The Electrocardiographic Pattern of Right Ventricular Hypertrophy in Chronic Cor Pulmonale

By Ralph C. Scott, M.D., Samuel Kaplan, M.D., N. O. Fowler, M.D., Robert A. Helm, M.D., Richard N. Westcott, M.D., Isom C. Walker, M.D., and William J. Stiles, M.D.

The electrocardiographic patterns encountered in 28 patients with pulmonary hypertension secondary to chronic lung disease have been studied. Thirteen had neither right ventricular hypertrophy nor right bundle-branch block; eight had either right ventricular hypertrophy alone or right ventricular hypertrophy and right bundle-branch block (complete or incomplete); and seven had incomplete right bundle-branch block. A statistical comparison is made between these various groups of the means of the following physiologic measurements: mean pulmonary artery pressure, total pulmonary resistance, arterial oxygen saturation, right ventricular work, arterial carbon dioxide content, and cardiac index. The significance of the pattern of right ventricular hypertrophy is discussed.

JOHNSON AND ASSOCIATES found in their study of patients with cor pulmonale secondary to chronic pulmonary disease that if the electrocardiographic pattern of right ventricular hypertrophy was present the mean pulmonary artery pressure usually exceeded 30 mm. Hg. Dexter and co-workers have pointed out in their study of cases of cor pulmonale without hypoxia that the pattern of right ventricular hypertrophy did not appear until the total pulmonary resistance exceeded 750 dynes seconds cm.  

We have had the opportunity to study by cardiac catheterization 28 patients with pulmonary hypertension secondary to chronic lung disease. It was thought that a comparison of our results with those of Johnson and co-workers and of Dexter and associates would be of interest.

Material and Methods

The patients studied were from the medical service of the Cincinnati General Hospital and from the Veterans Administration Hospital, Dayton, Ohio. Some of these patients were included in a previous report from this laboratory. All had the diagnosis of chronic lung disease established from history and/or chest x-ray study (tables 1 and 2).

Cardiac venous catheterization was carried out according to the method of Courmand and Ranges. Cardiac outputs were determined by the direct Fick principle. Pulmonary “capillary” pressures were measured according to the method of Hellem, Haynes and Dexter. Pressures were recorded by means of a five channel optical oscillograph (Hathaway). The zero point for pressures was 10 cm. anterior to the back of the patient when supine. The mean pulmonary artery and pulmonary “capillary” pressures were measured by planimetric integration.

Pulmonary arteriolar resistance was calculated according to the formula:

\[ R = \frac{PA_m - "PC"}{CO} \times 1332 \text{ dynes seconds cm}^{-2} \]

Total pulmonary resistance was calculated as follows:

\[ R' = \frac{PA_m - O}{CO} \times 1332 \text{ dynes seconds cm}^{-2} \]

Where \( PA_m \) = pulmonary arterial mean pressure in millimeters Hg; "PC" = pulmonary "capillary" mean pressure in millimeters Hg; \( CO \) = cardiac output, cubic centimeters per second; 1332 = conversion factor from millimeters Hg to dynes per square centimeter.

Right ventricular work was calculated as follows:

\[ W_r = \frac{(CI, \times 1.055)([PA_m - RA_m] \times 13.6)}{1000} \text{ Kg M./min./sq.M.} \]
### Table 1.—Physiologic Measurements in Thirteen Patients with Chronic Cor Pulmonale without Right Ventricular Hypertrophy or Right Bundle-Branch Block (Complete or Incomplete)

