The Electrocardiogram in Chronic Bronchitis and Emphysema

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A study has been made of the electrocardiogram in patients primarily suffering from chronic bronchitis. Airways obstruction and emphysema existed to a variable degree in these patients, and an attempt is made to assess the influence of these factors on the electrocardiogram.

Earlier papers relating to electrocardiographic changes in chronic bronchitis, emphysema, and other chronic forms of pulmonary disease are numerous. Scott (1961), in a good summary, refers to P pulmonale, prominent Tα waves, rightward deviation of the P axis and mean frontal QRS axis, low voltage QRS complexes, negative W shaped, or transitional (QS, rSR, QRS, RS) complexes in the praecordial leads, rSR' in V1, incomplete or complete right bundle-branch block, classical right ventricular hypertrophy with dominant R in V1 with or without T wave inversion in right praecordial leads, and abnormal left axis deviation.

Factors thought to explain these changes include dilatation and/or hypertrophy of the right atrium and right ventricle, diminished electrical conductivity by the emphysematous lung (supported by the experimental studies of Schwan and Kay, 1956), and altered position of the heart (clockwise rotation, vertical position, and backward rotation of the apex consequent on low diaphragms and hyperinflation of the lungs). The heart may certainly lie low in the chest in emphysema, thus accounting for predominantly negative QRS complexes to V5 or V6 ("clockwise rotation"). However, attempts to assess rotation in coronal and sagittal planes by correlation of the anatomical axis of the heart, obtained from anterior and lateral radiographs of the chest, with the electrical axis (Hyman, Failey, and Ashman, 1948; Fowler and Braunstein, 1951), are clearly subject to considerable error, since there is no way of locating the interventricular septum. Grant (1953), in a series of careful anatomical studies, found little correlation between the electrical (mean QRS) and anatomical axes, and Guntheroth, Ovenfors, and Ikoss (1961) reached similar conclusions as a result of biplane angiocardiographic studies in children.

The continued search for electrocardiographic signs of right ventricular hypertrophy reflects the difficulty of diagnosing the condition especially in acquired as opposed to congenital heart disease. The subject has been critically reviewed by Scott (1960). F. J. C. Millard (1964, personal communication) has made a detailed electrocardiographic and pathological study of right ventricular hypertrophy in adult patients with chronic pulmonary disease, using an accurate method of assessment of ventricular weight (Fulton, Hutchinson, and Jones, 1952). A mean QRS axis between +91° and +180° was found in 85 per cent of patients with isolated right ventricular hypertrophy and in no patients without right ventricular hypertrophy. There was little correlation between the degree of QRS axis shift to the right and the severity of right ventricular hypertrophy. The electrocardiogram was not helpful in diagnosing right ventricular hypertrophy in patients with biventricular hypertrophy. Millard concluded that in chronic pulmonary disease the position of the mean QRS axis provided as good an indication of right ventricular hypertrophy as any other electrocardiographic sign.

SUBJECTS AND METHODS

The 112 patients studied, 91 men and 21 women, were aged from 24 to 72 years. All had chronic bronchitis, as defined at the Ciba Guest Symposium (1959), but no other known or suspected pulmonary disease, rheumatic or ischemic heart disease, or hypertension (defined as a diastolic blood pressure of 110 mm. Hg or more). The
patients had all been referred to the hospital out-patient department by their practitioners, and they were studied when in their optimum clinical state. Since the investigation began in 1961, 10 have died. Histories were recorded on the M.R.C. Short Questionnaire on Respiratory Symptoms (1960).

Radiographs were assessed independently for emphysema and classified as normal, possible slight, localized, or widespread, according to criteria (signs of hyperinflation, pulmonary vascular changes, avascular areas) described by Simon (1964). The validity of this interpretation has been confirmed by the radiographic and pathological study of Reid and Millard (1964).

