Studies of Circulation Time During the Valsalva Test in Normal Subjects and in Patients with Congestive Heart Failure

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Circulation times (P2m from the antecubital and femoral veins to a peripheral artery) and roentgenographic studies of the pattern of venous distribution of a radio-opaque substance (Diodrast introduced through a cardiac catheter into the axillary vein and the inferior vena cava below the diaphragm) have been performed during the expiratory effort of the Valsalva maneuver. In normal subjects the circulation times were increased by the duration of the expiratory effort and the Diodrast injections were stagnated in the veins outside the thoracic cavity. These effects were in striking contrast to those in patients with congestive failure in whom the circulation times were retarded not only partially if at all and the Diodrast injections continued to flow freely towards the right atrium during the expiratory effort. Thus, in patients with congestive failure the Valsalva maneuver does not interrupt the venous return to the right atrium as it does in normal subjects.

In 1949 during studies on the circulatory effects of ganglionic blocking agents in patients with congestive heart failure (1) it was found that such patients have abnormal arterial pressure responses to the Valsalva test. In contrast to the normal subject who, after a brief initial rise in arterial pressure, has a fall in systolic, diastolic and pulse pressure during the straining period (fig. 1), the patient in congestive failure has a rise of systolic and diastolic pressure and a maintenance of pulse pressure throughout the expiratory effort (fig. 2). On relaxing the strain, the patient in congestive failure simply has a return of arterial pressure to control levels without the characteristic normal overshoot. This response pattern of the patient in congestive heart failure has been called the “failure response.” Occasional patients in moderate, but not severe, congestive failure have an “intermediate failure response”, showing less than normal decrease in pulse pressure during, and less than normal (or absent) overshoot of pressure after the strain (fig. 3). In a large group of cases all variations between the full “failure response” and the normal response are seen.

The purpose of the present paper is to report the effect of the Valsalva maneuver upon circulation times in patients with and without congestive heart failure. In addition, radiographic Diodrast studies during the Valsalva maneuver in two patients are presented.

Methods

The Valsalva maneuver was performed by having the supine subject take a full inspiration and forcibly attempt to exhale for a certain period of time, usually 10 seconds, into a closed manometer at a pressure of 40 to 60 mm. Hg. The systemic arterial pressure was recorded continuously with a Sanborn electromanometer connected to an inlying needle in a brachial or femoral artery.

Circulation time was measured from an antecubital vein, a femoral vein, or both, to a brachial or femoral artery before and during the Valsalva test. In three patients a cardiac catheter was placed in the pulmonary artery and the circulation time was measured from the pulmonary artery to a brachial or femoral artery.

Approximately 30 microcuries of essentially car-
carrier-free radio-active phosphorus (P³²) as sodium phosphate in 1 to 2 cc. of physiological saline were injected intravenously. Arterial blood was then sampled from the intra-arterial needle at two second intervals. The first injection was made with the subject in the resting state in order to obtain a "control" circulation time. After 3 to 5 minutes a second injection of P³² was made at the onset of the Valsalva maneuver, care being taken that the P³² was not injected before the patient had started to strain.

From each two-second sample of arterial blood 0.2 cc. were pipetted into planchettes and dried overnight. The radioactivity of all samples from each patient was quantitated on the same day with a thin-window Geiger-Mueller tube (Tracerlab—TGC-2) and scaling circuit, sufficient counts being taken in each instance to reduce the error of counting to less than 2 per cent. The calculated counts per second were plotted against time in seconds. The curve thus obtained (fig. 4) showed a sudden rise of the radioactivity above the background count when the first P³² reached the site of sampling. The last point before a definite and continuous rise over the baseline was taken arbitrarily as the circulation time. In the same patient the circulation time by this method may be determined repeatedly to within two seconds. The method is entirely objective and was particularly advantageous in this study because it required no cooperation on the part of the patient.

In two patients 10 cc. of a 30 per cent Diodrast solution were injected through a cardiac catheter into the inferior vena cava below the diaphragm (in one also in the axillary vein) during the Valsalva maneuver. X-ray films of the abdomen and thorax were taken at different intervals after the beginning of the maneuver.

