Pulmonary Heart Disease

With Emphasis on Electrocardiographic Diagnosis

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The diagnosis of cor pulmonale presents considerable difficulty in its earlier stages. Clinical symptoms are not available until the right heart fails and the only reliable sign is right ventricular hypertrophy, which is not easy to demonstrate. The purpose of this investigation is to evaluate electrocardiographic patterns of these patients as a more practical means of diagnosis. Sixty-seven patients with cor pulmonale are included in this study, which endeavors to point out degrees of correlation between various electrocardiographic patterns and clinical factors, such as stages of the disease, its severity, its etiology, and the anatomic changes of the right ventricle. It also evaluates the degrees of specificity of the various patterns encountered in these patients.

The clinical diagnosis of early right-sided heart disease presents greater difficulties than does that of its counterpart, left-sided heart disease. Once the full-blown pattern of venous engorgement, hepatic congestion, and peripheral edema dominates the clinical picture, no substantial difficulty is encountered in the recognition of right-sided decompensation. At this stage the clinician has practically no need for further laboratory or clinical tests; the physical signs suffice. However, without the clear-cut picture of congestive failure, and especially in borderline stages, or before the advent of early failure, it takes more than the clinical skill of the physician to establish the diagnosis of pulmonary heart disease. This difficulty is often compounded by the presence of the signs and symptoms of the chronic pulmonary disease itself, the etiologic factor responsible for the right-sided heart disease. In a great majority of cases chronic pulmonary disease is characterized by pulmonary emphysema or some form of pulmonary fibrosis. The third major cause of pulmonary heart disease, namely, the various forms of disease in the pulmonary vessels as described by Mack and Snider, has no bearing on the subject of this paper and will not be discussed in detail.

Short of the stage of congestive failure it becomes almost impossible to determine clinically where chronic chest disease ends and right-sided heart disease begins, unless one can demonstrate hypertrophy of the right ventricle, or abnormal pressure changes in the right side of the heart or in the pulmonary artery by means of cardiac catheterization. This latter means of diagnosis is not generally available except in larger medical centers and is not easily practicable. Therefore, methods of recognition of the early hypertrophy of the right ventricle remain the only practical means of establishing the advent of clinical right-sided heart disease. In the great majority of cases early recognition of right ventricular hypertrophy is uncertain. The only means are by radiologic or fluoroscopic methods and by electrocardiography. Radiographic determination of right ventricular hypertrophy poses considerable difficulty and requires special training and special technics. Of all the cardiac chambers, enlargement of the right ventricle is the most difficult to demonstrate roentgenologically.2 Obvious right ventricular enlargement demonstrable in a posteroanterior or oblique chest film is mostly due to dilatation of the chamber rather than actual hypertrophy alone of the wall of the right ventricle.2 By the time dilatation is demonstrable the right side has probably already failed or is in actual failure and, therefore, clinical signs of failure will have been in evidence to establish the diagnosis. In other words, demonstrable enlargement by radiologic means is probably not evidence of early pulmonary heart disease but of heart disease in progress or in failure.

In our opinion chronic chest disease may be in existence for long periods before the advent of right-sided heart disease, just as systemic
hypertension may persist for years alone without any evidence of hypertensive heart disease. Actually pulmonary heart disease does not necessarily follow chronic pulmonary disease, just as hypertensive heart disease does not always follow systemic hypertension. It appears to us more logical to disregard the fine differentiation between pulmonary disease and pulmonary heart disease and to consider them together as comprising one “single disease-complex,” beginning with pulmonary disease and ending in severe cor pulmonale with cardiac failure. We offer the following simple functional classification of this disease-complex: I, severe pulmonary disease and mild anoxia; II, severe pulmonary disease with anoxia plus right-sided heart disease without failure; III, chronic cor pulmonale with cardiac failure. Stages I and II differ very little as far as cardiopulmonary function is concerned, the only difference being the advent of hypertrophy of the right ventricle in stage II. The symptoms manifested in stage II are the symptoms of the primary pulmonary disease, the early hypertrophy of the right ventricle causing no symptoms. We have not been impressed by substernal pain as a symptom in this stage. It is in stage III that one notes severe deterioration of the cardiopulmonary function and clinical evidence of right-sided failure.

