The Effect of the Valsalva Maneuver on Echocardiographic Dimensions in Man

David Robertson, M.D., Rose Marie Stevens, M.D., Gottlieb C. Friesinger, M.D., and John A. Oates, M.D.

SUMMARY Physiologic changes in the circulatory system caused by performing the Valsalva maneuver are blunted or absent in patients with congestive heart failure. Previously there has been no noninvasive method for examining cardiac chamber size during this maneuver. M-mode echocardiography was used to evaluate possible changes in cardiac chamber dimensions in 12 normal subjects (group I) and 15 patients with cardiovascular disease (group II). In group I, the left ventricular end-diastolic dimension decreased 11.2% (±1.5%) and the end-systolic dimension 9.5% (±1.32%), with a fall in stroke volume of 29%. The left atrial (LA) dimension decreased 30%.

The Valsalva maneuver, raising intrathoracic pressure against a closed glottis ("forced expiration"),1 and the Mueller maneuver, reducing intrathoracic pressure behind a closed glottis ("forced inspiration"),2 have been used in the investigation of cardiovascular hemodynamics and autonomic nervous system function.3-5 Both produce marked hemodynamic changes, resulting in widespread alterations in the central and peripheral circulation. Disease states alter these responses.6 The echocardiogram permits the noninvasive determination of an internal dimension of the left atrium, aorta, and left ventricle. We investigated the echocardiogram during the Valsalva and Mueller maneuvers in normal volunteers and in patients with heart disease to determine the change in cardiac chamber size that such maneuvers might produce and the correlation of this change with abnormalities of cardiovascular function.

Methods

Twenty men and seven women were studied. Group I was composed of 12 normal subjects (age range 21 to 58 years, mean = 32) who had no history of cardiovascular or other disease. These subjects were chosen from a group of 24 volunteers because they had satisfactory ventricular and atrial echograms throughout the Valsalva maneuver. Group II consisted of 15 patients (19 to 65 years, mean = 47) admitted to Vanderbilt University Hospital for cardiac evaluation and included patients with ischemic heart disease, aortic stenosis, mitral regurgitation, mitral stenosis and one with severe hypertension. These patients (excepting the hypertensive patient) underwent cardiac catheterization within one week of the study described below. Their clinical status and medications were stable between the two studies.

All echocardiograms were performed in the supine posture with patients in the postabsorptive state. Echocardiograms were done using an Ekoline 20A, Hoffrel 101C, or Unirad Series C ultrasonoscope, and recordings were made on a Honeywell 1858 or Cambridge strip chart recorder. A 2.25 MHz, 13 mm diameter, nonfocused transducer with a repetition rate of 1,000 cycles per second was used. Transducer position was maintained constant throughout control and experimental maneuvers after initial echocardiographic exploration for rotation of the left atrium did not suggest that lateral or cephalo-caudal motion of the atrium accounted for the alteration in dimension observed.

The Valsalva and Mueller maneuvers were calibrated using a standard mouthpiece connected in parallel to a graduated manometer and a Statham transducer. The manometer displayed the positive or negative pressure generated by the patient and was placed in view of the patient so that he could maintain the desired pressure. The pressure generated was recorded on the echocardiogram (via a pulse amplifier) or the time during which it was maintained was marked by a separate observer using a marker channel on the strip chart recorder. All subjects were thoroughly instructed and practiced each maneuver several times. Movement and tension of the thoracic and abdominal musculature were observed to ensure valid performance.

Left atrial diameter was measured as an internal dimension from the posterior aortic root echo to the atrial wall echo at both maximal and minimal points for each cardiac cycle. Measurements were made only from echocardiograms that demonstrated aortic valve leaflets within the root, so as to ensure stability of transducer position throughout the Valsalva maneuver. Also, the diameter of the aorta was monitored for alteration in size, since lateral motion of the heart at the level of the aorta would give an apparent decrease in aortic diameter as the ultrasonic beam traversed the aorta eccentrically. Significant changes in the aortic dimension did not occur.

Left ventricular end-diastolic dimensions were taken just below maximal mitral valve excursion, where septum and posterior endocardium were clearly defined, and were timed to coincide with the Q wave of the simultaneous electrocardiogram. End-systolic dimensions were taken at the point of most anterior movement of the posterior endocardium. All echocardiograms were measured by two observers with an average difference in dimension change measurement of 1

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ventricular end-diastolic dimension ultimately decreased an average of 11.2% (±1.5%) while the end-systolic dimension was reduced 9.5% (±1.3%) and the echocardiographic estimate of stroke volume was reduced by 29% (fig. 2). The dimensions changed little during the first three seconds of the Valsalva maneuver, but then began to decline progressively over an average of 12 seconds, after which the dimensions remained stable until the positive intrathoracic pressure was released. A slight and statistically insignificant increase in left ventricular dimension was recorded in 8 of 12 subjects. During the "overshoot phase" (5–7 seconds after release of positive intrathoracic pressure), increased respiratory activity prevented recording of clear ventricular wall echoes.

