Pressure Flow Studies in Patients Having a Pressor Response to the Valsalva Maneuver

By Jerome Ruskin, M.D., Alexander Harley, M.B., M.R.C.P., and Joseph C. Greenfield, Jr., M.D.

SUMMARY
Blood pressure and flow were measured in the ascending aorta by means of the pressure-gradient technique in five patients with congestive heart failure manifesting a pressor response to the Valsalva maneuver. Beat-to-beat changes in stroke volume, blood pressure, pulse pressure, peak blood flow, duration of systole, duration of ejection, and heart rate were calculated. Stroke volume and blood pressure rose with the onset of straining; blood pressure rose slightly further as stroke volume fell to below control levels at the end of the straining period. Pulse pressure, peak blood flow, and duration of ejection tended to follow stroke volume. Heart rate was unchanged throughout. All parameters returned toward control after release of the strain.

Additional Indexing Words:
Pulsus alternans    Congestive heart failure    Filling pressure-cardiac output curve

The abnormal circulatory response to the Valsalva maneuver, which occurs in some patients with heart failure, has been recognized for over 20 years,[1,2] yet despite the continued interest of circulatory physiologists, its mechanism remains incompletely understood. The abnormal response is characterized either by an unchanged or an increased pulse pressure during straining, and by either a "square wave" pattern in the arterial pressure tracing, or by a fall in the diastolic blood pressure following release of the strain. There is no associated change in the heart rate. It has been suggested that the increase in pulse pressure during the straining phase in some patients may be due to an increased stroke volume.[3] However, observations of instantaneous aortic blood flow in patients manifesting this abnormal pressor response to the Valsalva maneuver have not been previously reported. The following study was undertaken to measure the beat-to-beat changes in aortic blood pressure and flow during the Valsalva maneuver in five patients with heart failure.

Method
Five adult male patients in the Durham Veterans Administration Hospital, ranging in age from 35 to 52 years (mean 45 years), were studied during the course of diagnostic cardiac catheterization. The informed consent from each patient was obtained. Four patients had clinical histories and physical findings compatible with idiopathic myocardial hypertrophy. The remaining patient was thought to manifest a myocardial infarction of undefined etiology. All patients had elevated central venous pressures, cardiomegaly, and loud third heart sounds; none was found to have significant valvular disease. All were symptomatically class III or IV according to the New York Heart Association Functional Classification.

Phasic blood flow and pressure were estimated in the ascending aorta during the Valsalva maneuver by the pressure-gradient technique.[4] This method is based on an approximate solution of the Navier-Stokes equations of fluid motion which relate the pressure gradient to the
The duration of the phase of straining ranged from 10 to 26 seconds (mean 16 sec- 
onds); none of the patients was able to main- 
tain a prolonged strain. Pertinent hemody-
namic data are summarized in Table 1 and 
figure 2. According to pressure is measured at 
the aortic root. Although the pressure difference 
is small and its accurate measurement is 
problematic, and it has been experimentally 
and clinically described. 6.5 French double- 
lobe catheter, having lateral pressure taps 
separated in 4 cm was inserted percutaneously 
through the femoral artery and advanced 
under fluoroscopic control until the tip was 
about 2 cm above the aortic valve. The 
pressure differences between the aortic 
and femoral pressure were continuously 
monitored simultaneously by a Donner Model 
3400 electronic pressure recorder. 