During the past 3 years we have been particularly interested in the electrocardiographic diagnosis of pulmonary heart disease as a more practical means of diagnosis. Located as we are in the heart of one of the main hard coal centers of the country, we have had the opportunity of seeing and studying a large number of cases of chronic pulmonary disease, especially anthracosilicosis, emphysema, and the often accompanying pulmonary heart disease. We have developed the study with the following questions in mind: 1. Does clinical cor pulmonale often occur either in the absence of electrocardiographic manifestations or in the presence of manifestations that are not specific for cor pulmonale? 2. If nonspecific electrocardiographic changes are often seen, to what extent may these be taken in conjunction with the known presence of appropriate pulmonary disease to make a reasonable presumptive diagnosis? 3. Is there any correlation between the type of electrocardiographic changes and the clinical severity of the heart disease?

**MATERIALS AND METHODS**

The cases of cor pulmonale investigated in this study were obtained from patients currently hospitalized for either their cardiopulmonary disease or for unrelated conditions, and from review of the hospital records of the last 50 consecutive cases whose final discharge diagnoses included cor pulmonale. Each case was thoroughly reviewed, and was accepted for this study only if satisfactory evidence existed to implicate chronic pulmonary disease as the cause for the cardiac disorder. Acceptance was based on either postmortem findings when available, or on clinical grounds in accordance with the criteria of the etiology of heart disease as set forth by the American Heart Association. A total of 67 cases was found to be suitable for the purposes of this study.

Each of the patients selected was suffering from some form of chronic pulmonary disease complicated by the coexistence of heart disease with right ventricular involvement. The latter was indicated by clinical findings such as signs of right-sided heart failure, or by radiologic or electrocardiographic changes consistent with such an interpretation.

The diagnosis of the underlying pulmonary disease was dependent on the history, especially in regard to exposure to industrial dusts, the physical findings, changes in the lungs found on x-ray, pulmonary ventilatory tests, and in one case by pulmonary biopsy. Hematologic studies were also carried out to determine the presence or absence of secondary polycythemia. For the purpose of this paper, this is defined as a red blood count in excess of 6,000,000 or a hematocrit value of more than 55 volumes per cent.

The coexistence of heart disease was easily established when signs of cardiac failure had developed. In addition to the common clinical manifestations of venous engorgement, hepatomegaly with or without ascites, and edema, most cases were also investigated by determinations of the venous pressure, and Decholin and ether circulation times. Radiographic evidence of enlargement of the outflow tract of the right ventricle and dilatation of the pulmonary arteries was also accepted as an indication of right-sided heart disease (fig. 1). In some cases cardiac enlargement without any specific changes in chamber configuration was found and was considered as evidence of heart disease. Cardiac catheterization was not performed on any patient.

During the period of this study 7 patients under investigation were subjected to necropsy examination. Although the normal thickness of the right ventricular wall is stated to be 3 mm., right ventricular hypertrophy was diagnosed only when the
muscle mass measured 5 mm. or more. Because of the difficulty in obtaining entirely accurate measurements of the thickness of the right ventricle, only those were included who died during the period of this study and whose measurements were made personally by one or another of the authors with this investigation in mind. This has necessarily led to a limitation of the number of autopsied cases.

**Results**

**Sex and Age**

Consistent with the usual hospital population all patients studied were males. Ages varied from 31 to 87, but with the exception of 2 patients in the early thirties, all were above 50 years of age.

**Underlying Chronic Pulmonary Disease**

Because of local industrial conditions the majority of the patients studied had been engaged in mining anthracite coal for varying, but usually prolonged periods. Pneumoconiosis due to anthracosilicosis was therefore very common. Some degree of pulmonary emphysema coexisted in almost every instance, and, in many, signs of acute or chronic bronchitis were also found. This combination of lung diseases was present in 41 (61 per cent) cases. Pulmonary emphysema of undetermined etiology was found in 8 (12 per cent), and in an equal number it was secondary to chronic bronchitis. In addition, emphysema was present in 2 cases of bronchiectasis, and in 1 case each of lung abscess, pulmonary tuberculosis, and chronic diffuse pneumonitis of unknown etiology. The remainder of the patients consisted of 3 of uncomplicated bronchiectasis, 1 of chronic bronchitis, and 1 of pulmonary tuberculosis.

**Incidence of Polycythemia**

Secondary polycythemia was found in 9 of the cases studied. In each, a diagnosis of pulmonary emphysema had been made with or without one of the associated forms of chronic lung disease mentioned. In addition all of these cases showed signs of heart disease, and 7 were in cardiac failure.

**Electrocardiographic Findings**

In general, the electrocardiographic patterns in the 67 patients conformed to those already described in the literature, and fell into 1 of 6 groups, as illustrated in figure 2. There was no correlation between the type of tracing and the underlying pulmonary disease. Among the 9 patients with polycythemia, however, 3 fell in group II and 4 in group III.

