Significance of Abnormal Phase II Response to Valsalva Maneuver in Cardiac Patients

By ROBERT W. BURROUGHS, M.D., AND ROBERT A. BRUCE, M.D.

Abnormal responses to a standardized Valsalva maneuver have been proposed as additional criteria for heart failure. The present study was undertaken to determine the variability of these responses in normal subjects as well as frequency of abnormal responses in cardiac patients. Only phase II responses could be considered reliable for this purpose. Discrepancies between phase II responses were observed in relation to other criteria.

VARIOUS effects of forced expiration against a closed glottis (Valsalva maneuver) have been recognized, virtually since ancient times. Abnormal responses in patients with heart failure have been observed recently by Elisberg and colleagues and by Judson, Hatcher, and Wilkins. Indeed Sharpey-Schafer has suggested the use of the Valsalva maneuver as another method of evaluating cardiac patients with respect to heart failure.

For purposes of analysis, the several responses of Valsalva maneuver (hereafter designated as Vm) were separated into 4 phases by Hamilton, Woodbury, and Harper, as follows: (fig. 1)

I. The blood pressure initially rises with a slight increase in pulse pressure. II. A fall in blood pressure, smaller pulse pressure, and acceleration of the heart rate ensue. As this phase continues, mean blood pressure may turn upward. III. As the strain is abruptly discontinued, blood pressure falls precipitously. IV. Post-Vm systolic, diastolic, and pulse pressures quickly rise; bradycardia may result. Subsequent studies during cardiac catheterization have shown elevated right atrial, right ventricular, and pulmonary arterial pressures during the strain with a less conspicuous pulmonary arterial post-Vm overshoot.

Patients with manifestations of heart failure in contrast, according to Sharpey-Schafer, maintain the strain (phase II) pulse pressure without a post Valsalva (phase IV) diastolic pressure overshoot (fig. 2). ("Failure response" of Judson and co-workers.)

The present study of 19 control subjects and 44 patients with heart disease was undertaken to assess validity and limitations of the Vm as an ancillary method for diagnosis of cardiac insufficiency.

The latter was evaluated by clinical criteria of appropriate symptoms and physical signs, estimation of functional capacity (according to criteria of New York Heart Association) and, in addition, objectively by means of a standard exercise tolerance test in 25 of these patients.

MATERIAL AND METHODS

The 19 control subjects included 4 healthy physicians and 15 ambulatory patients ranging in age from 19 to 64, with a median of 39 years. These patients were hospitalized for miscellaneous diseases but none had either heart or pulmonary disease. Forty-four selected patients with heart disease ranged in age from 12 to 79, with a median of 46 years. Three of the controls and 14 of the cardiac patients were women. Twenty-nine of the patients had rheumatic heart disease, 7 had hypertensive or arteriosclerotic heart disease, 4 had congenital heart disease, 2 had cor pulmonale, and 2 had heart disease of uncertain etiology.

Direct brachial, radial, or femoral arterial pressure was recorded with a Statham strain gage and a direct-writing (Sanborn) oscillograph. After a deep inspiration, an intra-oral pressure of 40 mm Hg (monitored by an aneroid manometer) was sustained for 10 seconds, and then abruptly released. The tracing
Fig. 1. Four phases of arterial pressure responses to Valsalva maneuver in a normal subject. Ratios A and B were derived from changes in pulse pressure and diastolic pressure. Ratio A = \( \frac{(32 - 71) \times 100}{71} \) = -55%, ratio B = \( \frac{(80 - 60) \times 100}{60} \) = +33%.

