Ascending Aortic Impedance Patterns in the Kangaroo: Their Explanation and Relation to Pressure Waveforms

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This study seeks to explain mechanisms responsible for the peculiar ascending aortic pressure waveform and impedance spectral pattern in kangaroos. Pulsatile pressure and blood flow velocity were measured and input impedance calculated in the ascending aorta, descending thoracic aorta, and brachiocephalic artery of 15 rock kangaroos. Pressure and velocity waveforms and impedance spectral patterns were interpreted with the aid of an asymmetric uniform T-tube model of the systemic arterial tree. The ascending aortic pressure waveform displayed a very large secondary wave that began in late systole or early diastole and continued throughout most of diastole. The peak of this secondary wave (which almost always occurred in diastole) was often greater than peak systolic pressure and results from apparently intense wave reflections from peripheral vascular beds. This contention is supported by the configuration of the impedance spectral pattern that is explained on the basis of a single (or dominant) functionally discrete reflecting site in the lower part of the body. These findings are explicable on the basis of body size and shape and the extreme eccentric location of the heart within the body. Wave reflections from the diminutive upper body are so small that they are totally dominated by intensive wave reflections from the large muscular lower body. These conclusions are supported by results obtained from the asymmetric T-tube model. (Circulation Research 1986;59:247-255)

There is general agreement on the contour of arterial pressure and flow waves in different animal species.1-4 The observed blood flow (or velocity) waveforms in the ascending aorta of various animals (including man) are similar, but the waveforms of pressure are markedly different. For example, in man three different types of pressure waveforms have been recorded.5,6 The pressure waveforms appeared to be age-related with peak systolic pressures occurring in late systole in older subjects and in early systole in younger subjects. In the Australian wombat, an animal with a short body and short stubby limbs, a large secondary diastolic wave was observed.2 Recordings in the diamond python, an animal with an extreme degree of body taper and no limbs, showed no secondary diastolic wave under any circumstances.6 In dogs, a secondary diastolic wave was observed, but it was lower in amplitude than that observed in the wombat.1 From these studies it became apparent that the body size and shape and the location of the heart within the body were important determinants of the arterial pressure waveform. The differences in pressure waveforms in the different animals were attributed to the intensity of waves reflected from peripheral reflecting sites, the distance over which the waves travelled, the velocity of the incident and reflected waves, and the relative contribution of waves reflected from the upper and lower parts of the body. These interpretations of experimental data were supported by studies of pressure and flow waveforms and of vascular impedance in models of different vascular beds and of the whole systemic circulation.6,10 From information obtained from impedance spectral patterns, pressure and flow waveforms could be explained on the basis of two functionally discrete reflecting sites; one in the upper part of the body and the other in the lower part.2,4,7,10,11,13,14

To further study the relation between body shape and pressure waveform, it seemed desirable to obtain data from an animal with marked differences in relative length and muscle bulk of upper and lower body. The Australian kangaroo appeared to be an ideal animal to study because of its relatively small head and short upper limbs (used primarily for feeding) and neck and relatively large, powerful lower limbs used for locomotion. The kangaroo also has a long, large circular tail that is used for stabilization in hopping and support when resting.

It was suspected that this animal would show a relatively small blood supply to the upper part of the body, abbreviated systolic flow into the brachiocephalic and left subclavian arteries, and similar flow waveform and impedance patterns in the ascending and descending thoracic aorta.

Very little information is available on vascular hemodynamics in kangaroos and other macropodinae. Initial cardiovascular physiology studies by Maxwell et al.15 Dawson and Needham,16 and Needham17 in kangaroos, wallaroos, and wallabies only measured arterial pressure and cardiac output. To our knowledge pulsatile pressure and flow and vascular impedance have not been reported by others in these animals.

The objectives of this study were to
1. record high-fidelity pulsatile pressure and blood flow velocity in different arteries (ascending aorta, descending thoracic aorta, brachiocephalic artery) of the Australian kangaroo, using a multisensor catheter;
2. calculate vascular impedance in these arteries from simultaneously recorded pressure and flow velocity waveforms;
3. interpret the peculiar ascending aortic pressure waveform and impedance spectral patterns with the aid of an asymmetric uniform T-tube model of the systemic arterial tree.
4. investigate the changes in pressure waveforms and impedance spectral patterns produced by vasodilation and vasoconstriction.

