Exertional Hypotension in Cardiac Patients

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Hemodynamic studies during steady states of sitting and walking on a treadmill have shown a fall in mean arterial pressure in 10 of 16 cardiac patients. This was associated with a greater increase in peripheral resistance during sitting, and a failure to increase stroke index during running. Cardiac index during exercise increased somewhat, largely because of greater acceleration of heart rate.

In contrast to the expected rise in systolic pressure with exercise, in a previous study,1 hypotensive responses were often recorded in 168 cardiac patients who were submitted to a test of exercise tolerance. Under the circumstances of the test, which required walking on a treadmill at 1.7 m.p.h. up a 10 per cent grade, normal subjects increased systolic pressure by a least 10 per cent of the pressure recorded during the fourth minute of sitting before standing and walking. Consequently “exertional hypotension” was defined as a failure to increase systolic pressure during walking by at least 10 per cent of the final value while sitting at rest; in some instances during exercise the pressure fell below the resting level. Forty-four per cent of patients with stenosis of either the mitral or aortic valves had exertional hypotension, as did 22 to 28 per cent of patients with either hypertensive vascular disease or coarctation of the aorta. The incidence was lower in patients who had flow loads due to either valvular regurgitation or to left-to-right shunts.

In reviewing previous experimental studies, Skouby pointed out that direct recordings of arterial pressure in horses and dogs during work on a treadmill sometimes showed a fall in mean pressure.2 Skouby studied direct recordings of aortic pressure in dogs as they ran on a treadmill. Despite initial oscillations and transient falls of 10 mm. for less than 10 seconds, both systolic and diastolic pressures increased in parallel with the rise in oxygen consumption. Holmgren3 also observed oscillations in pressure in human subjects during the initial phase of exercise, which he attributed to effects of immediate vasodilatation and decreased ventricular blood volume. Fraser and Chapman4 observed in normal subjects an average rise in systolic pressure of 15 mm. Hg, an average fall in diastolic pressure of 7 mm. Hg, and no change in mean systemic pressure when arterial pressures were recorded directly during standing and during walking on a treadmill at 3 m.p.h. up a 5-per-cent grade. The same authors reported that 1 out of 9 patients with a healed myocardial infarction had a fall in mean arterial pressure during the same work load.5 Donald and associates also noted a fall in systolic pressure in 1 out of 16 patients with mitral valve disease who were studied while they performed leg exercises in the supine position.6 In both of these instances, cardiac index increased somewhat during exercise.

Because of the paucity of directly recorded hemodynamic data on cardiac patients during exercises in the upright posture, further studies were made to determine the mechanisms of exertional hypotension.

Material and Methods

Twenty-six patients were studied, of which 16 were placed in group I because they either exhibited no change or showed an increase in mean arterial pressure during exertion; 10 were placed in group II because they demonstrated a decrease in mean arterial pressure during exertion. The clinical and physical characteristics of these 2 groups are presented in table 1. An additional

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### Table 1.—Physical Characteristics, Cardiovascular Diagnosis, Preliminary Evaluation of Exercise Tolerance and Blood Volume

<table>
<thead>
<tr>
<th>Group</th>
<th>Patients</th>
<th>Age</th>
<th>Sex</th>
<th>Wgt. (Kg.)</th>
<th>Weight area (M²)</th>
<th>Cardiac diagnosis</th>
<th>Rhythm</th>
<th>Functional capacity</th>
<th>ECG</th>
<th>Symptoms</th>
<th>Max. sys. pres. (mmHg)</th>
<th>Max. HR</th>
<th>PFI</th>
<th>Bl. vol. (ml/Kg.)</th>
<th>Sedation for cardiac catheterization</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>R.D.</td>
<td>61</td>
<td>M</td>
<td>73.5</td>
<td>1.88</td>
<td>HMI</td>
<td>NS</td>
<td>II</td>
<td>2+</td>
<td>O</td>
<td>30</td>
<td>140</td>
<td>20</td>
<td>77</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>V.W.</td>
<td>41</td>
<td>M</td>
<td>58.6</td>
<td>1.40</td>
<td>HCVD</td>
<td>NS</td>
<td></td>
<td></td>
<td></td>
<td>30</td>
<td>140</td>
<td>20</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>B.W.</td>
<td>36</td>
<td>M</td>
<td>65.6</td>
<td>1.78</td>
<td></td>
<td>NS</td>
<td>III</td>
<td>3+</td>
<td>ST</td>
<td>54</td>
<td>135</td>
<td>7</td>
<td>80</td>
<td>+</td>
</tr>
</tbody>
</table>