*Group I (fig. 2, col. 1)*. This group was characterized by tall peaked P waves in leads II, III, and aVr, and sometimes in the right chest leads, associated with marked clockwise rotation. It included 11 cases (16 per cent). Two of these cases also showed an r/S ratio exceeding 1/2 in lead V6. Peaked P waves, however, were often associated with the other electrocardiographic patterns to be described. Also many of the cases of chronic pulmonary disease at this hospital showed the same P-wave changes and clockwise rotation without associated heart disease. The rotation alone may account for the change in the P wave.

*Group II (fig. 2, col. 2)*. Eighteen cases (27 per cent) fell into this group showing the classic pattern of right ventricular hypertrophy with a tall R wave in leads V1 or Vm with or without a preceding q wave, and followed by an inverted T wave.

*Group III (fig. 2, col. 3)*. Incomplete right bundle-branch block characterized by an rR' with a delayed intrinsicsoid deflection but with a QRS duration of less than 0.12 second, and
Fig. 2. Representative electrocardiographic patterns found among 67 cases of chronic cor pulmonale.
TABLE 1.—Association of the Electrocardiographic Pattern in 67 Cases of Chronic Cor Pulmonale with the Severity of the Heart Disease

<table>
<thead>
<tr>
<th></th>
<th>Peaked P, clockwise rotation</th>
<th>R in V₁ or V₅₇</th>
<th>rR' in V₁ or V₄₄</th>
<th>R.B.</th>
<th>Inverted T over right chest</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart disease with failure . . . . . .</td>
<td>10</td>
<td>14</td>
<td>12</td>
<td>2</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>Heart disease without failure . . . . . . . .</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>No other evidence of heart disease . . . . . . . . . .</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

An inverted T wave in leads V₁ or V₄₄ occurred in 18 cases (27 per cent). In 3 of the cases right ventricular hypertrophy could be diagnosed by the criteria of Barker 19, 20 as well as incomplete right bundle-branch block.

Group IV (fig. 2, col. 4). The characteristic electrocardiogram of complete right bundle-branch block was present in 5 cases (7 per cent), and in 3 of these Barker's criteria indicated the co-existence of right ventricular hypertrophy.

Group V (fig. 2, col. 5). T-wave inversions alone, without the previously described R or rR' in V₁ or V₄₄ were present in the right chest leads V₁,2,3 and sometimes V₄ in 8 cases (12 per cent). All these cases showed in addition moderate to marked clockwise rotation with or without peaking of the P waves. The T-wave abnormalities were persistent in 5 patients, one of whom was on maintenance digitalis even prior to the first tracing. They were not permanent in the remaining 3 cases, being present only intermittently in 2, and in the third having disappeared during a 1-year interval between tracings.

Group VI (fig. 2, col. 6). Miscellaneous types of electrocardiograms were obtained from 7 patients (10 per cent). The patterns included 3 normal, 1 with only clockwise rotation, 1 with chronic atrial fibrillation, and 2 with T-wave inversions in the left chest leads.

In all 67 cases of cor pulmonale studied, only 3 cardiac arrhythmias were found. These include chronic atrial fibrillation, paroxysmal atrial fibrillation, and atrial flutter.

Correlation of Electrocardiographic Patterns with Other Signs of Heart Disease

The criteria, aside from the electrocardiogram, accepted in this study as indicative of chronic cor pulmonale have already been described. In short, they are the presence of a pulmonary disease acceptable as an etiologic factor and evidence of congestive failure, or radiologic or postmortem evidence of hypertrophy of the right ventricle. Table 1 indicates the frequency with which the various electrocardiographic patterns were associated with other evidences of heart disease.

This table reveals that of the 11 patients whose electrocardiograms showed only peaked P waves and marked clockwise rotation, 10 (91 per cent) exhibited signs of cardiac failure. The degree of failure as judged by the response to therapy for both the pulmonary and heart disease, was considered mild to moderate, and in no case was it intractable.

Among the 18 cases in group II were 2 cases in which the diagnosis of cor pulmonale was based solely on this characteristic change in the presence of an acceptable etiologic factor. The other 16 all showed other evidences of heart disease, and in 14 (78 per cent) cardiac failure existed. This was mild to moderate in degree in 12 cases, but was intractable in the 2 others. On postmortem examination in 2 cases with mild cardiac failure clinically the right and left ventricles measured 8 and 13 mm. in one instance, and 7 and 12 mm. respectively, in the other. In a third case with no clinical evidence of heart disease the right ventricle was 5 mm. thick.

