Vasodepressor Syncope
Factors Influencing Cardiac Output

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H. D. McIntosh, M.D., and J. J. Leonard, M.D.

Vasodepressor syncope is characterized hemodynamically by a sudden fall in total peripheral resistance with little associated change in cardiac output. The failure of the cardiac output to compensate for the fall in peripheral resistance is a striking feature of the fainting reaction. Possible explanations for this phenomenon are the presence of neurogenic myocardial inhibition or markedly limited volume of blood available to the heart. The effects of atropine injections, inflation of antigravity suit, negative pressure breathing, and albumin infusions on the syncopal reaction were studied. Results favor the causative role of limited venous inflow in the cardiac output response.

VASODEPRESSOR syncope offers an interesting experimental situation for the study of the physiologic mechanisms controlling blood pressure and cardiac output. Heretofore, only a relatively small number of observations on the cardiac output during the acute hypotensive phase have been reported.\(^1\)\(^2\) The difficulties in producing syncope at will, coupled with technical complications in measurement of cardiac output under such unstable circumstances, have made experiments difficult. The dye-injection method of determining cardiac output has enabled us to study not only the syncopal episode itself, but the effect of modifying experimental situations. These observations, obtained during the course of an over-all assessment of vasodepressor syncope, are the subject of the present report.

METHOD

The subjects were all university students. Syncope was induced by passively tilting to a 60° head-up position. Spontaneous syncope occurred in 20 per cent of the subjects. Of the remaining subjects, syncope was induced in 80 per cent by the oral administration of sodium nitrite 10 to 15 minutes prior to tilting. The entire procedure was explained to the subjects and precautions were taken to allay anxiety. Preliminary practice tilts were made in all cases to acquaint the subjects with the procedure, and to detect those who faint spontaneously.

The cardiac output was determined by the dye method of Hamilton,\(^3\) as modified by Doyle and colleagues.\(^4\) T-1824 in 0.5 per cent solution was injected into the central circulation through a catheter introduced into the superior vena cava or subclavian vein. The position of the catheter was determined by fluoroscopy. Arterial blood was collected in siliconized tubes from femoral or brachial arteries at 2-second intervals, and the undiluted dyed serum was promptly read on a Beckman model DU spectrophotometer. Calculations of cardiac output were made from concentration-time plots of the serum samples on semilogarithmic paper. The dye curves were scrutinized closely. Only those determinations demonstrating good delineation of the dye curves and the point of recirculation were included in the study.

Arterial blood pressures were obtained by Statham strain-gage, or Sanborn elecmameter, and recorded on direct writing or photographic multi-channel recording systems.

All cardiac output data are referred to body area, and are expressed as liters per minute per square meter of body surface. Mean arterial pressures were calculated by addition of one third of the pulse pressure to the diastolic levels. Because of the slow moving camera speed utilized for prolonged observations, planimetric integration was not feasible. Total peripheral resistance (PR) was calculated from the formula \(PR = (BAm \times 1332/CO)\), where \(BAm\) = mean brachial artery pressure, 1332 = conversion factor to dyne/cm\(^2\), \(CO\) = cardiac output in ml per second. The “pulmonary blood volume” was calculated according to the Hamilton formula.\(^4\)

A single balloon half-body antigravity suit* of the type reported by Beckman and co-workers\(^5\) was employed in the studies of the effects of body com-

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pression. With this apparatus, inflation to 60 ± 5 mm. Hg of body pressure was achieved within 10 seconds (1 pound per square inch of body pressure = 51.7 mm. Hg). Dye injections for cardiac output were made 5 seconds after initiation of suit inflation.

The carbon dioxide content of whole blood was determined by the method of Van Slyke and Neill. The data were analyzed statistically according to methods outlined by Snedecor.

**Results**

Syncope was observed in 40 subjects. The clinical picture during the course of the reaction was similar in all subjects and consisted of vague epigastric distress, nausea, pallor, sweating, mydriasis, yawning, sighing, belching, and hyperventilation followed by impaired consciousness, visual blurring, and finally unconsciousness. At times, clonic head and neck movements were in evidence. The presence of anxiety or apprehension during the syncopal reaction was unusual.