<table>
<thead>
<tr>
<th>Patient</th>
<th>X-ray</th>
<th>Pressures (mm. Hg)</th>
<th>Resistances (Dynes sec. cm.&quot;⁻¹)</th>
<th>Art. O₂ Sat. (% of Capacity)</th>
<th>Art. CO₂ Content (Vol. %)</th>
<th>RV Work (Kg. M./min. M.³)</th>
<th>Cardiac Index (L./min. M.²)</th>
<th>Electrocardiogram</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.Be. 63, W.M.</td>
<td>Pneumonia at apex LLL with abscess cavity</td>
<td>RA 12 to +5</td>
<td>3/D 8-15</td>
<td>13</td>
<td>433</td>
<td>94.4</td>
<td>47.0</td>
<td>2.7</td>
<td>S</td>
</tr>
<tr>
<td>G.AI. 60, W.M.</td>
<td>Congenital diaphragmatic hernia. Cardiac silhouette not well seen</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
<tr>
<td>M.L. 63, W.F.</td>
<td>Marked emphysema</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
<tr>
<td>H.J. 63, W.M.</td>
<td>Emphysema, severe</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
<tr>
<td>E.J. 61, W.M.</td>
<td>Chronic. bronch. infection and fibrosis. Possibly with bronchiectasis. Emphysematous blebs</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
<tr>
<td>A.B. 45, C.M.</td>
<td>—</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
<tr>
<td>J.Sm. 62, W.M.</td>
<td>Marked emphysema</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
<tr>
<td>L.N. 62, W.M.</td>
<td>Chronic lung disease with emphysema. Cor pulmon.</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
<tr>
<td>J.W. 63, C.M.</td>
<td>Chronic lung disease with emphysema. Prominent pulmonary artery</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
<tr>
<td>M.S. 53, W.F.</td>
<td>Chronic lung disease with fibrosis and emphysema. Card. enlarge.</td>
<td>RA 12</td>
<td>3/D 8-10</td>
<td>34</td>
<td>471</td>
<td>96.5</td>
<td>0.19</td>
<td>0.9</td>
<td>S</td>
</tr>
</tbody>
</table>

For normal values, see References 1-8. 16.

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Table I.—Continued

<table>
<thead>
<tr>
<th>Patient</th>
<th>X-ray</th>
<th>Pressures (mm Hg)</th>
<th>Resistances (Dynes sec. cm.**)</th>
<th>Art. O2 Sat. (% of Ca- pacity)</th>
<th>Art. CO Content (Vols. %)</th>
<th>RV Work (Khr. M./min. M.**)</th>
<th>Electrocardiogram</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>H. Br. 62, W. M.</td>
<td>Mild general, card. enlarge. (Bronch. asthma)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J. Bat. 55, C. M.</td>
<td>Emphysema</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H. St. 51, C. M.</td>
<td>Emphysema</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

For normal valves, see References 5, 6, 11, 12.

RA: right atrium; FC: "pulmonary capillary"; PA: pulmonary artery; PAR: pulmonary arterioles resistance; TPR: total pulmonary resistance; RV: right ventricular or pulmonary; RVH: right ventricular hypertrophy; IRBBB: incomplete right bundle-branch block; CRBBB: complete right bundle-branch block; VC: vital capacity, per cent of predicted; MBC: maximum breathing capacity, per cent of predicted.

* Right ventricular pressure.
† Right ventricular mean systolic ejection pressure.
‡ Total pulmonary resistance calculated using right ventricular mean systolic ejection pressure instead of mean pulmonary artery pressure.

Where \( W_r \) = work of right ventricle against pressure; \( CI \) = cardiac index, liters per minute per square meter; 1.055 = specific gravity of blood; \( P.A. \) = pulmonary arterial mean pressure, millimeters Hg; \( R.A. \) = right atrial mean pressure, millimeters Hg; 13.6 = specific gravity of mercury.

In three cases (R. P., E. J., R. W.) where no mean pulmonary artery pressures were available, the right ventricular mean systolic ejection pressure by equations was substituted in the above equations.

All patients had electrocardiograms consisting of the three standard leads, aVR, aVL, aVF, V1 through V6 (and V4R and V4R in some), usually taken either on the same day or within a few days of the time of catheterization.

The criteria used for right ventricular hypertrophy1, 6-9 were those of a QR pattern in V4R or V1, an R wave in V1 of 7 mm. or greater, an R to S ratio in V1 greater than 1, or a delay in the onset of the intrinsicoid deflection in V1 of from 0.035 to 0.05 second. Incomplete right bundle-branch block10 was diagnosed on the basis of a prolongation of the QRS interval in the standard leads to 0.12 second or greater with an rSR' pattern in V1 or V4 and a delay in the onset of the intrinsicoid deflection of R' to 0.08 second or greater. If the height of R' exceeded 15 mm. it was taken as evidence of right ventricular hypertrophy in addition to complete right bundle-branch block. The P waves in the standard leads were considered to be abnormal if they were 0.12 second or greater in duration or 3 mm. or more in height, or were notched.14 Heart position was classified according to the criteria of Wilson.8

