Input Impedance of the Systemic Circulation

By Michael F. O’Rourke, M.B., B.S., and Michael G. Taylor, M.D., Ph.D.

ABSTRACT

The hydraulic load presented to the left ventricle by the systemic circulation was characterized by expressing pressure-flow relationships in the ascending aorta as input impedance. This was determined by spectral or Fourier analysis of simultaneously recorded pressure and flow waves in 1 unanesthetized and 27 anesthetized dogs. Impedance modulus fell steeply from its value at zero frequency (the peripheral resistance) and its value was lowest (less than 1/20th of the peripheral resistance) over that band of frequencies (usually between 1.5 and 10 cycle/sec) which contained most of the energy of the left ventricular ejection (flow) wave. The patterns of modulus and phase of ascending aortic impedance were found to result from the presence of two functionally discrete reflecting sites in the systemic circulation, one in the upper part of the body and the other in the lower. The presence of these two sites appears to be an important factor in maintaining a low impedance modulus between 1.5 and 10 cycle/sec, and so in providing a favorable impedance to pulsatile flow from the heart. Both modulus and phase of impedance in the ascending aorta showed changes similar to those seen in other arteries when blood pressure was altered and when vasodilation occurred in the vascular bed.

ADDITIONAL KEY WORDS

Fourier and spectral analysis wave reflection pulse wave velocity ascending aorta vasodilation blood pressure design of vascular system external left ventricular work pressure and flow waves anesthetized dogs

The hydraulic load presented to the left ventricle may conveniently be expressed as the input impedance of the systemic circulation. This is determined by relating in modulus and phase the corresponding frequency components of the pressure and flow waves recorded in the ascending aorta (Fig. 1). Consideration of ascending aortic impedance is of great value in studying not only the properties of the systemic arteries but also the effect on the left ventricle of a variety of physiological and pathological conditions affecting the arteries and arterioles.

Studies of vascular impedance in various arteries have been reported by a number of authors (1-5), but the only comprehensive report on ascending aortic impedance in the dog is that published by Patel, deFreitas and Fry (6). The results presented here in general confirm their findings, but appear to be more consistent, and in addition show significant differences during alterations in mean blood pressure and peripheral resistance. The greater detail seen in these results is attributed to the fact that they were plotted separately for different animals, so that allowance was made for differences in pulse wave velocity and body length.
The principal purposes of this report are (A) to show how the input impedance characteristics of the systemic circulation are favorably adapted to the intermittent ejection of blood from the left ventricle; (B) to examine the factors which determine these favorable characteristics; and (C) to show how the load presented to the ventricle changes under different conditions. The most important finding relates to the gross anatomical design of the systemic arterial system. The favorable input impedance characteristics of the systemic circulation are due in part to the anatomical design of the arterial system which results in the ventricle being presented with two functionally discrete reflecting sites, one in the upper and the other in the lower part of the body.

**Methods**

**ANIMALS AND ANESTHETIC**

Experimental data were obtained from 28 dogs ranging in weight from 14.3 to 35.8 kg. Most of these were greyhounds or stray street dogs of the Alsatian or Boxer type. Allowing for variations in size, there was no obvious difference in the results obtained from different breeds. In all experiments but one the dogs were anesthetized. Twenty-three dogs were given morphine sulphate (1.0 to 1.5 mg/kg im), and anesthesia was then induced with pentobarbital sodium (20 to 40 mg/kg iv), and maintained with small doses (2 to 4 mg/kg per hour). Four dogs were anesthetized with 50 mg chloralose and 500 mg urethane per kg iv. In most experiments, the dogs were given small initial (1.5 to 2.0 mg/kg iv) and maintenance (less than 0.25 mg/kg per hour) doses of gallamine. The animals were anesthetized with 50 mg chloralose and 500 mg urethane per kg iv. In most experiments, the dogs were given small initial (1.5 to 2.0 mg/kg iv) and maintenance (less than 0.25 mg/kg per hour) doses of gallamine. The animals were ventilated with air (8 to 12 liters/min). One conscious dog had a flowmeter transducer previously implanted about the ascending aorta and a catheter then inserted in a femoral artery under local anesthesia. Results were not significantly different from those in the anesthetized dogs.

