

THE ELECTROCARDIOGRAM IN CHRONIC BRONCHITIS AND EMPHYSEMA*

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There have been a considerable number of reports on the subject of the electrocardiogram in chronic lung disease. Generally, the authors have included a variety of lung diseases, including bronchiectasis and pneumoconiosis, though the majority of cases have been diagnosed as chronic bronchitis and emphysema. Some articles included various lung function tests, but these usually provided evidence of airway obstruction and we could find no paper in which the degree of hyperinflation (as determined by the measurement of lung volumes) was taken into account. The purpose of this communication was to study the electrocardiograms of patients suffering from chronic obstructive airways disease and to relate the electrocardiogram to the degree of obstruction and of hyperinflation of the lung. A review of relevant findings in the literature is included.

MATERIAL AND METHODS

Fifty cases were selected from the files of the pulmonary function laboratory. They all had detailed clinical data, chest radiographs, a 12- or 13-lead scalar electrocardiogram, measurement of the forced expiratory volume in 1

second (FEV_1) and the forced vital capacity (FVC), and in 34 of these there were also measurements of the subdivisions of lung volume. All were White subjects over the age of 30 years. Most of the cases were outpatients, but some were convalescing in the wards. Patients were excluded who had a definite history of paroxysmal attacks of dyspnoea which could be due to asthma, if there was any evidence of ischaemic heart disease, pneumoconiosis, rheumatic heart disease or hypertension, or if any other major disease was present. The patients, therefore, were a selected group with chronic obstructive airways disease due to bronchitis and emphysema.

The forced vital capacity was performed on a Godart Pulmotest and a Collins spirometer was used for the closed-circuit helium dilution method for the measurement of lung volumes. The 50 cases in the series with measurements of $FEV_1/FVC\%$ and 34 with measurements of residual volume (RV) were subdivided into the categories mild, moderate and severe obstruction and hyperinflation as indicated in Table I. The predicted value for residual volume is based on normal subjects studied in this laboratory.¹

The electrocardiograms were analysed for the following

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TABLE I. CATEGORIES OF PATIENTS WITH OBSTRUCTION AND HYPERINFLATION

	Obstruction		Hyperinflation	
	FEV ₁ /FVC%	No. of cases	RV % pred.	No. of cases
Mild	>60	8	120-149	10
Moderate	51-60	12	150-179	9
Severe	≤50	30	≥180	15
Total		50		34

data: rhythm; P wave—mean frontal plane axis, shape, height, duration and PR interval; QRS complex—mean frontal plane axis, duration, evidence of right ventricular hypertrophy, incomplete or complete right bundle-branch block, and the degree of clockwise rotation. The position of T wave inversion in the chest leads was noted. The ECG findings for P wave axis and shape and QRS axis were subdivided, using the criteria of Spodick *et al.*² Single-peaked P waves were termed P pulmonale (amplitude >0.24 mV) or Gothic P (amplitude <0.24 mV).³ P wave axis was divided into 3 groups: (a) less than +40°, (b) 40°-60°, and (c) 70°-90° (Figs. 1 and 2). QRS axis was also divided into 3 groups: (a) -10° to -60°, (b) 0°-60°, and (c) indeterminate or positive axes of 70° or more, or negative axes of -70° or less (Figs. 5 and 6).

RESULTS

Four cases with arrhythmias were present in the series: 1 with atrial and ventricular ectopic beats, 1 with nodal rhythm and 2 with atrial fibrillation. The case with ectopic beats had moderate obstruction and hyperinflation. The case with nodal rhythm had mild obstruction and mild hyperinflation, but both cases with atrial fibrillation had severe obstruction and 1 of them had severe hyperinflation as well. There was 1 case of complete right bundle-branch block, the patient having severe obstruction and mild hyperinflation.

