Manipulation of Ascending Aortic Pressure and Flow Wave Reflections with the Valsalva Maneuver: Relationship to Input Impedance

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SUMMARY Dramatic changes in the shape of pulsatile ascending aortic pressure and flow wave forms occur during the Valsalva maneuver in man. To study these changes, aortic pressure and flow signals were recorded in eight patients using a multisensor catheter. Aortic input impedance was derived during the control, strain and postrelease phases of the Valsalva maneuver. During control, well-defined minima and maxima occurred in the spectral plots of impedance moduli. This pattern was accentuated during the postrelease phase. In contrast, input impedance during strain was almost equal to the characteristic impedance for all harmonics. These results imply that during the control and postrelease phases, strong reflections return to the ascending aorta, but during the strain phase, reflections are minimal, absent or more diffuse. From wave transmission theory, it also follows that pulsatile pressure and flow wave forms should be similar in shape in the absence of reflections and dissimilar in the presence of reflections. This was observed in all eight patients. By provoking changes in the arterial tree during the Valsalva maneuver, the magnitude and timing of wave reflections were significantly altered, resulting in marked changes in the shape of pulsatile aortic pressure and flow wave forms. This study demonstrates the importance of reflections in determining the shape of the arterial pulse.

THE SHAPES of ascending aortic pressure and flow wave forms are determined by cardiac function and the mechanical and geometric properties of the systemic arteries. The contribution of the arterial vascular tree to aortic pressure and flow wave shapes has been evaluated by calculation of aortic input impedance in the experimental animal and in man. These studies have demonstrated that arterial wave forms are influenced by the presence of wave reflections in the arterial system.

The Valsalva maneuver results in dramatic changes in the aortic pressure and flow wave shapes. The hemodynamic changes during the Valsalva maneuver have been studied by numerous investigators; however, studies of the effects of this intervention on pulsatile pressure and flow have been extremely limited, and an analysis of the changes in aortic input impedance in normal man has not been reported. Mills et al. analyzed input impedance of the ascending and descending aorta in a patient with ischemic heart disease during the Valsalva maneuver. Although the pattern of impedance spectra changed dramatically, these investigators did not focus on the relationship of the changes in impedance to the variations in arterial wave forms.

The purposes of this study were (1) to evaluate the sensitivity of impedance measurements in response to an intervention that markedly changes arterial hemodynamics; (2) to examine the relationships of changes in aortic pressure and flow wave forms to the changes in impedance; and (3) to investigate the contribution of wave reflections to the aortic pressure and flow wave shapes.

Methods

Patient Selection and Catheterization Techniques

Seven men and one woman underwent cardiac catheterization to evaluate chest pain syndromes. No cardiovascular disease was found by hemodynamic measurements during rest and exercise, left ventricular cineangiography or coronary arteriography. All patients were studied in a basal state and were either unsedated or very lightly sedated (diazepam, 10 mg orally 1 hour before the procedure). Right- and left-heart catheterizations were performed using special multisensor catheters during both rest and the performance of at least two complete Valsalva maneuvers as described below.

Steady-state conditions during rest were determined by a stable heart rate and stable sequential pulmonary artery hemoglobin oxygen saturation measurements. Oxygen consumption was then determined by collecting expired air with a Douglas bag for 5 minutes and measuring oxygen content using the Scholander technique. During the air collection, arterial venous oxygen content difference was derived from blood specimens collected from the aorta and pulmonary artery. Cardiac output was then calculated using the
direct Fick method. Duplicate determinations were made to evaluate reproducibility.

Each patient then performed at least two complete Valsalva maneuvers. A deeper-than-normal inspiration was followed by a forceful exhalation into a mouthpiece attached by plastic tubing to a hand-held mercury manometer. Intraoral pressure was monitored and each patient maintained a steady level between 20–40 mm Hg for a minimum of 20 seconds. Nose clips prevented air leaks and all patients had intact tympanic membranes. Three phases of the Valsalva maneuver were analyzed to calculate impedance in each subject (fig. 1): (1) the control period before the initiation of strain; (2) the mid- to late strain period after the transient response and the appearance of a reflex tachycardia; and (3) the post-release period after the transient pressure overshoot but while systemic hypertension and relative bradycardia were still present. All hemodynamic measurements, including the pressure and flow velocity signals used to derive impedance as described below, were recorded during these periods.

