Circulatory Changes Produced by the Valsalva Maneuver in Normal Subjects, Patients with Mitral Stenosis, and Autonomic Nervous System Alterations

By H. D. McIntosh, M.D., J. F. Burnum, M.D., J. B. Hickam, M.D., and J. V. Warren, M.D.

The cardiac output has been measured during the immediate post-Valsalva maneuver recovery period in normal subjects, patients with clinically significant mitral stenosis, and patients with alterations of the autonomic nervous system. When compared with the resting value, it was found that normal subjects had a decrease in cardiac output during the immediate post-Valsalva recovery period, while patients with mitral stenosis had an increased output following the same stress. Three patients with alterations of the autonomic nervous system had a variable response. Changes in total peripheral vascular resistance usually were directionally opposite to changes in the cardiac output. The difficulties of attaching clinical significance to a patient’s response to the Valsalva maneuver are considered.

RECENT reports suggest that patients with cardiovascular abnormalities, especially mitral stenosis, may respond differently, when compared with normal subjects, to the stress of a Valsalva maneuver1–4 (a forced expiration with a closed glottis, after a full inspiration). It has been reported that the absence of bradycardia following the Valsalva maneuver may serve as an index for the degree of mitral stenosis.1 It has also been observed that, unlike normal subjects, patients with mitral stenosis can maintain their systolic blood pressure equal to the resting level during the forced expiratory phase of the Valsalva maneuver when performed under effective autonomic blockade.4

Early observations on the altered circulatory dynamics produced by the Valsalva maneuver have been adequately reviewed5–7; however, few actual measurements of the cardiac output and peripheral resistance during and following this maneuver have been reported. Such measurements were made in this study in normal subjects, patients with mitral stenosis, and patients with alterations of the autonomic nervous system.

MATERIALS AND METHODS

Nine patients with normal sinus rhythm and stenosis of the mitral valve were studied by the technic of cardiac catheterization.8 None of these patients had clinically significant involvement of other valves. Pulmonary “capillary” pressure9 was recorded at rest and at the end of a three-minute period of leg exercise in all but one patient (WG). With the tip of the catheter in the pulmonary artery, a resting cardiac output, usually in duplicate, was measured, utilizing the direct Fick principle. (The Fick principle is routinely used in this laboratory in diagnostic catheterizations to determine the cardiac output.) The resting cardiac output was then measured by the T-1824 dye-dilution method.10 It has been shown11 that, allowing for the level of the cardiac output, there is no distinct difference in the contour of dye curves obtained from patients with mitral disease and normal subjects.

The cardiac output was then determined by the dye-dilution method during the recovery period immediately following a Valsalva strain which was maintained with an intraoral pressure of 40 to 50 mm Hg for 22 to 30 seconds. Duplicate cardiac outputs were determined during successive post-
Valsalva periods in four patients. The dye was introduced into the catheter, the tip of which was in the pulmonary artery, one second after release of the increased intrathoracic pressure and the systemic arterial pressure was recorded by an indwelling needle simultaneously with the determination of the cardiac outputs in all but two patients (MB and EB). In these two patients the arterial pressure was recorded under apparently the same conditions before or after the output determination.

The cardiac output was also determined by the dye-dilution technic during the period of sustained increased intrathoracic pressure in one of the above (MB) and two patients with mitral stenosis. In this phase of the study the dye was introduced into the catheter, the tip being in the pulmonary artery, two seconds after commencing the strain of a Valsalva maneuver.

Similar studies, with the following modifications, were carried out on eight normal subjects and three patients with alterations of the autonomic nervous system. In these 11 subjects, the catheter was passed only to the superior vena cava and cardiac outputs were determined only by the dye-dilution method. The increased intrathoracic pressure (40 to 50 mm. Hg) was maintained somewhat longer (average 34 seconds, range 25 to 73 seconds) in this group than by the patients with mitral stenosis.

