Input Impedance of the Systemic Circulation in Man

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SUMMARY To determine the systemic input impedance, pulsatile pressure and flow were measured in the ascending aorta in 16 human subjects who were undergoing diagnostic cardiac catheterization. Blood flow was measured with a catheter-tip electromagnetic velocity meter, and pressure with an external transducer connected with the fluid-filled lumen of the catheter. Five subjects were found to have no evidence of cardiovascular disease (group A, mean age 32 ± 2 years, mean aortic pressure 97 ± 4 mm Hg). Seven had clinical and angiographic signs of coronary arterial disease, and mean pressures less than 100 mm Hg (group B, mean age 48 ± 2 years). Four subjects had signs of coronary disease and mean pressures greater than 100 mm Hg (group C, mean age 48 ± 3 years). The frequency spectra of impedance were qualitatively similar in all three groups and resembled those previously observed in the canine aorta. Characteristic impedance was lower in the normal subjects (group A, average 53 dyn sec cm⁻⁵) than in the subjects with coronary artery disease (groups B and C, average 129 dyn sec cm⁻⁵). Among the subjects with coronary disease, characteristic impedance was higher in the hypertensive subjects (group C, average 202 dyn sec cm⁻⁵) than in those with lower mean pressures (group B, average 95 dyn sec cm⁻⁵). External left ventricular work per unit time (hydraulic power) averaged 1715 milliwatts (mW) in group A, 1120 mW in group B, and 2372 mW in group C. Cardiac outputs were within normal limits in all subjects, but tended to be lower in group B than in group C. These results suggest that the subjects of group C were better able to meet the increased energy demands imposed by an abnormally high aortic input impedance. Further investigation is needed to learn whether the high impedances in subjects with coronary disease represent an increase with age and transmural pressure alone, or whether some additional factor is involved. The data on relatively normal subjects permit a tentative definition of the normal limits for aortic input impedance in man: 26-80 dyn sec cm⁻⁵.

CLINICAL investigations of cardiovascular function have until recently been based on what might be called "steady flow hemodynamics," in that mean blood flow (cardiac output) has been measured, rather than pulsatile flow. The time-varying contours of pressure pulsations have been studied intensively, but the lack of methods for measuring instantaneous blood flow in human subjects prevented similar studies of flow waves. The first direct measurements of pulsatile flow in the ascending aorta of man were obtained with a perivascular electromagnetic probe placed around the vessel at the time of surgery.²,³ Advances in design have now made it possible to place the probe at the tip of a cardiac catheter that can be inserted into a peripheral vessel and then advanced to the site where the flow is to be measured.²,³ As a result, pulsatile pressure and flow can be measured simultaneously during clinical aortic catheterization, and from these data the input impedance of the arterial tree and the total hydraulic energy required to move blood into the system can be calculated. The input impedance of an arterial system, defined as the ratio of pressure harmonics to flow harmonics at the entrance to the system, depends on the dimensions and viscoelasticity of the artery involved, on the physical properties of the blood, and on waves reflected from more distal parts of the arterial tree. Measurements of aortic input impedance provide information about the physical state of the arteries, an assessment of the external "afterload" faced by the left ventricle.⁴-⁸ and complete data for calculating the external work of the ventricle.⁹-¹¹

The present investigation was undertaken to determine the aortic input impedance spectra, and the hydraulic power associated with aortic blood flow, in human subjects who had no cardiovascular disease, to the extent such individuals could be found among patients referred to a diagnostic catheterization laboratory, and in patients with coronary arterial disease. Studies of this kind have been reported previously on only a few subjects.¹,²,⁶,¹²,¹³ Our results provide a tentative definition of the normal impedance spectrum, based on five relatively normal subjects, and show higher values of impedance moduli in 11 patients with coronary arterial disease. The linearity of pulsatile pressure-flow relationships was tested in some of these patients by measuring aortic impedance at different heart rates.