Materials and Methods

This study was conducted on 15 healthy adult Australian rock kangaroos or wallaroos (Macropus robustus) ranging in weight from 12 to 38 kg. There were 7 males and 8 females. The animals were caught by running them into a large net suspended between two trees. They were premedicated with ketamine (10–15 mg/kg, i.m.) and later (approximately 20 minutes) anesthetized with sodium pentobarbitone (20–25 mg/kg, tail vein). After the animal was anesthetized, the left external jugular vein was isolated and catheterized for drug administration and supplemental doses of sodium pentobarbitone. High-fidelity pulsatile arterial pressures and blood flow velocity were measured using a specially designed 8F Millar multisensor catheter (model VPC-684D). One pressure manometer was mounted at the catheter tip. The electro-magnetic velocity sensor and another pressure manometer were mounted 5 cm from the tip. The velocity sensor was connected to an SE Medic flowmeter (model 275) and the pressure manometers to Millar preamplifiers. The velocity sensor was calibrated in vitro in a steady-flow hydraulic model using isotonic saline or whole blood. The model consisted of a reservoir attached to a 100-cm length of Tygon tubing (i.d. 2 cm). The velocity sensor was positioned midstream in the tubing 5 cm from the reservoir. Flow rates ranging from 6 to 121 cm/second were used and a calibration factor determined.

The multisensor catheter was inserted into the left common carotid artery and advanced until the pressure manometer at the tip was in the ventricle and the other pressure manometer and velocity sensor were in the ascending aorta. This arrangement tends to stabilize the sensor in the central axis of the ascending aorta, eliminates the spurious signal that appears if the sensor comes to lie against the wall of the vessel, and minimizes artifacts in the recorded velocity waveform caused by motion of the catheter. The ventricle was found to be extremely sensitive and fibrillated on several occasions in the early experiments when the catheter was manipulated inside the ventricular cavity. In subsequent experiments this was minimized by intravenous administration of lidocaine (2 mg/kg) before introducing the catheter into the ventricle. Recordings of pressure and blood flow velocity were collected during a control period and after intravenous administration of nitroglycerin (30 μg) and norepinephrine (4 μg). Pulsee velocity from the ascending aorta to the femoral artery was determined from the foot-to-foot delay time between the two pressure tracings. Femoral arterial pressure was recorded with a short fluid-filled stiff catheter connected to a P23Db statham transducer. The frequency response of the catheter–manometer system was flat (± 5%) from 0 to 40 Hz. The dynamic frequency characteristics of this pressure measuring system is sufficient for accurate measurements of pulsewave velocity. After recordings were obtained in the ascending aorta, the multisensor catheter was withdrawn and advanced into the descending thoracic aorta. The animal was then placed on a positive pressure respirator and a thoracotomy performed through the fourth intercostal space. This was done so that the position of the velocity (and pressure) sensor, which had been withdrawn to the brachiocephalic artery, could be verified. In three animals, the flow velocity waveform was verified by using a cuff probe to measure pulsatile flow.

Vascular impedance was determined from analysis of simultaneously recorded pressure and flow velocity waves with techniques described in detail previously. Impedance spectra (modulus and phase) were determined from corresponding harmonic components of pressure and flow waveforms by Fourier analysis on a PDP 11/03 computer. The valid representation of a continuous function such as a pressure or flow wave by Fourier series requires that these waves be periodic and that the system be in a steady state. These conditions are satisfied in the kangaroo. The use of Fourier analysis to define impedance also assumes linearity of the system in which the pressure and flow waves are measured. Several techniques have been employed to test this assumption in the arterial system, and all of them have shown approximately linear relationships. Pairs of pressure and flow pulses were analyzed individually, and results were averaged over 7–10 pulses. Background instrument noise was estimated by analyzing signals obtained with zero pressure and flow. The noise level was determined to be less than 0.5 mm Hg for pressure and less than 0.3 cm/sec/second for flow. Impedance values were discarded if determined from pressure or flow harmonic components below these levels. This procedure eliminated all data above about 15 Hz. Appropriate corrections were made for flowmeter frequency response and phase delay as described previously. Impedance modulus was expressed in terms of linear flow velocity (dyne·sec/cm² as recommended by McDonald and used by O'Rourke and Avolio in comparative studies).
Results

