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SPECIAL ISSUE - ORIGINAL ARTICLE

An introduction to wave intensity analysis

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Abstract Wave intensity analysis applies methods first used to study gas dynamics to cardiovascular haemodynamics. It is based on the method of characteristics solution of the 1-D equations derived from the conservation of mass and momentum in elastic vessels. The measured waveforms of pressure P and velocity U are described as the summation of successive wavefronts that propagate forward and backward through the vessels with magnitudes $dP_{\pm}$ and $dU_{\pm}$. The net wave intensity $dPdU$ is the flux of energy per unit area carried by the wavefronts. It is positive for forward waves and negative for backward waves, providing a convenient tool for quantifying the timing, direction and magnitude of waves. Two methods, the PU-loop and the sum of squares, are given for calculating the wave speed c from simultaneous measurements of P and U at a single location. Given c, it is possible to separate the waveforms into their forward and backward components. Finally, the reservoir-wave hypothesis that the arterial and venous pressure can be conveniently thought of as the sum of a reservoir pressure arising from the total compliance of the vessels (the Windkessel effect) and the pressure associated with the waves is discussed.

1 Introduction

Wave intensity analysis was introduced 20 years ago for the study of cardiovascular dynamics. In many ways it is a departure from the traditional Fourier methods of analysis that have dominated the field since the 1960s [7]. It represents the waveforms of pressure and velocity as successive wavefronts rather than the summation of sinusoidal wavetrains. This means that the analysis is carried out in the time domain rather than the frequency domain which can be advantageous for many applications.

The analysis is based upon sound mechanical principles, the conservation of mass and momentum, and rigorous mathematical analysis. This means that the mathematics are not always accessible to those whose mathematical training does not extend to partial differential equations, eigenvalues and eigenvectors. The results of the analysis, however, are intuitive and accessible and I will attempt to demonstrate this in the following. For the non-mathematical reader, I have marked the sections containing the more difficult mathematics {**detailed mathematics**}, and given a non-mathematical summary of the results at the end of the section {**non-mathematical summary**}. It should, therefore, be possible to skip these sections, if desired, while still being able to follow the development of the method.[1]

2 Foundations

Wave intensity analysis is rooted in the development of gas dynamics during and after the Second World War. The advent of supersonic flight, jet engines and rockets required a new approach to aerodynamics that could explain the 'new' phenomena that were being observed; particularly shock waves. For low Mach number (defined as the speed of convection divided by the speed of sound, $m = U/c$)

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[1] The material in this paper form the basis for a web site *An Introduction to Wave Intensity Analysis* http://www.bg.ic.ac.uk/research/intro_to_wia. The site contains some additional material and a number of examples which could not be included in this work for reasons of space.

flows, air could be considered to be incompressible with reasonable accuracy, but this was no longer true as we neared the 'sound barrier' when the Mach number approached and exceeded one. For supersonic and hypersonic flow, it became important to track the propagation of waves through the flow field. The mathematical tools for solving these problems were provided nearly a century earlier by Riemann who introduced the method of characteristics for the solution of hyperbolic equations [16].

Although arteries have complex geometries, for many purposes it is sufficient to consider them as long, thin tubes; the 1-D approximation. This approximation ignores the variation of velocity across the cross-section, necessarily abandoning the no slip condition at the wall. It is, therefore, not suitable for the calculation of the detailed distribution of wall shear stress, for example, but does provide information about the axial distribution of pressure and velocity.

2.1 What do we mean by a wave?

Before proceeding to the mathematical theory of wave intensity, it is necessary to clarify what is meant by a 'wave.' Because of the success of impedance methods, most haemodynamicists think of waves in arteries as sinusoidal wavetrains, the fundamental element of Fourier analysis. An example of the Fourier decomposition of a pressure waveform measured in a human aorta is shown on the left of Fig. 1. The waveform shown at the top is given by the summation of the Fourier components which are the sinusoidal waves at the fundamental and higher harmonic frequencies shown below. The decomposition is exact if all of the harmonics are summed, but the higher harmonics are dominated by noise and the first 16 components, shown in the figure give an excellent approximation to the original waveform.

There are, however, other waves such as tsunamis and shock waves (the sonic boom) that are best described as solitary waves. For these waves it is more convenient to consider them as a sequence of small 'wavelets' or 'wavefronts' that combine to produce the observed wave. These wavefronts are the elemental waves in wave intensity analysis.[2] In the digital era, it is convenient and accurate to describe these wavefronts as the change in properties during a sampling period Δt; e.g. $dP = P(t + \Delta t) - P(t)$. Differences such as this are commonly used in gas dynamics instead of the more familiar differential because they can cope with discontinuities such as shock waves where the differential is ill-defined. The difference, unlike the differential, depends upon the sampling period and this must be remembered if differences are used.

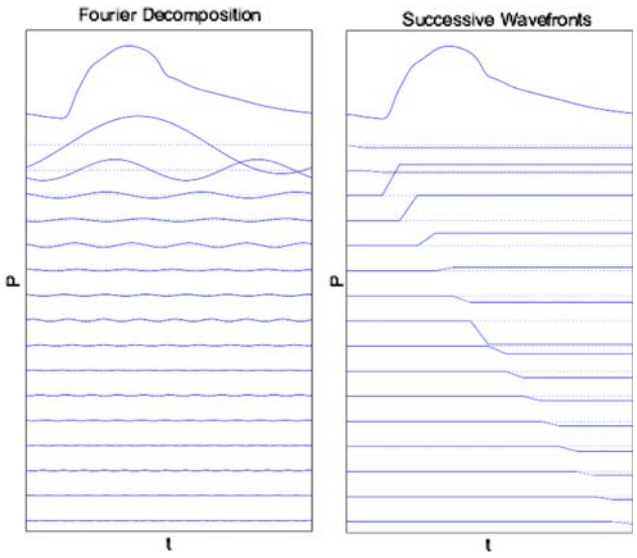

Fig. 1 The decomposition of the pressure waveform measured in a human aorta into sinusoidal wavetrains (*left*) and successive wavefronts (*right*). In each figure, the measured pressure is shown at the *top*. In the Fourier representation, the fundamental and first 15 harmonics are shown (the mean value is suppressed in this sketch). The successive wavefronts are obtained by dividing the cardiac period into 16 time intervals and plotting the change in pressure during the successive time intervals

The plot on the right of Fig. 1 shows the same pressure waveform decomposed into 16 successive wavefronts. This representation of the original waveform is rather crude but serves to illustrate the principle. Higher resolution can be obtained simply by using more wavefronts occurring at smaller intervals during the cardiac period. An exact representation of a digitised waveform can be obtained simply by using one wavefront per sampling period.

This difference in the interpretation of what is meant by a wave is fundamental to the understanding of wave intensity analysis. Both Fourier and wave intensity analysis give unique, complete representations of the measured waveform and the choice of representation is determined solely by convenience; wavefronts can be represented by Fourier components and sinusoidal waves can be represented by successive wavefronts. To avoid possible confusion in this work, '*wave*' will be used in a completely general way, '*wavetrain*' will be used to describe sinusoidal waves and '*wavefront*' will be used to describe the incremental wavelets.

Finally, it is important to observe that the fact that a waveform can be decomposed into a particular form does not imply that that form is in any way intrinsic to the initial waveform. Any waveform can be decomposed without any

[2] The elemental wavefronts should not be confused with solitons, which are solitary wave solutions of the nonlinear Korteweg–de Vries equation originally derived to model shallow water hydrodynamics. The soliton is another example of a solitary wave that cannot be analysed easily using Fourier methods although it can be described easily as successive wavefronts.

loss of information in an infinite number of different ways using any complete, orthogonal basis function or wavelet. No particular decomposition is inherently better than any other; their value depends solely on their utility.

