9 Pulsatile Haemodynamics and Arterial Impedance

David Fitchett and Michael F. O'Rourke

The arterial system acts as a conduit to deliver oxygenated blood to the tissues, and as a compliant cushion to dampen pressure and flow oscillations and convert the intermittent flow input from the left ventricle into a near-continuous flow output at tissue level. The intermittent ejection of blood from the left ventricle into the aorta results in pulsatile pressure and flow throughout the arterial system. The time sequence and magnitude of left ventricular ejection is determined by the coupling between the hydraulic load imposed by the arterial system and the contractile status of the left ventricle. The instantaneous value of the pulsatile pressure and flow in the arterial system is, in turn, determined by both the left ventricular ejection and the properties of the arterial system.

In this chapter, we will consider the properties and models of the arterial system that provide a quantitative description of the observed physiology. The chapter will discuss how changes to both the arterial system and cardiac function influence pressure and flow waves measured at various locations in the arterial tree, to better understand problems of left ventricular/arterial interaction which occur with ageing and in disease.

The elastic properties of the arterial system afford a buffer or cushion, to limit the rise in pressure that occurs as the heart ejects the stroke volume into the aorta. Stephen Hales in the eighteenth century likened the arterial system to the air reservoir of a fire engine which changed the intermittent input of water from the hand pump, to a steady flow at the nozzle. (Fig. 9.1) – now termed the Windkessel model.

While the Windkessel model of the arterial system provides a simple quantitative description of pressure measurements resulting from intermittent flow, it fails to reproduce observed changes in arterial pressure and flow waves throughout the arterial network. In particular, pressure and flow waves travel at a finite speed in the arterial tree, and are reflected at points of discontinuity, particularly at the entry to high resistance, low-calibre arterioles (as originally suggested by William Harvey 1957).

Figure 9.2 shows pressure in the ascending aorta of a rabbit under three conditions, which are also seen in humans when (1) blood pressure is high, the aorta is stiff and the rate at which the pressure wave travels (the pulse wave velocity) is high, (2) normal conditions where reflection from points of discontinuity is apparent as a prominent diastolic wave, and (3) an arteriolar dilating drug has decreased wave reflection (Wetterer 1954; O'Rourke 1970a). These pressure and flow waves

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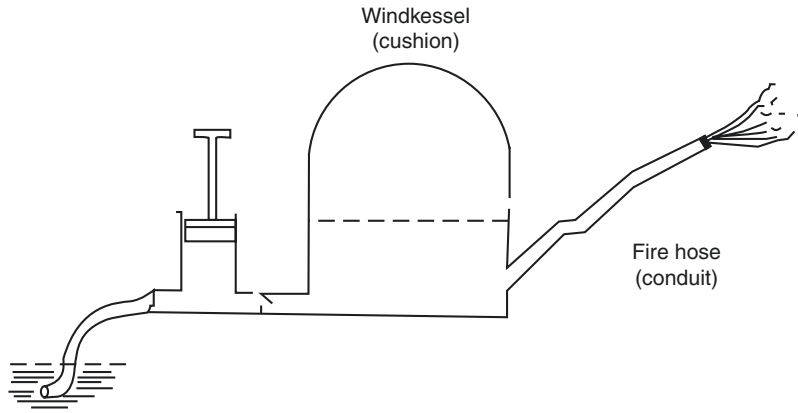

Fig. 9.1 The arterial system was compared to the fire engine of the eighteenth century by Stephen Hales. The ejection of water into the dome results in compression of the air – storing elastic energy which is returned between pump strokes converting the intermittent flow from the pump into a continuous flow delivered to the fire hose. (Reprinted from Hales 1769)

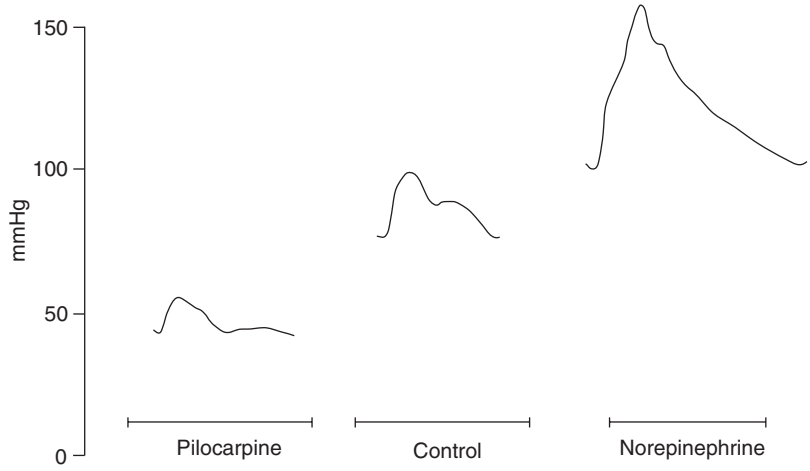

Fig. 9.2 Changes in ascending aortic pressure and flow in a rabbit (1) with severe hypertension induced with norepinephrine, (2) Control state (3) with hypotension induced with pilocarpine. Under normal resting states there is a prominent diastolic wave due to wave reflections. With severe hypertension the reflected wave returns earlier enhancing systolic pressure. During hypotension reflected waves are reduced. (Data from Wetterer (1954). Reproduced from O'Rourke (1970a) with permission of Wolters Kluwer Health, Inc.)

were recorded in the same animal just minutes apart. It is clear that the Windkessel model is inappropriate when blood pressure, heart rate, vasomotor tone, and cardiac properties are changing. The Windkessel model is of no value where it is needed most – in humans monitored during surgery and in intensive care units.

Arterial Wall Properties and Pulsatile Haemodynamics

Increased arterial stiffness and an increase of peripheral resistance are the hemodynamic determinants of hypertension (Ting et al. 1995). Hypertension increases the load on the heart and

stress on the arteries, and results in left ventricular hypertrophy, an increased risk of stroke, heart failure, renal failure and dementia.