The typical arterial pressure and pneumographic changes in vasodepressor syncope are illustrated in the record of subject LB in figure 1. Of particular interest in the arterial pressure tracings are the prominent Traube-Hering waves, the forward migration and diminution in size of the dicrotic notch, and the progressive fall in systolic, diastolic, and mean arterial pressure. The hyperventilation and relative bradycardia are additional features of the reaction.

**Cardiac Output During Vasodepressor Syncope**

In 8 subjects cardiac outputs were determined at 60° head-up tilt before and during the syncopal reaction. Control outputs were determined 3 to 10 minutes after tilting, when the subjects were relaxed with stable blood pressure and pulse. Since presyncopal changes, e.g., yawning, nausea, pallor, sweating, mydriasis, and hyperventilation, occur at varying and unpredictable intervals before the hypotension and bradycardia, dye injections for the syncopal cardiac outputs were made only after the latter findings were in evidence on monitored records. With the onset of convulsive movements in any subject, the tilt was terminated immediately. In 7 of these subjects undamped pressure recordings at the time of the syncopal reaction revealed systolic arterial

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**FIG. 1. Vasodepressor syncope with recovery following return to recumbency (marked "tilt"), demonstrating hypotension, bradycardia, Traube waves, and hyperventilation. Recording system was flushed at the point indicated on the illustration.**

**Table 1.—Hemodynamic Changes in Vasodepressor Syncope**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Cardiac index (L./min./M.²)</th>
<th>Mean arterial pressure (mm. Hg)</th>
<th>Peripheral resistance (Dyne-sec./cm.²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control Syncope Change</td>
<td>Control Syncope Change</td>
<td>Control Syncope Change</td>
</tr>
<tr>
<td>GH</td>
<td>3.5 2.2  -1.3</td>
<td>100 38  -62</td>
<td>1128 689  -439</td>
</tr>
<tr>
<td>BW</td>
<td>2.5 1.6  -0.9</td>
<td>74 30  -44</td>
<td>1302 812  -490</td>
</tr>
<tr>
<td>BJ</td>
<td>3.2 2.5  -0.7</td>
<td>85 26  -59</td>
<td>1125 426  -690</td>
</tr>
<tr>
<td>DF</td>
<td>2.9 2.3  -0.6</td>
<td>80</td>
<td>1108</td>
</tr>
<tr>
<td>LD</td>
<td>2.4 1.9  -0.5</td>
<td>82 55  -27</td>
<td>1407 1226  -181</td>
</tr>
<tr>
<td>JF</td>
<td>2.7 2.5  -0.2</td>
<td>87 57  -30</td>
<td>1388 956  -402</td>
</tr>
<tr>
<td>MS</td>
<td>2.2 2.5  +0.3</td>
<td>67 27  -40</td>
<td>1132 392  -740</td>
</tr>
<tr>
<td>LB</td>
<td>3.8 4.3  +0.5</td>
<td>95 32  -63</td>
<td>1064 320  -744</td>
</tr>
<tr>
<td>Mean</td>
<td>2.9 2.5  -0.4</td>
<td>84 38  -46</td>
<td>1217 689  -528</td>
</tr>
<tr>
<td>Sd*</td>
<td>0.21</td>
<td></td>
<td>80</td>
</tr>
<tr>
<td>p</td>
<td>&gt;.05</td>
<td></td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

All data obtained while the subjects were in the 60° head-up tilt position.

* Refers to standard error of the difference between means.
pressure levels ranging from 84 mm. to 33 mm. Hg, and diastolic levels of 54 mm. to 24 mm. Hg, with a mean of 54/33 for the group. The mean arterial pressure data are recorded in table 1.

The cardiac outputs observed in the 8 subjects in this series are summarized in table 1. Subjects DF and LB received no nitrite. Although the mean fall in cardiac index of 0.4 L is not statistically significant, the data indicate a tendency for the cardiac output to fall slightly during syncope. In 3 additional subjects in whom control tilted outputs for comparison were not obtained, the cardiac indices during fainting were 2.6, 2.6, and 2.4 L respectively. The mean resting cardiac index at 60° head-up tilt in a group of 30 subjects in this laboratory is 2.7 L. (S.D. ± 0.3).

Stroke volume changes were variable and revealed no consistent trends. During syncope a relative bradycardia is always observed when the pulse rate is compared to the usual presyncopal tachycardia.

