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The importance of wave reflection: a comparison of wave intensity analysis and separation of pressure into forward and backward components*

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Abstract

Waves and wave reflections play an undoubted role in arterial hemodynamics. Wave intensity analysis and separation of pressure into forward and backward components can both be used to analyze wave phenomena in arteries, but result in different interpretations regarding the contribution of wave reflections to the aorta blood pressure waveform. We compare these approaches using pressure and flow measurements made in the human aorta and discuss why the interpretations might differ.

I Introduction

The importance of waves and wave reflection in the arterial system is well recognized [1;2], although some aspects of the topic remain controversial [3]. Waves transport energy without the necessity for *net* transport of material [4]. In the arterial tree propagation of waves depends on the compressibility of the system [4] which is almost wholly attributable to the compressibility of arteries. Wave intensity analysis is a time-domain based approach for the analysis of waves and wave power, but the use of impedance analysis [3] and separation of pressure into forward and backward components as originally described by Westerhof *et al.* [5] probably remains more widely used. The two approaches view waves in different ways. Wave intensity analysis considers waves as being composed of small ‘wavefronts’ that combine to produce the observed wave. Analyses based on Fourier decomposition treat the measured waveform as a superposition of sinusoidal wavetrains at the fundamental frequency and all of its harmonics. These two approaches can lead to different interpretations regarding the importance of wave reflections in the aorta. The aim of this study was to compare these approaches, discuss their limitations and consider why the derived interpretations differ.

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II Mathematical theory

A Wave intensity analysis

Wave intensity analysis was first described by Parker and Jones [6]. It is based on the one-dimensional equations of conservations of mass and momentum in elastic tubes

$$A_t + (UA)_x = 0$$

$$U_t + UU_x + \frac{P_x}{\rho} = 0$$

where P, U, are pressure and velocity over time, t, A is the cross-sectional area of the tube, ρ is density and x is the distance travelled by the wave. These equations can be solved by the method of characteristics yielding several simple results. One of the most useful results is the 'Water hammer' equation

$$dP_{\pm} = \pm \rho c dU_{\pm}$$

where $dP_{\pm}$ and $dU_{\pm}$ denote the forward and backward wavefronts of pressure and velocity respectively. c is wave speed. Wave intensity (dI) is defined as

$$dI = dP dU$$

where dP is the change of pressure and dU is the change in velocity across a wavefront.

Wave intensity quantifies the power per unit cross sectional area of a wave and is positive for forward travelling wavefronts and negative for backward wavefronts. The integral of wave intensity is termed wave energy. In addition to direction, waves can also be characterized by their effect on pressure: compression waves increase pressure and decompression (expansion or rarefaction) waves decrease pressure.

B Pressure separation

Pressure separation was performed essentially as described by Westerhof *et al.* [5]. Using the notation employed by Laxminarayan [7], forward and backward components of each Fourier component of P and U are calculated as

$$P_f = \frac{1}{2}(P + Z_c AU)$$

$$P_b = \frac{1}{2}(P - Z_c AU)$$

where Z_c is the characteristic impedance, P_f is the forward component and P_b the backward component of pressure. If characteristic impedance is calculated as

$$Z_c = \frac{\rho c}{A}$$

both impedance and wave intensity methods of separation give identical results for P_f and P_b [8].

III Experimental Materials and Methods

Measurements were made in individuals undergoing coronary angiography, details have been published previously [9]. All subjects gave written informed consent in accordance with the protocol approved by the local research ethics committee. Arterial access was via the femoral approach with a Judkins right coronary catheter positioned in the aorta and each participant received intra-arterial heparin (5000u) and no other drugs during the procedure. Pressure and velocity recordings were made using .014 inch diameter Wavewires and Flowwires (Volcano Therapeutics) passed just beyond the end of the catheter. Simultaneous recordings of pressure and velocity were made at 10-cm intervals along the length of the aorta for 1 minute at each location. The timing and magnitude of incident and reflected waves were identified using wave intensity analysis of simultaneous measures of pressure and velocity [9] using the R wave of the ECG as a fiducial marker. Data, including an ECG, were digitized using a National Instruments DAQ-Card AI-16E-4 and acquired at 1 kHz using Labview. Care was taken to ensure accurate alignment of pressure and velocity wires within the vessel and to ensure appropriate orientation of the flow wire to acquire optimal Doppler recordings of blood velocity. Data were ensemble averaged and analyzed off-line using custom software written in Matlab (Mathworks, Natick, MA) as previously described [9].

IV Results

Typical traces of aortic blood pressure and flow velocity and net wave intensity are shown in Fig 1. The net wave intensity plot shows a characteristic forward compression wave (FCW) followed by a small reflected backward compression wave (BCW) which corresponds to ~5% of the intensity of the FCW. In late systole there is a forward decompression wave (FEW) that precedes closure of the aortic valve and is due to deceleration of the rate of myocardial contraction [10]. It is noticeable that there is no detectable wave intensity after ~0.5s once the aortic valve has closed despite a continuing decline in pressure during diastole. The separated pressure is shown in Fig 2. In contrast to the wave intensity analysis there is a large backward pressure (P_b) the peak magnitude of which is ~40% of the peak magnitude of the forward pressure (P_f) and this extends throughout diastole, at which time P_b comprises almost half of the total pressure. By the end of diastole both P_b and P_f are close to zero. The arrival times of the incident and reflected wave at various locations in the aorta are shown in Fig 3. Based on the estimated wave speed the calculated distance to an 'apparent' reflection site (~40cm) remained relatively constant over the length of the aorta.