The arterial wall shows viscoelastic properties, which can be quantified in vivo. Arterial compliance is the change in volume for a unit change in pressure ($\Delta V/\Delta P$). Distensibility is the compliance normalized for the initial volume of the arterial segment ($\Delta V/\Delta P)/V_0$. Peterson's elastic modulus (E_p) is the distensibility described in terms of arterial diameter: $E_p = D_o\,(P_s - P_d)\,/(D_s - D_d)$: where P_s and P_d are the systolic and diastolic arterial pressures, and D_s and D_d the systolic and diastolic diameters of the artery. The arterial stiffness or elastance is the inverse of arterial compliance or distensibility. Calculation of local arterial E_p is readily determined by vascular ultrasound measurements of systolic and diastolic arterial diameter (Baltgaile 2012) with simultaneous pressure measurements and is applicable to measurements in the aorta (ascending, arch, and descending), carotid, brachial, radial, iliac, and femoral arteries. Ideally, pressure changes should be measured at the same location as the dimension change, or using calculated central pressure determined from peripheral pressure as described below.

Arterial distensibility decreases in the axial direction (increasing when measured at a distance further from the heart) and as mean arterial pressure increases. At lower levels of distension, the elastic properties of the arterial wall result from extension of the arterial elastin, yet with greater stretch the less distensile collagen fibres take the load. Consequently, comparative measurements of elasticity, distensibility, or pulse wave velocity should be made at the same distending pressure.

Pulse wave velocity (C_o) in an elastic tube is related to the arterial elastance by the Moens-Korteweg or Bramwell-Hill equations:

$$C_o = \sqrt{V\left(\Delta P / \Delta V\right)} \quad \text{or} \quad C_o = \sqrt{\left(Eh / 2r\rho\right)}.$$

where E is Young's modulus of elasticity of the artery, h wall thickness, V arterial volume, ΔP pulse pressure, and ΔV change in arterial volume. Pulse wave velocity should be distinguished from blood flow velocity: Pulse wave velocity measures the speed of transfer of energy through the arterial wall (velocity range 4–12 m/s), whereas blood flow velocity measures the transfer of mass along the blood column (velocity range 10–100 cm/s).

Using measurements of the change of arterial radius (dR) between diastole and systole in an arterial segment, pulse pressure (dP), and blood density (ρ), the wave velocity can be calculated:

$$C_o = \sqrt{RdP\,/\,\rho 2dR}.$$

Arterial pulse wave velocity is usually measured using external pressure transducers, from the time difference between the foot of the pressure wave over long arterial segments (e.g. carotid (as a surrogate for central pressure wave) to femoral) (Nichols and O'Rourke 1998). Local arterial pulse wave velocity can be determined using either ultrasound or MRI measurements of arterial flow and dimensions, in the aorta and peripheral arteries. From the central aorta to the conduit arteries in the legs, the arteries become far more numerous, smaller, and have less elastin and more smooth muscle: consequently, pulse wave velocity increases. These changes are most marked in the thoracic and abdominal aorta, and least marked in the upper limb vessels. Increasing arterial pressure reduces distensibility of the arteries, resulting in an increase in pulse wave velocity.

With ageing and hypertension, arteries become less distensile, consequently pulse wave velocity increases.

Current indices of arterial distensibility are non-linear. We seek indices which have linear relationships or where non-linearities are small, or can be controlled or allowed for. Even peripheral resistance (Mean pressure / Mean flow) is non-linear.

Up until 1960, the approach to pulsatile haemodynamics in arteries concentrated on measurements of arterial stiffness. Anatomists however pointed out how the dimensions of animals could alter wave transmission and result in reflected waves as well as waves traveling forward by increasing the number of peripheral

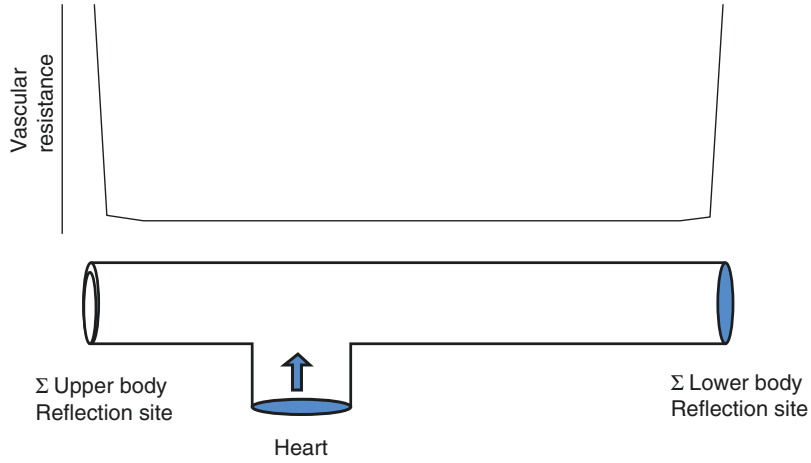

Fig. 9.3 Asymmetric T model of the arterial circulation showing the location and very abrupt change of resistance over a very short distance. (Resistance is shown on a logarithmic scale). These marked impedance mismatches provide multiple reflecting sites that can be lumped into upper and lower body sites. In this model, the lower body is the major reflection site with less intense reflections returning from the upper body as a consequence of the low resistance cerebral vasculature. The measured pressure and flow waves result from the interaction of forward and reflected waves from both sites. (Reproduced based on diagram from O'Rourke et al. (2018) with permission of Oxford University Press)

branches, their distance from the heart and their cross-sectional area (Milnor 1979). Moreover, such reflected waves have the potential to interfere with outgoing pulse wave in the proximal arteries augmenting systolic pressure and reducing mean diastolic pressure on the pulse wave velocity. The nature and degree of such augmentation will depend on the pulse wave velocity.

Of great significance is the recent recognition of the importance of the location of the peripheral resistance vessels being just a few millimetres from the low resistance conduit arteries (O'Rourke et al. 2018). This confirms the work of Hamilton, Dow (Hamilton 1939), Remington (Remington and Wood 1956), and others that the peripheral resistance is the site of strong wave reflection (Fig. 9.3).

Vascular Impedance

A large jump in the field took place in the 1960s with the introduction of frequency domain analysis of the arterial pulse by an English group of McDonald, Womersley, and Taylor, and a US group at the NIH in Bethesda led by Donald Fry. Michael Taylor liaised with the UK and US groups on his return from London to Sydney. Up until 1960, Taylor sought information on optimal function of the arterial tree as based upon optimal distensibility (Taylor 1967). He progressed to anatomical as well as physical preparations as required for a comprehensive study. Taylor pressed the search for answers through determination of vascular impedance in animals of different size and shape, and in humans; Michael O'Rourke was his graduate student with training in anaesthesiology and intensive care.