As expected from the above data, decreases in peripheral resistance occurred in all of the subjects (table 1). The mean change from 1217 to 689 units is significant (p < .01). Two additional subjects, in whom peripheral resistance in the tilted position before fainting could not be calculated, demonstrated falls of 924 and 620 units respectively when compared to their peripheral resistance in the recumbent position.

The estimated "pulmonary blood volume" was calculated in the 8 subjects in this series. The results are tabulated in table 2, and demonstrate a close correlation with the cardiac output changes. The mean circulation time during syncope averaged 18.0 seconds, which is not significantly different from the control average of 17.6 seconds.

Cardiac Index During Recovery from Syncope Following Resumption of Recumbency

Upon resumption of the recumbent posture in the course of vasodepressor syncope, there is prompt reversal of the blood pressure and pulse changes, and recovery of consciousness (fig. 1). On 2 occasions inordinately elevated cardiac indices were registered when dye output determinations were made immediately after return to the horizontal position. In order to investigate this phenomenon further, 5 additional studies were obtained in which syncope was induced in the usual manner and output determinations were made immediately after reverse tilt. To accomplish this, the dye was injected during the course of the reverse tilt. The data are summarized in table 3. A mean cardiac index rise of 2.4 (S.E. ± 0.9)* L. per minute per M.² above resting recumbent levels was observed during the first 30 seconds following return to the horizontal position. In all of the above subjects complete recovery, save persistent pallor and occasional headache, occurred within 45 seconds of the reverse tilt, so that the cardiac output rise occurred during the period of recovery. When compared to the cardiac index during syncope (table 1), the rise in cardiac index following return to recumbency is highly significant (p < .01). Of additional interest in all subjects following the reverse tilt was a transient but marked flush and tachycardia, each lasting no longer than 15 seconds.

Effects of Inflation of Antigravity Suit

In an attempt to assess the role of diminished venous inflow to the heart in the hemodynamic

* Refers to standard error of the difference between means.

Table 2.—"Pulmonary Blood Volume" in Vasodepressor Syncope

<table>
<thead>
<tr>
<th>Subject</th>
<th>Control (ml.)</th>
<th>Syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>GH</td>
<td>2336</td>
<td>1423</td>
</tr>
<tr>
<td>BW</td>
<td>1067</td>
<td>821</td>
</tr>
<tr>
<td>BJ</td>
<td>1601</td>
<td>1236</td>
</tr>
<tr>
<td>DF</td>
<td>1552</td>
<td>1356</td>
</tr>
<tr>
<td>LD</td>
<td>1662</td>
<td>1560</td>
</tr>
<tr>
<td>JF</td>
<td>1365</td>
<td>1301</td>
</tr>
<tr>
<td>MS</td>
<td>1679</td>
<td>1778</td>
</tr>
<tr>
<td>LB</td>
<td>1794</td>
<td>1355</td>
</tr>
</tbody>
</table>

Mean.......................... 1682  1358
Sd*.......................... 115
p..........................<.05

All data obtained while subjects were in the 60° head-up tilt position.
* Refers to standard error of the difference between means.

* Refers to standard error of the mean.
TABLE 3.—Cardiac Index during Recovery from Vasodepressor Syncope Following Resumption of Recumbency

<table>
<thead>
<tr>
<th>Subject</th>
<th>Before syncope (L./min./M.²)</th>
<th>During recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>RT</td>
<td>2.9</td>
<td>9.9</td>
</tr>
<tr>
<td>DT</td>
<td>2.5</td>
<td>5.0</td>
</tr>
<tr>
<td>DL</td>
<td>3.4</td>
<td>4.0</td>
</tr>
<tr>
<td>RH</td>
<td>3.7</td>
<td>3.8</td>
</tr>
<tr>
<td>RK</td>
<td>2.8</td>
<td>6.4</td>
</tr>
<tr>
<td>AW</td>
<td>3.4</td>
<td>5.0</td>
</tr>
<tr>
<td>FA</td>
<td>2.7</td>
<td>4.0</td>
</tr>
<tr>
<td>Mean</td>
<td>3.1</td>
<td>5.4</td>
</tr>
</tbody>
</table>

All of the above determinations were performed while the subjects were in the horizontal position.

