The Circulatory Response to the Valsalva Maneuver of Patients with Mitral Stenosis with and without Autonomic Blockade

By David G. Greene, M.D., and Ivan L. Bunnell, M.D.

Patients with clinically significant mitral stenosis have been demonstrated to differ from other subjects in their circulatory responses to the Valsalva maneuver. During forced expiration most patients with mitral stenosis are able to maintain systolic pressure in the brachial artery at a level equal to or greater than the resting value, whereas most control subjects cannot. Autonomic blockade does not affect the ability of patients with mitral stenosis to maintain systolic pressure, whereas it abolishes such ability, when it is present, in control subjects. The unique response of patients with mitral stenosis under autonomic blockade is attributed to the physiologic consequences of the volume of blood in the dilated left atrium.

INTERPRETATION OF hemodynamic data may be facilitated by comparison of observations made before and after autonomic blockade. The present study is based upon the circulatory responses to the Valsalva maneuver of patients with mitral stenosis. Analysis of the brachial arterial pressure pulses obtained during the maneuver reveals that the responses of patients with mitral stenosis differ from those of other patients, and that the differences are accentuated under autonomic blockade.

METHOD

Fifty-four patients* have been studied, among whom were 20 with rheumatic heart disease; these 20 have been classified as to clinical status and valvular involvement on the basis of history and physical examination, fluoroscopic and electrocardiographic interpretation, cardiac catheterization and the findings at surgery, when performed. Fifteen of the 20 patients were catheterized and nine underwent valvuloplasty. Three patients were discovered to have a predominantly aortic valvular lesion and 17 were discovered to have mitral disease predominantly. Of the 17 patients with mitral disease 15 have been classified as having predominantly mitral stenosis and two as having predominantly mitral insufficiency. These 17 patients have been further divided according to clinical status. All were disabled by their disease; 13 were clinically progressive, while four were stable. The data on three of the patients with mitral stenosis were obtained postoperatively. One of the three had developed a marked degree of mitral insufficiency as a result of valvuloplasty.

The control group is composed of 39 individuals. The group is heterogeneous. It includes patients with and without heart disease, as well as subjects with no known disease. The patients classified as having aortic valvular lesions or mitral insufficiency have been included among the controls for the purposes of this study, since their responses followed the normal pattern.

An indwelling needle was placed in the brachial artery and the blood pressure recorded through a Hathaway pressure recording system.³ A mouthpiece was inserted into which were connected an aneroid manometer and a pressure gage which recorded mouth pressure graphically. The subject was instructed to blow the indicator needle of the aneroid manometer to about the 40 mm. mark for 15 seconds. Whenever feasible, the same procedure was followed after autonomic blockade. Blockade was achieved by intravenous infusion of tetraethylammonium chloride at rates of 0.22 to 0.79 mg. per kilogram per minute. Records analyzed include those recorded after 6 to 50 minutes of continuous intravenous infusion at the above rates.⁴

Each subject performed more than one Valsalva maneuver. The particular record selected for tabulation was the one which was best performed, that is, in which the forced expiration was performed evenly for the full 15 seconds, and which produced a circulatory response typical for that particular patient, as judged from analysis of each of his records. Not all patients were able to achieve a mouth pressure of
40 mm. Hg. The absolute level attained did not appear to affect the type of circulatory response.

This communication will be concerned with measurements of systolic and pulse pressures and heart rate, at three points (A, B, C) in each record (fig. 1). Point A is two complete respirations during the resting period, before any forced expiration has occurred. Point B is selected as the last few beats during the period of forced expiration. Point C is represented by the second and third respirations following the cessation of forced expiration.

RESULTS

Description of the contrasting circulatory responses of the two groups to forced expiration will be arbitrarily limited to the levels of brachial artery pressure and heart rate at points B and C during the Valsalva maneuver. The response of the controls will be considered first. Figure 1 exemplifies the commonest response seen among the controls. Either with the onset of forced expiration, or shortly thereafter, systolic pressure falls below the resting value. At point B, near the end of the period of forced expiration, the majority of the controls (64 per cent) had a systolic pressure lower than the resting value, point A (table 1). The remaining 36 per cent of controls had either maintained or regained their resting systolic pressure at point B; this rise in pressure was, in some instances, a late phenomenon (fig. 2) occurring during the latter half of forced expiration, at a time when all but one of the subjects exhibited a tachycardia. All but one control subject showed a reduced pulse pressure during this period of forced expiration.

