Diagnostic Signs in Compressive Cardiac Disorders

Constrictive Pericarditis, Pericardial Effusion, and Tamponade

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The combination of two physical signs, increasing venous pressure and palpable decrease in arterial systolic and pulse pressure with inspiration in constrictive pericarditis was pointed out by Kussmaul. Wood alluded to these signs as evidence for pericardial effusion and attributed the rise in venous pressure to an inspiratory rise in intrapericardial pressure. Dock subscribed to this view, supporting his hypothesis by studies in cadavers. A recent monograph by Spodick treating compressive cardiac disease and encompassing chronic constrictive pericarditis and pericardial effusion, reported that Kussmaul’s venous sign is present in pericardial effusion.

In contrast, Hitzig examined the venous pressure in constrictive pericarditis, right heart failure, and a small number of patients with pericardial effusion and tamponade. Kussmaul’s venous and arterial signs were present in 25% of the patients with constrictive pericarditis; however, both constrictive pericarditis and congestive heart failure were associated with a rise in directly recorded venous pressure at the end of deep inspiration. Kussmaul’s venous sign was absent in pericardial effusion and tamponade. More recently, Lange and Tsagaris reported that intrapericardial and venous or right atrial pressure uniformly fell with normal or exaggerated inspiration in patients with effusion and tamponade. Subsequently, Morgan and associates reported similar findings in animal studies.

The work cited suggests that discrimination between various disorders of cardiac function due to extracardiac compression might be possible by clinical criteria. Therefore, systematic investigation of the physical findings, extended when possible by appropriate laboratory procedures, was carried out on 37 patients. Sixteen had constrictive pericarditis, seven had pericardial effusion without circulatory compromise, seven suffered from cardiac tamponade, and seven exhibited physical findings suggestive of compressive heart disease but in these the disorder was related to respiratory disease, pancarditis, or extreme obesity. In the majority of cases without obliteration of pericardial space, intrapericardial pressures were measured. Correlation of physical findings and functional disorders were made.

Definition of Physical Findings and Related Pressure Phenomena

1. Kussmaul’s venous sign—a regular inspiratory rise in venous pressure with tranquil breathing observed in the cervical veins or recorded in the right atrium and subclavian veins.
2. Friedreich’s sign—an early diastolic pres-
Table 1

**Clinical and Hemodynamic Findings in Constrictive Pericarditis (Group I)**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr) &amp; Sex</th>
<th>Etiology</th>
<th>Venous pressure, mm Hg</th>
<th>Rhythm</th>
<th>ECG Voltage</th>
<th>Third heart sound</th>
<th>Friedreich's sign</th>
<th>Kussmaul's sign</th>
<th>Pericardial calcification</th>
<th>Respiratory vari.* mm Hg</th>
<th>Diagnosis</th>
<th>Cardiac index</th>
</tr>
</thead>
<tbody>
<tr>
<td>H.W.</td>
<td>57 M</td>
<td>Idiopathic</td>
<td>18</td>
<td>AF</td>
<td>L</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>12</td>
<td>S</td>
<td>1.9</td>
</tr>
<tr>
<td>R.B.</td>
<td>55 F</td>
<td>Tuberculosis</td>
<td>8</td>
<td>AF</td>
<td>N</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>15</td>
<td>S</td>
<td>1.8</td>
</tr>
<tr>
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<td>Tuberculosis</td>
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<td>AF</td>
<td>N</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
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<td>S</td>
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<tr>
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<td>Tuberculosis</td>
<td>16</td>
<td>Sinus</td>
<td>L</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>S</td>
<td>1.7</td>
<td></td>
</tr>
<tr>
<td>H.B.</td>
<td>61 M</td>
<td>Tuberculosis</td>
<td>14</td>
<td>AF/sinus</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
<td>S</td>
<td>1.6</td>
</tr>
<tr>
<td>L.G.</td>
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<td>Post surgery</td>
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<td>AF</td>
<td>L</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>13</td>
<td>PM</td>
<td>1.9</td>
</tr>
<tr>
<td>G.B.</td>
<td>10 M</td>
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<td>18</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>PM</td>
<td>2.6</td>
</tr>
<tr>
<td>W.B.</td>
<td>62 M</td>
<td>Idiopathic</td>
<td>10</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>8</td>
<td>S</td>
<td>1.9</td>
</tr>
<tr>
<td>R.J.</td>
<td>26 M</td>
<td>Idiopathic</td>
<td>9</td>
<td>Sinus</td>
<td>L</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>12</td>
<td>S</td>
<td>3.3</td>
</tr>
<tr>
<td>J.C.</td>
<td>75 M</td>
<td>Idiopathic</td>
<td>17</td>
<td>AF/sinus</td>
<td>L</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>10</td>
<td>—</td>
<td>1.3</td>
</tr>
<tr>
<td>T.K.</td>
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<td>Tuberculosis</td>
<td>14</td>
<td>AF</td>
<td>N</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>8</td>
<td>—</td>
<td>3.9</td>
</tr>
<tr>
<td>H.M.</td>
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<td>AF</td>
<td>N</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>12</td>
<td>—</td>
<td>2.7</td>
</tr>
<tr>
<td>W.M.</td>
<td>74 M</td>
<td>Idiopathic</td>
<td>15</td>
<td>AF</td>
<td>N</td>
<td>+</td>
<td>+</td>
<td>+†</td>
<td>+</td>
<td>2</td>
<td>—</td>
<td>2.3</td>
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<tr>
<td>C.C.</td>
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<td>Tuberculosis</td>
<td>18</td>
<td>AF</td>
<td>L</td>
<td>+</td>
<td>+</td>
<td>+†</td>
<td>+</td>
<td>6</td>
<td>—</td>
<td>2.4</td>
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<tr>
<td>C.F.</td>
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<td>Idiopathic</td>
<td>18</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>14</td>
<td>S</td>
<td>2.6</td>
</tr>
<tr>
<td>R.H.</td>
<td>45 M</td>
<td>Idiopathic</td>
<td>27</td>
<td>Sinus</td>
<td>L</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>16</td>
<td>S, PM</td>
<td>2.7</td>
</tr>
</tbody>
</table>

