Input Impedance of the Pulmonary Arterial System in Normal Man

Effects of Respiration and Comparison to Systemic Impedance

Joseph P. Murgo and Nico Westerhof

With the technical assistance of Stephen A. Layton and John Paul Giolma

From the Cardiology Service, Department of Medicine, Brooke Army Medical Center, Fort Sam Houston, Texas, and the Laboratory for Physiology, Free University, Amsterdam, The Netherlands

SUMMARY. Input impedance of the pulmonary arterial system was determined in 10 subjects undergoing elective cardiac catheterization. No cardiovascular or pulmonary disease was found in these patients. In five of the subjects, systemic arterial input impedance was also obtained, so that both systems could be compared. Pulmonary and systemic peripheral resistances were 79 ± 9 dynes sec/cm$^5$ (mean ± SEM) and 1016 ± 50 dynes sec/cm$^5$, respectively. Characteristic impedance of the pulmonary circulation was lower than the characteristic impedance of the systemic circulation: 20 ± 1 dynes sec/cm$^5$ vs. 47 ± 9 dynes sec/cm$^5$, respectively. Pulmonary pressure and flow spectra for both systems are also presented. The amplitudes of the harmonics of pressure and flow are smaller for the pulmonary circulation, which is consistent with the lower pressures and more rounded waveforms of the normal pulmonary circulation. In all 10 subjects, input impedance of the pulmonary system was examined during both the inspiratory and expiratory phases of respiration. There was no difference between inspiration and expiration in either pulmonary vascular resistance (77 ± 10 dynes sec/cm$^5$ vs. 80 ± 9 dynes sec/cm$^5$, respectively), characteristic impedance (20 ± 1 dynes sec/cm$^5$ vs. 20 ± 1 dynes sec/cm$^5$) or in the overall impedance spectrum. Quiet respiration, thus, has no effect on the pulmonary arterial load, and changes in pressure and flow must result from alterations in right ventricular performance.

(Circ Res 54: 666-673, 1984)

UNLIKE the systemic circulation, the pulmonary vascular system in normal man is a low pressure system which is entirely exposed to changes in intrathoracic pressure during respiration. The effects of these changes on the physical characteristics of the human pulmonary circulation are unknown. It is of physiological and clinical importance to know how the pulmonary vasculature is affected by respiration, especially when evaluating the relationships between this vascular bed and right ventricular function. To study such effects, it is necessary to describe the pulmonary circulation in quantitative terms. One method is to calculate pulmonary input impedance. The input impedance of a vascular bed not only provides information about the pulsatile pressure-flow relationships, but also yields information regarding the physical characteristics of that bed (Randall and Stacey, 1956; Noordergraaf, 1969; Gessner, 1972; McDonald, 1974, pp 380–383; Westerhof et al., 1979; O’Rourke, 1982a, 1982b; Milnor, 1982).

Previous studies of pulmonary artery input impedance have been primarily restricted to animal experiments (Caro and McDonald, 1961; Patel et al., 1963; Bergel and Milnor, 1965; Milnor et al., 1966; O’Rourke, 1968; Elkins and Milnor, 1971; Reuben et al., 1971; Pace, 1971; Elkins et al., 1974; Piene, 1976a, 1976b; Hopkins et al., 1979, 1980). Pouleur et al. (1978) analyzed the effects of artificial lung inflation on anesthetized dogs, but the effects of normal respiratory mechanics on the input impedance of the pulmonary arterial system have not been studied.

Due to limitations in obtaining simultaneous pulsatile pressure and flow signals in the pulmonary artery in humans, only two studies on pulmonary input impedance in man have been reported (Milnor et al., 1969; Mills et al., 1970). Technological capabilities at the time of these studies were limited, since pulmonary artery pressure was measured by means of fluid-filled catheter-manometer systems and, in the study of Milnor et al. (1969), flow was derived from differential pressure techniques. None of the patients in those studies were free of organic heart disease, and the effects of respiration on pulmonary impedance were not evaluated. Recently, it has been suggested that respiration-induced changes in right ventricular ejection may be a result of changes in the impedance characteristics of the pulmonary vascular bed, as well as changes in right
ventricular filling (Shaver et al., 1974; Curtis et al., 1975; Pouluer et al., 1978).