Peak expiratory flow rates (PEF) were measured with Wright's flowmeter (Wright and McKerrow, 1959). The forced expiratory volume in 1 second (FEV₁) was also recorded since it is a more discriminating test of respiratory function (Fairbairn et al., 1962).

Electrocardiograms were recorded with Sanborn direct writers employing conventional standardization (1 cm. = 1 mV). They were analysed for the following features:

1. P pulmonale (peaked P > 2.5 mm. high in any standard limb lead).
2. P axis calculated by the method of Grant (1957). The normal limits of P axis were taken as +15° to +75° (Chevalier and Thaon, 1949; Masini and Rossi, 1951; His, Lamb, and Allen, 1960).
3. Mean frontal QRS axis by the method of Grant (1950). The normal range was considered to be −30° to +90°. QRS axes between −30° and −90° indicate abnormal left axis deviation and between +91° and +180° right ventricular hypertrophy (F. J. C. Millard, 1964, personal communication).
4. Other abnormalities. The criteria for right bundle-branch block (RBBB) were: QRS duration > 0.1 sec., terminal slurred S wave in lead I (terminal QRS vector between +90° and −90°), and terminal R waves in V₁ and V₂. S₁S₂S₃ syndrome refers to a condition in which the QRS complex is not prolonged, and there are terminal S waves in leads I, II, and III (terminal QRS vector between −90° and −150°).

**TABLE I**

<table>
<thead>
<tr>
<th>Radiographic classification</th>
<th>No. of patients</th>
<th>Age (mean and range)</th>
<th>Mean frontal QRS axis</th>
<th>P pulmonale</th>
<th>Mean P axis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Widespread emphysema</td>
<td>22 male, 2 female</td>
<td>58.3 (42-72)</td>
<td>+91° to +180°</td>
<td>13</td>
<td>+76° to +90°</td>
</tr>
<tr>
<td>Possible slight emphysema</td>
<td>6 male, 5 female</td>
<td>55.4 (24-72)</td>
<td>−90° to +90°</td>
<td>3</td>
<td>0° to +75°</td>
</tr>
<tr>
<td>Localized emphysema</td>
<td>15 male, 48 female</td>
<td>57.1 (24-72)</td>
<td>−30° to +90°</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>No emphysema</td>
<td></td>
<td></td>
<td>−90° to +180°</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

* 1 QRS axis indeterminate.
† 2 QRS axes indeterminate.
‡ Includes 1 case of right bundle-branch block.
Electrocardiogram in Chronic Bronchitis and Emphysema

TABLE II

<table>
<thead>
<tr>
<th>Widespread emphysema</th>
<th>No. of patients</th>
<th>Right ventricular hypertrophy</th>
<th>Normal QRS axis</th>
<th>Left axis deviation</th>
<th>P pulmonale</th>
<th>Rightward deviation of P axis</th>
<th>Normal P axis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>24</td>
<td>4 (17%)</td>
<td>14</td>
<td>5 (21%)</td>
<td>6 (25%)</td>
<td>13 (54%)</td>
<td>11</td>
</tr>
<tr>
<td>Possible slight, localized, or no emphysema (PEF &lt; 200 l/min, FEV₁ &lt; 1,200 ml.)</td>
<td>43</td>
<td>7 (16%)</td>
<td>23</td>
<td>3 (7%)</td>
<td>5 (12%)</td>
<td>16 (37%)</td>
<td>27</td>
</tr>
<tr>
<td>Possible slight, localized, or no emphysema (PEF &gt; 200 l/min, FEV₁ &gt; 1,200 ml.)</td>
<td>45</td>
<td>0</td>
<td>44</td>
<td>0</td>
<td>0</td>
<td>3 (7%)</td>
<td>42</td>
</tr>
</tbody>
</table>

or no airways obstruction had virtually normal electrocardiograms. As expected from these findings, the correlations of these four main electrocardiographic abnormalities with severe breathlessness (Grades 4, 5 dyspnoea), PEF < 200 l/min., and FEV₁ < 1,200 ml. are statistically significant (p varying from 0.0005 to 0.05). There was, however, no significant association between P pulmonale and vertical P axis and the various QRS abnormalities (right ventricular hypertrophy, left axis deviation, right bundle-branch block, S₁S₂S₃ syndrome, and indeterminate QRS axis).

