The Electrocardiogram in Chronic Lung Disease

F. J. C. MILLARD

From St. George's Hospital, London S.W.1

The objects of this study were to assess the value of the electrocardiogram in the diagnosis of right ventricular hypertrophy caused by chronic lung disease and to determine the effect of emphysema on the electrocardiogram.

Criteria for right ventricular hypertrophy have been suggested by many workers, for example, Myers, Klein, and Stofer (1948), Sokolow and Lyon (1949), Carouso et al. (1951), Scott et al. (1955), Braunwald et al. (1955), Milnor (1957), Goodwin and Abdin (1959), and Roman, Walsh, and Massie (1961). However, except for the study of Roman et al., the majority of patients included in these investigations suffered from either congenital heart disease or mitral stenosis, and comparatively few had right ventricular hypertrophy due to chronic lung disease. In some instances the presence of right ventricular hypertrophy was assumed without confirmation at necropsy, and in the remainder it was assessed by measuring the thickness of the wall of the right ventricle. There has been no previous investigation of patients with chronic lung disease in which electrocardiographic changes have been compared with ventricular weights.

Grant (1956), Spodick (1959), Wasserburger et al. (1959), Littmann (1960), and Selvester and Rubin (1965) have reported electrocardiographic changes due to emphysema. It was suggested that these were a direct effect of emphysema and were not due to cardiac abnormalities. However, there are very few cases recorded in which the diagnosis of emphysema is certain and in which cardiac abnormalities which could account for the electrocardiographic changes have been excluded.

Patients and Methods

Selection of Patients and Diagnosis. The electrocardiograms from 46 patients (35 men and 11 women) were studied. All had a history of chronic lung disease for 3 years or more, and all had had an electrocardiogram within 3 months of death. Emphysema was diagnosed from the chest radiograph, using the criteria of Simon (1964). The radiographs were read by Dr. Simon, and were divided into three groups: no emphysema, localized emphysema, and widespread emphysema.

Recording and Measurement of Electrocardiograms. A standard 12 lead electrocardiogram was recorded. The following measurements were made from each. (1) The frontal plane axis; (2) the height of the tallest P wave and the P wave axis; (3) the height of the tallest R wave in V1 and V2; (4) the width of the intrinscoid deflection over the right ventricle; (5) the depth of the deepest S wave in V5 and V6; and (6) the magnitude of the total deflection in the limb leads and in V6.

In the patients with hypertrophy of both ventricles the R wave in V5 and V6 and the S wave in V1 and V2 were also measured. Measurements were made with the naked eye to the nearest 0-5 mm. and the nearest 0-02 second. The frontal plane axis was calculated by the method of Carter, Richter, and Greene (1919) to the nearest 5°. Accurate measurement of the P wave axis was not attempted. If the P wave in aVL was positive, the axis was recorded as less than +60°; if it was negative and low or flat in lead I it was recorded as greater than +60°.

In addition, the criteria of Goodwin and Abdin (1959) for right ventricular hypertrophy were applied to each electrocardiogram. Goodwin and Abdin's criteria involve the use of V4R, which was not recorded. It was assumed, however, that if the R wave was dominant in aVR and V1, it must be dominant in V4R. Conversely, if the R wave was not dominant in either aVR or V1, it could not be dominant in V4R. It was also assumed that if the R wave was dominant in V1 but not in aVR, a dominant R was present in V4R. This assumption may not have been correct and could have led to the diagnosis of a higher grade of right ventricular hypertrophy than was actually present. However, in only one instance did an electrocardiogram show a dominant R in V1 and not in aVR, and this patient had a right bundle-branch block. Without V4R it was impossible to separate Goodwin and Abdin's Grades III and IV, and these were recorded as Grade III or IV.

Measurement of Ventricular Weight. The ventricles were dissected and weighed by the method of Fulton,
Hutchinson, and Jones (1952). The heart was removed at necropsy and inspected for any evidence of congenital or valvular disease. The coronary arteries were dissected and examined. The heart was fixed by immersion in 5 per cent formal saline. After fixation the atria and great vessels were separated from the ventricles. The tricuspid and mitral valves were cut away and the epicardial fat and superficial coronary vessels removed. The free wall of the right ventricle was separated from the septum with scissors. The blades of the scissors were held so that they were parallel to the septum. Trabeculae were cut as close to the septum as possible. The left ventricle and septum were not divided. The ventricles, that is, the free wall of the right ventricle and the left ventricle plus septum, were weighed to the nearest 0·5 g. The ventricles were sliced and the cut surface inspected for evidence of myocardial fibrosis.