Biplane left ventricular cineangiography and selective coronary arteriography were performed after all hemodynamic data were collected and the patients returned to a control state. If the ascending aortic root was not adequately visualized during the ventriculographic study, a selective aortic root angiogram was performed to measure the mean systolic radius of the ascending aorta. The aortic radius was determined to evaluate the proximal geometry of the arterial tree and to establish an aortic flow velocity calibration as described below. 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. All patients gave informed consent.

Custom-designed left- and right-heart multisensor catheters were used. A left-heart catheter contained two solid-state pressure sensors (Millar Mikrotip, Millar Instruments, Inc.) and an electromagnetic flow velocity probe (Carolina Medical Electronics, 1973-1975; Millar Instruments, Inc., 1975-1978). Both pressure sensors were mounted laterally with one sensor located at the tip of the catheter and the second located 5 cm away. The catheter was designed so that the sensing electrodes of the velocity probe and the aortic pressure sensor were mounted at the same site. After a brachial arteriotomy, this catheter was retrogradely passed across the aortic valve so that the tip sensor was within the left ventricular cavity and the second sensor and associated electromagnetic flow velocity probe were 3–5 cm above the aortic valve. This arrangement maximized the stability of the transducers in the ascending aorta while simultaneously providing left ventricular pressure. Details of the technical characteristics of these sensors, including frequency response, drift characteristics and calibration techniques have been described.

The following variables were also recorded: ECG, respiratory signal derived from an abdominal pneumatic belt, pulmonary capillary pressure from a fluid-filled Cournand catheter via a Statham P23Db strain gauge, and high-fidelity pressures from the right atrium, right ventricle and main pulmonary artery, using a single multisensor catheter.

![Figure 1](http://circ.ahajournals.org/)

**FIGURE 1.** Example of a slow-speed (2.5 mm/sec) multisensor catheter recording during the Valsalva maneuver. ECG, aortic (AO) flow velocity, and aortic and left ventricular (LV) pressures are recorded.
Signal Processing and Computational Techniques

The flow velocity probes were used in conjunction with either a Carolina Medical Electronics square-wave flowmeter (Model 501, Carolina Medical Electronics) or a Biotronex sinewave flow meter (Model BL-613, Biotronex Laboratory, Inc). The frequency response of the flow velocity measurements was determined primarily by the electronics with low-pass characteristics down 3 db at 100 Hz. Pressure and flow signals were low-pass-filtered with corner frequencies at 100 Hz. Data were recorded on a Honeywell 5600 analog tape recorder and an 1858 fiberoptic strip-chart recorder. Digital processing was obtained using a 13-bit analog-to-digital converter at 200 samples/sec coupled to a Honeywell 316 minicomputer. The technical details of the analog and digital processing are described elsewhere.

The spatial flow velocity profile in the proximal ascending aorta was assumed to be blunt, and cyclic changes in the diameter of the ascending aorta were considered to be negligible, so that the aortic flow velocity signal was used to represent instantaneous volumetric flow. In vivo calibration of this signal during the control state was accomplished by integrating the area under the curves and setting those areas equal to the stroke volume as determined by the direct Fick method, described above. This technique established a volumetric flow scale during the control period so that beat-to-beat variations in aortic flow could be measured and used to calculate impedance.