Other Abnormalities. Excluding right ventricular hypertrophy, 4 tracings showed isoelectric or inverted T waves in various leads, presumably due to ischaemia, and in a further 9 there were small secondary r waves in the right præcordial leads. Ventricular ectopic beats occurred in 10 electrocardiograms. During acute exacerbations of bronchitis one patient had atrial flutter, and another had paroxysmal atrial fibrillation, but there were no persistent arrhythmias. There were 2 cases of right and one of left bundle-branch block. The mean QRS axis was indeterminate in 3 patients. One of these and 2 others with mean QRS axes of -100° and -120° were examples of the S₁S₂S₃ syndrome. Left axis deviation deserves special mention. The mean QRS axis was -35° (later -85°), -35°, -50°, -60°, and -60°, respectively, in 5 patients with widespread emphysema. Two of these had isoelectric T waves in lead I (T vector +90°; wide QRS-T angle), possibly indicating myocardial ischaemia. In the other 3 patients, there was no obvious cause for the left axis deviation. Three patients with localized or no emphysema had mean QRS axes of -40°, -45° and -70°. The first also had a T vector of +90° and the second showed antero-lateral peri-infarction block.

Discussion

Rightward deviation of the P axis in chronic pulmonary disease has been noted by Novelo (1945), Zuckermann et al. (1948), Vaquero (1948), Lenègre, Maurice, and Scebat (1954), and Littmann (1960). Caird and Wilcken (1962) who also confined their investigation to patients with chronic bronchitis, found a P axis of +80° or more in 29 (50%) of 58 cases, but there was no relation between the P axis and ventilatory function as measured by FEV₁. Spodick et al. (1963) observed a P axis of +70° to +90° in 220 (73%) of 301 cases of "pulmonary emphysema", and there was significant correlation between vertical P axis and increasing severity of airways obstruction. In our study, a P axis between +75° and +90° occurred in only 32 (29%) of 112 patients. It correlated well with severe dyspnoea and airways obstruction but not with cardiographic right ventricular hypertrophy. The significantly higher incidence of vertical P axis in patients with widespread emphysema as opposed to those with slight, localized, or no emphysema, was a reflection of the uniformly severe airways obstruction found in the former group. P pulmonale, which was found in only 10 per cent of our patients, was always associated with marked impairment of ventilatory capacity.

The small (< 5 mm.) secondary r waves found in V₁ or V₂ in the absence of signs of right ventricular hypertrophy, right bundle-branch block, peri-infarction block, or other evidence of coronary artery disease, were presumably a normal finding (Camerini and Davies, 1955; Said and Bryant, 1956) though Mounsey, Ritzmann, and Selverstone (1952) found that their occurrence in chronic pulmonary disease was often a stage in the development of a classical right ventricular hypertrophy pattern. Right bundle-branch block occurred in 2 patients. It has been suggested that right ventricular hypertrophy may be diagnosed in the presence of RBBB if the secondary r wave in V₁ exceeds 15 mm. in amplitude (10 mm. if QRS duration less than 0·12 sec.—Barker and Valencia, 1949). However, the findings of Dodge and Grant (1956) and those of Booth, Chou, and Scott (1958) do not support this view.
The occurrence of left axis deviation in chronic pulmonary disease has been recorded by Lenègre et al. (1954), Coelho et al. (1962), and Banta, Greenfield, and Estes (1964). It may be due to associated left ventricular hypertrophy or ischemic heart disease with myocardial fibrosis. There was no clinical or electrocardiographic evidence of these conditions in 4 of our 8 patients with left axis deviation. Two of these patients have died: at necropsy one was found to have normal coronary arteries and a normal myocardium, and in the other myocardial fibrosis was present. Other suggested mechanisms of left axis deviation include altered position of the heart (Wasserburger et al., 1959; Burch and DePasquale, 1963), right ventricular hypertrophy (Lasser and Grishman, 1951; Curd, Hicks, and Gyorkey, 1961), and poor electrical conduction by the emphysematous or hyperinflated lung with “verticalization” of the electric field, and the axis illusion phenomenon (Grant, 1956; Talbot and Leonard, 1958; Spodick, 1959; Littmann, 1960). Some of these authors also mention the occurrence of indeterminate mean QRS axes, and S1S2S3 patterns. In patients with chronic pulmonary disease, the S1S2S3 syndrome may indicate right ventricular hypertrophy (Schwartz and Marcus, 1942; Carousou, Tilmant, and Lenègre, 1949; Pryor, 1964), but it may also occur in normal people, in congenital heart disease with right ventricular hypertrophy, and in myocardial infarction (Sodi-Pallares, 1956; Grant, 1957).