Results

Part I

Our cases were divided into four groups: Group I, those without right ventricular hypertrophy or right bundle-branch block, complete or incomplete (13 cases); group II, those with right ventricular hypertrophy and/or right bundle-branch block, complete or incomplete (15 cases); group III, those with right ventricular hypertrophy alone or with both right ventricular hypertrophy and
TABLE 2.—Physiologic Measurements in Fifteen Patients with Chronic Cor Pulmonale with Right Ventricular Hypertrophy and/or Right Bundle-Branch Block (Complete or Incomplete)

<table>
<thead>
<tr>
<th>Patient</th>
<th>X-ray</th>
<th>Pressures (mm. Hg)</th>
<th>Resistances (Dynes sec. cm. -2)</th>
<th>Art. O2 Sat. (% of Capacity)</th>
<th>Art. CO2 Content (Vol. %)</th>
<th>RV Work (Kg. M./min./M.2)</th>
<th>Cardiac Index (L./min./M.2)</th>
<th>Electrocardiogram</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>C. B.</td>
<td>Emphysema.</td>
<td>17</td>
<td>90/45</td>
<td>59</td>
<td>1469</td>
<td>51.3</td>
<td>65.7</td>
<td>1.26</td>
<td>2.1</td>
</tr>
<tr>
<td>M. T.</td>
<td>Emphysema and fibrosis.</td>
<td>10</td>
<td>45/27</td>
<td>37</td>
<td>744</td>
<td>83.8</td>
<td>46.8</td>
<td>1.03</td>
<td>2.6</td>
</tr>
<tr>
<td>H. F.</td>
<td>Fullness of pulmon. are</td>
<td>7</td>
<td>11</td>
<td>29</td>
<td>345</td>
<td>94.0</td>
<td>0.68</td>
<td>2.2</td>
<td>S + + + 0 0 Polycythemia He-   Hemat. 66</td>
</tr>
<tr>
<td>D. H.</td>
<td>Pulmonary fibrosis.</td>
<td>12</td>
<td>85/40</td>
<td>67</td>
<td>1050</td>
<td>72.3</td>
<td>60.1</td>
<td>1.71</td>
<td>2.6</td>
</tr>
<tr>
<td>A. W.</td>
<td>Mod. general. card. enlarge;</td>
<td>-</td>
<td>18</td>
<td>94/52</td>
<td>71</td>
<td>598</td>
<td>63.8</td>
<td>63.8</td>
<td>3.5</td>
</tr>
<tr>
<td>R. W.</td>
<td>Emphysema.</td>
<td>-</td>
<td>5/5*</td>
<td>35</td>
<td>4892</td>
<td>88.4</td>
<td>50.7</td>
<td>1.65</td>
<td>3.8</td>
</tr>
<tr>
<td>C. C.</td>
<td>Chron. lung disease with emphysema. Cor Pulmon.</td>
<td>-</td>
<td>-</td>
<td>145/60</td>
<td>89</td>
<td>-</td>
<td>48.6</td>
<td>90.3</td>
<td>- + 0 + 0 Autopsy: Emphysema. Cor Pulmon. RV: 15 mm. LV: 18 mm.</td>
</tr>
<tr>
<td>J. Sh.</td>
<td>Emphysema.</td>
<td>4</td>
<td>56/34</td>
<td>40</td>
<td>651</td>
<td>81.3</td>
<td>52.1</td>
<td>1.57</td>
<td>3.0</td>
</tr>
<tr>
<td>G. An.</td>
<td>Emphysema.</td>
<td>20</td>
<td>19</td>
<td>119/54</td>
<td>1358</td>
<td>50.2</td>
<td>40.3</td>
<td>1.41</td>
<td>1.8</td>
</tr>
<tr>
<td>G. M.</td>
<td>-</td>
<td>10</td>
<td>92/43</td>
<td>63</td>
<td>1372</td>
<td>84.7</td>
<td>44.7</td>
<td>1.67</td>
<td>2.1</td>
</tr>
</tbody>
</table>

For normal values, see References 6, 7, 26, 27, 28.
right bundle-branch block, complete or incomplete (8 cases); and group IV, those with only incomplete right bundle-branch block and no right ventricular hypertrophy (7 cases).

