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The reservoir-wave model☆

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Abstract This paper is based on a talk given at the Arterial Hemodynamics: Past, Present and Future symposium in June 2016. Like the talk it is divided into three different but related parts. Part 1 describes the calculation of reservoir and excess pressure from clinical pressure waveforms measured at 5 different aortic sites in 40 patients. The main results are that the reservoir pressure waveform propagates down the aorta and is effectively constant from the aortic root to the aortic bifurcation. Part 2 describes a low-frequency asymptotic analysis of the input impedance of an arterial tree. Neglecting terms of second order, the results show that the low-frequency component of the pressure waveform is uniform throughout the arterial tree and is delayed by an effective wave travel time that depends on the properties of the network. The low-frequency pressure waveform shares all of the properties of the reservoir pressure waveform, but it is premature to say that they are identical. Part 3 describes the analysis of arterial hemodynamicsusing wave fronts. It shows that every wave front introduced at the root of the aorta generates an exponentially increasing number of reflected and transmitted waves with exponentially decreasing amplitudes. The long-time response of the arterial tree can be described by a number of exponentially decaying eigen-modes, each with a different time constant. The analysis is applied to a 55-artery model of the human circulation and the modes and their time constants are shown. This theory provides an alternative method for studying arterial hemodynamics and helps in the interpretation of reservoir and excess pressure.

Introduction

This paper is an outline of the talk given at the meeting Arterial Hemodynamics: Past, Present and Future held at University College London, 14–15 June 2016. It is not a transcript of the talk. It does follow the structure of the talk and is rather more wide ranging than the usual scientific paper, being divided into three slightly disjointed parts. Part 1 describes some recent work analysing clinical arterial pressure measurements taken at five different locations along the aorta using the reservoir-wave model. The full paper describing this work has recently been submitted

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for publication and so this section takes the form of a brief outline of the main findings. Part 2 describes some recent work analysing the low-frequency component of the pressure wave in an arterial tree using impedance methods. This may seem out of place in a talk dealing with the reservoir pressure hypothesis, but I hope that its relevance is clear in the end. Part 3 concerns current work looking at arterial hemodynamics using wave fronts as the basis of the analysis. Some of the analysis presented at the meeting has now been revised and this outline is based on the most recent results. This analysis shows potential but is not completed and so this part of the paper should be considered to be work in progress rather than final results.

Reservoir and excess pressure along the human aorta

The definition and separation of pressure

Pressure is a such a familiar concept that we frequently forget how it is defined scientifically. In thermodynamics pressure P is defined as

$$P = -\left(\frac{\partial U}{\partial V}\right)_{S,N_k}$$

where U is the free energy of the system, V is its volume, S is the entropy and N_k are the mole numbers of the different chemical components of the system. This fundamental definition is essentially useless in the clinic because of the impossibility of measuring or controlling the intrinsic and extrinsic parameters.

In mechanics the formal definition of P is

$$P = \frac{1}{3}\sigma_{ii}$$

where $\mathbf{T} = \sigma_{ij}$ is the stress tensor where $i, j = [1, 2, 3]$ indicate the three cartesian coordinates and we use the summation convention for indices. In the jargon of mechanical analysis, P is the average of the trace of the stress tensor or, more accessibly, the normal component of force per unit area. Definition of the stress tensor is not straightforward in a system involving blood, an extremely complex fluid, and distensible arterial walls. However, this expression is the basis of all of the clinically useful definitions of pressure. Amazingly, despite much effort, no one has been able to show that the thermodynamic and mechanical pressures are equivalent.

Pressure is frequently divided into component parts. Probably the most common division of pressure is the gauge pressure

$$P_{gauge} = P_{absolute} - P_{reference}$$

It is possible to define an absolute pressure $P_{absolute}$. However, this is frequently inconvenient because we usually function in a sea of atmospheric pressure. For this reason we generally use pressure to mean the pressure relative to some reference pressure, i.e. a gauge pressure. This is common practice in the catheter lab where the pressure transducer is calibrated to some pressure relative to the heart which includes the atmospheric pressure. I do not know of any clinic that routinely records the atmospheric pressure which means that it is practically impossible to explore the effect of absolute pressure in hemodynamics.

The most famous separation of pressures into different components is undoubtedly the Bernoulli equation

$$P + \frac{1}{2}\rho U^2 + \rho g H = P_0$$

where P is the hydrodynamic pressure, ρ is the density and U is the velocity of the fluid, g is the gravitational constant, H is the height and P_0 is a constant generally called the total pressure. This equation holds along a streamline in steady flow of an inviscid fluid. In unsteady flows it is necessary to add another term involving the velocity potential that accounts for the effects of acceleration and is difficult to evaluate except in the simplest of flows. This is an energy equation (pressure has the units of energy per unit volume) that divides the pressure into potential and kinetic energy. Despite the formal restrictions in its derivation, this equation is very useful clinically and is the basis of estimates of pressure in the cardiac chambers in echocardiographical investigations. Even though blood is not inviscid and the arterial system is highly dynamic, this equation provides a very useful way of interpreting various observations of arterial hemodynamics.

In the context of this meeting, undoubtedly the most common separation of pressure into different components is the separation of the arterial pressure waveform into its forward and backward components shown in Fig. 1. This follows from the work by Westerhof and his colleagues who showed that simultaneous measurements of the pressure and flow waveform could be used for the separation through calculation of the reflection coefficient.[1] A few years later Laximinarayan, working in Westerhof's group, showed that the separation could be made more conveniently using the characteristic impedance.[2] I suspect that everyone attending this meeting has made use of this result in their work. In the separation, it is unclear how to apportion the zeroth component (the steady pressure and flow) between the forward and backward waveforms. Westerhof et al. cleverly got around this problem by letting the forward and backward waveforms drift relative to the scales of their measured counterparts. Laximinarayan resolved the problem by not showing any scales at all. This observation, seemingly trivial, is actually important and has a bearing on the definition of the reservoir pressure.

More than a decade ago, we formulated the reservoir-wave hypothesis that it might be useful to divide the measured arterial pressure into a reservoir pressure and an excess pressure defined as the difference between the measured pressure and the reservoir pressure.[3] Our argument was based on the success of the Windkessel model in describing the diastolic pressure waveform and in the original paper we separated the pressure into a 'Windkessel' pressure and a 'wave' pressure. After publishing that paper we realised that the Windkessel pressure was, by definition, uniform throughout the arterial system and could not describe the observed propagation of this component of the pressure down the aorta. For this reason