*Respiratory variation in pulse pressure in brachial or femoral artery.
†With exercise only.
Abbreviations: Voltage: N - normal; L - low; diagnosis: S - surgery; PM - postmortem.
<table>
<thead>
<tr>
<th>Case</th>
<th>Age, yr &amp; sex</th>
<th>Etiology</th>
<th>Venous pressure, mm Hg</th>
<th>Rhythm</th>
<th>ECG Voltage</th>
<th>Pulsus paradoxous</th>
<th>Kussmaul's sign</th>
<th>Diagnosis</th>
<th>Pericardial pressure, mm Hg</th>
<th>Control</th>
<th>Cardiac index</th>
<th>After tap</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.B.</td>
<td>65 F</td>
<td>Myxedema</td>
<td>10</td>
<td>NSR</td>
<td>N</td>
<td>0</td>
<td>0</td>
<td>A</td>
<td>--</td>
<td>2.0*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.T.</td>
<td>73 F</td>
<td>Myxedema</td>
<td>6</td>
<td>NSR</td>
<td>L</td>
<td>0</td>
<td>0</td>
<td>A,T</td>
<td>--</td>
<td>1.75*</td>
<td></td>
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<td>T.R.</td>
<td>29 M</td>
<td>Projectile wound</td>
<td>5</td>
<td>NSR</td>
<td>N</td>
<td>0</td>
<td>0</td>
<td>S,E,T</td>
<td>--</td>
<td>4.0</td>
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<td>4.0</td>
</tr>
<tr>
<td>V.A.</td>
<td>27 F</td>
<td>Idiopathic pericardium</td>
<td>10</td>
<td>NSR</td>
<td>N</td>
<td>+</td>
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<td>2</td>
<td>3.6</td>
<td></td>
<td>3.8</td>
</tr>
<tr>
<td>P.B.</td>
<td>7 M</td>
<td>Post cardiotomy</td>
<td>11</td>
<td>NSR</td>
<td>N</td>
<td>0</td>
<td>0</td>
<td>T</td>
<td>10</td>
<td>--</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.B.</td>
<td>4 F</td>
<td>Post cardiotomy</td>
<td>11</td>
<td>NSR</td>
<td>N</td>
<td>0</td>
<td>0</td>
<td>T</td>
<td>7</td>
<td>3.75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.S.</td>
<td>56 F</td>
<td>Myxedema</td>
<td>10</td>
<td>NSR</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>A</td>
<td>--</td>
<td>1.56*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Cardiac index was reduced proportionally with minute oxygen intake.

Abbreviations: Rhythm: NSR-normal sinus rhythm; Voltage: N-normal; L-low; diagnosis: A-angiography; E-ultrasonic echo; S-scan after intravenous administration of radioisotope; T-pericardiocentesis.

Table 2

Clinical and Hemodynamic Findings in Lax Effusion (Group IIA)

Group II Pericardial Effusion

In the cases in group IIA, pleural fluid was present in the pericardial space but it was not unusual for patients to have had signs of pericardial disease. Pericardial effusion was made when the pericardial space was greater than 20 mm Hg. This group had pericardial effusion. The diagnosis of cardiac tamponade was made by ultrasonic devices. Pericardial effusion was not followed by pericardiocentesis.

Table 3 provides a summary of clinical and laboratory findings in group II.