Control data from all experiments are summarized in Table 1. Table 2 summarizes pooled results during administration of nitroglycerine and norepinephrine. In the 15 animals studied, crown-to-rump length averaged 72 cm (range, 57-86 cm) and body weight averaged 23 kg (range, 12-38 kg). In the control state heart rate ranged from 49 to 137 beats/minute (average 92 ± 6.2 SE b.p.m.) and mean arterial blood pressure ranged from 56 to 154 mm Hg (average 111 ± 7.5 mm Hg) - ranges similar to those recorded by others in adult kangaroos and similar to those found in other mammals of equivalent size. Ascending aortic circumference (and radius) was measured with umbilical tape in 5 animals (Nos. 1, 3, 4, 14, 15) so that mean volumetric blood flow could be obtained from the velocity signal. In these 5 animals cardiac output averaged 2.64 l/minute and stroke volume averaged 35 ml. These values are in agreement with those reported previously for anesthetized kangaroos of similar size. Mean blood flow velocity averaged 13 ± 0.8 cm/second (range, 6-20 cm/sec) for the entire group of animals.

The ascending aortic pressure waveform, which was markedly different to that seen in other animals, demonstrated a very large exaggerated secondary wave that peaked late in systole or early diastole. Three different types of observed waveforms (with impedance spectral patterns) are shown in Figure 1. On occasion, when the secondary wave peaked in diastole, maximum diastolic pressure was greater than maximum systolic pressure (5 animals, Figure 1A). In 4 animals maximum diastolic pressure was essentially the same as maximum systolic pressure (Figure 1B), and in 6 animals maximum diastolic pressure was less than maximum systolic pressure (Figure 1C). The three different pressure patterns shown in Figure 1 correspond to Type A, B, and C pressure waveforms reported in humans by Murgo et al12 and Nichols et al.9 In adult human subjects, however, the secondary wave always occurred in systole, whereas in kangaroos and other experimental animals the secondary wave almost always occurred in diastole.1,4,21 In the entire group of kangaroos maximum systolic aortic pressure averaged 126 ± 7.8 mm Hg (range, 76-175 mm Hg); maximum diastolic pressure averaged 125 ± 6.8 mm Hg (range, 68-175 mm Hg); and minimum diastolic pressure averaged 97 ± 6.8 mm Hg (range, 52-137 mm Hg). A prominent diastolic wave (but of lesser magnitude than seen here) in the ascending aortic pressure waveform has also been observed in the Australian wombat under control conditions and in dog models when both femoral arteries, both brachial arteries, and both carotid arteries were momentarily occluded and during occlusion of the descending aorta at the trifurcation.23 Blood flow velocity waveforms recorded in kangaroos were similar to those reported previously for other experimental animals and man,14,6,11,13,22 but the ascending aortic impedance spectral patterns were markedly different. The impedance patterns showed large fluctuations in both modulus and phase as a function of frequency for all three types of aortic pressure waveforms.

TABLE 1. Pulsatile Hemodynamics in Kangaroos (Control)

<table>
<thead>
<tr>
<th>Kangaroo</th>
<th>Sex</th>
<th>Wt. (kg)</th>
<th>Crown rump length (cm)</th>
<th>HR (b/min)</th>
<th>Aortic pressure (mm Hg)</th>
<th>Mean velocity (cm/sec)</th>
<th>Zc (dsc⁻¹)</th>
<th>R (dsc⁻²)</th>
<th>PWV (cm/sec)</th>
<th>fmax (Hz)</th>
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</table>