Among the 18 cases of incomplete right bundle-branch block were 2 in which the diagnosis of heart disease was based on the electrocardiogram and the presence of chronic pulmonary disease. There were 4 cases in which heart disease without cardiac failure was present, and 12 (67 per cent) with definite signs of cardiac failure. In 2 of these the tracing was consistent with the interpretation of co-existent right ventricular hypertrophy. The severity of the cardiac failure was mild to moderate in all 12 cases. Two postmortem examinations were performed on cases with cardiac failure in this group: in them the thickness of the right
ventricular wall was increased to 8 and 10 mm., respectively; both hearts were otherwise normal.

A pattern of complete right bundle-branch block was present in the tracings of 5 patients. Two of these showed no other evidence of heart disease, but in both electrocardiographic changes were also consistent with right ventricular hypertrophy. Three other cases showed evidence of heart disease, and in 2 (40 per cent) cardiac failure was present, moderately severe in one and intractable in the other. The electrocardiogram in the latter instance was also compatible with the presence of right ventricular hypertrophy.

In the 8 cases showing T-wave inversions in the right chest leads and clockwise rotation, 2 had no other evidence of heart disease. The remaining 6 (75 per cent) all were in cardiac failure that was mild to moderate in 5 and intractable in 1. This group of 6 included the 3 cases with inconstant T-wave abnormalities. On postmortem examination in 1 case with changing T waves the right ventricle measured 6 mm., the left 14; the mitral orifice was narrowed to 1 finger and the mitral ring was calcified.

All 7 cases in the group with miscellaneous types of electrocardiograms showed other evidence of heart disease, and all were in cardiac failure. In no case was any etiology other than the chronic pulmonary disease found to account for the cardiac disorder. It is interesting to speculate that the 2 cases with T-wave inversions in the left chest leads might represent examples of left ventricular hypertrophy resulting from abnormal collateral circulation between the bronchial and pulmonary circulations secondary to the pulmonary hypertension. Postmortem examination in 1 patient of this type did reveal thickening of the left ventricular wall to 21 mm., the right to 8 mm., and an increase in the weight of the heart to 650 Gm. The heart was otherwise normal.

The group of the 7 cases that came to autopsy is too small to permit statistically valid conclusions, but there was some correlation between the more significant electrocardiographic changes of right ventricular hypertrophy and its presence anatomically. Similar correlation was reported by Myers et al.\(^\text{18}\) in a much larger series of autopsy material.

**DISCUSSION**

To simplify the discussion of the findings in the 67 patients investigated, the groups with heart disease and cardiac failure, heart disease without failure, and no other evidence of heart disease, will be examined separately.

In all, there were 51 cases of cor pulmonale that showed varying degrees of heart failure. The electrocardiogram in 10 of these (20 per cent) showed only the clockwise rotation and peaking of the P waves that extensive experience with chronic pulmonary disease and emphysema has demonstrated to be a very common change, even in the absence of heart disease. Like many others, we are inclined to accept this type of tracing as indicative of change in electric axis, and in no way specific for cardiac disease. Nevertheless, these 10 patients had chronic cor pulmonale of sufficient severity to produce heart disease.

There was another group of 7 patients (14 per cent) in whom the electrocardiograms varied from normal to T-wave abnormalities in leads overlying the left ventricle only, but in no instance showing changes even suggestive of right ventricular damage. Each of these patients was in heart failure, attributable, on the basis of presently accepted criteria, to chronic cor pulmonale. The only postmortem examination performed in this group confirmed this; and in the remaining 6 cases clinical investigation failed to suggest any other etiology.

Therefore, in 17 instances (34 per cent) of pulmonary heart disease sufficiently advanced to result in cardiac failure, the electrocardiogram was of no diagnostic assistance. Conversely stated, the absence of electrocardiographic patterns currently assumed to be characteristic of right ventricular disease does not eliminate such disease even in an advanced stage. It should be emphasized at this point that the very method of selection of these patients required the presence of signs of heart disease other than the electrocardiogram. Therefore, the perfect correlation between these nondiagnostic electrocardiograms and other evidence of cardiac involvement resulted from the
method of selection, and should not be assumed to indicate any degree of specificity for the electrocardiographic patterns described.

Among the other patients in cardiac failure, there were 6 (12 per cent) whose tracings showed T-wave inversions over the right leads V1 through V3 or V4, accompanied by clockwise rotation. In this situation, the T-wave abnormalities cannot be accepted as a merely normal variation—the so-called juvenile pattern. In half of these patients, the T-wave abnormalities were inconstant, suggesting that they appeared only during periods of increased right ventricular “strain” secondary to a temporary change in the pulmonary disease. They would then not be indicative of chronic right ventricular damage. The 1 postmortem examination in this type of case, however, does not support this interpretation, since the right ventricle was hypertrophied. In the remaining 5 cases, the possibility of coronary artery disease producing the abnormal T waves cannot be excluded, but the usual supportive evidence for such a diagnosis was lacking in every instance. In this group of 6 patients, the electrocardiogram suggested the possibility of right-sided heart disease but could not be considered diagnostic, despite the fact that again the heart disease had advanced to the point of cardiac failure.