The P Wave

P wave axis. In Fig. 1 it can be seen that with increasing severity of airways obstruction, more cases show 'verticalization of the mean frontal plane P wave axis', i.e. an axis of 70° or more.⁴

The effect of hyperinflation on the P wave axis (Fig. 2) appears to be less marked. In fact, most of the cases with the vertical P wave axis and severe hyperinflation had severe airways obstruction also, whereas 3 cases with severe hyperinflation and a normal P wave axis had only mild obstruction.

P wave shape. In Fig. 3, P wave shape (normal, Gothic or P pulmonale) is related to the degree of airways obstruction. The peaking of the P wave becomes frequent (32 out of 40 cases) when the obstruction is moderate or severe, though a normal shape is retained in one-fifth of such cases.

When P wave shape is related to the degree of hyperinflation of the lungs there is no obvious trend for increased peaking to be present with increasing hyperinflation (Fig. 4). All 3 cases with mild obstruction and severe hyperinflation had normal P waves, while 3 of the 4 cases with severe obstruction and mild hyperinflation had peaked P waves.

P WAVE AXIS

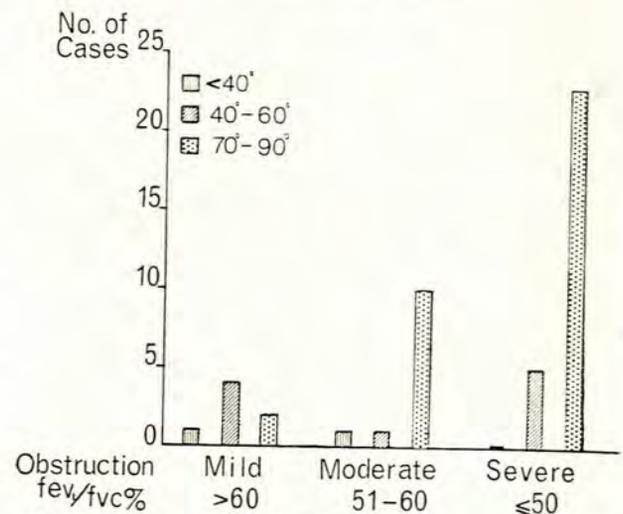


Fig. 1. Mean frontal plane P wave axis moves to the right with increasing severity of airways obstruction (see text).

P WAVE AXIS

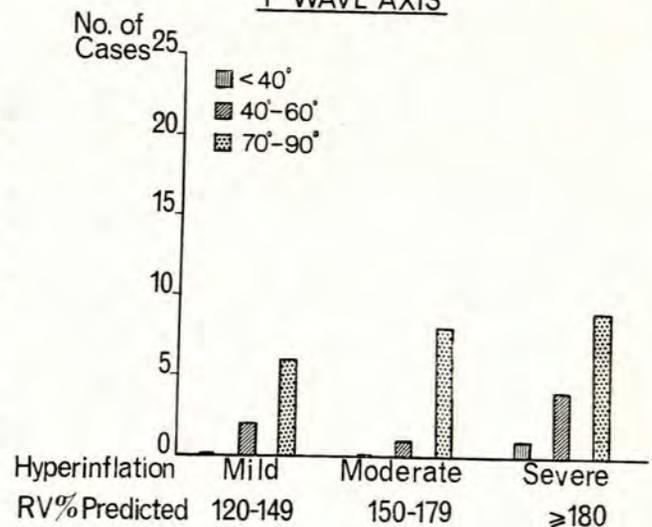


Fig. 2. Mean frontal plane P wave axis is not clearly related to the degree of hyperinflation of the lung (see text).

The QRS Complex

The QRS axis. The mean frontal plane QRS axis tends to become more vertical with increasing degrees of airway obstruction (Fig. 5). We have followed Spodick *et al.*² in grouping cases with a QRS axis of +70° or more to the right and -70° or further to the left together with those of indeterminate axis. These are all regarded as having a 'verticalization' of the QRS complex. Twenty-four of the 29 cases with severe obstruction fell into this latter group (1 case had right bundle-branch block).