**RESULTS**

The circulation times of the subjects with normal Valsalva responses are given in table 1. It can be seen that in all cases the circulation times were greater during the Valsalva maneuver than during the control period. In other words, there was a definite delay of the circulation time as a result of the strain. Thus, in the subjects who strained for a 10-second period, the delay from the arm (antecubital vein) as well as from the leg (femoral vein) averaged 10 seconds, ranging between 8 and
12 seconds. In subjects E. D. and J. B., who strained for longer than 10 seconds, there was also a definite delay. Percentagewise the delay in these two subjects amounted to 100 per cent of the straining period measured from the arm, but to only 41 to 75 per cent from the leg. However, calculated on the basis of 10 seconds, the delay from the leg was at least 80 per cent, as in the other normally responding subjects. This, as well as previous experience, has convinced us that a Valsalva test with a 10-second straining period is about optimal for producing blockage of the circulation in normal subjects. If the strain is prolonged beyond 10 seconds, the venous return, particularly from below the diaphragm, apparently may break through the blockage (see “Discussion”).

Table 2 gives the results obtained in patients with various types of “failure response.” The control circulation times in these patients were, with three exceptions, greater than normal. A Valsalva maneuver of 10 seconds produced a variable delay of the circulation time when measured from the arm to a peripheral artery, ranging between 0 and 100 per cent of the actual straining time. On the average, the delay was considerably less than in the normal group shown in table 1. However, there were two patients who had a 100 per cent delay of the arm vein to artery time as did the normal subjects. It is interesting that both these patients had a normal control circulation time. Both had undergone mitral valvuloplasty with considerable improvement of their circulatory status.

The results measured from a femoral vein to a peripheral artery in the failure group were much more consistent than from the antecubital vein. With one exception the Valsalva maneuver produced no significant delay of the leg to artery time; i.e. the delay did not exceed 20 per cent of the blowing period, although in some patients it did cause a delay in the arm to artery time.

The patient G. G. (tables 1 and 2) deserves special comment. Clinically this man had a normal cardiovascular status. After rapid infusion of 650 cc. of whole blood within eight minutes this patient had a moderately elevated right ventricular end-diastolic pressure of 8 mm. Hg and an increased arm-to-artery circulation time of 24 seconds. The Valsalva maneuver was then characterized by an abnormal “intermediate failure response”. The delay in the arm to artery circulation time during the Valsalva maneuver was only 20 per cent of the
TABLE 2.—Summary of Circulation Time (P<sub>100</sub>) from Antecubital and Femoral Veins to a Peripheral Artery Before and During the Valsalva Maneuver in Patients with Congestive Heart Failure

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Antecubital Vein to Peripheral Artery</th>
<th>Femoral Vein to Peripheral Artery</th>
<th>Blood Press. Resp. to Valsalva Man.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Circ. Time Control (sec.)</td>
<td>Blowing Time (Val-salva Man.) (sec.)</td>
<td>Circulation Time during Val-salva (sec.)</td>
</tr>
<tr>
<td>I. K.</td>
<td>H.C.V.D. Cong. failure (r. &amp; l.)</td>
<td>24</td>
<td>10</td>
<td>24</td>
</tr>
<tr>
<td>G. L.</td>
<td>H.C.V.D. Cong. failure (r. &amp; l.)</td>
<td>40</td>
<td>10</td>
<td>42</td>
</tr>
<tr>
<td>G. B.</td>
<td>A.S.H.D. Cong. failure (r. &amp; l.)</td>
<td>32</td>
<td>10</td>
<td>38</td>
</tr>
<tr>
<td>A. G.</td>
<td>A.S.H.D. Cong. failure (r. &amp; l.)</td>
<td>26</td>
<td>10</td>
<td>30</td>
</tr>
<tr>
<td>E. C.</td>
<td>R.H.D. M.S. Cong. fail.</td>
<td>24</td>
<td>10</td>
<td>26</td>
</tr>
<tr>
<td>A. Ls.</td>
<td>R.H.D. M.S. Postoper. valvuloplasty, cong. failure</td>
<td>10</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>C. G.</td>
<td>R.H.D. M.S. Clinically no cong. failure</td>
<td>10</td>
<td>10</td>
<td>14</td>
</tr>
<tr>
<td>J. C.</td>
<td>R.H.D. M.S. Postop. valvuloplasty, cong. failure</td>
<td>12</td>
<td>10</td>
<td>24</td>
</tr>
<tr>
<td>G. G.</td>
<td>Duodenal ulcer. Right ventricular press. 29/8 mm. Hg after infusion of 650 cc. whole blood within 8 minutes.</td>
<td>24</td>
<td>10</td>
<td>26</td>
</tr>
</tbody>
</table>

