

THE HEART IN RHEUMATOID ARTHRITIS

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Recent reports suggesting the presence of cardiac lesions at autopsy in a high proportion of patients with rheumatoid arthritis appear to be in conflict with clinical experience. In view of the scarcity of comprehensive clinical, and the absence of radiological studies of the heart in rheumatoid arthritis, and also of the contradiction between clinical and pathological findings, there appears to be a need of further clinical and radiological material.

The incidence of heart disease at autopsy in patients suffering from what is variously called nodular rheumatism, chronic rheumatism, and genuine arthritis deformans is, in earlier studies, given as between 30 to 50 per cent (Charcot, 1881; Kast, 1901; Grzimek, 1932; Klinge, 1933), the heart disease being more frequent in secondary than in primary chronic rheumatism. Changing conceptions of the ætiology and classification of rheumatic diseases in more recent years are an obstacle to the direct evaluation of these findings.

More recently cardiac lesions "indistinguishable from those of rheumatic fever" were described in 56 per cent of 24 patients with rheumatoid arthritis at necropsy by Baggenstoss and Rosenberg (1941 *a, b*), in 22 per cent of 23 patients by Bayles (1943), and in 66 per cent of 38 patients by Young and Schwedel (1944).

In a clinical study Dawson (1933) found 7 cases of manifest rheumatic heart disease in 100 patients suffering from rheumatoid arthritis. Colver (1937) in 69 children with Still's disease detected carditis during life in only 1; of 4 studied at autopsy none had carditis. Hensch and Rosenberg making a study of all their arthritic patients on a given date (1941) found none with clinical or electrocardiographic evidence of carditis. The incidence of clinically manifest rheumatic heart disease in a series of rheumatoid arthritis patients, examined by

Bayles and McGinn (1943) was 5 per cent. Young and Schwedel's series (1944) occupies a unique place here, since the diagnosis of rheumatic heart disease was made during life in 18 of 32 patients with rheumatoid arthritis: this, together with the high number of positive cardiographic findings, suggests selective accumulation of patients with heart disease. Elman (1944) in 100 patients with rheumatoid arthritis found 8 with "rheumatic carditis," 5 of whom gave no history of rheumatic fever; he also observed 22 patients in whom rheumatic carditis coexisted with rheumatoid arthritis. Rogen (1947) found only one patient with mitral disease in a group of 33 with rheumatoid arthritis.

MATERIAL AND METHODS

The 60 patients studied here were selected from 150 patients with rheumatoid arthritis, after eliminating those where the clinical picture was not typical, where rheumatic fever occurred in the past, where chest deformity or inability to stand upright prevented satisfactory radioscopy, and where records were not sufficiently complete. The criteria of diagnosis were those laid down by the Committee of the International League against Rheumatism (1934), Poynton and Schlesinger (1937), and a Committee of the American Rheumatism Association (1942). The clinical picture was regarded as typical if the smaller joints of the hand were characteristically involved in addition to the larger joints, and if joint involvement was accompanied by all or most of the following signs of general systemic involvement: anæmia, weight loss, general malaise, easy fatigability, and increased sedimentation rate. Patients in whom the process was limited to the larger joints or to a single joint, those in whom its onset coincided with the menopause, and those with a history of rheumatic fever or with

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raised blood pressure were excluded. Selection in this manner resulted in a group of patients with normal blood pressure, suffering from advanced rheumatoid arthritis of considerable duration.

Forty-three of the 60 patients were women. Ages ranged from 25 to 69 years, 5 being in the third, 6 in the fourth, 13 in the fifth, 21 in the sixth, and 15 in the seventh decade. Healed pulmonary tuberculosis was found in 2 patients (Cases 4 and 32), and duodenal ulcer in 1 (Case 49). From each patient a relevant history was taken and a careful physical examination made, including X-ray and electrocardiogram.

CLINICAL MANIFESTATIONS OF HEART DISEASE

Only 3 of these 60 patients presented clinical manifestations of heart disease.

Case 9, male, aged 59, with extensive articular involvement, was the only one in the series who had a mitral diastolic murmur. Radiologically his heart showed moderate left ventricular enlargement. Displacement of the barium filled gullet as a result of left auricular enlargement was not present and the electrocardiographic record was normal. Subjective symptoms related to the heart were absent.

Two patients, both women, aged 69 and 62 respectively (Cases 2 and 30), displayed signs of heart failure with congestion. There were no acoustic abnormalities, the cardiac silhouette was normal, and satisfactory response to digitalis and mercurial diuretics was obtained in both. The electrocardiogram in Case 2 showed auricular fibrillation. In the absence of signs of valvular involvement and in view of the patient's age, heart failure due to coronary disease appears the leading possibility with rheumatic heart disease not excluded.

Faint systolic apical murmurs were found in a number of patients. Some complained of slight shortness of breath and palpitation. Apart from the patients with frank heart failure these symptoms were mild, and it seems that their presence is of no specific significance in view of the anæmia and general debility characteristic of rheumatoid arthritis.