TABLE 4.—Cardiac Index in Normal Subjects after Antigravity Suit Inflation

<table>
<thead>
<tr>
<th>Subject</th>
<th>Before inflation (L./min./M.²)</th>
<th>After inflation*</th>
</tr>
</thead>
<tbody>
<tr>
<td>RN</td>
<td>1.8</td>
<td>2.4</td>
</tr>
<tr>
<td>HY</td>
<td>2.3</td>
<td>3.7</td>
</tr>
<tr>
<td>RK</td>
<td>2.8</td>
<td>2.5</td>
</tr>
<tr>
<td>JM</td>
<td>2.7</td>
<td>3.8</td>
</tr>
<tr>
<td>ST</td>
<td>2.3</td>
<td>3.0</td>
</tr>
<tr>
<td>MM</td>
<td>2.6</td>
<td>3.6</td>
</tr>
<tr>
<td>CH</td>
<td>3.1</td>
<td>3.0</td>
</tr>
<tr>
<td>TP</td>
<td>2.1</td>
<td>2.2</td>
</tr>
<tr>
<td>TH</td>
<td>2.2</td>
<td>3.5</td>
</tr>
<tr>
<td>RD</td>
<td>2.3</td>
<td>2.4</td>
</tr>
<tr>
<td>JG</td>
<td>1.8</td>
<td>2.7</td>
</tr>
<tr>
<td>Mean</td>
<td>2.4</td>
<td>3.0</td>
</tr>
<tr>
<td>Sd</td>
<td>0.18</td>
<td></td>
</tr>
<tr>
<td>p</td>
<td>&lt;.01</td>
<td></td>
</tr>
</tbody>
</table>

All data obtained while subjects were in the 60° head-up tilt position.

* Data obtained during the first 30 seconds following inflation to 60 ± 5 mm. Hg body pressure.

† Refers to standard error of the difference between means.

picture of vasodepressor syncope, the effects of application of external pressure in the normal and syncopal state were studied.

Inflation of the antigravity suit to 60 ± 5 mm. Hg body pressure in the standing position is attended by little or no discomfort to the subject. Because of the alarm of sudden inflation and the tendency of the subject to tense the abdominal muscles, practice inflations had been carried out before the actual studies were initiated. In the normal subject at 60° head-up tilt, antigravity suit inflation results in a prompt increase in pulse pressure (5–10 mm.), predominantly a result of elevated systolic pressure and a moderate slowing of the pulse. In table 4 are summarized the cardiac output data in 11 subjects in whom determinations were made with subjects at 60° head-up tilt before and after antigravity suit inflation. The mean increase of 0.6 L. per minute per M.² following antigravity suit inflation, representing a 25 per cent elevation above the control output for the group, is significant (p < .01). Peripheral resistance following antigravity suit inflation tends to fall slightly, averaging 206 units below control levels (p = 0.1).

Inflation of the antigravity suit in the course of a syncopal reaction is attended by dramatic recovery of consciousness and resolution of symptoms within 30 seconds. The typical hemodynamic response to inflation during syncope is illustrated in figure 2. In the left side of the figure, the usual pressure, pulse, and respiratory changes of vasodepressor syncope are seen. Of additional interest in this illustration is the central venous pressure, which remained unaltered throughout the course of the syncopal reaction until its rise after the suit was inflated. Immediately following inflation of the antigravity suit there is a rise in systolic, diastolic, and mean arterial pressure, and disappearance of the relative bradycardia.

A summary of the effects of antigravity suit inflation on the cardiac output in the course of syncope appears in table 5. In each of the subjects, the determination of cardiac output
followed immediately (5 seconds) after inflation of the antigravity suit and coincided with the improvement in the course of the syncope. Comparison with table 4 demonstrates the slightly more marked effect of antigravity suit inflation in syncope as compared to the normal standing posture. When compared to the cardiac index during syncope (table 1), the rise in cardiac index following antigravity suit inflation during syncope is significant ($p < .05$).

The mean peripheral resistance following antigravity suit inflation during syncope in 7 of the above subjects (819 units S.E. 96) was not significantly different from the mean peripheral resistance during unaltered syncope, despite the fact that the syncopal changes were reversed at the time of the postinfusion determinations.

In summary, antigravity suit inflation in the normal subject at 60° head-up tilt is accompanied by an increase in cardiac output and slight fall in peripheral resistance. In vasodepressor syncope, the prompt improvement after antigravity suit inflation is associated with an increased cardiac output and little or no change in peripheral resistance.