3 Is the cardiovascular system in steady-state oscillation?

It is commonly believed that the cardiovascular system is normally in steady-state oscillation. This view is promoted by the standard texts on arterial mechanics [8, 9] and is reinforced by the observation of very regular, near-periodic behaviour of the arterial pressure during stable conditions. However, periodic behaviour does not necessarily mean steady-state oscillation and this belief deserves investigation.

All macroscopic systems experience some form of damping, be it friction or viscosity, so it is not possible to have steady oscillations without some form of forcing of the system. Forced oscillations are divided into two categories, *under-damped* oscillations which are characterised by a slow decay of the oscillations when the forcing is stopped, and *over-damped* oscillations which cease oscillating immediately when the forcing is stopped. The boundary between these two conditions is termed a *critically damped* oscillation. Critical damping is important in engineering because critically damped systems exhibit the fastest possible transient between the forced and stationary state when the forcing starts or stops and most measuring instruments are designed to exhibit this behaviour to increase their temporal resolution in transitory states. A critically damped system will decay to the stationary, stable state within approximately one period of its natural oscillation when the forcing is stopped.

As long as a periodic forcing is applied to the system, it is impossible to tell whether the system is under- or over-damped because it will continue to oscillate in response to the forcing. If the forcing is stopped, however, it is very easy to differentiate between the two conditions: an under-damped system will continue to oscillate with ever decreasing amplitude until it finally decays to the new stationary state while an over-damped system will stop oscillating immediately and decrease smoothly to the new stationary state at a rate dependent upon the degree of over-damping.

Missing or ectopic beats are commonly observed, even in healthy subjects, when some irregularity in the pacing of the heart occurs which interrupts the regular contraction of the heart for a single beat. This 'natural' stopping of the periodic forcing of the arterial system by the heart provides a convenient way to assess the level of damping in the cardiovascular system. A typical missing beat measured in the left main stem coronary artery of a patient undergoing routine catheterisation is shown in Fig. 2.

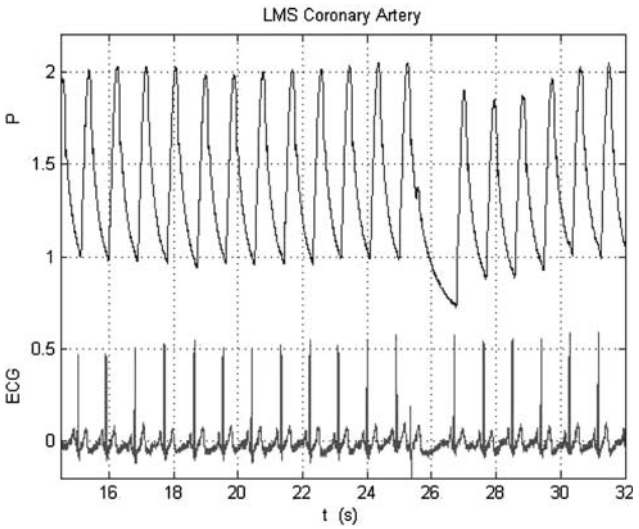

Fig. 2 Pressure response measured in the left main stem coronary artery during a missing beat. The *top trace* shows the pressure in kPa and the *bottom trace* shows the simultaneously measured ECG. Just before 26 s the ECG shows a premature QRS complex resulting in a contraction of the left ventricle that was barely able to create enough pressure to open the aortic valve. The small notch on the *pressure signal* indicates that the valve was opened very briefly but that there was negligible blood ejected during that cardiac cycle. The response to this 'missing' beat is a smooth continuation of the exponential fall-off of pressure that is normally observed during diastole. Following the missing beat, the ECG is normal and the pressure is close to normal. The pulse pressure of the beat immediately following the missing beat has a slightly increased pulse pressure consistent with the potentiation of the ventricular contraction produced by the increased filling due to the preceding missing beat (the Frank–Starling mechanism). There is also a decrease in mean pressure which persists for about 4–5 beats before the oscillation returns to its state prior to the missing beat

After the missing beat we see a smooth continuation of the exponential fall-off of pressure that is normally observed during diastole. This is typical of an over-damped system. There is no hint of a slightly damped oscillation at the normal heart frequency that would be characteristic of an under-damped system. This behaviour indicates that the cardiovascular system is over-damped and, by definition, over-damped systems cannot exhibit steady-state oscillation.

We must conclude, therefore, that the cardiovascular system is not in steady-state oscillation. It is probably better to think of each heart beat as an isolated event that just happens to occur periodically because of the regularity of the normal heart beat under constant conditions.

4 The method of characteristics

It is impossible to describe the development of wave intensity analysis without some discussion of the method of

characteristics. Although the method is rather complex mathematically, its results are simple and easy to comprehend.

{**detailed mathematics**} The 1-D equations describing flow in an elastic tube were formulated by Euler [3]. Although they can and have been generalised to consider viscous effects and compressible fluids, we will consider only the inviscid, incompressible case treated by Euler. The conservation of mass applied to a differential element of the tube requires that the change in volume of the element is equal to the difference between the volume flow rates into and out of the element

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

where A is the cross-sectional area of the tube, U is the velocity averaged over the cross-section, x is the distance along the tube, t is time and we are using the subscript notation for partial derivatives. Similarly, the conservation of momentum requires that the acceleration of the fluid within the element is equal to the net momentum flux into the element plus the net force acting on the element due to the pressure

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

where P is the hydrostatic pressure averaged over the cross-section and ρ is the density of blood which is assumed to be constant. These conservation equations involve three dependent variables A, U and P and so it is necessary to specify some further relationship between them. This is provided by a 'tube law' which relates the local area of the tube to the pressure within it. For our purposes, it is possible to express this relationship in a very general form

$$A(x,t) = A(P(x,t);x)$$

This functional equation just says that the local area is some function of the local pressure which can vary at different locations along the tube x. It is possible to generalise the tube law to account for temporal variations in the local relationship between area and pressure which would be necessary if, say, the effects of temporally changing arterial tone were to be considered or if the theory was being applied to vessels such as the coronary arteries where the state of the myocardium around the artery is changing in time. However, this generalisation introduces considerable complexity into the analysis and is not considered here for simplicity. Note, however, that the temporal variation of pressure means that there is still a temporal variation in the area.

In this development of wave intensity analysis, we choose to eliminate A and to retain P and U as the independent variables, primarily because those are the variables most frequently measured in the clinic. Other choices may be more convenient for particular applications. For example, in our recent numerical work it has proven to be most convenient to solve the problem in terms of the volume flow rate $Q = UA$ and A [4, 6]. Similarly, a clinical application of wave intensity analysis has been developed which uses ultrasound measurements of vessel diameter d and velocity, in which case it is most convenient to express the theory in terms of $d = \left(\frac{4A}{\pi}\right)^{1/2}$ and U.

With our assumption of the tube law, it is possible to write the partial derivatives of A

$$\left(\frac{\partial A}{\partial x}\right)_t = A_P P_x + A_x \quad \text{and} \quad \left(\frac{\partial A}{\partial t}\right)_x = A_P P_t \quad \text{where}$$
$$A_P = \left(\frac{\partial A}{\partial P}\right)_x \quad \text{and} \quad A_x = \left(\frac{\partial A}{\partial x}\right)_P$$

Note that A_P is the local compliance of the artery, i.e. the local change in area caused by a change in pressure, which is a measure of the local stiffness of the artery.

Substituting and rearranging terms, the mass and momentum conservation equations take the form

$$P_t + UP_x + \frac{A}{A_P}U_x = -\frac{UA_x}{A_P}$$

$$U_t + \frac{1}{\rho}P_x + UU_x = 0$$

Written in matrix form, the matrix of coefficients of the x-derivative terms has the eigenvalues

$$\lambda_\pm = U \pm \left(\frac{A}{\rho A_P}\right)^{1/2}$$

which are important for the method of characteristics.