The arterial pressure and pulse rate changes produced by the Valsalva maneuver were recorded by an indwelling arterial needle over 300 times in a heterogeneous group of 80 subjects. These records were analyzed in the four phases suggested by Hamilton:12 phase 1, the initial rise of blood pressure following the onset of increased intrathoracic pressure; phase 2, the period of sustained strain; phase 3, the period of release of the intrathoracic pressure; phase 4, the recovery period (fig. 1). All studies were done with the subject in the recumbent position.

Oxygen consumption was measured by a Pauling oxygen analyzer. Blood samples were analyzed for oxygen content by the method of Hickam and Frayser.13 The optical density of the dye samples was read against a serum blank on a Coleman Junior spectrophotometer at a wave length of 620 mu.14 At least four or more points were obtained for plotting the downward slope of the post-Valsalva dye-dilution curves in all subjects.

Arterial and venous pressures were measured by a Sanborn electromanometer or a suitable Statham strain gage and recorded on a four-channel direct-writing Sanborn polysoiellograph.

The area of the mitral valve was calculated by the formula of Gorlin.14 Total peripheral resistance was calculated:

\[
\text{Mean arterial B.P.} - 0 \\
\text{Cardiac output cc/sec.} \times 1332 \text{ dynes seconds cm.}^{-5}
\]

The arterial pressure for this determination was measured at the mean circulation time obtained from the dye-dilution curve. The mean arterial pressure was determined by the addition of the diastolic pressure and one-third the pulse pressure. The vasopressor response to the Valsalva maneuver was graded 0 to 4 by the method of Wilkins.15

Observations and Results

The values for the cardiac index and total peripheral resistance are integrated determinations. This is particularly true during the recovery phase following the Valsalva maneuver when the pulse rate and blood pressure varied from stroke to stroke. The arterial pressure used for calculating the total peripheral resistance was the pressure at the mean circulation time of the dye-dilution curve despite the fact that the peak overshoot of blood pressure might have occurred some seconds earlier. The determination of total peripheral resistance, therefore, does not reflect instantaneous changes in resistance occurring immediately after release of the strain.

Normal Subjects. Primary attention in this study was focused on phase 4 of the Valsalva maneuver (fig. 1), which was compared with the previous resting state. This phase occurred after the release of the strain when in most instances the systolic and diastolic pressure rose rapidly, the former more than the latter, so that both the mean arterial and pulse pressures were elevated above resting levels. The maximal rise in systolic pressure usually, but not invariably, occurred within three to four seconds. The magnitude of the vasopressor response did not appear related to the duration of the strain, provided it exceeded seven seconds, or the magnitude of the increase of the intrathoracic pressure, provided it exceeded 30 mm. Hg. The vasopressor responses in the normal subjects are recorded in table 1. Slight or no vasopressor response was also observed in a number of other subjects with no obvious cardiovascular abnormality who were not studied in detail. The response of these subjects was similar to that reported by other investigators.4

The rise in systolic pressure was usually followed within 15 seconds or less by a brady-
**Table 1.—Physiologic Data of Normal Subjects, Patients with Mitral Stenosis and Alterations of the Autonomic Nervous System at Rest and during the Post-Valsalva Recovery Period**

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<th>Mean Circulation Time sec.</th>
<th>Mean Arterial Pressure at Overload mm. Hg</th>
<th>Vasopressor Response</th>
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**Normal Subjects**

**Patients with Mitral Stenosis**

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* Determined by the dye-dilution method.
† Six-second period: (a) Prior to the Valsalva maneuver. (b) Maximal period of bradycardia within 15 seconds of the maximal rise of systolic pressure following the release of the strain.
‡ Determined at the mean circulation time of the dye-dilution curve.
§ According to the method of Wilkins.¹⁵
¶ Only normal subject not a medical student.

Cardiac. To be considered significant the pulse rate had to be reduced 10 per cent of the resting rate for a six-second interval within 15 seconds of maximal systolic overshoot. No bradycardia was observed without at least a grade 1 vasopressor response. However, it was not uncommon to observe grade 4 vasopressor responses without a significant bradycardia; nor
was it uncommon to observe marked bradycardia following grade 1 or 2 vasopressor responses.

