The Emphysema Response to Forced Straining (Valsalva’s Maneuver)

By Harold Mills, M.D., and Albert A. Kattus, Jr., M.D.

Changes in arterial pressure were recorded during and after forced straining (Valsalva’s maneuver) in 29 patients with emphysema. The response in these patients differed greatly from that found in normal subjects and in patients with cardiac disease. Additional physiologic data did not serve to define the mechanisms of this unique response in emphysema to forced straining.

Recently there has been a renewed interest in the physiologic effects of forced straining (Valsalva’s maneuver). Particular emphasis has been placed on the blood pressure changes during and immediately after forced straining in an attempt to define more accurately the diagnosis of certain circulatory abnormalities, particularly hemodynamically significant mitral stenosis and the presence of congestive failure.1-7 Little attention has been given to the manner in which pulmonary disease modifies the response to straining. The purpose of this paper is to present data obtained from observing circulatory changes during and immediately after Valsalva’s maneuver in patients with pulmonary emphysema and to contrast these changes with responses in patients with normal and diseased hearts. The “emphysema response” is unique and to our knowledge has not been described except in our recent preliminary report.8

Historic Background

In an excellent historical review Dawson8 summarized the early studies on the use of forced straining for investigation of various cardiovascular problems. Valsalva (1666–1723) is credited with the earliest description of the effect of forced straining on the circulation. In 1850, Weber described the effect of “chest compression” and noted syncope and convulsions after a particularly vigorous effort on his part. Approximately 35 years ago, Flack and Burton10 utilized the Valsalva test for appraising physical fitness in Royal Air Force officers. In 1936, Hamilton and co-workers11 divided the response to forced straining into 4 phases, a division subsequently utilized by others2 in the diagnosis of “dynamically significant” mitral stenosis: (I) During the initial straining period a rise in the systolic and diastolic blood pressures; (II) as straining continues a gradual fall in systolic, diastolic, and pulse pressures; (III) immediately after cessation of straining a marked drop in systolic and diastolic pressures; (IV) within a few beats an increase in systolic and diastolic pressures to greater than control levels immediately followed by bradycardia.

Particular attention has been given to the pressure overshoot and bradycardia occurring in phase IV. Numerous studies have pointed out that an intact autonomic nervous system is needed for the overshoot and bradycardia, for if autonomic function is abolished by organic disease or drugs, these responses do not occur.4, 5, 12-14 The overshoot has been attributed to blood entering a constricted arterial system after having been dammed back on the venous side.18 McIntosh and co-workers5 thought that the response is secondary to the interplay of cardiac output, total peripheral vascular resistance, and other poorly understood mechanisms. The bradycardia will never occur without the systolic overshoot, but a marked overshoot without significant bradycardia may occur. Bjork and associates15 recorded a fall in left atrial pressure during straining. However, Yu and co-workers3 noted that pulmonary capillary and pulmonary arterial pressures rose considerably at the onset of straining and then were maintained at approximately twice the...
control value. In general, the pulmonary artery pressure rose to a greater extent than the pulmonary capillary pressure with no differences discernible between subjects with normal or high pulmonary capillary pressures. Lee and associates\textsuperscript{15} have demonstrated that the effect of Valsalva's maneuver upon pulmonary artery pressure may be accounted for by the increased intrathoracic pressure during straining and by the post-straining increase in cardiac output.

The duration of straining is not important provided it exceeds 7 seconds with an intrathoracic pressure of 30 mm Hg or greater.\textsuperscript{5} Intraoral pressure may give a falsely high measure of intrapleural pressure due to the tension of the distended cheeks.\textsuperscript{17}

Elisberg's group\textsuperscript{1-2} have utilized the absence of a systolic overshoot and bradycardia in phase IV as a test for "dynamically significant" mitral stenosis although the validity of this method has been questioned.\textsuperscript{6, 18} Greene and Bunnell\textsuperscript{4} have emphasized that patients with mitral stenosis maintain their systolic pressures throughout the period of straining. They
attributed this phenomenon to the large reservoir of blood in the left atrium of these patients and were unable to abolish this response with tetraethyl ammonium chloride. Sharpey-Schafer and Knowles and co-workers have utilized the Valsalva maneuver for detecting pulmonary congestion of varying etiology. They noted that persons with left heart failure and pulmonary congestion maintain a persistently elevated arterial pressure during straining and have no post-straining overshoot or bradycardia. This failure was attributed to an elevated right ventricular diastolic pressure and peripheral venous pressure that prevented the usual obstruction of venous return caused by forced straining in normal subjects. Recently the observations of Sharpey-Schafer and Knowles and associates have been confirmed, although false negative and false positive responses were noted.