4.1 Wave speed

{**detailed mathematics**} The square root term in the equation for the eigenvalues has the dimensions of velocity and is, as we will see below, the speed at which changes propagate along the tube; i.e. the wave speed. One of the advantages of the method of characteristics is that it gives us an expression for the wave speed in terms of the physical parameters of the problem. Recognising that $D = \frac{A_P}{A}$ is the distensibility of the artery (fractional change in area with a change in pressure), the definition of the wave speed reduces to the expression given by Bramwell and Hill [2]

$$c = \left(\frac{A}{\rho A_P}\right)^{1/2} = \frac{1}{\sqrt{\rho D}}$$

In general, the wave speed will be a function of both pressure and position in the arteries

$$c = c(P(x,t);x)$$

This introduces considerable difficulties in wave intensity analysis and so we generally assume that the wave speed at any particular position is a constant, i.e. $c = c(x)$. We will make this assumption implicitly in most of the following analysis. It should, however, be kept in mind that it is an approximation to the behaviour of real arteries which generally become stiffer at higher pressures.

4.2 Solution by the method of characteristics

{**detailed mathematics**} Riemann observed that the *characteristic directions* defined as $\frac{\mathrm{d}x}{\mathrm{d}t} = \lambda_{\pm} = U \pm c$ play an important role in hyperbolic systems of equations, for which the eigenvalues are real. Along these directions the total derivative with respect to time can be written

$$\frac{\mathrm{d}}{\mathrm{d}t} = \frac{\partial}{\partial t} + \frac{\mathrm{d}x}{\mathrm{d}t}\frac{\partial}{\partial t} = \frac{\partial}{\partial t} + (U \pm c)\frac{\partial}{\partial t}$$

Substituting into the conservation equations

$$\frac{\mathrm{d}P}{\mathrm{d}t} - (U \pm c)P_x + UP_x + \rho c^2 U_x = -\frac{UA_x}{A_P}$$

$$\frac{\mathrm{d}U}{\mathrm{d}t} - (U \pm c)U_x + \frac{1}{\rho}P_x + UU_x = 0$$

Dividing the first equation by ρc and adding and subtracting it from the second equation, we obtain the ordinary differential equations along the characteristics

$$\frac{\mathrm{d}U}{\mathrm{d}t} \pm \frac{1}{\rho c}\frac{\mathrm{d}P}{\mathrm{d}t} = -\frac{UcA_x}{A}$$

Finally, we can write these equations very simply in terms of the Riemann variables $R_{\pm}$

$$\frac{\mathrm{d}R_{\pm}}{\mathrm{d}t} = \mp\frac{UcA_x}{A} \quad \text{where} \quad R_{\pm} \equiv U \pm \int \frac{\mathrm{d}P}{\rho c}$$

This remarkable result says that along the characteristic directions, we can solve for the Riemann variables by solving a simple ordinary differential equation in time.

For the purposes of describing the physical meaning of this rather subtle mathematical result, let us consider the simple case of a uniform vessel. For this case, $A_x = 0$ and so the Riemann variables are constant along the characteristic directions.[3] If there is no velocity in the vessel, then the Riemann variables are constant along lines that propagate upstream and downstream with speed $\pm c$. This justifies our identification of c with the wave speed. If there is a velocity in the vessel, the waves propagate downstream with velocity $U + c$ and upstream with velocity $U - c$. That is, the waves are convected with the flowing fluid, just as ripples caused by throwing a stone in a river get carried along with the river. If $U < c$, then one of the waves travels downstream and the other upstream. If $U > c$, then both of the waves propagate downstream and there is no way that changes produced in the vessel at any point can have an effect on the flow upstream. This is what happens in supersonic (or supercritical) flows and explains why subsonic and supersonic flows behave so differently. The convective velocity of blood in the arteries seldom, if ever, exceeds the wave speed and so we will consider only subcritical flows.

If we are interested in what is happening at a particular location x at a particular time t, we simply have to find the waves that intersect at (x, t), determine the value of the Riemann variables $R_{\pm}$ and then solve for P and U using the above expression for $R_{\pm}$. Conceptually this is very easy, but in practice it is not so simple. First of all, the path of the wave depends upon the local velocity and the local velocity depends upon the waves arriving there from upstream and downstream. Secondly, the expression for the wave speed depends on the pressure and so we have to solve integral equations to find P and U from the values of $R_{\pm}$. Making the assumption, discussed above, that c is constant, P and U at (x,t) are simply

$$P = \frac{\rho c}{2}(R_+ - R_-)$$

$$U = \frac{1}{2}(R_+ + R_-)$$

where $R_{\pm}$ are the values of the Riemann variables associated with the forward and backward characteristics that intersect at (x, t). Generally, the Riemann variables are given by the boundary conditions that are applied at the inlet and outlet of the vessel. In more complicated circumstances, changes can be imposed upon the vessel, for example, by applying external compression to it at some particular point. In these cases, the Riemann variables are also determined by the conditions imposed everywhere along the vessel, not just at its boundaries.

{**non-mathematical summary**} Any perturbations introduced into an artery will propagate as a wave with the speed $U + c$ in the forward direction and speed $U - c$ in the backward direction. U is the velocity of the blood and c is the wave speed which depends on the elastic properties of the artery.

5 Wave intensity

With this rather extensive background, we are finally in a position to describe the origin of wave intensity. In practice, we generally make measurements over time at a particular point in the artery. Given that we only know $P(t)$ and $U(t)$ at that particular point x, what can we learn about

[3] In this case, they are generally referred to as Riemann invariants.

the waves there? Since the arterial system is very complex and we generally do not know how the properties, or even the anatomy, of the arteries varies upstream and downstream of the measurement site, we are obviously limited in how we can apply the general solution that we have just derived.

{**detailed mathematics**} From the definition of the Riemann variables, we can write the differences

$$\mathrm{d}R_{\pm} = \mathrm{d}U \pm \frac{\mathrm{d}P}{\rho c}$$

where $\mathrm{d}P$ and $\mathrm{d}U$ are the differences in the measured P and U during the interval $\mathrm{d}t$, which can be conveniently taken as the sampling interval. Solving these two equations for $\mathrm{d}P$ and $\mathrm{d}U$

$$\mathrm{d}P = \frac{\rho c}{2}(\mathrm{d}R_{+} - \mathrm{d}R_{-})$$

$$\mathrm{d}U = \frac{1}{2}(\mathrm{d}R_{+} + \mathrm{d}R_{-})$$

The wave intensity $\mathrm{d}I$ is defined simply as the product of the measured $\mathrm{d}P$ and $\mathrm{d}U$

$$\mathrm{d}I(t) \equiv \mathrm{d}P(t)\mathrm{d}U(t) = \frac{\rho c}{4}(\mathrm{d}R_{+}^2 - \mathrm{d}R_{-}^2)$$

It has the useful property that forward waves make a strictly positive contribution to the wave intensity while backward waves make a strictly negative contribution. Thus, if the instantaneous wave intensity is positive it means that the forward waves are bigger than the backward waves at that time, and *vice versa*. Furthermore, this can be determined solely from measurements made at a single site, although it does require the simultaneous measurement of P and U. This simple observation was the genesis of wave intensity analysis.