The pressure developed by the contracting heart is determined by the force of contraction of the heart muscle and the external opposition to ventricular outflow. Characterization of the external opposition to left ventricular ejection needs to take into account the major components of the arterial load: (1) resistance, (2) blood viscosity, (3) arterial wall visco-elastance, (4) inertia, and (5) wave reflection. The implications of pulsatile haemodynamics on left ventricular/arterial *coupling will be discussed in a later section of this chapter*.

Arterial resistance is determined by blood viscosity and is inversely proportional to the fourth power of the radius of the vessel.

$$R = 8\mu L / \pi r^4 \quad \mu \text{ viscosity}, \quad L \text{ vessel length}, \quad r \text{ radius}$$

Consequently, the most important component of peripheral arterial resistance is at the level of the smallest vessels or arterioles. If flow from the heart was constant, the steady pressure (P_{mean}) generated would depend only on arterial resistance: $P_{mean} = R\,Q_{mean}$ where P_{mean} and Q_{mean} are the mean pressure and flow, respectively. However, the intermittent nature of left ventricular ejection presents a more complex case. In addition to the peripheral resistance, in the intermittent case, there is the possibility of reflected waves from more distant points in the vascular tree as well as the elastic nature of the arteries (which distend and recoil in systole and diastole respectively, resulting in a time-dependent elastic contribution to pressure). The pulsatile hydraulic load on the heart will reflect contributions from all of these.

The pulsatile left ventricular hydraulic load on the heart could be described in either the time or frequency domains. Time domain assessments of the pulsatile arterial load include measures of total arterial compliance (e.g. stroke volume/pulse pressure) or effective arterial elastance (end systolic pressure / stroke volume).

However, frequency domain assessment of the arterial load by the aortic input impedance provides the best description of the components of steady state and pulsatile arterial load. Impedance is a frequency-dependent measure of opposition to pulsatile flow. Resistance is the opposition to steady or non-oscillatory flow and is the impedance at zero frequency. Input impedance is determined by the properties of the arterial system, which include peripheral arteriolar resistance, the viscoelastic properties of the arteries, inertial forces associated with changing flow, the viscosity of the blood and the impact of reflected waves, as well as the size and shape of the animal.

Characteristic Impedance (Z_c) It is the relationship between pulsatile pressure and pulsatile flow ($\Delta P/\Delta V$) measured at the same site in the absence of reflected waves. *Z*c is determined by the physical properties of the arterial system such as the elastic modulus of the artery and the inverse of the cross-sectional area. Consequently, Z_c is linearly related to the pulse wave velocity (Co) and is also directly related to blood density.

$$Z_c = \rho c_o / \pi r^2$$

Wave velocity (C_o), in turn, is directly related to $\Delta P/\Delta V$ by the "Water Hammer" equation

$$C_o = \Delta P / \rho \Delta V$$

where ΔV is the change in blood flow velocity, ΔP the change in pressure, and ρ is blood density.

Z_c can be estimated from the change in pressure occurring simultaneously with peak flow.

$$Z_c = \Delta P_Q / \Delta Q = \left(P_i - P_d\right) / \text{peak flow}$$

where P_i is the pressure at the inflection point of aortic pressure where peak flow occurs (see Fig. 9.4), P_d is end diastolic pressure. This estimate assumes that aortic pressure up to this time point is not changed by wave reflections.

Z_c may also be obtained by Fourier analysis of arterial pressure and flow (as described below) by averaging the moduli of impedance at frequencies above the first minimum. There is good correlation between the peak flow method and the results obtained by Fourier analysis (Dujardin and Stone 1981).

When Z_c is determined from flow change in volume units of cm^3/s, Z_c has the same units as arterial resistance (dyne-s/cm^5). However, characteristic impedance is not a true resistance and can only be considered in the context of oscillatory phenomenon. Z_c is best expressed in terms of velocity (cm/sec) as dyne.s.cm^{-3}. Z_c numerically (in volume units) is about 5–7% of peripheral vascular resistance. Measurements of Z_c in man show increasing Z_c (in velocity units) with age, hypertension, and in patients with heart failure, as one would expect, on account of the relationship of Zc with pulse wave velocity and arterial stiffness.

Input Impedance (Z_I) It describes the actual relationship between observed pressure and flow,

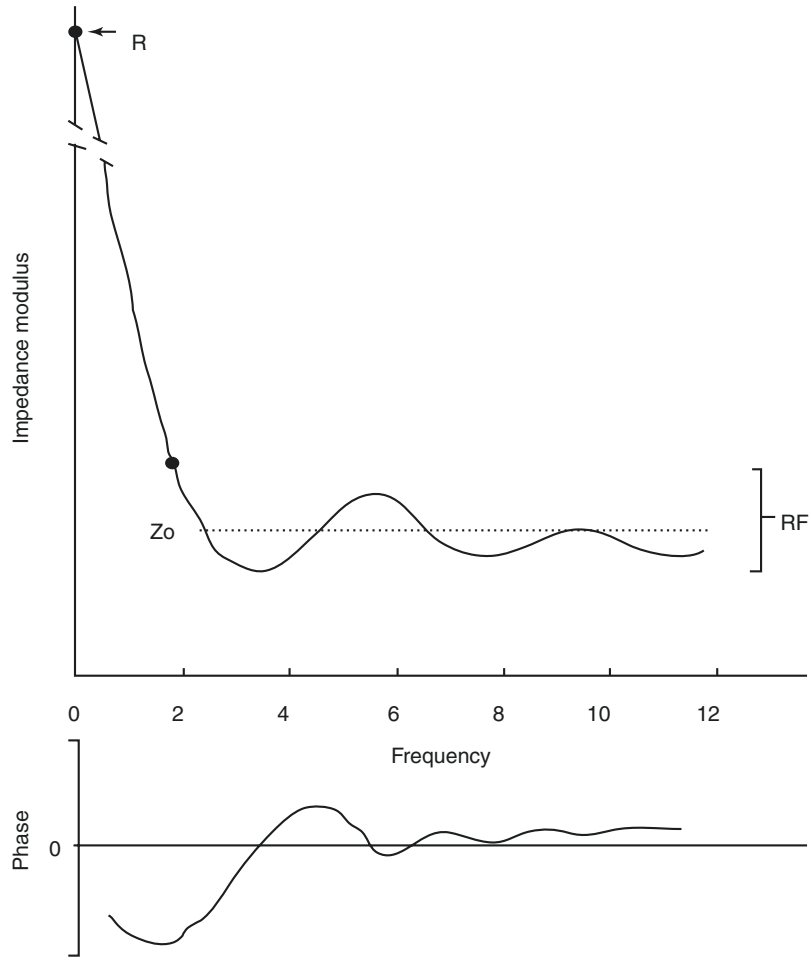

Fig. 9.4 Hypothetical aortic input impedance spectrum. The first minimum of the impedance modulus occurs close to the frequency where the phase crosses zero. The characteristic impedance (Z_c) is the average of the impedance moduli beyond the first minimum. A measure of wave reflection (RF) is the amplitude of the impedance moduli variation above the first minimum. (From Yin 1987. Reproduced with permission of Springer Nature)