* Failure.
† Intermediary.

blowing time, resembling that in patients with congestive failure. Seven days later, when he had recovered from the blood infusion, the control circulation time had returned to a normal value of 12 seconds and the arterial pressure response to the Valsalva maneuver was normal. In keeping with these findings the delays of circulation time during the maneuver from the arm and the leg were now 80 per cent and 100 per cent, respectively.

A similar experiment was carried out in a dog under Nembutal anesthesia. The Valsalva maneuver was performed by passive inflation of the lungs with a pressure of 40 mm. Hg for a 10-second period. The control circulation time from the femoral vein to the femoral artery was 8 seconds and the delay of the circulation time during the Valsalva maneuver was 100 per cent. After rapid infusion of 1650 cc. of saline within 15 minutes into the right auricle, the “control” circulation time was unaltered (8 seconds), but the Valsalva response had changed from normal to an “intermediate failure” type. There was now only a 40 per cent delay of the circulation time during the Valsalva maneuver.

Thus in a patient and in a dog, in whom the Valsalva response was changed from the normal to the intermediate type after large rapid intravenous infusions, the delay of the circulation time during the maneuver underwent corresponding changes from complete (normal) delay to less or none at all.

Table 3 gives the circulation times from the pulmonary artery to a peripheral artery before and during the Valsalva maneuver in one normal subject and in two patients with mild congestive failure who were studied after mitral
CIRCULATION TIME DURING VALSALVA TEST

Table 3.—Effect of Valsalva Maneuver on Circulation Times (P³) from Pulmonary Artery to Peripheral Artery and from Antecubital and/or Femoral Veins to Peripheral Artery in Patients with and without Congestive Failure

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Pulmonary Artery to Peripheral Artery</th>
<th>Antecubital and/or Femoral Veins to Peripheral Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>W. A.</td>
<td>Normal</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>A. Lm.</td>
<td>R.H.D. M.S. Postop. valvuloplasty, cong. failure</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>J. C.</td>
<td>R.H.D. M.S. Postop. valvuloplasty, cong. failure</td>
<td>8</td>
<td>10</td>
</tr>
</tbody>
</table>

* Normal.
† Failure.

**Fig. 5.** Roentgenogram taken 10 seconds after the injection of Diodrast solution through an intravenous catheter below the diaphragm in a patient with compensated heart disease (R. M.). The absence of any visible radio-opaque substance in the area below the diaphragm indicates that it has left the inferior vena cava and distributed itself in the general circulation.

**Fig. 6.** Roentgenogram taken of the same patient (R. M.) during the injection of Diodrast solution through an intravenous catheter below the diaphragm while the patient was performing the expiratory effort of the Valsalva maneuver. The radio-opaque material is now observed to be puddled below the diaphragm with none appearing above it suggesting venous obstruction at the level of the diaphragm.
valvuloplasty. None of these individuals showed a significant delay of the pulmonary to peripheral artery time during the Valsalva maneuver.

Figures 5 to 9 inclusive show the results obtained in two patients by Diodrast studies before and during the Valsalva maneuver. Patient R. M. had a moderately severe arterial hypertension but no elevation of the pulmonary arterial or right ventricular diastolic pressure at rest. He had a normal arterial pressure response, and a normal full delay of the circulation times during the Valsalva maneuver. With the patient resting quietly, 10 cc. of 30 per cent Diodrast were injected through a cardiac catheter into the inferior vena cava below the diaphragm over a period of 10 seconds. The roentgenogram (fig. 5) taken at the eleventh second after the beginning of the injection, showed no significant amount of radio-opaque material at the site of injection, indicating that the Diodrast had already left the vein at that time. There were only a few residual streaks of Diodrast adhering to the wall of the vein, giving a rather poor outline of the vessel.

