, τ – discharge time constant.

Formula (14) can in turn be expressed as:

$$TPR = \frac{\ln\left(\frac{P_{start}}{P_a(t)}\right) V_w}{t \cdot PWV^2 \cdot \rho} \quad (15)$$

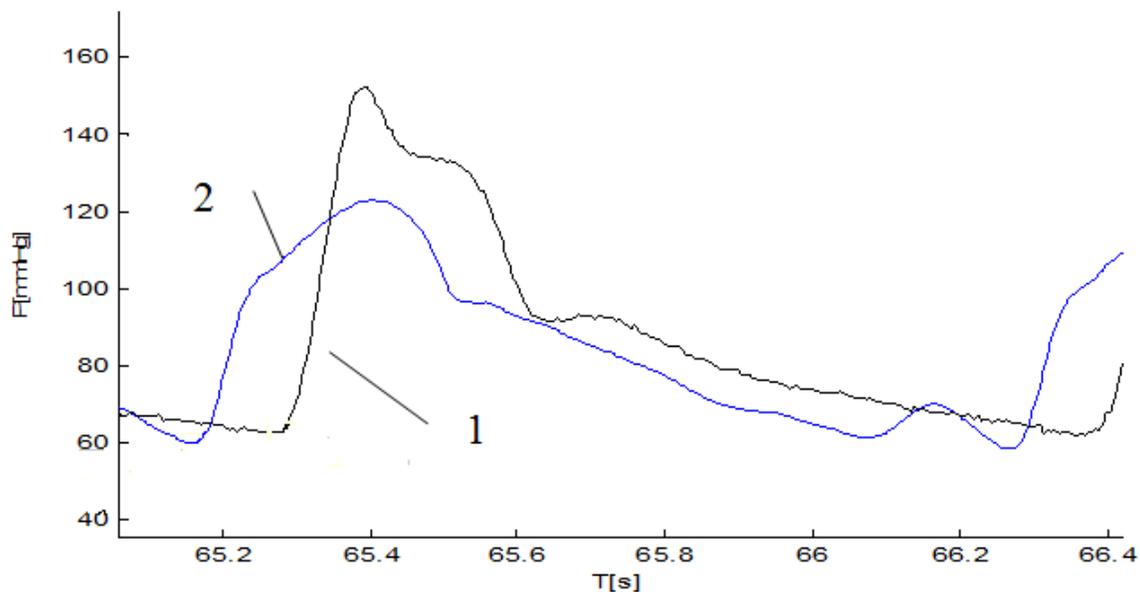


Figure 2: Difference between shapes of graphs of (1) radial and (2) carotid pulse pressure P [mmHg] over time T [s]. (Own data).

It is well known that the shape of the pulse pressure wave in the radial artery is remarkably different (Fig. 2) from that in the aorta. The three-elements Windkessel model implies usage of arterial pressure. In case of Modelflow® method, the radial pressure has to be transformed into aortic pressure using a predefined transfer function which is assumed to be the same for all subjects and therefore may be a source of error. In order to obtain shape of pulse pressure curve similar to that of aortic pressure, it seems reasonable to use pulse pressure obtained in a point as close to heart as possible. It may be reasonably assumed that the shape of the carotid pulse pressure graph is similar to the shape of aortic pressure. We may thus substitute aortic pressure with carotid pressure.

3. CONCLUSION

Despite its limitations, the Windkessel model is elegant description of phenomena in the circulatory system. The model was applied to calculate stroke volume in the Modelflow® method.

The main weakness of the Wesseling *et al.* [4] solution applied in this method is the use of non-individualized parameters. It is shown here that such individualization is possible using pulse pressure velocity. The pulse pressure velocity could be measured if, besides recording radial pressure with Portapres, carotid pressure was also recorded [11]. With these two signals, it is possible to obtain the values of C_w , Z_o and TPR, updated for every heart cycle, and to avoid using a transfer function currently necessary to convert radial pressure into aortic pressure.