Input impedance of the systemic and pulmonary arterial systems has been shown to be sensitive to changes in those systems, either within a given subject under different physiological conditions (Murgo et al., 1981b), as a result of pharmacological interventions (Gundel et al., 1981; Van den Bos et al., 1982), or between groups of subjects under different physiological or pathological states (Nichols et al., 1977; Murgo et al., 1980). In recent years, the development of multisensor catheterization techniques (Murgo and Millar, 1972; Murgo, 1975) and the application of dedicated minicomputers for human hemodynamic research (Murgo et al., 1977) have greatly facilitated the calculation of input impedance in the clinical laboratory environment. Using these techniques, the present study was designed to analyze the input impedance of the pulmonary arterial system in normal man for the first time, compare the impedance spectra of the pulmonary vascular tree to the systemic tree in the same patients, and investigate the effects of respiration on pulmonary input impedance.

Methods

Patient Selection and Catheterization Techniques

Ten patients were electively catheterized for a variety of clinical indications, the most common of which was a chest pain syndrome. No cardiovascular disease or pulmonary disease was found. Left and right heart hemodynamic measurements obtained during rest and exercise, left ventricular angiography, and coronary arteriography all were normal. All patients were studied in a basal state and were either unsedated or very lightly sedated with diazepam, 10 mg per os (Valium, Roche Laboratories), 1 hour prior to the procedure. Hemodynamic measurements were performed only during rest and steady state conditions, which were determined by a stable heart rate and stable sequential pulmonary artery hemoglobin oxygen saturation measurements. Cardiac output was measured at rest, using the direct Fick method (Slonin et al., 1967) with duplicate determinations performed for the evaluation of reproducibility. All hemodynamic measurements, including the pressure and flow velocity signals used to derive impedance, were recorded during this period before any angiography was undertaken. The study protocol was approved by the Clinical Investigation and Human Use Committees at Brooke Army Medical Center, and the United States Army Surgeon General's Office. Informed consent was obtained from all patients.

Custom-designed left and right heart multisensor catheters were utilized in all patients. The right heart catheter contained three solid state pressure sensors and an electromagnetic flow velocity probe (Millar Instruments, Inc.). The pressure sensors were mounted laterally with the most distal sensor located 7 cm from the tip, the middle sensor, 5 cm from the distal, and the most proximal sensor, 11.5 cm from the middle. The electromagnetic flow velocity probe was constructed in the same housing containing the distal pressure sensor and designed so that the sensing electrodes were mounted at the same site as the pressure sensor. The catheter was introduced into the basilic vein from the right antecubital fossa and advanced under fluoroscopic control and pressure monitoring through the right heart chambers into the pulmonary artery. The catheter was positioned so that the distal pressure sensor and electromagnetic flow probe was placed in the main pulmonary artery, the middle sensor in the right ventricular outflow tract, and the proximal sensor in the right atrium. The catheter was 8F in size up to the distal pulmonary artery pressure sensor. The 7-cm section distal to the pulmonary artery sensor was 7F in size and provided flexibility for passage through the right heart and, once in the right pulmonary artery, provided stabilization of the pressure and flow velocity transducers in the main pulmonary artery. A second, balloon flotation catheter was passed into the pulmonary artery to obtain blood specimens and provide a hydrostatic reference point for the microanometer. Systemic pressure and flow signals were obtained from a left heart catheter containing two pressure sensors and an electromagnetic flow probe. Details of the use of this catheter, and the methods and techniques for calculating systemic input impedance, were published earlier (Murgo et al., 1980, 1981a, 1981b). Details of the technical characteristics of pressure and velocity sensors, including frequency response, drift characteristics, calibration techniques, etc., have been described previously.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Yrs.</th>
<th>Height (in.)</th>
<th>Weight (lb.)</th>
<th>BSA (m²)</th>
<th>Resp (breaths/min)</th>
<th>HR (beats/min)</th>
<th>CO (liters/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-1</td>
<td>M</td>
<td>47</td>
<td>66</td>
<td>174</td>
<td>1.88</td>
<td>10</td>
<td>68</td>
<td>7.0</td>
</tr>
<tr>
<td>P-2</td>
<td>M</td>
<td>43</td>
<td>68</td>
<td>160</td>
<td>1.85</td>
<td>8</td>
<td>76</td>
<td>7.0</td>
</tr>
<tr>
<td>P-3</td>
<td>M</td>
<td>38</td>
<td>71</td>
<td>182</td>
<td>2.02</td>
<td>16</td>
<td>61</td>
<td>7.2</td>
</tr>
<tr>
<td>P-4</td>
<td>F</td>
<td>54</td>
<td>67</td>
<td>166</td>
<td>1.85</td>
<td>13</td>
<td>60</td>
<td>6.3</td>
</tr>
<tr>
<td>P-5</td>
<td>M</td>
<td>29</td>
<td>72</td>
<td>185</td>
<td>2.04</td>
<td>12</td>
<td>76</td>
<td>8.2</td>
</tr>
<tr>
<td>P-6</td>
<td>F</td>
<td>53</td>
<td>65</td>
<td>188</td>
<td>1.92</td>
<td>16</td>
<td>59</td>
<td>4.0</td>
</tr>
<tr>
<td>P-7</td>
<td>M</td>
<td>48</td>
<td>66</td>
<td>172</td>
<td>1.86</td>
<td>12</td>
<td>61</td>
<td>7.9</td>
</tr>
<tr>
<td>P-8</td>
<td>M</td>
<td>23</td>
<td>73</td>
<td>209</td>
<td>2.19</td>
<td>13</td>
<td>64</td>
<td>7.3</td>
</tr>
<tr>
<td>P-9</td>
<td>F</td>
<td>40</td>
<td>67</td>
<td>160</td>
<td>1.86</td>
<td>12</td>
<td>79</td>
<td>7.4</td>
</tr>
<tr>
<td>P-10</td>
<td>F</td>
<td>33</td>
<td>67</td>
<td>155</td>
<td>1.86</td>
<td>21</td>
<td>72</td>
<td>7.0</td>
</tr>
</tbody>
</table>