5.1 The water hammer equations

{**detailed mathematics**} An important relationship between the change of P and U across a wavefront, the so-called 'water hammer' equations, follows easily from the method of characteristics. The differences $\mathrm{d}P$ and $\mathrm{d}U$ going from one forward characteristic to another depend upon the imposed conditions. $\mathrm{d}P$ can be positive (compression) or negative (decompression) and, similarly, $\mathrm{d}U$ can be positive (acceleration) or negative (deceleration). However, the Riemann variable on the backward characteristic that intersects the two forward characteristics must be preserved. That is, for a forward wave

$$\mathrm{d}R_{-} = 0 = \mathrm{d}U_{+} - \frac{\mathrm{d}P_{+}}{\rho c}$$

Similarly, for differences between the Riemann variables in a backward wave

$$\mathrm{d}R_{+} = 0 = \mathrm{d}U_{-} + \frac{\mathrm{d}P_{-}}{\rho c}$$

This gives us the water hammer equations,

$$\mathrm{d}P_{\pm} = \pm\rho c\mathrm{d}U_{\pm}$$

very simple, but important and useful relationships for arterial waves.

It should be emphasised that P and U are not independent of each other in arterial waves; they are inextricably linked. The theory tells us that any change in P must be accompanied by a change in U. All waves rely upon the exchange of energy from one form to another as the wave propagates. In arterial waves this exchange is between P, the potential energy stored in the elastic walls, and U, the kinetic energy in the moving blood. The difference between the waveforms measured for P and U that often gives rise to the assumption that P and U are independent is, in fact, the result of simultaneous forward and backward waves which, according to the water hammer equations, have a different relationship to each other.

{**non-mathematical summary**} There is a simple relationship between changes in pressure and velocity in any wavefront given by the *water hammer* equations

$$\mathrm{d}P_{+} = \rho c\mathrm{d}U_{+} \quad \text{for forward wavefronts}$$

$$\mathrm{d}P_{-} = -\rho c\mathrm{d}U_{-} \quad \text{for backward wavefronts}$$

The *wave intensity* is defined as the product of the change in pressure times the change in velocity during a small interval. It is positive for forward waves and negative for backward waves. Therefore, the net wave intensity reveals immediately whether forward or backward waves are dominant and how big they are at any particular time during the cardiac cycle. The relationships for forward and backward waves are indicated in the table.

	$\mathrm{d}P$	$\mathrm{d}U$	$\mathrm{d}I$
Forward	>0 compression	>0 acceleration	>0 positive
	<0 decompression	<0 deceleration	
Backward	>0 compression	<0 deceleration	<0 negative
	<0 decompression	>0 acceleration	

Wave intensity has the dimensions of power/unit area and SI units $\mathrm{W/m^2}$. It is essentially the flux of energy per unit area carried by the wave as it propagates. This dimensional interpretation of wave intensity contributes some meaning to it, but its usefulness relies most heavily on its ability to 'measure' the importance of forward and backward waves at every time during the cardiac cycle.

A problem with this definition of wave intensity is that its value depends upon the sampling time. Doubling the

sampling time will double the value of dP and dU increasing the magnitude of dI. This problem can be eliminated by using the alternative definition [10]

$$\mathrm{d}I' = \frac{\mathrm{d}P\mathrm{d}U}{\mathrm{d}t\ \mathrm{d}t}$$

Wave energy. In wave intensity analysis, the time of arrival and the magnitude of a wave are given by the start and the magnitude of the peak when dI is plotted as a function of t. Sometimes, however, it has proved useful to characterise a wave by the integral of the peak, $I = \int_{t_{\mathrm{start}}}^{t_{\mathrm{end}}} \mathrm{d}I\mathrm{d}t$, since weaker but longer duration waves can be equally important as stronger but shorter waves. I is generally called the *wave energy* and has units J/m^2. It should be remembered that this quantity is associated with the energy flux carried by the wave and is generally much less than the total kinetic and potential energy associated with the wave.

Another important property of wave intensity that has proved valuable clinically is that it is calculated in the time domain. With this interpretation of waves, it is very easy to determine when waves are present at the measurement site, their time of arrival and their magnitude. In methods based on Fourier techniques, the results are given in the frequency domain and it is frequently very difficult to determine wave arrival times with this approach. Wave-trains do not 'arrive' they are always there.

An example of wave intensity analysis applied to measurements made in the human ascending aorta is shown in Fig. 3 [12]. The instantaneous pressure, P, and velocity, U, are shown as the top two curves and the net wave intensity, dI, calculated from them is shown as the bottom curve. Positive values of dI correspond to dominant forward waves and negative values to dominant backward waves. The first peak of dI corresponds to the initial compression (or acceleration) wavefront caused by the contraction of the left ventricle. In mid-systole there is a negative peak indicating a dominant reflection of the initial contraction wavefront. This is followed by a second positive peak indicating a dominant forward wavefront at the end of systole. Because P and U are both falling at the time of the second positive peak, it is clear that this represents a decompression (or deceleration) wave generated by the relaxation of the left ventricle.

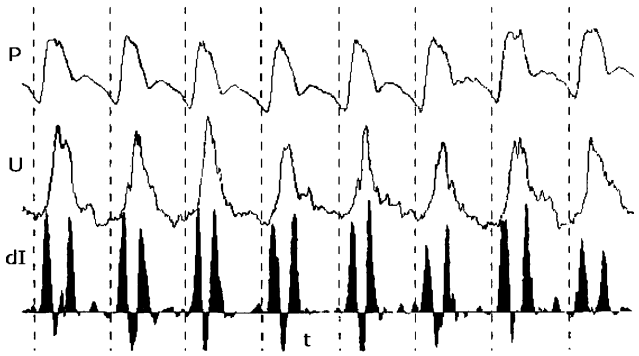

Fig. 3 Our first measurement of wave intensity in man. Instantaneous pressure P and velocity U are plotted as the *top two curves*, and net wave intensity dI is the *bottom curve*. The *dotted lines* represent the peak of the R-wave of the ECG. The heart rate at the time of measurement was approximately 74 beats per min. The velocity measurements were made with a catheter based EM-flow meter with a low signal to noise ratio by the standards of more modern in vivo methods. Despite this, the wave intensity calculated beat-by-beat shows consistent patterns which, during the course of the full measurements, varied regularly with the respiratory cycle deduced from the changes in the measured systolic pressure (approximately one respiratory cycle is shown in the figure)

6 Separation of forward and backward waves

6.1 Wave separation

{**detailed mathematics**} Up to this point, the analysis has been very general, admitting a number of nonlinearities into the analysis; the nonlinearities due to the convective term and the wave speed being a function of the pressure. In spite of this, we can still show that the wave intensity calculated from the measured P and U is the net intensity due to the forward (positive definite) and backward (negative definite) waves. If we now make the partially linearising assumption that the forward and backward waves are additive when they intersect, it is possible to extract even more information from the measurements.

The assumption of additivity is not generally true for nonlinear waves; solitons, for example, do not interact additively when they meet. The solution given by the method of characteristics allows for the separation of waves without making any linearising assumption [15]. However, the method involves the implicit solution of integral relationships, making it far from trivial to implement, and the authors conclude that it generally makes only a small difference compared to the linearised theory. We will therefore restrict ourselves to the linearised method of separation which is relatively easy in theory and in practice. Furthermore, since we can always make the amplitude of the wavefronts as small as desired by increasing the sampling frequency, we can make the linear separation more accurate by increasing the sampling rate. We also note that with these linearising assumptions, the following separation of forward and backward waves is formally identical to the method using Fourier analysis introduced by Westerhof et al. [22].

If we define d$P_{\pm}$ and d$U_{\pm}$ as the changes in pressure and velocity in the forward '+' and backward '−' waves, additivity requires

$$dP = dP_+ + dP_- \quad \text{and} \quad dU = dU_+ + dU_-$$

These equations, together with the waterhammer equations for the forward and backward waves

$$dP_\pm = \pm dU_\pm$$

can be solved for changes in the forward and backward waves

$$dP_\pm = \frac{1}{2}(dP \pm \rho c dU)$$

or, equivalently,

$$dU_\pm = \frac{1}{2}\left(dU \pm \frac{dP}{\rho c}\right)$$

The forward and backward wave intensity for the separated waves is

$$dI_\pm \equiv dP_\pm dU_\pm = \frac{\pm 1}{4\rho c}(dP \pm \rho c dU)^2$$

It may not be immediately obvious, but a bit of simple algebra shows that the forward and backward wave intensity sum to the measured wave intensity

$$dI = dI_+ + dI_-$$

which is convenient analytically.