ELECTROCARDIOGRAPHIC CHANGES IN RHEUMATOID ARTHRITIS

Master and Jaffee (1932) taking daily cardiograms in 17 patients for 53 days, found only the slightest evidence of myocardial involvement. Logue and Hanson (1944) in 100 cases with prolonged P-R interval found that rheumatoid arthritis has been present in 5. Young and Schwedel (1944) found changes in 7 of 22 patients, 4 having auricular fibrillation. The changes in rheumatic carditis are transient and very variable, most cardiographic abnormalities having been encountered and a prolonged P-R interval being the most frequent.

We have examined 131 cardiograms in these 60

patients, and except for low voltage curves have found only 3 with significant changes. A woman, aged 46, with typical active rheumatoid arthritis had a normal curve, but a year later, when she was improving after chrysotherapy, had S-T depression in the first and elevation in the third lead. A man, aged 69, had a normal curve first, but later developed auricular fibrillation and congestive heart failure. A male, aged 69, with some cardiac enlargement, had a P-Q interval of 0.22 seconds. As the second and third patients were 69, these findings have little, if any, significance.

Low Voltage. Low voltage in the standard leads has occurred more frequently in the present series than any other single cardiographic abnormality. A voltage of all deflections in all leads of 0.5 mV or less is regarded as abnormal, and was found in 5 of the 60 patients (8 per cent). Low voltage has been associated with other cardiographic abnormalities in 2 patients, and with radiological cardiac enlargement and congestive heart failure in one of these. The 3 other patients of this group have presented no further evidence of heart disease. The ages of the 5 patients with low voltage were 57, 67, 67, 69, and 69 years. In 18 patients (30 per cent) the voltage was 0.8 mV or less.

The incidence of low voltage in healthy subjects shows considerable variation from author to author. It ranges from none (Shipley and Hallaran, 1936; Hoskin and Jonescu, 1940; and Larsen and Skulason, 1941), 1.8 (Viscidi and Geiger, 1943), and 3 per cent (Levitt, 1939; Chamberlain and Hay, 1939) to 6 per cent (Leach, Reid, and White, 1941), and 8 per cent (McFarland *et al.*, 1939). A voltage of less than 0.8 mV was found in 30 per cent by McFarland *et al.* (1939). That age does not determine the occurrence of low voltage is suggested by the fact that both the Viscidi and Geiger (1943) and the McFarland (1939) groups consisted of young, healthy airmen.

In order to obtain further information, minimum, maximum, and average values of R I, R II, and R III in the first tracing obtained from each patient were determined. Table I shows a comparison between values found in this series and normal controls reported by other workers in healthy subjects and also a control series of 50 healthy subjects.

In the present series the incidence of voltage below 0.5 and below 0.8 mV respectively is as high as the highest in published work. It is higher than in a series of 50 healthy controls. The voltage of R in the present series is less than in any reported by other authors, with the exception of Lewis and Gilder (1912) and less than in a series of 50 normal controls examined by us. In view of the elasticity of normal standards it is difficult to decide whether

TABLE I

COMPARISON OF THE VOLTAGE TO R IN RHEUMATOID ARTHRITIS WITH PUBLISHED DATA AND NORMAL CONTROLS

	Chamberlain and Hay (1939)	Hoskin and Jonescu (1940)	Larsen and Skulason (1941)	Wilson and Nyboer (1937)	Lewis and Gilder (1912)	Present study	
						Arthritis group	Control group
No. patients ..	302	50	100	104	52	60	50
Minimum							
R I ..	1.5	—	1.8	1.5	2.0	1.0	2.1
R II ..	3.6	—	4.1	4.0	5.5	2.0	2.0
R III ..	0.5	—	0.7	1.0	—	1.0	0.5
Maximum							
R I ..	18.6	—	12.4	19.4	12.0	17.0	14.3
R II ..	23.6	—	23.4	23.6	16.5	21.0	21.0
R III ..	20.5	—	20.8	20.0	13.5	14.0	20.2
Average							
R I ..	8.0	9.8	7.7	5.73	5.16	4.85	7.07
R II ..	12.7	13.5	10.2	11.90	10.32	8.61	9.28
R III ..	7.3	6.5	4.6	7.99	6.61	3.95	4.03

the above findings really show a tendency to lower voltage, in patients with rheumatoid arthritis.

Of the conditions associated with rheumatoid arthritis, anæmia (Turner, 1932; Szekely, 1940) and generalized wasting (Speckman and Rich, 1931; Steuer, 1934; Leach *et al.*, 1941) were found to coincide with low voltage. Considering that pericarditis is one of the known causes of low voltage, it is of interest that pericarditis was found at autopsy in a proportion of patients with rheumatoid arthritis by Baggenstoss and Rosenberg (1941 *a, b*) Bayles (1943), and Young and Schwedel (1944).

RADIOLOGICAL STUDY OF THE HEART IN RHEUMATOID ARTHRITIS

Published work on the heart in rheumatoid arthritis, as indicated above, deals chiefly with anatomical and clinical findings. Extensive search of the journals has failed to reveal a comprehensive radiological study. Rogén (1947) seems the only author who attempted cardiac X-ray examinations in some of his 33 rheumatoid arthritis patients, but gave it up because of difficulties in positioning, due to inability of the patients to stand upright.