During strain, the radius of the ascending aorta is expected to change significantly so that the changes recorded by the electromagnetic aortic flow velocity probe during this phase (fig. 1) represent changes in velocity and do not quantitate changes in instantaneous volumetric flow. We chose to calculate impedance using absolute volumetric flow, and a volumetric flow scale during the strain period was established using the following approach. An aortic flow velocity scale during the control period was established by dividing the Fick-calibrated volumetric flow scale by the cross-sectional area of the ascending aorta. The latter was calculated using the angiographically determined aortic radius (with the patient in the control state). This velocity scale (fig. 1) was used to determine aortic flow velocity during the strain period. To calculate instantaneous volumetric flow during this phase, certain assumptions of the changes induced in the aortic cross-sectional area were necessary. Greenfield and co-workers demonstrated a 17% decrease in aortic cross-sectional area during this phase of the Valsalva maneuver in normal man. This approximation was used for all eight subjects in this study after being supported by mid strain aortic angiograms in one of our subjects. An estimate of absolute volumetric flow could then be established by converting the flow velocity scale back to a volumetric scale using these decreased aortic cross-sectional areas.

The pressure-diameter changes of the ascending aorta are nonlinear, so marked changes in diameter are expected to occur during decreased pressures, but smaller changes will occur during increased pressures. Accordingly, the same volumetric scale used during the control period was applied to the postrelease state to estimate volumetric changes during that period.

Calculation of Input Impedance

Ascending aortic input impedance was calculated during the control, strain and postrelease states of the Valsalva maneuver. Fourier analysis and impedance calculations were not attempted during transient responses where heart rate and other physiologic parameters were rapidly changing. Signals were chosen from the "quasi-steady states" of the maneuver during mid- to late stage after reflex tachycardia and maximum stabilization of pressure and flow had appeared, and during the postrelease period after the early hypertensive overshoot while hypertension and reflex bradycardia were still present. Using the electrocardiographic RR interval as the fundamental period, the Fourier series of aortic pressure and flow were determined for the first 10 harmonics. Impedance should be calculated from a pressure difference across the system (aortic pressure minus right atrial or venous pressure) and aortic flow.

Right atrial pressure for these patients was low compared with aortic pressure during the control and postrelease states, so the aortic pressure alone was used for calculation of impedance during these periods. However, during the strain phase, the marked effect of increased intrathoracic pressures on the systemic venous and right atrial pressures was accounted for in the calculations of impedance by subtracting mean right atrial pressure from aortic pressure. Amplitude and phase spectra of impedance were calculated by dividing amplitudes and subtracting the phase angles of the aortic pressure and flow components. A spectral averaging algorithm was used to remove the effects of noise in the physiologic signals, especially flow velocity signal, which inherently has a poorer signal-to-noise ratio than pressure. Characteristic impedance ($Z_c$) was defined as the average of all impedance moduli above 2 Hz.

To characterize the patterns of the impedance moduli plots for the three phases of the Valsalva maneuver, each subject's amplitude spectra were normalized to that subject's characteristic impedance. The normalized moduli and the phases were then averaged by harmonic for the entire group during each of the three states.

Wave Form Classification

Although Fourier analysis and impedance calculations were not attempted during transient responses, it is interesting to examine the changes in aortic wave forms during the transient response that occurs with the initiation of strain (fig. 2). Within a few beats, the aortic pressure wave forms change dramatically. To relate changes induced by the
Valsalva maneuver in the patterns of the impedance spectral plots to changes induced in the shape of the ascending aortic wave forms, pressure wave forms were classified into three groups, as previously described by this laboratory. In the prestrain period and the first beat after the initiation of strain, the ascending aortic pressure wave form revealed a peak systolic pressure in late systole after a mid-systolic inflection (type A beat). By the second beat during strain, this late systolic peak is reduced to a level equal to early systole (type B beat). In the third and subsequent beats, peak systolic pressure occurred in early systole, and the late systolic portion of the wave form no longer had the characteristics seen during the prestrain (control) phase (type C beat).