The pressure and velocity waveforms for the forward and backward waves can be found by summing these wavefronts determined from the measured P and U

$$P_\pm(t) = \sum_0^t dP_\pm(t) + P_0 \quad \text{and}$$

$$U_\pm(t) = \sum_0^t dU_\pm(t) + U_0$$

where P_0 and U_0 are pressure and velocity at $t = 0$, effectively integration constants. This linearised form of separation of the waves into forward and backward components is formally identical to the Fourier method first proposed by Westerhof and his co-workers [22] and it produces essentially identical results [refer to paper in this issue by van den Wijngaard et al.].

{**non-mathematical summary**} If the local wave speed is known, it is possible to use simultaneously measurements of pressure and velocity to determine the magnitude and type of waves arriving at the measurement site at any given time. This is particularly important if the timing of wave arrival is important since a large forward wave can mask the arrival of a smaller backward wave so that the net wave intensity is positive. Forward waves in the arteries are largely caused by the heart and backward waves are the result of reflections. Because the reflected waves can be re-reflected as forward waves (and *vice versa*), this is not entirely true. In some cases, particularly the coronary arteries, there are backward waves generated at distal sites that are not due to reflections.

An example of the separation of the waveforms into their forward and backward components is shown on the left in Fig. 4. For ease of comparison, the diastolic pressure is subtracted from the measured pressure.

The importance of the separation of waves is evident when, as often happens, the forward and backward waves are of similar magnitude so that the net wave intensity is small even though the waves can be big. It is particularly important when determining the arrival time of waves when there are both forward and backward waves present.

7 The reservoir-wave hypothesis

Figure 4 illustrates one of the problems with the separation of the arterial pressure and velocity into their forward and backward components using either impedance or wave intensity analysis. During systole the separation seems to be reasonable with an initial forward compression wave produced by left ventricular contraction followed by backward, reflected waves. During diastole, however, the prediction is invariably that there are large, simultaneous forward and backward waves whose pressures add to give the exponential fall-off of pressure that is regularly seen during diastole accompanied by large velocities that cancel each other out to give the low diastolic flow velocities that are also observed. This is the only way that the wave theory can explain a falling pressure and a zero velocity.

This problem gave rise to the reservoir-wave hypothesis that the pressure in the arteries is made of two components: a reservoir pressure produced by the expansion of the elastic arteries during systole followed by their contraction during diastole (the Windkessel effect) and a wave pressure that drives the arterial waves. Using this hypothesis, the anomalous behaviour of the separated waves during diastole does not occur since the reservoir pressure arising from the Windkessel effect describes the pressure fall-off during diastole very well so that there is little or no wave pressure which is consistent with the little or no velocity that is observed. The right side of Fig. 4 shows the difference between the separated waves calculated without the reservoir pressure and after separating out the reservoir pressure.

The reservoir-wave hypothesis has been applied to arterial system [20] and the venous system [21] with interesting and far-reaching results. It is the subject of another paper in this issue which presents the experimental evidence for it [18]. This approach seems very promising and may be very useful in understanding arterial mechanics more fully.

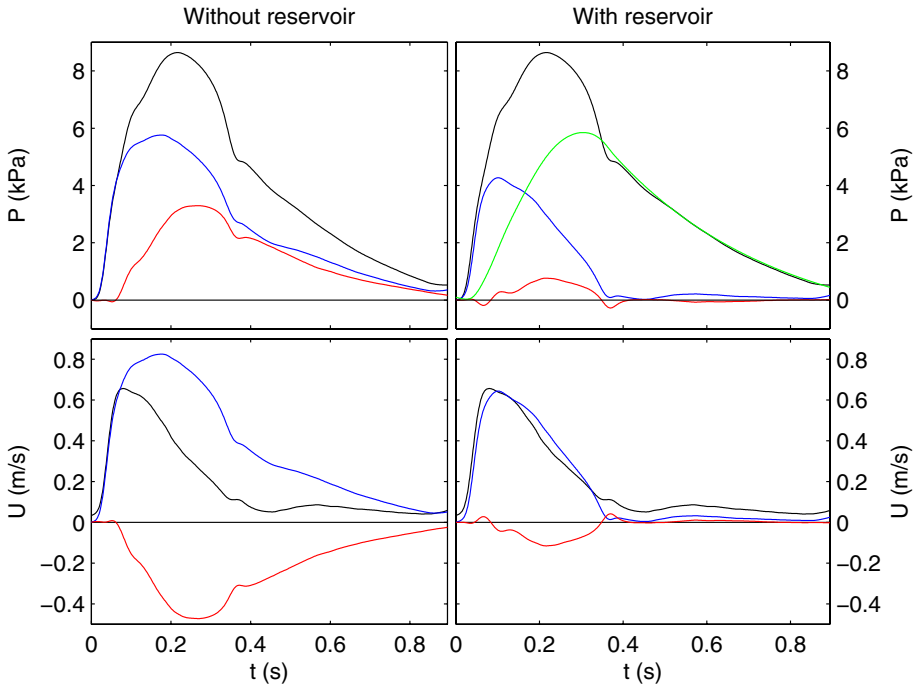

Fig. 4 The separation of measured P and U waveforms into their forward and backward components. The data were measured in the human descending aorta and are shown in *black*. The forward waveforms are shown in *blue* and the backward in *red*. On the *left* the separation is performed on the measured signal; the forward and backward waveforms add to give the measured waveforms. On the *right* the reservoir contribution to the waveforms (shown in *green*) is calculated and subtracted from the measured waveforms to give the total wave contribution to the measured waveforms. The separation is then carried out on the wave P and U. In this case the reservoir, forward and backward waves sum to give the measured waveforms. For ease of comparison, the diastolic pressure is subtracted from the measured pressure

The rate of propagation of the waves through the arterial system is indicated in Fig. 5. The figure shows the pressure measured every 10 cm down the aorta in man. On the left, the data are plotted in the traditional way as $P(t)$ at different x. On the right, the same data are plotted as $P(x)$ at different t, every 20 ms; the solid lines represent the period of rising pressure $100 < t < 360$ ms and the dotted lines the period of falling pressure $t > 360$ ms. The initial compression wave (indicated by the solid arrows) can be seen propagating down the aorta, starting at $t = 100$ ms

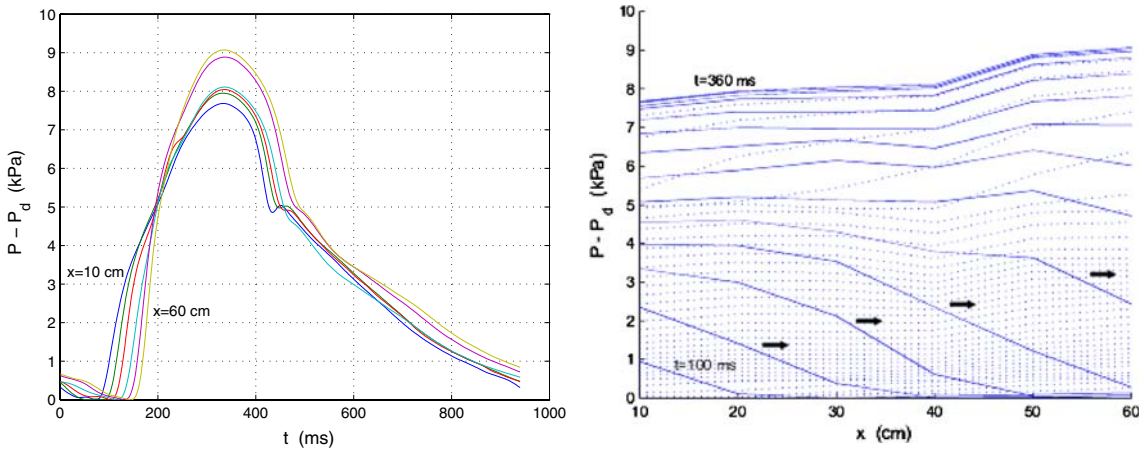

Fig. 5 Aortic pressure measured 10, 20, 30 , 40, 50 and 60 cm downstream from the aortic valve plotted on the *left* as a function of time at different distances and on the *right* as a function of distance at different times. The *solid lines* indicate 20 ms intervals from 100 to 360 ms (the period of ascending pressure) and the *dotted lines* indicate 20 ms intervals from 360 ms to the end of diastole. The *arrows* indicate the progression of the initial compression wave distally during early systole. The diastolic pressure has been subtracted from the measured pressure to emphasise the increasing pulse pressure with distance along the aorta