Table 1

Pressure and Flow Data during Valsalva Maneuver in Five Patients with Pressor Responses

<table>
<thead>
<tr>
<th>Period</th>
<th>Stroke Volume (cm³)</th>
<th>Systolic Pressure (mm Hg)</th>
<th>Diastolic Pressure (mm Hg)</th>
<th>Mean Pressure (mm Hg)</th>
<th>Pulse pressure (cm Hg)</th>
<th>Peak Flow (cm/sec)</th>
<th>Peak Velocity (cm/sec)</th>
<th>Duration Systole</th>
<th>Duration Ejection</th>
<th>Heart Rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>26 ± 3</td>
<td>110</td>
<td>83</td>
<td>92 ± 3</td>
<td>27 ± 6</td>
<td>238 ± 22</td>
<td>35 ± 6</td>
<td>0.33 ± 0.02</td>
<td>0.20 ± 0.01</td>
<td>93 ± 7</td>
</tr>
<tr>
<td>3 Beats after onset</td>
<td>33 ± 5</td>
<td>138</td>
<td>107</td>
<td>117 ± 10</td>
<td>31 ± 3</td>
<td>304 ± 48</td>
<td>49 ± 11</td>
<td>0.34 ± 0.01</td>
<td>0.21 ± 0.03</td>
<td>94 ± 6</td>
</tr>
<tr>
<td>Mid-strain</td>
<td>30 ± 3</td>
<td>138</td>
<td>109</td>
<td>118 ± 6</td>
<td>31 ± 3</td>
<td>276 ± 30</td>
<td>44 ± 8</td>
<td>0.33 ± 0.01</td>
<td>0.21 ± 0.01</td>
<td>93 ± 7</td>
</tr>
<tr>
<td>Before release</td>
<td>25 ± 3</td>
<td>139</td>
<td>115</td>
<td>123 ± 8</td>
<td>25 ± 4</td>
<td>248 ± 18</td>
<td>39 ± 6</td>
<td>0.33 ± 0.02</td>
<td>0.20 ± 0.01</td>
<td>94 ± 7</td>
</tr>
<tr>
<td>6 Beats after release</td>
<td>27 ± 3</td>
<td>110</td>
<td>82</td>
<td>91 ± 3</td>
<td>27 ± 4</td>
<td>260 ± 20</td>
<td>41 ± 6</td>
<td>0.34 ± 0.01</td>
<td>0.20 ± 0.01</td>
<td>95 ± 7</td>
</tr>
</tbody>
</table>

*Average data and standard error from five patients. Data derived from a single beat in each period listed except for one patient developing pulsus alternans during the Valsalva maneuver in whom paired beats were used.
PRESSURE FLOW STUDIES

Mean data, expressed as percent of control values, and standard error (crossed bars) for the following parameters: SV, stroke volume; pulse pressure; MAP, mean arterial pressure; peak flow; and heart rate. The periods in which the data were obtained are listed at the top of the figure.

illustrated in figure 1. Under control conditions the mean stroke volume for all five subjects was 26 cm³ se ± 3 and the simultaneously recorded mean blood pressure was 92 mm Hg se ± 3. Three beats after the onset of straining, the stroke volume rose to 33 cm³ se ± 5 and the mean arterial pressure to 117 mm Hg se ± 10. The patient with the cardiomyopathy of obscure origin demonstrated a response of an intermediate nature. In this patient although the systemic arterial pressor response was maintained throughout the phase of straining, stroke volume did not change appreciably from the control value. In the four patients in whom stroke volume increased with the onset of straining, paired data analysis revealed significant changes in both stroke volume (P < 0.01) and mean arterial pressure (P < 0.05). By mid-strain, the stroke volume had fallen to 30 cm³ se ± 3; the mean arterial blood pressure was unchanged at 118 mm Hg se ± 8. By the end of the straining period, just prior to release, stroke volume had fallen below control values (25 cm³ se ± 3), whereas mean arterial blood pressure remained elevated at 123 mm Hg se ± 8. Six beats following release, all parameters returned to control values. Duration of systole and heart rate were essentially unchanged throughout the Valsalva maneuver. Both during and after the Valsalva maneuver, peak velocity, peak flow, pulse pressure, and duration of ejection tended to follow stroke volume (table 1 and fig. 1).

