multilevel atrioventricular block. These interpretations can be made from the clinical electrocardiogram and do not require complex electrophysiologic studies. This phenomenon is not rare and occurs under a variety of clinical circumstances. It is likely that multilevel block is far more common than previously suspected, since stable 4:1 block in atrial flutter probably represents two levels of 2:1 block. The more complex patterns are often transient, or may simply not be recognized. The patterns presented in this series are not exhaustive of the permutations of multilevel block that can occur. Recognition that block can occur at more than one level in the A-V conduction system may facilitate the interpretation of hitherto obscure rhythm disturbances and thereby aid in the management of the patient with arrhythmia.

References
1. Lewis T: Atrial flutter. Heart 4: 171, 1912

Echocardiographic Evaluation of the Valsalva Maneuver in Healthy Subjects and Patients with and without Heart Failure

ALFRED F. PARISI, M.D., JAMES J. HARRINGTON, B.S., JOSEPH ASKENAZI, M.D., RICHARD C. PRATT, M.D., AND KEVIN M. McINTRYE, M.D.

SUMMARY  The Valsalva maneuver was evaluated by echocardiography in three groups: A) 10 normal volunteers, B) 10 patients with no history of heart failure and normal ejection fractions, and C) 10 patients with heart failure and depressed ejection fractions.

Groups A and B had a significant fall in left ventricular internal dimensions and calculated stroke volume by end strain which returned rapidly to baseline in recovery without significant overshoot. Arterial pressure showed a sigmoidal strain pattern with a normal overshoot in early recovery in all group B patients. In group C ventricular dimensions did not diminish during strain; arterial pressures showed a "square wave" pressure elevation during strain without an overshoot in recovery. Echocardiography allows a new approach to evaluate further the left ventricular response to the Valsalva maneuver. Patients with severely depressed ejection fractions, unlike those with normal ventricular function, are unable to alter stroke output in response to acutely increased intrathoracic pressure. A square wave pressure response is a likely consequence of a fixed stroke output during the strain maneuver.

THE VALSALVA MANEUVER is commonly employed clinically and far more often practiced unwittingly in the course of biological and occupational activities. The blood pressure response to the Valsalva maneuver has been extensively studied.1-5 Individuals without heart failure have a sigmoidal strain blood pressure response, i.e., a rise above resting pressures during early strain followed by a progressive fall to below control pressure in late strain. At release blood pressure overshoots baseline measurements. In patients with heart failure there is a square wave response, i.e., blood pressure rises above control during the entire strain phase and falls to resting blood pressure levels at release.

There has been little opportunity to examine the effects of acutely raising intrathoracic pressure on left ventricular chamber dimensions and beat-to-beat stroke volume. With the advent of echocardiography it has become possible to assess rapidly and continuously the left ventricular response to this maneuver. Accordingly this study was designed to further evaluate with echocardiography the effect of the Valsalva maneuver on the left ventricular dimensions of normal subjects as compared to those of patients with and without heart failure. A report of our initial findings has already been made.4

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Received May 20, 1976; revision accepted July 6, 1976.
Methods and Materials

This study was carried out on 10 normal volunteers (group A) with no evidence of cardiovascular disease; 10 patients (group B) with no history of heart failure and with normal sized hearts who were referred for cardiac evaluation, all of whom proved to have normal ejection fractions at cardiac catheterization; and 10 patients (group C) with a history of heart failure and cardiomegaly who had depressed ejection fractions at cardiac catheterization. All groups were studied by echocardiography just before, while performing, and immediately after the strain phase of the Valsalva maneuver.

M-mode strip chart echocardiograms were made on a Kent-Cambridge multichannel recorder interfaced with a Smith Kline Ecoline 20 A echograph. Either an Aerotech 1.6 MHz or 2.25 MHz ultrasound uncollimated transducer was used to obtain the echoes.

The transducer was placed along the left sternal border at the third or fourth intercostal space. The anterior leaflet of the mitral valve was detected and a scan of the left ventricle from base to apex was performed to locate the posterior leaflet. The echocardiogram was recorded at the distal tip of the mitral leaflets and left ventricular internal dimensions were measured at this level. Care was taken to delineate the left side of the interventricular septum and the endocardium of the posterior left ventricular wall. A simultaneous electrocardiogram (lead II) was also recorded. Records were made at a paper speed of 50 mm/sec with time lines every 0.04 sec.

