RIGHT VENTRICULAR HYPERTROPHY IN THE PNEUMOCONIOSIS OF COALMINERS

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Right ventricular hypertrophy has been recorded in the necropsy records of proven cases of coalminers' pneumoconiosis (Coggins *et al.*, 1938; Scott and Garvin, 1941; Gooding, 1946); but its frequency has not been well defined, partly because the degree of hypertrophy is not easy to establish in any particular case. The small size of the heart, clinically and radiologically, and the frequent absence in advanced cases of the classical cardiographic pattern of right ventricular hypertrophy have often caused comment (Thomas, 1948). This investigation, attempting to clarify some of these points, is presented in two parts, a pathological study of the degree of right ventricular hypertrophy present in pneumoconiosis, and a clinical study of the variation from normal of the cardiographic patterns at various stages of the disease. In some instances it has been possible to correlate the findings because the cases have appeared in both parts of the investigation.

RIGHT VENTRICULAR HYPERTROPHY AT NECROPSY

The hearts of fifty unselected cases of pneumoconiosis have been studied post mortem and the ventricular weights ascertained. These hearts had already been examined by a pathologist and had then been placed in formalin. The method of ventricular separation used was that described as Method B by Hermann and Wilson (1922). The auricles were removed, the ventricles were divided into sections at right angles to their long axis and then separated by dissection through the septum, and the complete right and left ventricles weighed. The normal range of the ratio of left ventricle to right ventricle, LV/RV ratio, by this method is 1.46 to 2.14 (Hermann and Wilson, 1922) and by the slightly different method of Lewis 1.57 to 2.18 (Lewis, 1914). For this study ratios below 1.40 and above 2.20 are considered to be abnormal.

The LV/RV ratios for fifty unselected cases of pneumoconiosis range from 0.61 to 2.66 with a mean of 1.16. The marked shift down the scale of this ratio is shown in Fig. 1; thirty-eight hearts have LV/RV ratios that are below the lowest limit of normal. This is a significant variation from the normal and indicates the high degree of right ventricular hypertrophy.

The mean heart weight post mortem was 361 g. with a range of 180 g. to 800 g. The ventricular weights show that the right preponderance is present even in the smallest hearts, e.g. LV/RV, 58 g./88 g., and many of the hearts in this series are small. The mean body weight of the patients was 48.1 kg. (range 29 to 71 kg.) and their mean age at death 58.4 years (range 39 to 71 years). It is possible to relate ventricular weight to body weight both in grams; the controls in the Hermann and Wilson series ranged from 0.0017 to 0.0039 and they considered ratios above 0.0050 indicative of ventricular hypertrophy. In the present series the ratio is available in 29 instances; the mean ratio is 0.0051 with a range from 0.0033 to 0.0078, and 16 have a ratio above 0.0050.

In many of the cases the right ventricular hypertrophy was visible before dissection and the right ventricle was clearly bigger than the left in some of them (Fig. 2). In others the relative

FIG. 1.—The ratio of the weights of left ventricle to right ventricle in fifty unselected cases of pneumoconiosis of coalminers.

increase in weight of the right ventricle became evident only after dissection, and often the LV/RV ratio was lower than the appearance of the whole heart suggested. The hypertrophy is first evident in the thickening of the muscle around the base of the outflow tract, and particularly the supraventricular crest. There is accentuation of all the normal features of the right ventricle so that the trabeculæ carneæ and the papillary muscles are thickened (Fig. 2 and 3). The left ventricle appears relatively small and the cavity of the right ventricle extends further into the apex of the heart than is normal.

Analysing the lung lesions and the causes of death, 30 cases had massive fibrotic dust lesions; 20 of these died of pure pulmonary heart failure, the mean LV/RV ratio of this group being 0.90 as against the mean of the series 1.16; only one had a ratio above the lower limit of normal (1.48: 1.40). Three others died of heart failure due to pneumoconiosis plus atheroma, tuberculosis, and emphysema respectively; two without heart failure died of pneumoconiosis and tuberculosis, and the remaining five with massive lesions had other unrelated diseases.

Apart from the 30 dealt with above, there were 6 deaths from heart failure without massive lesions; three of these had massive emphysema with some dust foci and an LV/RV ratio below the lower limit of normal; the other three had hypertensive heart disease, aortic stenosis, and syphilitic aortitis respectively with LV/RV ratios of 1.82, 2.57, 2.66 in that order.

