SEPARATION OF THE RESERVOIR AND WAVE PRESSURE AND VELOCITY FROM MEASUREMENTS AT AN ARBITRARY LOCATION IN ARTERIES

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Abstract

Previous studies based on measurements made in the ascending aorta have demonstrated that it can be useful to separate the arterial pressure $P$ into a reservoir pressure $\bar{P}$ generated by the windkessel effect and a wave pressure $p$ generated by the arterial waves; $P = \bar{P} + p$. The separation in these studies was relatively straightforward since the flow into the arterial system was measured. In this study we extend the idea to measurements of pressure and velocity at sites distal to the aortic root where flow into the arterial system is not known. $\bar{P}$ is calculated from $P$ at an arbitrary location in a large artery by fitting the pressure fall-off in diastole to an exponential function and assuming that $p$ is proportional to the flow into the arterial system. We also define a local reservoir velocity $\bar{U}$ that is proportional to $\bar{P}$. The separation algorithm is applied to \textit{in vivo} human and canine data and to numerical data generated using a one-dimensional model of pulse wave propagation in the larger conduit arteries. The results show that the proposed algorithm is reasonably robust, allowing for the separation of the measured pressure and velocity into reservoir and wave pressures and velocities. Application to data measured simultaneously in the aorta of the dog shows that the reservoir pressure is fairly uniform along the aorta, a test of self-consistency of the assumptions leading to the algorithm. Application to data generated with a validated numerical model indicates that the parameters derived by fitting the pressure data are close to the known values which were used to generate the numerical data. Finally, application to data measured in the human thoracic aorta indicates the potential usefulness of the separation.
1 Introduction

The mechanics generating the characteristic shape of the arterial pressure waveform measured in normal subjects were first described quantitatively by Frank in 1899 using an air chamber or windkessel model [1]. This model highlights the importance of the arterial compliance in transforming the discontinuous cardiac output into a more steady pressure and flow in the arteries and microcirculation. Indeed, if arteries were rigid, pressure changes would occur at the same time everywhere in the system, and the blood flow into the microcirculation would be instantaneously equal to the blood flow out of the ventricle, leading to zero blood flow during diastole.

The windkessel model excels in explaining the diastolic part of the pressure waveform, but it fails to reproduce the systolic part because pressure changes are assumed to occur synchronously throughout the arteries without accounting for the wave nature of pulse wave propagation, as also noticed by Frank [2]. The basic equations describing flow in the flexible arteries were first introduced by Euler in 1775, who derived the one-dimensional partial differential equations expressing the conservation of mass and momentum for inviscid flow [3]. These equations can be solved in the time domain using the method of characteristics developed by Riemann [4] or in the frequency domain using Fourier analysis [5]. Both solutions show that pressure and flow waveforms can be understood as forward running waves originated at the left ventricle, and backward or reflected waves [6, 7].

Although forward and backward waves are able to explain changes in pressure and flow waveforms through the arterial system during systole, including their alterations due to ageing, vascular diseases and invasive clinical procedures, they fail in explaining pressure and flow waveforms during diastole. Indeed, the equal and opposite waves predicted during diastole are difficult to explain physiologically at the time when the aortic valve is closed. Wang et al. [8] proposed a unifying windkessel-wave theory that provides a suitable explanation of central aortic pressure and flow waveforms during the whole cardiac cycle. In this work, central aortic pressure was decomposed into the sum of a time-varying reservoir pressure \( \bar{P}(t) \), governed by Frank’s windkessel model, and a wave pressure \( p(x, t) \) that varies in time \( t \) and with the distance along the arteries \( (x) \). This decomposition shows a striking similitude between measured wave pressure and central aortic flow during the whole cardiac cycle (see Figure 5 A in [8]).

\[1\] The notation in Wang et al. [8, 9] is different from that used here. They referred to the ‘reservoir’ pressure as the ‘windkessel’ pressure and the ‘wave’ pressure as the ‘excess’ pressure. Apart from this difference in names, the definitions are the same.
separation of measured pressure into a reservoir pressure and a wave pressure has also been
applied to measurements in the central venous system with equally striking results [9].

The model of Wang et al. provides a suitable explanation of the mechanics of pulse wave
propagation, although it can only be applied to measurements made in the ascending aorta
when the measured flow is equal to the flow into the arterial system. In this work, we propose a
new algorithm, based on empirical hypotheses, to determine the reservoir pressure from pressure
measurements alone at an arbitrary arterial location. Furthermore, we propose a decomposition
of measured flow at the same location into a reservoir and a wave component that are directly
related to their pressure counterparts. The implications of this new separation technique are
explored using in vivo and numerical data.

