Pressure-Flow Studies in Man During the Valsalva Maneuver with Observations on the Mechanical Properties of the Ascending Aorta

By Joseph C. Greenfield, Jr., M.D., Ronnie L. Cox, M.D., Rafael R. Hernandez, M.D., Corinna Thomas, B.S., and Fred W. Schoonmaker, M.D.

SUMMARY

The pressure-gradient technique was used to obtain continuous measurements of both blood pressure and flow in the ascending aorta of eight normal subjects who were performing a standardized Valsalva maneuver. From these recordings the beat-to-beat changes in stroke volume, peak blood flow, peripheral vascular resistance, duration of ejection, and an index of total systolic duration were calculated. Stroke volume and peak blood flow were not changed with the onset of straining (phase I), but were decreased to approximately 50% of control values immediately prior to release (phase II). During the overshoot period (phase IV) stroke volume and peak flow were increased above control levels. Both the duration of ejection and the duration of systole were shortened during straining (phase II). The concomitant changes in blood pressure in these subjects were similar to previously reported pressure recordings. An estimate of the pressure-radius relationships in the ascending aorta of these patients was obtained angiographically. The mean cross-sectional area of the ascending aorta changed by 17% during the Valsalva maneuver.

ADDITIONAL INDEXING WORDS:
Indicator-dilution technique  Aortic compliance  Peripheral vascular resistance
Ejection period  Systolic period

The effect of the Valsalva maneuver on the cardiovascular system has been of interest to circulatory physiologists for many years. In order to characterize these effects, one should know beat-to-beat values for both aortic pressure and flow. Continuous recordings of blood pressure during the Valsalva maneuver are well documented; however, measurements of instantaneous flow have not previously been available because of the technical problems encountered in quantitating phasic aortic blood flow in the intact human subject. Consequently, alterations in blood flow during the Valsalva maneuver have either been inferred from pressure data or estimated with indicator-dilution techniques. Since these methods require several seconds for completion, they cannot be used to estimate beat-to-beat changes in flow during an unsteady state. At present the pressure-gradient technique provides the only practical method for measuring instantaneous aortic blood flow in the intact human subject. This report presents blood pressure and flow data recorded continuously in the ascending aorta.

From the Division of Cardiology, Department of Medicine, Duke University Medical Center, and the Durham Veterans Administration Hospital, Durham, North Carolina.

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of eight normal subjects during a standard Valsalva maneuver. In addition, the pressure-radius relationships for the ascending aorta of these subjects were estimated by angiographic techniques.

Method

Eight adult male patients, ranging in age from 36 to 47 years, were studied. All of these subjects had minimal hypertension at the time of admission to the hospital but demonstrated no other clinical evidence of cardiovascular disease. The data described in this report were obtained during the course of arterial catheterization for diagnostic renal arteriography. The nature of these studies was fully discussed with the patients. They had not received antihypertensive therapy for several weeks prior to the study, and no precatheterization sedation was given.

The phasic blood flow and pressure were measured simultaneously in the ascending aorta using the pressure-gradient technique. This method depends on the laws of fluid motion which, with suitable restrictions, can be simplified to the following approximate relationship between the axial pressure gradient, $-\partial p/\partial z$, and the instantaneous blood flow, $q$:

$$ -\frac{\partial p}{\partial z} = \frac{(1.1) \rho}{g\pi r^2} \frac{\partial q}{\partial t} + \frac{(1.6) 8\mu}{8\pi r^4} q $$

where $p$ is the lateral pressure, $z$ is the axial coordinate along the vessel, $\rho$ is the blood density, $\mu$ is the blood viscosity, $g$ is the gravitational constant, $r$ is the vessel radius, and $t$ is time. The factors 1.1 and 1.6 are experimentally derived correction values that account for, among other things, the nonuniformity of the flow profile. A complete discussion of the validity of these equations appears elsewhere. Note that the mean sectional blood velocity, $u$, is related to blood flow, $q$, by the equation