in the frequency domain. The measured pressure and flow waves are the summation of incident and reflected backward traveling waves. Thus, input impedance (Z_i)

$$Z_I = \left(P_f + P_b\right) / \left(Q_f + Q_b\right)$$

in contrast to Z_c reflects the effect of reflected waves in addition to the physical properties of the arterial system such as vessel radius and viscoelastic properties. In the ascending aorta, the aortic input impedance describes the actual arterial hydraulic load on the ejecting left ventricle.

Fourier transform analysis of pressure and flow recordings taken in the arterial system yields a Fourier series – a weighted sum of sine waves at specified frequencies which would yield the observed pressure and flow recordings. These frequencies are harmonics – multiples of the fundamental frequency (60/HR where HR is heart rate in beats/minute). The observed waveforms can be reconstructed with increased accuracy by including more harmonics.

Z_i is determined for each individual harmonic (h) as an amplitude (modulus) $Z_i(h)$ and a phase angle ($\theta(h)$). The phase angle ($\theta(h)$) is the phase difference between the pressure (β) and flow (ϕ) harmonic and is positive when the flow harmonic leads the pressure harmonic.

$$Z_i\left(h\right) = \left|P\left(h\right)\right| / \left|Q\left(h\right)\right| \quad \theta\left(h\right) = \phi - \beta$$

An aortic input impedance spectrum is a graphical representation of the phase angle and impedance moduli for each frequency (Fig. 9.4).

Impedance values decline from a high value at 0 Hz (the peripheral vascular resistance) to a minimum at approximately 3.5 Hz. At about this frequency, the phase angle crosses zero. A negative phase angle indicates that the flow harmonic leads the pressure harmonic. The impedance modulus over a range of frequencies oscillates around a mean value due to the impact of wave reflections. The magnitude of the oscillations of impedance moduli approximates the reflection index. The mean value of input impedance at frequencies above approximately 3 Hz is taken to represent the characteristic impedance (Z_c).

The frequency at which the first minimum of impedance modulus and the zero crossing point of the phase angle allows calculation of the wavelength of the travelling wave and, consequently, the distance to the major site for wave reflection. The reflection site is at a quarter of a wavelength ($\lambda/4$) where $\lambda = C_o/f$, C_o is the pulse wave velocity and f the frequency of the first impedance modulus minimum. The distance to the effective reflecting site is $d = C_o/4f$. In normal human studies, with an impedance minimum at 3.5 Hz and an average aortic pulse wave velocity of 750 cm/sec, the distance from the ascending aorta to the effective reflecting site is 54 cm.

The significance of the effect of wave reflections on the measured pressure depends upon their magnitude and the phase difference between reflected and incident waves, which in turn depends upon both the distance from the reflection site and the wave velocity. If the phase difference is 0°, the reflected wave will increase the net pressure (constructive interference), whereas if the phase difference is 180° the reflected wave will decrease the measured pressure (destructive interference).

The validity of impedance as a measure of the relationship between pressure and flow and the application to transmission line theory depends upon the linearity of the relationship between pressure and flow. For example, in the Fourier analysis, each harmonic of pressure and flow is considered to be uniquely related and not dependent upon other frequency components, that is, there is no harmonic interaction. Theoretical and experimental studies have demonstrated that any non-linearities between pressure and flow are surprisingly small and can be neglected as a first-order approximation.

Arterial Wave Reflections

The arterial system is a network of distensile tubes with multiple branches. The arterial pressure wave is transmitted along the conduit arteries at the local pulse wave velocity. On encountering the branching points and the arteriolar network, a proportion of the wave energy is reflected back. Such reflecting points are termed points of impedance mismatch. As above, the reflected backward travelling wave interacts with the forward wave with the resulting observed wave dependent on both the magnitude and the phase difference of the forward and backward waves. Using parameters acquired from impedance data, the arterial pressure and flow waves can be analysed into forward and reflected waves (Fig. 9.5).

Reflected waves are seen in arterial pressure recordings, especially in animals and young people in whom the amplification is more pronounced and the wave velocity slower such that reflected waves return later and after the initial impulse (Fig. 9.6).

Wave reflection is responsible for the increase in systolic pressure observed as the pressure wave travels from the central aorta to the periphery. Over the same distance, diastolic pressure falls with a consequent greater fluctuation of pressure around a slightly lower mean pressure (by just 1–2 mmHg) (Pauca et al. 1992).

Wave reflection can be quantified by the magnitude of the variation of the input impedance at frequencies above the first minimum. An estimate of wave reflection can also be made by calculation of a reflection coefficient (Γ): $\Gamma = (Z_T - Z_C)/(Z_T + Z_C)$, where Z_T is the peripheral resistance and Z_C is characteristic impedance. However, Γ is frequency dependent and falls from a value of approximately 0.8 at the fundamental heart rate frequency to very low levels above the first impedance maximum.