<table>
<thead>
<tr>
<th>Category of Patients</th>
<th>Criteria</th>
<th>Patients</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericardial tamponade</td>
<td>Criteria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac pain</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cardiac tamponade</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac murmur</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3. Pulses paradoxus—palpable decrease in strength of pulse of 20 mm Hg or greater in systolic pressure by sphygmomanometry or direct recording during inspiration or expiration.

4. Third heart sound suggested by Cohn and Katz.

5. Palpable pericardial knock as per the criteria suggested by Mecklenburg and Mosses. Timing for sound was by phonocardiogram.
### Table 3

**Clinical and Hemodynamic Findings in Tamponade (Group IIIB)**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, yr, &amp; sex</th>
<th>Etiology</th>
<th>Venous pressure, mm Hg</th>
<th>ECG Rhythm</th>
<th>ECG Voltage</th>
<th>Pulsum paradoxus</th>
<th>Kussmaul's sign</th>
<th>Diagnosis</th>
<th>Pericardial pressure, mm Hg</th>
<th>Control</th>
<th>Cardiac index</th>
<th>After tap</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.M.</td>
<td>67 M</td>
<td>Metastatic carcinoma</td>
<td>25</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>T</td>
<td>21</td>
<td>1.7</td>
<td>3.0</td>
<td></td>
</tr>
<tr>
<td>A.L.</td>
<td>29 M</td>
<td>Stab wound</td>
<td>20</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>T</td>
<td>18</td>
<td>1.6</td>
<td>2.4</td>
<td></td>
</tr>
<tr>
<td>M.Y.</td>
<td>39 F</td>
<td>Cardiac catheterization</td>
<td>28</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>T</td>
<td>26</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>C.B.</td>
<td>80 F</td>
<td>Idiopathic pericardium</td>
<td>19</td>
<td>AF</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>E,T</td>
<td>16</td>
<td>1.5</td>
<td>2.3</td>
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</tr>
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<td>C.K.</td>
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<td>Acute right failure</td>
<td>18</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>T</td>
<td>15</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>S.L.</td>
<td>65 F</td>
<td>Dissecting aneurysm</td>
<td>22</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>E,T</td>
<td>18</td>
<td>1.9</td>
<td>1.9</td>
<td></td>
</tr>
<tr>
<td>G.L.</td>
<td>45 M</td>
<td>Cardiac catheterization</td>
<td>16</td>
<td>Sinus</td>
<td>N</td>
<td>0</td>
<td>0</td>
<td>A,T</td>
<td>—</td>
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</tr>
</tbody>
</table>

Abbreviations: Voltage: N - normal; diagnosis: E - ultrasonic devices; A - angiography; T - pericardiocentesis.

### Table 4

**Clinical and Hemodynamic Findings in Groups IIIA, IIIB, IIC**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, yr, &amp; sex</th>
<th>Etiology</th>
<th>Venous pressure, mm Hg</th>
<th>ECG Rhythm</th>
<th>ECG Voltage</th>
<th>Pulsum paradoxus</th>
<th>Kussmaul's sign</th>
<th>Diagnosis</th>
<th>Pericardial pressure, mm Hg</th>
<th>Control</th>
<th>Cardiac index</th>
<th>After tap</th>
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<tbody>
<tr>
<td><strong>Group IIIA</strong></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>T.C.</td>
<td>71 M</td>
<td>Emphysema</td>
<td>5</td>
<td>Sinus</td>
<td>L</td>
<td>+</td>
<td>0</td>
<td>A,T</td>
<td>5</td>
<td>4.0</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td>S.K.</td>
<td>66 M</td>
<td>Emphysema</td>
<td>5</td>
<td>AF</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>T*</td>
<td>2</td>
<td>2.6</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td><strong>Group IIIB</strong></td>
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<td></td>
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<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>L.M.</td>
<td>38 M</td>
<td>Pancarditis</td>
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<td>Sinus</td>
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<td>0</td>
<td>0</td>
<td>T</td>
<td>22</td>
<td>2.3</td>
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<td>A.S.</td>
<td>37 M</td>
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<td>AF</td>
<td>L</td>
<td>0</td>
<td>0</td>
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<td>2.3</td>
<td>2.3</td>
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</tr>
<tr>
<td>R.P.</td>
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<td>Chronic myocarditis</td>
<td>5</td>
<td>Sinus</td>
<td>N</td>
<td>0</td>
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<td>A,E</td>
<td>—</td>
<td>2.7</td>
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<td><strong>Group IIC</strong></td>
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<td></td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>R.W.</td>
<td>52 M</td>
<td>Obesity</td>
<td>13</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>2.6</td>
<td>—</td>
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</tr>
<tr>
<td>J.R.</td>
<td>55 M</td>
<td>Obesity</td>
<td>10</td>
<td>Sinus</td>
<td>N</td>
<td>+</td>
<td>0</td>
<td>—</td>
<td>—</td>
<td>2.8</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

*Pericardial space entered during cardiac catheterization.*

Abbreviations: Voltage: N - normal; L - low; diagnosis: A - angiography; T - pericardiocentesis; E - ultrasonic echo.
carditis, and group IIIC patients were extremely obese.