$$ q = u \pi r^2 $$

where $\pi r^2$ is the vessel lumen area. Thus equation 1 can be used to compute the blood velocity, $u$. Since in practice the pressure gradient, $-\partial p/\partial z$, cannot be obtained, a pressure difference, $\Delta p$, is measured over a finite distance, $\Delta z$, and $\Delta p/\Delta z$ is substituted for $-\partial p/\partial z$. Equation 1 can be solved continuously for the flow, $q$, with an analog computer by using $\Delta p/\Delta z$ as the "forcing function." The pressure-gradient technique has been evaluated in both a pulsatile flow generator where the "true" flow was known and in the descending thoracic aorta of dogs in which the "true" flow was estimated with an electromagnetic flowmeter. The correlation between the flows obtained with the pressure-gradient technique and the monitored "true" flows in both of these experimental situations was good. These data indicate that the pressure-gradient technique can be used to measure the major features of both phasic blood pressure and flow in the ascending aorta. The instrumental techniques, manometric accuracy requirements, and calibration procedures used to obtain valid pulsatile flow and pressure recordings in man have been described previously.

A double-lumen catheter with lateral pressure taps, 4 cm apart, was used to obtain the pressure gradient. The catheter was inserted percutaneously into the femoral artery and advanced under fluoroscopic control until the tip was 2 to 3 cm above the aortic valve. A second cardiac catheter was introduced percutaneously into the cephalic vein and passed into the right atrium. Simultaneous right atrial pressure, aortic pressure, and aortic flow were recorded during a 5-minute control period, during a Valsalva maneuver, and for 2 minutes following release. During the control period cardiac output was obtained using an indicator-dilution technique: Indocyanine-green dye was injected into the right atrium and sampled from the brachial artery. In order to standardize the Valsalva maneuver, the patients were required to maintain an intra-oral pressure of 40 mm Hg for approximately 25 seconds. The electrocardiogram was monitored throughout the procedure. All recordings of data were carried out on both an Electronics for Medicine optical recorder and a Sanborn Model 2007 F.M. tape recorder.

As can be seen, the solution of equation 1 for the flow, $q$, requires a knowledge of the aortic radius. In studies of aortic flow in which the blood pressure does not vary widely, a single angiographic determination of aortic radius is adequate. However, during the Valsalva maneuver the distending pressure of the aorta (aortic pressure to intrathoracic pressure) changes considerably, and it is necessary to obtain an estimate of the aortic radius as a function of this distending pressure. Prior to the flow study an Elema-Schonander angiographic unit was used to take three films a second after the injection of 35 cm³ of Renovist (sodium and methylglucamine diatrizoate) into the ascending aorta. The injection was made using a no. 8 Lehman catheter. In order to vary the aortic pressure as much as possible during filming, the contrast material was injected immediately after the release phase of a Valsalva maneuver. Prior to the injection simultaneous pressures in the ascending aorta and the brachial artery were recorded. After correction for catheter transmission delay, the phase relationship between these two pressures was established and the brachial artery pressure was used.
to estimate pressure in the ascending aorta during filming. The exposure times of the films were recorded simultaneously on the optical recorder with a photocell. The technique used to correct for x-ray distortion and to obtain the true vessel radius from the angiograms has been described in detail by Luchsinger and associates. From three to 10 films from each patient were considered to be technically acceptable. These data were used to construct a graph of the aortic radius as a function of distending pressure for each subject. In calculating the aortic distending pressure (aortic-intrathoracic pressure) during the Valsalva maneuver, right atrial pressure was substituted for the intrathoracic pressure. From this graph the area of the vessel lumen for any systolic pressure could be calculated and used in the solution of equation 1. In addition \( \Delta P/\Delta t \) for each patient was calculated from these data.

In evaluating the data, zero flow was assumed to be present at the end of diastole. Peak blood flow was measured directly from the flow tracing as the maximum deflection, and the stroke volume was obtained by planimetry. Flow per second was calculated from the stroke volume and heart rate. Systolic and diastolic pressures were measured directly from the pressure recording, and the mean arterial pressure was computed by planimetry. The duration of ventricular ejection was measured from the flow tracing as the period of forward flow. The period from the onset of the Q wave of the electrocardiogram to the end of forward flow was used as an index of the duration of total systole. Peripheral vascular resistance, PVR, in dynes sec cm\(^{-5} \) was computed from the equation:

\[
PVR = \frac{MAP - RAP}{Flow/sec} \times 1332
\]  