The Valsalva maneuver was performed by blowing a mercury column to a height of 40 mm and maintaining it for 20 seconds.9 The subjects were instructed to perform the maneuver near end expiration to minimize the air interface between the ultrasound transducer and the heart. Echocardiograms were continuously recorded from rest (just prior to the performance of the maneuver), through the strain phase of the Valsalva maneuver and after release.

The echocardiograms taken were carefully measured by two observers who determined left ventricular end-diastolic and end-systolic dimensions during the maneuver. The baseline phase of the maneuver, which included both phases of respiration, was taken as the five to ten second interval immediately before the onset of the Valsalva maneuver. The strain phase was marked on the echogram and confirmed by the presence of muscle tremor on the ECG. It was subdivided into three intervals, early (0-5 sec), mid (10-15 sec) and late (16-20 sec) strain phase.

At release of strain the mark was removed from the strip chart and the recovery phase was similarly subdivided. The early, mid and late phases of recovery were measured at 0-5 sec, 6-10 sec and 15-25 sec, respectively. During the stated intervals at least four consecutive beats were measured and an average dimension for that beat sequence was tabulated. Left ventricular volumes were derived using the formula of Teichholz et al.6

A high quality echocardiogram was obtainable in two-thirds of subjects with well defined left ventricular endocardial landmarks at rest. The major problem encountered was a tendency to gasp at beginning strain and particularly at release — an act which introduced the air interface of lung between the transducer and the heart. Possibly for this reason patients with cardiomegaly were somewhat easier to study than individuals with normal sized hearts.

Group A consisted of six men and four women with a mean age of 28.4 years (range 23 to 39). They were selected as a reference group because they were free of cardiovascular disease as determined by detailed history, physical examination, chest X-ray and electrocardiogram. All were studied in the resting supine postabsorptive state.

Group B were men selected because they had no history of heart failure or cardiomegaly on physical examination. Their mean age was 46.9 (range 19 to 63). Their clinical problems and angiographic findings are listed in table 1. All ten in this group were studied at cardiac catheterization. Echocardiograms were made with hemodynamic measurements before angiography. All had normal ejection fractions. Brachial artery or aortic and right atrial pressures were measured simultaneously during the Valsalva maneuver on an Electronics for Medicine DR8 recorder. The ECG was displayed simultaneously on both the echocardiograms and pressure recordings. Standardization marks were introduced on the ECG every 5-10 sec so that measurements could be made for simultaneous beats.

Group C consisted of 10 men chosen because they had a history of dyspnea and physical findings indicating left ventricular failure (S3, rales and cardiomegaly). Their mean age was 48.2 (range 24 to 64). Nine had severely depressed ejection fractions at cardiac catheterization (table 1). One patient (E.F.) was a 38-year-old black man with a history of severe hypertension and heart failure; he did not have a cardiac catheterization study. The remaining nine came to cardiac catheterization to exclude quantitatively significant mitral regurgitation or a ventricular aneurysm. Coronary angiography was not performed in PT, AK, WP, and JM when the above lesions were not found. HR and JCa had normal coronary arteriograms. SS subsequently died of heart failure; he had an enlarged heart consistent with a cardiomyopathy, mild rheumatic mitral disease and normal coronary arteries at postmortem examination. In five of these subjects the echocardiographic studies were conducted with simultaneous pressure measurements at catheterization as in group B. In the remainder the Valsalva maneuver was performed after diagnostic catheterization. None of this latter subgroup had had a clinical event or surgical procedure in the postcatheterization interval to suggest a significant change in their left ventricular performance.

Pressure measurements were made from fluid-filled catheters connected to Statham P23Ia pressure transducers. Beat-to-beat resistance in arbitrary units was calculated by dividing the echocardiographic stroke volume into the difference of the simultaneous mean systemic arterial and mean right atrial pressures. Ejection fractions were determined from 30° right anterior oblique 35 mm cineventriculograms using the length-area method.4 Coronary angiography was performed by either the Sones or Judkins technique. Statistical analyses were done according to standard formulae using a PDP 8/1 computer.7 Informed consent was obtained from all participants in writing before each study.