Fig. 2. Abnormal response to Valsalva maneuver observed in a patient with congestive heart failure. Note increased, rather than decreased pulse pressure in phase II. Ratio A = \( \frac{(72 - 60) \times 100}{60} \) = +20%, ratio B = \( \frac{(70 - 86) \times 100}{86} \) = -19%.

of this standardized Vm was analyzed according to criteria established by Sharpey-Schafer as follows: (figs. 1 and 2)

\[
\text{Ratio A} = \frac{\text{phase II } PP - \text{pre-Vm } PP}{\text{pre-Vm } PP} \times 100
\]

\[
\text{Ratio B} = \frac{\text{phase IV } DP - \text{pre-Vm } DP}{\text{pre-Vm } DP} \times 100
\]

where, \( PP = \) pulse pressure  
\( DP = \) diastolic pressure

In 15 of the control subjects, 2 successive tracings were analyzed to determine variance.

Twenty-five of the cardiac patients performed a standard exercise tolerance test, walking a 10 per cent grade at 1.73 mph on a motor driven treadmill for 10 minutes if tolerated. The cardiorespiratory performance was expressed in terms of the Physical Fitness Index (PFI). Normal values average 19 with a range from 13-26.

For purposes of this paper, decompensated patients are defined as those with physical signs of heart failure, such as rales, elevated venous pressure, hydrothorax, hepatomegaly, ascites, or edema. The term compensated implies absence of these signs.

RESULTS

Control Subjects. The mean change of phase II pulse pressure (ratio A) in 19 control subjects was \(-47 \pm 20\) per cent, with a range from +3 to -81 per cent (fig. 3). The mean change of phase IV diastolic pressure (ratio B) was +18 \pm 19 per cent, with a range from -25 per cent to +51 per cent. The phase II pulse rate was 13 beats per minute higher, on the average than the pre-Vm pulse rate. Contrary to expectations, average of the slowest phase IV pulse rate was only one beat per minute less than the pre-Vm pulse rate.

Analysis of 2 successive Vm determinations in 15 control subjects revealed a coefficient of variation for phase II pulse pressure change (ratio A) of 11 per cent, and for phase IV diastolic pressure change (ratio B) of 40 per cent. Thus, the former ratio was more reproducible than the latter.

Patients. Ratio A deviated by more than 2 \( \sigma \) from mean of control subjects in 10 of 44 patients. Pulse pressure in phase II was essentially maintained or paradoxically increased in each
of these 10 patients. Five had decompensated chronic heart failure, manifested by combinations of rales,\(^3\) increased venous pressure,\(^5\) hepatomegaly, hydrothorax, ascites, or edema.\(^4\) Etiology in 3 of these 5 was rheumatic heart disease, whereas it was hypertensive and arteriosclerotic in 1, and possibly related to hemochromatosis in the last individual.

Of the remaining 5 patients out of 10 with abnormal phase II responses to Vm, 1 had rheumatic heart disease with borderline compensation, 1 had digitalis toxicity and compensated chronic cardiac insufficiency due to rheumatic heart disease, 1 had cyanotic congenital heart disease, 1 had congenital infundibular stenosis, and 1 had an old myocardial infarction.

Seven of the 10 had normal sinus rhythm and 3 had chronic atrial fibrillation.

The mean values for ratios A and B in various subgroups of the 44 cardiac patients are shown in table 1, for comparison with control values. Noteworthy is the maintenance of phase II pulse pressure in the 11 patients with decompensated chronic heart failure, in contrast to 7 patients with borderline compensation, and with 19 control subjects. The criteria failed to discriminate between 9 cardiac patients with a functional capacity of either class I or II, and 17 patients in class III. Phase II responses in 13 patients in class IV were similar to the values obtained in the decompensated patients; actually 8 of the decompensated patients also were included in the class IV group.

Both ratios A and B failed to discriminate between 9 cardiac patients with normal exercise tolerance (mean PFI 19) and 16 cardiac patients with subnormal exercise tolerance (mean PFI 6.3).

The mean phase II response (ratio A) in 11 patients with average circulation time (arm to tongue with decholin) of 14 seconds was \(-46\) per cent. Eight patients with an average circulation time of 39 seconds had a mean ratio of 0 per cent (p.006).