FIG. 2.—The powerful right ventricle is noted on inspection and the structures forming the outflow tract are thickened and prominent. The left ventricle appears relatively small. Case No. 167, aged 39 years, body weight 64 kg. Left ventricle 140 g., right ventricle 190 g. $LV/RV=0.74$.

Right ventricular hypertrophy and death from right heart failure are associated with massive fibrotic lesions in 21 cases, but with massive emphysema in the presence of focal dust lesions in only 3 cases. Furthermore, there are cases with massive fibrotic lesions dying of other diseases before heart failure appears, which show a progressively increasing degree of right ventricular hypertrophy.

There are 18 necropsies of cases that had been subjected to complete clinical and cardiographic examinations, but not all were seen in the final phase. Three deaths resulted from coronary artery disease: this had been postulated a year earlier in one patient aged 47 years; in the second there was proven cardiac infarction present some weeks before death in heart failure supervened; and in the third, followed clinically for a long period with recurring cardiac failure and cardiographic evidence of considerable right ventricular hypertrophy, the terminal event was a cardiac infarction but the gross right ventricular hypertrophy was mainly due to the massive pulmonary lesions. One

FIG. 3.—The left and right ventricles are shown after dissection to illustrate the change in size and structure of the right ventricle with advanced complicated pneumoconiosis. Left ventricle 77 g. Right ventricle 98 g. The chest lead pattern recorded a few weeks before death is shown in Fig. 6A. Case No. 339, aged 48 years.

patient had mitral stenosis and severe pulmonary disease: another presented for a long period with advanced pulmonary disease, moderate hypertension, persistent cardiac failure, and a cardiographic pattern of left bundle branch block; post mortem the heart weighed 520 g. and both ventricles were dilated.

The remaining 13 deaths were due to the pulmonary disease. Eleven died in right heart failure due to pneumoconiosis and had electrocardiographic evidence of right ventricular hypertrophy for many months before death; four of these hearts, which were weighed, gave ratios below 1·00, viz. 0·61, 0·71, 0·79, and 0·88. The other two died of emphysema and pneumoconiosis without heart failure, and the LV/RV ratios were 1·00 and 1·02: the cardiographic patterns of these two, recorded at intervals during the last two years, failed to show any right ventricular hypertrophy and radiologically the heart of one was very small, the right and left ventricles each weighing 91 g. (Fig. 4). These two show that severe pneumoconiosis and emphysema may terminate before right heart failure develops and before right hypertrophy is sufficient to appear on the chest lead pattern.

FIG. 4.—The open right ventricle is shown. Left ventricle 91 g. Right ventricle 91 g. LV/RV=1.00. This patient had complicated pneumoconiosis and massive emphysema. Case No. 348, aged 42 years.

It is suggested that the LV/RV ratio has to drop below 1.00 before the pattern of right hypertrophy can be expected.

The earlier hypertrophy is found in the walls of the outflow tract and a chest lead from the medial end of the third left interspace anteriorly has been used in an effort to detect this hypertrophy at an earlier stage. This lead is shown as V2 (3 ics.) in Fig. 5; it is abnormal in the first, normal in the second. An r' wave higher than 4 mm. is regarded as abnormal and indicative of hypertrophy of the right ventricular outflow tract. Harris (1941) has shown that this region is one of the last to be activated and the late appearance of the r' conforms with this fact, yet the delay, 0.07 sec., is not that of a conduction defect. The height of the r' has been found not to exceed 4 mm. in the normal, as shown later. The presence of the abnormal r' wave in V2 (3 ics.) has coincided with an hypertrophied outflow tract of the right ventricle in the seven cases in this series in which it was recorded.

The method of assessing ventricular hypertrophy post mortem as described above, is considered to be of value in correlating the cardiographic findings, particularly when the heart is of normal or small overall size.

FIG. 5.—(A) Tracing to illustrate extreme abnormality of V2 (3ics.); the r' pattern is present in V2 in this case. Lesser degrees of abnormality show in V2 (3ics.) when not present in V2. LV/RV=0.95. Case No. 279.

(B) Tracing to illustrate the limit of normality accepted in V2 (3ics.) This tracing is from a patient in the clinical series with complicated pneumoconiosis stage B and no other evidence of involvement of the right heart. Case No. 40, C.