Wave reflection can be modified by physiological, pathological, and pharmacological influ-

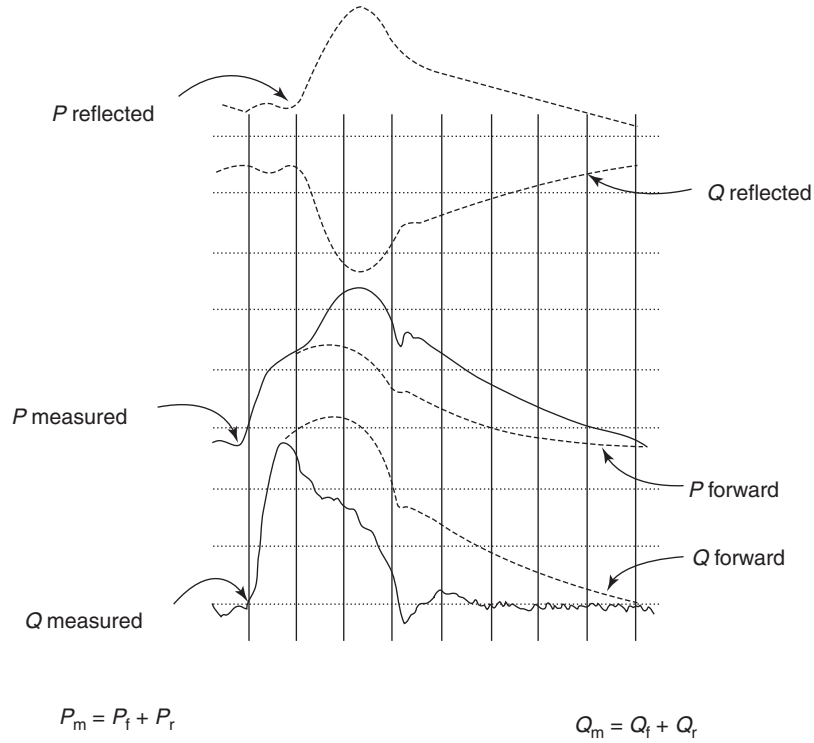

Fig. 9.5 Measured aortic pressure is analysed into forward and backward (reflected) waves. The reflected wave results in the late systolic peak of pressure. (From Yin 1987. Reproduced with permission of Springer Nature)

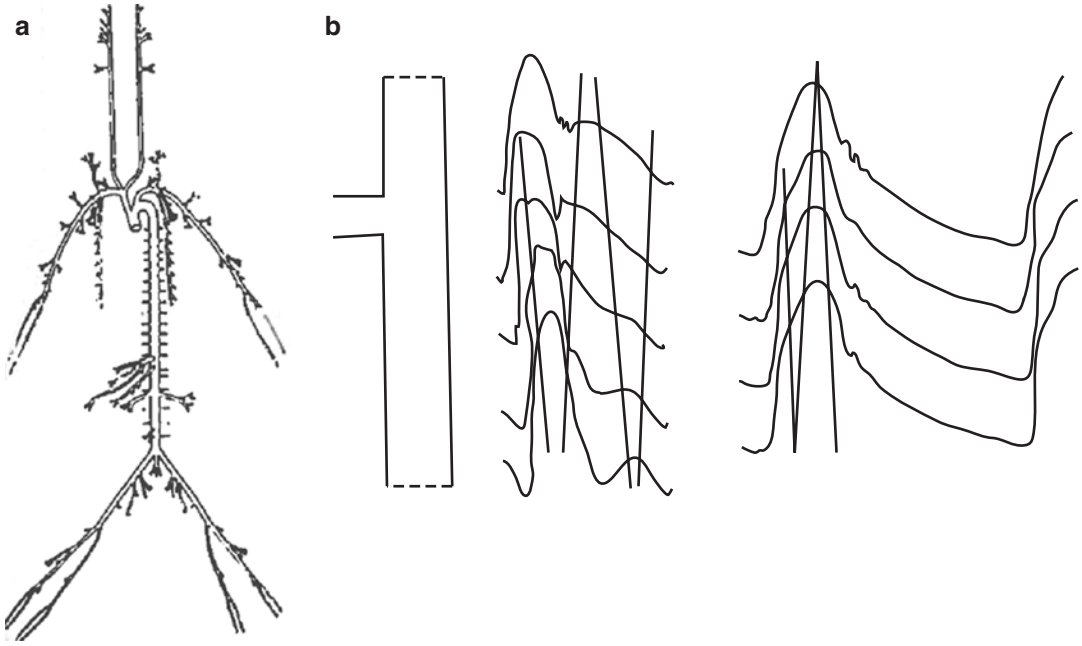

Fig. 9.6 The arterial system is modelled as an asymmetric *T* with reflections originating in the upper and lower parts of the body. (**a**) In a young subject or in animals, systolic and pulse pressure increase as the pressure wave is transmitted from the central aorta to the periphery due to the impact of wave reflections. (**b**) In an older human subject with stiffened arteries wave reflections return earlier augmenting late systolic pressure and a lesser effect on pressure amplification between central and peripheral pressures. (From Wave Travel and Reflection in the Arterial System. O'Rourke 1971. Modified with permission of Elsevier Science & Technology Journals)

ences. Ageing and arterial disease are associated with an increase in late systolic aortic pressure due to increased wave reflection arriving early during systole as consequent to the increased pulse wave velocity.

Laskey and Kussmaul (1987) show that exercise reduces the magnitude of the reflected wave. The reduction of the late systolic peak of aortic pressure observed during a Valsalva maneuver is associated with reduced fluctuation of the impedance spectrum indicating a reduction of wave reflections (Murgo et al. 1981).

Vasodilators such as nitroprusside reduce mean arterial pressure and peripheral vascular resistance (Merillon et al. 1982). As a result of the reduction of pulse wave velocity, the input impedance curve is shifted to the left, suggesting a delay in the timing of wave reflections. In addition, the amplitude of the impedance modulus of the first two harmonics is reduced, indicating an overall reduction of wave reflections. In contrast, nitroglycerin reduces late systolic pressure in the ascending aorta as well as the fluctuations of the modulus of the impedance spectrum with little effect on peripheral arterial resistance or change in the frequency of the modulus minima (Fitchett et al. 1988a; Yaginuma et al. 1986). These observations indicate that nitroglycerin reduces wave reflection without changing peripheral arteriolar resistance.

Changes of Waveforms in the Arterial System

Differences between peripheral and central arterial pressures have important practical consequences. Pressure measured in the radial artery differs from both central aortic and intra-cranial arterial pressure. Central aortic pressure is both the pressure load encountered by the ejecting left ventricle and the driving pressure for coronary perfusion in diastole, while carotid arterial pressure is the cerebral perfusion pressure.

Differences in central and peripheral pressures become accentuated during procedures that cause hypotension as shown in Fig. 9.7.