**Results**

General patient characteristics and basic hemodynamic data appear in table 1. The patients were classified according to the shape of the ascending pressure wave form during the control period and are a subset of a larger group of patients previously described. Average age was 36 years (range 19–54 years). Significant changes occurred in heart rate during the strain and postrelease phases of the Valsalva maneuver compared with the control state. Cardiac output was significantly depressed during the strain phase but was essentially the same as the control period during the postrelease phase. Due to the residual bradycardia in the postrelease phase, stroke volume during that state was increased compared with the control period. Systolic ascending aortic pressures were significantly different in all three phases of the Valsalva maneuver. During the strain phase, diastolic pressures in all cardiac chambers and in the pulmonary capillary wedge position equilibrated, reflecting the marked increase in intrathoracic pressures. As an indication of the intrathoracic pressure during this phase, the pulmonary capillary wedge pressure is shown in parentheses next to the mean aortic pressure in table 1. Equilibration of pressures occurred, so this column also gives the strain phase value of mean right atrial pressure.

Figure 3 illustrates the steady-state phases of the Valsalva maneuver that were subjected to analysis to calculate input impedance in one patient. Patient A5 transformed the ascending aortic pressure wave form from a type A beat during control to a type C beat during strain, and back to an “accentuated” type A beat in the postrelease phase (fig. 3). There is a marked difference between the pattern seen in the control and postrelease phases compared with the pattern
TABLE 1. Hemodynamic Data

<table>
<thead>
<tr>
<th>Patient data</th>
<th>Heart rate (beats/min)</th>
<th>Flow data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td>Cardiac output (l/min)</td>
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<tr>
<td>Sex</td>
<td>BSA (m²)</td>
<td>C</td>
</tr>
<tr>
<td>A1 54</td>
<td>1.86</td>
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<tr>
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<td>C2 30</td>
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</tr>
<tr>
<td>C3 29</td>
<td>1.87</td>
<td>85</td>
</tr>
</tbody>
</table>

Mean 36 1.95 1.26 80 105 65 7.1 3.3 7.1

* SEM = 4 0.03 0.04 0.04 6 4 4 0.3 0.4

*p < 0.05, analysis of variance with Student-Newman-Keuls multiple comparison test, strain and postrelease states compared with control.

†Valsalva release signals technically inadequate.

Mean intrathoracic pressure given in parentheses.
Abbreviations: BSA = body surface area; Ao R = aortic radius; C = control; S = strain; PR = postrelease.

FIGURE 3. (top) Pressure and flow waves from the "quasi-steady state" portions of control, strain, and postrelease phases of the Valsalva maneuver. FV = flow velocity; AO = aortic pressure; LV = left ventricular pressure. The LV pressure was discontinued in the latter portion of each of these recordings in order to more clearly display the primary aortic wave forms used to calculate impedance. (bottom) The corresponding impedance plots for each of the three states are illustrated. The 0-Hz term during control is 1059 ± 127 dyn-sec-cm⁻⁵; during strain, 1659 ± 174 dyn-sec-cm⁻⁵; and during the postrelease phase, 1393 ± 160 dyn-sec-cm⁻⁵.
Table 1. (Continued)

<table>
<thead>
<tr>
<th>Flow data</th>
<th>Aortic systolic</th>
<th>Pressure data</th>
<th>Aortic mean</th>
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<td>C  S  PR</td>
<td>C  S  PR</td>
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<td>=2</td>
<td>4*</td>
<td>5*</td>
<td>=4*</td>
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</table>

during the strain phase. The pattern of the amplitude spectra during the control and postrelease states demonstrates very distinct minima between 3-4 Hz, followed by well-defined maxima between 6-8 Hz. In contrast, an extremely flat amplitude spectrum is observed during the strain period. More negative phase angles are also present in the lower harmonics during the control and postrelease states. Note that the impedance spectra with well-defined minima and maxima during control and postrelease are associated with type A beats, whereas the flatter spectrum during the strain period is associated with the type C beat.