Note that the three-element windkessel model proposed by Westerhof et al. [11] and widely
used for arterial modelling [12] is an alternative way to express the reservoir component of the
model we propose. However, we are unaware of any model that uses the electrical analogy and
is equivalent to our hypothesis on a combined reservoir and wave components.

2 Methodology

2.1 Previous work on the calculation of the reservoir pressure

The work by Wang et al. [8] on the reservoir-wave separation calculated the reservoir pressure,
$\bar{P}$, using pressure, $P_{in}$, and flow, $Q_{in}$, measured at the inlet to the arterial system, neglecting the
flow through the coronary system. This method solves the conservation of mass in the arterial
system,

$$\frac{dV}{dt} = Q_{in} - Q_{out},$$  \hspace{1cm} (1)

where $V$ is the volume of the arterial system and $Q_{out}$ is the flow out of the arteries through
the microcirculation, by assuming that: (i) the compliance of the whole arterial tree, $C = \frac{dV}{dP}$, is constant; and (ii) the flow through the microcirculation can be written in terms of a
simple resistive relationship $Q_{out} = \frac{\bar{P} - P_{\infty}}{R}$, where $R$ is the effective resistance of the peripheral
systemic circulation and $P_{\infty}$ is the pressure, assumed to be constant, at which flow through the
microcirculation is zero. We note that $P_{\infty}$ is not necessarily the venous pressure, but could be
related to the tissue pressure surrounding the microcirculation.
Using (i) and (ii) we can write Equation (1) as

\[ \frac{d\bar{P}}{dt} + \frac{\bar{P} - P_\infty}{RC} = \frac{Q}{C}. \]  

This equation can be solved by multiplying by the integrating factor \( e^{t/\tau} (\tau \equiv RC) \) with the result

\[ \bar{P} - P_\infty = (\bar{P}_0 - P_\infty)e^{-t/\tau} + \frac{e^{-t/\tau}}{C} \int_0^t Q_{in}(t')e^{t'/\tau}dt', \]

where \( \bar{P}_0 \) corresponds to the pressure at the onset of the ejection. During diastole, where the inflow \( Q_{in} \) is zero, Equation (3) is used to fit \( P_\infty \) and the time constant \( \tau \) to the experimentally measured pressure \( P_{in} \). Approximately the last two thirds of diastole of \( P_{in} \) is used, when waves are believed to be minimal. An estimate of the resistance is calculated as \( R = \frac{<P_{in}>}{<Q_{in}>} \), where \( <P_{in}> \) and \( <Q_{in}> \) are the time-averaged pressure and flow over the cardiac cycle at the ascending aorta.

The wave nature of the system derives from the one-dimensional theory of flow in elastic tubes. A propagating wave is a disturbance generated by an excess pressure \( p \) over an undisturbed pressure \( \bar{P} \) [10]. The measured aortic pressure waveform was defined as having a reservoir component and wave component as \( P_{in} = \bar{P} + p \).

### 2.2 Extension of the theory to measurements at an arbitrary location

We propose an extension of the previous theory based on the following observations:

1. The pressure waveform decay measured at different locations in the arterial system is very similar in diastole. For example, the pressure measured in the carotid arteries is very similar to the pressure waveform measured in the ascending aorta, although the flow waveforms are qualitatively different (see, for instance, Figure 4 in [13]). Recent measurements in the coronary arteries also show pressure waveforms that are very similar to those measured in the aorta in the same subject [14].

2. When the wave pressure in the ascending aorta (the difference between the measured pressure and the reservoir pressure calculated from the measured flow) is plotted against the measured flow waveform, an almost straight line is obtained (see Figure 2 in [8]). This indicates that the flow in the ascending aorta is dominated by forward travelling waves. Thus, arterial bifurcations should be close to well-matched for forward waves and, hence, the wave pressure measured at any aortic location is approximately proportional to the
cardiac output. Observation of well-matched bifurcations for forward waves in humans has been reported in [15].

If both of these observations are true, then it should be possible to determine a reservoir pressure, $P(t)$, from a pressure waveform, $P(x, t)$, measured at an arbitrary location in the arterial tree. Assuming that $P$ is determined from the conservation of mass described by Equation (2) using the same assumptions about the compliance of the whole arterial system and the resistive nature of flow through the microcirculation, the last term of this equation can be written in terms of $P$. By the second observation (assumption) above, we can approximate $Q_{in}$ as being proportional to the difference between $P$ and $P$ that we are trying to calculate. This can be written simply $Q_{in} = \gamma(P - \bar{P})$, where $\gamma$ is a constant which will depend upon a number of factors, such as the local wave speed and cross-sectional area at the root of the aorta. Therefore, Equation (2) leads to

$$\frac{d\bar{P}}{dt} = a(P - \bar{P}) - b(\bar{P} - P_\infty),$$

where $a = \gamma/C$ and $b = 1/\tau$ are rate constants of the system with units $s^{-1}$.