Mean ± SD 41 ± 10 68 ± 3 175 ± 16 1.93 ± 0.11 13 ± 4 68 ± 8 6.9 ± 1.1

BSA = body surface area; Resp = respiratory rate; HR = heart rate; CO = Fick cardiac output.
FIGURE 1. Physiological signals obtained during multisensor catheterization. Displayed from the top: electrocardiogram (ECG), pulmonary artery (PA) flow velocity, pulmonary artery, right ventricular (RV), and right atrial (RA) pressures, aortic (AO) flow velocity, aortic and left ventricular (LV) pressures, and a respiratory signal (unlabeled) at the bottom of the figure. Inspiration is indicated by a downward direction of the respiration signal. In the last cardiac cycle, a pulmonary artery pressure is displayed both from the micromanometer and a conventional fluid-filled catheter system. This signal was used to reference the micromanometer pressure accurately.

(Murgo and Millar, 1972; Millar and Baker, 1973; Nichols and Walker, 1974; Murgo, 1975; Murgo et al., 1977).

Signal Processing and Computational Techniques

The flow velocity probes were used in conjunction with a Biotronex sinewave flow meter (model BL-613, Biotronex Laboratory, Inc.). The technical details of the analog and digital processing are described elsewhere (Murgo et al., 1977, 1980).

The spatial flow velocity profile in the main pulmonary artery was assumed to be blunt, as was shown for the aorta, by Schultz et al. (1969), so that for transducer movements over the cross-sectional area, calibration was assumed to be constant. The pulmonary flow velocity signal was used to represent instantaneous volumetric flow, and the input impedance of the pulmonary vascular bed was calculated by techniques identical to those applied by our laboratory to the systemic circulation (Murgo et al., 1980, 1981a, 1981b). The input impedance of a vascular bed should be calculated from a pressure difference [pulmonary artery pressure minus pulmonary venous pressure (Westerhof et al., 1979)] and pulmonary artery flow. Direct pulmonary venous or left atrial pressures were not measured, but pulmonary capillary wedge pressure was obtained with a Swan-Ganz catheter. The mean wedge pressure on a beat-by-beat basis was utilized in calculating the mean component (resistance) of the pulmonary input impedance spectrum. In five of the 10 patients, aortic input impedance was also determined so that the input impedance spectra from both systems could be compared. Details on the derivation of systemic input impedance were given earlier (Murgo et al., 1980, 1981a, 1981b). Respiration was monitored with a strain gauge belt around the lower thorax, and beats used for processing were identified as inspiratory or expiratory by their temporal relationship to the respiratory signal. If a cardiac cycle happened to occur in such a manner as to overlap both

