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# Influence of an Asymptotic Pressure Level on the Windkessel Models of the Arterial System

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**Abstract:** Windkessel models are lumped-parameter models of the arterial system that describe the dynamic relation between blood flow and pressure in the aorta. Despite their simplicity, they are used in many applications including methods for the non-invasive stratification of cardiovascular risk. However, even though they have been studied extensively, there is still disunity regarding the question if pressure should be modelled as resulting solely from the ejection of the heart or if an asymptotic pressure level should be included which is independent from cardiac beating. The aim of this work is to mathematically analyse the influence of such a pressure level $P_\infty$ on the model behaviour of the four most widely used Windkessel models (two-, three- and four-element Windkessel, the latter in a series as well as a parallel configuration). Therefore, the model equations are introduced and Fourier analysis is performed to clarify the impact of $P_\infty$ on the other parameters. Then, a typical aortic flow wave is used as input to the models and simulation experiments with varying values of $P_\infty$ are performed. Theoretical considerations as well as numerical results show that, in all four models, including $P_\infty$ mainly affects the diastolic part of the modelled pressure wave and could potentially improve the fitting performance during diastolic decay. However, further research is needed to clarify the physiological interpretation of $P_\infty$ as well as its appropriate size.



*Keywords:* Modelling, Physiological models, Fourier transforms, Parameters, Differential equations, Windkessel models, Arterial system

## 1. INTRODUCTION

The mechanisms shaping pressure and flow in the arterial system have been studied since the first measurements were performed almost three centuries ago. The german physiologist and anatomist Ernst Heinrich Weber was one of the first to propose an explanation based on the Windkessel effect (Hildebrandt and Weber, 1831): during systole, when the heart is ejecting, the walls of the large elastic arteries such as the aorta expand and act as a reservoir storing blood. When the pressure decreases during diastole, the walls contract again thereby enabling a steady blood flow during the entire cardiac cycle.

In 1899, this concept was formalized in a mathematical model by Otto Frank (Westerhof et al., 2009). His so called two-element Windkessel consisted of the arterial compliance, describing the distensibility of the large arteries, and the peripheral resistance, representing the total resistance of the arterial system mostly caused by the small arteries and arterioles (Kokalari et al., 2013). The number of parameters in Frank's Windkessel was later extended to improve the accuracy of the modelled pressure waves, resulting, among many more, in the very popular three-element Windkessel as well as different configurations of four-element Windkessel models (Burattini and Gnudi, 1982; Segers et al., 2008).

Because of their simple yet (to a certain degree) accurate description of the arterial system, these models are still used in many applications. Especially, since all parameters have a physiological meaning, methods to determine characteristics of the arterial system of a specific person are often based on Windkessel models (Stergiopulos et al., 1999a; Liu et al., 1986) and they have therefore become an important tool for the non-invasive stratification of cardiovascular risk (Wassertheurer et al., 2008; Hametner et al., 2012).

However, even though the Windkessel models have been investigated extensively, there is still disunity regarding the question if an asymptotic pressure level should be included, i.e. a pressure that is not caused by the ejecting heart itself but is sustained by the vascular system. The aim of this work is to investigate the influence of such a pressure level on the behaviour of the most commonly used Windkessel models.


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## 2. METHODS

### *2.1 Two-element Windkessel*

Windkessel models describe the dynamic relation between pressure and flow in the arterial system assuming that the system is in steady state, i.e. that both pressure and flow are periodic functions. They are so called lumped-parameter models, which means that the whole arterial system is modelled as one compartment and space is therefore not considered. The ejection pattern of the left ventricle represents the input, the resulting pressure wave the output of the models.

Otto Frank originally considered two parameters to describe the mechanisms in the arterial system: the arterial compliance $C_a$ and the peripheral resistance $R_p$. The mathematical formulation of his model is based on conservation of mass, i.e. the change of blood volume $V$ contained in the compartment must be equal to the amount of blood flowing in from the heart $q_{in}$ minus the amount of blood leaving the system to the periphery $q_{out}$, see (1).

