SOLITON-WINDKESSEL ARTERIAL MODEL

Emmanuelle Crépeau

Résumé. — In this work, we propose a new model of arterial blood pressure, combining differential equations (windkessel terms) and partial differential equations (Korteweg-de Vries terms). The objective of this study is to define some characteristics of the vessels obtained from non-invasive measurements.

This is a jointed work with Michel Sorine and Taous Meriem Laleg (Inria Rocquencourt).

1. Introduction

The cardiovascular system can be seen as consisting of the heart, a complex double chamber pump, pumping the 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. Such reduced mathematical models are usually a set of coupled ordinary differential equations, each of them representing the input-output behaviour of a subsystem : conservation law of the blood quantity for short time-intervals and specific behaviour laws. They can be used for understanding the global hydraulic behaviour of the system during a heartbeat. They can also be used to study the short-term control by the autonomous nervous system [**4, 3, 2**].

The pulse pressure plays an important rule in the circulatory system. It undergoes an increase in its amplitude and a decrease in its pulse width when it propagates along the arterial tree. These observed phenomena are called "peaking" and "steepening" and are usually explained by the existence of reflected waves. The pulse pressure is decomposed into forward and backward waves associated to some linearized models. In this article we propose to decompose the solution into some nonlinear waves.

2. Governing equations.

In this section, we suppose that for normal space and time scales, the windkessel model predomines but, for small time and small space scales, there appears a boundary layer where the windkessel model is no more convenient. This ansatz is used in

singular perturbation computations to develop a corrector of the motion of the fluid in this boundary layer.
The idea of a boundary layer where a corrector of the motion of the fluid satisfies a KdV equation is a conjecture to represent the wave phenomena rather fast when compared to the windkessel effect. We derive formally the equations satisfied by this corrector. We still need to prove that the solutions converge (in a sense to be defined) to the solutions of equations (1), (2), (3) and (4).
We suppose that the arteries can be identified with an elastic tube, and blood flow is supposed to be an incompressible fluid.
Thus, we consider a one dimensional elastic tube of mean radius R_0. The Navier Stokes equation can read as

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

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

where, $A(T, Z)$ is the cross-sectional area of the vessel, $Q(T, Z)$ is the blood flow and $P(T, Z)$ is the blood pressure. Moreover ρ is the blood density and ν a coefficient of viscosity of blood.
Furthermore, the motion of the wall satisfies, (see for example [**6**])

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

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

Remark : Usually the term $\frac{\rho_w h_0 R_0}{A_0} A_{TT}$ is neglected because A_{TT} is small.

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

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

where $\Delta A = A - A_0$, with A_0 the cross-sectional area at rest, and E is the coefficient of elasticity. By hypothesis, let $\epsilon = \left(\frac{R_0}{L}\right)^{2/5}$.

$$\frac{\rho_w h_0 R_0}{\rho L^2} = \frac{\rho_w}{\rho}\frac{h_0}{R_0}\frac{R_0^2}{L^2} = O(\epsilon^5) = \lambda\epsilon^5.$$

We first adimensionalize the variables to get a quasi 1D Navier-Stokes equation,

$$a_t + q_z = 0, \tag{5}$$

$$q_t + \left(\frac{q^2}{1+a}\right)_z + (1+a)p_z = -\eta\frac{q}{1+a}, \tag{6}$$

$$\lambda\epsilon^5 a_{tt} + a = p. \tag{7}$$

Then, we decompose formaly the solutions in series with slow and fast terms, namely,

$$a^\epsilon(t,z) = \epsilon a_1(t,z,\frac{t}{\epsilon},\frac{z-t}{\epsilon^2}) + ...$$
$$p^\epsilon(t,z) = \epsilon p_1(t,z,,\frac{t}{\epsilon},\frac{z-t}{\epsilon^2}) + ...$$
$$q^\epsilon(t,z) = \epsilon q_1(t,z,,\frac{t}{\epsilon},\frac{z-t}{\epsilon^2}) + ...$$

In this type of method we introduce the following notations, with $\tau = \frac{t}{\epsilon}$, $\xi = \frac{z-t}{\epsilon^2}$.

$$f(t,z,\tau,\xi) = \bar{f}(t,z) + \tilde{f}(t,z,\tau,\xi), \text{ with } \bar{f}(t,z) = \lim_{\tau,\xi\to+\infty} f.$$

We suppose that all the functions $\tilde{f}$ are rapidly decreasing at infinity in τ,ξ and carries the rapid variations of f^ϵ in the layer $0 \le t_d = O(\epsilon)$, $0 \le z'_d - t'_d = O(\epsilon^2)$. The terms $\tilde{f}$ correspond to the boundary layer and the terms $\bar{f}$ correspond to the outer part.