Figure 2 illustrates the development of pulsus alternans in a patient manifesting an abnormal pressor response during a Valsalva maneuver. During the control period (Panel A) the stroke volume was 33 cm³, and mean blood pressure, 100 mm Hg. Pulsus alternans began following a premature ventricular beat occurring early during straining, and persisted after release. At mid-strain (Panel B) both flow and pressure were increased in both the weaker and stronger beats (34 cm³, 127 mm Hg and 38 cm³, 126 mm Hg, respectively).

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Just prior to release (Panel C) flow had fallen below control on the weaker beat (26 cm³), and to control on the stronger (33 cm³), yet the increased mean pressure (132 mm Hg) was maintained essentially unchanged in both beats. After release, stroke volume was 31 cm³ and 35 cm³ on the weaker and stronger beats, respectively, while mean pressure was slightly lower than control.

Discussion

In normal subjects performing a Valsalva maneuver, the onset of straining is accompanied by an increase in systemic arterial pressure that lasts for 6 to 8 seconds and is proportional to the rise in intrathoracic pressure. The change in blood pressure is not the result of “squeezing blood out of the heart and lungs,” as was formerly believed. Studies have shown that the stroke volume remains unchanged with the onset of straining in the normal subject. Mean arterial blood pressure, pulse pressure, and stroke volume decrease markedly during the continued strain; a secondary rise in blood pressure occurs later in the straining phase. A further fall in all three parameters is noted upon release, and is then followed by an overshoot in both blood pressure and stroke volume. Elevation of the diastolic blood pressure after release is regarded as evidence for vasoconstriction during the late portion of straining. The finding of a late rise in blood pressure during straining in the presence of a decreasing stroke volume tends to confirm this interpretation.

The mechanisms responsible for the pressor response during the Valsalva maneuver have not been defined. On the basis of an observed increase in pulse pressure during the Valsalva maneuver, Sharpey-Schafer postulated that stroke volume was increased. According to his hypothesis, the Valsalva maneuver reduces effective ventricular filling pressure thereby shortening myocardial fiber length. In the failing heart that is on the descending limb of a Starling (filling pressure-stroke volume) curve, this will augment stroke volume. It would follow that in patients in whom the stroke volume did not decrease, the ventricle was functioning on the flat portion of this curve. A second hypothesis does not require a significant change in filling pressure or myocardial fiber length. Instead, because of an already full venous bed, blood can neither be sequestered in the venous system during the straining period, nor suddenly released upon discontinuing the strain. Thus, the increase in arterial pressure simply reflects the change in intrathoracic pressure. This report documents for the first time that the rise in arterial blood pressure is accompanied by an increase in stroke volume both with the onset of straining and at midstrain. This finding is consistent with the first hypothesis, but does not altogether exclude the second.

The further late rise in mean arterial pressure in the presence of a falling stroke volume is of interest and suggests that vasoconstriction occurs, as in normal subjects, near the termination of the straining phase. However, the lack of an overshoot in blood pressure after release and the constant heart rate seem incompatible with a significant degree of vasoconstriction. A steadily rising intrathoracic pressure during the strain might account for this late rise in arterial blood pressure. In three of the five patients reported herein, oral pressure was monitored during the straining and remained constant. In the other two patients oral pressure was not monitored, but because their results were qualitatively similar, it was assumed that these patients performed the maneuver in the same way as the other three. The arterial blood pressure responses are similar to those seen in normal subjects performing a Valsalva maneuver during norepinephrine infusion, although in the latter no secondary increase in blood pressure occurs during the late portion of straining. At present there appears to be no alternative than vasoconstriction by which to explain the rise in arterial blood pressure during late straining.

A possible explanation for the fall in stroke volume near the end of straining depends upon the redistribution of blood to the extrem-
ities secondary to improved ventricular performance during the strain. When peripheral venous pressure rises sufficiently to overcome the intrathoracic venous pressure, venous return to the heart increases, and ventricular filling pressure and myocardial fiber length correspondingly increase. This causes the failing heart to fall back to the descending limb of a Starling curve, resulting in the decrease in stroke volume.

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