The left atrial chamber size was even more sensitive to changes in intrathoracic pressure. Atrial dimensions were reduced 30% and 28% respectively when measured at their maximal and minimal size during the cardiac cycle (fig. 3). Since there was little difference between the changes in maximal and minimal dimensions, we considered only the maximal dimension in further investigations. The decrease in

![Diagram](http://circ.ahajournals.org/doi-figures/1975/597)

**FIGURE 1.** These unretouched echocardiograms illustrate a normal response to the Valsalva maneuver with a line drawing of the control and intra-maneuver echoes. In the top strip, the solid line appearing in the middle of the aortic root marks the attainment of 40 mm Hg pressure noted by an observer watching the manometer into which the patient is blowing. $AVO =$ aortic valve open in systole; $AVC =$ closure line of the aortic valve; $AR =$ aortic root; $LA_{max} =$ maximal left atrial dimension; $LA_{min} =$ minimal left atrial dimension. Note that the left atrial size decreases markedly as the maneuver progresses. In the bottom strip, the waving line is the representation of the positive intrathoracic pressure generated by the subject's blowing into a manometer. 40 mm Hg was achieved in the Valsalva. $RVW =$ right ventricular wall; $IVS =$ interventricular septum; $LVD_{D} =$ left ventricular end-diastolic dimension; $LVD_{S} =$ end-systolic dimension; $MV =$ mitral valve; $CH =$ chordae; $PW =$ left ventricular posterior wall. Note that both $LVD_{D}$ and $LVD_{S}$ diminish as the maneuver proceeds.
atrial size began at about 3 seconds into the maneuver and continued for about 9 seconds. While the atrial dimensions appeared to increase to greater than control values during the overshoot phase, accurate measurement was again hindered by respiration.

The heart rate increased an average of 20 beats/min during the Valsalva maneuver (P < 0.001). The acceleration in heart rate for the 12 normal subjects ranged from 8–34 beats per minute. The correlation coefficient between atrial size change and heart rate change was 0.76. In general, those whose atrial dimension changed the most also had the greatest changes in heart rate.

The unexpectedly large change in left atrial dimension with 40 mm Hg positive intrathoracic pressure prompted investigation of lower levels of pressure. On 14 occasions in four subjects, we determined left atrial dimension change with positive pressures of 40 mm Hg, 30 mm Hg, 20 mm Hg, and 10 mm Hg. Significant alterations in left atrial dimension were noted even at 10 mm Hg, with left atrial dimension decreasing by 13%. The other positive pressures affected the

Figure 2. Change in left ventricular dimension and stroke volume with a 40 mm Hg Valsalva maneuver in 12 normal subjects. Each point represents the mean percent change in dimension from control for the 12 subjects. On the abscissa, C = control value (three beats averaged), and numbers 1–10 indicate cardiac cycles after the beginning of the maneuver. Minimum indicates the lowest value reached, usually attained between 12 and 20 cycles. Bars indicate one standard error. * P < 0.05; ** P < 0.01; *** P < 0.005: change vs. control.

Figure 3. Change in left atrial maximal and minimal dimensions (see fig. 1 for technique and measurement) with a 40 mm Hg Valsalva maneuver in 12 normal subjects. Each point represents the mean percent change in dimension from control for the 12 subjects. On the abscissa, C = control value (three beats averaged), and numbers 1–10 indicate cardiac cycles after the beginning of the Valsalva maneuver. Minimum indicates the lowest value reached, usually attained between 12 and 20 cycles. Bars indicate one standard error. * P < 0.05; ** P < 0.01; *** P < 0.005: change vs. control.
atrial dimension in a linearly progressive fashion (fig. 4).

Compared to the Valsalva maneuver, the Mueller maneuver produced very modest alterations. Left atrial maximal dimension and the left ventricular dimension remained stable during the maneuver. The left atrial minimal dimension increased an average of 4% (statistically insignificant), with 11 of 12 subjects demonstrating this response.

The heart rate fell an average of 5 beats/min during the Mueller maneuver (P < 0.01), but no overshoot phenomenon was observed.