Under normal conditions, $Q_{in} = 0$ for $T_N \leq t \leq T$, where $T_N$ is the time when the aortic valve shuts at the end of systole and $T$ is the time when the heart beat ends. During this period of time, Equation (4) reduces to

$$\frac{dP}{dt} = -b(\bar{P} - P_\infty), \quad T_N \leq t \leq T.$$  

(5)

We consider that $T_N$ corresponds to the time when the dicrotic notch appears in $P$. In case of a pressure waveform without a clearly marked notch, $T_N$ is taken to be the time of the first zero-crossing of the second derivative of pressure at the end of systole. The general solution of Equation (5) is

$$\bar{P} - P_\infty = (\bar{P}(T_N) - P_\infty)e^{-b(t-T_N)}, \quad T_N \leq t \leq T.$$  

(6)

Considering that the reservoir effect is the main driver of the exponential pressure fall off in diastole, and waves are minimal, we can estimate the constant parameters $\bar{P}(T_N)$, $P_\infty$ and $b$ by fitting them to experimental data $P$ using Equation (6). We note that data was non-dimensionalised for better performance of the unconstrained nonlinear optimization algorithm selected. The Matlab routine fminsearch was used with non-dimensionalised initial conditions equivalent to $\bar{P}(T_N) = P(T_N)$ and $b = 1 \text{ s}^{-1}$, and a tolerance of $1e^{-12}$. This function uses the simplex search method described in [16]. We assume $P_\infty = 0$ for experimental data. There
is a suggestion [9], however, that $P_\infty$ should not be the venous pressure, but an intermediate pressure associated with the waterfall or the mean circulatory pressure. In the numerical model, $P_\infty = 432.6$ Pa, which corresponds to the venous pressure previously established as a boundary condition.

During systole, Equation (4) can be solved explicitly using the integration factor $e^{(a+b)t}$,

$$\bar{P} = \frac{b}{a+b}P_\infty + e^{-(a+b)t} \left[ \int_0^t aP(t')e^{(a+b)t'} dt' + \bar{P}_0 - \frac{b}{a+b}P_\infty \right], \quad 0 \leq t \leq T_N, \quad (7)$$

where $\bar{P}_0$ corresponds to the start point of the onset of $P$, since little wave activity is expected at the end of the previous beat. Note that the second observation needs only to be true during the systolic period, $0 \leq t \leq T_N$. To determine the unknown parameter $a$ we enforce continuity of $\bar{P}$ at $t = T_N$, which yields

$$\bar{P}(T_N) = \frac{b}{a+b}P_\infty + e^{-(a+b)T_N} \left[ \int_0^{T_N} aP(t')e^{(a+b)t'} dt' + \bar{P}_0 - \frac{b}{a+b}P_\infty \right]. \quad (8)$$

The parameter $a$ is fitted from experimental data using the fminsearch Matlab routine described above, with non-dimensionalised data, an initial value equivalent to $10 \, s^{-1}$, and a tolerance of $1e^{-12}$. $\bar{P}$ is determined for the entire period from Equations (6) and (7).

### 2.3 Extension to reservoir velocity

The velocity due to the reservoir pressure, $\bar{U}$, is assumed to be zero in the model by Wang et al. [8]. This is a good assumption in the ascending aorta, as shown by experimental measurements where the velocity is invariably zero during the later part of diastole. However, it should not be true in more distal vessels. We now assume that both the pressure and the velocity can be resolved into a reservoir and a wave component; $P = \bar{P} + p$ and $U = \bar{U} + u$. However, while $\bar{P}$ is only time-varying, $\bar{U}$ also depends upon position; $\bar{U}(x,t)$.

To determine $\bar{U}$ we establish that it has to be directly proportional to $(P - P_\infty)$ at the end of diastole when the reservoir effect is dominant and, hence, wave activity is expected to be minimal. This can be written as

$$\bar{U} = \frac{P - P_\infty}{R}, \quad (9)$$

where $R$ is the effective resistance of the vessels downstream of the measurement site. Note that the $PU$-loop proposed in [17] states there is a linear relationship between $P$ and $U$ when only unidirectional waves are present. The slope is equal to $\pm \rho c$, where $\rho$ is the blood density, $c$ is
the pulse wave speed and the sign indicates the direction of the waves.