FIGURE 2. Individual pulmonary input impedances of 10 normal subjects during inspiration (thin lines) and expiration (thick lines). Vertical bars in the right-hand side of each panel indicate characteristic impedance (± 1 se) as determined from averaging the impedance moduli above 2 Hz. (Pulmonary wedge pressure was not available in patient 5; thus, resistance value is not reported.)
The determination of impedance requires that the system under evaluation be in a steady state [McDonald, 1974 (pp 161-173)]. Both to determine the effects of respiration on input impedance and to minimize errors introduced by analyzing non-steady state beats, a maximum difference of 2 mm Hg was accepted between the start and end of a single pressure tracing. Any beats exceeding this limit were not analyzed. An estimation of the errors allowed could be obtained by considering the addition, to the pressure tracing, of a "saw-tooth" waveform. The amplitudes of the moduli resulting from this saw-tooth waveform [McDonald, 1974 (pp 147-149)] were less than 5% of the amplitudes of the pulmonary pressure harmonics when the height of the saw-tooth was less than 2 mm Hg. The use of pressures with less than 2 mm Hg pressure difference from cycle to cycle resulted in discarding no more than 10% of the total number of cardiac cycles analyzed. In total, 89 ± 15 beats (mean ± SD) per patient were analyzed; 42 ± 8 beats in inspiration and 47 ± 13 beats during expiration.

In determining the actual impedance spectra, we utilized a spectral averaging algorithm to remove the effects of noise in the physiological signals, especially of the flow velocity signal which inherently has a poorer signal:noise ratio than the pressure signals. The details of this algorithm are described elsewhere (Murgo et al., 1980).

Results

The study group consisted of 10 patients whose basic hemodynamic data are given in Table 1. Respiratory rate, heart rate, cardiac output, and main pulmonary artery and pulmonary capillary wedge pressures were within normal limits for all patients. Mean pulmonary artery flow increased from expiration (112 ± 6 ml/sec) to inspiration (123 ± 7 ml/sec). However, mean pulmonary artery pressure decreased from expiration (14.9 ± 0.8 mm Hg) to inspiration (13.7 ± 0.9 mm Hg). Mean pulmonary capillary wedge pressure also decreased from expiration (8.9 ± 0.7 mm Hg) to inspiration (6.8 ± 0.9 mm Hg).

Figure 1 illustrates the pressure and flow waveforms obtained in one of the patients from the right and left heart catheters. The individual pulmonary artery input impedance spectra of the 10 patients during both inspiration and expiration are shown in Figure 2. In eight of the patients, a minimum and maximum pattern in the modulus spectra was seen, with the first minimum occurring between 2 and 4 Hz. In six of these patients, the minima and maxima were accompanied by zero crossings in the phase spectra. There was very little difference in the impedance spectra in each patient between inspiration and expiration, including the zero frequency term (pulmonary vascular resistance) and pulmonary characteristic impedance.

Figure 3 illustrates pulmonary artery pressure moduli, pulmonary artery flow moduli, and the input impedance spectra during both inspiration and expiration averaged by harmonics (Murgo et al.,
1980) for all 10 patients. The higher harmonics for the pulmonary artery pressure signal averaged less than 1 mm Hg so that the major components of pulmonary artery pressure are formed by the mean term plus the first two harmonics. With the exception of the mean pulmonary artery pressure, there are no statistically significant differences (Wilcoxon paired signed ranks test, two tailed, \( P < 0.05 \)) between the various harmonics during inspiration and expiration (Fig. 3). The first two harmonics of flow are larger in amplitude than the mean term. The mean flow and first two harmonics are statistically larger during inspiration than expiration, but the flow harmonics at higher frequencies are similar during both phases of respiration. The average input impedance spectra are also shown in Figure 3. There are no significant differences between the inspiratory and expiratory phases of respiration in either pulmonary vascular resistance (77 ± 10 dynes sec/cm² vs. 80 ± 9 dynes sec/cm², respectively), or in the characteristic impedance (20 ± 1 dynes sec/cm² vs. 20 ± 1 dynes sec/cm²). The average characteristic impedance for the group is approximately 25% of the pulmonary vascular resistance. The minima and maxima observed in the individual curves shown in Figure 2 are lost in the process of averaging the impedance spectra. Similarly, there are no significant differences in phase angles between inspiration and expiration. The phase remains negative to approximately 4 Hz, after which it is close to zero or slightly positive.