and ending before $t = 200$ ms. Similarly, the rapid fall in pressure shortly after peak pressure is attained can also be seen to propagate down the aorta. Apart from these two periods, the pressure is remarkably uniform all along the aorta. This is particularly true during diastole, where the falling pressure is very uniformly distributed along the aorta.

8 Reflection and transmission of waves

So far, the theory has been confined to the propagation of waves in a single tube. The arterial system is a very complex network of arteries and it is therefore necessary to consider what happens to the waves as the anatomy or the properties of the arteries change from place to place. Briefly, when a wave encounters a discontinuity of conditions; a change of area, a bifurcation, or simply a change in the local wave speed; reflected and transmitted waves are generated that satisfy the boundary conditions at the discontinuity.

The effect of a bifurcation can be described by a reflection coefficient $\Gamma = \frac{\delta P}{\Delta P}$ where δP is the magnitude of the pressure change due to the reflected wave and ΔP is the pressure change due to the incident wave. An expression for Γ can be found by requiring

1. the net volume flux into the bifurcation is equal to the net volume flux out and (conservation of mass)
2. the total pressure $P_T = P + \frac{1}{2}\rho U^2$ is constant across the bifurcation (conservation of energy).

The results depend upon the areas and wave speeds of the different vessels. The value of Γ assuming that $c \sim A^{1/4}$ is shown as a function of the daughter-parent area ratio $\alpha = \frac{A_1+A_2}{A_0}$ for different values of the asymmetry ratio $\gamma = \frac{A_2}{A_1}$ where A_0, A_1 and A_2 are the areas of the parent, major and minor daughter vessels.

We see from Fig. 6 that the $\Gamma = 1$ for a closed tube, $\alpha = 0$, and that $\Gamma \to -1$ for an open tube, $\alpha \to \infty$. We also see that there is no reflection, $\Gamma = 0$ for symmetrical bifurcations if $\alpha \approx 1.15$, This is generally referred to as the *well-matched condition*. Interestingly, extensive measurements of area ratios of human arterial bifurcations found a mean value $\alpha = 1.14 \pm 0.03$ [11]. Looking only at the coronary circulation, they found a mean value $\alpha = 1.18 \pm 0.04$. The correspondence between the measured area ratios of arterial bifurcations and the well-matched condition could be a coincidence, but it could also be taken as evidence that the arteries are designed to be well-matched for waves generated by the heart.

If arterial bifurcations are well-matched for forward wavefronts, they are necessarily poorly matched for backward wavefronts in one of the daughter vessels. This

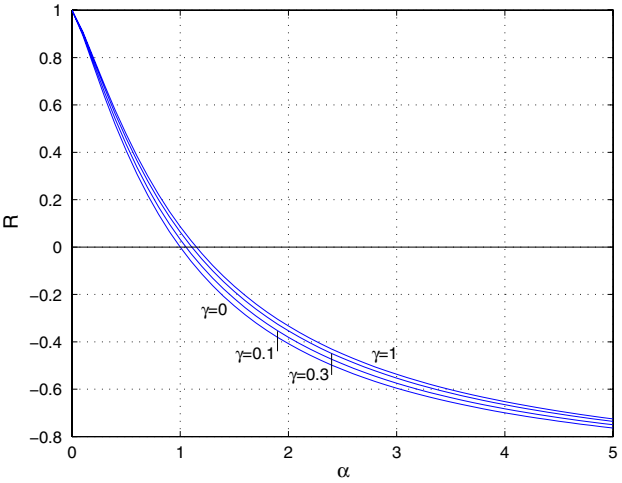

Fig. 6 The reflection coefficient for a bifurcation as a function of α the ratio of daughter to parent areas for different symmetry ratios $\gamma \cdot \gamma = 0$ corresponds to a straight tube with no bifurcation and $\gamma = 1$ corresponds to a symmetrical bifurcation

observation has very important implications in arterial mechanics and leads to a phenomenon that is described as *wave trapping*.

Very briefly, The waves generated by the ventricles propagate forward through the well-matched bifurcations to the periphery where they are reflected by the mismatch in impedance in the small arteries and arterioles. These backward reflected waves must traverse the same bifurcations to return to the heart but, because the bifurcations are poorly matched for backward waves, they suffer significant reflections *en route*. The reflections that occur at the poorly matched bifurcations are forward waves that again travel to the periphery without loss where they too are reflected. These re-reflected backward waves again suffer reflections and so on ad infinitum. This mechanism, arising from the asymmetry in the reflection of forward and backward waves at arterial bifurcations, is very important in understanding arterial haemodynamics.

The wave trapping phenomenon can explain the apparently puzzling results of many of the experiments performed in the arterial system in an effort to elucidate the nature of wave reflections. To cite just two; Peterson and Shephard created a large pressure wave in the femoral artery by the rapid injection of blood and failed to measure any detectable effect in the ascending aorta [13]. Westerhof et al. completely occluded the aorta just proximal to the aorto-iliac bifurcation and measured pressure and flow in the ascending aorta of dogs and found that the effects of the occlusion were so small that they were not tabulated [19]. In the introduction to his chapter on wave reflections, McDonald cites a remark by Wormersley, '*If you wanted to design a perfect sound-absorber you could hardly do better than a set of tapering and branching tubes with*

considerable internal damping such as the arterial tree' [7] (Chap. 12).

9 Determination of wave speed

The local wave speed in arteries is notoriously difficult to measure. In arteries, the wave speed is known to vary from artery to artery and from place to place in the aorta. Methods for the determination of wave speed rely either on measurements of the transit time of a wave from one site to another or upon the calculation of the wave speed from measurements of the elastic properties and the dimensions of the artery wall. Transit time measurements can give only average values over the distance between the two sites of measurement. Calculation of the wave speed from elastic property generally involves invasive measurements of wall properties that are difficult or impossible in the clinic.

Because net wave intensity does not involve knowledge of the wave speed, but only the simultaneous measurement of P and U, it is a very robust and reliable measure of net wave properties. It should be used preferentially whenever possible. However, there are cases when the properties of the separated waves are important and these turn out to be very sensitive to the wave speed that is used. Also, since the local wave speed is inversely related to the local distensibility of the artery, it is also a clinically meaningful property in its own right. For these reason, a lot of effort has been expended on ways to determine the local wave speed, ideally from the measurements of P and U.

Two approaches have been developed for determining the wave speed from simultaneous measurements of P and U; the PU-loop and the sum-of-squares.

9.1 The PU-loop method

If there are only forward waves in the artery, $dP = dP_+$ and $dU = dU_+$ which means, using the water hammer equations, that $dP = \rho c dU$. Thus a plot of P versus U should be linear during any period when there are only forward waves present, and that the slope of the line should equal ρc. In the systemic and pulmonary arteries, we expect that there should be a period right at the start of systole, after the initial contraction wave has passed but before any reflections can get back to the measurement site, when there are only forward waves. Plots from clinical measurements show that this is true and this provides our most secure way of determining the local wave speed.