Although the pattern of right bundle-branch block may appear in otherwise normal individuals, its occurrence in the presence of chronic pulmonary disease is very strongly suggestive of cor pulmonale. Whether this change signifies hypertrophy, dilatation, or delayed conduction in the right ventricle will be discussed. Therefore, in the group of 14 patients (27.5 per cent) with cardiac failure, 12 with incomplete and 2 with complete right bundle-branch block, the electrocardiogram was consistent with a diagnosis of right-sided heart disease.

The pathognomonic pattern of right ventricular hypertrophy existed in an additional 14 patients (27.5 per cent) with signs of cardiac failure. The relative infrequency of this pattern again emphasizes that severe cor pulmonale often exists in the absence of classical electrocardiographic changes. However, combining the patients in this group with those showing complete and incomplete right bundle-branch block presents 55 per cent of the patients with chronic pulmonary disease and cardiac failure in whom the electrocardiogram showed right ventricular involvement.

The number of cases diagnosed as having chronic cor pulmonale without cardiac failure is considerably smaller than those with failure. This, in part, may be due to the recognized difficulty in making the clinical diagnosis in the early stages of right-sided heart disease. In the absence of postmortem examination and, for the purpose of the present investigation, excluding assistance from the electrocardiogram, the “early” diagnosis frequently depends on radiographic changes in heart size and configuration. Accentuation of the pulmonic second sound is indicative of pulmonary hypertension but this may be difficult to evaluate in patients with pulmonary emphysema in whom heart tones are often distant and muffled.

In each of the 8 cases with compensated heart disease the presence of right ventricular enlargement was confirmed by either x-ray or necropsy examination. Table 1 indicates that in 1 of these the electrocardiogram showed only the nondiagnostic peaking of P waves and clockwise rotation; in 5 the complete or incomplete right bundle-branch block consistent with right-sided heart disease in this type of patient; and in 2, the pathognomonic configuration of right ventricular hypertrophy (R in V1). Therefore, in this group, the electrocardiogram furnished acceptable evidence of right ventricular involvement in 87 per cent of the cases. Among them were 3 cases without clinical or x-ray evidence of heart disease during life, but with definite right ventricular hypertrophy on postmortem examination.

The final group of 8 patients (table 1) includes those in whom a diagnosis of heart disease was based solely on the electrocardiogram. In 2 of these, the tracing was diagnostic, and in the remaining 6 it was consistent with cor pulmonale. Originally, this group also included the 3 patients mentioned above who, on the basis of autopsy findings, were subsequently placed in the category of those with heart disease without failure. It is anticipated that continued observation of the remainder
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will eventually disclose other evidence of heart disease besides the electrocardiogram. Nevertheless, since at present these 8 cases show only electrocardiographic changes, they are not included in the final statistical tabulation in the conclusions.

Review of the 7 autopsied cases from among the total of 67 patients investigated showed the anatomic presence of right ventricular hypertrophy in each. The electrocardiogram had been diagnostic of right ventricular hypertrophy in 3 and consistent with the diagnosis in 2. It had been merely suggestive in another, and actually misleading in the last, where it had denoted left ventricular disease. Hypertrophy of both ventricles was found at post-mortem examination in this case.