The quest for the exact portion with the best linear relationship of \( P \) and \( U \) in diastole was implemented using a Bayesian statistics based method for linearity testing, programmed using Matlab. However, for some cases, particularly at high heart rates, the linear portion is difficult to identify, since waves may still be present. For such cases, and to ensure robustness and applicability of our algorithm to cases in which \( U \) is not available or data is noisy, \( \bar{R} \) is determined as 
\[
\bar{R} = \frac{\langle P \rangle - P_\infty}{\langle U \rangle},
\]
where \( \langle P \rangle \) and \( \langle U \rangle \) are the time-averaged pressure and velocity at \( T_N \leq t \leq T \).

Once \( \bar{R} \) has been determined, we can calculate \( \bar{U} \) at any time \( 0 \leq t \leq T \) as
\[
\bar{U} = \frac{\bar{P} - P_\infty}{\bar{R}}.
\]

The wave velocity is then \( u = U - \bar{U} \), which will generally be non-zero except at the root of the aorta during diastole. This velocity (\( \bar{U} \)) is equal to the velocity due to the reservoir pressure if the compliance downstream the measuring site is negligible.

3 Results

3.1 In vivo data

Simultaneous \( P \) and \( U \) measurements from dogs were provided by Wang et al. [8] for analysis. Measurements were performed at four different sites: ascending aorta (Asc), aortic arch (Arch), 11 cm after the aortic valve, thoracic aorta (Thor), at 24 cm, and abdominal aorta (Abd), at 45 cm. Area was calculated as 2.6 cm\(^2\), 1.5 cm\(^2\), 1.5 cm\(^2\) and 0.5 cm\(^2\), respectively, from diameter measurements at each site. Results from the algorithm applied to invasive measurements in paced dogs at a heart rate of 110 beats per minute (bpm) are presented. Figure 1 shows \( P, \bar{P}, p \) and \( u \) calculated for two measurement sites: the aortic arch and thoracic aorta. Note that the scales for \( p \) and \( u \) have been adjusted for a better comparison. It is important to point out the similarity of \( \bar{P} \) in both locations, and the similarity of \( p \) and \( u \) curves at the beginning of systole. Differences between them are due to wave interaction, since they are located at a different distance to main reflection sites. In Figure 2 the overall set of results are presented and their calculated time constants can be found in the first column of Table 1. The mean and standard deviation of the time constants calculated are \( \tau = 2.3 \pm 0.2 \text{ s} \). All graphs have similar scale ranges to better observe the contribution of each component to the measured \( P \) and \( U \).
waveforms. \( \dot{U} \) generated by \( \dot{P} \) is generally low and tends to increase with distance. Note that viscous dissipation accounts for the fall in mean \( P \), mostly noticeable in the reservoir component; while wave activity is responsible for the increase in pulse pressure.

Figure 3 shows the \( PU \)-loop from data at the thoracic aorta as suggested by Khir et al. [17] plus two new curves using the separated components of \( P \) and \( U \) waveforms. The slope at the beginning of systole corresponds to the wave speed. The \( pu \)-loop maintains the same slope as the \( PU \)-loop, retaining the wave speed and wave propagation information. Note that the \( pu \)-loop was plotted as \( p + P_0 \) for convenience, with \( P_0 \) the measured pressure at the onset of ejection. The wave speed calculated using the slope of the \( pu \)-loop at the beginning of systole increases with distance from the aortic valve indicating the change of stiffness of the aorta at the different locations measured. The slope of the \( P\dot{U} \)-loop corresponds to \( R \) that varies at different locations due the distribution of resistances and compliances.

The separation algorithm is also applied to invasive \( P \) and \( U \) measurements in the descending aorta of human subjects obtained in [14]. Figure 4 shows the resulting reservoir and wave components of \( P \) and \( U \) using \( P_\infty = 0 \). The calculated time constant is \( \tau = 1.60 \text{ s} \).