Radial arterial pressure is frequently used for patient monitoring in critically ill patients. With vasoconstriction in patients with shock, wave reflections can be increased, potentially increasing pressure amplification and exaggerating the difference between central aortic and radial arterial systolic pressure (Fig. 9.8).

However, radial arterial pressure can underestimate central aortic pressure in these patients possibly due to peripheral arterial vasoconstriction. Consequently, measurement of peripheral arterial pressure by radial arterial pressure monitoring provides only a limited assessment of aortic systolic pressure. There is a need for fur-

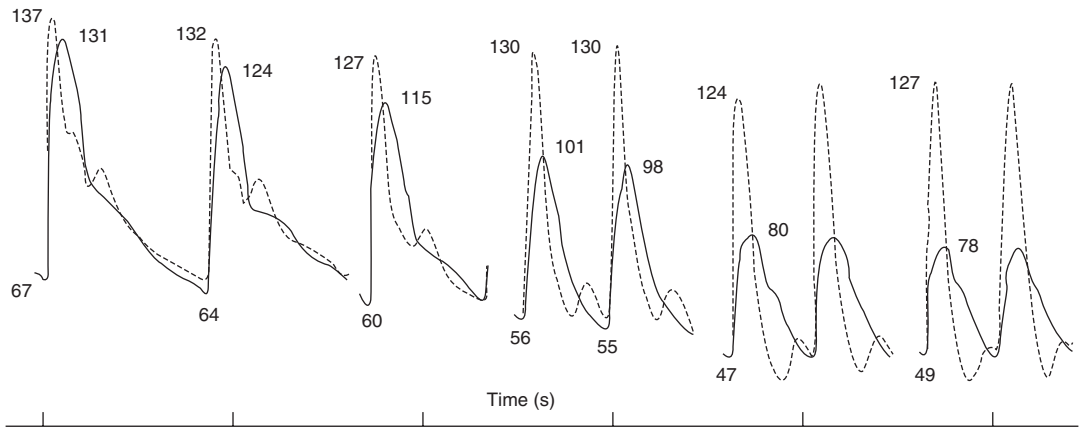

Fig. 9.7 Pressure waves simultaneously recorded from the radial artery (dashed line) and from the abdominal aorta (solid line) during the course of a hypotensive reaction to rapid intra-aortic injection of isotonic electrolyte solution (numbers represent mmHg). (From Remington and Wood 1956. Reproduced with permission of American Physiological Society)

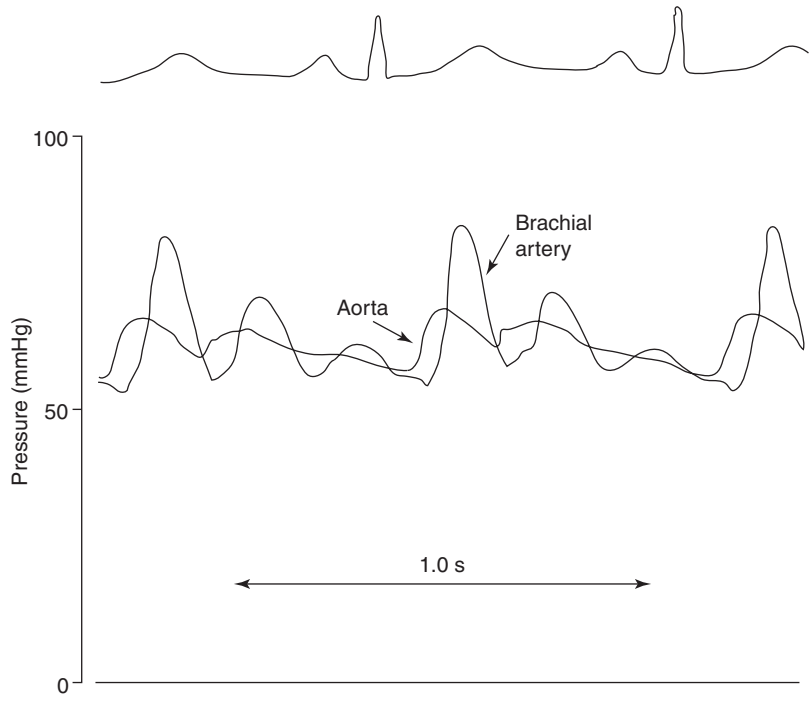

Fig. 9.8 Radial and aortic pressures in a patient with hypotension, vasoconstriction, and an obstructed carotid artery, showing the exaggerated diastolic pressure waves in the radial arterial pressure due to delayed wave reflection. Note the substantial difference between peak radial and aortic pressure indicating that radial arterial systolic pressure can overestimate central systolic pressure. (From O'Rourke 1970b. Reproduced with permission of Oxford University Press)

ther studies of pressure wave transmission between the central aorta and radial artery in patients with shock, to better understand the changes and clinical implications. Anaesthetists and intensivists are well aware of such problems which can also damp the pressure waveform. The appropriate action is to replace the radial cannula with a short catheter and advance it to the subclavian artery.

Vasodilators such as nitroglycerine reduce ascending aortic systolic pressure more than is apparent from changes of radial arterial pressure (Fig. 9.9) due to the reduction of reflected waves as indicated by the changes of aortic input impedance without any significant reduction in systemic vascular resistance (Fig. 9.10).

Non-invasive Assessment of Pulsatile Haemodynamics and Central Aortic Pressure

Analysis of the radial arterial pressure contour can provide information about the transmission of the pressure pulse from the aorta to the periphery. The radial arterial pulse characteristically has an early systolic peak followed by a late systolic shoulder and another wave after the dicrotic notch as shown in Fig. 9.9.

The relationship between recordings of aortic and radial arterial pressure waves and the moduli and phase of the individual Fourier harmonics – the output of any given input – can be expressed as a transfer function for each harmonic. In the upper limb, the transfer function is remarkably constant over a wide range of ages (Karananoglu et al. 1997) and after the administration of vasodilators such as nitroglycerine (O'Rourke et al. 1990). Consequently, the same transfer function can be used to generate central aortic pressure from recordings of the radial pressure wave in a wide range of conditions.