Fulton's criteria of ventricular hypertrophy are given in the Appendix. The patients with isolated right ventricular hypertrophy were divided arbitrarily into three groups.

Group 1: Mild hypertrophy: ratio of left to right ventricle less than 2, but right ventricular weight less than 80 g.

Group 2: Moderate hypertrophy: right ventricular weight 80–100 g.

Group 3: Severe hypertrophy: right ventricular weight over 100 g.

Results

The clinical details, ventricular weights, and electrocardiographic findings from the 46 patients are recorded in Tables I to VI. In the Tables the patients are arranged in ascending order of right ventricular weight. When reference is made in the text to an individual patient the initials and right ventricular weight (RVW) are given so that they can be identified rapidly in the Tables. Of the 46 patients, 13 had no hypertrophy of either ventricle, 20 had isolated right ventricular hypertrophy, and 7 had hypertrophy of both ventricles. The remaining 6 had electrocardiographic or necropsy evidence of myocardial ischaemia. Of the 20 patients with isolated right ventricular hypertrophy, 4 had mild, 7 moderate, and 9 severe hypertrophy.

One of the patients (A.W.) without ventricular hypertrophy (RVW, 42·5 g.) had a ventricular ratio (L/R) of 3·5 and a history of thyrotoxicosis which had been successfully treated 5 years before death. Although a ventricular ratio of 3·5 is above the normal range quoted by Fulton et al. (1952), no myocardial abnormality was found and the left ventricle plus septum weighed 149 g., which is well within the normal range.

With one exception, the 7 patients with hypertrophy of both ventricles had a right ventricle of 80 g. or more, a left ventricle of 190 g. or more, and a ventricular ratio (L/R) greater than 2·0. The exception was A.K. (RVW, 127 g.) who had a ratio of 1·7 and therefore had relative right ventricular preponderance. However, he had a left ventricle of 212·5 g. and a history of systemic hypertension. According to Fulton et al. this degree of left ventricular hypertrophy is more than can be accounted for by hypertrophy secondary to an increase in the size of the right ventricle. He was, therefore, included as a case of hypertrophy of both ventricles.

Frontal Plane Axis. None of the patients without hypertrophy of either ventricle (see Table I) had right axis deviation. Of the 20 patients with isolated right ventricular hypertrophy (see Tables II–IV), 16 had right axis deviation (91° to 180°). The 4 exceptions were T.H. (RVW, 60 g.) who had an axis of +60° and the smallest right ventricle in the group, B.B. (RVW, 81 g.) who had an axis of +90° which is borderline, A.S. (RVW, 98 g.) whose axis could not be calculated because the positive and negative deflections were almost equal, and H.J. (RVW, 78 g.) who had an axis of −80°.

There was a tendency for the axis to increase with increasing degrees of right ventricular hypertrophy, but for individual patients the correlation between the axis and the degree of hypertrophy was poor. Individual correlations were no better if the axes were compared with the ventricular ratios.

None of the patients with hypertrophy of both ventricles had right axis deviation. Of the 6 patients with myocardial ischaemia, 4 had moderate or severe right ventricular hypertrophy, but only W.W. (RVW, 114 g.) had right axis deviation.

The results show that right axis deviation is a reliable sign of right ventricular hypertrophy, but the extent of the axis deviation is a poor guide to the degree of hypertrophy. The absence of right axis deviation is good evidence against moderate or severe hypertrophy, providing left ventricular hypertrophy and myocardial ischemia can be excluded.

Prercordial Pattern. The height of the R wave in V1 and V2 in the patients without hypertrophy of either ventricle ranged from 0·5 to 5·0 mm. Only 1 patient in this group had a secondary R wave, and this patient C.M. (RVW, 75 g.) had an axis of −165°. Among the 20 patients with isolated right ventricular hypertrophy, 6 had R waves of more than 5·0 mm. and 4 had secondary R waves. The height of the R wave tended to increase with increasing right ventricular hypertrophy, but there was considerable variation. None of the patients with hypertrophy of both ventricles had an R wave of more than 5·0 mm. Of the patients with ischaemia and right ventricular hypertrophy, 2 had R
### TABLE I