HR = heart rate, S = systolic pressure, Dmin = minimum diastolic pressure, Dmax = maximum diastolic pressure, M = mean aortic pressure, Zc = characteristic impedance (dyne·sec/cm²), R = peripheral resistance, PWV = pulsewave velocity, fmax = frequency of minimum impedance modulus.
forms. An indicator of the amplitude of fluctuations of the impedance moduli about the characteristic impedance ($Z_0$) (and therefore of the degree of pulse wave reflection) is given by the equation

\[
\frac{Z_{\text{max}} - Z_{\text{min}}}{Z_0}
\]

where $Z_{\text{max}}$ and $Z_{\text{min}}$ are values of the maximum and minimum input impedance moduli. This ratio was different for the three types of pressure waveforms shown in the examples of Figure 1: 1.71 for Type A, 1.21 for Type B, and 1.08 for Type C. These values are higher than those normally found in healthy humans and dogs under control resting conditions. The differences observed in individual aortic pressure waveforms and fluctuations in impedance moduli in the kangaroos may be related to the age of the animal. The second minimum of modulus occurred at three times the frequency of the first minimum, and the intervening maximum occurred at approximately twice this frequency, while impedance phase crossed zero at close to these frequencies. This pattern was preserved even when the impedance spectra from all animals were grouped together (Figure 2). Because of large differences in heart rate, impedance data (modulus and phase) were grouped for averaging by frequency rather than harmonic number. This type of impedance pattern is similar to that measured in a rubber tube with a closed end or a uniform transmission line and suggests the presence of a single (or dominant) functionally discrete reflecting site. By applying this simple model to the arterial tree, it is possible to derive an "effective" length, i.e., the distance ($L_e$) to the...
reflecting site from the point of pressure and flow measurement by the quarter wavelength relationship:

\[ L_e = \frac{PWV}{4f_{\text{min}}} \]

where \( PWV \) is the pulsewave velocity obtained from ascending aorta and femoral arterial pressure recordings and \( f_{\text{min}} \) is the frequency of the first aortic input impedance modulus minimum. Using this relation, the calculated "effective" reflecting site distances for the three kangaroos (Nos. 1, 7, 3) of Figure 1 were 39, 46, and 37 cm respectively. Since reflection site distance influences the shape of the pressure waveform, an attempt was also made to derive it from the pressure wave alone using the relationship:

\[ L_p = \frac{PWV \Delta t}{2} \]

where \( L_p \) is the effective length and \( \Delta t \) is the time from the initial upstroke of the pressure wave to the inflection point (or the up-stroke of the reflected wave) and represents the time of travel of the wave from the heart to the reflection site and back. If the uniform tube model can be used as a first approximation of the arterial system, then the distances \( L_e \) and \( L_p \) should be equal. The distances \( L_e \) derived for kangaroos 1, 7, and 3 were 38, 50, and 39 cm respectively, which are very close to the values \( L_p \) obtained from the impedance spectra.

Vasodilation (nitroglycerine) and vasoconstriction (norepinephrine) caused marked changes in the ascending aortic pressure waveform and impedance spectral pattern. An example of recordings obtained in kangaroo No. 10 under control conditions and during drug administration is shown in Figure 3. With vasodilation, the secondary wave was decreased in amplitude and displaced later in diastole presumably as a result of the observed decrease in pulsewave velocity. Vasoconstriction, on the other hand, increased the amplitude of the secondary wave, but its position on the pressure waveform did not change since pulsewave velocity remained the same. Thus, in this example, norepinephrine caused a change in the pressure waveform from a Type C configuration to a Type A. A similar change in the pressure waveform occurs in humans immediately after release from the Valsalva maneuver.39 The major alterations in the impedance spectral patterns were changes in oscillations about the characteristic impedance as a result of wave reflections. During control, in this example, \( \frac{Z_{\text{max}} - Z_{\text{min}}}{Z_0} \) was 1.16; with vasodilation \( \frac{Z_{\text{max}} - Z_{\text{min}}}{Z_0} \) was 0.8 and with vasoconstriction \( \frac{Z_{\text{max}} - Z_{\text{min}}}{Z_0} \) was 1.55. The greatest change between control and vasoactive drugs occurred at low harmonics, with impedance moduli tending to be lower with vasodilation and higher with vasoconstriction. Similar changes were also observed in apparent phase velocity spectral patterns.21 In the animals \( n = 12 \) administered drugs, pulsewave velocity averaged 487 ± 29 cm/second in the control state; 437 ± 24 cm/second with vasodilation \( (p < .001) \) and 540 ± 40 cm/second with vasoconstriction \( (p = \text{NS}) \). The observed changes in PWV are presumably due to changes in smooth muscle tone and arterial pressure.