Table 1 shows the cardiac index and total peripheral resistance as determined in eight normal male subjects during the resting and immediate post-Valsalva recovery period. When compared with the resting cardiac index, the index during the immediate post-Valsalva recovery period was decreased an average of 12 per cent (range +8 to −34 per cent). In no subject was there a significant increase of the cardiac output following the Valsalva. No correlation between the degree of the vasopressor response, the magnitude of the increased intrathoracic pressure or the duration of the strain and the resultant change of cardiac output was apparent. In five of the eight subjects in whom the cardiac index was decreased 20 per cent or more following the Valsalva maneuver, the total peripheral resistance was increased 38 per cent or more (table 1).

Patients with Mitral Stenosis. Pertinent data on the circulatory dynamics of the patients with mitral stenosis are contained in table 2. Eight of the 11 patients had calculated valve areas of 1.1 sq. cm. or less, and all but three of the group subsequently had a mitral valvotomy. The valve areas estimated at surgery are also recorded in table 2.

During phase 4 of the Valsalva maneuver, patients with mitral stenosis, usually had an increased systolic and diastolic pressure when compared with the resting level. The former increased more than the latter so that there was an increase in both the pulse and mean pressure. However, as has been previously observed, the patients with mitral stenosis attained the maximal rise of systolic pressure somewhat later than did normal subjects (figs. 1 and 2). Whereas normal subjects had the maximal vasopressor response within four seconds following the release of the strain, patients with mitral stenosis usually required six to eight seconds to reach the peak of the vasopressor response. The vasopressor response of these patients is recorded in table 1.

* Performed by W. C. Sealy, M.D., and J. P. Collins, M.D.
Patients with mitral stenosis frequently also had a bradycardia following the maximal rise of systolic pressure. Seven of the 11 cases in this study had a pulse rate during phase 4 of 10 to 23 per cent less than the resting level in one or all of a number of Valsalva maneuvers. This slowing of the pulse was frequently of short duration (at least six seconds) and, like the maximal rise in systolic pressure, did not always occur as promptly as did the bradycardia in normal subjects. However, to be considered significant, the bradycardia had to occur within 15 seconds of the maximal rise of systolic pressure. This bradycardia was frequently preceded by a relative tachycardia. It should be noted that the maximal vasopressor and bradycardic response for a given patient to a Valsalva maneuver is not necessarily recorded in table 1. The recorded vasopressor responses accompanied the reported cardiac output. A significant bradycardia following the Valsalva maneuver was also observed in three additional patients subjected to mitral valvotomy.

Table 1 shows the cardiac index and total peripheral resistance measured during the recovery phase of the Valsalva maneuver in nine patients with mitral stenosis. The cardiac index during this recovery period, when compared with the resting value, increased an average of 12 per cent (range +35 to −23 per cent). In only one patient was the index during the recovery phase considerably decreased below the resting level (JC, −19 and −23 per cent). Of five patients who, following the Valsalva maneuver, had a cardiac output greater than 16 per cent of the resting output, only one (AF), had a significant increase of the total peripheral resistance (+34 per cent). The total peripheral vascular resistance was unchanged or increased in the other four patients who had no change or a decrease in the cardiac output during this period.

The cardiac output was also measured during the period of increased intrathoracic pressure in three patients with mitral stenosis. During this period it was decreased 25, 52 and 65 per cent, respectively.

Patients with Autonomic Nervous System Alterations. The etiologic mechanism of the

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<td>FW</td>
<td>R</td>
<td>83</td>
<td>24</td>
<td>6.0</td>
<td>1.6</td>
<td>35</td>
<td>0.81</td>
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<td>E</td>
<td>100</td>
<td>37</td>
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</tbody>
</table>

* Surgeon could not estimate because of calcification.
† Not operated.
‡ Fick principle.
§ Determined by the Gorlin formula.14
R. Resting. E. Exercise.