To compare the input impedance spectra obtained from the pulmonary artery to that measured in the ascending aorta, Figure 4 illustrates impedance spectra from both vascular beds in the five patients in whom measurements were obtained. For this subgroup, mean aortic pressure was 93 ± 4 mm Hg, mean aortic flow was 123 ± 4 ml/sec, mean systemic vascular resistance was 1016 ± 50 dynes sec/cm², and mean characteristic impedance was 47 ± 9 dynes sec/cm². The individual values for systemic vascular resistance are separately listed in each modulus plot of Figure 4. Thus, the pulmonary vascular resistance is approximately 8% of the systemic vascular resistance and pulmonary characteristic impedance approximately 43% of aortic characteristic impedance.

In Figure 5, the averaged frequency spectra of pressure, flow, and impedance are shown for the pulmonary arterial system in the 10 patients reported in this paper and to the systemic arterial system in 13 patients previously reported by our laboratory (Murgo et al., 1981a). Although marked similarities in overall shape of the pressure spectra are present, it is apparent that all harmonics of aortic pressure are of greater magnitude than the corresponding harmonics of pulmonary artery pressure. With the exception of the mean flow terms which, by necessity and calibration techniques, are set equal to each other, all of the harmonics of the pulmonary flow moduli are lower in magnitude than the respective harmonics of the aortic flow moduli. The average systemic resistance term is listed in the modulus spectral plot, since the harmonics are plotted on the same scale to compare the differences in the two circulations. In general, the impedance moduli of the systemic circulation appear to oscillate more than the impedance moduli of the pulmonary circulation. The phase components of pulmonary artery input impedance are less negative than the ascending aortic input impedance spectra.

Discussion

This study has examined the input impedance of the pulmonary vascular system in 10 human subjects without evidence of cardiovascular or pulmonary disease, with special emphasis on evaluating the effects of respiration. Particular care was taken to examine cardiac cycles in which pressure and flow signals were sufficiently stable to allow for a proper application of the Fourier series.

The results of our study showed no difference in pulmonary vascular resistance between the inspiratory and expiratory phases of respiration. As mean flow increases during inspiration through the circuit, the pressure difference between pulmonary artery
and left atrium (as indicated by pulmonary capillary pressure) also increases, so that resistance remains constant. Thus, during quiet respiration in the recumbent subject, additional recruitment of vascular channels as a result of the mechanics of inspiration and collapse of channels during expiration cannot be determined by calculating peripheral resistance.

Pouleur et al. (1978) evaluated the effects of lung inflation in dogs and obtained results similar to ours. These investigators reported a difference in the zero Hz term of the pulmonary impedance (the quotient of mean pulmonary artery pressure and mean pulmonary flow) with lung inflation, but no change in pulmonary vascular resistance (calculated from pressure difference and flow) occurred. The change in the zero Hz term was interpreted by these investigators as a change in the state of the pulmonary circulation which they presumed affected right ventricular performance. Traditionally, when the systemic circuit has been evaluated, venous pressure has been ignored, because of its low magnitude in comparison to central aortic pressure. However, in the pulmonary circulation, the magnitude of the mean left atrial pressure may be significant when compared to mean pulmonary arterial pressure. Thus, when addressing the steady term (resistance), left atrial pressure (or an indirect assessment by pulmonary capillary wedge pressure) should be utilized (Bergel and Milnor, 1966). In this study, peripheral resistance was calculated in this manner, and the impedance spectrum was plotted using this term.

The values of pulmonary vascular resistance (79 ± 9 dynes sec/cm$^5$) found in our study are similar to the values reported by Milnor et al. (1969) for three patients with mild mitral valve disease and minimal hemodynamic embarrassment (97 dynes sec/cm$^5$). Mills et al. (1970) reported a value of 1920 dynes sec/cm$^2$ in one patient with ischemic heart disease. Assuming a cross-sectional area of 8 cm$^2$ of the main pulmonary artery, this would yield a pulmonary vascular resistance of 240 dynes sec/cm$^5$. However, in this latter study, left atrial pressure was not subtracted from pulmonary artery pressure, which may explain the difference.