Figure 4 is an illustration of the average impedance spectra for the entire group after normalization to characteristic impedance during each of the three states of the Valsalva maneuver. Although averaging by harmonics may cause smoothing of spectra if heart rates and patient size vary considerably, such an average serves to compact the data for groups of patients and was used to examine the patterns of the spectral plots for the three states. When so organized, the differences between the three phases of the Valsalva maneuver were found to be similar to the individual example shown in figure 3. Six of the eight patients demonstrated either type A or type B beats during the control period (table 1), so that the average impedance spectra for the control state demonstrate the characteristics typically found with the type A beat, i.e., well-defined minima and maxima and relatively large oscillations about the characteristic impedance. Although there were some differences between patients in the shape of the ascending aortic pressure wave form during the control state, all eight patients had type C beats during the strain period and type A beats during the postrelease state. An extremely flat average spectrum is again shown during
strain and a return to the oscillatory behavior present during control is illustrated in the postrelease state.

The major features of input impedance, i.e., the peripheral resistance and the characteristic impedance, are listed in Table 2. All but two patients increased their peripheral resistance during the strain phase of the maneuver, but the mean value for the group was not significantly different from the control state. In that portion of the postrelease phase that was chosen for analysis (20–30 seconds after the transient response), no significant difference from the control state was found in peripheral resistance. No consistent change in characteristic impedance was found during the various phases; the average values in the strain and postrelease states were not significantly changed from the control period.

Discussion

This study demonstrates that the Valsalva maneuver can induce dramatic changes in the patterns of aortic input impedance, which are associated with marked changes in the shape of the ascending aortic pressure wave form. The oscillatory behavior of the impedance spectra for type A beats suggests that there is considerable reflection in the arterial system, as we and others have emphasized. The flatter impedance spectra associated with type C beats imply smaller or more diffuse reflections. Another indication of the amount of reflection in a system with a given reflection site is the magnitude of the first harmonic of the normalized impedance moduli. The amplitude of the first harmonic of the normalized impedance moduli is much higher than Zc during the control and postrelease states, indicating large reflections (fig. 4). During the strain period, the value of the first harmonic has already approximated Zc, indicating less influence of reflected waves. Examination of the phase spectra also yields information related to the influence of reflections. In a system without reflections, the phase angle would be close to zero. During the strain phase, the phase angle is much less negative for the first few harmonics, and approaches zero at lower frequencies than the phase spectra during the control and postrelease states.

During control, the majority of pressure wave forms were type A beats, and during the postrelease phase all patients demonstrated type A beats. These wave forms possess a secondary midsystolic rise in pressure and are always associated with oscillatory impedance spectra. These results imply that the secondary rise in the aortic pressure wave is primarily the result of a less well-matched arterial system, i.e., a system with considerable reflection. The type C beats, always seen during strain, are present when the system demonstrates little reflection and show small or no secondary rise in pressure. Such changes in the degree of reflection are easily induced by the Valsalva maneuver (fig. 5). In the course of only six beats from the end of the strain period to the hypertensive bradycardia stage, the aortic pressure pulse changes from a beat without a secondary rise in late systolic pressure (type C beat) to a beat with a very pronounced late systolic wave (type A beat). As stroke volume is re-established and mean aortic distending pressure increases, this late systolic wave gradually becomes larger and appears progressively earlier in systole. The earlier appearance is demonstrated by the progressively shorter time intervals from the onset of the aortic pressure pulse to the later systolic wave (fig. 5). Less clearly shown, but suggested in the flow velocity signals, is a negative inflection point that corresponds in time to the late systolic wave in the pressure pulse. Other investigators have postulated that such events are most likely caused by reflected pressure and flow waves returning to the ascending aorta from the peripheral systemic circulation.