Results

1. Group A — Normal Volunteers

Figure 1 illustrates segments of a typical record from a normal volunteer obtained during the Valsalva maneuver.
During strain, left ventricular dimensions diminished markedly and returned to normal once the strain was released. Mitral elements often were more prominently represented in late strain phase recordings, a feature which disappeared immediately upon release of the Valsalva effort.

Table 2A lists the mean measured diastolic, systolic dimensions and heart rate in these volunteers during the early, mid and late strain and recovery phases of the maneuver.

Diastolic dimension was 4.6 ± 0.2 cm at rest and fell progressively to 3.5 ± 0.2 cm by termination of strain. This dimension rapidly returned to 4.5 ± 0.1 cm during early recovery, a value not significantly different from control. The value of 4.7 ± 0.2 cm in the mid-recovery phase was also not significantly different from the resting value.

Systolic dimension was 3.0 ± 0.2 cm at rest and paralleled diastolic dimension in its gradual progressive diminution with strain and rapid return to baseline in early recovery.

Figure 2 illustrates the percent change in diastolic and systolic dimensions, and heart rate during the Valsalva maneuver.
systolic dimensions for group A patients. Groups B and C are similarly plotted for comparison. Systolic dimensions decreased proportionately more than diastolic dimensions by end strain in group A patients.

Figure 3 shows the mean derived values for systolic and diastolic volumes, stroke volume and heart rate for this group. Maximum heart rate increase (25%) was achieved by the mid-strain phase, while maximum depression of stroke volume occurred during the terminal strain phase. After release heart rate fell promptly and reached a low of 85.7% of control at mid-recovery. At this time the stroke volume was 108% of control (NS). Alternative derivations of left ventricular volumes (Appendix I) yielded a similar significant fall in stroke output by terminal strain, with values of 50%-67% of rest depending on the formula chosen. No statistically significant stroke volume overshoot occurred after release regardless of the regression equation chosen.

2. Group B — Patients without Heart Failure

The ten subjects in group B had a resting mean heart rate of 59, 16 beats lower than the volunteers in group A (table 2B). Five of these subjects were taking propranolol up to the time of cardiac catheterization; the mean dose was 40 mg four times daily (total dose range 80-240 mg/day). Their resting end-diastolic dimensions were slightly larger than group A, but their response to the Valsalva maneuver was similar to the normal volunteers (fig. 2). Figure 4 illustrates the mean derived values for systolic and diastolic volumes in this group. Systemic and right atrial mean pressures are also illustrated. Every individual in this group had a sigmoidal systolic blood pressure response to Valsalva strain with the normal release overshoot.

Derived beat-to-beat stroke volumes and simultaneous arterial and atrial pressures allowed computation of beat-to-beat peripheral vascular resistance of this group in arbitrary units. The variation in these parameters during the course of the Valsalva maneuver, expressed as percent change from resting values, is illustrated in figure 5. These interrelationships suggest that peripheral resistance is elevated

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** The mean diastolic (dark bars) and systolic (hatched bars) volumes (± 1 se) of group A subjects during the Valsalva maneuver. The mean heart rate (± 1 se) is shown at the top. Stroke volume is apparent as the difference between diastolic and systolic volumes. E, M and L refer to the early, mid and late portions of strain and recovery.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Percent change in diastolic and systolic dimensions during the Valsalva maneuver in each of the study groups. Resting dimensions in each group were used as control values. E, M and L refer to the early, mid and late portions of strain and recovery.