A.L. 47 F 59.0 1.70 HCVD NS III 2+ T 30 88 †4.2 74 +

K.S. 36 F 51.8 1.52 MS 1+ ST 20 146 8.3 62 +

K.E. 29 F 57.3 AR NS II 2+ 42 175 10.7 57 +

A.S. 51 M 80.5 1.96 MR NS II 4+ VPS 10 138 †2.6 — +

J.S. 45 F 84.0 2.05 HMI NS I 0 T† 38 68 26.2 63 +

W.H. 16 M 56.0 1.65 AR NS II 0 15 116 14.3 58 +

T.L. 47 M 105.0 2.20 HMI NS II 0 12 100 16.1 40 +

F.C. 57 M 66.4 1.77 MS NS II 0 32 114 8.1 72 +

V.L. 23 M 89.1 2.11 AS NS I* 0 — 30 117 23.2 66 +

E.He. 22 M 77.0 1.85 HCVD NS I* 0 T† 35 100 22.0 70 +

M.E. 34 F 60.0 1.66 MS NS II 1+ 24 115 17.6 67 +

M.E. 30 F 60.0 1.66 MS NS II 3+ 20 108 19.2 63 +

Mean 38.6 70.3 30 120 13.9 64.6±10.2 +

Mean 42.2 64.1 1.71 9.4 146 60.4±10.6 +

*Not digitalized.
†Poor motivation.

HMI, healed myocardial infarction; HCVD, hypertensive cardiovascular disease; AS, aortic stenosis; MS, mitral stenosis; AR, aortic regurgitation; MR, mitral regurgitation; NS, normal sinus rhythm; AF, atrial fibrillation; Max. HR, maximal heart rate; PFI, physical fitness index.

5 patients who were sedated became weak or dizzy on sitting up and were eliminated from the study.

A preliminary test of exercise tolerance was done on each patient to determine blood pressure response by means of a sphygmomanometer, and to note any symptoms or signs resulting from exertion. Cardiorespiratory performance was expressed in terms of the Physical Fitness Index.7

Thirteen patients were sedated with 25 mg. of meperidine hydrochloride (Demerol), and 25 mg. of dimenhydrinate (Dramamine) in preparation for surgical exposure of the median basilic vein and the radial artery under local anesthesia with procaine. They were then given 25 mg. of ephedrine sulfate intramuscularly to prevent postural hypotension during sitting and standing as a result of previous sedation. The remaining patients received only 1 ml. of saline subcutaneously as a placebo.

A polyethylene catheter (PE 90) was inserted into the radial artery, and the venous catheter (6F) was passed into the pulmonary artery.
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TABLE 2.—Effects of Upright Posture and Exercise (Mean ± Standard Deviation and Probability of Difference between Means Being due to Chance Alone*)