Methods

The observations reported here were made on adults who were undergoing diagnostic cardiac catheterization. Patients with evidence of congenital or valvular heart disease were excluded, but the subjects were otherwise unselected. The study was approved by the appropriate institutional committees for clinical investigation, and informed consent was obtained from each patient. Sodium pentobarbital (100 mg p.o.) was given prior to catheterization. Clinical and angiographic signs of coronary arterial disease were found in 11 of the 16 patients studied. The remaining five gave a history of nonspecific chest pain, but had normal coronary angiograms, normal hemodynamics, and no objective indications of cardiovascular disease.

Velocity of blood flow was measured with electromagnetic catheter-tip velocity transducers. A Mills catheter-tip probe²,⁶-¹⁰, and flowmeter (model 275, S.E. Laborato-
verted pressure and velocity data to Fourier series, applied to the system was determined by the free vibration tech-

calculator (Hewlett-Packard, model 9820A) which con-

analog-to-digital converter (Technical Instruments, model 400B). Data analysis was carried out on a programmable calculator (Hewlett-Packard, model 9820A) which converted pressure and velocity data to Fourier series. applied

corrections for the measured dynamic responses of the transducers, and computed aortic impedance and hydraulic power as functions of frequency. Input impedance modulus at each harmonic frequency was computed by dividing flow modulus by pressure modulus, and impedance phase by subtracting the phase angle of flow from that of pressure. The impedance of 0 Hz, or "input resistance," was calculated by dividing mean flow into mean pressure. Impedances were not calculated for harmonics in which the pressure modulus was less than 0.6 mm Hg, or flow modulus less than 1 cm²/sec; these are values that probably represent the noise levels of our measurement systems. In effect, this procedure eliminated all data above about 12 Hz. Characteristic impedance was estimated by averaging impedance moduli between 2 and 12 Hz. Theoretically, characteristic impedance depends on the physical properties of the vessel under study, while input impedances oscillate around the characteristic value because of waves reflected from more distal points.

The hydraulic energies associated with aortic blood flow were computed by methods previously reported.

Results

The data obtained from each subject are summarized in Tables 1–3. Clinical and angiographic signs of coronary arterial disease were found in 11 of the 16 subjects studied, including two who had previously been treated surgically for coronary disease. No cardiovascular abnormality was found in the other five individuals. For purposes of comparison, the subjects were divided into three groups:

Group A ("normals"): Five subjects who had given a history of nonspecific chest pain, but who were found to have normal hemodynamics, normal coronary angiograms, and no objective indications of cardiovascular disease. Their ages ranged from 28 to 37 years and averaged 32 years. Mean aortic pressure averaged 97 + 4 (SEM) mm Hg.

Group B: Seven subjects with evidence of coronary disease and mean aortic pressures <100 mm Hg. Their average age was 48 years, and average aortic pressure 85 ± 1 (SEM) mm Hg.

Group C: Four subjects with coronary disease. and mean aortic pressures >100 mm Hg. Average age was 48 years, and average pressure 120 ± 5 (SEM) mm Hg. Peripheral vascular resistance in these subjects was not significantly different from that in group B, but cardiac outputs were higher in group C than in group B.

The waveforms of the measured flow curves were similar to those previously recorded by electromagnetic flowmeters in the ascending aorta of the dog and man. An example from each group is shown in Figures 1–3. Peak flows were much lower in subjects with coronary artery disease (average in groups B and C = 433 ± 27 ml/sec) than in the normal subjects (average = 712 ± 53 ml/sec). The low peak flows in group C were associated with prolonged ejection times (0.367 ± 0.022 seconds as compared to 0.313 ± 0.019 seconds for the normals).

The average aortic input impedance spectrum for each group is shown in Figures 4–6. Because of the large differ-