**INSTRUMENTATION AND EXPERIMENTAL PROCEDURE**

Flow was measured with a sine-wave electromagnetic flowmeter which was a modified version of the instrument described by Kolin and Kado (7) and similar to the currently available Medicon instrument. The flow transducer was placed about the ascending aorta distal to the origin of the coronary arteries. Application of the transducer necessarily entailed some constriction of the artery, but by careful selection of size this effect was minimized. Zero flow baseline was taken to be the integrated level during diastole. Calibration varied little from day to day and was determined by passing saline through a piece of excised artery enclosed within the transducer. Dynamic calibration of the flowmeter was determined by resolving the output signal when sinusoidal flow of known characteristics was pumped through the transducer (8). Allowance was made for flowmeter frequency response in the calculations.

Pressure was measured through a fine nylon catheter by a Sanborn P287B manometer, from the ascending aorta immediately downstream from the probe. Two techniques were used for measuring pressure in the ascending aorta. In the first, the sawed-off end of a 23 gauge hypodermic needle was attached to the catheter and the needle inserted directly through the aortic wall. In the second, the catheter was inserted in a peripheral artery (usually the superior thyroid branch of the carotid artery) and threaded down until its tip was in the ascending aorta. The catheter tip was located by palpation or, when the chest was closed, with the aid of an x-ray image intensifier or by passing the catheter into the ventricle and withdrawing it a little more than the width of the flow transducer past the aortic valves. The catheter or needle tip was manipulated to face the direction of flow to measure impact and not lateral pressure. The frequency responses of the catheter-manometer systems were determined by the pressure-step or "pop" method, and allowance was made for these in the subsequent calculations. The damped natural frequencies were usually between 80 and 100 cycle/sec.

The effects of an increase in mean arterial pressure on ascending aortic impedance were studied in 5 animals by infusing norepinephrine intravenously (norepinephrine, 2 μg in 1 ml at rates up to 5 μg/min). In these experiments as in those of Patel et al. (6), norepinephrine infusions led to an increase in both cardiac output and mean arterial pressure so that peripheral resistance was not appreciably altered. The effects of a decrease in peripheral resistance were studied in 5 animals by infusing intravenously acetylcholine (50 μg/ml at rates up to 100 μg/min), and isoproterenol (1 μg/ml at rates up to 2 μg/min). Vasodilation was always accompanied by a fall in mean arterial pressure.

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1The image intensifier was kindly donated by Watson Victor Ltd., aided by a grant from the National Heart Foundation.

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In most of the 28 experiments, vascular impedance was determined in a number of other arteries, including the descending thoracic aorta (8 dogs), brachiocephalic artery (7 dogs), and left subclavian artery (3 dogs). As for the ascending aorta and femoral artery (9), impedance was calculated from records of pressure and flow; flow was measured with an electromagnetic flowmeter and pressure with a Sanborn manometer through a needle or catheter whose tip was located just beyond the flow transducer. In six experiments, the impedance in the ascending aorta was measured during occlusion of the brachiocephalic artery and descending thoracic aorta, and in two during occlusion of both the brachiocephalic and left subclavian arteries. The two arteries were occluded with snares close to their origin from the aorta, and the descending thoracic aorta was occluded by hand in the mid-thoracic region. When the brachiocephalic artery was occluded, records were taken before the reflex rise in pressure occurred and when the aorta was compressed, after the initial sharp rise in pressure had subsided. Attempts were made to take all records at the same mean pressure levels, since alterations in mean pressure caused significant changes in impedance patterns. Comparisons of vascular impedance in different arteries under control conditions and in the same artery under different conditions were facilitated in some experiments by maintaining blood pressure at the same level by an intravenous infusion of norepinephrine (2 μg in 1 ml at rates up to 2 μg/min). Intra-arterial norepinephrine in high doses was found to have little effect on impedance patterns in other vessels.

RECORDING AND ANALYSIS

Impedance was determined either from Fourier analysis of single pairs of pressure and flow waves with the heart beating regularly, or from spectral analysis of long trains of pressure and flow waves with the heart beating irregularly. In all, many thousands of pairs of waves were analyzed in the 28 experiments.

For Fourier analysis of pressure and flow waves, the methods of recording, data processing and analysis were the same as those reported earlier (9). Pressure and flow were recorded together with ECG and digitizing pulses (120/sec or 160/sec) at 30 inches/sec on magnetic tape. Where data were to be analyzed, a long train of digitizing pulses (2000 to 2400) was recorded on the tape and one channel of data at a time transferred at slow speed to paper tape. With digitizing signals at 100/sec, data were taken from an interval of 20 to 24 sec of recording, and impedance determined over a range of frequencies from 0.25 to 25.0 cycle/sec.