To pursue these observations further, the measured pressure and flow waves were dissected into their forward and reflected (or backward) components using mathematical techniques described in detail elsewhere. The pertinent equations are summarized as follows:

\[ P_m = P_f + P_b \]  
\[ F_m = F_f + F_b \]  
\[ P_f = Z_c \cdot F_f \]  
\[ P_b = -Z_c \cdot F_b \]

where P and F are pressure and flow and m = measured wave, f = forward wave, b = backward wave and Zc = characteristic impedance. Thus, the measured pressure wave (Pm) as sensed by a transducer is equal to the sum of a forward (Pf) and a backward (Pb) travelling wave. Similarly, the measured flow wave (Fm) is equal to the sum of the forward and backward travelling flow waves. The forward pressure and flow waves are related by the characteristic impedance of the aorta as given by equation 3. If Zc is a real number, this relationship predicts that the forward pressure and flow waves are

<table>
<thead>
<tr>
<th>Table 2. Impedance Data</th>
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<td>Patient</td>
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<tr>
<td>Total</td>
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*All impedance measurements in dyn-sec-cm⁻⁴.
†Valsalva release signals technically inadequate.

No significant differences at 0.05 level between C, S and PR in both Rp and Zn with Student-Newman-Keuls multiple comparison test.

Abbreviations: Rp = peripheral resistance; Zn = characteristic impedance; C = control; S = strain; PR = postrelease.
identical in shape. The same relationship holds for the backward waves, but flow is inverse with respect to pressure, as indicated by the minus sign (equation 4). Because \( P_m \) and \( F_m \) are measured quantities and the characteristic impedance \( Z_c \) may be derived from the calculated impedance spectra, equations 1-4 may be solved to yield the forward and backward flow and pressure waves:

\[
P_f = Z_c F_f = (P_m + Z_c F_m)/2 \tag{5}
\]

\[
P_b = -Z_c F_b = (P_m - Z_c F_m)/2 \tag{6}
\]

Using these equations and a digital computer, measured pressure and flow waves can be dissected into their forward and backward components in a patient with a type A beat (fig. 6). At a given location in the ascending aorta, a forward travelling pressure wave first appears but, while systole is still in progress, a backward travelling (reflected) pressure wave arrives at this location. This reflected wave is added to the forward wave and yields the archetypical ascending aortic pressure wave form with a late systolic wave. Similarly, the forward flow wave (which has the same shape as the forward pressure wave) is added to

\[
P_m = P_f + P_b
\]

\[
F_m = F_f + F_b
\]
the backward flow wave (also identical in shape to the backward pressure wave, but inverse in direction). The sum of these two waves yields the archetypical flow wave, which is markedly different from the measured pressure wave.

This same analysis is applied to the control, strain and postrelease phases of the Valsalva maneuver (fig. 7). During the strain phase, almost no reflections exist. This is in agreement with the flat impedance spectra discussed above. The absence of reflections yields a measured pressure wave \( P_m \), which is almost identical to the forward pressure wave \( P_f \). Likewise, the absence of flow reflections yields a measured flow wave \( F_m \), which is very similar to the forward flow wave \( F_f \). The forward pressure and flow waves are identical in shape, so the measured pressure and flow waves are similar in shape. In contrast, significant reflections exist during the control and postrelease states and the measured pressure and flow waves are considerably different. The backward waves during the postrelease phase are significantly larger than during control, so that the differences in pressure and flow waves shapes are accentuated.

These observations are emphasized in figure 8, which is an illustration of aortic flow and pressure in another patient during the strain and postrelease states. Marked similarities in aortic pressure and flow are present during strain and the wave forms are essentially indistinguishable from one another. During the poststrain phase, when strong reflections are present, the aortic flow and pressure wave forms are markedly dissimilar.

Several mechanisms could be proposed to explain the changes in both the impedance spectra and the ascending aortic wave forms during the strain phase of the Valsalva maneuver. First, a change in the reflection coefficient \( r \) (i.e., a change in impedance matching) at the major effective reflection site would be the most obvious possible cause. We have shown that the major effective reflection site in man appears to be in the region of the terminal abdominal aorta. We could not measure the factors contributing to the reflection coefficient at that location. It would therefore be of interest to compute the reflection coefficient by studying the characteristic impedance of the abdominal aorta and the input impedance of both iliac arteries during the Valsalva maneuver. A second mechanism could be a change in the location of the effective reflection site. The time of arrival of the reflected wave depends on the distance of the effective reflection site and the pulse wave velocity. The latter parameter was not measured during the Valsalva maneuver, but investigators have reported that it increases with increasing pressure. Thus, the observation that the time of arrival decreases during the postrelease state (130 ± 11 msec postrelease vs 141 ± 11 msec control, \( p < 0.05 \)) may be explained from the increase in pulse wave velocity rather than a change in reflection site.