<table>
<thead>
<tr>
<th></th>
<th>Group†</th>
<th>Lying supine</th>
<th>Sitting</th>
<th>p</th>
<th>Walking</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventilation (L/min.)</td>
<td>I</td>
<td>7.2±1.8</td>
<td>11.0±2.5</td>
<td>&lt;.001</td>
<td>26.7±7.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>7.9±3.0</td>
<td>10.0±3.1</td>
<td></td>
<td>26.6±7.8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>O₂ removal rate (ml/L)</td>
<td>I</td>
<td>39.5±14.0</td>
<td>28.0±6.6</td>
<td>&lt;.006</td>
<td>34.5±10.6</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>34.5±12.5</td>
<td>28.0±7.3</td>
<td></td>
<td>30.7±13.9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>O₂ consumption index (ml/min./M²)</td>
<td>I</td>
<td>151±36</td>
<td>164±19</td>
<td></td>
<td>487±104</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>150±56</td>
<td>153±26</td>
<td></td>
<td>442±45</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Arterial O₂ content (vol. %)</td>
<td>I</td>
<td>17.3±2.8</td>
<td>18.4±2.3</td>
<td></td>
<td>18.6±2.7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>16.9±1.9</td>
<td>17.9±1.6</td>
<td></td>
<td>18.2±1.6</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mixed venous O₂ content (vol. %)</td>
<td>I</td>
<td>12.4±3.8</td>
<td>11.7±2.5</td>
<td></td>
<td>8.4±2.7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>11.3±2.1</td>
<td>10.7±2.0</td>
<td></td>
<td>5.8±1.7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>A-V O₂ difference (ml/L)</td>
<td>I</td>
<td>52±10</td>
<td>66±14</td>
<td>&lt;.001</td>
<td>101±27</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>56±4</td>
<td>71±18</td>
<td></td>
<td>124±27</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cardiac index (L/min./M²)</td>
<td>I</td>
<td>2.95±0.59</td>
<td>2.56±0.58</td>
<td></td>
<td>5.05±0.46</td>
<td>&lt;.001</td>
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<tr>
<td></td>
<td>II</td>
<td>2.73±0.94</td>
<td>2.22±0.47</td>
<td></td>
<td>3.61±0.35</td>
<td>&lt;.001</td>
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<tr>
<td>Heart rate</td>
<td>I</td>
<td>71±14</td>
<td>83±17</td>
<td></td>
<td>112±21</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>71±18</td>
<td>80±16</td>
<td></td>
<td>129±31</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Stroke index (ml/M²)</td>
<td>I</td>
<td>43±10</td>
<td>32±9</td>
<td>&lt;.001</td>
<td>50±6</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>40±14</td>
<td>29±7</td>
<td></td>
<td>30±13</td>
<td></td>
</tr>
<tr>
<td>Mean pulm. press. (mm Hg)</td>
<td>I</td>
<td>23±11</td>
<td>24±14</td>
<td></td>
<td>35±19</td>
<td></td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>25±10</td>
<td>26±14</td>
<td></td>
<td>36±22</td>
<td></td>
</tr>
<tr>
<td>Mean syst. press. (mm Hg)</td>
<td>I</td>
<td>108±25</td>
<td>115±22</td>
<td></td>
<td>131±22</td>
<td></td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>102±24</td>
<td>124±26</td>
<td></td>
<td>114±28</td>
<td></td>
</tr>
<tr>
<td>Total pulm. resist.‡</td>
<td>I</td>
<td>65±42</td>
<td>75±42</td>
<td></td>
<td>65±53</td>
<td></td>
</tr>
<tr>
<td>dyne sec. cm.⁻⁵ × M²</td>
<td>II</td>
<td>80±38</td>
<td>107±82</td>
<td></td>
<td>95±74</td>
<td></td>
</tr>
<tr>
<td>Total syst. resist.‡</td>
<td>I</td>
<td>303±84</td>
<td>375±34</td>
<td>&lt;.002</td>
<td>228±84</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>dyne sec. cm.⁻⁵ × M²</td>
<td>II</td>
<td>325±107</td>
<td>463±121</td>
<td>&lt;.007</td>
<td>276±139</td>
<td>&lt;.002</td>
</tr>
</tbody>
</table>

*In changing from lying to sitting, or from sitting to walking, for the same group of patients.
†I, 16 patients with normal pressor response to exercise; II, 10 patients with hypotensive response to exercise.
‡Expressed as 1/10 of calculated values.

Cardiac outputs were determined by direct Fick technic during steady states of sitting and walking. Oxygen consumption was measured with a 13-liter respirometer filled with 100-per cent oxygen. Blood oxygen contents were determined by the Van Slyke-Neill method.

Blood volume was estimated from T-1824 plasma concentration, at equilibrium, 2 to 3 minutes after directly recording the primary dilution curve with an ear oximeter and logarithmic amplifier;³ venous hematocrit was used without any correction for trapped plasma.

Blood pressures were recorded with Statham P23D transducers and a Sanborn oscillograph. Zero reference levels were located 10 cm. above the table when the patient was supine, and at the level of the fourth interspace anteriorly when the patient was upright. Transducers were taped to the arm, which was held dependent, to maintain constant positions during walking. Catheters were flushed at frequent intervals to prevent damping. No corrections were made for respiratory variations, and mean pressures of more than one respiratory cycle were determined by planimetric integration.

Pulmonary and systemic resistances were calculated from mean pressure and flow corrected for body surface areas, as follows:

\[ \text{Resistance Index (dynes sec. cm.}^{-5} \times M^2) = \frac{\text{Mean pressure} \times 1332 \times 60}{\text{Cardiac index}} \]
A Valsalva maneuver, standardized by holding orotracheal air pressure at 40 mm. Hg for 10 seconds, was recorded in several patients to observe responses in systemic arterial pressure during phases II and IV.