**Effects of Negative Pressure Breathing and Albumin Infusions**

Full-phase negative-pressure breathing (minus 16 to minus 19 mm. Hg) was maintained by having the subjects breathe from a counterweighted Tissot apparatus. A face mask was secured in place before the experimental tilt, and by a stopcock device the subjects could be maintained on atmospheric pressure until the maximum hypotension was reached. The apparatus was filled with 50 per cent oxygen in air. Carbon dioxide accumulation was prevented by allowing expired air to pass through a potassium hydroxide chamber before re-entering the negative-pressure chamber. In 3 subjects prompt recovery and reversal of the hypotension was noted when negative pressure was applied at the height of syncope, despite the fact that the subjects were maintained in the upright position.

In 3 additional subjects, all of whom experienced previous syncope, induction of syncope was attempted by the usual nitrite and tilt procedure immediately following completion of a rapid infusion (20 minutes) of 1000 ml. of 5 per cent albumin in saline. In none of the individuals was a syncopal or presyncopeal change noted, despite the fact that each was maintained at 60° tilt for 30 minutes following nitrite administration. In 2 of these subjects syncope was subsequently induced by the tilt-nitrite procedure.

**Effect of Atropine**

In 5 individuals 1.6 to 2.4 mg. of atropine were injected into the central circulation via the venous catheter 2 to 5 minutes prior to the onset of syncope induced by the tilt-nitrite procedure. Dramatic hastening of the pulse indicative of vagal blockade occurred in each case. Syncope progressed in the usual manner, despite the absence of bradycardia. Cardiac output determinations at the time of syncope in these subjects revealed a mean cardiac index of 2.5 L per minute per M.² (S.E. ±0.1), and demonstrated no significant deviation from the cardiac index during syncope in the non-atropinized subjects.

**Arterial Carbon Dioxide**

In 10 subjects arterial carbon dioxide content was determined in the 60° head-up tilt position 1 to 3 minutes before, and again, at the height of syncope. The results revealed a uniform fall

**Table 5.—Cardiac Index in Vasodepressor Syncope after Antigravity Suit Inflation**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Before syncope (L./min./M²)</th>
<th>Syncope and inflation*</th>
</tr>
</thead>
<tbody>
<tr>
<td>RN</td>
<td>1.8</td>
<td>2.9</td>
</tr>
<tr>
<td>RK</td>
<td>2.8</td>
<td>4.2</td>
</tr>
<tr>
<td>LD</td>
<td>2.4</td>
<td>3.1</td>
</tr>
<tr>
<td>JM</td>
<td>2.7</td>
<td>4.4</td>
</tr>
<tr>
<td>CH</td>
<td>3.1</td>
<td>3.8</td>
</tr>
<tr>
<td>TP</td>
<td>2.1</td>
<td>2.2</td>
</tr>
<tr>
<td>TH</td>
<td>2.2</td>
<td>3.9</td>
</tr>
<tr>
<td>RD</td>
<td>2.3</td>
<td>2.5</td>
</tr>
<tr>
<td>Mean</td>
<td>2.4</td>
<td>3.4</td>
</tr>
</tbody>
</table>

All data obtained while subjects were in the 60° head-up tilt position.

* Data obtained during the first 30 seconds following inflation to 60 ± 5 mm. Hg body pressure during the syncopal reaction.
Table 6.—Arterial Carbon Dioxide Content in Vasodepressor Syncope

<table>
<thead>
<tr>
<th>Subject</th>
<th>Before syncope* (Vol.%</th>
<th>During syncope† (Vol.%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>JC</td>
<td>48.8</td>
<td>45.3</td>
</tr>
<tr>
<td>JH</td>
<td>45.8</td>
<td>41.7</td>
</tr>
<tr>
<td>JT</td>
<td>44.7</td>
<td>42.7</td>
</tr>
<tr>
<td>TP</td>
<td>43.1</td>
<td>39.2</td>
</tr>
<tr>
<td>DR</td>
<td>50.9</td>
<td>44.7</td>
</tr>
<tr>
<td>RF</td>
<td>47.8</td>
<td>37.2</td>
</tr>
<tr>
<td>CL</td>
<td>42.4</td>
<td>36.8</td>
</tr>
<tr>
<td>JL</td>
<td>46.9</td>
<td>43.1</td>
</tr>
<tr>
<td>DS</td>
<td>46.7</td>
<td>45.5</td>
</tr>
<tr>
<td>GW</td>
<td>46.4</td>
<td>43.4</td>
</tr>
</tbody>
</table>

Mean ................ 46.4 42.0
Sd† ................ 0.83
p ..................<.01

All samples obtained while subjects were in the
60° head-up tilt position.