Figure 5 compares the forward and backward waves obtained using the total \( P \) and \( U \) measurements in the human descending aorta (left panels) against the corresponding values obtained using \( p \) and \( u \) (right panels). Waves are separated using the technique proposed in [7]. \( P_0 \) was subtracted from the data prior to separation for ease of graphical comparison. If the wave separation is performed using \( P \) and \( U \), significant wave activity is observed during diastole to produce the exponential decay of \( P \), which consists of self-cancelling waves with equal pressures (\( P_+ \) and \( P_- \)) and opposite velocities (\( U_+ \) and \( U_- \)) throughout the whole diastolic period. On the other hand, the right panels show that the reservoir components \( \dot{P} \) and \( \dot{U} \) are responsible for the exponential decay of \( P \) during diastole. If \( p \) and \( u \) are used to separate forward and backward travelling waves, the resulting \( p_+ \), \( p_- \), \( u_+ \) and \( u_- \) waves are almost zero during diastole, when wave activity is expected to be minimum. The backward travelling waves, \( p_- \) and \( u_- \), arrive at mid-systole to produce the characteristic shape of \( P \) and \( U \). Note that much of the skewing of \( U \) is due to the backward wave \( u_- \); the forward velocity generated by the ventricle is much more symmetrical.

Two of the main advantages of separating the reservoir and wave components from the measured pressure waveform are illustrated in Figures 8 and 9. Figure 8 shows results from separating waves measured in the same experimental dog in the thoracic aorta paced at different heart rates, 110 bpm on left panel and 60 bpm on the right panel. Several features of these
results are worth noting. The pulse pressure more than doubles as the heart rate is decreased, but most of this difference is due to the change in the amplitude of the reservoir pressure. The wave pressure component of the measured pressure is remarkably similar at the two heart rates except for an approximately 50% increase in the peak wave pressure at the lower heart rate, consistent with an increased contraction of the ventricle due to an increased filling time according to the Frank-Starling mechanism. It is also noticeable that $p$ and $u$ are similar in shape during the first half of systole and they deviate from each other in a very similar way due to the arrival of reflected waves. Calculated $\tau$ values can be found in the first and fourth columns of Table 1.

Figure 9 shows $P$, $\bar{P}$, $u$, $p$ and the electrocardiogram (ECG) at four simultaneously measured locations of a dog paced at 110 bpm during a period when an irregularity, a missing beat, occurred. In this case, we analyse a normal heart beat, followed by a long beat caused by the missing beat, subsequently followed by a potentiated beat. These data are rich in content, showing the response at different locations in the arterial tree under normal and irregular conditions. We will mention only a few of the salient features. First, these data show the robustness of the algorithm which performed convincingly at all locations throughout the period. First, we note that the reservoir pressures calculated at the different measurement sites are very similar, consistent with our first hypothesis. Second, the wave pressure in the ascending aorta is very similar in shape to the velocity, as observed in [8]. This is true during the first part of systole at all sites, but during the latter part of systole $p$ and $u$ waveforms begin to differ with the differences increasing with distance from the heart. This means that the second assumption gets progressively worse as we move further and further from the heart valve, where we expect larger reflected waves. Finally, these data are an excellent illustration of the temporal nature of the analysis that enables us to analyse highly irregular events such as this. The time constant $\tau$ calculated for each measurement can be found in Table 1.

### 3.2 Numerical data

The separation algorithm is applied to numerical data obtained using a nonlinear, time-domain, one-dimensional model of pulse wave propagation [18, 19] in a bifurcating network that represents the 37 largest conduit arteries in the human. This numerical model has been validated against a physical representation of the systemic arteries in the human made of silicone tubes [20]. With these data, the separation technique can be tested not only in the aorta but also in other large arteries, without any measuring error and with an exact knowledge of the total compliance, terminal resistance and $P_\infty$ of the system.
Figure 6 shows the total pressures simulated by the model and the calculated $\bar{P}$ at four locations along the aorta and at the left brachial and femoral arteries. The following boundary conditions have been considered. (i) The inflow at the ascending aorta is a half-sinusoidal wave during systole followed by a period of reversal flow, and zero flow during diastole. Therefore, the assumption $Q_{in} = 0$ for $T_N \leq t \leq T$ (see Section 2.2) is perfectly satisfied. (ii) The total compliance of the system has been increased to 1 ml/mmHg by introducing some peripheral compliance to reduce peripheral wave reflections. (iii) The cardiac cycle has been increased to 1 s.

Disregarding the time delay of the onset of pressure at different locations, all sites present very similar reservoir pressures (Figure 7, middle left), which is in accordance with the assumption of a time-varying reservoir pressure in large arteries. Differences arise, mainly, because of the reduction in mean pressure caused by viscous dissipation. Moreover, reservoir pressures have a larger contribution to the total pressure waveforms than their wave counterparts (Figure 7, left), which highlights the importance of arterial compliance on pulse wave propagation. On the other hand, reservoir velocities are less significant compared to their wave components during systole (Figure 7, right). Reservoir velocities become dominant during diastole. Indeed, wave pressures and velocities are almost zero during diastole, which suggests that wave activity is occurring mainly during systole.