There was no significant difference in the incidence of right ventricular hypertrophy in chronic bronchitis with widespread emphysema and similarly disabled patients with less severe or no emphysema. This finding is contrary to commonly held views and comparable published work, which indicate a much higher incidence of right ventricular hypertrophy in chronic bronchitics without radiographic emphysema (Fletcher et al., 1963; Burrows et al., 1964). The electrocardiographic criteria of Goodwin and Abdin (1959) were used for the diagnosis of right ventricular hypertrophy in these authors’ series, but if applied to the 112 patients in this study an even higher incidence of right ventricular hypertrophy would result (Table III) in those with widespread emphysema.

The finding is clearly not due to the selective inclusion of a large number of patients with widespread emphysema in the present study, since the proportion with this radiographic abnormality (21%) is similar to that found (14–29%) in 4 other series comprising 1,431 patients with chronic bronchitis (Simon and Galbraith, 1953; Simon and Medvei, 1962; Fletcher et al., 1964; Burrows et al., 1965). There were no significant differences in age, mean transverse diameter of heart, and number of incidents of cardiac failure in our patients with right ventricular hypertrophy in association with widespread emphysema, and right ventricular hypertrophy with less severe or no emphysema, though the duration of symptoms was greater in the former group.

### TABLE III

<table>
<thead>
<tr>
<th>Radiographic classification</th>
<th>No. of patients</th>
<th>Right ventricular hypertrophy</th>
<th>Right ventricular hypertrophy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Widespread emphysema</td>
<td>24</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Slight, localized, or no</td>
<td>88</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>emphysema</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### SUMMARY

An electrocardiographic study has been made of 112 hospital out-patients suffering from chronic bronchitis. Rightward deviation of the P axis occurred in 32 (29%) patients, P pulmonale in 11 (10%), right ventricular hypertrophy in 11 (10%), and left axis deviation in 8 (7%). Less common abnormalities included small secondary r waves in right precordial leads, right and left bundle-branch block, indeterminate mean QRS axes, and S1S2S3 syndrome. Rightward deviation of the P axis, P pulmonale, and right ventricular hypertrophy were confined to patients with severe airways obstruction, and the presence or absence of widespread emphysema assessed radiographically did not appear to influence these electrocardiographic abnormalities. In so far as right ventricular hypertrophy is concerned, this finding is contrary to previously published evidence. Left axis deviation may be the sole electrocardiographic abnormality and the possible mechanisms concerned are discussed.

I am grateful to Dr. J. C. Batten for permission to study his patients at the Brompton Hospital, to Dr. G. Simon for interpreting the X-rays, and to Dr. Batten, Dr. W. Phillips, and Dr. A. Leatham for much helpful criticism and advice in the preparation of this paper.

### REFERENCES


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