In all cases of group I, right heart catheterization was carried out. In two of the seven cases in group IIA, right heart catheterization was accomplished. In all remaining cases, peripheral and central venous pressures and arterial pressure were measured. Cardiac output was measured in six cases. Pericardial pressures were obtained in five. In group IIB, venous, arterial, and peripheral pressures were recorded in all cases and pericardial pressures were measured in six of the seven cases. Cardiac output was estimated before and after tap in four. Right heart catheterization was carried out in group IIIA and B, and pericardial pressures were obtained. In group IIIC, right heart catheterization and arterial pressures were obtained, but pericardial pressures were not. Respiratory activity was indicated by mercury silastic gauge attached circumferentially about the chest, by esophageal balloon pressure, spirometry or impedance plethysmography.17

Pressures were measured by Statham (P23DB) gauges and optimally damped galvanometers. The zero reference point for pressure was at the mid-chest at the level of the second rib anteriorly. Either the Fick principle was applied for a cardiac output or indocyanine green-dilution curves were recorded by a compensated densitometer (Waters Corporation). Blood samples were analyzed by the method of Neill and Van Slyke and expired air was analyzed by the Scholander method.

Studies were carried out with the patients supine. The patients undergoing pericardiocentesis were placed in a slightly head-elevated position and a no. 20 spinal needle was inserted into the subxiphoid area after local anesthesia. In patient 7 of group IIA, general anesthesia was used. Observations were made during tranquil or involuntary breathing and at times during a prolonged deep inspiratory action with the glottis open.

Representative Cases

Group I. Constrictive Pericarditis

The right heart phenomena are well recognized and the present work does not add to the previous descriptions.10, 11, 18

Group II.A. Lax Pericardial Effusion

Case T. R. The patient, a 29-year-old married Negro, had received several projectile wounds in the right chest and right humerus. Surgical drainage was carried out and a cast was applied to the right arm. During follow-up examination 8 days later, an enlarging cardiac silhouette was noted. This increased over the next 2 weeks. There was no evidence of circulatory embarrassment. Venous pressure did not exceed 6 mm Hg and pulsus paradoxus was not apparent. A pericardial friction rub was noted at times, and the electrocardiogram showed flattening and inversion of T waves. Effusion was confirmed by ultrasonic methods and by cardiac scan after intravenous injection by radioiodinated albumin. After venous and arterial cannulation and measurement of control cardiac output, pericardiocentesis allowed the removal of 250 ml of fluid having the appearance of old blood. Pericardial pressure was −2 mm Hg and dropped slightly after tap. Cardiac index remained unchanged at approximately 4.0. Subsequently, the heart silhouette reduced to normal. Figure 1 shows femoral artery and intrapericardial pressures before and after fluid removal. Tranquil respiration caused a fall in intrapericardial pressure. No diastolic dip was visible in the venous pressure recording.

Group IIB. Tamponade

Case S. L. (Table 3). A 65-year-old Negress entered the emergency ward complaining of dull, substernal chest pain. She had hypotension, elevated venous pressure, and pulsus paradoxus. Chest x-rays showed aortic dilatation. A presumptive diagnosis of aortic hematoma with dissection into the pericardial space was made and, after infusion of 1 unit of plasma, pressor therapy was no longer required. After venous and arterial cannulation, pressures were measured, and the cardiac index was estimated by indicator-dilution methods. Pericardiocentesis, with removal of 45 ml of blood, was carried out. Before fluid removal, venous pressure was 22 mm Hg and intrapericardial pressure was 18 mm Hg. Both showed a decrease in pressure on inspiration. No diastolic dip was present in the venous pressure. Fluid removal was followed by a drop in venous pressure to 14 mm Hg and intrapericardial pressure to 7 mm Hg. Pulsus paradoxus was no longer present (fig. 2). Subsequently, bleeding occurred into the right pleural space and abdominal cavity. The patient expired on the ninth hospital day with acute tamponade.