The radial arterial pressure wave can be reliably recorded non-invasively using applanation tonometry (Drzewiecki et al. 1983). By applying the transfer factor to the radial pressure harmonics, central aortic pressure may thus be synthesized from recordings of peripheral arterial pressure. Using this technique, it is possible to determine the impact of therapy (e.g. antihyper-

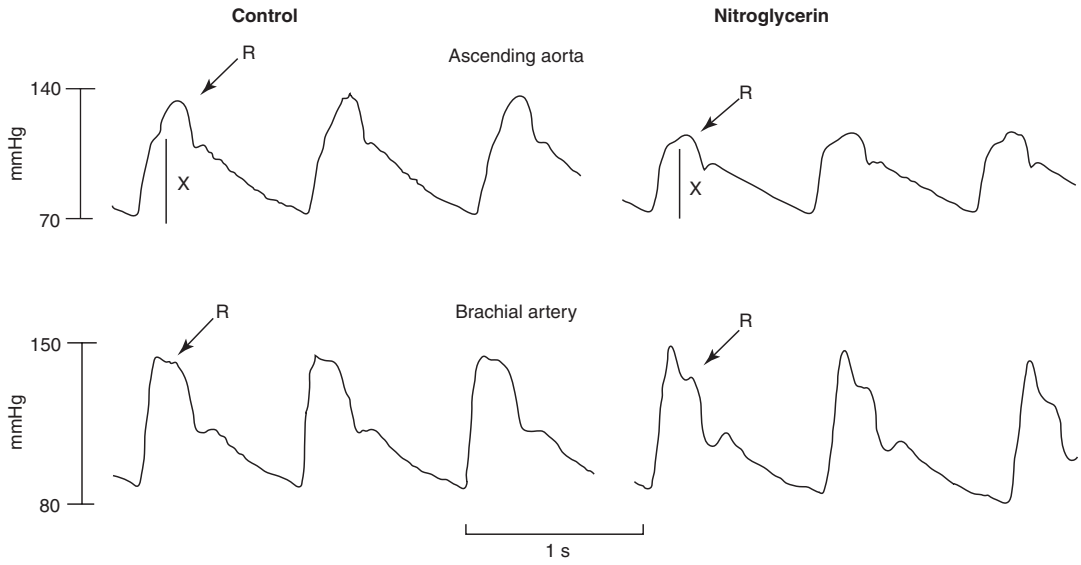

Fig. 9.9 Pressure waves recorded in the ascending aorta and brachial artery under control conditions (left) and after 0.3 mg sublingual nitroglycerine (right) in a human adult. *X*, height the pressure pulse would be without reflection (*R*). (From Kelly et al. 1990, Fig. 9.2. Reproduced with permission of Oxford University Press)

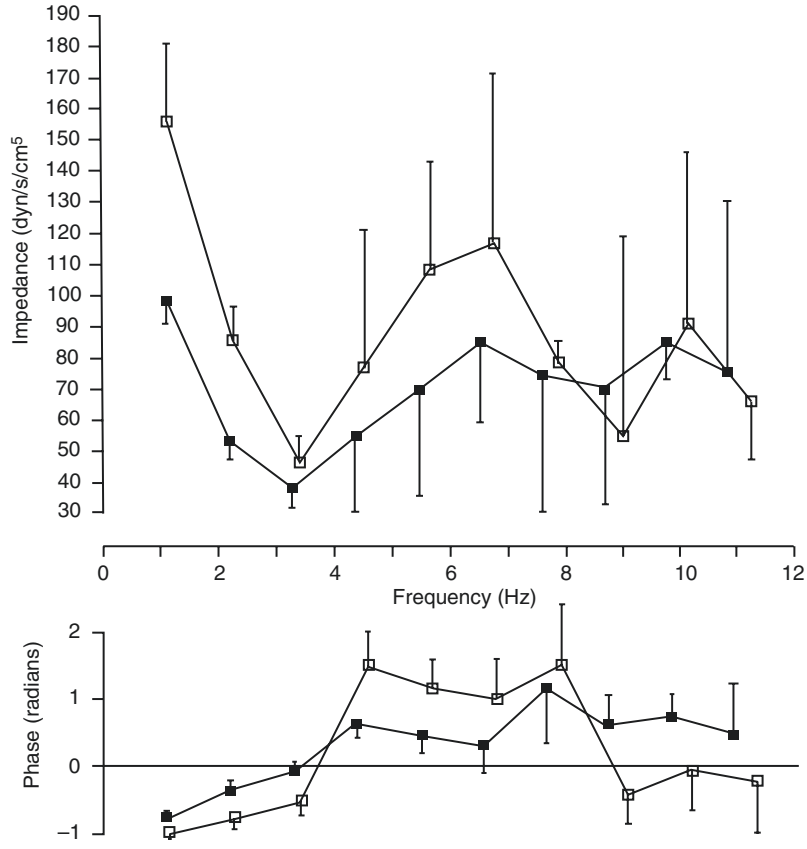

Fig. 9.10 The effect of nitroglycerin 15 μg on the aortic input impedance modulus and phase, before and during nitroglycerine infusion. Systemic vascular resistance was unchanged. There was a significant reduction in the modulus of impedance of the first harmonic and less oscillation of the modulus and phase angle spectrum indicating nitroglycerine reduces peak systolic pressure consequent to a reduction of reflections with no reduction of systemic vascular resistance. (From Fitchett et al. 1988b. Reproduced with permission of Oxford University Press)