Method. As it was intended to use heart size prediction tables based on height and weight, the 60 patients with rheumatoid arthritis in this series were selected from patients able to stand upright. In addition a control group of 100 patients, seen in the course of daily radiological routine by one of us (F. J. G.) was also investigated; they were selected to show an age distribution similar to the rheumatoid arthritis group, and patients with raised blood pressure were again excluded. Using prediction

tables based on weight in patients in whom weight loss is a prominent feature, the possibility arose that the impression of cardiac enlargement might be produced where discrepancy between predicted and actual heart measurement was due to discrepancy between the patient's present and past weight. To test and minimize this source of error, the control group consisted of patients most of whom had shown considerable loss of weight.

The heart was examined by frontal teleradiogram and fluoroscopy including study of the barium-filled gullet. To determine heart size three standards were used.

(a) The transverse cardiac diameter was compared with prediction tables based on height and weight, as devised by Ungerleider and Clark (1939). Generally 80 per cent of normals fall within ± 10 per cent of the predicted transverse diameter; not more than 10 per cent exceed $+10$ per cent. This means that a few normal hearts are classified as being enlarged, and others with minor degrees of enlargement are not recognized. With these qualifications enlargement of the heart exists when the actual transverse measurement deviates from the predicted measurement by 10 per cent or more. (b) According to Comeau and White (1942) in the great majority of cases a transverse diameter greater than 13.4 cm. in males or 12.4 cm. in females is a sign of cardiac enlargement. Considered as a group the transverse diameter will be below 13.4 cm. in 90 per cent of normal males and below 12.4 cm. in 94 per cent of normal females. (c) In using radiological methods for estimating heart size the difficulty of applying mathematical methods to biological

problems must be remembered (Gwynne, 1933; Comeau and White, 1942). The diagnosis of cardiac enlargement was therefore made only if in addition to showing increased measurements, the cardiac silhouette was judged to be enlarged on inspection.

Table II shows that subjective judgment and measurement were not always in accord. That radiographic measurement alone should not be relied upon to diagnose cardiac enlargement is indicated by the fact that different hearts are shown as enlarged by different standards (Table II).

Heart Size. As measurements of heart size and subjective judgment (Table II) have given divergent results they will be discussed separately.

A transverse diameter of more than 10 per cent in excess of the predicted figure, was found in 15 patients (25 per cent) in the rheumatoid group, with only 8 per cent in the control series. In 8 patients (13 per cent) in the rheumatoid group the transverse diameter was 15 per cent or more in excess of the predicted figure, with not a single instance in the control series. The figure of 12.4 cm.

TABLE II
PATIENTS IN WHOM THE HEART WAS FOUND ENLARGED ACCORDING TO ONE OR MORE OF THE THREE STANDARDS EMPLOYED

Case No. and type of enlargement	Transverse cardiac diameter in excess of predicted +10% of 12.4 in women, 13.4 in men				Heart judged enlarged
	Predicted (cm.)	Actual (cm.)	Excess (percentage)	Actual (cm.)	
1	—	—	—	13.3 F	—
5 LA*	—	—	—	— F	—
6 LV,LA	11.9	13.2	11	13.2 F	+
7 G (Fig. 1)	—	—	—	14.0 F	++
9 LV	11.6	14.0	21	14.0 M	+
19 LV	11.6	13.1	13	13.1 F	+
20 LV (Fig. 2)	—	—	—	15.0 M	++
24 G (Fig. 3)	13.1	15.1	15	15.1 M	++
27	—	—	—	13.5 F	—
28	11.7	13.2	13	13.2 F	—
30	—	—	—	13.2 F	—
32 G	11.5	13.2	15	13.2 F	+
35 LV,LA (Fig. 4)	11.1	13.6	23	13.6 F	++
36 LV,LA	—	—	—	15.0 M	+
43 LV (Fig. 5)	11.7	14.0	20	14.0 M	++
46 LV (Fig. 6)	—	—	—	14.7 M	++
48 LV (Fig. 7)	13.6	15.6	15	15.6 M	++
51	11.8	13.5	11	13.5 F	—
53 G	11.6	14.5	25	14.5 F	+
56	10.9	12.3	13	— F	—
57	11.2	12.4	11	— F	—
58	10.4	11.7	13	— F	—
60 LV (Fig. 8)	11.2	13.1	17	13.1 F	++

* LV=enlargement mainly left ventricular. G=enlargement generalized. LA=left auricular enlargement causing gullet displacement. F=female. M=male. +=moderate enlargement. ++=pronounced enlargement.

Eight women with a transverse diameter between 12.5 and 13.0 cm., but with no other evidence of cardiac enlargement, are not included in this table.

Discrepancy was particularly obvious with regard to the standard of Comeau and White (1942); the number of hearts with transverse diameters in excess of their figures was strikingly high (45 per cent), while on inspection many appeared to be normal. For this reason the relation of each heart examined to this standard was recorded, but the result was not regarded as decisive in judging heart size in any given patient, although considered significant in group comparison.

in females and 13.4 cm. in males was exceeded by 27 patients (45 per cent), the corresponding figure in the control series being 10 per cent.