Results

The principal features of the ascending aortic impedance curves under control conditions are shown in Figure 1. From its value at zero frequency, impedance modulus fell steeply to a minimum which occurred at a lower frequency than in any other artery; from this first minimum the modulus rose slightly, then fell to a second minimum after which it usually rose to settle at a constant value above 12 to 15 cycle/sec. Impedance modulus at frequencies greater than 1.5 cycle/sec was about one twentieth of the impedance modulus at zero frequency, i.e. the "peripheral resistance" to steady flow. Phase angle was negative at low frequencies and showed two fluctuations which corresponded to the two minima of modulus. Phase angles either did not become positive at all, or became positive only over a narrow band of frequencies; thereafter, as frequency increased, phase angle tended to become progressively more negative. These features were seen in 27 of 28 animals; in the remaining dog, there was only one broad minimum of impedance modulus,
Vascular impedance under control conditions in the ascending aorta of 2 dogs. Left: impedance in a small (14.3 kg) dog determined from Fourier analysis of a series of 25 waves. Each point is the mean of 25 values and the bars represent ±2 standard errors of the mean. Peripheral resistance (the modulus at zero frequency) is $8.53 \times 10^3$ dyne sec cm$^{-5}$. Right: Impedance in a moderately large (23.0 kg) dog determined from spectral analysis of a long series of waves with the heart beating irregularly. Peripheral resistance is $7.71 \times 10^3$ dyne sec cm$^{-5}$. Between 12 and 25 cycle/sec (not shown in graph) impedance modulus settled about a constant value, $0.34 \times 10^4$ dyne sec cm$^{-5}$, and this was taken to be the characteristic impedance. Coherence, plotted above, is a type of correlation coefficient, indicating the stability of the relationship between pressure and flow at each frequency (10).

and this probably represented two fused minima.

Fluctuations in modulus and phase of impedance are due to wave reflection at the arterial terminations (2, 9). The frequencies at which they occur depend on transit time between the recording and reflecting sites—on the length of the arterial system and the velocity of the pulse wave. Thus, fluctuations in modulus and phase of impedance should occur at frequencies which depend on the size and shape of the animal. If wave velocity is the same, transit time will be shorter in small than in large animals so that the fluctuations will occur at higher frequencies. This is evident in Figure 1 which shows impedance in a small 14.3-kg dog and a larger 23-kg greyhound. In the former case, minima of impedance modulus occurred at 4 and 8 cycle/sec, and in the latter at 2.25 and 5.5 cycle/sec. The first impedance minimum in the small dog corresponded to the first maximum in the larger animal, and the first maximum in the smaller dog to the second minimum in the larger. Although statistical analysis of a large number of experimental results is desirable, it is obvious that features of the impedance curves can be lost if data are pooled.
from a large number of dogs of different shapes and sizes with different mean arterial blood pressures. For this reason, individual examples only are given here as illustrations; these, however, are typical of the findings in all experiments under all conditions.

The two separate minima in the graph of ascending aortic impedance appeared as a

FIGURE 2

A flow wave (upper right) recorded in the ascending aorta of a 17.9 kg dog (dog 7), and plotted from the digital voltmeter output. Graph shows its first 25 harmonics (solid black bars) after correcting each for instrumental errors. The approximate noise level is indicated by the small diagonally striped bars; these were obtained by submitting to Fourier analysis the diastolic part of the wave. Heart rate is 1.25/sec.

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broad trough in the frequency range of 1 to 12 cycle/sec. The trough corresponded to the range of frequencies which contained the greatest energy of the ventricular ejection wave. This is illustrated in Figure 2 which shows the moduli of the first 25 harmonics of the left ventricular ejection wave in a 17.9-kg dog which had chronic surgically induced heart block and whose heart was paced artificially. The greater part of the energy of the wave was in the first 5 harmonics at frequencies between 1 and 7 cycle/sec, and this was the frequency range over which impedance modulus was least. Figure 3 shows the changes which occurred in the first 10 harmonics of the ejection wave in this animal when its heart rate was increased from 60 to 186/min. As the heart rate increased, the energy in the

*Moduli of the first 10 harmonics of the ascending aortic flow waves at heart rates between 60 and 186/min. Each point is the average of 5 waves. Standard error is not plotted since it was extremely small, there being little difference in the shape of the waves at the same frequency. These data were obtained from dog 7 (17.9 kg) which had a surgically induced heart block, and an artificially paced heart.*
wave shifted from the higher to the lower harmonics so that over this wide range of heart rates the greater part of the energy of the wave remained in the band of frequencies 1 to 7 cycle/sec.