**Discussion**

The arterial system of a variety of different animals (including man) has been likened to an asymmetrical T-tube whose short arm represents arteries supplying the head, neck, and upper limbs, and whose long arm represents arteries supplying the trunk and lower limbs. It has been suggested that the arterial system is a Type C configuration to a Type A. A similar change in the pressure waveform occurs in humans immediately after release from the Valsalva maneuver.39 The major alterations in the impedance spectral patterns were changes in oscillations about the characteristic impedance as a result of wave reflections. During control, in this example, \( \frac{Z_{\text{max}} - Z_{\text{min}}}{Z_0} \) was 1.16; with vasodilation \( \frac{Z_{\text{max}} - Z_{\text{min}}}{Z_0} \) was 0.8 and with vasoconstriction \( \frac{Z_{\text{max}} - Z_{\text{min}}}{Z_0} \) was 1.55. The greatest change between control and vasoactive drugs occurred at low harmonics, with impedance moduli tending to be lower with vasodilation and higher with vasoconstriction. Similar changes were also observed in apparent phase velocity spectral patterns.21 In the animals \( n = 12 \) administered drugs, pulsewave velocity averaged 487 ± 29 cm/second in the control state; 437 ± 24 cm/second with vasodilation \( (p < .001) \) and 540 ± 40 cm/second with vasoconstriction \( (p = \text{NS}) \). The observed changes in PWV are presumably due to changes in smooth muscle tone and arterial pressure.
tense the reflections and the greater change in impedance spectra. When the aorta was occluded at the diaphragm, the single (uniform) tube appeared to be a much better model than the 3-element Windkessel model.

Therefore, a logical explanation for the observed findings in the kangaroo come from consideration of body shape and the eccentric location of the heart within the body and the effects of this on dispersion of reflecting sites.

Impedance Spectral Pattern

Fluctuations in impedance modulus and phase are due to wave reflections at the arterial terminations. The frequencies at which they occur depend on transit time between the recording and reflecting sites — on the length and configuration of the arterial system and pulsewave velocity. Thus, fluctuations (i.e., maxima and minima) in impedance modulus and phase should occur at frequencies that depend on the size and shape of the animal under study. In the studies of O'Rourke and Taylor the impedance spectra measured in the ascending aorta of dogs displayed two distinct minima. The second minimum occurred at less than three times the frequency of the first and the intervening maximum occurred at less than twice the frequency of the first minimum. The finding in the impedance pattern suggested that the systemic circulation presented to the left ventricle two discrete reflecting sites, one in the upper and the other in the lower part of the body. In dogs and young adult humans reflections from these two sites interact so as to cause fluctuations in aortic input impedance spectra (modulus and phase) that are relatively low in amplitude. In man these fluctuations are attenuated during the Valsalva maneuver or peripheral vasodilation when reflections from the lower body are delayed (as a result of lower aortic pulse wave velocity) so that functional dispersion of reflecting sites is accentuated. In older adult humans and patients with cardiovascular disease, fluctuations are somewhat more marked compared to young adults with no cardiovascular disease. The increased amplitude of impedance fluctuations is apparently due to increased pulsewave velocity as a result of decreased arterial distensibility. This reduces dispersion of upper and lower body reflecting sites so that reflected waves from the lower body arrive almost simultaneously with
Fluctuations in the ascending aortic input impedance spectra (modulus and phase) of kangaroos were found to be more marked than in any other animal studied. The observed impedance spectral patterns in the ascending aorta of kangaroos are similar to those measured in a uniform transmission line,\textsuperscript{27} a rubber tube model with a closed end\textsuperscript{28} or a single uniform tube computer model.\textsuperscript{14,28} That is, the second impedance minimum occurs at three times the frequency of the first and the intervening maximum occurs at twice the frequency. These findings suggest that the arterial system of kangaroos appears to behave like a single tube with a single (or dominant) reflecting site (representing the resultant of all individual reflecting sites in the lower part of the body). This conclusion is supported by measurements of impedance spectra in the descending thoracic aorta (just distal to the left subclavian artery) and the brachiocephalic artery (Figure 4) and in the asymmetric T-tube model (Figure 5). Large fluctuations (maxima and minima) of approximately the same amplitude and frequency appear in both the ascending and descending aortic impedance spectra while fluctuations in the brachiocephalic spectra are very attenuated, and the moduli spectrum shifted upward. If brachiocephalic impedance had been lower than that of the descending aorta, then ascending aorta impedance would have been smoother (parallel tubes). Using the above formula, the distance to peripheral reflecting sites from the points of pressure and flow measurements in kangaroo No. 14 can be calculated [ascending aorta, $484/(4 \times 2.3) = 53$ cm; descending thoracic aorta, $484/(4 \times 2.5) = 48$ cm; brachiocephalic artery, $550/(4 \times 11.5) = 12$ cm]. The asymmetric T-tube model was run using these measurements — short upper limb 12 cm; long lower limb 48 cm; wave velocity in short limb 550 cm/second and long limb, 484 cm/second; attenuation in short limb 0.01 nepers/cm, and long limb 0.007 nepers/cm; reflection coefficient 0.8 at each end. Impedance spectral patterns obtained using this T-tube model showed remarkable agreement with those measured in the kangaroo (Figure 5). These findings support the contention that the arterial system of the kangaroo can be likened to a single tube with a single reflecting site in the lower part of the body that dominates all other reflection phenomena.