### TABLE 2. Mean Ventricular Dimensions and Heart Rate (± 1 se) in Study Populations

<table>
<thead>
<tr>
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<th>Rest</th>
<th>Strain</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
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<td>Early</td>
<td>Mid</td>
<td>Late</td>
</tr>
<tr>
<td><strong>A. Group A</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>75 ± 5</td>
<td>83 ± 7</td>
<td>94 ± 6*</td>
</tr>
<tr>
<td>End-diastolic dimension (cm)</td>
<td>4.6 ± 0.2</td>
<td>4.3 ± 0.2</td>
<td>3.8 ± 0.2*</td>
</tr>
<tr>
<td>End-systolic dimension (cm)</td>
<td>3.0 ± 0.2</td>
<td>2.6 ± 0.2*</td>
<td>3.8 ± 0.2*</td>
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<tr>
<td><strong>B. Group B</strong></td>
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<tr>
<td>Heart rate (beats/min)</td>
<td>50 ± 2</td>
<td>65 ± 3</td>
<td>74 ± 4*</td>
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<td>End-diastolic dimension (cm)</td>
<td>5.2 ± 0.2</td>
<td>4.8 ± 0.2*</td>
<td>4.1 ± 0.2**</td>
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<tr>
<td>End-systolic dimension (cm)</td>
<td>3.5 ± 0.2</td>
<td>3.1 ± 0.2**</td>
<td>3.8 ± 0.2**</td>
</tr>
<tr>
<td><strong>C. Group C</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>94 ± 6</td>
<td>97 ± 5</td>
<td>98 ± 6</td>
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<tr>
<td>End-diastolic dimension (cm)</td>
<td>7.1 ± 0.3</td>
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<td>End-systolic dimension (cm)</td>
<td>6.1 ± 0.3</td>
<td>6.0 ± 0.3</td>
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</table>

*P < 0.01

**P < 0.001
throughout strain because stroke volume falls proportionally more than the mean arterial-right atrial pressure difference. This elevation is most marked at end strain when stroke output reaches its lowest point. At release the normal blood pressure overshoot reflects persistent elevation of resistance at a time when stroke output is rapidly normalizing.

3. Group C — Patients with Heart Failure

The response of this group is shown in table 2C and figure 2. They had higher resting heart rates than subjects in groups A and B and abnormally large resting ventricular dimensions. In contrast to subjects with normal ventricular function, their internal ventricular dimensions did not diminish significantly even by the terminal phase of the Valsalva maneuver (fig. 2). The largest individual dimensional change (irrespective of systole or diastole) in this group was 3 mm. This value was less than the smallest dimensional change encountered in any individual in groups A or B during the Valsalva strain phase.

Figure 6 illustrates the mean derived values for left ventricular systolic and diastolic volumes in this group. In marked contrast to groups A and B there was only a small increase in heart rate (6% by end strain) and stroke output was virtually fixed. In the five subjects studied by cardiac catheterization, right atrial pressures were maintained close to 30 mm Hg throughout strain, a response similar to subjects in group B. These five patients all showed a square wave arterial pressure response to the Valsalva maneuver.

**Discussion**

This study demonstrates that echocardiography can be used to evaluate the Valsalva maneuver in a manner heretofore impossible. Because the technique is noninvasive yet allows continuous beat-to-beat recording, we have been able to study the Valsalva response in healthy subjects and patients with heart disease over a period of 40 to 50 seconds. The results depict directly the effect of acutely raising intrathoracic pressure on left ventricular dimensions.

1. Response in Normal Subjects

By the end of the strain phase, our normal subjects (group A) showed a 1.1 cm mean decrease in left ventricular diastolic dimension and an accompanying 0.9 cm decrease in end-systolic dimension. Utilizing the Teichholz formula these findings are consistent with a 40% fall in stroke volume by end strain. This falls near the middle of the 33-50% range of decrease which can be derived with alternative formulae (Appendix I). Since the major and minor axis relationships of the left ventricle have not been established under conditions of the Valsalva maneuver it can be argued that none of the formulations utilized is appropriately quantitative. Data from two invasive studies suggest that several of these echocardiographic formulae may underestimate the stroke volume decrease which occurs during the Valsalva strain phase.
2. Response in Patients with and without Heart Failure

The response of patients with normal ejection fractions (group B) was nearly identical to group A. The main difference lay in slower mean baseline heart rates and larger resting left ventricular dimensions in the patient group. The slower resting heart rate was likely influenced by propranolol therapy in half of this group. The fact that group B was all men, some of whom had underlying heart disease, probably explains the somewhat larger, though still normal resting left ventricular internal dimensions when compared to group A. Nevertheless with strain group B patients had a proportional decrease in ventricular dimensions (fig. 2) and calculated stroke volumes comparable to the group A volunteers (figs. 3, 4). Likewise there was a rapid increase of these values to baseline measurements once strain was released. An overshoot in release phase stroke volumes did not occur despite an overshoot in release phase blood pressures.