**PATIENTS WITHOUT RIGHT VENTRICULAR HYPERTROPHY**

<table>
<thead>
<tr>
<th>Initials</th>
<th>Age (yr.)</th>
<th>Pulmonary diagnosis</th>
<th>Non-pulmonary diagnosis</th>
<th>RVW (g.)</th>
<th>Ratio (L/R)</th>
<th>Frontal plane axis</th>
<th>P wave (mm.)</th>
<th>P axis</th>
<th>Right</th>
<th>Left</th>
<th>Maximum total deflection</th>
<th>Goodwin and Abdin grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.M.</td>
<td>46</td>
<td>Widespread emphysema (primary); aspergillosis</td>
<td>Peritonitis following mesenteric infarct</td>
<td>28</td>
<td>3:2</td>
<td>+20° 1:5 &gt;60°</td>
<td>1 — 0:02</td>
<td>5 8 8</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>W.M.</td>
<td>62</td>
<td>Pulmonary tuberculosis</td>
<td>Carcinoma of larynx; syphilis (non-cardiac)</td>
<td>30-5</td>
<td>2:8</td>
<td>+90° 2:0 &gt;60°</td>
<td>2 — 0:02</td>
<td>7 12 17</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B.F.</td>
<td>75</td>
<td>Chronic bronchitis; localized emphysema</td>
<td>Carcinoma of stomach and colon</td>
<td>33</td>
<td>3:1</td>
<td>+75° 1:0 &gt;60°</td>
<td>3 — 0:02</td>
<td>3 13 10</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.W.</td>
<td>63</td>
<td>Asthma</td>
<td>Thyrotoxicosis 5 years before death</td>
<td>42-5</td>
<td>3:5</td>
<td>+30° 2:0 &gt;60°</td>
<td>3 — 0:02</td>
<td>2 12 12</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I.E.</td>
<td>67</td>
<td>Chronic bronchitis</td>
<td>Reticulum cell sarcoma</td>
<td>43</td>
<td>3:0</td>
<td>+30° 1:0 &gt;60°</td>
<td>0:5 — 0:02</td>
<td>12 5</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.J.</td>
<td>73</td>
<td>Asthma</td>
<td>Senile dementia</td>
<td>45</td>
<td>2:8</td>
<td>+75° 1:5 &lt;60°</td>
<td>3 — 0:04</td>
<td>12 8</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.Z.</td>
<td>77</td>
<td>Chronic bronchitis; localized emphysema</td>
<td>Carcinoma of pancreas</td>
<td>46:5</td>
<td>2:8</td>
<td>+60° 1:0 &gt;60°</td>
<td>5 — 0:02</td>
<td>5 9 15</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.M.</td>
<td>67</td>
<td>Chronic bronchitis; carcinoma of bronchus</td>
<td>Carcinoma of bile-ducts</td>
<td>45</td>
<td>3:0</td>
<td>+20° 1:0 &gt;60°</td>
<td>0:5 — 0:02</td>
<td>10(S) 6 12</td>
<td>I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R.H.</td>
<td>71</td>
<td>Asthma; localized emphysema</td>
<td></td>
<td>57:0</td>
<td>2:3</td>
<td>+65° 1:5 &gt;60°</td>
<td>4 — 0:02</td>
<td>8 11 10</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E.H.</td>
<td>57</td>
<td>Chronic bronchitis; asthma; widespread emphysema</td>
<td></td>
<td>60</td>
<td>2:0</td>
<td>+70° 2:0 &gt;60°</td>
<td>0:5 — 0:02</td>
<td>14 8</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.B.</td>
<td>37</td>
<td>Chronic bronchitis; widespread emphysema</td>
<td></td>
<td>60:5</td>
<td>2:5</td>
<td>-30° 1:0 &gt;60°</td>
<td>3 — 0:02</td>
<td>7 6 10</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.H.</td>
<td>44</td>
<td>Chronic bronchitis; widespread emphysema</td>
<td></td>
<td>62:5</td>
<td>2:1</td>
<td>+55° 1:0 &gt;60°</td>
<td>2 — 0:02</td>
<td>4 13 10</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C.M.</td>
<td>63</td>
<td>Chronic bronchitis; widespread emphysema</td>
<td></td>
<td>75</td>
<td>2:0</td>
<td>-165° 1:0 &lt;60°</td>
<td>2 1 0:04</td>
<td>4 10 4</td>
<td>II</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RVW: right ventricular weight.  
Int: intrinsicoid deflection.  
(S): Dominant S in all precordial leads.