Case G. L. (Table 3). A 45-year-old Caucasian was undergoing cardiac catheterization for investigation of severe calcific aortic stenosis. The aortic pressure was 118/62 mm Hg, and the left ventricular pressure was 195/5 mm Hg. Angiographic studies of the left ventricle resulted in penetration of the ventricular wall and a portion of the injectate entered the pericardial space. A few minutes after the episode, the aortic pressure dropped to 103/90 mm Hg during expiration and 92/80 mm Hg during inspiration. At the same time the left ventricular pressure was 190/18 mm Hg in expiration and 138/16 mm Hg in inspira-

Group IIIA. Respiratory Disease

Case T. C. (Table 4). A 71-year-old Negro with a history of respiratory distress and chronic cough for 10 years entered the hospital because of recent respiratory infection which had failed to clear. Chest x-rays showed increased cardiac silhouette over previous studies. There was a paradoxical pulse, but venous pressure was not observed above 5 to 7 mm Hg. A presumptive diagnosis of pericardial effusion was made and, after preliminary studies, the pericardial space was tapped with removal of 300 ml of fluid. The venous pressure and pericardial pressure were slightly reduced by the fluid removal, but the paradoxical pulse remained (fig. 4) and cardiac index did not change. Three weeks later the heart size returned to normal; however, the paradoxical pulse continued. Spirometry revealed severe respiratory obstructive airway disease.

Group IIIB. Myocardial Disease with Incidental Pericardial Effusion

Case L. M. (Table 4). A 38-year-old Caucasian entered the hospital giving a 2 weeks' history of progressive dyspnea on exertion and edema following upper respiratory infection. He showed engorged neck veins with a diastolic dip. There was an inspiratory decrease in venous pressure. Prominent third heart sound and tachycardia were noted. A chest film showed extreme generalized cardiac enlargement. There was no pulsus paradoxus. Pericardiocentesis of 300 ml of bloody
Cardiac tamponade: Case M. B., group IIB, aortic wall dissection. Top to bottom: Electrocardiogram, respiration (by mercury-silastic strain gauge), femoral arterial, venous and pericardial pressure. Left panel: Pulsus paradoxus in femoral artery pressure before tap. Time lines are 1 second. Voluntary prolongation of inspiration and slow expiration is accompanied by prompt rise in pulse pressure in late inspiration. Right panel: Relief of pulsus paradoxus by removal of 45 ml of blood. Intrapericardial pressure is 7 mm Hg with slight but regular decrease with inspiration.

Group IIIC. Obesity

Case R. W. (Table 4). A 52-year-old Caucasian (height 72% inches, weight 287 pounds) exhibited cervical venous distention and pulsus paradoxus with quiet respiration. Figure 6 shows the paradoxical pulse and the evidence of thoracic inlet obstruction. During early inspiration, right atrial pressure fell rapidly, and the pressure in the inferior vena cava rose gradually. Subclavian vein pressure tended to fall slightly with inspiration; however, there was an inspiratory gradient between the subclavian vein and the right atrium. Catheter exploration indicated that the inspiratory pressure gradient developed over a few centimeters of length of the vessel. This case demonstrates pulsus paradoxus and elevated venous pressure due to extrathoracic obstruction. Kussmaul’s sign was present in the inferior vena cava but not in the superior vena cava tributaries.

Results

The coincidence of the selected physical signs with the disease states are represented by the modified Venn diagrams in figures 7 to 9.

Cardiac Output, Third Heart Sound, and Venous Pulse

Figure 7 indicates that a reduction in resting cardiac output did not allow good diagnostic discrimination since a cardiac index of less than 2.5 was present in some members of groups I, II, and III. The occurrence of an early diastolic pressure dip (Friedreich’s sign) and a third heart sound was confined to groups I and IIIB.

Venous Pressure and Kussmaul’s Sign

Venous (or right atrial) pressure elevation of more than 12 mm Hg was common in group I, was seen in all cases of subgroup
Figure 3
Tamponade: Case G. L., group IIB, severe calcific aortic stenosis. Following penetration of left ventricular wall by injection of contrast material, pulsus paradoxus of the left ventricular pressure was not reflected by equivalent changes in aortic systolic or pulse pressure.

IIB, and was also present in subgroups IIIB and IIIC. Kussmaul’s venous sign, present clinically or during SVC or RA catheterization and defined as a recognizable and regular inspiratory increase in venous pressure with tranquil respiration, was observed in only six of the 37 cases, in approximately one third of the cases of constrictive pericarditis. Kussmaul’s sign as defined herein was not apparent in group II. Similarly, in respiratory disease and primary cardiac disease with associated effusion, an inspiratory rise in venous pressure was not seen (fig. 8).

Inspiratory Drop in Venous Pressure and Pulsus Paradoxus
When the converse sign, inspiratory drop in venous pressure, was examined by clinical observation and direct central measurement of the venous pressure in the subclavian vein and right atrium, a positive sign was present in 21 cases of group IIA and B and group IIIA, B and C (fig. 9). Intrapericardial pressures were obtained in 16 of these cases and an inspiratory decrease in pressure was observed. All of group I were excluded.