The pulmonary artery characteristic impedance values found in our patients are also of the same order of magnitude as reported by Milnor et al. (1969) (average of 23 dynes sec/cm$^5$), in the same three patients referred to above. The effects of respiration on characteristic impedance found in our study are consistent with the findings of Bergel and Milnor (1965) and Pouleur et al. (1978), who also found no change in characteristic impedance with artificial lung inflation.

Shaver et al. (1974) and Curtis et al. (1975) suggested that changes in right ventricular ejection characteristics may be a result of changes in the impedance characteristics of the pulmonary vascular bed, as well as of changes in right ventricular filling.

![Figure 5](http://circres.ahajournals.org/)

**Figure 5.** Averaged data from 10 patients for the pulmonary vascular tree (thick lines) and five patients for the systemic vascular tree (thin lines). Top two panels: averaged pressure and flow spectra for pulmonary artery and aorta. Bottom two panels: averaged impedance modules and phase angles for the pulmonary and systemic arterial beds.
However, in the study, impedance was not measured and changes were inferred from an analysis of right and left heart systolic time intervals during the various phases of respiration. The results of our study do not support the implications that respiration-induced changes of the pulmonary arterial tree contribute to changes in the timing of pulmonary valve closure during quiet respiration in normal man.

Characteristic impedance ($Z_c$), expressed in terms of local compliance ($C' = \Delta A/\Delta P$) is equal to:

$$Z_c = \sqrt{\frac{\rho}{AC'}}$$

where $\rho$ is density of blood and $A$ is cross-sectional area. The observation that characteristic impedance does not change with respiration may be a result of the cancellation of effects due to increasing radius and decreasing compliance (Patel et al., 1962). This reasoning was also utilized in explaining the lack of change in aortic characteristic impedance with exercise-induced changes in ascending aortic pressure (Murgo et al., 1981a). In contrast, Elkins and Milnor (1971) observed an increase in pulmonary artery characteristic impedance with exercise in the dog. However, these investigators pointed out that loading effects of the external flow transducer may have affected the values of pulmonary characteristic impedance in their study. With serotonin infusion, Bergel and Milnor (1965) and Van den Bos et al. (1982) reported no change in characteristic impedance.

Bargainer (1967), using pulsewave velocity to calculate characteristic impedance, found an increase with serotonin infusion. However, the increase in cross-sectional area of the pulmonary artery would have minimized or negated any increase in characteristic impedance, had this parameter been expressed in terms of volume flow rather than linear velocity. However, Pace (1971) was able to induce an increase in pulmonary characteristic impedance with sympathetic stimulation. Accordingly, there is still uncertainty as to what happens to characteristic impedance of the pulmonary artery with increasing transmural pressure.

The advent of multisensor catheterization techniques has allowed us to evaluate dynamic pressure-flow relationships in both the systemic and pulmonary circulations. When compared to ascending aortic pressure and flow, the harmonic spectra of pulmonary pressure and flow contain less information in all harmonics (Fig. 5). As a result, more potential error in calculating input impedance of the pulmonary circulation is present due to a lower signal-to-noise ratio. Our results are in agreement with previous studies summarized by Milnor (1982). The pulmonary artery impedance spectrum is qualitatively similar to systemic input impedance, but the ratio of peripheral resistance and characteristic impedance is smaller than in the systemic bed. An other dissimilarity is seen in the low frequency range, where the phase angles of the pulmonary impedance spectra are less than in the systemic tree. These results are consistent with less reflection in the pulmonary system (Van den Bos et al., 1982).

The results of this study indicate that alterations in right ventricular output as a consequence of respiration are due primarily to changes in right ventricular function, rather than alterations of pulmonary input impedance.
O'Rourke MF (1968) Impact pressure, lateral pressure, and impedance to the proximal aorta and pulmonary artery. J Appl Physiol 25: 533-341
O'Rourke MF (1982b) Vascular impedance in studies of arterial and cardiac function. Physiol Rev 62: 570-623

INDEX TERMS: Input impedance • Pulmonary circulation • Systemic circulation • Respiration • Pressure-flow relationships
Input impedance of the pulmonary arterial system in normal man. Effects of respiration and comparison to systemic impedance.

J P Murgo and N Westerhof

*Circ Res*. 1984;54:666-673
doi: 10.1161/01.RES.54.6.666

*Circulation Research* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1984 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/54/6/666

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation Research* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Circulation Research* is online at:
http://circres.ahajournals.org/subscriptions/