On inspection the heart was judged to be enlarged in 14 patients (23 per cent). Enlargement was regarded as pronounced in 8 (13 per cent), and moderate in 6 (10 per cent). The radiograms showing pronounced enlargement are reproduced in Fig. 1 to 8. Subjective judgment differed from that based on prediction tables. In Cases 7 and 46,

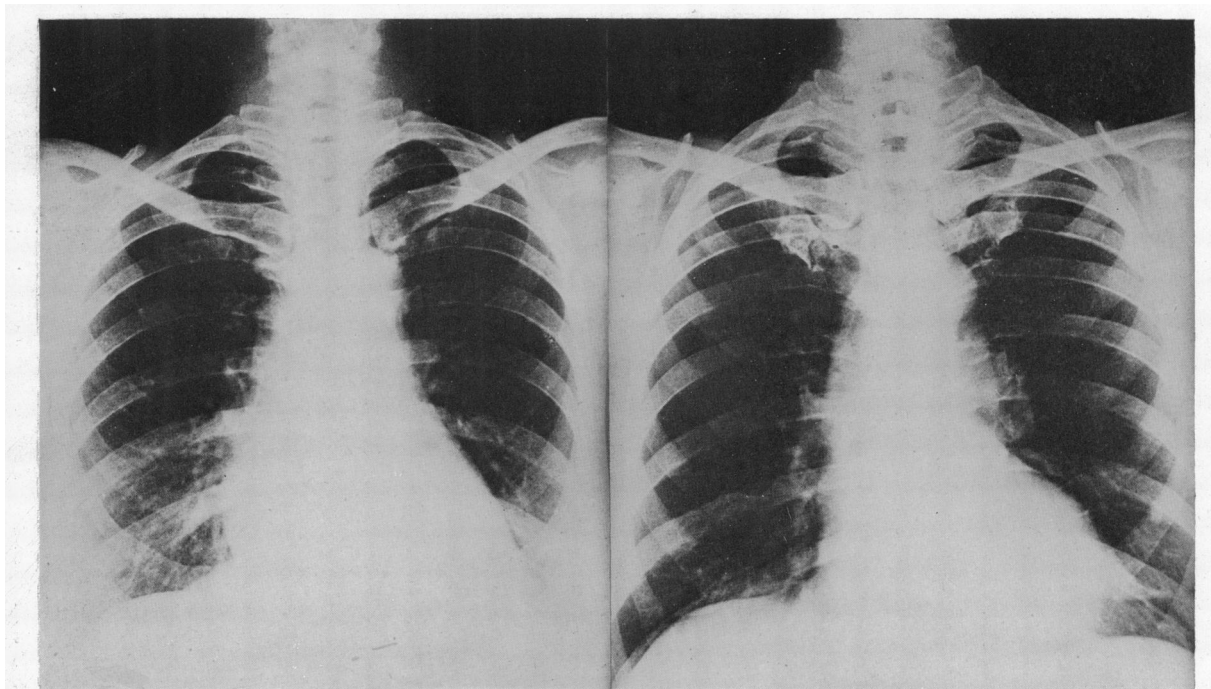


FIG. 1.—Case 7, female, 52 years. Generalized enlargement by inspection, although measurement at upper limit of predicted range. Clinically normal heart. Cardiogram normal.

FIG. 2.—Case 20, male, 63 years. Mainly left ventricular enlargement by inspection, although measurement at upper limit of predicted range. Clinically normal heart. Cardiogram normal.