METHODS

All subjects were studied while supine and fasting. The majority were given a mild sedative prior to the procedures. All of the normal subjects and those with emphysema were adult males. The miscellaneous cardiac group included 78 adult males and 2 adult females. A no.-17 needle was placed within the brachial artery and after good pulsatile flow was established, a stiff 24-inch (3 mm. internal diameter) polyethylene tube was attached to the needle and to a Statham pressure transducer. The arterial pressures were recorded on a Sanborn Twinviso direct-writing recorder. Each subject was directed to blow into a mouthpiece attached to a mercury manometer for visual recording of straining pressure. In most cases the manometer was also connected through a strain gage to the Sanborn apparatus in order to record the pressure and duration of straining. All subjects maintained forced straining for at least 10 seconds at a pressure of 30 to 40 mm. Hg. The terminal straining systolic and diastolic blood pressures were measured for the cardiac cycles immediately prior to the cessation of straining. In most subjects spirometric measurements were made in conjunction with these procedures and in all cardiac outputs were determined by the indicator-dilution technic. Pulmonary blood volume was estimated by the method of Newman employing the down slope of the dye-dilution curve. All patients in the group with emphysema had unequivocal clinical and laboratory evidence of the disease. The miscellaneous cardiac group included many forms of acquired and a few cases of adult congenital heart disease. Most, but not all, were compensated at the time of study.

RESULTS

Data were obtained from 9 normal subjects, 29 patients with pulmonary emphysema, and 80 patients with miscellaneous cardiac diseases. Figures 1 and 2 illustrate the 2 striking characteristics of the response to forced straining in emphysema: (1) marked narrowing of the terminal straining pulse pressure; and (2) marked prolongation of the time required for the post-straining systolic pressure to return to the control value. In table 1 the mean values relevant to these 2 characteristics are summarized for the 3 groups. Many of the patients with emphysema had virtually complete obliteration of the peripheral pulse during performance of the Valsalva maneuver. Figures 3 and 4 depict the types of responses to the Valsalva maneuver obtained from normal subjects and patients with severe heart disease.

Table 2 summarizes physiologic data obtained on the patients with emphysema in conjunction with the Valsalva test. The average hematocrit, venous pressure, and circulation time are in the high normal range. The severity of the pulmonary disease is indicated by a decrease in arterial oxygen saturation and reduction in the 3-second, vital capacity. Although the mean cardiac index for the entire

<table>
<thead>
<tr>
<th>Table 1.—The Effect of Forced Straining</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>No. of cases</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Emphysema</td>
</tr>
<tr>
<td>Miscellaneous cardiac</td>
</tr>
</tbody>
</table>

* pp = pulse pressure.
emphysema group is within normal limits, the 12 patients who had the most marked "emphysema responses" were those who had low cardiac outputs and low pulmonary blood volumes. Venous pressure and the degree of arterial oxygen unsaturation did not appear to be important as determinants of the intensity of the emphysema response.

Table 3 correlates cardiac index with the time required for the post-straining systolic pressure to return to baseline. The lower the cardiac index, the longer is the time required for the return of systolic pressure to control level. Furthermore, 17 of 23 subjects with cardiac indices greater than 2.5 L./M.² had distinct post-straining overshoots while only 1 of the 6 with cardiac indices below this level had a distinct overshoot.

**DISCUSSION**

The emphysema response to the Valsalva maneuver appears to be simply an exaggerated form of the normal response. It consists mainly of a sharp diminution of the cardiac output, in some cases almost to the vanishing point. A similar response can be brought about in the normal individual if the straining is forced to sufficiently high pressure. We have seen it in a few normal individuals who were able to force
EMPHYSEMA RESPONSE TO FORCED STRAINING

TABLE 2.—Physiologic Data Obtained on Twenty-nine Patients with Pulmonary Emphysema

<table>
<thead>
<tr>
<th>No.</th>
<th>P.P.s</th>
<th>P.P.c ( \times 100^* )</th>
<th>Time required for post-straining systolic pressure to reach control (sec.)</th>
<th>Hematocrit</th>
<th>Arterial O₂ sat. (%L.)</th>
<th>V.P.(^t) (mm. H₂O)</th>
<th>Circulation time(^†) (sec.)</th>
<th>C.I.(^†)</th>
<th>SIVI</th>
<th>V.C.(^†)</th>
<th>Mean (\times 100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients in whom terminal strain pressure &lt; 15 per cent of control.</td>
<td>12</td>
<td>8.8</td>
<td>13.8</td>
<td>47.8</td>
<td>88.9</td>
<td>119.6</td>
<td>17.9</td>
<td>2.58</td>
<td>.402</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Patients in whom terminal strain pressure &gt; 15 per cent of control.</td>
<td>17</td>
<td>34.7</td>
<td>9.9</td>
<td>47.1(^*)</td>
<td>87.4</td>
<td>102.2</td>
<td>16.5(^*)</td>
<td>3.32</td>
<td>.585</td>
<td>64.6(^b)</td>
<td></td>
</tr>
<tr>
<td>Mean (\times 100)</td>
<td>20</td>
<td>24.0</td>
<td>11.5</td>
<td>47.4</td>
<td>88.1</td>
<td>105.3</td>
<td>17.1</td>
<td>3.01</td>
<td>.495</td>
<td>64.8</td>
<td></td>
</tr>
</tbody>
</table>

\(*\) P.P.s. \(\times 100\) = Terminal strain pressure — Control pulse pressure \(\times 100\).
\(^†\) V.P.—Venous pressure
\(^†\) Circulation time—Time required for indicator to appear at sampling site in brachial artery after injection into brachial vein.