Pulsus paradoxus (>20 mm Hg decrease in systolic pressure with quiet respiration) was found only in group IIA and B and IIIA and C. It was present in all but one of group IIB, in two of seven of group IIA, and in all of group IIIA and C. The finding was not observed in any of group I or group IIIB. The sole case of group IIB in which pulsus paradoxus did not appear despite significant tamponade was a case of severe calcific aortic
stenosis in which cardiac tamponade occurred incident to perforation of the ventricular wall during angiographic studies. Figure 3 indicates that respiration induced a 50-mm variation in left ventricular systolic pressure; however, the severe aortic valvular obstruction prevented reflection of the pressure variation in a simultaneously recorded aortic pulse.

![Graph](image-url)

**Figure 4**

Obstructive airway disease with incidental pericardial effusion, Case S. K., group IIIA. Upper panel: Top to bottom: Brachial artery, right atrial pressure, electrocardiogram. Left panel: Before removal of 250 ml of fluid. Right panel: After fluid removal. Arterial and atrial pressures fall with inspiration. Time intervals are indicated between arrows.

Lower panel: Brachial artery, pulmonary artery, intrapericardial pressures and electrocardiogram before (left) and after (right) fluid removal. Systemic and pulmonary arterial intrapericardial pressures fall with inspiration. Time interval is indicated between arrows.

**Discussion**

**Venous and Auscultatory Phenomena**

Several authors have reported that the venous diastolic dip (Friedreich's sign) along with an early diastolic sound is very common in constrictive pericarditis, but they also have reported that these phenomena may exist in heart failure. The present report is
Figure 5
Acute myocarditis with pericardial effusion. Case L. M., group IIIB. Left panel: Before removal of fluid; top to bottom, respiration, femoral artery, pulmonary and right atrial pressure with common pressure reference and sensitivity. Pulsus paradoxus is not present. Right panel: After removal of 300 ml of fluid. Intrapericardial pressure calibrations at right. Inspiration is accompanied by a greater reduction in intrapericardial than atrial pressures with a consequent rise in transmural pressure.

Figure 6
Spurious signs of cardiac compression: Case R. W., obesity, group IIIC. Top to bottom: Respiration (impedance method, inspiration downward), brachial artery pressure (calibration at left), inferior vena cava and right atrial pressures, electrocardiogram. Time lines, 1 second. Pulsus paradoxus and inspiratory IVC and RA pressure gradient are evident. A voluntary prolongation of expiratory pause, left and center, is accompanied by a rise and fall in arterial pressure.6, 27
in agreement, since 15 of 16 cases of group I and all cases of group IIIB show these signs. Although the third heart sound tends to occur closer to the second sound and often is rather brisk in pericardial disease as compared with heart failure, the quality of the sound did not allow discrimination in the majority of our cases. In groups IIA and B and IIIA and C in which pericardial effusion, tamponade, respiratory disease, or obesity were the primary disorders, a third heart sound was not reported. Similarly, a diastolic dip in cervical venous pressure was not observed nor was it seen on direct measurement. Although the numbers are not large, the conclusion seems allowable that Friedreich's sign or prominent third heart sound would exclude the presence of lax cardiac tamponade or pericardial effusion in the absence of other cardiac disease. Our findings are in agreement with Fowler who stated that a diastolic dip in the cervical veins is not found in pericardial effusion. The report of Connolly and associates contains the suggestion that diastolic sounds are not present in pericardial effusion. McKusick and Goldner and Kroop reported diastolic sounds in chronic pericarditis due to tuberculosis. The reports do not discount the possibility that the sound may have been a friction rub, or a combination of visceral pericardial constriction and effusion may have existed.

**Transmission of Cardiac, Pericardial, and Chest Wall Retractive Forces**

The venous pressure phenomena during diastole are conditioned by various factors as Brecher has discussed. The early diastolic forces are comprised of restorative myocardial action and the negative intrapleural pressure. These forces, closely coupled mechanically by normal pericardial structures, are not commonly reflected in the cervical veins because of the low venous pressure. Elevation of venous and right heart diastolic pressures in congestive heart failure allow emergence

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**Figure 7**

A modified Venn diagram. **Top:** The set of cases with cardiac index less than 2.5 contains portions of groups I, II, and III. **Lower:** Friedreich's sign and third heart sound were not present in groups II or IIIA.

**Figure 8**

**Top:** The set of cases with venous pressure > 12 mm Hg includes all cases of IIIB, IIIB, and IIIC. **Lower:** The set of cases with Kussmaul's venous sign includes only group I.
to clinical recognition as the early diastolic and inspiratory drop in venous pressure. The similarity of venous pressure signs and third heart sound in constrictive pericarditis and heart failure is related to the myocardial and mediastinal coupling by fibrous structures and consequently is an exaggeration of restorative diastolic forces. In consequence, clinical delineation between congestive heart failure and constrictive pericarditis is difficult from the venous and auscultatory phenomena.