(3)

where MAP is the mean arterial pressure (mm Hg), and RAP is the right atrial pressure (mm Hg). Data from five consecutive heart beats were averaged during the control period. Cardiac output (cm\(^3\)/min) was computed as the product of stroke volume and heart rate. Although continuous measurements of phasic pressure and flow were obtained throughout the Valsalva maneuver, specific times were chosen in order to compare the results from patient to patient. The following periods were selected: the third heart beat after the beginning of the Valsalva maneuver, the midpoint of the straining phase, the heart beat just before release, and the first, third, sixth, and ninth heart beats following Valsalva release. The heart beat having the greatest systolic pressure during the overshoot phase was also used. The previously described data were calculated for each of these periods. Standard statistical techniques were employed to evaluate the data.\(^{15} \)

**Table 1**

<table>
<thead>
<tr>
<th>Heart rate (beats min(^{-1} ))</th>
<th>Duration of ejection (sec)</th>
<th>Duration of systole (sec)</th>
<th>Area of systolic pressure (cm(^2))</th>
<th>Peak flow (cc sec(^{-1} ))</th>
<th>Peak flow (cm(^3)/sec)</th>
<th>Flow/sec</th>
<th>Systolic pressure (mm Hg)</th>
<th>Diastolic pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>72 ± 12</td>
<td>62 ± 10</td>
<td>82 ± 10</td>
<td>104 ± 20</td>
<td>353 ± 54</td>
<td>45 ± 3</td>
<td>7.6 ± 1.1</td>
<td>88 ± 19</td>
<td>66 ± 1.2</td>
</tr>
<tr>
<td>97 ± 12</td>
<td>97 ± 12</td>
<td>114 ± 15</td>
<td>120 ± 20</td>
<td>480 ± 78</td>
<td>48 ± 10</td>
<td>7.9 ± 1.2</td>
<td>90 ± 20</td>
<td>64 ± 1.1</td>
</tr>
</tbody>
</table>

*Average data along with the standard deviation for the eight patients are recorded in columns 2 through 11. The data in row 1 are the average of five consecutive heart beats obtained from each patient during the control period. Data in the other four rows represent a single heart beat for each patient obtained in the periods noted in column 1.*
Results

The mean cardiac output for the group computed by pressure-gradient technique was 4,476 cm³/min, sd ± 611. At the same time cardiac output obtained with the indicator-dilution technique had a value of 4,815 cm³/min, sd ± 974. The mean of the difference between these two methods was 425 cm³, sd ± 720. The average stroke volume was 50 cm³, sd ± 7 and flow per second was 72 cm³/sec during the control period. Simultaneously recorded aortic pressure was 132/88 mm Hg for the group with a mean of 104 mm Hg, sd ± 20. The cross-sectional area of the ascending aorta was 7.6 cm², sd ± 1.1, and the heart rate was 87 beats/min, sd ± 13.

Pertinent information obtained during the Valsalva maneuver is tabulated in table 1 and illustrated in figure 1. The crossed bars in figure 1 represent the standard deviation. A typical recording from one of the subjects obtained during the various phases of the Valsalva maneuver is illustrated in figure 2.

As can be seen in panel A of figure 2, immediately following onset of straining, the arterial pressure increased by approximately the same amount as did the right atrial pressure. Neither stroke volume nor aortic cross-sectional area changed during this time (see fig. 1). Immediately thereafter both pressure and stroke volume fell rather rapidly. By the time the midpoint of the straining phase was reached, the average stroke volume had decreased to 28 cm³, sd ± 4 (42 cm³/sec) and the mean arterial pressure to 88 mm Hg, sd ± 19 (see table 1). Calculated PVR for the group increased by approximately 25%, and aortic area decreased to 6.6 cm², sd ± 1.2. Both the duration of total systole and the duration of ejection decreased. At the midpoint of the straining phase mean right atrial pressure for the group was 36 cm H₂O.