### TABLE II

**PATIENTS WITH MILD RIGHT VENTRICULAR HYPERTROPHY**

<table>
<thead>
<tr>
<th>Initials</th>
<th>Age (yr.)</th>
<th>Pulmonary diagnosis</th>
<th>RVW (g.)</th>
<th>Ratio (L/R)</th>
<th>Frontal plane axis</th>
<th>P wave (mm.)</th>
<th>P axis</th>
<th>Right</th>
<th>Left</th>
<th>Maximum total deflection</th>
<th>Goodwin and Abdin grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>T.H.</td>
<td>74</td>
<td>Chronic bronchitis</td>
<td>60</td>
<td>1:7</td>
<td>+60° 3:0 &lt;60°</td>
<td>2 — 0:02</td>
<td>3 13 10</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.B.</td>
<td>56</td>
<td>Asthma; chronic bronchitis</td>
<td>67</td>
<td>1:9</td>
<td>+110° 0:5</td>
<td>* 0:5 — 0:02</td>
<td>2 6 4</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S.W.</td>
<td>39</td>
<td>Chronic bronchitis; widespread emphysema</td>
<td>75</td>
<td>1:7</td>
<td>+100° 3:0 &gt;60°</td>
<td>0:5 1 0:06</td>
<td>15(S) 13 12</td>
<td>I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H.J.</td>
<td>66</td>
<td>Chronic bronchitis; widespread emphysema</td>
<td>78</td>
<td>1:8</td>
<td>-80° 1:0 &lt;60°</td>
<td>2 — 0:02</td>
<td>9 16 13</td>
<td>II</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* P waves too small for axis to be calculated.
waves of more than 5·0 mm., and 1 of the remaining 2 had a secondary R wave of more than 5·0 mm. However, 1 of the patients with ischaemia but no right ventricular hypertrophy had an R wave of 8·0 mm.

The depth of the S wave in V5 and V6 in the patients without hypertrophy of either ventricle (excluding L.Z., RVW 45 g., who showed a dominant S in all the precordial leads) ranged from 0 to 8·0 mm. Among the 20 patients with isolated right ventricular hypertrophy, 14 had an S wave of 10 mm. or more. As with the other electrocardiographic changes, the depth of the S wave tended to increase with increasing right ventricular hypertrophy, but the correlation in individual cases was poor. Among the patients with hypertrophy of both ventricles, only 1 had an S wave of 10 mm. or more, and of the 4 with right ventricular hypertrophy and ischaemia, 2 had S waves of 10 mm. or more.