4 Discussion

This work has shown that it is possible to interpret the measured arterial pressure, $P$, and velocity, $U$, at an arbitrary location in a large artery as the sum of a reservoir components, $\bar{P}(t)$ and $\bar{U}(x,t)$, governed by Frank’s windkessel model, and wave components, $p(x,t)$ and $u(x,t)$, that vary in time and with distance along the arteries. This separation technique is based on the work by Wang et al. at the ascending aorta [8] and in the central venous system [9]. We have expanded their *ad hoc* ideas to the larger systemic arteries by proposing a separation algorithm that does not require knowledge of the flow into the arterial system. Pressure can be separated into $\bar{P}$ and $p$ from pressure measurements alone at an arbitrary arterial location. In addition, if simultaneous flow measurements are available at the same location, our algorithm also allows us to determine $\bar{U}$ and $u$.

The applicability of our algorithm depends on the extent of validity of the following empirical assumptions: (i) the pressure waveform decay measured at different locations in the arterial
Table 1: Time constant values $\tau$ (s) calculated from simultaneous \textit{in vivo} pressure measurements in dogs. Three consecutive beats are shown: normal, long, and potentiated (Figure 9) and paced at different heart rates (Figure 8). The mean and standard deviation are $\tau = 2.3 \pm 0.2$ s.

<table>
<thead>
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<th>Location</th>
<th>Normal 110 bpm</th>
<th>Long 60 bpm</th>
<th>Potentiated</th>
<th>Normal</th>
</tr>
</thead>
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<tr>
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<td>2.50</td>
<td>2.43</td>
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<td>2.58</td>
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<td>2.16</td>
</tr>
<tr>
<td>abdominal aorta</td>
<td>2.52</td>
<td>2.72</td>
<td>2.67</td>
<td>2.37</td>
</tr>
</tbody>
</table>

The method that we propose is carried out in the time domain and makes no assumption about the periodicity of the data. This is illustrated in the results for the sequence of beats including a missing beat shown in Figure 9. The algorithm was applied to each beat separately and the calculated time constants $\tau$ for each beat are shown in Table 1. The values are reasonably constant at the different locations, which is consistent with the basic assumptions, and for the different beats, which would be expected for the acute nature of the change. The same is true for the difference between normal beats in the same dog with pacing rates of 60 and 110 bpm, although the heart rate change should be considered more of a chronic than an acute change.

In general, our algorithm cannot be applied to any part of the arterial system or any pathological state in which wave reflections affect our two empirical assumptions significantly. For this reason, the algorithm performs better with a slow heart rate and in arteries close to the ascending aorta. The coronary arteries are a prime example of a region where we would not expect our algorithm to work. Although pressure waveforms in the coronary arteries are very similar to those measured in the aorta of the same subject [14], our algorithm cannot be applied to coronary flows because wave activity prevails during all the cardiac cycle, as indicated by the
very different velocity waveforms measured in the coronary arteries. The concept of separating the measured pressure into a reservoir pressure and a wave pressure could, however, still be useful in cases, like the coronaries, where our algorithm cannot be used to separate the different pressure contributions. In those cases, it is necessary to have independent knowledge of the flow into the arterial system.

5 Conclusions

We have shown how pressure and flow waveforms measured at an arbitrary location in arteries can be separated into a reservoir and a wave components. Our model expands and reinforces the work by Wang et al. [8, 9], who proposed the wave-reservoir separation of the central aortic pressure provided that the cardiac output is known. Our separation technique enhances the understanding of the mechanics behind the characteristic shape of the arterial pressure and flow waveforms. Using in vivo and numerical data, we have shown that the highest contribution of the wave components to the total waveforms occurs during the early systolic rise. Later on the cardiac cycle, the reservoir components become dominant. Wave activity is almost inexisten during the last period of diastole. Overall, the reservoir components have a higher contribution to the total waveforms than the wave components, which highlights the importance of arterial compliance on pulse wave propagation. Wave components are tightly related to the inflow at each heart beat in early systole.

This work has concentrated on the development of an algorithm that would enable us to separate the pressure measured at an arbitrary point in the arterial system into a reservoir pressure and a wave pressure. The demonstration of the utility of this separation remains to be explored fully. Wang et al. [8, 9] have explored some of the implications of the separation, but there are many other possibilities. For example, the separation implies that the work done by the left ventricle can be divided into work done to ‘fill’ the reservoir and work done to generate the arterial waves. This separation raises interesting possibilities in the interpretation of different physiological and pharmacological interventions. Most of the perfusion of the microcirculation is a result of the reservoir pressure (note that the reservoir velocities tend to increase towards the periphery). The waves, on the other hand, are necessary to generate the reservoir pressure but have little effect on net perfusion.