A statistical comparison* was made between the means of the various groups for the following items: mean pulmonary artery pressure, total pulmonary resistance, arterial oxygen saturation, right ventricular work, arterial carbon dioxide content, and cardiac output. A summary of these results is presented in table 3.

A significant difference was demonstrated between the means of group I and group II for mean pulmonary artery pressure \((p < 0.001)\), total pulmonary resistance \((p > 0.001 < 0.01)\), arterial oxygen saturation \((p > 0.01 < 0.02)\), and level of right ventricular work \((p > 0.02 < 0.05)\). No significant difference was demonstrated between the means of arterial carbon dioxide content \((p > 0.9)\) or of cardiac index \((p > 0.8 < 0.9)\) table 3).

Groups I and III were compared and a statistically significant difference was found between the means of the two groups for mean pulmonary artery pressure \((p < 0.001)\), total pulmonary resistance \((p < 0.001)\), arterial oxygen saturation \((p > 0.001 < 0.01)\), and right ventricular work \((p > 0.02 < 0.05)\). No significant difference was found for the mean arterial carbon dioxide content \((p > 0.6 < 0.7)\), or the mean cardiac index \((p > 0.1 < 0.2)\) (table 3).

A significant difference was demonstrated between the means of group I and group IV for mean pulmonary artery pressure \((p > 0.001 < 0.01)\). No statistically significant dif-

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* Standard statistical methods were used throughout. 

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**TABLE 2.—Continued**

<table>
<thead>
<tr>
<th>Patients</th>
<th>X-ray</th>
<th>Pressures (mm Hg)</th>
<th>Resistances (Dynes sec cm(^{-2}))</th>
<th>Art. O(_2) Sat. (% of Capacity)</th>
<th>Art. CO(_2) Content (Vol. %)</th>
<th>RV Work Kg. min/ M(^3)</th>
<th>Cardiac In- dex (L/ min/ M(^3))</th>
<th>Electrocardiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>RA 5-13</td>
<td>PC 3-13</td>
<td>PA 11-29</td>
<td>Mea 4-13</td>
<td>P.A. 47-100</td>
<td>T. 185.9</td>
<td>S/D 94-50</td>
</tr>
<tr>
<td>R. P. 46</td>
<td>C.M.</td>
<td>Chron. lung disease, Cor pulmonale, Milary infiltrate.</td>
<td>12 --</td>
<td>45-62/ 4-10*</td>
<td>45</td>
<td>--</td>
<td>11711</td>
<td>75.9</td>
</tr>
<tr>
<td>D. M. 46</td>
<td>W.M.</td>
<td>Prom. pulmon. art. Card. enlarge. Cor pulmonale.</td>
<td>10</td>
<td>8</td>
<td>70/26</td>
<td>42</td>
<td>466</td>
<td>576</td>
</tr>
<tr>
<td>J. D. 67</td>
<td>W.M.</td>
<td>Chron. lung disease, Mod. card. enlarge.</td>
<td>--</td>
<td>34/23</td>
<td>23</td>
<td>--</td>
<td>276</td>
<td>85.9</td>
</tr>
<tr>
<td>J. L. 55</td>
<td>W.M.</td>
<td>Cystic bronchiectasis, LUL, and LLL.</td>
<td>--</td>
<td>82/32</td>
<td>35</td>
<td>--</td>
<td>269</td>
<td>72.7</td>
</tr>
<tr>
<td>H. Sa. 59</td>
<td>C.M.</td>
<td>Pulmon. congest. Prominence of pulmon. art. segment. Fibrotic infiltrate in LUL</td>
<td>12</td>
<td>82/30</td>
<td>50</td>
<td>--</td>
<td>1573</td>
<td>65.7</td>
</tr>
</tbody>
</table>

Abbreviations as table 1.