**Electrocardiogram in Chronic Lung Disease**

**TABLE V**

PATIENTS WITH HYPERTROPHY OF BOTH VENTRICLES

<table>
<thead>
<tr>
<th>Initials</th>
<th>Age (yr.)</th>
<th>Pulmonary diagnosis</th>
<th>Non-pulmonary diagnosis</th>
<th>RVW (g.)</th>
<th>LVW (g.)</th>
<th>Ratio (L/R)</th>
<th>Frontal plane axis</th>
<th>P wave (mm.)</th>
<th>P axis</th>
<th>Right</th>
<th>Left</th>
<th>Maximum total deflection</th>
</tr>
</thead>
<tbody>
<tr>
<td>H.P.</td>
<td>49</td>
<td>Chronic bronchitis</td>
<td>Anterior myocardial infarct</td>
<td>81</td>
<td>216</td>
<td>2.5</td>
<td>+50°</td>
<td>2.5</td>
<td>&gt;60°</td>
<td>0.5</td>
<td>5</td>
<td>15</td>
</tr>
<tr>
<td>G.S.</td>
<td>60</td>
<td>Chronic bronchitis; healed pulmonary tuberculosis; localized emphysema</td>
<td>Myocardial ischemia</td>
<td>80</td>
<td>190.5</td>
<td>2.4</td>
<td>+10°</td>
<td>2.0</td>
<td>&gt;60°</td>
<td>2</td>
<td>—</td>
<td>22</td>
</tr>
<tr>
<td>V.V.</td>
<td>50</td>
<td>Bronchiectasis; asthma</td>
<td>Systemic hypertension; amyloid (non-cardiac)</td>
<td>96</td>
<td>281</td>
<td>2.9</td>
<td>+65°</td>
<td>1.0</td>
<td>&lt;60°</td>
<td>5</td>
<td>—</td>
<td>29</td>
</tr>
<tr>
<td>F.S.</td>
<td>72</td>
<td>Chronic bronchitis</td>
<td>Myocardial ischemia</td>
<td>102</td>
<td>270</td>
<td>2.6</td>
<td>+50°</td>
<td>1.5</td>
<td>&lt;60°</td>
<td>1</td>
<td>—</td>
<td>40</td>
</tr>
<tr>
<td>P.W.</td>
<td>57</td>
<td>Chronic bronchitis</td>
<td>Myocardial ischemia</td>
<td>102</td>
<td>272.5</td>
<td>2.6</td>
<td>↑</td>
<td>1.0</td>
<td>&gt;60°</td>
<td>1</td>
<td>—</td>
<td>25</td>
</tr>
<tr>
<td>A.K.</td>
<td>60</td>
<td>Chronic bronchitis</td>
<td>Systemic hypertension</td>
<td>127</td>
<td>212.5</td>
<td>1.7</td>
<td>-50°</td>
<td>4.0</td>
<td>&lt;60°</td>
<td>1</td>
<td>—</td>
<td>32</td>
</tr>
<tr>
<td>H.S.</td>
<td>64</td>
<td>Bronchiectasis</td>
<td>Myocardial ischemia</td>
<td>138</td>
<td>314</td>
<td>2.2</td>
<td>-80°</td>
<td>1.0</td>
<td>&gt;60°</td>
<td>2</td>
<td>—</td>
<td>19</td>
</tr>
</tbody>
</table>

**TABLE VI**

PATIENTS WITH MYOCARDIAL DISEASE BUT WITHOUT LEFT VENTRICULAR HYPERTROPHY

<table>
<thead>
<tr>
<th>Initials</th>
<th>Age (yr.)</th>
<th>Pulmonary diagnosis</th>
<th>Non-pulmonary diagnosis</th>
<th>RVW (g.)</th>
<th>LVW (g.)</th>
<th>Ratio (L/R)</th>
<th>Frontal plane axis</th>
<th>P wave (mm.)</th>
<th>P axis</th>
<th>Right</th>
<th>Left</th>
<th>Maximum total deflection</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.F.</td>
<td>67</td>
<td>Chronic bronchitis; localized emphysema</td>
<td>Myocardial ischemia</td>
<td>52.5</td>
<td>145</td>
<td>2.8</td>
<td>+30°</td>
<td>0.5</td>
<td>*</td>
<td>0.5</td>
<td>—</td>
<td>0.02</td>
</tr>
<tr>
<td>B.M.</td>
<td>60</td>
<td>Chronic bronchitis and bronchiectasis; localized emphysema</td>
<td>Posterior myocardial infarct</td>
<td>56.5</td>
<td>165-5</td>
<td>2.8</td>
<td>+30°</td>
<td>1.0</td>
<td>&lt;60°</td>
<td>8</td>
<td>—</td>
<td>0.02</td>
</tr>
<tr>
<td>R.W.</td>
<td>59</td>
<td>Chronic bronchitis</td>
<td>Anterior myocardial infarct; right bundle-block</td>
<td>85.5</td>
<td>145</td>
<td>1.7</td>
<td>+80°</td>
<td>1.0</td>
<td>&gt;60°</td>
<td>11</td>
<td>—</td>
<td>0.1</td>
</tr>
<tr>
<td>G.P.</td>
<td>62</td>
<td>Idiopathic pulmonary fibrosis</td>
<td>Posterior myocardial infarct</td>
<td>88</td>
<td>153</td>
<td>1.1</td>
<td>-165°</td>
<td>2.5</td>
<td>&gt;60°</td>
<td>3</td>
<td>14</td>
<td>0.06</td>
</tr>
<tr>
<td>H.D.</td>
<td>66</td>
<td>Chronic bronchitis; carcinoma bronchus</td>
<td>Myocardial ischemia</td>
<td>103</td>
<td>180</td>
<td>1.8</td>
<td>+10°</td>
<td>1.0</td>
<td>&gt;60°</td>
<td>11</td>
<td>—</td>
<td>0.04</td>
</tr>
<tr>
<td>W.W.</td>
<td>55</td>
<td>Chronic bronchitis</td>
<td>Anterior myocardial infarct</td>
<td>114</td>
<td>185</td>
<td>1.6</td>
<td>+120°</td>
<td>3.0</td>
<td>&gt;60°</td>
<td>1</td>
<td>5</td>
<td>0.06</td>
</tr>
</tbody>
</table>