![Figure 1](image-url)

Mean data obtained from the eight patients including the standard deviation (crossed bars) are given for the following parameters: S.V., stroke volume; P.V.R., peripheral vascular resistance; M.A.P., mean arterial pressure; aortic area; and peak flow. The data were normalized prior to averaging by dividing the particular parameter by the control data, for example, stroke volume by control stroke volume. The periods in which the data were obtained are listed at the top of the figure.
Figure 2

Pressure and flow recordings obtained during a control period (panel A), at the midpoint of the straining phase (panel B), during release (panel C), and during the overshoot phase (panel D). The electrocardiogram (ECG), pressure gradient (Δp/Δz), ascending aortic blood velocity, aortic pressure, and right atrial pressure appear from the top down in each panel. Note that the calibration for the phasic flow tracing is given in terms of blood velocity. This is necessary since each heart beat must be independently calibrated due to changes in the area of the aortic lumen (see text).

The stroke volume continued to fall slowly; immediately before release the stroke volume had decreased to an average of 22 cm³, SD ± 6 (35 cm³/sec). Mean arterial pressure rose slightly during the latter part of the straining phase and just prior to release was 94 mm Hg, SD ± 20. The PVR continued to rise during the straining phase and before release it was approximately 50% greater than the control values. The average duration of the Valsalva maneuver was 26 (range, 18 to 35) sec. Immediately following release the stroke volume remained essentially unchanged at 21 cm³ (34 cm³/sec); however, mean arterial pressure fell to 77 mm Hg. PVR also...
did not change from the level present just before release. Following release both stroke volume and arterial pressure rose rapidly, reaching control values six to nine heart beats after release. The PVR fell to control levels by the third heart beat after release, where it remained during the overshoot period. During the overshoot phase the maximum stroke volume was 62 cm$^3$, sd $\pm$ 12 (81 cm$^3$/sec) with a rise in mean arterial pressure to 114 mm Hg. The maximum stroke volume occurred six to 15 heart beats after release. The mean aortic area for the group at this time increased to 7.9 cm$^2$ and heart rate slowed to 79 beats per minute. During the Valsalva maneuver both peak blood flow and velocity changed in the same direction as did the stroke volume (table 1).

The pressure-radius relationships obtained in the ascending aorta (table 2) revealed that the mean lowest radius, $r_d$, for the group was 1.45 cm, sd $\pm$ 0.12. The $\Delta r/\Delta p$ (cm/cm H$_2$O) calculated from these data had a mean value of 1.74 $\times$ 10$^{-3}$ sd $\pm$ 0.289 $\times$ 10$^{-3}$. The maximum change during the Valsalva maneuver in aortic cross-sectional areas was 17% of the control area.

**Discussion**

The correlation between the values for cardiac output measured with the pressure-gradient technique and by the indicator-dilution method in these patients compares favorably with similar studies reported by Snell and associates.$^{16}$ The mean of the difference between the two methods was 384 cm$^3$, sd $\pm$ 702 in the normal patients studied by Snell and co-workers$^{16}$ and 425 cm$^3$, sd $\pm$ 720 in the present group. Since the double-lumen catheter is placed in the ascending aorta above the coronary ostia, coronary flow will not be measured with the pressure-gradient technique. Thus, the cardiac output will be somewhat less than that obtained with an indicator-dilution method. It should be noted that the estimate of stroke volume by means of the pressure-gradient technique is subject to greater error due to the improper selection of zero flow base line than is the measurement of peak blood flow or velocity. The control values for peak blood flow, peak blood velocity, and cross-sectional aortic area recorded in these subjects are comparable to similar data obtained in this laboratory$^{17}$ and by others$^{16, 18}$ in normal subjects.

In the following discussion of the pressure-flow relationships during the Valsalva maneuver in our patients, only those findings that differ from the results obtained by other workers will be dealt with in detail. For the purposes of this presentation the Valsalva maneuver will be divided into phases I through IV as described by Hamilton and associates.$^{1}$

The earlier hypothesis that following the onset of straining (phase I) the cardiac output is transiently increased by blood being "squeezed" from the heart and lungs into the systemic arterial circulation, has been refuted by Sharpey-Schafer.$^3$ He reached this conclusion because neither the pulse pressure nor the cardiac rate changes appreciably during this period. The results of our study corroborate his interpretation in that for the first three to four heart beats after straining begins, there is no change in either stroke volume or aortic distending pressure. Therefore, the increase in blood pressure noted at this time is due solely to transmission of the elevated intrathoracic pressure to the great vessels.