Patients

Because of the difficulty in recording a clear left ventricular echo throughout the Valsalva maneuver, and because the left atrial dimension was far more sensitive to the maneuver, this latter parameter was examined in patients with various forms of heart disease (table I). Patients receiving sublingual nitroglycerin, sublingual isosorbide dinitrate, or propranolol were studied at the end of a dose interval, no less than six hours after the last dose of either of these medications. (Patient L. C. was receiving propranolol for otherwise intractable angina despite the coexistence of severe congestive failure. This latter condition predated the use of propranolol and was not clinically worsened by this medication.)

In contrast to the 30% reduction in left atrial dimension induced by the Valsalva maneuver in normal subjects, nine patients in NYHA functional class III or IV reduced their left atrial dimension by an average of only 3.8% (P < 0.001 compared with group I response). All these patients had diminutions in left atrial size of 6% or less except patient R. H. who was receiving α-methyldopa. Two representative responses of patients in failure are shown in figure 5. The four asymptomatic patients, those in NYHA functional Class I, did not differ from normal subjects in their response to the Valsalva maneuver. In general, subjects with little change in atrial dimension with Valsalva also had little or no increase in heart rate during the maneuver.

Because the Mueller maneuver produced no significant change in normal subjects, it was not investigated in patients.

Discussion

The Valsalva maneuver is best understood in terms of its four phases. At the onset of the maneuver, when intra-

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**TABLE 1. Clinical and Echocardiographic Data for Patients in Group II**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age/Sex</th>
<th>Diag</th>
<th>NYHA Class</th>
<th>Medications</th>
<th>Echo LA Dimension</th>
<th>% change*</th>
<th>Mean % change by CHF class</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.B.</td>
<td>64F</td>
<td>IHD</td>
<td>I</td>
<td>TNG</td>
<td>33 mm</td>
<td>-30%</td>
<td></td>
</tr>
<tr>
<td>R.B.</td>
<td>56M</td>
<td>unstable angina</td>
<td>I</td>
<td>TNG, propranolol</td>
<td>36 mm</td>
<td>-31%</td>
<td></td>
</tr>
<tr>
<td>C.J.</td>
<td>57M</td>
<td>IHD</td>
<td>I</td>
<td>quinidine, propranolol</td>
<td>28 mm</td>
<td>-29%</td>
<td></td>
</tr>
<tr>
<td>J.L.</td>
<td>62M</td>
<td>hypertension</td>
<td>I</td>
<td>—</td>
<td>40 mm</td>
<td>-32%</td>
<td></td>
</tr>
<tr>
<td>J.R.</td>
<td>47M</td>
<td>AR</td>
<td>II</td>
<td>digoxin</td>
<td>39 mm</td>
<td>-25%</td>
<td></td>
</tr>
<tr>
<td>R.O.</td>
<td>44M</td>
<td>MR</td>
<td>II</td>
<td>—</td>
<td>38 mm</td>
<td>-13%</td>
<td></td>
</tr>
<tr>
<td>T.E.</td>
<td>37F</td>
<td>MS</td>
<td>III</td>
<td>thiazide, digoxin</td>
<td>32 mm</td>
<td>-5%</td>
<td></td>
</tr>
<tr>
<td>E.M.</td>
<td>61F</td>
<td>AS</td>
<td>III</td>
<td>—</td>
<td>33 mm</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>P.M.</td>
<td>33F</td>
<td>MS</td>
<td>III</td>
<td>thiazide, digoxin</td>
<td>37 mm</td>
<td>-5%</td>
<td></td>
</tr>
<tr>
<td>M.A.</td>
<td>31F</td>
<td>MS</td>
<td>III</td>
<td>thiazide, digoxin</td>
<td>49 mm</td>
<td>-2%</td>
<td></td>
</tr>
<tr>
<td>C.W.</td>
<td>65F</td>
<td>AR</td>
<td>IV</td>
<td>furosemide, digoxin</td>
<td>38 mm</td>
<td>-3%</td>
<td></td>
</tr>
<tr>
<td>L.M.</td>
<td>65F</td>
<td>MR</td>
<td>IV</td>
<td>furosemide, digoxin</td>
<td>42 mm</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>D.A.</td>
<td>47M</td>
<td>cardiomyopathy</td>
<td>IV</td>
<td>thiaside, digoxin</td>
<td>46 mm</td>
<td>+4%</td>
<td></td>
</tr>
<tr>
<td>L.C.</td>
<td>43M</td>
<td>IHD</td>
<td>IV</td>
<td>TNG, propranolol, digoxin</td>
<td>38 mm</td>
<td>-6%</td>
<td></td>
</tr>
<tr>
<td>R.H.</td>
<td>53M</td>
<td>IHD</td>
<td>IV</td>
<td>isosorbide dinitrate, α-methyldopa, digoxin</td>
<td>47 mm</td>
<td>-17%</td>
<td></td>
</tr>
</tbody>
</table>

*Percent change in left atrial dimension with the Valsalva maneuver.