When significant fluid is present in the pericardial space and pericardial structure and compliance are not altered by fibrosis or infiltrative disease, the retractive forces due to a negative intrapericardial pressure are still transmitted. The decrease in pericardial pressure on inspiration reported previously in humans and animal studies is normal in direction if not in magnitude. The decreased intrapericardial pressure and augmented right ventricular output combine to reduce right atrial and venous pressure during inspiration. Although the pericardial fluid does not prevent the transmission of low frequency pressure variation of respiratory origin, damping of the restorative myocardial forces of early diastole by the inertia of extracardiac fluid mass may prevent the formation of discernible venous pressure phenomena.

**Kussmaul’s Sign**

In certain patients with elevated venous pressure, late inspiration may be associated with a further rise in venous pressure. This is presumably related to the late inspiratory increase in abdominal pressure. The elevation of extramural (abdominal) pressure in venous circulation may be reflected in a rise in cerebral venous pressure or Kussmaul’s sign. Kussmaul’s venous sign may be considered evidence of failure of transmission of intrapleural pressure to the right heart chambers and lack of increased right heart output during inspiration. Both factors may operate in constrictive pericarditis. The latter factor would be operative in severe heart failure. Therefore, we conclude, as did Hitizig and Wood, that Kussmaul’s sign does not discriminate between constrictive pericarditis and congestive heart failure. It may, however, be evidence against cardiac compression by fluid.

**Pulsus Paradoxus**

Although several patients of group I showed respiratory pulse pressure variation greater than that noted in normal subjects, pulsus paradoxus as defined was absent (table 1). Perhaps our rather strict adherence to the criteria of Gauchat and Katz is in part responsible for this. Many authors who have described pulsus paradoxus have implied that deep inspiration was employed in the test or that a 10-mm Hg decrease in arterial systolic pressure was a criterion. Extensive recordings of arterial pressure in several cases of constrictive pericarditis by Beck and associates show respiratory variation in pulse pressure not exceeding 12 mm Hg. The high incidence of atrial fibrillation, nine of 16, with possible differences in diastolic filling due to variable rate would make recognition of pulsus paradoxus difficult in the majority of our cases. Wood, however, in reporting 30 cases of constrictive pericarditis stated that pulsus paradoxus occurs, but he did not comment on the incidence, nor did he define the criteria employed.
Kussmaul originally described his venous sign and pulsus paradoxus in constrictive pericarditis. It is possible that his patient had coexistent airway disease or that respiration was not tranquil. We would conclude that pulsus paradoxus greater than 20 mm Hg is not characteristic of constrictive pericarditis.

Pulsus paradoxus was inconstant in lax pericardial effusion (two of seven cases), but could be easily induced by voluntary deep inspiration as Dornhorst and associates reported. Pulsus paradoxus was seen in six of seven cases of tamponade and the severity was reduced or eliminated entirely with removal of fluid. The occurrence in the two cases of obstructive airway disease with no obvious respiratory distress or voluntary exaggeration of respiration represents the "dynamic" form of pulsus paradoxus. We have previously reported the intrapericardial pressure variation in emphysema without effusion and have suggested a mechanism for pulsus paradoxus.

The biphasic character of pulsus paradoxus, consisting of an early inspiratory drop in arterial pressure and pulse pressure followed promptly by a subsequent rise in arterial and pulse pressure as inspiratory efforts are maintained, was present when sustained inspiratory pauses could be carried out (figs. 2 and 6). In a previous communication we considered this a result of a transient drop in left ventricular stroke volume due to an increased capacity of the pulmonary vascular bed, but the subsequently brisk rise in arterial systolic and pulse pressures occurs as the inspiratory augmentation of right ventricular output is ultimately delivered to the left ventricle. The "respiratory pump" continues to function, although possibly imperfectly. Figure 6 shows that voluntary prolongation of respiratory cycle unmasks the biphasic character of pulsus paradoxus, independent of expiration.

Of interest is the absence of pulsus paradoxus in group IIIB, in which pericardial effusion was an incidental process superimposed on acute or chronic heart disease with right and left ventricular dilatation demonstrated at surgery or by angiography. The absence of pulsus paradoxus in these three cases with elevated venous pressure along with Friedreich's sign and prominent third heart sounds provided important evidence that effusion was not the cause of circulatory dysfunction.

Pulsus paradoxus was noted in the two cases of extreme obesity of group IIIC. We have reported this finding as a spurious sign of pericardial disease and feel that it may reflect venous collapse at the thoracic inlet with the resultant late inspiratory reduction of right ventricular output. In a recent report, pulsus paradoxus in an obese patient led to the erroneous diagnosis of cardiac tamponade.