We get the following pulse pressure model. For the rapid variations, we get a Korteweg de Vries equation in $\tilde{p}_1$

$$2\tilde{p}_{1\tau} + (2q_1 + p_1)\tilde{p}_{1\xi} + \lambda\tilde{p}_{1\xi\xi\xi} = 0. \tag{8}$$

For the slow terms,we get an equation in $\bar{p}_1$

$$\bar{p}_{1tt} + \eta\bar{p}_{1t} - \bar{p}_{1zz} = 0. \tag{9}$$

A low frequency approximation of (9) gives a 2 or 3-element windkessel system for each position, ($\bar{P}$ is the slow pressure term in initial variables),

$$\frac{d\bar{P}(T)}{dT} + \frac{\bar{P}(T)}{R_p C} = \frac{P_\infty}{R_p C} + \frac{\tilde{P}(T,Z_m)}{R_H C} \tag{10}$$

where C and P_∞ are respectively the arterial compliance and the asymptotic pressure,R_H and R_p are the heart and peripheric resistances.

Therefore, we propose to estimate the measured arterial blood pressure as the sum of a N-soliton ($\tilde{P}$) describing the fast phenomena and a windkessel model ($\bar{P}$) representing the slow phenomena.

$$P(T,Z) = \tilde{P}(T,Z) + \bar{P}(T,Z).$$

The description of the obtention of the model can be found in the detailed article [**1**].

3. Numerical validation of the model

The pressure P is the sum of a N-soliton (the fast component) and a 2-element windkessel solution (the slow one). The identification is done for a 2 or 3-solitons and a 2-element windkessel. The figure 1 illustrates the rather good results obtained from real arterial blood pressure data measured at the finger level with a FINAPRES sensor.

4. Conclusion

In this article we have proposed a reduced model of the input-output behaviour of an arterial compartment, including the short systolic phase where wave phenomena are predominant. The close form formulae of these non-linear models of propagation in conjunction with windkessel models are rather easy to use to represent wave shapes at the input and output of an arterial compartment. Some very promising preliminary comparisons of numerical results obtained along this line with real pressure data have been shown.

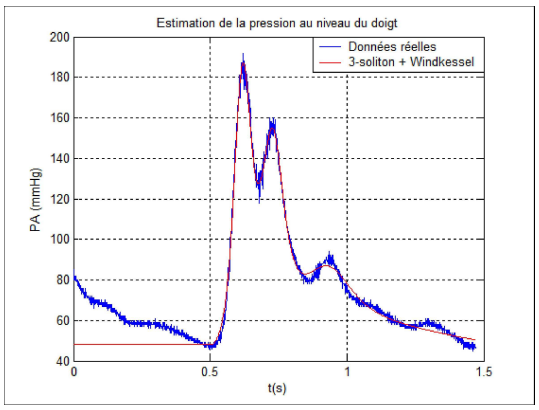

FIGURE 1. Pressure at the finger : real and estimated data

Références

[1] E. Crépeau and M. Sorine, *A reduced model of pulsatile flow in an arterial compartment*, To appear in Chaos, Solitons and Fractals, accepted in March 2006.

[2] A. Monti and C. Médigue and M. Sorine, *Short-term modelling of the controlled cardiovascular system*, ESAIM : Proceedings, 12, pp115-128, 2002.

[3] J.T. Ottesen, *General Compartmental Models of the Cardiovascular System. In Mathematical models in medicine* Ottesen, J.T. and Danielsen, M. IOS press, pp121-138, 2000.

[4] C.S. Peskin, *Lectures on Mathematical Aspects of Physiology*, F.C. Hoppensteadt, AMS, 19, 1981.

[5] G.B. Whitham, *Linear and nonlinear waves*, J. Wiley and Sons, 1999.

[6] S. Yomosa, *Solitary waves in large blood vessels*, J. of the Physical Society of Japan, 56, pp506-520, 1987.

Emmanuelle Crépeau
UVSQ, Avenue des Etats-Unis, 78035 Versailles.
E-mail : `crepeau@math.uvsq.fr`