* Samples drawn within 3 minutes before onset of
syncopeal changes.

† Samples drawn at the time of maximum hypo-
tension.

‡ Refers to standard error of the difference be-
tween means.

averaging 4.4 volumes per cent (p < .01)
(table 6).

Discussion

In the normal individual the arterial blood
pressure is maintained within narrow limits by a balance between the cardiac output and
the effective peripheral resistance. In the event
of a fall in peripheral resistance, the arterial
pressure is maintained by an increased cardiac
output. This response of the cardiac output to a
sudden fall in peripheral resistance has been
well demonstrated in individuals with large areas of reactive hyperemia,* and in patients
with arteriovenous fistula. A striking feature of
the observations on vasodepressor syncope
reported here is the failure of the cardiac output
to rise in the face of a decreased peripheral
resistance. It is upon the nature of this ap-
parent dissociation of the cardiac output and
peripheral resistance responses that our interest
is centered in the present study.

The absence of a compensatory rise in cardiac
output in the presence of a diminished pe-
ripheral resistance may be explained by reflex
myocardial inhibition or marked limitation on
the volume of blood available to the heart.

The observations on the effect of return to
recumbency demonstrate that the cardiac
output often increases above resting levels
during the recovery process, indicating a
removal of those factors responsible for the
restriction of the cardiac output. Since the
rise in cardiac output during the termination
of a syncopeal reaction could be accomplished
by removal of either or both of the aforemen-
tioned mechanisms restricting cardiac output,
it was necessary to evaluate the relative sig-
nificance of each factor individually by study-
ing the cardiac output responses under various
experimental conditions while the subjects were
maintained in the tilted posture.

Observations on the existence of myocardial
vagal inhibition10 suggested that a vago-
inhibitory effect might explain the cardiac
output response during syncope. In addition,
slowing of the heart, long recognized as oc-
curring during syncope, might of itself be a
restrictive factor on the heart's ability to
compensate. The effect of atropine on the
cardiac output response in syncope was there-
fore studied. Despite the presence of marked
tachycardia and apparent vagal blockade at
the height of syncope, the cardiac output
response to the fall in peripheral resistance was
no different from that in the nonatropinized
subjects. The factor of bradycardia therefore
does not appear to be the limiting one in
restricting the cardiac output. In addition,
atropine-sensitive cardio-inhibition probably
does not play a significant role in the cardiac
output response in syncope.

Attention was next directed to the second
possible mechanism of cardiac output restric-
tion. The effects of increasing the volume of
blood available to the heart by antigravity suit
inflation, negative-pressure breathing, and
albumin infusion were studied. Each of these
factors was found either to abort or to prevent
the hypotensive phase of syncope. Because of
the ease of experimental control and the
efficacy of its action, the antigravity suit was
studied most extensively. Inflation of the anti-
gravity suit resulted in termination of the
hypotension and simultaneous elevation of the
cardiac output. These changes occurred despite
the fact that the subjects were maintained in
the upright position, and strongly suggest that
the factor of limited venous inflow plays a significant role in the restriction of the cardiac output in syncope. Although the data concerning the effects of situations designed to enhance venous inflow to the heart do not eliminate the possibility of concomitant neurogenic myocardial inhibition, it appears unnecessary to invoke the latter factor as an additional explanation for the diminished cardiac output response in syncope.

The fall in arterial carbon dioxide content is probably secondary to the hyperventilation during syncope. Hypocapnia secondary to hyperventilation has been demonstrated to result in mild to moderate hypotension, increased forearm blood flow, and diminished cerebral blood flow.\textsuperscript{10, 11} Despite these changes, all of which are well known components of the circulatory picture of vasodepressor syncope, acute hyperventilation alone uncommonly produces typical vasodepressor syncope. It is thought therefore that the hypocapnia during syncope is an important factor in accentuating the circulatory embarrassment, but probably is not an essential component of the reaction. Supporting this view is the finding that inhalation of 5 per cent carbon dioxide tends to diminish or eliminate the hypocapnia without otherwise altering the syncopal reaction.\textsuperscript{10}

The observed fall in total peripheral resistance in our subjects is of interest in light of studies on specific vascular beds. Although blood flow effects have varied, the syncopal reaction has been found to result in a decreased vascular resistance in the muscle,\textsuperscript{14} splanchnic,\textsuperscript{15} and renal\textsuperscript{18} circulations.