The influence of hyperinflation again appears to be less than that of obstruction in affecting the QRS axis (Fig. 6). Two out of 3 cases with mild obstruction and

P WAVE SHAPE

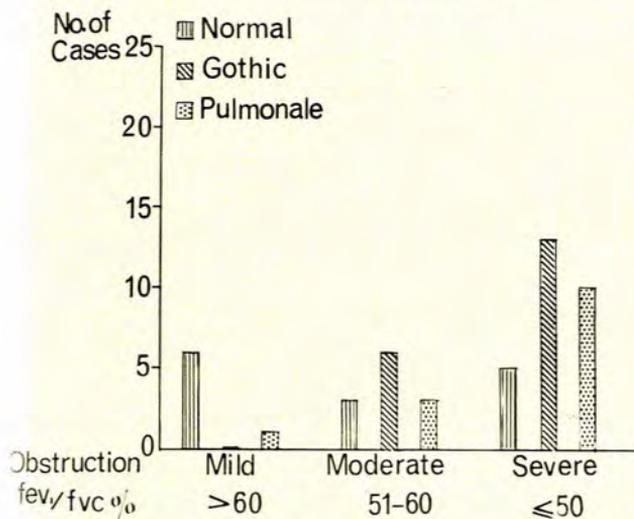


Fig. 3. Peaking of the P wave becomes frequent when airways obstruction is moderate or severe (see text).

QRS AXIS

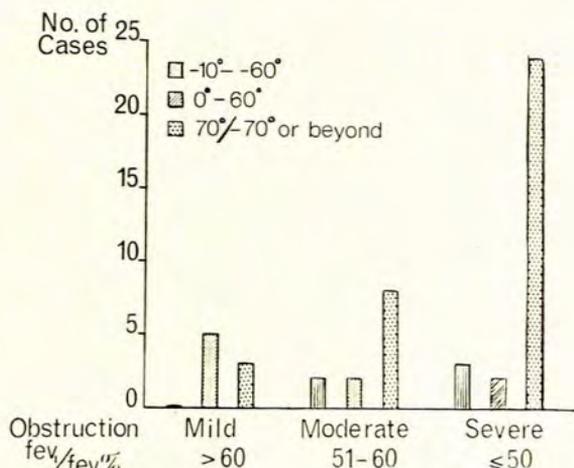


Fig. 5. Mean frontal plane QRS axis becomes more vertical with increasing severity of airways obstruction (see text).

P WAVE SHAPE

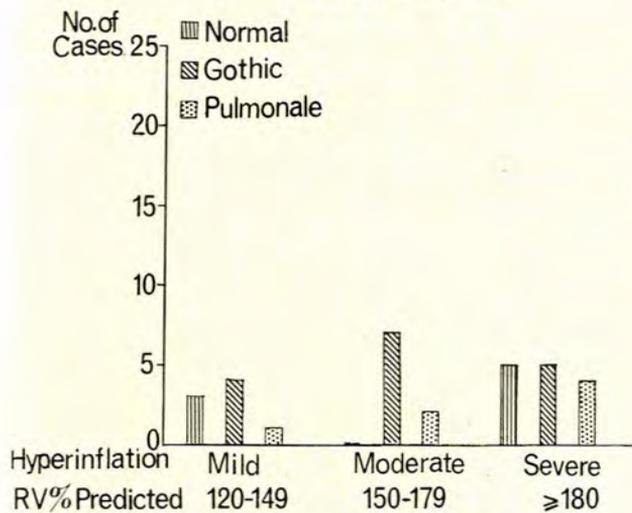


Fig. 4. Peaking of the P wave is not clearly related to the degree of hyperinflation of the lung (see text).