Recently Brooker et al. studied the Valsalva response of five patients with heart disease during left ventricular angiography.\(^\text{9}\) Utilizing slow intermittent bursts of contrast agent into the left ventricle during a 12–15 sec strain phase they found that stroke volume decreased from 60 ml to 27 ml. After release stroke volume overshot slightly to 70 ml, primarily because one of their five subjects had a 50% increase in stroke volume at that time. Our results are similar to the above findings, although our calculated fall in stroke volume by end strain was somewhat less striking.

Group B patients afforded the opportunity to gain further insight into the physiology of the sigmoidal strain and overshoot release Valsalva responses characteristic of individuals with normal ventricular function. Our data suggest that peripheral resistance is elevated in this group throughout strain as blood pressure falls proportionately less than stroke volume. It is possible that the left ventricle changes shape significantly under the influence of increased intrathoracic pressure so that standard volume formulae are less applicable under these conditions. Such a change, however, was not noted in the study of Brooker et al. The rapid return of ventricular dimensions and thus stroke output to baseline values upon release accompanied by an overshoot blood pressure response strongly suggests an elevated peripheral resistance at this time. We are currently pursuing an angiographic evaluation of the Valsalva maneuver to examine these points further.

Group C patients were strikingly different from groups A and B in that their ventricular dimensions were large at rest and did not change significantly even by the termination of the Valsalva strain phase. The constant ventricular dimensions imply a fixed stroke output. In five patients in whom simultaneous blood pressures were recorded there was a square wave response. In the face of a fixed stroke volume this response may directly reflect a transient rise in peripheral resistance which falls abruptly at release. Failure of stroke volume to fall after initial strain explains the lack of a sigmoidal strain blood pressure response in this group. Furthermore, upon release stroke output is still fixed rather than increasing rapidly from a relatively depressed state imposed by prolonged straining. Inability to alter stroke volume at release also suggests that this is the mechanism
for lack of overshoot in systolic pressure at the early recovery phase in these subjects with poor ventricular function.

3. Clinical Considerations

Our data suggest that the increase in intrathoracic pressure which accompanies forceful lifting, straining at urination and defecation or even spasmodic coughing can potentially cause as much as a 50% drop in left ventricular stroke output. This fall may be even more striking with upright postures. Dizziness and even syncope may ensue, particularly if vasoconstriction and compensatory tachycardia are inadequate to maintain cerebral perfusion. Indeed a striking diminution in stroke volume may be an important initiating or contributing factor to the mechanism of sudden death which sometimes occurs after straining at stool.

Angina pectoris has been referred to as relieved by the Valsalva maneuver. In our patients with normal ventricular function (group B) the later phases of straining were accompanied by a depressed arterial systolic pressure as well as decreased left ventricular volume (fig. 4). Under these conditions left ventricular wall tension is considerably diminished. Presumably in those individuals whose angina is relieved by the Valsalva maneuver the oxygen requirements of the heart are reduced significantly despite an increase in heart rate.

Our results also suggest that a fixed ventricular volume response is not an accompaniment of normal ventricular function. Thus echocardiographic evaluation using the Valsalva maneuver may be an additional aid in distinguishing patients with normal from those with impaired ventricular performance. We are currently investigating 1) whether a fixed volume response can occur in patients other than those with severely depressed ejection fractions and 2) the frequency of association of a fixed volume response with a square wave pressure response.

References


APPENDIX I. Alternative Derivations of Left Ventricular Volumes in Group A

<table>
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<th>Early (早)</th>
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<th>Late (晚)</th>
<th>Early (早)</th>
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<th>Late (晚)</th>
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<td>A. Pombo et al. 11</td>
<td>EDV = 0.962 Ud^3 + 11.53</td>
<td>ESV = 1.041 Us^3 + 0.25</td>
<td>EDV = 0.962 Ud^3 + 11.53</td>
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<td>EDV = 0.70 Ud^3 + 28.1</td>
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<td>C. Feigenbaum et al. 13</td>
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<td>EDV = 0.922 Ud^3 + 72.42</td>
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*Values in parentheses indicate % of resting value.

Abbreviations: EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; EF = ejection fraction; Us = Ultrasound systolic dimension; Ud = ultrasound diastolic dimension.
A F Parisi, J J Harrington, J Askenazi, R C Pratt and K M McIntyre

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