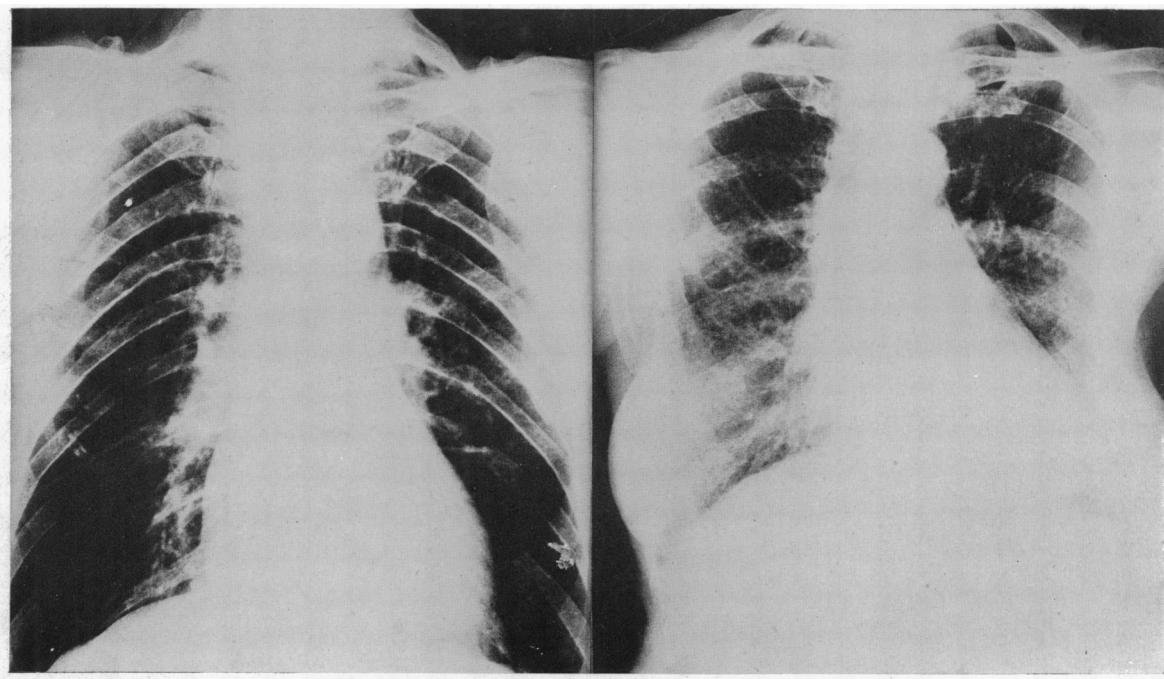


FIG. 3.—Case 24, male, 69 years. Generalized cardiac enlargement by inspection and measurement. Clinically normal heart. P-Q phase 0.22 sec.

FIG. 4.—Case 35, female, 63 years. Left auricular and left ventricular enlargement by inspection and measurement. Clinically moderate shortness of breath on exertion. Cardiogram normal.

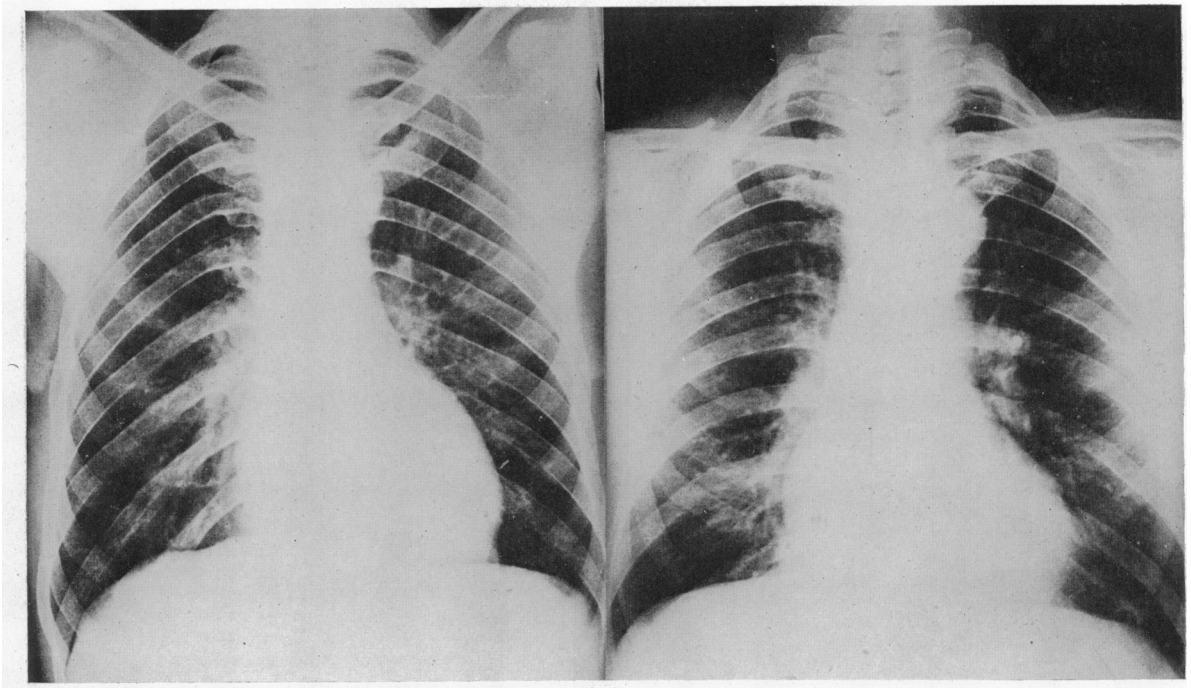


FIG. 5.—Case 43, male, 26 years. Mainly left ventricular enlargement by inspection and measurement. Heart clinically normal. Cardiogram normal.

FIG. 6.—Case 46, male, 46 years. Mainly left ventricular enlargement by inspection, although measurement at upper limit of predicted range. Clinically normal heart. Cardiogram normal.