V Discussion and Conclusion

Wave intensity measures the power/unit area carried by waves, whereas pressure separation resolves pressure into forward and backward (i.e. by implication incident and reflected components). These two different approaches to the analysis of arterial waves give rise to different interpretations regarding the importance of wave reflection, with separated pressures according much more importance to wave reflection in the generation of the blood pressure waveform. The biggest difference between these approaches is in diastole where wave intensity shows negligible wave power (once the perturbation associated with valve closure is over). Wave intensity analysis indicates that discernible reflections are restricted to systole. In contrast pressure separation is generally interpreted to show that a substantial part of pressure throughout diastole is due to reflections.

The lack of convergence between timing of P_b and P_f with more distal measurements in the aorta (Fig 3) appears to conflict with the idea of substantial discrete reflections from distant sites contributing to the aortic pressure or flow waveform. If recordings are made closer to an impedance mismatch responsible for reflections then the interval between incident and reflected wave should get smaller. Evidently this does not happen. Davies *et al.* [9] have suggested that this may be due to a 'horizon effect'; where the inevitable high degree of impedance mismatch in the backward direction at bifurcations that are well matched in the forward direction markedly attenuates the intensity of reflected waves from peripheral locations. These reflections will undergo multiple re-reflections and contribute to the impedance to outflow from the aorta, although the wave power will be very small. Wave intensity analysis does show large reflections in systole in peripheral arteries such as the radial artery [11], but such large reflections are not evident in the aorta. It has been proposed that backward pressure in the pressure separation approach usually attributed to reflections arise from minor impedance mismatches at bifurcations [12] and/or tapering [13]. Both suggestions are plausible. The suggestion that tapering accounts for diffuse reflections is interesting, but it is not clear that the aorta is anatomically tapered under physiological conditions [14], although gradual changes in arterial distensibility or wave speed may be important. Nevertheless, a lack of a marked reduction in the intensity of the forward compression wave due to the cardiac ejection in the radial artery [11] is not consistent with the existence of major reflection sites between the aorta and this location, at least in healthy individuals.

We suggest that comparisons of wave intensity analysis and pressure separation data are complicated by the assumptions involved in pressure separation. Notably regarding mean arterial pressure (for Fourier-based analysis) or end-diastolic pressure (for wave intensity-based separation approach or the Fourier approach if diastolic pressure is subtracted before analysis). In a recent review Westerhof and Westerhof [12] state that 'reflections do not exist for mean flow'. This is implicit in Fourier analysis but seems to be an inappropriate way to view the arterial system. Where, if not from waves generated by the heart does mean pressure ultimately derive? A similar difficulty relates to end-diastolic pressure which is used as an integration constant for wave intensity-derived separated pressures. What accounts for the pressure at this time? End diastolic pressure is not the equilibrium state of the circulation, even if it remains relatively consistent under conditions of regular heart rate.

If another heart beat is not initiated diastolic pressure will continue to fall for up to 3–5s, although it does not reach zero [15]. The lack of explanation of the origins of mean or diastolic pressure would seem to be a fundamental problem with the pressure separation technique.

Arguably another problem with pressure (or flow) separation is the assumption that all flow or energy can be resolved into forward and backward components. Some wave energy generated by the heart is converted into potential energy due to the transverse expansion of the elastic vessel. The assumption that the measured flow waveform is the sum of a forward and backward flow wave [5] neglects the volume of blood stored in the vessel segment and implies that the vessel segment has no significant transverse admittance (or a near infinite transverse impedance) [16]. Reflections during systole are likely to contribute to storage of blood in the aorta and the large elastic arteries during systole when influx exceeds efflux through the resistance vasculature [17], but during diastole there is a net efflux of ‘stored’ blood out of the aorta and large elastic arteries.

Wang *et al.* [17] have proposed that a wave-reservoir model may be useful. In this model reservoir pressure and flow take account of the potential energy stored in a segment of blood vessel [18]. If the segment is assumed to be short in relation to the wavelength (i.e. hydrodynamically compact) then 2-element ‘Windkessel’ theory can be applied to model its behavior (a series of such reservoirs or ‘Windkessels’ equates to a simple transmission line [16;18]). While a simplification, use of this approach has the attraction that subtraction of reservoir pressure results in the near abolition of the ‘self-cancelling’ forward and backward pressures in diastole [19]. However the wave-reservoir approach has been criticized by Mynard *et al.* [20] and, despite a defense of the method [21], reservations [22;23] still remain about the appropriateness of this method.

In summary, there is a consensus that wave reflections are important in arterial physiology and pathophysiology, however use of wave intensity and pressure separation leads to differing interpretations of their magnitude; when they occur; and their overall importance to the pressure waveform. We conclude that some assumptions made in pressure separation are questionable.

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Figure 1.

Aortic pressure (P), flow velocity (U) and wave intensity. Forward compression wave (FCW), backward compression wave (BCW) and forward expansion wave (FEW) are indicated.

Figure 2.
Aortic pressure waveform (P) separated into forward (P_f) and backward (P_b) components.
Diastolic pressure was subtracted before separation.

Figure 3.

Time to arrival of reflected wave at various locations in the human aorta. Data are mean $\pm$ SD of observations from 19 individuals.