$$\frac{dV}{dt} = q_{in} - q_{out} \tag{1}$$

The parameter $C_a$ specifies the elasticity of the arteries and is defined as the change of volume $V$ arising from a change in pressure $p_{wk}$:

$$C_a = \frac{dV}{dp_{wk}} \tag{2}$$

$R_p$ describes the resistance the blood has to overcome in order to flow through the periphery, i.e. to leave the compartment. According to Ohm's law it relates pressure $p_{wk}$ to outflow $q_{out}$:

$$p_{wk} = R_p q_{out} \tag{3}$$

These equations can now be merged into one by first using the chain rule of differentiation and (2)

$$\frac{dV}{dt} = \frac{dV}{dp_{wk}}\frac{dp_{wk}}{dt} = C_a\frac{dp_{wk}}{dt} \tag{4}$$

and then substituting (4) and (3) in (1) which results in the following linear ordinary differential equation (ODE) for the pressure $p_{wk}$.

$$\frac{dp_{wk}}{dt}(t) + \frac{1}{R_p C_a}p_{wk}(t) = \frac{1}{C_a}q_{in}(t) \tag{5}$$

$p_{wk}$ represents the pressure resulting from the ejection of blood into the arterial system. The aortic pressure $p$ itself is then given by $p_{wk}$ plus an asymptotic pressure level $P_\infty$ which is maintained by the vascular system (at least for some time) even without activity of the heart.

*Model 1. 2-element Windkessel (WK2)*
For $R_p$, $C_a > 0$ and $P_\infty \geq 0$, the aortic pressure $p(t)$ corresponding to the flow $q_{in}(t)$ is modelled as

$$p(t) = p_{wk}(t) + P_\infty \tag{6}$$

with $p_{wk}(t)$ satisfying the ODE given in (5).

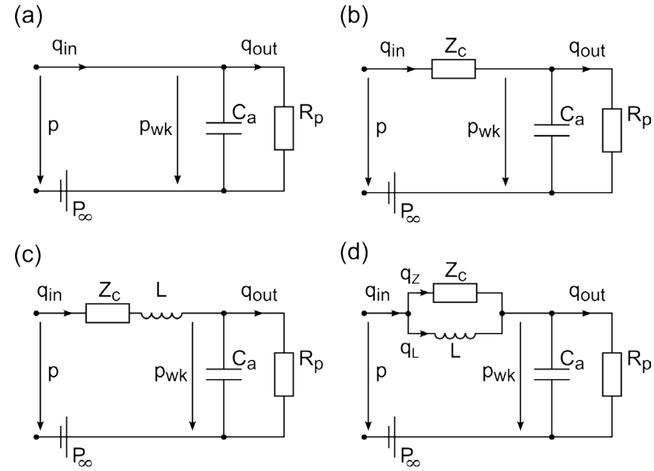


Fig. 1. Electrical analogue representation of the 2-element (a), 3-element (b) and the two configurations of the 4-element (serial c, parallel d) Windkessel models. Pressure corresponds to voltage, blood flow to current flow. Characteristic impedance $Z_c$ and peripheral resistance $R_p$ are represented by resistors, total arterial compliance $C_a$ by a capacitor, inertance $L$ by an inductor and asymptotic pressure $P_\infty$ by a pressure source.

An electrical analogue representation of this model is shown in fig. 1a. In order to solve the ODE (5) for a given flow profile in the time domain, an initial condition $p_{wk}(0) = p(0) - P_\infty$ has to be specified.

### *2.2 Three- and four-element Windkessel*

Comparison of the aortic pressure $p$ derived with the WK2 to measurements showed that the modelled pressure rise in the beginning of systole was too slow. Therefore the WK2 was extended by a third element, the characteristic impedance $Z_c$. $Z_c$ takes into account the compliance and inertance in the ascending aorta (Westerhof et al., 2009) and acts as an extra resistance on $q_{in}$ (see fig. 1b).

*Model 2. 3-element Windkessel (WK3)*
For $R_p$, $C_a > 0$ and $Z_c, P_\infty \geq 0$ the aortic pressure $p(t)$ corresponding to the flow $q_{in}(t)$ is modelled as

$$p(t) = Z_c q_{in}(t) + p_{wk}(t) + P_\infty \tag{7}$$

with $p_{wk}(t)$ satisfying the ODE given in (5).

Efforts to further improve the model resulted in the inclusion of a fourth element, the hydraulic inductance $L$, which represents the total arterial inertance (Stergiopulos et al., 1999b). $L$ specifies the augmentation of pressure caused by a change in flow or, in other words, by an acceleration or deceleration of blood. Two different configurations of this model exist, once $Z_c$ and $L$ are, electrically spoken, connected in series (WK4s) and once in parallel (WK4p), see fig. 1c, d. In the first case, the total pressure $p$ increases linearly with changes in $q_{in}$, whereas the second case is a little more complex and will be discussed in the paragraphs following the formulation of the WK4s.