§ C.I.—Cardiac index = Cardiac output

Body surface area

|| SIVI—Slope volume index = Slope volume

Body surface area

\(^*\) 3 sec. vital capacity, per cent of normal.
\(^b\) 15 cases only

intrathoracic pressure above 60 mm. Hg. Dawson\(^*\) reported that Weber succeeded in producing syncope and convulsions in himself, and he also gave an account of prisoners of ancient times using the same method to avoid torture.

Our data do not permit a definitive explanation of the exaggerated response in pulmonary emphysema, but one may speculate on several possible mechanisms. Failure of the cardiac output due to increased intrathoracic pressure may be caused by impingement of this pressure at several different sites. These might be the ventricular or atrial walls, the pulmonary vasculature, or the venae cavae.

High intrathoracic pressure could restrict the motion of the ventricles as in cardiac tamponade, or it might collapse the thin atrial walls. Neither of these 2 possibilities appears likely, since the pulse obliteration requires several seconds to reach a maximum after the pressure is applied. One would expect that if this mechanism were to operate, it would occur as soon as the pressure was applied.

It is possible that exertion of high pressure on the pulmonary blood vessels would narrow them to such an extent that the right ventricle would be unable to generate sufficient pressure to propel blood through them. This should cause acute dilatation of the right ventricle. We have no data on this point, but evidence bearing on the problem might be obtained by observing emphysema patients under fluoroscopy during the Valsalva maneuver.

Damming back the venous return to the right ventricle is known to be a major factor in the genesis of the normal response to forced straining. This is partially overcome by a rise of venous pressure above the intrathoracic pressure. It is possible that the veins of the emphysema patient are less reactive and less able to increase their pressures in response to the challenge of the Valsalva maneuver than are the veins of the normal subject. It is also
conceivable that barrel chest deformities in emphysema may expose more serosal surface area of the vena cava to the high intrathoracic pressure and thereby make it more susceptible to collapse.

Although the mechanisms leading to pulse obliteration remain obscure, our data provide some evidence to explain the delay in return to normal pressure following straining in the patient with emphysema. The rapid but not instantaneous decline in pulse pressure during straining and the slow recovery to control levels after release suggest that the lungs are probably emptied of their blood content more or less completely while pressure is being exerted. On release the right ventricle must first pump enough blood to fill the pulmonary vessels before enough can be delivered to the left ventricle to re-establish normal peripheral pulse pressure. It is significant that the cases showing the greatest delay in recovery were the ones with the lowest cardiac outputs and pulmonary blood volumes. It may be that those with higher pulmonary blood volumes were more resistant to having their lungs emptied of blood.

The post-straining overshoot was absent in several of the patients, particularly in the group with low cardiac output. Absence of the post-straining overshoot has been described as characteristic of congestive heart failure.\(^3\)\(^,\)\(^9\) In our cases, subclinical failure may have been present; however, the straining portion of our curves with a striking fall-off is entirely different from that observed in congestive failure in which there is a sustained rise in systolic pressure.

The mechanisms responsible for the emphysema response undoubtedly also account for the susceptibility of these patients to cough syncope, for coughing is but a series of Valsalva maneuvers.\(^2\)

**Summary**

In the majority of patients with pulmonary emphysema the forced straining of the Valsalva maneuver leads to a unique circulatory response.

This “emphysema response” has 2 characteristics: marked diminution in terminal straining pulse pressure frequently to the point of obliteration, and marked delay in the return of post-straining systolic pressure to the control level.

The emphysema response appears to be an exaggeration of the normal response to the Valsalva maneuver and is most marked in those patients with emphysema who have low cardiac output and small pulmonary blood volume.

**Summario in Interlingua**

In le majoritate de patientes con emphysema pulmonar le efforto del experimento de Valsalva produce un responsa circulatori de character unic.

Iste “responsa emphysematic” ha 2 characteristicas: marcate diminution—frequentemente al puncto de su obliteration—del pression del pulso al fin del efforto; e marcate retardation, post le effortio, in le retorno del pression systolic al nivello de controlo.

Le responsa emphysematic pare esser un exaggeration del responsa normal al experimento de Valsalva. Illo es le plus marcate in ille patientes con emphysema qui ha basse rendimento cardiac e parve volumine sanguinei pulmonar.

**Acknowledgment**

We are grateful to Drs. Allen Leslie and Daniel Simmons for performing the pulmonary function tests. Mrs. Mabel Pearson and Mrs. Edna O’Connor rendered invaluable technical assistance.

**REFERENCES**


10 Flack, M., and Burton, H. L.: An investigation into the physiological significance of the “40 mm. mercury test.” J. Physiol. 56: 1, 1922.


The Emphysema Response to Forced Straining (Valsalva's Maneuver)
HAROLD MILLS and ALBERT A. KATTUS, JR.

Circulation. 1958;17:65-71
doi: 10.1161/01.CIR.17.1.65
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1958 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/17/1/65

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/