Defect in the autonomic nervous system varied in these three patients. AM had persistent symptoms of postural hypotension following a lumbar sympathectomy four years prior to this study. MCB had diabetes mellitus and associated postural hypotension with a fixed pulse rate. Both of these patients were incapacitated by their postural symptoms. VB had a lumbodorsal sympathectomy for severe essential hypertension eight weeks prior to this study. She had no significant postural symptoms.

During phase 4 of the Valsalva maneuver these patients did not have an overshoot of
Fig. 2. Four tracings recorded from the same patient with mitral stenosis under similar resting conditions. The mitral valve in this patient was subsequently estimated at operation to be 0.4 sq. cm. The presence of a bradycardia in phase 4 does not appear to be related to the duration of the strain. Contrary to most patients with mitral stenosis in this series, the cardiac output was 23 percent below the resting level. Note the delay in reaching the maximal rise in systolic pressure following the release of the strain, as compared with normal subjects (fig. 1).

Fig. 3. The absence of the arterial overshoot following the release of the strain and a prolonged recovery period is associated with alterations of the autonomic nervous system.
systolic or diastolic pressure (fig. 3). The time required for the systemic blood pressure to return to the previous resting levels was prolonged. Both the systolic and diastolic pressures were below the resting level for three minutes following the release of the strain in one patient (AM). MCB had a fixed pulse rate which persisted during the recovery period. The other two patients had an increase in pulse rate during this period.

Table 1 shows the changes in the cardiac output and total peripheral resistance measured during the recovery phase of the Valsalva maneuver. Two patients had increased outputs during this period of 17 and 45 per cent, respectively. The patient with a fixed pulse rate (MCB) had a decreased output on two occasions of 19 and 33 per cent. An increase of total peripheral resistance during the recovery period was not observed in any patient.

Comment. The duration of phase 1 (fig. 1), indicating the initial maximal rise of the systolic and diastolic pressure in response to the increase of the intrathoracic pressure, varied from 1 to 5 pulse beats. The rapidity of the onset of this increase in arterial pressure was directly related to the speed of attaining the greatest rise of intrathoracic pressure. A sudden increase in intrathoracic pressure, as in a cough which was sustained, caused the maximal rise in both the systolic and diastolic pressure within one beat. If the intrathoracic pressure was increased slowly, phase 1 was more prolonged. This response was uniformly observed in all types of subjects.

**Discussion**

It has been generally accepted that when the intrathoracic pressure is increased by straining against the closed glottis or blowing against a column of mercury, the venous return to the heart is slowed and the cardiac output is considerably reduced.\(^1\), \(^7\), \(^12\), \(^16\) An obstruction to venous return produced by the Valsalva maneuver at the level of the first rib has recently been conclusively demonstrated\(^17\) by venograms.

Using radiographic technics, Natvig\(^18\) concluded that the volume of the heart decreased approximately 200 cc. during the strain. Utilizing the dye-dilution technic, three patients with mitral stenosis had an average reduction in cardiac output of 47 per cent (table 3). A similar reduction by this technic was observed in patients without mitral stenosis.\(^19\)

Following the release of the strain, it has been assumed\(^1\), \(^12\), \(^21\) that the blood previously dammed in the venous system was immediately presented to the right ventricle and resulted in a rapid increase in cardiac output. Hamilton\(^12\) stated that the blood dammed back in the extrathoracic reservoirs made its way through the lesser circulation and produced a maximal effect on the arterial pressure in four seconds. Few actual measurements of the cardiac output during this recovery period are available in the literature. Otis and co-workers\(^20\) reported observations on two subjects in whom the stroke volume, measured by the ballistocardiograph, decreased following the release of the strain, but the pulse rate increased so that the cardiac output increased 119 and 158 per cent above the resting output. Kay and associates,\(^21\) utilizing electrokytomographic technics, observed that 8 of 10 healthy subjects in the sitting position (the subjects in the present study were recumbent) had increased aortic pulsations within four beats after the release of the strain. These increased pulsations were interpreted as indicating an increase in cardiac output. Venhofen,\(^22\) using the pulse velocity method of Broemser and Ranke, reported the cardiac output in three of four patients to be decreased during the Valsalva recovery period 31, 41 and 31 per cent, respectively, when compared with resting values. Wilkins and Culbertson\(^19\) found that the cardiac output measured by the ballistocardiograph, when compared with the resting output, was decreased in the period immediately following termination of the Valsalva maneuver in patients with hypertensive vascular disease.