*1 See table 1.
TABLE 3.—Statistical Comparison between the Means of Various Physiologic Measurements in Twenty-eight Patients with Chronic Cor Pulmonale

<table>
<thead>
<tr>
<th>Physiologic Measurements</th>
<th>Group I: No RVH or RBBB (13 Cases)</th>
<th>Group II: RVH and/or RBBB (13 Cases)</th>
<th>Group III: RVH or RVH and RBBB (8 Cases)</th>
<th>Group IV: IRBBB (7 Cases)</th>
<th>Statist. Compar. of Diff. between Group Means</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean PA pressure (mm. Hg)</td>
<td>26.7 ± 6.3</td>
<td>49.9 ± 18.5</td>
<td>60.1 ± 19.0</td>
<td>38.1 ± 8.6</td>
<td>4.30†</td>
</tr>
<tr>
<td>Total pulmon. resistance (dynes sec. cm.−1)</td>
<td>448 ± 130</td>
<td>914 ± 481</td>
<td>1100 ± 463</td>
<td>728 ± 484</td>
<td>3.37†</td>
</tr>
<tr>
<td>Art. O2 saturation (% of capacity)</td>
<td>86.0 ± 8.5</td>
<td>74.2 ± 14.4</td>
<td>68.9 ± 16.8</td>
<td>80.2 ± 8.7</td>
<td>2.10*</td>
</tr>
<tr>
<td>Rt. vent. work (Kg.M./min./M.²)</td>
<td>0.95 ± 0.33</td>
<td>1.28 ± 0.35</td>
<td>1.37 ± 0.4</td>
<td>1.16 ± 0.33</td>
<td>2.32*</td>
</tr>
<tr>
<td>CO₂ content (vols. %)</td>
<td>56.7 ± 7.2</td>
<td>56.9 ± 13.0</td>
<td>59.6 ± 16.6</td>
<td>54.3 ± 8.7</td>
<td>.06</td>
</tr>
<tr>
<td>Card. index (L/min./M.²)</td>
<td>2.9 ± 0.7</td>
<td>2.8 ± 1.2</td>
<td>2.5 ± 0.6</td>
<td>3.2 ± 1.6</td>
<td>.23</td>
</tr>
</tbody>
</table>

RBBB: Right bundle branch block, complete or incomplete; IRBBB: Incomplete right bundle branch block; RVH: Right ventricular hypertrophy.

Significant probabilities are designated as follows: * 0.05-0.01; † 0.01-0.001; ‡ 0.001.

ference was demonstrated between the two groups for mean total pulmonary resistance (p > 0.05 < 0.1), arterial oxygen saturation (p > 0.1 < 0.2), right ventricular work (p > 0.2 < 0.3), arterial carbon dioxide content (p > 0.5 < 0.6), or cardiac index (p > 0.6 < 0.7) (table 3).

Finally, group III and group IV were compared. There was a significant difference found between the means of the two groups for mean pulmonary artery pressure (p > 0.01 < 0.02). No statistically significant difference was found for total pulmonary resistance (p > 0.1 < 0.2), arterial oxygen saturation (p > 0.1 < 0.2), right ventricular work (p > 0.3 < 0.4), arterial carbon dioxide content (p > 0.4 < 0.5) or cardiac index (p > 0.2 < 0.3) (table 3).

Part II

A correlation between right ventricular work and (1) the height of the R wave in millimeters in V₁, and (2) the R to S ratio in V₁ was carried out. A correlation of borderline significance was found between right ventricular work and the height of the R wave in V₁ (r = 0.423, p = 0.05). No significant correlation was found between right ventricular work and the R to S ratio in V₁ (r = 0.347, p > 0.1).

Part III

Our cases were next classified into three groups. (See fig. 1.)

Group A: Patients with the mean pulmonary artery pressure less than 30 mm. Hg and the total pulmonary resistance less than 750 dynes seconds cm.−5 (fig. 1). There were 12 patients in this group. Ten showed no electrocardiographic evidence of right ventricular hypertrophy. One (J. D.) showed the pattern of incomplete right bundle-branch block and one (H. F.) the pattern of right ventricular hypertrophy.

Group B: Patients with the mean pulmonary artery pressure greater than 30 mm. Hg and the total pulmonary resistance less than 750 dynes seconds cm.−5 (fig. 1). There were eight patients in this group. One showed the pattern of right ventricular hypertrophy, four showed incomplete right bundle-branch block, and three showed neither right ventricular hypertrophy nor bundle-branch block.