References

1. Feltham, Middlesex, England) were used in nine cases. The frequency response of this system at the filter settings used in this study, measured by applying an appropriate electrical input signal to the system was constant in amplitude (±5%) from 0 to 32 Hz. Phase shift was approximately linear and equivalent to a time delay of 17 msec. This response is adequate for accurate measurement of pulsatile velocities in the human aorta because more than 98% of the variance of the pulsations is included in the first seven harmonics (unpublished observations). In the seven other subjects, a Carolina probe flowmeter was used (model 601D, Carolina Medical Electronics). The dynamic response of this system was constant (±5%) in amplitude from 0 to 14.5 Hz, with a time delay of 35 msec. The catheter containing the Carolina probe included a flexible radiopaque tail extending 5 cm beyond the velocity sensor. The tail was advanced through the aortic valve and into the left ventricle, so that the sensing electrodes were at approximately the upper border of the sinuses of Valsalva. This arrangement tends to stabilize the probe in the central axis of the ascending aorta, eliminates the spurious signal that appears if the probe comes to lie against the wall of the vessel, and minimizes artifacts in the recorded velocity waveform caused by motion of the catheter.

Measurements of blood flow were calibrated by one of two different methods. In the first method, the velocity calibration of the probe was determined in vitro in a hydraulic model, and the internal cross-sectional area of the vessel was measured by radiography at the time of catheterization. Since the velocity profile of the ascending aorta is relatively flat, the product of measured velocity and cross section is volume of blood flow per unit time. In the second method, the output signal of the velocity meter was calibrated in cm³/sec by reference to a simultaneous determination of cardiac output by the dye-dilution method. A comparison of the two calibration techniques in eight subjects showed excellent correlation ($r = 0.97, y = 1.08x – 0.33$ liters/min, $P < 0.001$). The velocity signal averaged over the last third of diastole was taken to represent zero flow. Both kinds of velocity probe have been shown to be thermally and electrically safe for use in man. Under local anesthesia with 1% lidocaine, a sterilized velocity catheter was inserted percutaneously into a femoral artery, or else through a brachial arteriomy. With the aid of fluoroscopy, the velocity probe was then positioned in the ascending aorta.

Aortic pressure was measured through the fluid-filled lumen of the velocity catheter with a Millar strain gauge transducer attached externally. The frequency response of this system was determined by the free vibration technique and the damped natural frequency in these studies ranged from 27 to 36 Hz. with a damping ratio of 0.12–0.17.

The pressure and velocity signals were recorded on analog magnetic tape (Hewlett-Packard, model 3960) and later digitized at a sampling interval of 10 msec by an analog-to-digital converter (Technical Instruments, model 400B). Data analysis was carried out on a programmable calculator (Hewlett-Packard, model 9820A) which converted pressure and velocity data to Fourier series. applied
grouped for averaging by frequency rather than harmonic number. In all cases, the moduli of the input impedance fell steeply from a high value at zero frequency (the input resistance) to a minimum between 2.3 and 5.3 Hz, and then rose to a less well defined peak at 6–9 Hz. In the subjects of group A the minimum occurred at frequencies between 2.5 and 4.2 Hz (average, 3.1); in group B, between 2.3 and 5.3 Hz (average, 4.1); and in group C, between 3.7 and 5.0 Hz (average, 4.5). The impedance phase was negative (i.e., flow led pressure) for the first three harmonics in 14 of the 16 cases. The phase angle was positive for the higher harmonics, crossing zero at approximately the frequency of the modulus minimum.

The average characteristic impedances of the three groups differed to a striking degree. The value in the normal group was 53 ± 4 dyn sec cm⁻³, while the average in group B was almost twice that value, and the average in group C was 4 times the normal level (Table 2). Input resistances were approximately the same in all groups.

Total hydraulic power associated with aortic blood flow (including kinetic energy) averaged 1715 ± 240 milliwatts (mW) in group A (Table 3). Average total power was significantly lower in group B than in the normals (group