Cardiac Index and Venous Pressure

Our findings of a variable decrease in cardiac index in constrictive pericarditis agree with the report of Wood. Of interest is the normal cardiac index in group IIIA with lax effusion when the low cardiac index in myxedematous patients is corrected by increasing Vo2 to normal. In contrast, the cardiac index was reduced in IIB but increased to normal values after pericardial tap in three of the four cases in which it was measured.

Venous pressure was elevated more than 12 mm Hg in the majority of cases in group I, all of group IIB, and in groups IIIB and C. After pericardiocentesis, venous pressure decreased in group IIB but not in group IIIB. The increased venous pressure in group IIIC seems related to the "obligatory venous hypertension" associated with massive obesity. At the thoracic inlet, an abrupt drop in extramural vascular pressure occurs. Rapid depletion of a short extrathoracic venous segment during early inspiration causes local collapse, and the extrathoracic venous pressure does not reflect right atrial pressure. Inferior caval pressure may increase with inspiration (fig. 6). We do not find Kussmaul's sign in the cervical veins with tranquil respiration, but conceivably an inspiratory rise in cervical venous pressure could occur with vigorous respiration.

Burdine and Wallace reported findings of pulsus paradoxus and Kussmaul's sign in a
patient with circulatory and respiratory distress. Pericardiocentesis did not yield fluid and postmortem examination showed a pulmonary embolus. The patient reported on weighed 235 pounds and was 63 inches tall. Although by the criteria reported herein the respiratory variation of 10-mm systolic pressure which they recorded would not be considered pulsus paradoxus, Kussmaul's sign may have been secondary to venous collapse and the venous pressure would not necessarily reflect variations in right atrial pressure with inspiration.

Summary

Thirty patients with primary cardiac compression due to constrictive pericarditis, lax effusion, or cardiac tamponade and an additional seven patients with spurious evidence of cardiac compression or with pericardial effusion playing an unimportant role in the circulatory disorder were studied. Rather stringently defined physical findings were sought which might allow discrimination between cardiac disorders. The following conclusions are drawn from the results.

1. Constrictive pericarditis is associated with venous and auscultatory phenomena which do not allow separation from other forms of heart disease causing congestive heart failure. Kussmaul's sign is present in less than 40%; pulsus paradoxus as classically defined is rare.

2. In lax pericardial effusion, Kussmaul's sign and Friedreich's sign, along with third heart sounds, are not present. Pulsus paradoxus is inconstant with tranquil breathing but is regularly induced by deep inspiration. There is inspiratory decrease in venous pressure and pericardial pressure. Cardiac index is normal and venous pressure is less than 12 mm Hg. Circulatory distress is not apparent.

3. Tamponade induces signs of circulatory distress and is regularly characterized by pulsus paradoxus but Friedreich's sign, a third heart sound, as well as Kussmaul's venous sign, are absent. The venous pressure exceeds 12 mm Hg. There is an inspiratory decrease in venous pressure and pericardial pressure.

The low cardiac index is usually relieved by tap. When aortic stenosis is present, respiratory variation in left ventricular systolic pressure may not be reflected by clinical pulsus paradoxus.

4. Spurious signs of cardiac compression may be due to (1) respiratory disease, (2) severe myocardial disease and incidental effusion, or (3) obesity. In the respiratory disease pulsus paradoxus, normal cardiac index, low venous pressure, and venous and pericardial-pressure decrease with inspiration are present. The second group does not show pulsus paradoxus and the elevated venous pressure, diastolic dip, and third heart sounds are due to heart failure. Obesity may cause pulsus paradoxus and increased peripheral venous pressure, which does not reflect central venous pressure. These findings seem related to inspiratory collapse of extrathoracic vessels.

References

10. McKusick, V. A.: Chronic constrictive pericar-
COMPRESSION CARDIAC DISORDERS


Importance of Studies at the Bedside—"the Dublin School"

The human mind is so constituted, that in practical knowledge its improvement must be gradual. Some become masters of mathematics, and of other abstract sciences, with such facility, that in one year they outstrip those who have laboured during many. It is so, likewise, in the theoretical parts of medicine; but the very notion of practical knowledge implies observation of nature; nature requires time for her operations: and he who wishes to observe their development will in vain endeavour to substitute genius or industry for time. Remember, therefore, that however else you may be occupied—whatever studies may claim the remainder of your time, a certain portion of each day should be devoted to attendance at an hospital. . . . —ROBERT J. GRAVES: Clinical Lectures on the Practice of Medicine, ed. 2, vol. 1. Dublin, Fannin and Co., 1848, p. 2.
Diagnostic Signs in Compressive Cardiac Disorders: Constrictive Pericarditis, Pericardial Effusion, and Tamponade
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