Practically, there are problems with this determination of wave speed. Temporal delays between the pressure and velocity measurements have large and unpredictable effects on the slope of the PU-curve during early systole. Since the methods of measuring pressure and velocity are very different, there is a high probability that there will be some time lag introduced into the two measurements and it is essential to calibrate very accurately the measurement systems not only for magnitude but also for the temporal response to obtain consistent, reliable values for c. This is shown in Fig. 7 where shifts of 5 ms between P and U have a very large effect on the 'linear' portion of the PU-loop.

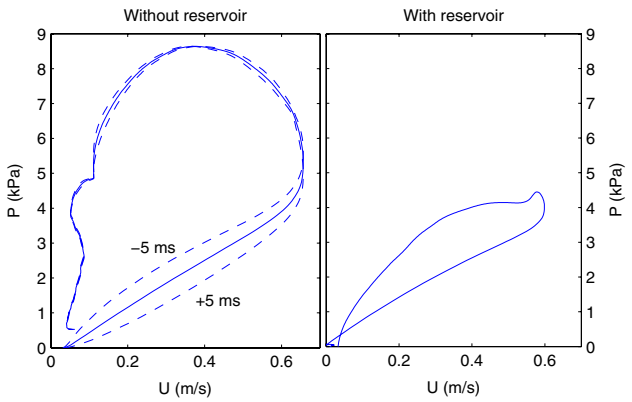

Fig. 7 An example of the determination of the wave speed using the PU-loop method. The *left figure* shows the measured P plotted against U for a single cardiac cycle. The data are the same as those shown in Fig. 4. The loop is traversed in the counter clockwise direction in time. The *linear* portion of the curve, corresponding to the early part of systole, indicates that there are only forward waves present during that period of the cardiac cycle and by the water hammer equation the slope is ρc, giving a measure of the wave speed since the density of blood is known. The *dotted lines* indicate the sensitivity of the PU-loop to shifts in measurements of P and U. They indicate the effect of 5 ms shifts of U relative to P. The same data after the subtraction of the reservoir pressure is shown on the *right* of the figure. The slopes of the linear portions of the two loops are nearly identical. The wave pressure obtained after the separation of the reservoir pressure is much smaller in the descending aorta and relatively small reflected wave seen in Fig. 4 means that the loop is much closer to the *linear line* predicted when only forward waves are present

Experience with well calibrated sensors has confirmed that there is a period in systemic and pulmonary arteries, often shorter than we expected, when the PU-loop is linear, confirming that there are only forward waves in very early systole. Incidentally, this is also consistent with our general observation that wave intensity goes to zero during the later parts of diastole in most cases. In practice, we find that we can often use the linearity between P and U during early systole to infer the relative lags in the measurement systems. This is done by shifting one signal relative to the other until the 'most linear' relationship is attained.

9.2 The sum of squares method

In some circumstances, particularly in the coronary arteries, it is not possible to be sure a priori that there are periods during the cardiac cycle when there are only forward waves present in the artery. For these cases we have

devised another algorithm. It is based on the observation, for both clinical and benchtop measurements, that the use of an incorrect wave speed, either too small or too big, usually results in the calculation of self-cancelling forward and backward wave intensity. Remember that the sum of the separate wave intensities must equal the net wave intensity which depends only on the measured P and U, independent of the wave wave speed. This suggests that minimising the magnitude of the separated wave intensities might give a way to determine the wave speed. Mathematically this involves the minimisation of the sum of the absolute values of the separated wave intensities as a function of c. Defining

$$W = \sum |\mathrm{d}I_+(t)| + \sum |\mathrm{d}I_-(t)| = \frac{1}{2}\sum\left(\frac{\mathrm{d}P^2}{\rho c} + \rho c \mathrm{d}U^2\right)$$

where the sum is taken over the cardiac cycle. Minimising with respect to c, we obtain

$$\rho c = \left(\frac{\sum \mathrm{d}P^2}{\sum \mathrm{d}U^2}\right)^{1/2}$$

which provides a second way to calculate c. Further analysis shows that this is only strictly true when the forward and backward velocities are not correlated, i.e.

$$\sum \mathrm{d}U_+ \mathrm{d}U_-.$$

A recent paper has demonstrated that the sum of squares method for calculating wave speed gives non-physiological results when applied to measurements made in the coronary arteries before and after interventional therapy [5]. The reasons for this may be the presence of large reflection sites close to the measurement site in the relatively short coronary arteries or the neglect of the reservoir pressure in the calculations. Because of the importance of the wave speed in the separation of forward and backward waves, which is particularly important in the coronary arteries, it is important to resolve these difficulties if wave intensity analysis is to contribute to our understanding of coronary artery dynamics [17].

{**non-mathematical summary**} The relationship between changes in pressure and velocity in an arterial wave enable us to calculate the local wave speed during periods when there are only forward waves present, e.g. during very early diastole before the initial compression wave has had time to be reflected. This period can be determined from the slope of the linear segment of a plot of the PU-loop during early systole.

$$c = \frac{1}{\rho}(\text{slope of the } PU\text{-curve})$$

If the PU-loop does not display a linear segment during early systole, as is the case in the coronary arteries, the wave speed can be determined alternatively from the sum of the square of the pressure change and the sum of the square of the velocity change over one cardiac period.

$$c = \frac{1}{\rho}\sqrt{\frac{\sum \mathrm{d}P^2}{\sum \mathrm{d}U^2}}.$$

Both methods for determining wave speed work well in in vitro experiments but they should be used with caution in vivo where the validity of the underlying assumptions about the uniformity of the vessel and the nature of the reflected waves is largely unknown.

10 The advantages and disadvantages of wave intensity analysis

Wave intensity analysis relies upon the simultaneous measurement of pressure and velocity, which is not trivial. In principle, it is very easy to calculate the wave intensity, particularly for digitally acquired data where $\mathrm{d}P$ and $\mathrm{d}U$ can be thought of as the difference between P and U over one sampling time. In practice, taking simple differences is very sensitive to noise in the measurements. Since wave intensity is the product of two differences, it is doubly sensitive to noise. For this reason, it is almost always necessary to filter the experimental measurements in some way before taking the differences, which means that the results can be sensitive to the nature of the filtering that is used.

A significant advance in the practical realisation of wave intensity analysis came with the use of Savitzky–Golay filters [14]. These filters were developed to smooth spectrographic data where it is important to preserve peaks in the data while smoothing. In brief, the filter fits a polynomial of chosen order to a chosen number of points about the centre point using least squares. The smoothing filter then returns the value of the fitted polynomial. Knowing the fitted polynomial, however, means that any derivative of the fitted polynomial can also be returned as the filter output. In particular, the filter coefficients can be determined so that the value returned by the filter is the first derivative of the fitted polynomial. This means that a filter can be implemented that calculates the first derivative of the best fit polynomial through the local data, differentiating and smoothing the data in a single operation. With this filter, it is relatively easy to calculate the net wave intensity in real time as the pressure and velocity data are being recorded.

Since wave intensity analysis is done in the time domain rather than the frequency domain, it is easy to relate the features of the analysis to the temporal changes in the measured pressure and velocity. It is very easy, for

example to relate shoulders and points of inflection on the measured pressure waveform to the arrival times of waves calculated using wave intensity analysis. This is not true for methods based on Fourier analysis. It is usually difficult to predict features of the measured waveform from the collection of waves determined by the Fourier decomposition. For example, cover the measured waveform at the top of the left hand side of Fig. 1 and try to work out the time at which the rapid rise of pressure at the beginning of systole begins by looking at the sinusoidal waves that sum to give the measured pressure. Using the successive wavefront representation shown to the right of the figure, the timing of the foot of the wave is very easily determined.