Figure 6 shows the result obtained by injecting Diodrast solution in the same patient (R. M.) during the Valsalva maneuver. The contrast substance was given over an eight-second period, the roentgenogram taken at the eighth second. In contrast to figure 5, a large quantity of radio-opaque material can be seen in the inferior vena cava distal to the tip of the catheter, superimposed partially on the shadow of the spine. No contrast medium is detectable proximal to the catheter tip. The radio-opaque material obviously did not move upwards in the direction of the right heart during the whole period of the injection.

Figure 7 gives the result obtained when the

Fig. 7. Roentgenogram taken of the same patient (R. M.) after the injection of contrast substance through a catheter into the right axillary vein during the Valsalva maneuver shows the pooling of the radio-opaque material in the veins outside, but not within, the thoracic cavity, indicating obstruction to the venous flow at this junction.

Fig. 8. Roentgenogram taken of patient I. K. with severe congestive failure four seconds after completion of the injection of Diodrast solution through a catheter below the diaphragm shows the presence of radio-opaque material both below and above the diaphragm.
Diodrast solution was injected through the catheter into the right axillary vein during the Valsalva maneuver in the same patient, R. M. The contrast substance is accumulated at the site where the vein enters the thoracic cavity, outlining clearly the venous network in the axilla. No radio-opaque material can be detected in the veins within the thoracic cavity.

Diodrast solution was injected similarly into the inferior vena cava of a patient in severe congestive failure (I. K., table 2). This patient had a markedly elevated control circulation time of 24 seconds, a "failure response" to the Valsalva maneuver and a delay of the circulation time during the maneuver of only 0 per cent and 20 per cent, respectively, from the arm and the leg. When 10 cc. of Diodrast solution were injected into the patient at rest over a six-second period and a film was taken at 10 seconds (fig. 8), the radio-opaque material was readily seen in the inferior vena cava both distal and proximal to the tip of the catheter with the head of the column just above the diaphragm. This was in contrast to the relatively quick disappearance of the contrast medium in the patient who was not in congestive failure, but was quite in keeping with the slow circulation time and the elevated right ventricular diastolic pressure in this patient.

When the injection was repeated during the Valsalva maneuver (injection over a period of six seconds), the roentgenogram taken at the fifth second revealed essentially the same pattern (fig. 9), i.e., the radio-opaque material could be detected distal and proximal to the catheter tip and passing just above the diaphragm towards the right heart. This was in striking contrast to the subject with a normal Valsalva response, in whom the Diodrast remained distal to the diaphragm and did not flow in the direction of the heart during the maneuver.

**DISCUSSION**

The results reported here indicate that in individuals with normal Valsalva responses the venous return from the arm and leg is blocked during a Valsalva maneuver for about a 10-second period. The data in table 3 also show that the circulation from the pulmonary artery to a peripheral artery is not blocked during the Valsalva maneuver. Identical results have been obtained by Matthes (4), using a different method. The Diodrast studies revealed that radio-opaque material deposited just outside the thoracic cavity does not flow in the direction of the right heart during a Valsalva maneuver in a normally responding subject.

Thus, direct and indirect evidence was obtained that the venous return is blocked during the strain of the Valsalva maneuver and that this causes a decrease in cardiac output and the consequent drop of the systolic, diastolic and pulse pressures. Thus, the theory originally forwarded by Buerger that a block of the circulation occurs in the pulmonary vascular bed during the Valsalva maneuver (due to an elevated intrapulmonary pressure which cannot
be overcome by the relatively weak right ventricle) must be discarded. Indeed, Buerger himself abandoned it, at least partially, in the later stages of his investigations of the Valsalva experiment.7

It was not the primary purpose of the present study to determine how long the circulation time can be delayed when a patient strains for longer than 10 seconds. Walz and Zimmermann8 (using a similar method with P32) have reported circulation times from arm vein to arm vein in five normal subjects who strained between 30 and 40 seconds. All five individuals demonstrated a delay of 100 per cent of the blowing period. This was true for our patient, J. B. (table 1), who strained for 18 seconds (delay 100 per cent). The situation, however, seems to be different for the circulation times from the femoral vein. The two individuals who strained for 16 and 19 seconds, respectively, showed a delay of the circulation time during the Valsalva maneuver of only 12 and 8 seconds. As already mentioned it would appear that the venous return from the arm can be impeded more readily and for a longer period of time than that from the leg. Experience in this laboratory has shown that the most striking and pronounced blood pressure changes are usually obtained by having the subject strain for a period of about 10 seconds. The majority of subjects begin to have a return of arterial pressure toward control levels when the Valsalva maneuver is carried out for longer than 10 seconds.