Group C: Patients with the mean pulmonary artery pressure greater than 30 mm. Hg and the
**PART IV**

The patients were next divided into two categories: (1) those with hypoxia (arterial oxygen saturation less than 85 per cent) and (2) those without significant hypoxia (arterial oxygen saturation above 85 per cent).\(^2\)\(^,\)\(^18\)

There were 15 patients in the first class and 13 patients in the second class (fig. 2). Eleven of the 15 patients in class 1 demonstrated right ventricular hypertrophy or right bundle-branch block (complete or incomplete). Only 4 of the 13 patients in class 2 showed right ventricular hypertrophy or incomplete right bundle-branch block.

**DISCUSSION**

Our findings confirm those of Johnson and associates.\(^1\) All of our cases, except one, with the pattern of right ventricular hypertrophy, had a mean pulmonary artery pressure greater than 30 mm. Hg. (The exception, H. F., had a pressure of 29 mm. Hg.) On the other hand, like Johnson and associates, we also had several patients (three in our series) with mean pulmonary artery pressures greater than 30 mm. Hg who did not have the pattern of right ventricular hypertrophy.

All of our cases whose total pulmonary resistance exceeded 750 dynes seconds cm.\(^{-5}\) had right ventricular hypertrophy, or right bundle-branch block (complete or incomplete) as did those of Dexter and co-workers.\(^2\) However, there were two cases of right ventricular hypertrophy and five of incomplete right bundle-branch block whose total pulmonary resistance was less than 750 dynes seconds cm.\(^{-5}\)

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**FIG. 1.** Relation of mean pulmonary artery pressure and total pulmonary resistance to the electrocardiographic pattern of right ventricular hypertrophy in patients with chronic cor pulmonale. Modified from Dexter and co-workers.\(^2\)

**FIG. 2.** Relation of hypoxia to the electrocardiographic pattern of right ventricular hypertrophy in patients with chronic cor pulmonale. Modified from Dexter and co-workers.\(^2\)
All of our seven cases whose total pulmonary resistance exceeded 750 dynes seconds cm.\(^{-5}\) had high mean pulmonary artery pressures (50 mm. Hg or greater), and all except one had low cardiac indices. These patients had advanced cor pulmonale.

Of interest is the high incidence of right ventricular hypertrophy in those patients with hypoxia. The reason appears to be that the patients with hypoxia usually have more advanced lung disease. It has been repeatedly pointed out that hypoxia tends to increase the pulmonary artery pressure.\(^{15-19, 24}\) There was a highly significant negative correlation in the present study between the mean pulmonary artery pressures and the arterial oxygen saturation \((r = -0.799, p < 0.001)\). This has also been pointed out by other workers in cases of chronic cor pulmonale.\(^{19, 20}\)

We did not find the close correlation between increasing levels of right ventricular work and the height of the R wave in V\(_1\) and the R to S ratio found by Cosby and co-workers in their cases of congenital heart disease and of mitral stenosis.\(^{7a, b}\) We did find, however, as they did in their cases of mitral stenosis, that the electrocardiogram was often normal at levels of right ventricular work above 1 kilogram meter per minute per square meter of body surface area.

Of interest was the relative rarity of auricular fibrillation in this series (one case out of 28) as contrasted with its frequency in a similar study\(^{23}\) of patients with mitral stenosis (11 cases out of 32). This feature of cor pulmonale has already been emphasized.\(^{22}\)

It has also been pointed out that hypercapnia may be of importance in the development of pulmonary hypertension.\(^{19, 20}\) Yu and co-workers\(^{19}\) and Harvey and associates\(^{20}\) have demonstrated a highly significant correlation between the arterial carbon dioxide tension and the mean pulmonary artery pressure. In the present study we were unable to demonstrate a significant correlation between the arterial carbon dioxide content and the mean pulmonary artery pressure \((r = 0.365, p > 0.03 < 0.1)\) and no significant difference between the mean arterial carbon dioxide content in those patients without right ventricular hypertrophy and those patients with right ventricular hypertrophy \((p > 0.6 < 0.7)\). We have no satisfactory explanation for this discrepancy between our findings and those of these other workers.\(^{19, 20}\) Stroud and Rahn\(^{23}\) also failed to demonstrate any significant increase in pulmonary artery pressure or resistance in anesthetized dogs subjected to 5 per cent carbon dioxide in the inspired gas mixture.