**RESULTS**

The results observed in 13 sedated and 13 nonsedated patients were combined and subdivided into 2 groups according to the pressor response to walking. Since 6 nonsedated patients developed exertional hypotension in contrast to only 4 of the sedated patients who were able to sit up, it is likely that sedation selectively eliminated several of the more vulnerable patients from complete study.* The hemodynamic effects of the upright posture and exercise will be described in relation to

*Tabulation of all individual data in a mimeographed appendix will be supplied on request.

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**Fig. 1.** Comparison of average percentage changes in 26 cardiac patients as a result of sitting upright and of walking. Patients in group I and II were separated on basis of change in mean arterial pressure from sitting to walking; note significance of changes in stroke and cardiac indices.

**Fig. 2.** Variations in arterial pressures observed during standing and walking in 3 different patients. Arrows indicate level of mean arterial pressure.
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Fig. 3. Two examples of exertional hypotension (top, M. H.; bottom, J. R.). Note the full in systolic as well as mean arterial pressure.

16 patients with a normotensive, and 10 patients (38 per cent) with a hypotensive response to exercise in the upright posture.

Effects of Sitting and Standing at Rest

Several adaptations in circulation occurred as a result of changing from the supine to a sitting position (table 2, fig. 1). Presumably, venous pooling affected venous return, widened the arteriovenous oxygen difference about 25 per cent, and lowered the cardiac index. There was a significant reduction in stroke index of more than 25 per cent, and only a slight acceleration of heart rate. Although the magnitude of increased pressure and peripheral resistance was greater in group II patients, the differences were not significant (p < .07 and .12 respectively).

Some patients exhibited slow oscillations, or undulations in systemic arterial pressure, when they stood up (fig. 2). These fluctuations were not well correlated with respiratory variations in pulmonary arterial pressure.

Fig. 4. Comparison of changes in average cardiac index, systemic and pulmonary arterial pressures, and total resistances as a result of sitting up and of walking.
Effects of Walking

For approximately a 3-fold increase in oxygen consumption above the level observed while supine, patients in group I almost doubled the cardiac index (table 2). They also showed about a 50 per cent increase in stroke index and arteriovenous oxygen difference in changing from sitting to walking (fig. 1). Since mean arterial pressure increased but slightly, the peripheral resistance showed about a 40 per cent fall due to vasodilation (fig. 3).

Patients in group II with exertional hypotension (figs. 3 and 4) showed a similar fall in resistance, but a significantly smaller increase in stroke index (p < .003) and cardiac index (p < .02) than did those in group I. The increase in arteriovenous oxygen difference was somewhat greater, but there were no appreciable differences in heart rate, oxygen consumption, ventilation, or oxygen removal rate.

Contributory Factors for Exertional Hypotension

The possible importance of abnormalities in total blood volume, distribution of blood volume (as reflected in pressor responses to the Valsalva maneuver), and mechanism of heart beat was reviewed.

There was neither a significant reduction in blood volume nor a significant difference in average total blood volume for patients in groups I and II (table 1).

Valsalva responses were recorded on 16 patients. Only the 2 hypertensive patients who were taking a sympathetic blocking agent orally showed any impairment of the post-Valsalva overshoot in diastolic pressure. There was no consistent difference in phase II responses between the 2 groups. Examples of maximal differences observed between patients in each group are shown in figure 5. Furthermore, there was no change in these responses.
when the Valsalva maneuver was repeated after the exercise test.

Three of 4 patients who had atrial fibrillation developed exertional hypotension. The exceptional patient, who increased mean arterial pressure during exertion (fig. 6), had predominantly mitral regurgitation. The diagnostic impression was confirmed later during open heart surgery (mitral annuloplasty performed by Dr. K. Alvin Merendino). Of the 3 others with atrial fibrillation and hypotensive responses to exertion, 2 had predominantly mitral stenosis; the other had mitral regurgitation and possibly myocarditis. Another patient, E. H., had normal sinus rhythm at rest, but exhibited a fall in mean arterial pressure due to the onset of ventricular bigeminy during exertion (fig. 6). Since he had a healed myocardial infarct, and was not treated with any digitalis preparation, this arrhythmia was presumably due to myocardial ischemia.