QRS AXIS

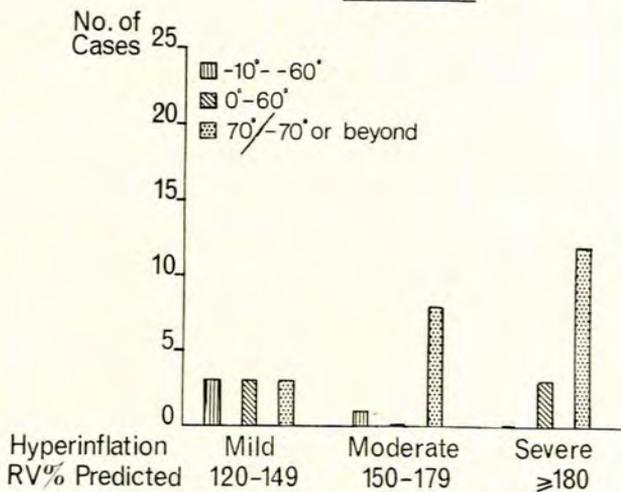


Fig. 6. Mean frontal plane QRS axis becomes more vertical with increasing degree of hyperinflation (see text).

severe hyperinflation had a normal QRS axis. Of 4 cases with severe obstruction and mild hyperinflation, 2 showed verticalization and 2 left axis deviation of the QRS.

Clockwise rotation. A dominant S wave was noted in V5 or further to the left in the chest leads, with increasing frequency as obstruction became more severe (Fig. 7). This trend is less distinct when clockwise rotation is related to degree of hyperinflation (Fig. 8). Clockwise rotation did not occur in any of the 3 cases with severe hyperinflation and mild obstruction, but was present in 2 of the 4 cases with severe obstruction and mild hyperinflation.

There were 4 cases with left axis deviation. Two of these had moderate obstruction, 1 with mild and 1 with

moderate hyperinflation. The other 2 cases both had severe obstruction and mild hyperinflation. Abnormalities of P wave duration, PR interval, QRS duration and T waves were too infrequent to be associated meaningfully with obstruction or hyperinflation. There was only 1 case with dominant R waves in V1 and V4R to suggest right ventricular hypertrophy.

DISCUSSION

Our results suggest that in chronic obstructive airways disease airway obstruction exerts more influence than hyperinflation on the P wave and shape, on the QRS axis and on the extent of clockwise rotation.

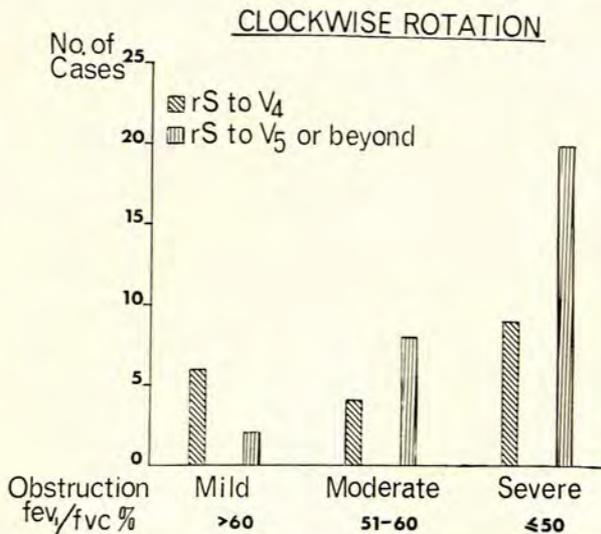


Fig. 7. Dominant S wave occurs more frequently in left chest leads with increasing severity of airways obstruction (see text).

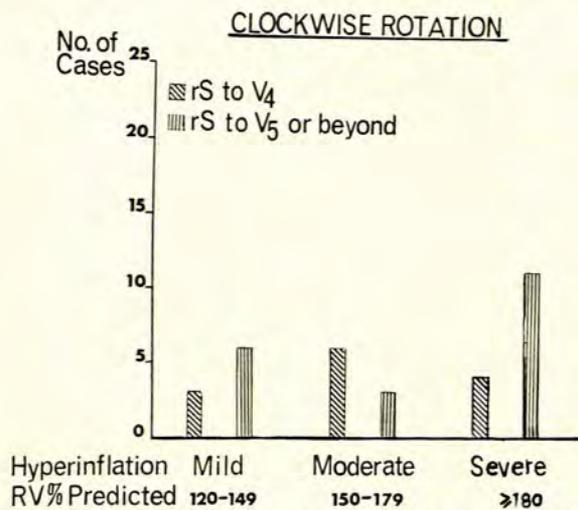


Fig. 8. Dominant S wave in left chest leads is not obviously related to degree of hyperinflation (see text).