A third possible explanation for the decreased reflections seen during strain is a change in the factors necessary for the transmission of the pulse wave to and from the reflection site. One major factor in a viscoelastic arterial system is damping. If both the

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**Figure 7.** Forward, backward and measured pressure and flow wave forms during each of the three phases of the Valsalva maneuver in one patient.
forward and backward waves are significantly attenuated in the process of travelling to and from the terminal abdominal aorta, both the reflection coefficient $R_f$ and the effective length of the system would remain unchanged, but reflections returning to the ascending aorta would be markedly reduced. Further studies to investigate the changes in damping induced by the Valsalva maneuver are necessary to confirm such a hypothesis.

To our knowledge, only one other study has addressed the changes induced in arterial wave forms by the Valsalva maneuver. Micromanometer techniques were not used in that study and the authors chose to focus on the changes induced in the aortic flow velocity wave forms in the innominate artery of two patients with ischemic heart disease and in the ascending aorta of one patient with mitral valve disease. However, changes in the pressure wave forms (shown in their illustrations of the two patients with ischemic heart disease) were almost identical to those described for the ascending aorta in our study. In the only patient in whom ascending aortic wave forms were described, no significant reflection effects were noted, and the authors concluded that reflection effects were not important in the ascending aorta. However, this patient had mitral stenosis and insufficiency, peak aortic systolic pressures less than 100 mm Hg, and pressure wave forms during both control and the postrelease phase similar to the type C beats described in our study (i.e., a system with little reflection). This patient with heart disease did not provide sufficient evidence to conclude that reflections do not play a significant role in determining the shape of ascending aortic wave forms. In none of these three patients was input impedance calculated. However, in a fourth patient subjected to the Valsalva maneuver, these investigators did calculate input impedance of the ascending and descending aorta. The results of this patient with ischemic heart disease were almost identical to those presented in this study, i.e., large oscillations in the impedance spectra were found during control and postrelease, while a flat spectrum was observed during strain. However, changes induced in the ascending aortic wave forms in that patient were not illustrated or discussed, although the changes induced in the impedance spectra suggested marked changes in reflection phenomena.

This study has demonstrated that the Valsalva maneuver induces significant changes in input impedance in man, with little or no change in characteristic impedance. The changes in the spectral patterns of aortic impedance are consistent with considerable reflection effects during the control and postrelease phases and weak or absent reflections during strain. The changes induced in ascending aortic pressure wave forms are consistent with the impedance measurements, because late systolic waves are present in most patients during control, disappear in all patients during strain and are accentuated in all during the postrelease state. The fact that pressure and flow wave shapes that are ordinarily dissimilar become similar with Valsalva strain was explained by analyzing the changes induced in the amount of reflection during this maneuver.

Acknowledgment

The authors gratefully acknowledge the valuable assistance of Thomas Dunne and the technicians of the Cardiac Catheterization Laboratory at Brooke Army Medical Center, SP5 Thomas R. Deojay and James W. Bagwell for their assistance in data processing, Dr. Barry R. Alter and the physicians of the Cardiology Service for their cooperation, and Bettye Jo Hairston for her outstanding assistance in typing this manuscript. The photography and graphics were provided by Earl Ferris, SP5 David Lovelace, SP5 Georgette Toshok, and Douglas Meyer.

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Manipulation of ascending aortic pressure and flow wave reflections with the Valsalva maneuver: relationship to input impedance.
J P Murgo, N Westerhof, J P Giolma and S A Altobelli

doi: 10.1161/01.CIR.63.1.122

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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