**Discussion**

Even though exertional hypotension has previously been observed directly in some experimental animals and indirectly in cardiac patients studied in this laboratory, the possibility of technical errors in these studies should not be disregarded without further comment. Due to the sampling of blood and flushing of catheters during the procedures, the possibility of artifactual damping is remote. Most important, however, is the fact that many patients exhibited a rebound to higher radial arterial pressures during recovery, as has been observed previously in...
older normal men by Fraser and Chapman.\textsuperscript{4}

Numerous mechanisms may contribute to a depressor response to muscular exercise. Undoubtedly neural regulation of vasomotor tone is important. This may be impaired by the syndrome of orthostatic hypotension, or by depression from the use of narcotics or sedatives.\textsuperscript{9} The latter was presumed responsible for eliminating 5 patients from complete studies, since without sedation, they could tolerate the upright posture and exercise in the preliminary test prior to cardiac catheterization. Further support of this is the fact that all of the nonsedated patients completed the experimental procedure. An effective blockade of peripheral ganglia may impair regulation of vasomotor tone also. Although 2 hypertensive patients (A.L. and L.M.) had been on prolonged therapy with pentolium bitartrate, one showed the hypotensive response to posture previously described by Smith and Hoobler\textsuperscript{10} while the other showed it in response to walking. Presumably marked reductions in blood volume from hemorrhage or dehydration also could assume major importance if neural regulation of vasomotor tone was not adequate. Pulmonary hypertension may be contributory in favoring a redistribution of the effective blood volume, but this could not be evaluated adequately from the 2 examples included in this study. Hypocapnia from hyperventilation may lower peripheral resistance,\textsuperscript{11} but the mean arterial carbon dioxide content during exercise of 43.0 volumes per cent for 4 patients with exertional hypotension was not significantly different from the mean (43.9 volumes per cent) for the other 9 patients who had lower levels of ventilation (23.1 versus 27.0 L./min.), and no fall in pressure.

Although the role of posture in relation to numerous cardiovascular syndromes has been reviewed by Silverman and Salomon, observations during physical activity in the upright posture were not available for analysis in their study.\textsuperscript{12} The cardiac patients in the present study who exhibited a depressor response to exercise usually demonstrated a greater pressor response to changing from supine to sitting posture. This was in compensation for a somewhat greater reduction in cardiac index. The decreased peripheral resistance from vasodilatation secondary to muscular exercise in individuals in group II was comparable to that observed in other cardiac patients in group I who had a pressor response to exertion. Thus exertional hypotension was a physical sign of impaired cardiac reserve, primarily due to inability to increase the stroke index because of myocardial ischemia, dilatation, valvular stenosis, or ventricular bigeminy. Tachycardia, although it increased cardiac index, was not a sufficient compensation for this defect. In fact, if tachycardia were excessive, particularly with atrial fibrillation, it would further limit stroke by the greater reduction in diastolic filling time. Valvular regurgitation, when not associated with significant cardiac dilatation, myocardial disease or depression of neural regulation of vasomotor tone, was unlikely to cause exertional hypotension. Similarly, a previous study of 13 congenital cardiac patients with left-to-right shunts and no significant myocardial disease did not demonstrate exertional hypotension.\textsuperscript{13}

**Summary and Conclusions**

Exertional hypotension, or a fall in mean systemic arterial pressure during walking, occurred in 10 of 26 patients tested with indwelling catheters and direct recordings of pressure in pulmonary and radial arteries.

Patients who developed exertional hypotension had slightly increased peripheral resistance and lowered cardiac indices while sitting at rest, and a marked limitation of stroke index during walking. Cardiac index usually increased somewhat due to acceleration of heart rate.

It is concluded that exertional hypotension may be a significant manifestation of impaired cardiac function whenever a major loss in blood volume or a marked impairment of neural regulation of vasomotor tone can be excluded.

**Summario in Interlingua**

Hypotension de effortio, i.e. un reduction del valor medie del tension arterial systemie in ambulation, occurreva in 10 ex 26 patientes
testate con catheteres in sito e registration
directe del tension in arterias pulmonar e
radial.

Patientes qui disveloppava hypotension de
effortio habeva levemente augmentate resis-
tentias peripheric e reducece indices cardiae
quando illes sedeva in stato de reposo e un
marecate limitation del indice de pulso quand
illes ambulava. Le indice cardiae cres-
ceva in general levemente in consequentia de
un augmento del frequentia cardiae.

Es concludite que hypotension de effortio
es possibilemente un manifestation significative
de dysfunction cardiae in omne casos in
que il es possibile escluder le explication per
major perditas de sanguine o per marcate
dysfunction del regulation neural del tono
vasomotori.

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