The finding of two separate minima of impedance modulus and phase at low frequencies was characteristic of the ascending aorta and was not seen in any other artery. The first maximum occurred at less than twice the frequency of the first minimum, and the second minimum at less than thrice the frequency of the first; this suggested that the systemic circulation presented to the left ventricle two discrete reflecting sites. Consideration of the anatomical arrangement of the systemic arteries led to the suggestion that one represented arterial terminations in the lower part of the body, and the other, arterial terminations in the upper part of the body. When impedance was determined in the descending thoracic aorta just below the left subclavian artery, there was only a single minimum of modulus at 2 to 4 cycle/sec, while in the brachiocephalic and left subclavian arteries, the minimum occurred between 5 and 10 cycle/sec. Again, as in the ascending aorta and the femoral artery (9), the minima occurred at lower frequencies in large dogs and higher frequencies in small dogs. Figure 4 shows the results of impedance determination in the ascending aorta, in the proximal part of the brachiocephalic artery and in the upper descending thoracic aorta of the same animal at the same mean arterial blood pressure. Both the modulus and the phase of impedance in the ascending aorta appeared to be composites of the modulus and phase of impedance in the brachiocephalic artery.
artery and the descending thoracic aorta. In this dog, impedance was not measured in the left subclavian artery (the second branch of the aortic arch), but in other animals the patterns were almost identical to those in the brachiocephalic artery. Figure 5A shows impedance in the ascending aorta of the same animal during occlusion of the descending thoracic aorta and Figure 5B, during occlusion of the brachiocephalic artery. When the aorta was occluded, the first minimum of modulus and the first fluctuation of phase disappeared so that the patterns of impedance came to resemble those seen in the brachiocephalic artery. Occlusion of the brachiocephalic artery caused accentuation of the first maximum of impedance modulus so that impedance patterns came to resemble those seen in the descending thoracic aorta. This was more obvious when both brachiocephalic and left subclavian arteries were occluded (Fig. 6).

The principal effects on ascending aortic impedance of a decrease in mean arterial blood pressure were a shift of the curves to the left when blood pressure fell, and to the right when blood pressure rose (Table 1). Two other features were sometimes evident. Firstly, when blood pressure rose, the separation of the two minima frequently became less distinct and secondly, the characteristic impedance often decreased as mean pressure
rose. Characteristic impedance is the modulus of impedance in the absence of wave reflection (2), and was taken to be the average value of impedance modulus between 15 and 25 cycle/sec.

Figure 7 shows impedance in the ascending aorta under control conditions (A) and during the infusion of norepinephrine (B) acetylcholine (C) and isoproterenol (D). Norepinephrine did not significantly alter peripheral resistance since blood pressure and cardiac output tended to rise proportionately. Vasodilation caused by infusion of acetylcholine or isoproterenol led to a fall in peripheral resistance, a less steep initial slope of impedance modulus, and smaller fluctuations
in modulus and phase angle than were seen under control conditions.

**Discussion**

**PHYSIOLOGICAL SIGNIFICANCE OF IMPEDANCE CURVES IN THE ASCENDING AORTA**

Input impedance of the systemic circulation expresses the opposition to blood flow from the left ventricle into the ascending aorta. For steady flow, opposition is the viscous resistance to blood flow through the systemic circulation. For pulsatile flow, opposition depends on the interaction of the inertial and viscous properties of blood, the elastic and viscous properties of the arterial walls, and wave reflection. Impedance modulus falls steeply from zero frequency, and above 1.5 cycle/sec is usually one twentieth (or less) of the peripheral resistance. The opposition to pulsatile flow is even less than this, since impedance modulus is pressure modulus divided by flow modulus, and pulsatile pressure is not in phase with pulsatile flow. Thus, a better index of the opposition to blood flow is the in-phase impedance, \(|Z| \cos \phi\), which is the relationship between oscillatory flow and the component of oscillatory pressure.

\[ \text{FIGURE 7 (continued)} \]
Impedance was determined before and during intravenous infusion of norepinephrine. In dogs 34 and 35, impedance was derived from spectral analysis of records obtained with the heart beating irregularly owing to irregular cardiac pacing. In dogs 20, 41, and 42, impedance was derived from Fourier analysis of records obtained when the heart rate was slowed by electrical stimulation of the right vagus nerve in the neck.