Pathologic Physiology

The process that terminates in pulmonary hypertension and right-sided failure may begin in several ways: (1) with rupture of interalveolar septa, loss of pulmonary capillary bed, and decreased distensibility of the pulmonary vascular circuit—the usual picture in emphysema; (2) with gradual obliteration of smaller pulmonary vessels by fibrosis secondary to pulmonary granulomata (a broad group including diffuse tuberculosis, sarcoid, silicosis, and berylliosis); (3) with disease in the vessels themselves resulting from such factors as pulmonary hypertension and arteriolar hypertrophy secondary to mitral stenosis of congenital cardiac lesions, multiple pulmonary emboli, carcinomatosis of the lungs, and other less common conditions. Whichever process is primary, the inability of the lung to clear itself of secretions leads to secondary infection and the production of both fibrosis and emphysema. The end result is therefore a composite of at least the first 2 of the above factors. Arterial anoxia results from diffusion defect secondary to fibrosis, as well as from failure to aerate vascularized portions of the lung involved in secondary infection and bronchiolar obstruction. Anoxia has been shown to produce a reflex rise in pulmonary artery pressure, presumably due to pulmonary arteriolar constriction. Factors contributing to pulmonary hypertension therefore include actual destruction of pulmonary vessels, compression of vessels by fibrous tissue, reflex vasoconstriction of the pulmonary arterioles, increased blood viscosity in instances of secondary polycythemia, and increased cardiac output (resulting from hypervolemia, the effect of anoxia on the chemoreceptors, and the stretching of myocardial fibers). Cardiac catheterization in this phase reveals an elevated systolic but normal end-diastolic pressure in a right ventricle, which has not yet failed and still empties completely; an elevated mean pulmonary artery pressure; and a normal “wedge” pressure. In the next phase the right ventricle no longer empties completely; its filling pressure increases; blood is dammed back into the systemic veins and the clinical picture of right heart failure ensues, still (in anoxic, emphysematous patients) with a cardiac output above normal. Prior to the onset of failure, emphysematous patients increase cardiac output at the expense of a disproportionate rise in pulmonary artery pressure and the normal arteriovenous oxygen difference is maintained. It is not our purpose to present the experimental evidence that has been presented in other papers in support of this concept. Of the factors mentioned, arterial anoxia is the one most consistently found in association with pulmonary hypertension and pulmonary heart disease in cases of “emphysema heart”; it is of course not a factor at all in those patients with heart disease secondary to primary disease of the pulmonary vessels. The central position of anoxia has been emphasized by Cournand, Lewis and co-workers, Harvey et al. and Gelfand. Mack and Snider commented that “by the time arterial blood gas abnormalities become demonstrable (in chronic obstructive emphysema), chronic cor pulmonale may be assumed to be present.”

Patients in failure with cor pulmonale were found by Lewis’ group to share many of the characteristics of other forms of heart disease in failure, as well as some basic differences. Similarities included increased arteriovenous oxygen difference and depressed renal function (diminished renal plasma flow and glomerular filtration rate). Lewis thought that oxygen transport per unit time rather than blood flow per unit time is the determinant in fixing the
level of renal blood flow. Patients in failure with cor pulmonale differed from other cardiaces with respect to (1) pronounced arterial oxygen unsaturation, probably the most important factor precipitating this form of failure for the reasons noted above; (2) frequent secondary polycythemia; and (3) normal or increased resting cardiac output that, however, fails to increase normally with exercise as it also fails to do in other forms of decompensated heart disease.

We have had 2 patients with cor pulmonale in whom there was left ventricular disease without the presence of any other form of heart disease ordinarily leading to increased work of the left ventricle. In one of the cases the findings were confirmed post mortem. Roosenburg and Deenstra\(^1\) have demonstrated the admixture of oxygenated blood in the smaller pulmonary arteries in patients with chronic lung disease, particularly those with bronchiectasis, indicating an extensive collateral circulation from the bronchial arteries. From the standpoint of the left ventricle this mixing represents an arteriovenous shunt and may be responsible for the left ventricular disease.

Pathophysiology of the Electrocardiographic Pattern

The interpretation of the electrocardiogram in emphysematous patients is subject to error caused by the position of the heart, which is in extreme clockwise rotation. Even \(V_{6}\) may overlie the right ventricle and it is necessary to take additional leads to the left to obtain the patterns of both ventricles.\(^1\) A QS pattern may appear in \(aV_{R}\) in the absence of myocardial infarction because with clockwise rotation the current of septal activation may be at right angles to \(aV_{R}\), and what would otherwise be an rS (in a horizontal heart) becomes a QS.

With minor points of difference, Wilson, Goldberger, Myers, and others have accepted the following criteria for right ventricular hypertrophy:

1. In the absence of excessive clockwise rotation, leads \(V_{3R}\) and \(V_{1}\) overlie the right ventricle as in the normal heart. With a hypertrophied right ventricular myocardium, the vector toward these leads is increased and is not dominated, as in the normal heart, by the greater muscle mass of the left ventricle. \(V_{3R}\) therefore shows an rs instead of the normal rS. \(V_{1}\) may also show an Rs. However, if \(V_{1}\) lies close to the septum, the later phase of ventricular activation in both ventricles is away from this electrode and an rS may appear despite the presence of a hypertrophied right ventricle. It is therefore important to obtain a \(V_{3R}\) or \(V_{4R}\) in every case of suspected right ventricular hypertrophy where the diagnosis cannot be made on a standard 12 lead electrocardiogram.\(^1\)