An intrinsicoid deflection of 0.04 sec. or more was present in half the patients with isolated right ventricular hypertrophy, but was also present in two of the patients without hypertrophy of either ventricle. Only one of the patients with hypertrophy of both ventricles had a prolonged intrinsicoid deflection. All the patients with right ventricular hypertrophy and myocardial ischemia had an intrinsicoid deflection of 0.04 sec. or more, and one had right bundle-branch block.

The results show that providing myocardial ischemia is excluded, an R wave over the right ventricle of 6 mm. or more indicates right ventricular hypertrophy. Providing emphysema is excluded, an S wave over the left ventricle of 10 mm. or more indicates right ventricular hypertrophy.
However, the absence of these signs does not exclude right ventricular hypertrophy. A prolonged intrinsicoid deflection is of little value in the diagnosis of right ventricular hypertrophy.

**P Wave.** Only 8 of the 20 patients with isolated right ventricular hypertrophy had P waves of 2.5 mm. or more. Two of the 7 patients with hypertrophy of both ventricles and 2 of the 4 with myocardial ischaemia and right ventricular hypertrophy had P waves of 2.5 mm. or more. A P wave axis of more than +60° occurred with almost equal frequency among the patients with and without right ventricular hypertrophy.

Therefore, a P wave of 2.5 mm. or more indicates right ventricular hypertrophy but its absence does not exclude it. A tall P wave may be of greater value than other electrocardiographic signs in the presence of hypertrophy of both ventricles or myocardial ischaemia, but more cases would have to be studied in order to prove this. A P wave axis of more than +60° is of no value in the diagnosis of right ventricular hypertrophy.

**Goodwin and Abdin’s Criteria.** Among the 20 patients with isolated right ventricular hypertrophy, 15 had electrocardiograms showing Grade I or more. The criteria gave 2 false positives among the patients without ventricular hypertrophy. One of these (L.Z., RVW, 45 g.) had a dominant S wave in all the precordial leads so that there was a dominant S in V5. The other had a right ventricle weighing 75 g. which is above the upper limit of normal given by Fulton et al. (1952), though less than 80 g. which is the lower limit for right ventricular hypertrophy. The correlation between the electrocardiographic grades and the degree of hypertrophy was poor.

**Effect of Emphysema on Electrocardiogram.** Table VII shows the findings in the 12 patients with radiographic evidence of widespread emphysema. Only one patient (H.J., RVW, 78 g.) had left axis deviation and his electrocardiogram is shown in the Figure. The T wave axis was +90° giving an angle of 170° between the QRS and T axes. A wide angle between the QRS and T axes is suggestive of myocardial ischaemia. Although there was no macroscopic evidence of this at necropsy, ischaemia may have been the cause of the left axis deviation. If emphysema had been the cause of the abnormal axis it would have been expected to cause deviation of the T axis in the same direction as the QRS. Only one patient (F.J.; RVW, 143 g.) had a low voltage electrocardiogram.

Of the 12 patients with widespread emphysema, 5 had dominant S waves in all the precordial leads. This pattern may be due to emphysema but was also seen in 2 patients without emphysema. There were no other common features which could be attributed to the presence of emphysema.

**DISCUSSION**

The present study demonstrates the value of right axis deviation in the diagnosis of right ventricular hypertrophy due to chronic lung disease. Einthoven (1906, 1908) was the first to suggest axis deviation as a sign of ventricular hypertrophy. Phillips (1958) and Roman et al. (1961) emphasized the value of right axis deviation as a sign of right ventricular hypertrophy, and Shipley and Hallaran (1936) and Packard, Graettinger, and Graybiel (1954) have shown that it is rare in normal adults over the age of 30.
Electrocardiogram in Chronic Lung Disease

Goodwin and Abdin's criteria depend on the ratio of the R and S waves in the præcordial leads, and in their original study the correlation between the electrocardiographic changes and the degree of right ventricular hypertrophy (measured by wall thickness) was fairly good. However, only 16 out of the 117 patients in their study had chronic lung disease. Caird and Wilcken (1962), using the same anatomical and electrocardiographic criteria, failed to find any correlation between the degree of hypertrophy and the electrocardiographic grade. The present study confirms this.