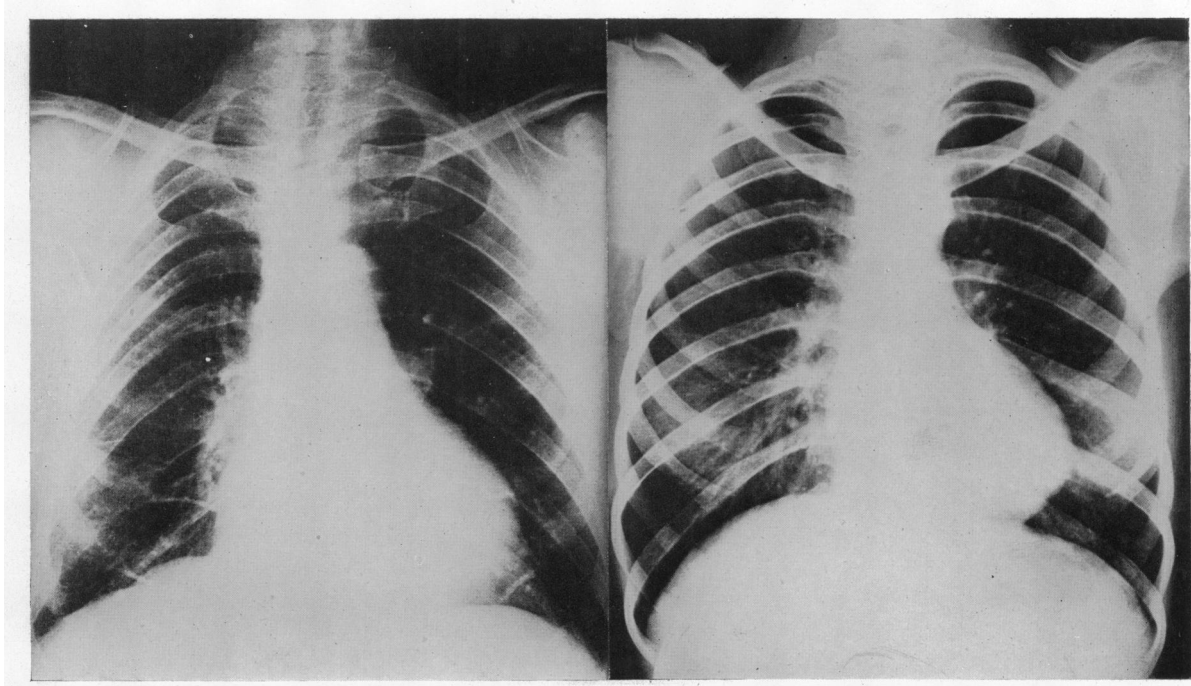


FIG. 7.—Case 48, male, 65 years. Mainly left ventricular enlargement by inspection and measurement. Triple rhythm with extra sound in mid-diastole, otherwise clinically normal. Normal cardiogram.

FIG. 8.—Case 60, female, 23 years. Mainly left ventricular enlargement. Palpitation and shortness of breath on moderate exertion, otherwise clinically normal. Normal cardiogram.

the actual transverse diameter was less than 10 per cent in excess of the predicted figure although on inspection pronounced enlargement was deemed to be present. On the other hand, of the patients whose transverse diameter was more than 10 per cent in excess of the predicted figure, 4 were not judged to be enlarged (Cases 28, 51, 56, 58). Of the 8 patients whose hearts were 15 per cent or more in excess of the predicted measurements all were judged to be enlarged, 3 moderately, and 5 considerably (Table II).

In clinical practice the radiological diagnosis of cardiac enlargement is usually made in a patient presenting signs and symptoms of a well-known heart disease and in most cases finally rests on a combination of radiological and clinical judgment. The situation is different here, a type of patient being examined in whom enlargement of the heart has not been demonstrated radiologically in the past, and who is suffering from a disease where the presence of cardiac involvement is regarded as questionable and is the unknown factor being investigated. Clinically these patients are characterized by the absence of signs or symptoms of heart disease. Pronounced cardiac enlargement such as is present in 8 patients in the present series can be diagnosed on radiological grounds alone. The diagnosis of slight to moderate enlargement on the other hand should be made only in the presence of supplementary evidence (Parkinson, 1933; Bramwell, 1933). Clinical manifestations of heart disease, which are the usual source of such evidence, are strikingly absent in patients with rheumatoid arthritis. It is suggested, on the other hand, that the evidence required to diagnose moderate cardiac enlargement in these patients is supplied from the following three sources.

Study of Patients as a Group. Prediction tables of heart size are composed of average figures derived from the study of large groups. The error involved in their use will therefore be smaller if applied to a group instead of to an individual. If in a single patient radiological determination of the transverse cardiac diameter in itself may not suffice to reveal the presence of moderate cardiac enlargement, in a group showing excessive measurements a tendency to cardiac enlargement may be assumed. In the single patient the impression of cardiac enlargement may be created or hidden by variations within the physiological range, in a group variations in the two directions will tend to neutralize each other. Comparison of the rheumatoid patients with the control group shows that a clear line of demarcation between patients with normal heart size and those with a tendency to enlarged hearts can be achieved by applying this method to groups instead of individuals.

Comparison with Anatomical Findings. Estimates of radiological enlargement in the present series are lower than the incidence rate of enlarged hearts reported at autopsy. Baggenstoss and Rosenberg (1941 *a, b*) found hearts of greater than normal weight in 11 of 25, Bayles (1943) in 13 of 23, and Young and Schwedel (1944) in 19 of 33 patients. These figures include cardiac enlargement due to rheumatic heart disease and to other causes, including hypertension. Considering that radiological

heart size estimation will inevitably miss a number of moderately enlarged hearts, and that the present series did not include patients with hypertension, there is no obvious discrepancy between the autopsy and radiological data.

Radiologically revealed cardiac enlargement although in disharmony with the absence of clinical manifestations, is in keeping with the only positive group of facts we possess about the heart in rheumatoid arthritis, namely the findings at autopsy. Discrepancies between radiological and clinical findings would appear no more "startling" (Hench, 1941, referring to the anatomical findings) than the much discussed discrepancy between anatomical and clinical findings. The question arises here as to what extent these two in themselves independent discrepancies will serve to explain each other. In the absence of symptoms and signs radiological enlargement may be the only clinical manifestation of the changes found at autopsy.