### Table 1 Hemodynamic Data

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, sex</th>
<th>Diagnosis</th>
<th>Ejection fraction</th>
<th>Cardiac index (liters/min/m²)</th>
<th>Aortic radius (cm)</th>
<th>Heart rate (beats/min)</th>
<th>Surface area (m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>37 M</td>
<td>Normal</td>
<td>0.71</td>
<td>2.6</td>
<td>1.42</td>
<td>64</td>
<td>2.28</td>
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<tr>
<td>2</td>
<td>32 M</td>
<td>Normal</td>
<td>0.65</td>
<td>2.5</td>
<td>1.54</td>
<td>90</td>
<td>2.23</td>
</tr>
<tr>
<td>3</td>
<td>28 M</td>
<td>Normal</td>
<td>0.61</td>
<td>2.5</td>
<td>1.54</td>
<td>90</td>
<td>2.23</td>
</tr>
<tr>
<td>4</td>
<td>35 M</td>
<td>Normal</td>
<td>0.66</td>
<td>3.0</td>
<td>1.91</td>
<td>83</td>
<td>2.10</td>
</tr>
<tr>
<td>5</td>
<td>30 M</td>
<td>Normal</td>
<td>0.57</td>
<td>2.9</td>
<td>1.71</td>
<td>77</td>
<td>1.90</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>32 ± 2</td>
<td></td>
<td>0.64 ± 0.02</td>
<td>3.3 ± 0.52</td>
<td>1.60 ± 0.10</td>
<td>76 ± 5</td>
<td>2.11 ± 0.07</td>
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</tbody>
</table>

### Table 2 Hemodynamic Data—continued

<table>
<thead>
<tr>
<th>Subject</th>
<th>Mean</th>
<th>Systolic</th>
<th>Diastolic</th>
<th>Pulse</th>
<th>SV (ml)</th>
<th>Peak (ml/sec)</th>
<th>Mean (ml/sec)</th>
<th>Ejection time (sec)</th>
<th>R</th>
<th>Zₐ</th>
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</thead>
<tbody>
<tr>
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<td>86</td>
<td>103</td>
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<td>36</td>
<td>94</td>
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<td>51</td>
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<td>917</td>
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<td>50</td>
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<td>36</td>
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<td>86</td>
<td>40</td>
<td>72</td>
<td>698</td>
<td>92</td>
<td>0.300</td>
<td>1530</td>
<td>53</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>97 ± 4</td>
<td>114 ± 4</td>
<td>78 ± 4</td>
<td>36 ± 2</td>
<td>95 ± 20</td>
<td>712 ± 53</td>
<td>114 ± 17</td>
<td>0.313 ± 0.019</td>
<td>1218 ± 147</td>
<td>53 ± 4</td>
</tr>
</tbody>
</table>

R = input resistance in dyn sec cm⁻³ (mean aortic pressure divided by mean flow); Zₐ = characteristic input impedance modulus (sec text) in dyn sec cm⁻³; SV = stroke volume.
A), and there was little overlap in the individual values. Six of the seven subjects in group B had levels of hydraulic power lower than any observed in the normal subjects. The group A average probably overestimates the total power to be expected in normal subjects, because of the relatively high cardiac output (11.0 liters/min) in one individual (subject 2). If this subject were excluded, the average would be 1,488 mW. Adopting this value for the normal average would probably overestimate the total power to be expected in man. There is no reason to believe that they are not satisfied in man.

The use of Fourier analysis to define impedance also assumes linearity of the system in which the pressure and flow pulses are measured. Several techniques have been employed to test this assumption in the arterial system of the dog.27 and all of them have shown approximately linear relationships. To test the linearity of the pressure-flow relationships in the human aorta, we measured impedance as the input waveforms were altered by electrically pacing the heart at different rates28-32 in five subjects. Changes in the fundamental frequency, or heart rate, in the range 1.4-2.6 Hz produced no consistent changes in the impedance spectrum, demonstrating that the system behaves in an almost linear fashion in this range.

**LINEARITY**

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 dog27 and there is no reason to believe that they are not satisfied in man.
range. Pacing was carried out in at least one subject from each group and an example from group A is shown in Figure 7. The relatively small shifting of points on the impedance spectrum with pacing appeared to be a random variation associated with errors of measurement rather than a systematic change related to the fundamental rates. Another conceivable source of error lies in the respiratory swings of intrathoracic pressure, which might alter aortic compliance and hence impedance. In all subjects studied there were small variations in the pressure and flow during the respiratory cycle. In four subjects pulses were analyzed throughout the respiratory cycle, including samples at maximum inspiration and expiration. The results of each test were similar to the example shown in Figure 8, demonstrating that respiration per se does not influence aortic input impedance.
FIGURE 7  Input impedance of the systemic circulation in a normal young adult at five different heart rates, showing no significant change of impedance pattern. Resting heart rate was 84 beats/min, and faster rates were produced by electrical pacing. The relative constancy of the impedance patterns with changes of heart rate is evidence that the arterial system is approximately linear. There is a well defined minimum in the modulus at 2.5 Hz.