RIGHT VENTRICULAR HYPERTROPHY IN THE ELECTROCARDIOGRAPH

A planned investigation of pulmonary function carried out by the Pneumoconiosis Research Unit (M.R.C.) on normal men and coalminers with all stages of pneumoconiosis has provided the opportunity for a cardiological examination of miners with pulmonary disease. This part is principally concerned with the results of the electrocardiographic investigations. The tracings were taken with the patient reclining at an angle of 45 degrees, standard limb leads, CR chest leads, and V chest leads being recorded with an instrument that gave three simultaneous traces. The subject then performed a standard amount of work—700 kg.m.—in the form of a two minute stepping test. The CR chest leads were recorded again at the termination of the test with the subject reclining. Then followed a cardioscopic examination and later a study of the standard chest radiographs. In all, 146 persons were examined, representing all the age groups, normals, and disease categories. The classification of the pneumoconiosis is that described by Fletcher *et al.*, 1949.

There were 44 normal men, of whom 16 were miners. The cardiographic findings conformed

closely to the normal series of Myers *et al.*, 1947 and of Sokolow *et al.*, 1949. The findings in 25 of these of ages comparable to the disease groups are given in Table I.

Forty-one patients with simple pneumoconiosis have been examined. Three had some evidence of left ventricular prominence and one other had paroxysms of auricular fibrillation; these findings were not related to the pulmonary disease. The remainder presented cardiographic patterns of normal form. In three instances there was respiratory variation of considerable degree in the tracings, especially after exercise, and in nine there was an increase in the height of the T wave after exercise. No suggestion of right ventricular hypertrophy could be found in this group.

TABLE I

ANALYSIS OF LEADS V I AND V 6 GIVING THE MEAN AMPLITUDES OF R AND S,
AND IN BRACKETS, THE STANDARD DEVIATIONS

Lead	Group	R wave	S wave
V I	25 NORMALS, Group N	4.4 mm. (2.1)	12.0 mm. (4.6)
	25 ABNORMALS, Group D	4.4 mm. (2.1)	13.3 mm. (5.8)
V 6	25 NORMALS, Group N	15.9 mm. (5.2)	1.06 mm. (1.5)
	25 ABNORMALS, Group D	10.0 mm. (3.9)	1.9 mm. (1.4)

There were 24 persons with complicated pneumoconiosis, Stage B, with massive shadows present in the chest radiographs. Four only had any evidence of cardiovascular abnormality and were classed as borderline cases of right heart involvement due to the pulmonary disease. Two of these had high resting pulse rates, which increased considerably on exercise, and marked respiratory variation in the cardiograph, especially in the right chest leads. One other had a CR1 suggesting right heart involvement and also considerable variation with breathing; the last subject with a doubtful CR1 pattern presented slight enlargement of the right ventricle on cardioscopic examination.

The last group to be considered contains 25 cases of complicated pneumoconiosis, Stage D, coalminers with advanced massive shadows, pulmonary distortion, and considerable emphysema. These patients were incapacitated with gross dyspnoea on effort, and the lung function studies revealed severe impairment; 9 of these had clear evidence of right heart involvement and a further 5 were borderline cases.

Comparison of the cardiographs of this group and an equally balanced group of normal men with ages ranging from 35 to 60 years is given, the normals for convenience are labelled group N and this group is D. The P waves of lead II in the normal group have a mean amplitude of 1.7 mm. and none exceed 3 mm.: in group D the mean amplitude is 2.3 mm. and in 2 records exceeds 3 mm. Lead III shows little difference between the groups either in P, QRS, or T forms. The more frequent vertical heart pattern in group D tends to lower the R wave in lead I.

The leads from positions C1 and C6 best represent the overall chest lead pattern and details of the V leads are given (Table I). The chief significant differences between the groups are the reduction in amplitude of the R wave and the increase in amplitude of the S wave in V6 in group D, changes that are statistically significant in this series. This change in V6 has been noted by other workers (Sokolow, 1949).

In all subjects a V chest lead from the inner part of the left third interspace anteriorly has been recorded, it is called V2 (3 ics.). In the normals the pattern resembles V2; in six traces there is an rSr' pattern, the highest r' being 4 mm. and the mean height 2.2 mm. In group D the r' was present in 9 instances with a mean height of 6.2 mm., and a range from 2 mm. to 16 mm., a significant difference. The peak of the r' wave falls an average of 0.07 sec. after the start of the QRS deflection and the r' wave is not due to a conduction defect. The suggestion made earlier that the abnormal r'

wave in V2 (3 ics.) is useful evidence of hypertrophy of the right ventricular outflow tract is supported by these findings.