After forced expiration was ended, and the first breath was taken, brachial artery pressure fell abruptly. This was followed by a rapid rise until during the second and third respirations, point C, the systolic pressure rose above the resting value (figs. 1 and 2). This constitutes the "overshoot" phenomenon, and was seen in

<table>
<thead>
<tr>
<th>Number of Subjects</th>
<th>Without TEAC</th>
<th>With TEAC*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Col. 1 Equal to or Greater than Resting Value</td>
<td>Col. 2 Less than Resting Value</td>
</tr>
<tr>
<td>Controls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Random Patients</td>
<td>13</td>
<td>21</td>
</tr>
<tr>
<td>Aortic Lesion</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Mitral Insufficiency</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>25</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>13</td>
<td>2</td>
</tr>
</tbody>
</table>

* Only certain of those subjects in column 1 received TEAC. Columns 3 and 4 include only subjects who originally were placed in column 1.

79 per cent of control subjects (table 2). During the second and third respirations, the heart rate usually slowed. However, in 17 of 32 controls in which an accurate measure of heart rate was obtainable, it did not fall below the resting value.

Figure 3 exemplifies the circulatory response of patients with mitral stenosis to the Valsalva maneuver. As can be seen from table 1, most of
the test group (13 out of 15 subjects or 87 per cent) have a systolic pressure at the end of forced expiration equal to or greater than the resting level. At the same time, the pulse pressure was increased in 6 out of the 15 subjects. During the second and third respirations after forced expiration was terminated, 11 of 15 subjects (73 per cent) exhibited a systolic overshoot. (See table 2.) It is difficult to make any statement about heart rate in the records obtained in mitral stenosis since more than half the subjects (8 out of 15) exhibited atrial fibrillation. Of those with regular sinus rhythm, ability to regain resting systolic pressure during forced expiration (fig. 4). Six subjects with mitral stenosis received tetroethylammonium chloride. All now failed to show an overshoot. Each had previously maintained systolic pressure during forced expiration, and now despite the action of the blocking agent, all six retained this ability (fig. 5).

![Fig. 3.](image-url)

**FIG. 3.** Response of patient with mitral stenosis to the Valsalva maneuver. Systolic pressure is maintained throughout the period of forced expiration, and in this instance there is no overshoot.

![Fig. 4.](image-url)

**FIG. 4.** Response of control patient to the Valsalva maneuver during autonomic blockade. Systolic pressure does not regain resting level, and there is no overshoot.

![Fig. 5.](image-url)

**FIG. 5.** Response of patient with mitral stenosis to the Valsalva maneuver during autonomic blockade. Although the blood pressure level is lower, the form of the tracing is similar to that of the same patient before autonomic blockade taken a few minutes earlier (fig. 3). Systolic pressure is maintained throughout the period of forced expiration.

**TABLE 2.** The Occurrence of Systolic Overshoot and Bradycardia during the Second and Third Respirations after Forced Expiration

<table>
<thead>
<tr>
<th></th>
<th>Systolic Overshoot</th>
<th>Bradycardia*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>11</td>
<td>4</td>
</tr>
</tbody>
</table>

* Includes only those patients with regular sinus rhythm, whose records could be accurately timed.

only one out of seven showed a heart rate at point C slower than at point A.

After autonomic blockade had been achieved by the administration of tetroethylammonium chloride (TEAC), subjects in both groups showed generally lower systemic arterial pressure. All 19 members of the control group who had received tetroethylammonium chloride failed to demonstrate an overshoot. Of the 14 controls that had been able, before autonomic blockade, to regain resting systolic pressures at point B, six received the blocking agent tetroethylammonium chloride. All six lost this

**DISCUSSION**

The classic circulatory response to the Valsalva maneuver has been well described. The responses of the control group, both with regard to brachial artery pressure and heart rate, are consistent with the accepted normal responses. Some normals are able to maintain or regain resting systolic pressure during expiratory pressure, while others are unable to do so. The elevated systolic pressure late in the period of forced expiration was accompanied by tachycardia, suggesting that both are of reflex origin. This concept is further supported by the fact that in each instance in
which it was achieved, autonomic blockade abolished this ability to maintain or regain resting systolic pressure.