**Discussion**

**NORMAL IMPEDANCES**

The aortic input impedance spectra in our subjects closely resemble the examples published previously by other investigators. In spite of the variety of methods and subjects, certain distinctive features of the aortic impedance spectrum have been noted consistently. Impedance moduli are usually less than 1/10 the amplitude of the input resistance, and there is a steep decline in modulus at low frequencies to a minimum between 2 and 6 Hz, followed by a maximum at about twice the frequency of the minimum. There is usually a well defined minimum in the modulus at 2.5 Hz.

The characteristic impedance in our normal subjects (53 ± 4 dyn sec cm\(^{-5}\)) is lower than that in previously published human aortic impedance spectra. The spectra previously reported were from subjects with cardiovascular disease. Patel and his colleagues measured impedance in the ascending aorta in three subjects with atrial septal defects, and the average characteristic impedance was approximately 82 dyn sec cm\(^{-5}\) (our estimate from their figures). The ages of their subjects were 24-35 years; mean aortic blood pressures were 74-94 mm Hg. Their data were obtained with a rigid velocity probe around the aorta, and under conditions of open thoracotomy. Gabe and his associates catheterized the aorta with a double-lumen catheter in three subjects with rheumatic mitral valvular disease, measured flow by a pressure-gradient method, and computed impedance. Their figures indicate an average characteristic impedance of about 100 dyn sec cm\(^{-5}\). Age ranged from 37 to 39 years, and mean pressure from 83 to 98 mm Hg. Mills and his group, who used methods similar to those in the present investigation, presented impedance spectra for the ascending aorta in two subjects, 40 and 51 years of age, both diagnosed as cases of ischemic heart disease. (Data from 21 other subjects were reported in Mills’ paper, but ascending aortic impedance measurements were presented for only two.) The characteristic impedance in these subjects was apparently much higher than in our normal subjects. Their impedances were expressed as pressure-velocity ratios.
and we estimate the characteristic impedance at approximately 600 dyn sec cm⁻³. Converted to the same units, our group A average is 416 dyn sec cm⁻³. Blood pressures and cardiac outputs were normal in their subjects.

We conclude from these reports and our own observations that many different pathological conditions can elevate the characteristic impedance in the ascending aorta. The number of normal subjects studied is as yet too small to permit firm conclusions about normal limits, but we suggest as a tentative guide that in the absence of cardiovascular disease or elevated blood pressure, under basal conditions, characteristic impedance in the ascending aorta in man is usually between 26 and 80 dyn sec cm⁻³ (group A mean ± 3 SD).

**EFFECTS OF AGE AND PRESSURE**

Transmural pressure and age are known to have an important influence on the stiffness of the vascular wall, and for that reason it has been widely assumed that these variables affect the characteristic impedance of arteries. Consideration of the characteristic impedance (Zₒ) in relation to mean aortic blood pressure and age in our subjects suggests that these two factors may account for the relatively high characteristic impedances in the individuals with coronary disease (Fig. 9). The graph on the left side of Figure 9 shows that the difference in Zₒ between groups A and C appears to be related to the higher pressures in the latter group. The right side of the figure indicates that the difference between groups A and B is associated with the older age of the subjects in the latter group. In addition, groups B and C have about the same age distribution, and the subjects with higher pressures tend to have the highest characteristic impedance. Whether age and distending pressure completely account for the observed differences, however, is a question that cannot be answered until data are available on older normal subjects, and on subjects with coronary disease who are under the age of 40.