The tall R (without preceding q) in the right chest leads, often with delayed intrinsicoid deflection, is the only direct evidence of right ventricular hypertrophy. Other evidence has been accepted as conclusive, though indirect, and depends on the assumption that extreme clockwise rotation (in the presence of a lesion competent to produce right heart hypertrophy) may be accepted as evidence of right ventricular hypertrophy.\(^1\)

2. The following evidence of extreme clockwise rotation has been accepted as indirect evidence of right heart hypertrophy under the conditions noted above: (a) A qR pattern in \(V_{1}\), or in \(V_{1}\) and \(V_{2}\), is usually considered to indicate that the heart has so rotated on its own long axis as to bring a portion of the left ventricle beneath these electrodes. The qR is therefore the normal recording of the posterior surface of the left ventricle, appearing in an abnormal position. (b) A qR in \(aV_{R}\) where the R is at least 3 times the magnitude of the q, in addition to a deep S wave in all precordial leads such that R/S in \(V_{6}\) is less than \(\frac{1}{2}\).

3. Difficulty is encountered in the diagnosis of right heart hypertrophy in the presence of right bundle-branch block. This may be manifested by: (a) An rSR\(^2\) pattern in the right precordial leads with a QRS duration of 0.08-0.11 second (experimental evidence to be recorded below indicates that this pattern does not invariably signify incomplete right bundle-branch block). (b) A tall R in the right precordial leads with QRS 0.12 second or more. Goldberger considered the diagnosis of right ventricular hypertrophy not to be possible in the presence of right bundle-branch block.\(^1\) Braunwald's group accepts the diagnosis of
right ventricular hypertrophy on the basis of the following criteria established by Barker and Valencia: 14.19.20 (a) If the R' is greater than 10 mm. in the presence of incomplete block. (b) If the R' is greater than 15 mm. in the presence of complete block. Braunwald selected 3 groups of patients and measured the time interval between the onset of ventricular activation as indicated by the onset of the QRS on the electrocardiogram and the onset of ventricular contraction as indicated by the first rise in pressure in the catheterized right ventricle. 1. In 15 patients with right heart hypertrophy clinically and electrocardiographically but with no electrocardiographic evidence of conduction defect, this study confirmed the absence of conduction defect. 2. In 6 patients with the electrocardiographic pattern of right bundle-branch block but with no other evidence of heart disease, this study confirmed the presence of right bundle-branch block. 3. In 15 patients similar to the patients in our study, in whom there was clinical reason to suspect right heart hypertrophy and in whom catheterization demonstrated pulmonary hypertension, but in whom the electrocardiogram showed bundle-branch block, two thirds were found to have normal conduction intervals. It would therefore appear that the rSR’ and prolonged QRS in these patients was a manifestation of right heart hypertrophy rather than of bundle-branch block. Vectorcardiograms, on the other hand, correctly differentiated the groups and corresponded to the results obtained on catheterization. 15

Other manifestations that may appear in the electrocardiogram in patients with emphysema heart disease have not been accepted as criteria for right ventricular hypertrophy. Peaked P waves may indicate atrial hypertrophy or simply change in the position of the heart. 1.16 Inverted T waves in leads subtending the right ventricle indicate “strain” rather than hypertrophy and are often reversible. Finally, superimposition of an acute pneumonic process on a restricted pulmonary bed may produce the electrocardiographic picture of acute cor pulmonale even when embolization has not occurred.

Various groups have found correlation between pulmonary artery hypertension and the electrocardiographic picture of right ventricular hypertrophy. 11.17 In general electrocardiographic changes were not found in the presence of a resting mean pulmonary artery pressure under 30 mm. Hg. In all groups studied a significant number of patients had a pattern generally attributed to complete or incomplete right bundle-branch block rather than hypertrophy. Correlation was also found by Scott et al. 17 between hypoxia and abnormality of the electrocardiographic pattern.

Postmortem studies by Myers and co-workers 18 again emphasized the importance of the pattern of bundle-branch block in these patients. In 40 patients proved post mortem to have right ventricular hypertrophy, 21 had met the classical criteria quoted above for the diagnosis of right ventricular hypertrophy; 12 others met only the electrocardiographic criteria for complete or incomplete right bundle-branch block; 7 met neither of these criteria.