Engel and Romano\textsuperscript{17} have suggested that fainting represents an incomplete response in the preparation for flight occurring in situations where muscular activity is repressed. The physiologic adaptations for motion are thus incomplete, with resultant circulatory disorganization. The findings reported in this paper are consistent with this thesis.

From a circulatory standpoint, therefore, fainting might be characterized by a series of events initiated by a precipitous fall in peripheral resistance and arterial blood pressure occurring in the face of a situation where the heart does not compensate by increased output. Although the present study has focused attention on these circulatory aspects of syncope, it is clear that other facets, such as emotional and humoral factors, are involved.

**Summary**

Postural syncope can be produced with facility by 60° head-up tilt with the aid of sodium nitrite. Syncope so induced is similar to spontaneous vasodepressor syncope, and offers an experimental technic for the study of regulation of blood pressure and cardiac output. The cardiac output in vasodepressor syncope tends to fall slightly. During recovery, the cardiac output rises at times to supernormal levels. Of particular interest is the failure of the cardiac output to compensate for the fall in peripheral resistance during syncope. The occurrence of either neurogenic myocardial inhibition or markedly limited venous inflow were investigated as possible explanations for this finding.

Atropinization prevents the relative bradycardia during syncope, but does not alter the cardiac output response.

Antigravity suit inflation or negative-pressure breathing rapidly reverses the syncopal reaction. The improvement following antigravity suit inflation is associated with a rise in cardiac output and insignificant changes in peripheral resistance. Albumin infusions were found to prevent syncope.

The arterial carbon dioxide content falls at the height of syncope. This fall is probably secondary to the hyperventilation during syncope, and contributes to the circulatory embarrassment by further lowering peripheral resistance and cerebral blood flow.

The major circulatory event of vasodepressor syncope would appear to be not only widespread loss of peripheral resistance, but its occurrence in the face of inability of the heart to compensate by an increase in output. Present evidence favors the causative role of limited inflow in restricting the response of cardiac output.

**Summario in Interlingua**

Syncope postural es facile a producer per basculation a 60° (capite in alto) con le adjuta de nitrito de natrium. Le syncope assi induce es simile al spontanee syncope vasodepressori e
vasodepressor syncope

representa un technica experimental pro le studio del regulation de pression sanguine e rendimento cardiac.

In syncope vasodepressori le rendimento cardiac tende a reduce se levemente. Durante le recovramento le rendimento cardiac cresce a vices usque a nivellos supernormal. De interesse special es le facto que le rendimento cardiac non compensa le reduce resistentia peripheric que ocurre durante le syncope. Esseva investigate, como explicationes possibile de ille phenomeno, le occurrentia de neurogene inhibition myocordial o de un marcate limitation del influxo venose.

Atropinisation preveni le bradycardia relativa durante le syncope, sed illo non altera le responsa del rendimento cardiac.

Le pression exercite per le inflation de un costume de gravitate o le effectuation de respiration de pression negative reverte le reaction syncope rapidemente. Le melioration que ocurre post le inflation del costume de gravitate es associate con un augmento del rendimento cardiac e non-significative alteratones del resistentia peripheric. Esseva trovate que infusiones de albumina preveni le syncope.

Le contento arterial de bioxydo de carbon se reduce al culmine del syncope. Iste reduction es probabilmente secundari al hyperventilation durante le syncope e contribue al disturbance circulatori per causar un reduction additional del resistentia peripheric e del fluxo de sanguine cerebral.

Le major evento circulatori in syncope vasodepressori es apparentemente non solo le perdisa extense de resistentia peripheric sed le occurrentia de iste perdisa in association con le incapacitate del corde de efectuar un compensation per augmentar su rendimento. Le datos nune disponibile pare attribuer un rolo causative al reduce influxo in le restriction del responsa del rendimento cardiac.

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