The net wave intensity, preferably calculated using the derivative rather than the difference to eliminate scale differences due to sampling rate, is a remarkably robust measure of net wave energy. It is based on sound mechanical principles, the conservation of mass and momentum, and can be calculated from measured data very easily. It is rather sensitive to any relative delay between the P and U measurements and it is important that the measurements are well calibrated not only in magnitude but in time. The magnitude and pattern of net wave intensity is potentially useful clinically and there have been some applications of it to clinical data [10].

The use of wave intensity analysis to separate forward and backward waves can provide much information about arterial mechanics, particularly when there are large reflections. Unlike the net wave intensity, the separation depends upon an accurate estimate of the wave speed which is not always easy to obtain. For this reason, separated wave analysis is now more of a research tool than a method that can be used clinically. Of course, if the results of the research prove to be useful, it could undoubtedly be developed into a robust clinical method with the appropriate effort.

11 Conclusions

Wave intensity analysis provides and alternative approach for the study of pressure and flow in the cardiovascular system. It is carried out in the time domain and so it is easy to relate the results of the analysis to particular times in the cardiac cycle. It is based on sound mechanical principles, the conservation of mass and momentum, and involves the general solution of the basic equations using the method of characteristics. Despite the complexity of the mathematical methods, the results are surprisingly simple to apply.

Wave intensity has the convenient property that it is positive for forward and negative for backward travelling waves, enabling rapid determination of proximal and distal effects on arterial haemodynamics. The theory also suggests ways of calculating the local wave speed from simultaneous measurements of pressure and velocity. Since the wave speed is directly related to the local distensibility of the vessel, the measurement of wave speed is also a measurement of the local elastic properties of the artery, which could be of clinical importance.

Once the wave speed is known, wave intensity analysis provides a simple way to separate the forward and backward components of the waves that make up the measured pressure and velocity waveforms. This provides further quantitative and temporal information about proximal and distal effects that provide much information about cardiovascular mechanics.

A recent development, arising from wave intensity analysis of measured data but with much wider implications, is the reservoir-wave hypothesis that it is informative to consider the pressure waveform in the arterial (and venous) system as the sum of a reservoir pressure arising from the capacitive effect of all of the elastic vessels and a wave pressure that is responsible for the waves that traverse the vascular system. This ad hoc hypothesis has only been validated in the aorta and venae cavae but it provides a resolution to several long-standing conundrums about arterial mechanics and deserves further consideration. The hypothesis, if valid, could have important implications in the study of coronary arterial mechanics where the separation of measured pressure and flow waveforms is particularly important. It also raises the clinically interesting possibility of measuring the reservoir pressure, a global property seen by the heart, from easily accessible peripheral arterial sites [1].

Finally, it is my belief that wave intensity and Fourier based analysis provide us with two alternative ways of looking at pressure and flow in the arteries. Neither is right (or wrong) and both are approximations to reality. They are based on profoundly different representations of waves, both of which are well-founded mathematically. Both methods of analysis have their advantages (and disadvantages) and ultimately the choice between them is one of convenience and utility.

References

1. Aguado-Sierra J, Alastruey J, Wang J-J, Hadjiloizou N, Davies J, Parker KH (2007) Separation of the reservoir and wave pressure and velocity from measurements at an arbitrary location in arteries. J Eng Med (Proc Inst Mech Eng Part H) 222:403–416. ISSN: 0954-4119
2. Bramwell JC, Hill AV (1922) The velocity of the pulse wave in man. Proc R Soc Lond B 93:298–306
3. Euler L (1775) Principia pro motu sanguinis per arterias determinando. In: Fuss PH, Fuss Petropoli N (eds) Opera posthuma mathematica et physica anno 1844 detecta, vol 2. Apund Eggers et Socios, pp 814–823

4. Franke VE, Parker KH, Wee LY, Fisk NM, Sherwin SJ (2003) Time domain computational modelling of 1D arterial networks in monochorionic placentas. ESAIM Math Model Numer 37: 557–580
5. Kolyva C, Spaan JAE, Pick JJ, Siebes M (2008) "Windkesselness" of coronary arteries hampers assessment of human coronary wave speed by single-point technique. Am J Physiol Heart Circ Physiol 295(2):H482–H490
6. Matthys KS, Alastruey J, Peiró J, Khir AW, Segers P, Verdonck PR, Parker KH, Sherwin SJ (2007) Pulse wave propagation in a model human arterial network: assessment of 1-D numerical simulations against in vitro measurements. J Biomech 40:3476–3486
7. McDonald DA (1974) Blood flow in arteries, 2nd edn. Edward Arnold, London
8. Milnor WR (1989) Hemodynamics, 2nd edn. Williams & Wilkins, Baltimore
9. Nichols WW, O'Rourke MF (2005) McDonald's blood flow in arteries: theoretical, experimental and clinical principles, 5th edn. Hodder Arnold, London
10. Ohte N, Narita H, Sugawara M, Niki K, Lkada T, Harada A, Hayano J, Kimura G (2003) Clincal usefulness of carotid arterial wave intensity in assessing left ventricular systolic and early diastolic performance. Heart Vessels 18:107–111
11. Papageorgiou GL, Jones NB, Redding VJ, Hudson N (1990) The area ratio of normal arterial junctions and its implications in pulse wave. Cardiovasc Res 6:478–484
12. Parker KH, Jones CJH (1990) Forward and backward running waves in the arteries: analysis using the method of characteristics. J Biomech Eng 112:322–326
13. Peterson LH, Shepard EW (1955) Symposium on applied physiology in modern surgery: some relationships of blood pressure to cardiovascular system. Surg Clin North Am 35:1613–1628
14. Press WH, Flannery BP, Teukolsky SA, Vetterling WT (2002) Numerical recipes in C++ : the art of scientific computing, 2nd edn. Cambridge University Press, Cambridge
15. Pythoud F, Stergiopulos N, Meister J-J (1996) Separation of arterial pressure waves into their forward and backward running components. J Biomech Eng 118:295–301
16. Riemann GFB (1859) Üeber die Fortzflanzung ebener Luftwellen von endlicher Schwingungsweite. Gesammelte mathematische Werke und wissenschaftlcher. Nachlass. Liepzig, Teubner BG (ed), pp 145–176 (originally published in Bande VIII, Abhandlungen der Königlechen Geselschaft der Wissenschaften zu Göttingen, pp 43–65)
17. Siebes M, Kolyva C, Piek JJ, Spaan JA (2009) Potential and limitations of wave intensity analysis in coronary arteries. Med Biol Eng Comput (this issue)
18. Tyberg JY, Davies JE, Wang Z, Whitelaw WA, Flewitt JA, Shrive NG, Francis DP, Hughes AD, Parker KH, Wang J-J (2009) Wave intensity analysis and the development of the reservoir-wave approach. Med Biol Eng Comput (this issue)
19. van den Bos GC, Westerhof N, Wlzinga G, Sipkema P (1976) Reflections in the systemic arterial system: effects of aortic and carotid occlusion. Cardiovasc Res 10:565–573
20. Wang JJ, O'Brien AB, Shrive NG, Parker KH, Tyberg JV (2003) Time-domain representation of ventricular-arterial coupling as a windkessel and wave system. Am J Physiol Heart Circ Physiol 284:H1358–H1368
21. Wang JJ, Flewitt JA, Shrive NG, Parker KH, Tyberg JV (2006) Systemic venous circulation. Waves propagating on a windkessel: relation of arterial and venous windkessels to systemic vascular resistance. Am J Physiol Heart Circ Physiol 290: H154–H162
22. Westerhof N, Elzinga G, Sipkema P (1972) Forward and backward waves in the arterial system. Cardiovasc Res 6:648–656