**Table 2**

<table>
<thead>
<tr>
<th>Cases</th>
<th>$\Delta r$ (cm) *</th>
<th>$\Delta p$ H$_2$O $\dagger$</th>
<th>$r_d$ (cm) ‡</th>
<th>$\Delta r/\Delta p \times 10^3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.05</td>
<td>32</td>
<td>1.35</td>
<td>1.56</td>
</tr>
<tr>
<td>2</td>
<td>0.08</td>
<td>45</td>
<td>1.50</td>
<td>1.77</td>
</tr>
<tr>
<td>3</td>
<td>0.08</td>
<td>38</td>
<td>1.28</td>
<td>2.10</td>
</tr>
<tr>
<td>4</td>
<td>0.10</td>
<td>68</td>
<td>1.45</td>
<td>1.47</td>
</tr>
<tr>
<td>5</td>
<td>0.10</td>
<td>63</td>
<td>1.66</td>
<td>1.58</td>
</tr>
<tr>
<td>6</td>
<td>0.14</td>
<td>66</td>
<td>1.46</td>
<td>2.12</td>
</tr>
<tr>
<td>7</td>
<td>0.08</td>
<td>41</td>
<td>1.48</td>
<td>1.95</td>
</tr>
<tr>
<td>8</td>
<td>0.11</td>
<td>80</td>
<td>1.36</td>
<td>1.37</td>
</tr>
</tbody>
</table>

*Data were obtained from the angiograms and represent the maximum changes in vessel radius which occurred during angiography.

†$\Delta p$ represents the concomitant changes in aortic pressure.

‡$r_d$ is the lowest measured value for aortic radius.
Rudewald18 with a modified pressure-gradient technique, the control stroke volume of 81 cm³ fell to 28 cm³ during phase II. As has been noted previously,1-5 mean arterial pressure tends to rise somewhat during the latter part of straining, presumably because of augmented peripheral resistance. In our subjects mean arterial pressure did increase during this period, but the stroke volume continued to decrease gradually. Booth and co-workers7 used an indicator-dilution technique to measure cardiac output during the latter part of phase II and reported an increase in flow. One possible explanation for these conflicting findings between patients and those studied by Booth and co-workers7 is that the duration of straining was 10 to 15 seconds longer in their subjects. However, in two of our patients in whom the straining period was prolonged to 35 seconds, no increase in stroke volume was noted during the latter part of phase II.

Immediately following release (phase III) the blood pressure dropped rapidly to its lowest level; presumably this decrease was due to the sudden decrease in intrathoracic pressure. Thereafter, both stroke volume and blood pressure increased rapidly as predicted by Hamilton and associates.1 It is interesting to note that flow and pressure increased in a manner such that calculated peripheral vascular resistance decreased within three heart beats after release to control levels where it remained during overshoot. Both stroke volume and blood pressure were elevated above control values during the overshoot (phase IV). An increase in pulmonary blood flow was reported during phase IV by Lee and DuBois,19 who used a body plethysmograph to obtain their data. However, neither McIntosh and associates6 nor Booth and co-workers7 measured an increased cardiac output during phase IV in normal male subjects. In both of these latter studies an indicator-dilution technique was used to measure cardiac output. In our patients mean flow increased from 34 to 82 cm³/sec during this period. It is reasonable to assume that an integrated value for cardiac output, as obtained by an indicator-dilution technique, would reflect only the average flow during this period. Thus, the fact that the flow was transiently increased during overshoot might not be recorded with these techniques.

The duration of total systole and duration of ejection changed during the Valsalva maneuver by 30 msec and 60 msec, respectively. One would expect the duration of ejection to reflect concomitant changes in stroke volume; however, the duration of total systole is not influenced by changes in flow.20 The alterations in the duration of total systole are secondary to the changes in heart rate and the inotropic stimulation by the sympathetic nervous system during the Valsalva maneuver.