Our study confirms the statement of Dexter and his group\(^2\) that, when the electrocardiogram demonstrates the pattern of right ventricular hypertrophy in cases of chronic cor pulmonale, the disease is far advanced. This is borne out by the significantly different mean values for such measurements as mean pulmonary artery pressure, total pulmonary resistance, arterial oxygen saturation and right ventricular work in groups I and III. On the other hand the significance of the occurrence of the pattern of incomplete right bundle-branch block (without right ventricular hypertrophy) is more difficult to assess. Table 3 indicates that the mean values of the above measurements for group IV are intermediate between those of groups I and III. There are no statistically significant differences between the means of these measurements in groups I and IV and groups III and IV except in the case of mean pulmonary artery pressure. The electrophysiologic explanation of the rSR' pattern in V\(_1\) remains controversial, some workers believing that it is a phase in the development of right ventricular hypertrophy and others that it is due to a conduction defect in the right bundle.\(^{1, 7a, 7b, 8, 25, 26}\) It is likely that either explanation may be correct depending upon the underlying pathologic process.

When this pattern occurs in chronic cor pulmonale, our data favor the former explanation. However, a larger number of cases would be required to establish our results on a firm statistical basis. As in our patients with chronic cor pulmonale, Cosby and co-workers\(^{7a, 7b}\) have shown that in mitral stenosis and congenital heart disease, incomplete right bundle-branch block may occur at all levels of mean pulmonary artery pressure and pulmonary resistance. This is not surprising when it is re-
called that incomplete right bundle-branch block is a purely electrocardiographic diagnosis which may be demonstrated by an individual whose heart is normal according to all other criteria. However, the presence of incomplete right bundle-branch block, particularly in a young individual without coronary artery disease, should always lead to the consideration of the possibility of a condition associated with increased pulmonary artery pressure.

Summary and Conclusions

(1) Twenty-eight patients with pulmonary hypertension secondary to chronic lung disease have been studied by cardiac catheterization.

(2) Thirteen patients did not show the electrocardiographic pattern of either right ventricular hypertrophy, or right bundle-branch block; 15 patients showed right ventricular hypertrophy and/or right bundle-branch block (complete or incomplete).

(3) A statistically significant difference was demonstrated between the means of these two groups for the following physiologic measurements: mean pulmonary artery pressure, total pulmonary resistance, arterial oxygen saturation, and right ventricular work.

(4) All cases except one with the pattern of right ventricular hypertrophy had a mean pulmonary artery pressure greater than 30 mm Hg.

(5) All cases whose total pulmonary resistance exceeded 750 dynes seconds cm⁻⁵ showed the electrocardiographic pattern of right ventricular hypertrophy or right bundle branch block (complete or incomplete).

(6) The development of the pattern of right ventricular hypertrophy in patients with chronic cor pulmonale usually indicates an advanced state of the disease.

Summario e Conclusiones in Interlingua

(1) Esseva studiate per catheterisation cardiac 28 patientes con hypertension pulmonar secundari a chronic morbo del pulmones.

(2) In 13 del casos nulle configuration electrocardiographic characteristica de hypertrophia dexteroventricular o de bloco de branca dextere esseva observate. Le remanente 15 casos exhibiva hypertrophia dexteroventricular e/o bloco de branca dextere (o complete o incomplete).

(3) Esseva constatate in le duo gruppos differentias de signification statistic inter le valores median pro le sequente mesurations physiologic: pression pulmono-arterial, resistentia pulmonar total, saturation oxygenic arterial, e labor dexteroventricular.

(4) Omne casos—con le exception de un que exhibiva le configuration characteristica de hypertrophia dexteroventricular—habea un valor median del pression pulmone-arterial de plus que 30 mm Hg.

(5) Omne le casos con un resistentia pulmonar total de plus que 750 dynas/sec/cm² exhibiva un configuration electrocardiographic characteristica de hypertrophia dexteroventricular o bloco de branca dextere (o complete o partial).

(6) Le disveloppamento del configuration de hypertrophia dexteroventricular in patientes con chronic cor pulmonale indica generalmente un avantiate studio del morbo.

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