The effect of the separation on wave intensity analysis also remains to be explored in detail. Figure 3 shows the difference between the $PU$-loop and the $pu$-loop, which could have significant
implications for the calculation of the local wave speed from the initial slope of the loop [17]. In this particular example, there is only a small difference in slopes, but this may not always be the case. The impact of pressure separation on the sum-of-squares method for calculation wave speed has not been studied [21]. Since the reservoir pressure is the result of a capacitive-like process, it responds less quickly to the initial compression phase of the left ventricle and so its effect on the wave intensity of the largest waves in the cardiac cycle will be minimal. For small waves resulting from wave reflections and re-reflections, the effect of subtracting the reservoir pressure from the measured pressure could have a relatively larger effect. This remains to be studied in detail.

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References


List of Figures

1 Measured $P$ (thick lines) and reservoir $\bar{P}$ (thin lines) are shown in the top panels and wave pressure $p$ (dashed lines) compared to wave velocity $u$ (thick lines) are shown in the bottom panels. Left panels show measurements in the aortic arch 11 cm from the aortic valve. Right panels show measurements in the thoracic aorta 24 cm from the aortic valve.

2 Simultaneous $P$ and $U$ recordings at four different aortic locations in a dog and their corresponding reservoir-wave separated components. Pressures in left panels, velocities in right panels. Ascending in thick solid lines, arch in thick dashed lines, thoracic in thin solid lines and abdominal in thin dashed lines.

3 $PU$-loop (thick solid line), $\bar{P}\bar{U}$-loop (thin solid line) and $pu$-loop (thin dotted line) from thoracic aorta. Note that $p + P_0$ is used for convenience.

4 Reservoir-wave separation in human descending aorta measurements. Pressure components $P$ in thick solid line, $\bar{P}$ in thin solid line, and $p$ in thin dashed line, $PU$-loop (thick solid line), $\bar{P}\bar{U}$-loop (thin solid line) and $pu$-loop (thin dashed line), and velocity components $U$ in thick solid line, $\bar{U}$ in thin solid line, and $u$ in thin dashed line.

5 Wave separation in human descending aorta measurements using the total $P$ and $U$ (left panels) in comparison to wave separation after extraction of $\bar{P}$ and $\bar{U}$ (right panels). Measured $P - P_0$ in thin solid line, in top panels. $P_+$ in thick solid line and $P_-$ in thick dashed lines in top left panel. Notice that $P_0$ was subtracted from $P$ before separation for ease of comparison. $\bar{P} - P_0$ in thick dash-dot line, $p_+$ in thick solid line and $p_-$ in thick dashed line in top right panel. Measured $U$ in thin solid line, in lower panels. $U_+$ in thick solid line and $U_-$ in thick dashed line in lower left panel. $\bar{U}$ in thick dash-dot line, $U_+$ in thick solid line and $u_-$ in thick dashed line in lower right panel. Subscripts $+$ and $-$ indicate forward and backward travelling waves, respectively, with $c = 5.88$ m/s.

6 Total (thick lines) and reservoir (thin lines) pressures at the ascending aorta (Asc), aortic arch (Arch), left brachial (Brach), upper abdominal (U Abd), lower abdominal (L Abd), and left femoral (Fem) arteries simulated using a numerical model containing the 37 largest conduit arteries in humans. $x$ indicates the distance from the inlet of the ascending aorta.

7 Total, reservoir and wave pressures (left) and velocities (right) at the locations shown in Figure 6. Asc in thick solid lines, Arch in thick dashed lines, Brach in thin dashed lines, U Abd in dotted lines, L Abd in thick dotted-dashed lines and Fem in thin solid lines.

8 Reservoir-wave pressure components from the thoracic aorta for the same dog (as shown in Figure 1) paced at two different heart rates: 110 bpm (left panel) and 60 bpm (right panel). Measured $P$ (thick lines) and reservoir $\bar{P}$ (thin lines) are shown in the top panels and wave pressure $p$ (thin dashed lines) compared to wave velocity $u$ (thick lines) are shown in the bottom panels.
Three consecutive beats simultaneously measured in four different locations in the aorta of a paced dog at a heart rate of 110 bpm. Measured $P$ thicklines and reservoir $\bar{P}$ (thin lines) are shown in the top panel; and wave $p$ (thin lines) in comparison to wave $u$ (thick lines) in the lower panel. $x$ refers to the distance from the aortic valve. A normal beat is followed by a long beat and a potentiated beat.