Most patients with mitral stenosis, on the other hand, would appear to be able to maintain systolic pressure without regard to reflex mechanisms. This is suggested by the fact that their systolic pressure at the end of forced expiration was equal to or greater than the resting value, whether or not autonomic blockade was induced. One is led to the conclusion that there is, in mitral stenosis, a mechanical reason for systolic pressure maintenance during forced expiration. In the control subject, forced expiration leads to increased intrathoracic pressure and a consequent reduction in venous return to the right atrium. After the first few beats, the venous return to the left atrium is also reduced, as is left ventricular filling. Without the compensatory reflex effect of tachycardia and peripheral arteriolar vasoconstriction, the reduced left ventricular filling and output inevitably lead to a fall in systemic arterial pressure as forced expiration is prolonged. In mitral stenosis, there is a large reservoir of blood in the dilated left atrium, which, trapped behind a stenotic orifice, can maintain good left ventricular filling during the 15 seconds of forced expiration. This ensures maintenance of left ventricular output and, with it, maintenance of systemic artery pressure, wholly apart from the effect of any reflexes.

It is only in the above regard that the control and mitral stenosis groups are clearly differentiated in their response. An overshoot was present in 79 per cent of control Valsalva maneuvers, and in only slightly fewer of the mitral group, 73 per cent. Tetraethylammonium chloride infusion abolished the overshoot in all subjects who received the drug in each group. Comparison of the test and control groups with regard to changes in heart rate after forced expiration is ended is obscured by two facts: (a) that more than half the test group were fibrillating, and (b) the control group shows no consistency of pattern with regard to heart rate. So although six of the seven patients with mitral stenosis with regular rhythm do not show a bradycardia at point C, neither do half the controls.

Recently Goldberg, Elisberg, and Katz have studied the circulatory responses to the Valsalva maneuver of clinically significant mitral stenosis, and have concluded that it differs from only acoustically significant mitral stenosis, and normalcy, in two regards. These are the lack of (a) the overshoot and (b) the bradycardia which normally accompanies it. The authors suggest that it may be possible to utilize the latter as a simple bedside test for clinically significant mitral stenosis. From our observations, neither of these criteria would appear to be critical in the evaluation of mitral stenosis.

In our particular series of subjects with mitral stenosis, all had lesions of sufficiently high grade to be of clinical significance. We can therefore say nothing about the circulatory response of only acoustically significant mitral stenosis.

**Summary**

Only patients with clinically significant mitral stenosis were able to maintain systolic pressure in a systemic artery during the forced expiratory phase of the Valsalva maneuver when performed under effective autonomic blockade. Resting systolic pressure was regained in certain other subjects during forced expiration when the autonomic nervous system was functionally intact, but this was not observed after blockade. The unique response of patients with mitral stenosis is attributed to the physiologic consequence of the volume of blood in the dilated left atrium.

**Sumario Español**

Ha sido demostrado que pacientes con estenosis mitral clínicamente significativa difieren de otros sujetos en el respondimiento circulatorio a la maniobra de Valsalva. Durante la expiración forzada la mayoría de los pacientes con estenosis mitral pueden mantener una presión sistólica en la arteria braquial a un nivel igual o mayor que los valores obtenidos durante reposo, mientras que la mayoría de los sujetos controles no pudieron mantener la presión a este nivel. Bloqueo autonómico no afecta la habilidad de los pacientes con estenosis mitral a mantener la presión sistólica, pero
si abole esta habilidad cuando se encuentra presente en sujetos controles. El respondimiento único en pacientes con estenosis mitral bajo bloqueo autonómico se atribuye a los resultados fisiológicos del volumen de sangre en el aurículo izquierdo dilatado.

REFERENCES


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