Chronic lung disease has been found to have the following effects on the electrocardiogram:^{5,6} P pulmonale; prominent Ta waves; rightward deviation of the P wave axis; rightward deviation of the mean frontal QRS axis; low voltage QRS complexes; negative, W-shaped or transitional (QS, rSR, QRS, RS) complexes in precordial leads; rSR' in V₁; incomplete or complete right bundle-branch block; classical right ventricular hypertrophy with dominant R in V₁ or V_{4R} with or without T wave inversion in right precordial leads; abnormal left axis deviation; and the S₁, S₂, S₃ syndrome.

A number of factors have been considered to explain these ECG changes and include dilatation and/or hypertrophy of right atrium and right ventricle, as well as the crista supraventricularis; hyperinflation of the lung which alters electrical conductivity and the position of the heart

and factors such as hypoxia, hypercapnia, respiratory acidosis and pulmonary arterial hypertension.

Spodick *et al.* in a series of studies showed that, in patients with chronic lung disease (mainly bronchitis and emphysema), verticalization of the P wave axis is frequent. Of 301 patients with emphysema, 77% had such verticalization,² which was well correlated with increasing degrees of airways obstruction assessed by the 2-second fraction of the timed vital capacity. Spodick also showed that P loop vectors were vertical in most of 25 cases with chronic bronchitis and emphysema.⁷ Chappell analysed 112 patients with emphysema (assessed radiologically) and chronic bronchitis.⁸ The 24 emphysematous subjects all had severe obstruction (assessed by peak expiratory flow-rate and forced vital capacity), while the 88 bronchitics were subdivided into those with severe obstruction and those with a lesser degree of obstruction. Severe obstruction, whether occurring with emphysema or bronchitis, was associated with P axis verticalization, right ventricular hypertrophy, left axis deviation and P pulmonale; cases with little obstruction showed virtually normal electrocardiograms. Calatayud *et al.*⁹ reported studies of the P wave in 173 patients with chronic obstructive pulmonary disease and demonstrated a good correlation between P wave verticalization and amplitude and increasing obstruction. They also showed that as the P wave axis shifted rightward, the atrial T wave (Ta) amplitude increased and the highest Ta values occurred in patients with most obstruction.⁹

Spodick⁷ showed that the shape of the P wave, as represented by a P pulmonale, correlated well with the increasing degree of obstruction, and that the P loop magnitude was much larger than in normal subjects. Patients with normal P waves in scalar tracings had normal P loop magnitude, those with Gothic P waves were intermediate in size and those with pulmonale P waves had the largest P loop amplitude. He suggested that, despite their normal scalar dimensions, Gothic P waves appeared to be intermediate between normal and pulmonale P waves and might indicate right atrial 'strain' or enlargement.

Spodick *et al.* also concluded that increasing QRS verticalization corresponded well with increasing airways obstruction,² while Millard showed that right axis deviation was good evidence of right ventricular hypertrophy provided left ventricular hypertrophy and myocardial ischaemia were excluded.¹⁰

We found left axis deviation in 4 cases. Chappell's series⁸ contained 8 cases, 5 of these having widespread emphysema, and 3 of these 5 had no other cause for this axis deviation. The mechanisms which may be related to this finding have been described as altered position of the heart; right ventricular hypertrophy especially involving the crista supraventricularis so that the terminal vector points superiorly and may even dominate the electrocardiographic picture; poor electrical conduction by emphysematous lung with verticalization of the electric field and the axis illusion phenomenon;⁴ and associated left ventricular hypertrophy or ischaemic heart disease with myocardial fibrosis. In a series of 535 cases with left axis deviation, at necropsy pulmonary emphysema was thought to be the only cause in 12 instances.¹¹