**TABLE 1**

<table>
<thead>
<tr>
<th>Dog</th>
<th>Mean arterial pressure (mm Hg)</th>
<th>Peripheral resistance $\times 10^4$ (dyne cm$^{-2}$ sec cm$^{-$$2}$)</th>
<th>Value of impedance modulus at first minimum $\times 10^4$ (dyne cm$^{-2}$ sec cm$^{-$$2}$)</th>
<th>Characteristic impedance modulus at second minimum $\times 10^4$ (dyne cm$^{-2}$ sec cm$^{-$$2}$)</th>
<th>Frequency (cycle/sec) [first minimum]</th>
<th>Frequency (cycle/sec) [first maximum]</th>
<th>Frequency (cycle/sec) [second minimum]</th>
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<td>0.195</td>
<td></td>
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<td>0.073</td>
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<td>0.17</td>
<td>4.2</td>
<td>5.2</td>
<td>7.0 - 9.5</td>
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</table>

FACTORS WHICH DETERMINE THE PATTERNS OF ASCENDING AORTIC IMPEDANCE CURVES

The patterns of impedance in the ascending aorta cannot be explained on the basis of wave reflection at a single peripheral site, although (with reservations about high frequencies) such an explanation has been satisfactory for explaining impedance patterns in other arteries (2, 9). When an artery "sees" a single reflecting site, the first maximum and second minimum occur respectively at twice and thrice the frequency of the first minimum (2, 9, 11). When an artery "sees" other arteries (2, 9) or the "vascular bed" was made up of two separate networks, the patterns of impedance were obtained in calculations on models of the arterial system. However, the patterns obtained in this way were similar to those obtained in the ascending aorta in all the frequency range. In the ascending aorta, the second minimum occurs at approximately twice the frequency of the first minimum, where, on the basis of high-frequency and low-frequency (2, 9) and low-frequency (11) patterns, such an explanation has been satisfactory for explaining the patterns in the descending thoracic aorta (Fig. 4). In the ascending aorta, the second minimum occurs at approximately twice the frequency of the first minimum, where, on the basis of high-frequency and low-frequency (2, 9) and low-frequency (11) patterns, such an explanation has been satisfactory for explaining the patterns in the descending thoracic aorta (Fig. 4).
The findings for the ascending aorta indicate that it sees two discrete reflecting sites. Consideration of the anatomical arrangement of the systemic arteries led us to suggest that the first minimum of impedance modulus in the ascending aorta results from wave reflection at arterial terminations in the lower part of the body, and the second minimum from reflection at arterial terminations in the upper part of the body. This hypothesis appears to be verified by the studies of impedance in the major arteries supplying the upper and lower parts of the body, and of impedance in the ascending aorta during occlusion of these vessels (Figs. 4-6). Such a hypothesis also appears to assist in explaining the patterns of pressure and flow waves in the systemic arteries (13).

Taylor (12, 14) has discussed the factors which determine the shape of impedance curves in the ascending aorta, and has stressed the importance of (a) the nonuniform elastic properties of the arterial system and (b) the distributed nature of the arterial terminations in providing a low input impedance to pulsatile flow from the heart. These factors are responsible for the steep initial fall in impedance modulus from its value at zero frequency, and for the absence of any marked rise in impedance at higher frequencies. The
position of the heart in relation to arterial terminations in the upper and lower parts of the body is seen as a special example of b. The arrangement of the major arterial branches appears to be desirable in maintaining a low impedance minimum at those frequencies which contain the greatest energy of the ventricular ejection wave. Without such an arrangement the impedance modulus would rise and fall in the same way as it does in the descending thoracic aorta, and this one would expect to cause large changes in the amplitude of the aortic pressure wave and in the magnitude of pulsatile external left ventricular work (vide supra) with alterations in heart rate; this, clearly, would be undesirable. The beneficial effects of having the heart pump into a long and a short reflecting system are seen in Figure 8 where the in-phase impedance \( |Z| \cos \phi \) is plotted against frequency for the ascending aorta under control conditions, the ascending aorta during occlusion of the brachiocephalic artery, and the proximal descending thoracic aorta.