Two patients with cor pulmonale, one decompensated, as well as 2 other patients with pulmonary emphysema not included in this study, had normal Vm responses (mean ratio A \(-54\) per cent, mean ratio B +20 per cent).

Only 2 of the 44 patients had values for ratio B barely deviating from the mean ratio of control subjects by greater than 2 standard deviations.

**DISCUSSION**

Either maintenance or paradoxic increase of pulse pressure during the strain of a standard Valsalva maneuver (Vm) was associated with manifestations of decompensated heart failure in 5 of 10 patients. Since 4 of the remaining 5

**Table 1.—Evaluation of Results of Standard Valsalva Maneuver**

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Incidence of abnormal responses</th>
<th>Ratio A</th>
<th>Ratio B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Phase II</td>
<td>Phase IV</td>
<td>Mean %</td>
</tr>
<tr>
<td>Controls</td>
<td>19</td>
<td>1</td>
<td>1</td>
<td>-47 ±20</td>
</tr>
<tr>
<td>Cardiac patients:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>class I + II</td>
<td>9</td>
<td>1</td>
<td>0</td>
<td>-30 +28</td>
</tr>
<tr>
<td>class III</td>
<td>17</td>
<td>3</td>
<td>1</td>
<td>-35 +12</td>
</tr>
<tr>
<td>class IV</td>
<td>13</td>
<td>5</td>
<td>1</td>
<td>-3 +27</td>
</tr>
<tr>
<td>Compensated</td>
<td>26</td>
<td>4</td>
<td>1</td>
<td>-25 +28</td>
</tr>
<tr>
<td>Borderline compensation</td>
<td>7</td>
<td>1</td>
<td>0</td>
<td>-36 +23</td>
</tr>
<tr>
<td>Decompensated</td>
<td>11</td>
<td>5</td>
<td>1</td>
<td>0 +27</td>
</tr>
<tr>
<td>Normal PFI* (mean 19)</td>
<td>9</td>
<td>2</td>
<td>0</td>
<td>-29 +12</td>
</tr>
<tr>
<td>PFI* 6-13</td>
<td>7</td>
<td>1</td>
<td>0</td>
<td>-32 +3</td>
</tr>
<tr>
<td>PFI* &lt; 6</td>
<td>9</td>
<td>1</td>
<td>0</td>
<td>-35 +28</td>
</tr>
</tbody>
</table>

* PFI = Physical Fitness Index.
with this abnormal response were definitely clinically compensated, it cannot be regarded as diagnostic of decompensated heart failure in all instances. With one exception, the 11 decompensated patients tended to maintain or increase phase II pulse pressure, though the response was statistically abnormal in only 5 of the 11.

Phase I of the Vm in normal subjects is thought to be due in part to forceful evacuation of blood from pulmonary vasculature and left heart. Phase II is related to diminished venous return to the right heart as demonstrated by venography, resulting in decreased cardiac output. Abnormal phase II is ascribed by Stucki and associates to reduction of the normal "venous blockade." They describe only slight increases in the circulation times (p 32) from antecubital and femoral veins to the brachial artery during the Vm in patients in congestive heart failure, in contrast to the marked delay observed in normal subjects during the Vm. Diodrast studies in one normal subject demonstrated blockade of inferior vena caval inflow during the Vm, whereas in a patient in congestive heart failure, diaphragmatic blockade of inferior vena caval inflow during the Vm was absent. The lack of a "venous blockade" is attributed by Judson and colleagues to an elevated right ventricular end-diastolic pressure. In regard to this concept, studies during cardiac catheterization in 2 of our patients may be of interest. Each had compensated congenital heart disease with an interatrial septal defect and a minor degree of pulmonic or infundibular stenosis. One, with a markedly abnormal phase II pulse pressure (ratio A +28 per cent), had virtually identical inferior vena caval and right atrial pressures during a Vm, suggesting little or no venous blockade; nevertheless the resting right ventricular end-diastolic pressure was only 5 mm. Hg. A gradient of 2 mm. Hg from the inferior vena cava to the right atrium during a Vm was demonstrated in the second patient by simultaneously recording the pressures with a double-lumen catheter; her ratio A was only –8 per cent, and her resting right ventricular end-diastolic pressure varied from 5 to 7 mm. Hg.