**Aortic Pressure Waveforms**

The peculiar pressure waveform recorded in the ascending aorta of kangaroos\textsuperscript{18,22} can be explained on the basis of apparently intense and unusually discrete peripheral wave reflection from the lower part of the body. This explanation is supported by large fluctuations in the impedance spectral patterns which are

![Figure 4](https://example.com/f4.png)

*Figure 4. (Top) Pressure and velocity waveforms recorded in the ascending aorta (AA), descending thoracic aorta (DTA), and the brachiocephalic artery (BCA) of the kangaroo. (Bottom) Impedance spectra calculated from pressure and velocity signals. The DTA impedance spectra is very similar to the AA spectra while the BCA spectra is markedly different.*
known to result from wave reflections. The difference between ascending aortic pressure waves in Figure 1 are explicable on the basis of timing of wave reflections. In Figure 1B the reflected wave returned during systole causing the incisura to fall on the peak of the secondary wave. In Figure 1A and 1C the reflected waves returned later. In Figure 1A the intensity and timing of the reflected or secondary wave was such that peak pressure occurred in diastole rather than systole. In Figure 1C the reflected wave returned later, so that the diastolic pressure wave, while of high amplitude, was not greater than systolic pressure. The intensity and timing of these reflected waves can be altered by vasoactive drugs (Figure 3). Vasoconstriction (noradrenaline) caused an increase in intensity (or amplitude) of the reflected wave but not its timing since pulse wave velocity did not change. Vasodilation (nitroglycerine), on the other hand, caused a decrease in the amplitude and a delay later in diastole of the reflected wave because of decreased pulse wave velocity.

The impedance spectral patterns derived from the three different pressure waveforms all showed large amplitude fluctuations. Pressure waves and impedance spectral patterns observed in the kangaroo, an animal with small muscle mass (and blood supply) cephalically and a large muscle mass (and blood supply) caudally, are unlike those seen in any other animal studied and are readily explained on the basis of wave reflection principally from one large reflecting site in the lower body.

In summary: The ascending aortic pressure waveform recorded in kangaroos shows very large secondary waves that are attributable to very intense wave reflections from the lower part of the body. This explanation is supported by the corresponding impedance spectral patterns that display very large fluctuations similar to those seen in a uniform transmission line or a rubber tube with a closed end.

References
2. O'Rourke MF: Pressure and flow waves in systemic arteries and the anatomical design of the arterial system. J Appl Physiol 1981;23:139-149

**Key Words** • wave reflection • arterial compliance • kangaroo
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