† = P < 0.001

†† = P < 0.005.

Abbreviations: Pt = patient; NYHA Class = New York Heart Association functional classification; IHD = ischemic heart disease; TNG = nitroglycerin; AR = aortic regurgitation; MR = mitral regurgitation; MS = mitral stenosis; MR = mitral regurgitation.
The chest and abdominal pressure rise suddenly, there is a corresponding transient increase in systemic blood pressure (Phase I). Phase 2 is characterized by a fall in blood pressure and pulse pressure accompanied by a reflex tachycardia. With release of the raised intrathoracic pressure, systemic blood pressure falls (Phase 3). About five seconds later, increased venous return causes pulse pressure and mean blood pressure to rise to greater than control levels. With this raised stroke volume, parasympathetic slowing of the heart occurs (Phase 4). If a trained subject exhales into a manometer rather than against a closed glottis, similar cardiovascular effects are observed. Although with practice, positive intrathoracic pressure of 150 mm Hg and negative pressure of 50 mm Hg can be attained by healthy adults, major hemodynamic effects are seen at 40 mm Hg, which is within the reach of almost all adults. The magnitude of the hemodynamic change induced can be great: after seven seconds of the Valsalva maneuver at 40 mm Hg, the pulse pressure may be reduced 80% and 1500 ml of blood pooled into the periphery.

Hamilton first observed that patients with left-sided congestive heart failure functioned abnormally during the Valsalva maneuver; while pulse pressure falls in normal persons, it remains unchanged in patients with congestive heart failure. Similar abnormalities were identified also in patients with right-sided failure or with severe mitral or aortic stenosis.

While cardiac chamber pressures and systemic blood pressures during the Valsalva maneuver have been investigated, changes in cardiac chamber size have been more difficult to assess. It was noted early that the cardiac silhouette on chest X-ray became smaller during the Valsalva maneuver, and during the overshoot phenomenon, sometimes became slightly overdistended in patients with mitral stenosis. This was thought to be due to the temporary dilatation of the left atrial appendage and was proposed as a radiographic sign of this valvular abnormality.

The technique of suturing metal markers to the surface of the ventricles during cardiac surgery and subsequently analyzing cineradiographic studies of such patients during various physiological and pharmacological interventions has been shown to correlate with but not estimate chamber volumes. By this method a right ventricular dimension decreased sharply during the Valsalva maneuver while the left ventricular dimension initially remained constant or increased slightly but then diminished. During overshoot, the ventricular end-diastolic dimension increased an average of
angiographic studies revealed a 50% decrease in stroke volume during the Valsalva maneuver, with shortening of the duration of ejection and reduction in aortic cross-sectional area by 17%. 

Conversely, echocardiographic exploration during the maneuver revealed a 50% decrease in left ventricular size and a 42% increase in cardiac output. 

However, since the view of the heart is limited to a single axis, one must eliminate artifactual causes of the phenomena observed. Possible alternative explanations for the apparent diminution in atrial echocardiographic dimension included certain potential technical difficulties: 1) rotation of the atrium during the maneuver, altering the dimension through which the ultrasound beam traveled, and 2) conformation change in the atrium induced by movement of the esophagus. As mentioned above, echocardiographic exploration during the maneuver did not reveal a larger atrial dimension in any direction. To observe the behavior of the esophagus during positive intrathoracic pressure, subject performed the maneuver in the course of an upper gastrointestinal survey. The barium-lined esophagus was observed radiographically and found to remain collapsed during the maneuver without any impingement on the left atrium. 

Because the echocardiogram permits noninvasive study of cardiac chamber size on a beat-to-beat basis, we applied this technique to patients with heart disease. Patients with most severe congestive heart failure had the least decrease in left atrial size with positive intrathoracic pressure. In several patients, the left atrial size did not change at all, and in one patient it increased slightly. It is conceivable that with greater intrathoracic pressures there would be a more "normal" response of left atrial size. The standard Valsalva maneuver, however, required considerable effort, and higher levels of pressure were not attempted in this study. 

The findings in this study may explain the so-called "square-wave" phenomenon previously noted in patients with congestive heart failure. Instead of the diminution in systolic pressure and pulse pressure seen in normal subjects, in patients with heart failure the pulse pressure often does not decrease, and the systemic blood pressure may be raised for the duration of the positive intrathoracic pressure. If it is possible to generalize from the response of the left atrium, our data suggest that the failure of the pulse pressure to diminish in these subjects may relate to the fact that their heart chambers do not change in size with the maneuver, and heart volume remains near control levels. 