When Judson and associates suddenly decreased the effective blood volume in patients in congestive heart failure by application of venous congesting cuffs on the thighs, or by venesection, their "failure response" became more nearly normal. Occurrence of a failure response in their normal subjects following an acute increase in circulating blood volume implied that impairment of cardiac function is not the sole explanation of abnormal phase II responses. Consequently, it would be of interest to correlate measured total blood volume with Vm response; unfortunately the data are thus far not available.

When there is effective blockade of venous inflow into the thorax during the Vm, as in the normal, or the usual compensated cardiac patient, the magnitude of phase II pulse pressure decline may be expected to vary inversely with the pulmonary blood volume. If so, this would be in accord with Sjöstrand's concept of the pulmonary blood "reserve" or "depot" serving as a regulator of the circulation.

Studies by Rushmer in normal subjects suggest that venous flow from the abdomen to the thorax may persist during the Vm if leakage of air occurs during the strain, or if it is not preceded by a deep inspiration. Either failure to obtain the prerequisite full inspiration preceding the Vm, or an undetected leakage of air during the maneuver may partially explain the abnormal phase II pulse pressure response found in several of our compensated patients.

Normal phase II responses (ratio A) were frequently noted despite either cardiac enlargement, severe stenosis or insufficiency of mitral or aortic valves, or a history of cardiac decompensation, provided no physical signs of heart failure were evident at the time of the Vm. These observations are in accord with those of Judson and associates. But phase II response fails to discriminate between class I, II, and III cardiacs (New York Heart Association) or between normal and markedly subnormal exercise tolerance (PFI), if manifestations of decompensation are absent.

Thus abnormal phase II, providing certain technical prerequisites are adhered to (preceding full inspiration, nonleakage of air during the strain), may reflect right ventricular failure.
The relative importance of physiologic acute and chronic alterations in the blood volume modifying phase II of the Vm remains to be elucidated. In a general sense, the degree of “venous blockade” attained during the Vm, and thereby the magnitude of phase II pulse pressure response, depends on the pressure gradient between increased intrathoracic air pressure during Vm and the pressure-volume relationships of the right heart and systemic veins entering the thoracic cavity. Consequently both false positive and false negative abnormal phase II responses to Vm may be observed in relation to heart disease.

**SUMMARY AND CONCLUSIONS**

Arterial blood pressure was directly recorded during the Valsalva maneuver (Vm) in 44 cardiac patients and 19 control subjects.

Patients with definite physical signs of cardiac decompensation may exhibit an abnormal response in phase II, but not necessarily.

Abnormal phase II response may be observed despite cardiac decompensation.

Abnormal phase II responses to the Valsalva maneuver in cardiac patients are more closely associated with physical signs of congestive heart failure than either impaired functional capacity or exercise tolerance, but both false positive and false negative responses may be observed.

**SUMMARIO IN INTERLINGUA**

Le pression de sanguine arterial esseva registra directemente durante le experimento de Valsalva in 44 patientes cardiac e 19 subjectos de controlo.

Patientes con definite signos physic de discompensation cardiac pote, sed non debe, exhibir un responsa anormal in phase II.

Anormal responsas in phase II pote esser observate in despecto de compensation cardiac.

Anormal responsas in phase II es associate plus nettement con signos physic de congestive disfallimento cardiac que con reduceit capacitate functional o tolerantia a exercitos, sed il occurre responsas pseudopositive e pseudo-negative.

**REFERENCES**

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