It has been pointed out that overt ECG changes of

Right ventricular hypertrophy are not common in chronic obstructive pulmonary disease,¹² an observation supported by our single case. Millard¹⁰ studied the ECG in 46 patients with chronic lung disease (of whom 12 showed radiographic evidence of widespread emphysema) and correlated this with right ventricular hypertrophy assessed by dissection and weighing. He concluded that in chronic lung disease, right axis deviation was the most reliable sign of right ventricular hypertrophy, but not in the presence of left ventricular hypertrophy or myocardial ischaemia. He concluded also that, provided that myocardial ischaemia is excluded, an R wave over the right ventricle greater than 6 mm indicated right ventricular hypertrophy while a left precordial S wave greater than 10 mm was also very suggestive, provided emphysema was excluded. A P wave of 2.5 mm or more indicated right ventricular hypertrophy but its absence did not exclude it. Chappell⁶ found ECG evidence of right ventricular hypertrophy in 9% of his series of 112 outpatients with chronic bronchitis (the incidence being no different in those with or without associated radiographic emphysema), the determining factor being the presence of severe airways obstruction. The mechanism by which airways obstruction affects the ECG has not yet been elucidated.

Persistent arrhythmias are uncommon findings in chronic obstructive airways disease and atrial arrhythmias suggest a superimposed acute respiratory complication.¹² Chappell found that almost 10% of his cases had ventricular ectopic beats but only 2 other arrhythmias occurred, both transiently during acute exacerbations of bronchitis. Thomas and Valabhji reported an incidence of important disorders of rhythm and rate in 7% of 1 482 hospital admissions for chronic pulmonary disease.¹³

Finally, it should be noted that although the electrocardiogram may be completely normal in patients with chronic obstructive airways disease,^{2,6,12} the ECG has been used to identify working men with ventilatory defects or with an increased risk of developing ventilatory defect.¹⁴

SUMMARY

In a carefully selected series of 50 White adults with chronic bronchitis and emphysema, it has been confirmed that P wave axis and shape, as well as QRS axis and degree of clockwise rotation, are related to the degree of airway obstruction, assessed by FEV₁/FVC%. The degree of hyperinflation assessed by RV% of the predicted value appears to be of much less importance in influencing the electrocardiogram. Arrhythmias and right ventricular hypertrophy are uncommon, occurring in less than 10% of this series.

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REFERENCES

1. Goldman, H. I. and Becklake, M. R. (1959): *Amer. Rev. Tuberc.*, **79**, 457.
2. Spodick, D. H., Haugher-Klevene, J. H., Tyler, J. M., Muench, H. and Dorr, C. A. (1963): *Amer. Rev. Resp. Dis.*, **88**, 14.
3. Zuckerman, R., Cabrera, C. E., Fishleder, B. L. and Sodi-Pallares, D. (1948): *Amer. Heart J.*, **35**, 421.
4. Spodick, D. H. (1959): *Circulation*, **20**, 1067.
5. Scott, R. C. (1961): *Amer. Heart J.*, **61**, 843.
6. Chappell, A. G. (1966): *Brit. Heart J.*, **28**, 517.
7. Spodick, D. H. (1968): *Amer. Rev. Resp. Dis.*, **98**, 634.
8. Calatayud, J. B., Abad, J. M., Ba Khoi, N. and Silver, H. M. (1968): *Circulation*, **38**, suppl. 6, 49.
9. *Idem* (1969): *Dis. Chest*, **56**, 5.
10. Millard, F. J. C. (1967): *Brit. Heart J.*, **29**, 43.
11. Bahl, O. P., Walsh, T. J. and Massie, E. (1969): *Ibid.*, **31**, 451.
12. Rodman, T. and Sterling, F. H. (1969): *Pulmonary Emphysema and Related Lung Diseases*, p. 124. St Louis: C. V. Mosby.
13. Thomas, A. J. and Va'abhji, P. (1969): *Brit. Heart J.*, **31**, 491.
14. Prineas, R. J., Tibblin, G. and Rose, G. (1968): *Ibid.*, **30**, 859.