The lack of correlation between the præcordial pattern and the degree of hypertrophy may be due to alteration of the position of the præcordial leads in relation to the heart. In emphysema or any lung condition in which there is overinflation of the chest, the heart tends to be displaced downwards and the antero-posterior diameter of the chest increased. These changes may disturb the balance of the R and S waves across the præcordium and partially mask the effects of ventricular hypertrophy. The dominant S wave, which was found in nearly half the patients with widespread emphysema, could be explained by extreme downward displacement of the heart in relation to the præcordial leads.

In the present study emphysema was diagnosed from the chest radiograph using the criteria of Simon (1964). Reid and Millard (1964) in a comparison of the radiograph during life with the lung at necropsy found that Simon's criteria were reliable, though severe and extensive emphysema must be present before radiographic changes can be detected. Thus, though minor degrees of emphysema may have been missed, it is likely that the 12 patients in whom emphysema was diagnosed had extensive changes in the lungs. However, apart from dominant S waves in the præcordial leads, the electrocardiograms in this group had little in common and did not show the changes commonly attributed to emphysema. Left axis deviation, which Grant (1956) suggested might be caused by emphysema, was present in only one patient and in this case may have been due to myocardial ischaemia. Wasserburger et al. (1959) and Littmann (1960) described a vertical electrical axis in emphysema, but a vertical axis was no more common in the patients with widespread emphysema than in those without. The low voltage in emphysema observed by Selvester and Rubin (1965) was found in only one instance.

The present series is small and does not exclude the possibility that emphysema may sometimes cause electrocardiographic changes. However, it is possible that some of the changes previously attributed to emphysema may have been due to undetected myocardial disease.

SUMMARY AND CONCLUSIONS
A comparison was made between the electrocardiograms and the ventricular weights of 46 patients with chronic lung disease. Right axis deviation (frontal plane axis +91° to ±180°) was
found to be the most reliable sign of right ventricular hypertrophy, but in the presence of left ventricular hypertrophy or myocardial ischaemia the electrocardiogram was of little value in the diagnosis of right ventricular hypertrophy. None of the electrocardiographic changes investigated were a reliable guide to the degree of ventricular hypertrophy.

Apart from dominant S waves in the precordial leads, the electrocardiograms from the patients with emphysema had nothing in common. It is suggested that some of the changes previously attributed to emphysema may be due to myocardial disease.

I should like to thank Dr. Lynne Reid, Professor A. C. Dornhorst, Dr. M. Towers, and my wife, Dr. R. E. Millard, for helpful advice and criticism, Dr. G. Simon, who read the chest radiographs, and Dr. K. F. W. Hinson and Dr. D. K. Henry of the Pathology Department of the Brompton Hospital, who supplied the pathological material.

Part of this work was done during the tenure of a Brompton Hospital Research Grant.

APPENDIX

VENTRICULAR WEIGHTS IN CARDIAC HYPERTROPHY
(Fulton et al., 1952)

"Criteria for normality: A heart may be classed as normal only if: (A) the total ventricular weight is less than 250 g. (B) the free wall of the right ventricle weighs less than 65 g. (C) the left ventricle and septum together weigh less than 190 g. (D) the ratio left ventricle plus septum to right ventricle lies between 2:3:1 and 3:3:1.

Criteria for right ventricular hypertrophy: Right ventricular hypertrophy is considered to be present when the free wall of the right ventricle weighs 80 g. or more. In isolated right ventricular hypertrophy the ratio left ventricle plus septum to right ventricle is always less than 2:1. If left ventricular hypertrophy is also present, the ratio may be within normal limits or even raised.

Criteria for left ventricular hypertrophy: Left ventricular hypertrophy is considered to be present when the weight of the left ventricle plus septum is 225 g. or more. The ratio left ventricle plus septum to right ventricle may be modified by secondary or independent right ventricular hypertrophy and the ratio alone is, therefore, not an indication either of the presence or degree of left ventricular hypertrophy."

REFERENCES


The electrocardiogram in chronic lung disease.

F J Millard

*Br Heart J* 1967 29: 43-50
doi: 10.1136/hrt.29.1.43

Updated information and services can be found at:
http://heart.bmj.com/content/29/1/43.citation

These include:

Email alerting service

Receive free email alerts when new articles cite this article.
Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/