A direct relation between age and arterial impedance is to be expected. Arteries unquestionably become less compliant with age, and experimental stiffening of the aortic wall by external constraints has been shown to increase characteristic aortic impedance. The effect of increased transmural pressure on vascular impedance is far from certain, however. Arteries become stiffer as they are distended, presumably because more and more of the wall stress is borne by collagen as the diameter increases. Vascular impedance is directly proportional to the stiffness (elastic modulus) of the vascular wall, but it is also inversely proportional to the cross-sectional area of the vessel. The net effect of increased transmural pressure on impedance is therefore difficult to predict, and the literature on the subject gives no definitive answer. The elevation of arterial pressure that follows intravenous infusion of norepinephrine, for example, is not accompanied by an increase in characteristic aortic impedance in the dog, though the impedance minimum is shifted to higher frequencies. Nevertheless, in two of the three subjects studied by Gabe and his associates there was a slight increase in aortic impedance during norepinephrine infusion.

In any case, the response to vasoactive agents is not an appropriate test, because they alter not only the distending pressure but also the active tension of smooth muscle in the vessel being examined. The evidence is inconclusive, and it may be that transmural pressure plays only a small part in determining characteristic impedance in vivo. Conceivably, our results may depend less on transmural pressure than on a correlation between disease of the coronary arteries and some pathological process in the aortic wall, perhaps an exaggeration of the intramural effects of aging on the whole arterial tree.

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**FIGURE 9** Variations in characteristic impedance (Zₒ) with age and mean arterial pressure. Values for the subjects with normal cardiovascular hemodynamics (group A) are indicated by the open circles (O). Values for the subjects with coronary artery disease and mean arterial pressures less than 100 mm Hg (group B) are indicated by the closed circles (●). Values for the subjects with coronary artery disease and mean arterial pressures greater than 100 mm Hg (group C) are indicated by the crosses (×). See text for discussion.

**FIGURE 10** Total external hydraulic power of the left ventricle in the three groups of subjects. The upper portion of the figure indicates the amount of power required to move blood through the systemic circulation in a pulsatile manner (oscillatory terms) and the lower portion indicates the amount of power associated with mean blood flow (mean terms).
HYDRAULIC POWER

The hydraulic power, or work per unit time, associated with blood flow at the root of the aorta depends not only on the ability of the left ventricle to do external work, but also on the properties of the arterial tree into which blood is ejected. Aortic input impedance is an expression of these properties. On the one hand, the stiffer the aorta, the higher the impedance moduli and the greater the amount of work required to produce a given blood flow. On the other hand, given a constant impedance spectrum, the smaller the pressure and flow generated by the ventricle, the lower the external work and power. Consequently, the performance of the heart and the state of the aorta must both be taken into account in interpreting measurements of hydraulic power. A useful “rule of thumb” for this purpose, derived from the equations for computing hydraulic power,16 states that power equals flow squared multiplied by resistance (or impedance). The “steady flow” component of power is calculated by using mean flow and input resistance as the elements in this expression. The pulsatile component for any one harmonic is calculated by inserting the appropriate flow modulus and real impedance.

The relatively low total hydraulic power in the subjects of group B (average, 1.120 mW, as compared to 1.715 mW in group A) (Table 3 and Fig. 10) thus indicates that the increased characteristic impedance in Group B was outweighed by a relatively low blood flow in that group. The subjects in group C, in contrast, had mean flows that were not significantly different from those in the normal subjects, in spite of a high impedance. They maintained normal cardiac outputs in spite of an increased afterload, in other words, though at the cost of a marked increase in work per unit time (2.372 mW). This ability to generate more power in the face of a high impedance suggests that ventricular performance in group C was in some sense better than in group B. The possibility that this relationship might be used to evaluate ventricular behavior is worth exploring.

The distinction between “oscillatory” and “mean” or “steady flow” components of hydraulic power16 is useful because the steady flow terms represent energy that is dissipated primarily in the arterioles and capillaries, while the pulsatile terms depend mainly on the elasticity of the aorta. One manifestation of diminished aortic compliance is an increase in pulsatile power, as is evident in group C. In the oldest subject of that group the pulsatile component was 588 mW, or 26% of total power. Whatever the cause of the elevated aortic impedance in these subjects, the physical state of the aorta has clearly increased the energy that must be supplied by the left ventricle to move blood into the systemic circulation.

References
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