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1. Time constant values $\tau$ (s) calculated from simultaneous in vivo pressure measurements in dogs. Three consecutive beats are shown: normal, long long and potentiated (Figure 9) and paced at different heart rates (Figure 8). The mean and standard deviation are $\tau = 2.3 \pm 0.2$ s.
List of Notations

\( \gamma \) local time constant
\( \rho \) blood density
\( \tau \) time constant of the exponential diastolic decay
\( a \) inverse of \( \gamma \)
\( b \) inverse of \( \tau \)
\( c \) pulse wave speed
\( C \) compliance of the arterial system
\( p \) wave pressure
\( p_+ \) separated forward travelling pressure wave using \( p \)
\( p_- \) separated backward travelling pressure wave using \( p \)
\( P \) measured pressure
\( P \) reservoir pressure
\( P_\infty \) pressure at which flow in the microcirculation is zero
\( P_0 \) measured pressure at the onset of ejection
\( P_0 \) reservoir pressure at the onset of ejection
\( P_{in} \) pressure at the root of the aorta
\( P_+ \) separated forward travelling pressure wave using measured \( P \)
\( P_- \) separated backward travelling pressure wave using measured \( P \)
\( Q_{out} \) blood out of the arteries through the microcirculation
\( Q_{in} \) inflow to the arterial system from the left ventricle
\( R \) effective resistance of the peripheral systemic arterial circulation
\( \dot{R} \) effective resistance of the vessels downstream of the measurement site
\( t \) time
\( T \) time at which a heart beat ends
\( T_N \) time at which the aortic valve shuts at the end of systole
\( u \) wave velocity
\( u_+ \) separated forward travelling velocity wave using \( u \)
\( u_- \) separated backward travelling velocity wave using \( u \)
\( U \) measured velocity
\( \dot{U} \) reservoir velocity
\( U_+ \) separated forward travelling velocity wave using measured \( U \)
\( U_- \) separated backward travelling velocity wave using measured \( U \)
\( V \) volume of the arterial system
\( x \) distance

Abd abdominal aorta
Arch aortic arch
Asc ascending aorta
Brach brachial artery
Fem femoral artery
Thor thoracic aorta
Figure 1: Measured $P$ (thick lines) and reservoir $\bar{P}$ (thin lines) are shown in the top panels and wave pressure $p$ (dashed lines) compared to wave velocity $u$ (thick lines) are shown in the bottom panels. Left panels show measurements in the aortic arch 11 cm from the aortic valve. Right panels show measurements in the thoracic aorta 24 cm from the aortic valve.
Figure 2: Simultaneous $P$ and $U$ recordings at four different aortic locations in a dog and their corresponding reservoir-wave separated components. Pressures in left panels, velocities in right panels. Ascending in thick solid lines, arch in thick dashed lines, thoracic in thin solid lines and abdominal in thin dashed lines.
Figure 3: $PU$-loop (thick solid line), $P\bar{U}$-loop (thin solid line) and $pu$-loop (thin dotted line) from thoracic aorta. Note that $p + P_0$ is used for convenience.
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Figure 6: Total (thick lines) and reservoir (thin lines) pressures at the ascending aorta (Asc), aortic arch (Arch), left brachial (Brach), upper abdominal (U Abd), lower abdominal (L Abd), and left femoral (Fem) arteries simulated using a numerical model containing the 37 largest conduit arteries in humans. $x$ indicates the distance from the inlet of the ascending aorta.
Figure 7: Total, reservoir and wave pressures (left) and velocities (right) at the locations shown in Figure 6. Asc in thick solid lines, Arch in thick dashed lines, Brach in thin dashed lines, U Abd in dotted lines, L Abd in thick dotted-dashed lines and Fem in thin solid lines.
Figure 8: Reservoir-wave pressure components from the thoracic aorta for the same dog (as shown in Figure 1) paced at two different heart rates: 110 bpm (left panel) and 60 bpm (right panel). Measured $P$ (thick lines) and reservoir $\bar{P}$ (thin lines) are shown in the top panels and wave pressure $p$ (thin dashed lines) compared to wave velocity $u$ (thick lines) are shown in the bottom panels.
Figure 9: Three consecutive beats simultaneously measured in four different locations in the aorta of a paced dog at a heart rate of 110 bpm. Measured $P$ thicklines and reservoir $\bar{P}$ (thin lines) are shown in the top panel; and wave $p$ (thin lines) in comparison to wave $u$ (thick lines) in the lower panel. $x$ refers to the distance from the aortic valve. A normal beat is followed by a long beat and a potentiated beat.