Figure 4 shows that the normal subjects in this study had a decrease or no change in cardiac output following a period of increased intrathoracic pressure when compared with the resting value; the patients with mitral stenosis, however, with one exception (JC),
had an increase or no change in cardiac output in response to the same stimuli.

Insufficient cardiac filling was not the cause of the decreased cardiac output in normal subjects during phase 4 of the Valsalva maneuver, for, as seen in figure 5, the central venous pressure remained elevated for a considerable time after the release of the strain. A decrease in cardiac output with an elevated right auricular pressure has previously been observed with other alterations of hemodynamic equilibrium.23

A bradycardia was not observed in any subject in this study without an associated increase in arterial pressure, although a marked increase in arterial pressure in phase 4 was not always accompanied by bradycardia. The bradycardia following the release of the increased intrathoracic pressure is similar to that observed following occlusion of an arteriovenous fistula.24 In the latter condition, the slowing of the pulse is also associated with a rise in arterial pressure. The bradycardia following occlusion of an arteriovenous fistula may be abolished by atropine, despite the fact that the increase in arterial pressure is greater than that observed before administration of the drug. Atropine also blocks the bradycardia following the Valsalva maneuver despite an even greater overshoot of arterial pressure than observed without the medica-

![Figure 4](image1.png)

**Fig. 4.** Change in cardiac index following the Valsalva maneuver. Subjects with low resting cardiac indexes tend to have an increased cardiac output following the Valsalva maneuver while those with high or normal resting indexes tend to have a decreased output following the same stress. The small circles indicate duplicate determinations of the cardiac index.

![Figure 5](image2.png)

**Fig. 5.** Elevation of central venous pressure following Valsalva maneuver. The arrows indicate the return of the arterial pressure to normal following the Valsalva maneuver.

As the cardiac output may be reduced in normal subjects in the immediate post-Valsalva recovery period, it is of interest that following the occlusion of an arteriovenous fistula, in subjects without evidence of heart failure, there is an immediate decrease in the cardiac output due to a decreased stroke volume.24 Atropine does not prevent this decreased stroke volume. It was thought that the decrease in stroke volume was due to variations in diastolic relaxation of the ventricle or to variations in the completeness of systolic emptying.

Examination of table 1 shows that in general, when compared with resting values, the subjects who had an increase of the total peripheral resistance during the post-Valsalva recovery period, usually had a decrease in cardiac output. Those who had a decrease in total peripheral resistance during this period had an increase in cardiac output. All subjects, however, usually had an increase in arterial pressure during this period.

The relationship between the cardiac output and peripheral vascular resistance in determining the degree of response to the Valsalva is further demonstrated by three patients with alterations of the autonomic nervous system. None had an increase in peripheral resistance in response to the stress of a Valsalva maneuver. Two of these patients had an increase in cardiac output and pulse rate during this period (table 1). However, despite a 45 per cent increase in cardiac output, the arterial pressure in one patient (AM) was below the
resting level. The third patient (MCB) had a decrease in cardiac output; however, he was further complicated by having a fixed pulse rate which did not vary during any phase of the Valsalva maneuver or in response to other stimuli which would usually alter the heart rate. His arterial pressure was considerably below the resting value.