Other physiological maneuvers have been used to differentiate normal subjects from patients with cardiovascular dysfunction. Lower body negative pressure (30 mm Hg) reduced cardiac output and stroke volume by 20% in normal subjects but these parameters were unchanged in two patients with advanced heart failure. Upright tilting reduced left ventricular end-diastolic volume index as estimated from echocardiographic dimensions in normal subjects, and this parameter also remained unchanged in subjects with left ventricular failure. These physiological maneuvers and the Valsalva maneuver are similar in at least one respect: all are associated with a peripheral pooling of blood in normal subjects, and this is accompanied by an increased heart rate and often a demonstrable fall in blood pressure. 

Possible explanations of the insensitivity of patients with heart failure to these maneuvers include: 1) alterations in blood volume; 2) functional and/or structural changes in the heart or vascular system; and 3) altered activity of the sympathetic nervous system. 

Increases in blood volume are known to occur in heart failure. This could interfere with the ability to pool blood efficiently in response to postural or pressure stimuli. Structural and functional changes in the cardiovascular system are also known to accompany heart failure. Increased arteriolar vascular stiffness is not unrelated to sympathetic nervous function has been described; should structural changes also occur in peripheral veins, alterations in venous capacitance might result and impede peripheral pooling. In patients with valvular heart disease, it might be supposed that simple obstruction to outflow could diminish the response to Valsalva. While this may indeed play a role, it does not account for those patients with the most abnormal responses, more than half of whom had no obstructive valvular lesions. It is, however, of interest that one patient with mitral stenosis had an abnormal left atrial and normal left ventricular response suggesting that outflow obstruction may in some cases account for a significant part of the abnormal response. It may be that in these cases both the severity of congestive failure and the abnormality of the response are related to the degree of obstruction to outflow. 

The increased activity of the sympathetic nervous system in severe heart failure is suggested by an increased urinary norepinephrine excretion, a raised plasma norepinephrine level at rest, and such clinical evidence as tachycardia, diaphoresis, suppression of urine formation and peripheral vasoconstriction. The increased venous tone seen in heart failure is due, at least in part, to this increased sympathetic activity. The combination of increased peripheral venous tone and elevated blood volume would seem to be plausible explanations for the lack of decrease in heart size during positive intrathoracic pressure in patients with heart failure. 

The echocardiographic atrial response to the Valsalva maneuver is an objective correlate of the severity of congestive failure. The sensitivity of this correlation needs further documentation; the observation, however, may provide an additional clinical tool for further evaluation of patients with cardiovascular disease.

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References


Intracardiac Pressure-Sound Correlates of Echographic Aortic Valve Closure

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GORDON BORKAT, M.D., AND CONNIE BORMUTH

SUMMARY Echographic aortic valve closure was compared to the dicrotic notch of the aortic pressure and intracardiac A2 to define the exact temporal relationship of the echographic, pressure, and sound parameters of aortic valve closure. Sixteen children, ages 3–20 years, were evaluated by simultaneous aortic valve echograms, micro-manometric root pressure tracings, and intracardiac phonocardiograms recorded at paper speeds of 200 mm/sec. Our observations demonstrated that echographic coaptation of the aortic valve leaflets coincides with the trough of the aortic pressure incisura and the onset of A2. The data suggest that A2 is a result of valve closure.

ALTHOUGH considerable controversy exists regarding the genesis of the aortic component of the second heart sound (A2), two major views prevail. One view contends that A2 is a direct result of aortic valve closure (AVC). The other theory relates the genesis of A2 to the vibrations that result from rapid acceleration and deceleration of blood within the cardiohematic system. The latter theory de-emphasizes the role of semilunar valve closure.

Cineangiographic studies in man and studies employing electrical techniques in animals have demonstrated AVC to be coincident with the incisura of the aortic pressure curve but preceding A2. Data from these studies supported the view that AVC does not generate A2. Echocardiographic studies which allow clear, instantaneous delineation of aortic leaflet closure have been brought recently into the controversy. Some investigators appeared to have demonstrated that echographic AVC preceded the surface recording of A2, while data from Craig suggested that echographic AVC and the surface recorded A2 were simultaneous. Consequently, a study was undertaken of three simultaneously compared intracardiac events in order to delineate exact temporal relationships of echographic, pressure, and sound parameters of AVC. The three events were echo-
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