ACKNOWLEDGMENTS

This work was supported 50% by part grant no. 2011/01/D/NZ7/04405 from the National Science Centre and 50% by the research program of the Institute of Metrology and Biomedical Engineering, Warsaw University of Technology

REFERENCES

- [1] Frank, O., Die Grundform des arteriellen Pulses [The basic form of the arterial pulse]. 1899 Z Biol 37:483–526

- [2] Wetterer, E., Quantitative Beziehungen zwischen Stromstärke und Druck im natürlichen Kreislauf bei zeitlich variabler Elastizität des Arteriellen Windkessels [Quantitative relationships between current and pressure in the natural cycle in time-varying elasticity of arterial air chamber]. 1940 Z Biol 100:260–317
- [3] Westerhof, N., Lankhaar, J.W., Westerhof, B.E., The arterial Windkessel. Med Biol Eng Comput. 2009 Feb; 47(2):131-41. doi: 10.1007/s11517-008-0359-2. Epub 2008 Jun 10. Review. PubMed PMID: 18543011.
- [4] Wesseling, K.H., Jansen, J.R., Settels, J.J., Schreuder, J.J., Computation of aortic flow from pressure in humans using a nonlinear, three-element model. J Appl Physiol (1985). 1993 May; 74(5):2566-73. PubMed PMID: 8335593.
- [5] Tam, E., Azabji Kenfack, M., Cautero, M., Lador, F., Antonutto, G., di Prampero, P.E., Ferretti, G., Capelli, C., Correction of cardiac output obtained by Modelflow from finger pulse pressure profiles with a respiratory method in humans. Clin Sci (Lond). 2004 Apr; 106(4):371-6. PubMed PMID: 14606953.
- [6] Azabji Kenfack, M., Lador, F., Licker, M., Moia, C., Tam, E., Capelli, C., Morel, D., Ferretti, G., Cardiac output by Modelflow method from intra-arterial and fingertip pulse pressure profiles. Clin Sci (Lond). 2004 Apr; 106(4):365-9. PubMed PMID: 14606952.
- [7] Dyson, K.S., Shoemaker, J.K., Arbeille, P., Hughson, R.L., Modelflow estimates of cardiac output compared with Doppler ultrasound during acute changes in vascular resistance in women. Exp Physiol, 95: 561–568 (2010).
- [8] Breithaupt, K., Belz, G.G., Sinn, W., Non-invasive assessments of compliance of the aortic wind-kessel in man derived from pulse pressure/storage volume ratio and from pulse wave velocity. Clin Physiol Biochem. 1992; 9(1):18-25. PubMed PMID: 1424435.
- [9] Strehler, E., Individuelle Norm der Aortenweite [Individual standard of aortic width]. Z Krsllfsschg 54:571-578 (1965)
- [10] Gauer, O., Haemodynamic des arteriellen systems [Haemodynamic of the arterial system]. pp 164-222 in Herz und Kreislauf Eds. Trautwein W. Gauer O.H.Koepchen H.P. Urban& Schwarzenberg, Muenchen – Berlin – Wien-1972
- [11] Chirinos, J.A., Arterial stiffness: basic concepts and measurement techniques. J Cardiovasc Transl Res. 2012 Jun; 5(3):243-55. doi: 10.1007/s12265-012-9359-6. Epub 2012 Mar 24. Review. PubMed PMID: 22447229.

LIST OF SYMBOLS

1. Pa [mm Hg] – aortic pressure
2. PP [mm Hg] – pulse pressure
3. Ia [cm³/s] – aortic blood flow
4. Ip [cm³/s] – pulsatile flow
5. Zo [mm Hg s/ cm³] – aortic input impedance
6. Cw [cm³/ mmHg] – arterial compliance
7. TPR [mm Hg s/ cm³] – total peripheral resistance
8. Vw [cm³] – arterial volume
9. PWV [cm/s] – pulse wave velocity
10. ρ [g/cm³] – blood density
11. Qo [cm²] – cross-sectional area of aortic arch
12. Lw [cm] – length of cylinder with constant cross-sectional area best matching total storage capability of all elastic arteries.
13. A [years] – age
14. H [m] – height
15. W [kg] – body mass
16. BSA [m²] – body surface area
17. η [Pa*s] – blood viscosity
18. l [cm] – length of vessel
19. r [cm] – radius of vessel