Pronounced Cardiac Enlargement. Cardiac enlargement of a pronounced degree was found in 8 out of 60 patients with rheumatoid arthritis. In addition, 6 patients had hearts suggesting enlargement. By themselves these may not be regarded as evidence of frequent cardiac enlargement. If, however, an increased number of hearts are found to be at the upper limit of normal and 8, as compared with none in the control group, are found to be enlarged to a pronounced degree, the suggestion emerges that the hearts are actually increased in size in more than 8 out of 60 patients.

Radiological Configuration of Enlarged Hearts. Of the 8 considerably and 6 moderately enlarged hearts, left ventricular enlargement alone was found in 7, left ventricular and left auricular enlargement in 3, and generalized increase in size in 4. Left auricular enlargement alone was present in 1, and with left ventricular enlargement in 3 patients.

Predominantly right-sided enlargement was not seen in any of the hearts examined. It appears that the left ventricle is the chamber chiefly affected, and it was enlarged in every patient with an abnormal heart, excepting the one in whom left auricular enlargement was the only change present.

DISCUSSION

The result of the above investigation suggests the presence of radiologically evident, mainly left ventricular cardiac enlargement in 23 per cent of patients with rheumatoid arthritis. On the other hand it appears that the cardiac abnormality underlying the enlargement is characterized by the absence of recognized clinical and electrocardiographic manifestations of heart disease.

In an attempt to explain this discrepancy, published reports regarding autopsy findings in rheumatoid arthritis will have to be taken into consideration. Three recent anatomical studies suggest that in patients with rheumatoid arthritis a disease of the heart is present which is "indistinguishable" from that found after

rheumatic fever (Baggenstoss and Rosenberg, 1941 *a, b*; Bayles, 1943; Young and Schwedel, 1944). The incidence of cardiac involvement in the three groups varies from 26 to 66 per cent, but unanimity exists with regard to certain points. It seems that the lesions found resemble rheumatic carditis more closely than any known cardiac lesion and that pericarditis, chronic or subacute myocarditis, gross valvular distortion and cardiac enlargement was present in a proportion of cases. The participation of the various chambers in the enlargement is not stated.

In addition to these changes, coronary sclerosis with or without myocardial infarction was found in a smaller proportion of patients in the three above groups. Coronary sclerosis as a cause of radiologically manifest cardiac enlargement in patients with rheumatoid arthritis is, in addition to its low incidence in the above three autopsy series, excluded by the following points. The role of coronary sclerosis alone in causing cardiac hypertrophy is still a matter of controversy. That it is able to cause enlargement is stated by Palmer (1937), denied by Jones (1930), Gross and Spark (1937), and Maun (1941). Clawson (1939) states that narrowing of the coronary orifices in syphilitic aortitis fails to produce hypertrophy in the absence of a valvular lesion. If only 1 out of every 2 or 3 patients with coronary sclerosis is expected to have an enlarged heart, to explain cardiac enlargement in 23 per cent, as found in the present series, it would have to be assumed that coronary sclerosis was present in 46 to 69 per cent. If coronary sclerosis were the chief cause of cardiac enlargement in rheumatoid arthritis, an increase of the incidence of cardiac enlargement with age could be expected. Table III shows that

TABLE III
INCIDENCE OF ENLARGED HEARTS DECREASING WITH
INCREASING AGE

Age group Years	All rheumatoid arthritis cases	Transverse diameter 10% in excess of predicted		Heart judged enlarged	
		No.	Percent- age	No.	Percent- age
20-39	11	4	36	3	27
40-59	34	8	24	8	24
60-79	15	3	20	3	20
20-79	60	15	—	14	—

this is not the case, as the percentage of enlarged hearts actually decreases with age. In the control group only 8 per cent exceeded predicted cardiac measurements. Neither clinical nor cardiographic evidence of coronary sclerosis was demonstrated in the arthritic patients.

If coronary sclerosis is excluded, only the changes "resembling rheumatic carditis" occur with enough regularity to need consideration as the basis of the radiological cardiac enlargement. Whether these anatomical changes mean that rheumatic carditis is actually present, or whether they are an expression of rheumatoid arthritis ("rheumatoid carditis" Rosenberg

et al., 1944) is undecided. This problem is closely linked with the controversy regarding the relationship of rheumatic fever and rheumatoid arthritis. Since the aetiology of these two diseases is unknown, proof of their identity must be indirect, and either attempting to show the similarity of the conditions under which they occur, or the similarity of certain pathological and serological changes found in both. The two diseases tend to occur in identical families and individuals, and their geographic distribution and seasonal incidence is similar. The identity of the rheumatic nodule in the two diseases has been pointed out by Coombes and Coates (1926), Dawson (1933) and by Poynton and Schlesinger (1937), but questioned by Bennett *et al.* (1940). Immunological evidence suggests the presence of a streptococcal antibody in both diseases, but in rheumatic fever it is a lysin (Todd, 1932; Coburn and Pauli, 1932), in rheumatoid arthritis an agglutinin (Dawson *et al.*, 1934). The response to salicylates is different in the two diseases. Inflammatory granulomatous changes regarded as specific for rheumatoid arthritis, were found by Steiner *et al.* (1946) in muscle tissue obtained by biopsy. Similar changes were also found (Curtis and Pollard, 1940; Freund *et al.*, 1942) in the peripheral nerves. These changes were not demonstrated in rheumatic fever. The above data suggest that while evidence of the pathological and immunological identity of the two diseases is incomplete and in view of more recent investigations open to doubt, the two diseases tend to occur under similar circumstances, and in the same individual. For this reason, and because the cardiac changes described at autopsy in rheumatoid arthritis resemble rheumatic fever more closely than any other known cardiac disease, the possibility that the radiological enlargement is due to rheumatic carditis deserves attention.