In the ascending aorta under control conditions, in-phase impedance remained low over the frequencies of interest, while in the descending thoracic aorta it fluctuated considerably. When the brachiocephalic artery was occluded there were greater fluctuations of impedance in the ascending aorta, and one would expect these to have approached those seen in the descending thoracic aorta if, in addition, the left subclavian artery were occluded.

**EFFECTS ON ASCENDING AORTIC IMPEDANCE CURVES OF CHANGES IN MEAN ARTERIAL BLOOD PRESSURE AND PERIPHERAL RESISTANCE**

The changes in impedance during alterations in mean arterial blood pressure are shown in Table 1 and Figure 7. The principal effects are seen to be a shift of the curves to the left when blood pressure falls and to the right when blood pressure rises. These findings are similar to those in the femoral artery (9), and are readily explained as being due to alterations in pulse wave velocity, wave velocity rising when blood pressure increases and falling when pressure decreases (15). There are two other features in these curves which were often, though not always, present in other animals when blood pressure was altered. The first was that the two minima of modulus and phase tended to be more distinct at low pressure and less obvious when blood pressure was high. At first sight this may appear to be due to progressively greater cancellation of reflected waves from different sites when the minima occurred at a higher frequency (12). This explanation, however, cannot be invoked since the relationship between path length and wave length at the resonant frequency should remain the same when wave velocity is altered by changes in blood pressure. There remains the possibility that an increase in blood pressure may cause relatively greater changes in wave velocity in the vessels supplying the lower part of the body than in those to the head, neck, and upper limbs. The second feature, which is difficult to reconcile with the above findings, is the change in characteristic impedance at different blood pressures. As blood pressure rises, the characteristic impedance may decrease despite the rise in wave velocity (indicated by an increase in the frequency at which impedance minima occur). One would expect that characteristic impedance would change in the same direction as pulse wave velocity, but in many animals the changes were clearly in the opposite direction. There is, however, a difference between characteristic impedance and wave velocity determined from the frequency of impedance minima. The latter is determined by wave velocity between the recording and peripheral reflecting sites, while characteristic impedance depends on the cross sectional area of, and wave velocity in, the artery immediately distal to the site of measurement (14, 16).

To explain the experimental findings, it is necessary to suggest that when mean blood pressure rises, the wave velocity in the ascending aorta and aortic arch does not increase as much as the cross sectional area.

When the peripheral resistance is reduced, the changes in ascending aortic impedance result from a decrease in reflection coefficient at the arterial terminations (9, 17). It has
been shown above that by causing impedance modulus to fall below the characteristic impedance at those frequencies which contain the greatest energy of the ventricular ejection wave, reflected waves from the arterial terminations help to reduce the extra external work the heart must do in pumping intermittently. One would expect that during peripheral vasodilation, pulsatile energy losses in the arterial system would increase; this has been shown to be true.1 These findings pose the question—is the arterial system at peak efficiency at rest and does it become less efficient during exercise when the peripheral resistance is low? The answer to this cannot be given from the findings in acute experiments. During exercise, the broad minimum of impedance modulus may be partly preserved since vasodilation occurs only in active muscle while arteriolar tone in other regions is maintained or even increased.

The results presented here extend the earlier findings of Patel et al. (6). These authors did not show a double minimum of impedance modulus and phase under control conditions and because of scatter in their experimental data, they were unable to evaluate the changes brought about by intravenous noradrenaline and isoproterenol. There are three principal reasons for the differences between the two studies. Firstly, the semiautomatic methods of data preparation we used were probably more accurate than the manual methods employed by Patel et al., and certainly enabled more data to be analyzed. Secondly, spectral analysis applied to data obtained with the heart beating irregularly proved a far better method for showing the details of impedance patterns and subtle changes under different conditions than Fourier analysis with the heart beating regularly; nonetheless, all the features described here were also seen when Fourier analysis was used. Thirdly, because the patterns of vascular impedance depend on body size, we graphed all experiments individually, while Patel and co-workers pooled their results. This, we consider, is the main factor responsible for the differences between the two studies.

However, on reviewing the published data of Patel et al. in the light of the work published here, it is possible to see certain features to which the previous workers did not draw attention—a double minimum of impedance modulus under control conditions and during vagal stimulation, smaller fluctuations in modulus and phase during the infusion of isoproterenol, and a shift of impedance curves to the left when blood pressure was decreased by vagal stimulation.

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References


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