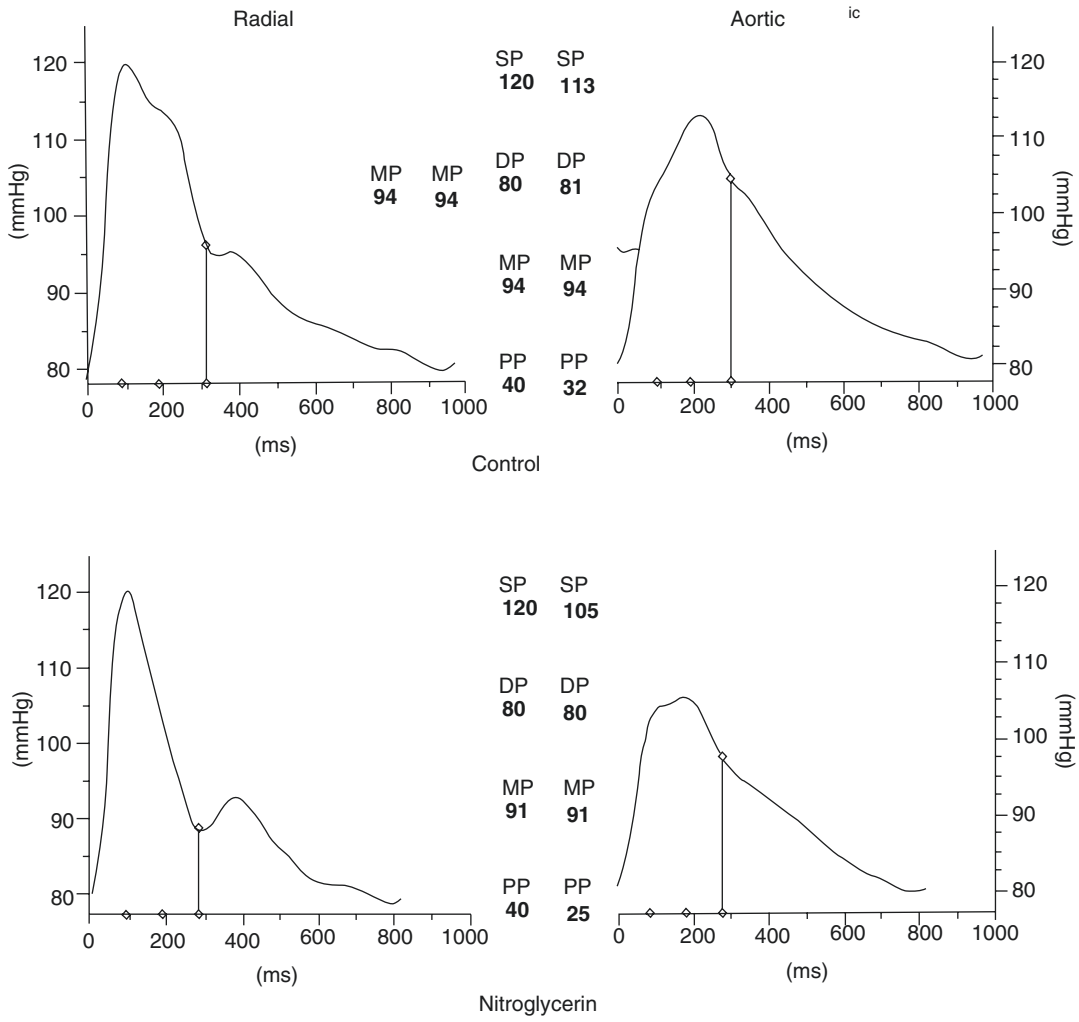

Fig. 9.11 Tonometric recordings of radial artery pressure with synthesized aortic pressure using the transfer function before and after the administration of *s/l* nitroglycerine. Similar pressure pulse changes in both the radial artery and aorta are observed to those measured with intra- arterial measurements as shown in Fig. 9.9. (From O'Rourke et al. 1993. Modified with permission of Informa UK Limited through PLSclear)

tensive and heart failure medications) on central BP and hence the impact on left ventricular loading (Fig. 9.11).

Pulsatile Haemodynamics and Ventricular Arterial Coupling

The external work performed by the heart in a cardiac cycle (or stroke work) is the integral of the pressure and flow over a cardiac cycle:

$$W\left(\text{stroke}\right) = \int PQdt$$

Total external work has two components: (1) steady flow power (W_s).

$W_s = P_m \bullet Q_m$, where P_m is the mean arterial pressure and Q_m the mean ascending aortic flow and (2) oscillatory power (W_o) $W_o = ½ \sum (Q_n)^2 Z_n \cos \theta_n$.

Where Q_n is the nth harmonic of flow and Z_n the nth harmonic of impedance, and θ_n the phase angle of impedance for the nth harmonic. The

total hydraulic power is the sum of W_s and W_o. In the left ventricle, the oscillatory contribution to total power output is less than 20% of the total power (Nichols et al. 1977).

The hydraulic power of blood flow (work/time) in the ascending aorta is dependent upon (1) the ability of the left ventricle to perform external work and (2) the hydraulic load of the arterial system. Consequently, the hydraulic power generated is dependent upon both ventricular performance and the impedance to flow. The steady state achieved defines the coupling of the ventricle to the arterial system.

As shown previously, ageing, the development of atherosclerosis, and hypertension decrease arterial distensibility and increase wave reflections which arrive back early in the ascending aorta during left ventricular ejection and boost systolic pressure. Consequently, both total and oscillatory power are increased, with a larger proportion of oscillatory power in the individuals with atherosclerosis and hypertension. (oscillatory/total power normal group 13 ± 1%, atherosclerosis and hypertension 19 ± 2.5%) (Nichols et al. 1977). In the oldest group with the highest characteristic impedance, the pulsatile component was 26% of total power.

Exercise increases both steady flow and oscillatory power. In dogs, there was a modest increase in the proportion of oscillatory to total power during exercise 19–22% (Unpublished data presented in (Milnor 1989)). In man especially with exercise-induced systolic hypertension, it is likely that there is a greater proportion of oscillatory power/total power during exercise.

Conclusions

1. The arterial system is more than a conduit to deliver oxygenated blood to the organs. It acts both as a buffer to limit the rise of pressure and to deliver a more constant flow to the tissues.
2. The hydraulic load encountered by the left ventricle is the consequence of arteriolar resistance (constant flow) and both the elastance of the arterial system and the impact of reflected waves (pulsatile flow).
3. The arterial system behaves as a distributed network of distensible vessels with wave reflections originating from impedance mismatches that occur mainly at the arterial/arteriolar interface.
4. Characterization of the arterial load is best expressed in the frequency domain as the aortic input impedance. It allows assessment of both steady flow impedance (arteriolar resistance) and pulsatile impedance as determined by the characteristic impedance and the impact of wave reflections.
5. Arterial pressure and flow waves change during their transit through the arterial system due to their interaction with reflected waves. Consequently, pressures measured distally in a limb (e.g. radial arterial pressure) may not accurately reflect central aortic pressure: the pressure determining left ventricular afterload, coronary and cerebrovascular perfusion. Central aortic pressures can be synthesized from recordings of radial artery pressure waveforms.

For further in-depth reading, see

McDonald's Blood Flow in Arteries. Theoretical, Experimental and Clinical Principles, 6th Edition, 2011. WW Nichols, MJ O'Rourke, and C Vlachopoulos. CRC Press, Boca Raton, USA.

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