In the foregoing, we have used by convention the term peripheral vascular resistance to denote data obtained by solving equation 3. However, these data in fact represent the resistive or nondynamic component of the hydraulic input impedance to the aorta.21 In a steady state these are analogous, and values for peripheral vascular resistance calculated from equation 3 have been found to be a most helpful tool by both circulatory physiologists and clinicians. Since the aorta is a collapsible tube, flow into the ascending aorta does not necessarily imply that an equal volume of flow occurs simultaneously at the arteriolar level. Thus for a few heart beats after the release of a Valsalva maneuver, the true peripheral vascular resistance may be extremely high because a considerable amount of blood is required to distend the aorta to its control volume, and little peripheral flow occurs. However, at this time values for peripheral vascular resistance computed from equation 3 are near control levels and do not reflect the peripheral pressure-flow relationships (fig. 1). In the present studies the ascending aorta decreased in cross-sectional area by 1.0 cm² during the straining phase.
Assuming the ascending aorta to be a cylinder, a segment 10 cm in length would have decreased in volume by 10 cm. Thus, a considerable portion of the cardiac output immediately following release will be used to dilate the thoracic aorta.

The pressure-radius relationships for the ascending aorta recorded in these subjects are remarkably consistent with similar data obtained by direct measurement at the operating table by Greenfield and Patel. In these patients \( \Delta r/\Delta p \times 10^8 \) was 1.82 cm/cm H2O, and in the present group (table 2) it was 1.74 cm/cm H2O. It, therefore, seems reasonable that the pressure-radius relationships of the ascending aorta can be estimated fairly well with the angiographic techniques used in this study.

To recapitulate we wish to stress the following observations: (1) Immediately after initiation of straining (phase I) mean arterial pressure increased by approximately the same amount as right atrial pressure. Stroke volume did not change during this period. (2) Both blood pressure and stroke volume fell rapidly shortly after onset of the Valsalva (phase II), and at the midpoint of the straining phase mean arterial pressure and stroke volume had decreased to 85% and 56% of the control values, respectively. Thereafter stroke volume continued to fall slowly, declining to 44% of control before release; mean arterial pressure rose slightly to 90% of control just before release. (3) Stroke volume did not change immediately after release (phase III), but mean arterial pressure fell to 74% of the control value (its lowest level). But stroke volume and arterial pressure rose to the control values at from six to nine beats after release. (4) The maximum stroke volume occurred six to 15 beats after release, and during the overshoot it reached a level 24% greater than control. (5) The cross-sectional area of the ascending aorta changed by 17% during the Valsalva maneuver.

Acknowledgment

The technical support rendered by Mrs. Joan Swain is greatly appreciated as is the invaluable help of the Medical Illustration Service at the Durham Veterans Administration Hospital, directed by Mr. Leonard Hart.

Addendum

Since this paper was submitted, a study of blood flow in the descending thoracic aorta of eight normal subjects performing a Valsalva maneuver has been published by Fox and associates. These workers used a constant-rate-injection-indicator-dilution technique to measure flow. Their results are similar to the findings presented in this present report.

References


Graves on Medical Education (1848)

This is an age of ambitious acquirement, and professional men seem to be ashamed unless they have the character of universal knowledge. Every body studies every thing, and the consequence is that few know any thing well. We live amidst the din of declamations in favour of general education; and are every where assailed by the ceaseless competition of those who vend cheap knowledge in the form of penny periodicals, lectures innumerable, and hosts of rival encyclopaedias; but ours is not an age of calm unpretending acquirement and severe precise study, without which, the effort to become good physicians and surgeons must prove vain and fruitless.—ROBERT J. GRAVES: Clinical Lectures on the Practice of Medicine, ed. 2, vol. 1. Dublin, Fannin and Co., 1848, p. 14.
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References


Correction

In the article on "Pressure-Flow Studies in Man During the Valsalva Maneuver with Observations on the Mechanical Properties of the Ascending Aorta" by Joseph C. Greenfield, Jr., Ronnie L. Cox, Rafael R. Hernandez, Corinna Thomas and Fred W. Schoomaker (April CIRCULATION 35: 653-661, 1967), formula #1 on page 654 should read:

$$- \cfrac{\partial p}{\partial z} = \cfrac{(1.1) \rho \cfrac{\partial q}{\partial t}}{g \pi r^2} + \cfrac{(1.6) 8\mu}{g \pi r^4} q \quad (1)$$

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