The recording of cardiograms at rest and immediately on terminating a standard exercise test revealed considerably higher resting rates in group D and a greater increase in rate with the same amount of exercise; thus a resting rate of 90 a minute with an increase to 115 a minute on the standard test, was not uncommon. Variations in the contour of the QRS can occur normally with breathing, but in complicated pneumoconiosis there is a much greater respiratory variation particularly in CR1 where the mean measured difference showed that group D had twice the degree of respiratory variation seen in the normals (Fig. 6). This curious phenomenon is under further study at present.

FIG. 6.—Chest leads CR1, CR2, CR4 illustrate the change with respiration. In CR1 and V1 on inspiration the S wave lessens in amplitude, the R wave simultaneously increases and the T wave lessens or inclines to negativity. The r' becomes prominent in (C). The three traces are recorded simultaneously. Case Nos. 339, 310, 51.

This graded series of miners, from normal men through the categories of simple pneumoconiosis to the most severe degree of complicated pneumoconiosis, shows no striking cardiographic change and that found is mainly in the most severe Stage D. Here the first change is in V6, a reduction of the R wave, an increase in the S wave. An occasional cardiographic pattern of classical right ventricular hypertrophy is found in Stage D. Also in Stage D an r' wave above normal amplitude in V2 (3 ics.), is rather frequent, a finding that is helpful in the diagnosis of outflow tract hypertrophy on the right side.

The increased resting pulse rate and the further abnormal increase in the rate with moderate exercise are not an indication of ventricular hypertrophy, but of a compensatory mechanism called forth by the effect of severe pulmonary disease on the heart.

Respiratory variation of the cardiogram is a normal finding but is here found in abnormal degree in the right chest leads in abnormal cases, and surprisingly in those whose chest wall and diaphragmatic movement are restricted. It is well known that there is respiratory variation of the peripheral arterial pressure, and several workers have demonstrated changes in the ballistocardiogram on respiration (Otis *et al.*, 1946). The electrocardiographic variation must be presumed to be due to positional changes for the present, but certain features justify further study. The same may be said of the increased height of the T wave after exercise which is most marked in the severe cases; clearly tachycardia is partly responsible but does not account for the total increase in some of the severe cases.

The relationships between the cardiological findings and the lung function studies must be the subject of another report. In general, there is over 50 per cent impairment of lung function in those patients who are found to have right ventricular hypertrophy, but there are also patients without evidence of hypertrophy who have the same impairment of lung function. It would seem that the duration of the severe stage of lung disorder is operative in addition to the degree of the disorder.

Clinically it may be said that evidence of right heart involvement is most uncommon in the pneumoconiosis of coalminers, until the disease is complicated and there is progressive massive fibrosis with emphysema and distortion. The pathological studies have shown the great degree of right ventricular hypertrophy usually found in these advanced cases, and have forced us to the admission that the electrocardiographic patterns are a poor test of early right ventricular hypertrophy. An effort has been made in this work to improve the cardiographic diagnosis of hypertrophy by assessing the changes in V1 and V6 and also by the introduction of V2 (3 ics.). Certain other pointers to the severity of the right heart stress are the increased resting pulse rate and the further increase on a standardized exercise test. The circulatory effects of respiration, both normal and abnormal, still require further elucidation.

SUMMARY

A series of 50 hearts from cases of coalminers pneumoconiosis have been dissected and the ventricular weights recorded. The ratio of left ventricle to right ventricle, LV/RV ratio, shows a great reduction, and there is a consistent relative increase in the weight of the right ventricle.

A cardiographic study of 146 persons (normal men, miners and non-miners, and patients with all stages of pneumoconiosis) has shown that V6 changes most with increasing severity of lung disorder. In the most severe groups the R wave of V6 is diminished and the S wave is increased in amplitude. A suggestion is made that a lead from the third interspace immediately above V2 may help in the diagnosis of right ventricular hypertrophy when it shows an r' wave of 4 mm. or more in height.

Certain effects of respiration on the electrocardiogram are described, particularly the respiratory variation of the chest leads on the right side. This is presumed to be a positional change, but some findings suggest that the mechanism of respiratory variation should be further investigated in relation to respiratory function.

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