In the patients with a "failure response" to the Valsalva maneuver the delay of the circulation time by the strain was definitely less than in the normal subjects. This was particularly true for the circulation time from the femoral vein, indicating that little or no block of the venous return from that area occurred during the Valsalva maneuver in these subjects. A similar situation was found for return from the arm vein; although in some patients the delay from that area was greater than from the femoral vein. As in the normal subject, it would appear that in patients with congestive failure, expiratory straining may impede venous flow from the arm more readily than from the leg. Thus, during the Valsalva maneuver the return from the inferior vena cava is apparently of greater importance than that from the superior vena cava in determining the type of arterial pressure response to the maneuver.

The fact that in subjects with a "failure response" the venous return to the right heart is not blocked from the femoral vein during the Valsalva maneuver and may not be even from the arm appears to explain the difference between the "normal" and the "failure" type of Valsalva response. In the latter the filling of the heart is maintained during the strain, at least to an extent adequate to insure that there is no, or only a partial, decrease of cardiac output and of systolic, diastolic, and pulse pressure in the peripheral arterial system.

**Summary**

The effect of the Valsalva maneuver on the circulation times from both the antecubital and femoral veins to a peripheral artery has been measured with P32 in normal subjects, compensated cardiac patients, and in patients with congestive failure.

In normal subjects and compensated cardiac patients the circulation times increase during the Valsalva maneuver by roughly the duration of the expiratory strain.

In patients with congestive failure the circulation times are not retarded, or are only partially retarded during the strain of the Valsalva maneuver.

Roentgenograms taken after the introduction of Diodrast solution through a venous catheter into the inferior vena cava below the diaphragm (or in the axillary vein outside the thoracic cavity) during the Valsalva maneuver indicate that in patients with congestive failure the expiratory effort of the Valsalva maneuver does not interrupt normally the venous return to the right atrium.

These observations on circulation time and the concentration and movement of the contrast substance (Diodrast) in the venous system suggest that the blood pressure responses during the Valsalva maneuver are determined by the height of the right ventricular end-diastolic pressure, which in-
fluences the venous return to the right side of the heart during the expiratory strain.

**Summary in Interlingua**

Le effecto del experimento de Valsalva super le tempore de circulation ab le venas tanto antecubital como etiam femoral usque a un arteria peripheric eseva mesurate per medio de P32 in individuos normal, in compensate patientes cardiac, e in patientes con dysfunctionamento congestive.

In individuos normal e in compensate patientes cardiac, le tempores de circulation se augmenta durante le experimento de Valsalva per grossiermente le duration del effortio expiratori.

In patientes con dysfunctionamento congestive le tempores de circulation non es retardate del toto o solo partialmente durante le effortio del experimento de Valsalva.

Roentgenogrammas facite post le introducction durante le experimento de Valsalva de un solution de Diodrast per un catheter venose a in le vena cave inferior infra le diaphragma (o a in le vena axillar al exterior del cavitate thoracic) indica que in patientes con dysfunctionamento congestive le effortio expiratori del experimento de Valsalva non interrumpe normalmente le retorno venose al atrio dextere.

Iste observationes in re le tempore de circulation e le concentration e movimento del substantia de contrasto Diodrast in le systema venose pare indicar que le responsas del pression sanguinee durante le experimento de Valsalva es determinate per le magnitude del termino-diastolic pression dexteroventricular que influe durante le effortio expiratori super le retorno venose al latere dextere del corde.

**Acknowledgement**

The authors gratefully acknowledge the assistance of Dr. Edward N. Burke in the roentgenographic studies.

**References**


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Circulation. 1955;11:900-908
doi: 10.1161/01.CIR.11.6.900

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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