One significant fact emerges from a comparison of our data (table 1) with the data of observers quoted above: In various series, only 50 per cent or fewer of the patients fulfilled the absolute electrocardiographic criteria for right ventricular hypertrophy. The diagnosis of pulmonary heart disease may be made more frequently if the criteria are expanded to include patients with right bundle-branch block (complete or incomplete) and significant T-wave abnormalities over the right chest, in the presence of advanced chronic pulmonary disease. The occasional patient with a combination of chronic pulmonary disease and some other unrelated type of heart disease, may be misdiagnosed on this basis, but the over-all accuracy of the diagnosis in a large series of patients will be greatly improved.

**Summary and Conclusions**

Sixty-seven patients with pulmonary heart disease were investigated in this study. Of these, 59 were found to be suitable for statistical analysis from which the following conclusions are drawn:

In about 30 per cent of the cases of pulmonary heart disease the electrocardiogram alone is not diagnostic; in about 27 per cent the elec-
trocardiogram alone is pathognomonic of right ventricular hypertrophy; in about 42 per cent it is only suggestive, but in the presence of an appropriate pulmonary disease, it becomes strongly suggestive. Clinical pulmonary heart disease of even advance I degree may be present with no more than mild nonspecific electrocardiographic changes and, conversely, pathognomonic electrocardiographic changes may appear in the presence of only mild clinical pulmonary heart disease.

On the basis of postmortem observations in 7 patients, the degree of hypertrophy of the right ventricle correlates well with the electrocardiographic patterns of right ventricular hypertrophy and incomplete right bundle-branch block. But there is no definite correlation between the thickness of the right ventricle and the clinical severity of the pulmonary heart disease.

If complete or incomplete right bundle-branch block, and significant T-wave abnormalities over the right chest leads are considered diagnostic in the presence of an acceptable etiologic factor, then the electrocardiogram might be considered fairly diagnostic in about 69 per cent of cases of pulmonary heart disease. If currently acceptable electrocardiographic criteria are to be chiefly relied upon for the diagnosis of pulmonary heart disease, even in the presence of an acceptable etiologic factor, this diagnosis may be missed in about 31 per cent of cases. For the satisfactory diagnosis of pulmonary heart disease, a combination of clinical, radiologic, and electrocardiographic evidence is necessary in the majority of cases.

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Summario in Interlingua

Esseva investigate in le presente studio 67 patientes con morbo cardiac pulmonar. Cinquantanove de illes se prestava al analyse statistic, resultante in le sequente conclusiones:

In circa 30 pro cento de casos de morbo cardiac pulmonar, le electrocardiogramma per se non es diagnostic. In circa 27 pro cento, le electrocardiogramma per se es pathognomonic pro hypertrophia dextero-ventricular. In circa 42 pro cento, le electrocardiogramma pote solmente suggerer le diagnose, sed in le presentia de un pertinente morbo pulmonar, su valor suggestive deveni multo forte. Clinic morbo cardiac pulmonar—mesmo de grades avantiate—pote esser presente in casos exhibente solmente leve e nonspecific alterationes electrocardiographic, e inversemente, pathognomonic alterationes electrocardiographic pote esser manifeste in le presentia de solmente leve grados de clinie morbo cardiac pulmonar.

A judicar per observationes post morte in 7 patientes, le grado de hypertrophia del ventriculo dextere es ben correlate con le configuration electrocardiographic typic pro hypertrophia dextero-ventricular e incomplete bloco de branca dextere. Sel il ha nulle correlation del spissitate del ventriculo dextere con le severitate clinie del morbo cardiac pulmonar.

Si, in le presentia de un aceptabile factor etiologic, complete o incomplete bloco de branca dextere e significative anormalitates de unda T in le derivaciones dextero-thoracic pote esser considerate como diagnostic, alora le electrocardiogramma es satis definite pro le establimento del diagnose in 69 pro cento del casos de morbo cardiac pulmonar. Si le currentemente acceptate criterios electrocardiographic es usate como base principal pro le diagnose de morbo cardiac pulmonar, alora—mesmo in le presentia de un aceptabile factor etiologic—iste diagnose escape al detection in circa 31 pro cento del casos. Pro le satisfacent diagnose de morbo cardiac pulmonar, un combination de indices clinie, radiologic, e electrocardiographic es requirite in le majoritate del casos.

REFERENCES


Twenty-four dogs were subjected to systemic hypothermia. Nine dogs were autopsied at the onset of fatal cardiac irregularities or at the termination of moderate (26 to 27.5 C.) or deep (21 to 22.5 C.) hypothermia of 1 to 4 hours' duration. In all cases the myocardium showed foci of necrotic muscle fibers with an occasional cellular reaction. Fifteen dogs were sacrificed and autopsied 3 days to 3 years after survival of moderate or deep hypothermia of the same duration. In 13 of 15 cases distinct areas of necrosis showing various stages of organization were detected.

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