If so, one must explain the discrepancy between the presence of the familiar clinical and radiological picture in chronic rheumatic carditis and its absence in these patients. This need still exists, if the anatomical changes are an expression of "rheumatoid carditis," because, regardless of aetiology, it is still not explained why gross cardiac disease remains without symptoms or signs.

The heart disease assumed to be present in rheumatoid arthritis is characterized by the presence of left ventricular enlargement, and the absence of gross congestive heart failure, acoustic abnormalities, auricular fibrillation, and cardiographic changes.

The absence of congestive failure is partly explained by assuming a milder myocardial process due to three causes. (1) A difference in the age at onset, which will alter the course of these two forms of heart disease owing to the greater sensitivity of the heart in youth to the rheumatic agent (Poynton and Schlesinger, 1937; Sangster, 1940; De Liee *et al.*, 1943), and the greater capacity of the young heart muscle to hypertrophy (Palmer, 1937). Hypertrophy, although initially increasing the efficiency of the heart eventually becomes a liability. (2) A reduction of physical performance of the patient due to joint involvement. Available evidence suggests that the progression and outcome of rheumatic heart disease is influenced by the amount of rest and

exercise during its course. (3) More gradual development of the rheumatic process. This would allow for smoother adjustment of the heart to the changes in circulatory dynamics.

The assumption of a milder form of cardiac disease is in keeping with the statement by Rosenberg *et al.* (1944) that the cardiac lesion associated with rheumatoid arthritis is not as severe or widespread as such a lesion in young persons who have rheumatic fever, and also with the fact that in the present series of patients with rheumatoid arthritis, radiologically evident cardiac enlargement, although frequently present, was in most cases not excessive.

There are two possible contributory causes of the absence of abnormal sound phenomena: (1) A milder myocardial process. Triple rhythm due to rapid diastolic inflow is the only acoustic abnormality in heart disease of the rheumatic carditis type, the absence of which may be explained on this basis, since it usually accompanies advanced disease (Weber, 1937; Evans, 1943). (2) Physical properties of the blood. The incidence and intensity of other murmurs here being considered is not dependent on the extent of cardiac involvement. If as we are informed by those who investigated such hearts anatomically valvular and muscular changes are identical in the two conditions, the question arises, whether the physical state of the blood accounts for some of the differences. Estimation of blood velocity and the B.M.R. in ten patients in this series gave normal results, nor did significant murmurs appear when blood velocity was increased by exertion. Information regarding the kinematic viscosity of full blood in rheumatoid arthritis is so far not available and is being studied at present by Sagar and Fischmann (1947).

Left ventricular enlargement was found in this series, instead of the characteristic cardiac silhouette of rheumatic valvular disease. In chronic rheumatic carditis the distribution of dilatation and hypertrophy of the cardiac chambers is determined by the combined effect of the inflammatory lesion and the distribution of mechanical strain, the latter according to the localization of the pathological process in the valves. In rheumatoid arthritis, on the other hand, with the lessening of physical exertion the importance of the mechanical factor will diminish and the distribution of enlargement will follow chiefly, or alone the distribution of the inflammatory process. In rheumatic carditis Gross and Erlich (1930)

found that Aschoff bodies were almost invariably present in the interventricular septum and the posterior wall of the left ventricle, while other parts of the heart were less frequently invaded. Also the enlargement of the heart found in patients dying in the early stages of rheumatic carditis is largely limited to the left ventricle (Boyd, 1931).

SUMMARY

The 60 rheumatoid arthritis patients in this series were selected to have normal blood pressure, no history of rheumatic fever, and no chest deformity, and to be able to stand upright for cardiac radiography.

Clinical and electrocardiographic findings were negative apart from a tendency to low voltage of the standard leads.

Radiological examination suggested the presence of cardiac enlargement in 23 per cent of patients. Enlargement was left ventricular in 50 per cent of these, left ventricular and left auricular in 21 per cent, generalized in 29 per cent, and right sided in none of the hearts judged enlarged. The gullet was displaced backward at the level of the left auricle in 29 per cent of enlarged hearts.

It is thought probable that radiologically manifest cardiac enlargement in rheumatoid arthritis is an expression either of chronic rheumatic carditis or a form of cardiac involvement due to the aetiological factor of rheumatoid arthritis.

An attempt is made to explain the prevalence of the left ventricle in cardiac enlargement and also the absence of clinical and electrocardiographic manifestations of heart disease in the presence of radiologically demonstrated enlargement.

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