Goldberg, Elisberg, and Katz have stated that patients with significant mitral stenosis do not have an overshoot of arterial pressure nor bradycardia following the release of a Valsalva maneuver. However, in this study and that of Greene and Bunnell, a significant overshoot of arterial pressure and associated bradycardia was frequently observed in such patients. Goldberg and associates felt that the overshoot of pressure in normal subjects was due to the rapid increase of cardiac output following the release of the strain and that the stenosed mitral valve prevented such an increase. The present study does not support such an opinion.

It was indeed surprising to observe that the cardiac output in patients with significant mitral stenosis, following a period of increased intrathoracic pressure, not only equaled but frequently exceeded the resting output. Flow through a stenosed mitral valve is dependent on the pressure gradient across that valve. An increase in flow through the valve may be produced by an increase of left auricular pressure, or a decrease in left ventricular end diastolic pressure. Goldberg and associates pointed out that patients with mitral stenosis reach the maximal rise of systolic overshoot following a Valsalva procedure slower than normal subjects. Such a delay may reflect a period in which the left auricular pressure is increasing. The increased left auricular pressure could thus increase the pressure gradient across the stenosed mitral valve and result in an increased cardiac output.

Further discussion as to the reason that patients with mitral stenosis frequently had an increased output following the stress of a Valsalva maneuver while normal subjects tended to have a decreased output would be purely speculative. The data obtained in this study simply permits one to state that following the Valsalva maneuver, the response observed in the arterial tracing is due to an interplay of cardiac output, total peripheral vascular resistance, and other at present poorly understood hemodynamic mechanisms. Alteration in venous tone has been observed in the isolated vein segment during this maneuver. Some subjects have been observed to have an increase over the resting forearm blood flow, as measured by the plethysmograph, when the cardiac output was actually decreased. Greene and Bunnell have observed with tetraethylammonium chloride (TEAC) a difference between normal subjects and patients with mitral stenosis during phase 2 of the maneuver. These and other variables may alter in importance from patient to patient and in the same patient from time to time.

The complexity and multiplicity of changes occurring during and following the period of increased intrathoracic pressure are apparent. Without simultaneous precise measurements of these variables, an interpretation, especially a clinical interpretation, of the phenomena producing the changes observed in an arterial tracing during and following a Valsalva maneuver should be made with caution.

Summary

1. Circulatory changes produced by the Valsalva maneuver have been investigated in normal subjects, in patients with mitral stenosis, and in patients with alterations of the autonomic nervous system.

2. Compared with resting values, the cardiac output as measured by the dye-dilution technic tended to be decreased during the post-Valsalva recovery period in normal subjects but increased in patients with mitral stenosis. Two patients with autonomic nervous system alterations had increased outputs and one had a decreased output during this period.

3. The total peripheral vascular resistance tended to change inversely to the cardiac output during this recovery period.

4. It is suggested that caution should be exercised in attaching clinical significance to the response to a Valsalva maneuver.

SUMARIO ESPAÑOL

1. Los cambios circulatorios producidos por la maniobra de Valsalva han sido investigados...
en sujetos normales, en pacientes con estenosis mitral, y en pacientes con alteraciones del sistema autónomo nervioso.

2. Comparado con los valores durante el reposo, la producción cardíaca medida por la técnica de dilución de tinte tendió a disminuir durante el período de recobro post-Valsalva en sujetos normales pero aumentó en los pacientes con estenosis mitral. Dos pacientes con alteraciones del sistema nervioso autónomo tuvieron producciones aumentadas y uno tuvo una producción disminuida durante este período.

3. La resistencia periférica vascular total tendió a cambiar inversamente con la producción cardíaca durante el período de recobro.

4. Se sugiere que se ejerza cautela en asignar significado clínico a la respuesta a la maniobra de Valsalva.

REFERENCES
19 Doyle, J. T.: Personal communication.
26 McIntosh, H. D.: Unpublished observations.
Circulatory Changes Produced by the Valsalva Maneuver in Normal Subjects, Patients with Mitral Stenosis, and Autonomic Nervous System Alterations
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