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CARDIAC FIBROSIS AS A RESULT OF CORONARY OBSTRUCTION.

By G. A. GIBSON, M.D., D.Sc., F.R.C.P.Ed.; and ROBERT MUIR, M.A., M.D.

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Cardiac Fibrosis as a Result of Coronary Obstruction.

By G. A. Gibson, M.D., D.Sc., F.R.C.P.Ed.; and Robert Muir, M.A., M.D.

The study of the morbid changes which the muscular wall of the heart undergoes, in consequence of local nutritive disturbances, is not only of great importance from the pathological point of view, but it is also of real value to the scientific physician. There can be no doubt that our appreciation of the consequences of such nutritive disorders renders much service in the explanation of obscure symptoms, and affords useful indications as to prognosis and treatment. An instance of such changes, which came under our observation lately, seems to us to be of so much clinical and pathological interest, that we have no hesitation in placing it on record and discussing its more important features.

D. S., a tinsmith, æt. 36, complaining of cough and breathlessness, was admitted to Ward 22 of the Royal Infirmary, under the care of Dr. Gibson, on 12th March 1893.

The patient seemed to have no hereditary tendencies to disease, and, until a short time before admission, his surroundings were good in all respects. His previous health was excellent, and, in particular, although he had complained of vague pains in the joints and muscles, he had never suffered from any definite rheumatic attack. For a few months before coming to the hospital he had been ailing, but, as his wife was confined to bed on account of a severe illness, he had struggled on until, a fortnight before his admission, he had been compelled to take to his bed. In addition to the cough and dyspnœa, the patient complained of weakness and sleeplessness.

The appetite was very poor, and the patient had fre-

quently vomited undigested food. The bowels were constipated. The liver was slightly enlarged, extending from the fourth rib to an inch below the costal margin in the mammary line. There was no ascites.

The patient was distinctly anæmic, and so pasty-looking as to suggest renal disease. There was no morbid appearance connected with the spleen or glandular apparatus.

Dyspnœa was constant, but with paroxysmal exaggeration, especially at night. Some præcordial pain and considerable palpitation were complained of.

On inspection and palpation of the neck and chest, no abnormal symptoms could be observed, except that the cardiac impulse was extremely weak. The radial arteries were somewhat hard, but the vessels were unfilled, and the tension was moderate. The pulse was extremely frequent, varying from 130 to 150; it was perfectly regular, and the wave small. Cardiac dulness extended from $2\frac{1}{2}$ in. to the right to $4\frac{1}{4}$ in. to the left of the midsternal line. On auscultation the heart sounds were extremely faint. A soft systolic murmur was heard over the præcordia, but it was so feeble that it was difficult to follow its distribution with accuracy. There could be no doubt that it was loudest at the lower end of the sternum, but there was a probability that there were two other points of maximum intensity, one towards the apex of the heart, the other over the manubrium.

Dulness was present at the bases of both lungs on account of double hydrothorax, and many crepitations were to be heard above the level of the fluid on either side.

The urine was scanty, varying from 18 to 24 oz. per diem. It contained no albumin or tube-casts.

Considerable œdema of the subcutaneous tissues was present in the lower limbs.

The patient complained much of sleeplessness and restlessness.

The diagnosis arrived at was cardiac failure, with mitral and tricuspid incompetence, in consequence of arterial degeneration, probably attended by some changes in the aortic cusps, from chronic atheromatous processes.

The patient was treated by means of cardiac tonics:

strophanthus was administered, as well as iron and strychnine, but his condition did not improve. Stimulants were also freely exhibited: alcohol, ether, and ammonia being given at short intervals. Although the patient slept better than before admission, the dyspnœa became worse, and on Sunday, 19th March, the breathlessness was so distressing, that the resident physician, Dr. Donald Macaulay, found it necessary to remove some fluid from the right pleural sac by aspiration. Some temporary relief was obtained, but in a very short time pronounced œdema of both lungs followed, with a copious expectoration of frothy, sanguineous fluid. The stimulation was increased, and gentle counter-irritation employed, but the patient's condition became rapidly worse, and he died suddenly on the following day.

The following is a summary of the chief facts observed at the post-mortem examination, which was performed by Dr. Muir on the 21st March.

The body was well nourished, and there was marked dropsy of the lower limbs.

Heart.—The pericardium was normal. The heart was considerably enlarged, especially its left side, and weighed 15 oz. The right side was greatly distended with dark clot; the left ventricle, on the contrary, was rather collapsed, and its wall could be felt to be thin, especially along the anterior aspect, where it also had a peculiar stiff consistence.

The aortic valve was very slightly incompetent, not sufficiently so to be of any importance.

Diameters of orifices: Aortic, ·9 in.; pulmonary, 1 in.; mitral, 1·2 in.; tricuspid, 1·8 in. The aortic cusps were slightly thickened at their margins, otherwise the valves were normal. The left ventricle was considerably dilated, measuring 4¼ in. in length, and its width was increased even more proportionately. Its wall was much thinned, especially along the anterior division of the coronary artery, which ran further from the septum than usual, where it measured only ¼ in. in thickness. This part was distinctly tough, and apparently fibrous, as was definitely found to be the case on microscopic examination. The musculi papillares were exceedingly atrophied, and partly fibrous at their extremities, and the

columnæ carneæ were much thinned, some of them being almost like paper. The myocardium was rather pale and soft where there was no fibrous change. There were some ante-mortem thrombi adherent to the endocardium in the apex of the ventricle.

The right ventricle was of normal size, but its wall was much hypertrophied, its thickness being $\frac{1}{4}$ in. at places, and the columnæ carneæ were thick and powerful, contrasting markedly with those of the left ventricle. The auricles were almost normal, the right being slightly dilated.

The aorta at its commencement showed extensive atheroma, chiefly in the form of soft cushion-like patches, with very little calcareous change. The orifice of the left coronary artery was in the centre of one of these patches, and was so much narrowed that it only admitted the entrance of a fine bristle. Immediately beyond, the coronary artery was of normal width, and in its course nothing abnormal was visible. The right coronary was not affected.

On injecting the right coronary with water, a very small quantity passed into the left, showing the existence of a slight anastomosis.

There was a slight hydrothorax on both sides, the left pleura containing 12 oz., and the right 20 oz. of serous fluid.

Lungs.—The lower lobe of the right lung showed commencing hypostatic pneumonia.

In both there was congestion, with some œdema posteriorly. Only slight emphysema was present.

The other organs showed chronic venous congestion.

Microscopic examination.—Portions of the left ventricle, taken chiefly from the anterior wall, were examined both in the fresh condition and after fixing in corrosive sublimate and in spirit. The changes found may be described as twofold, viz. extensive atrophic and degenerative changes in the muscle fibres, and marked interstitial growth of fibrous tissue in certain parts of the heart wall.

The fibrous change was most marked towards the inner part of the wall, and at many places a distinct though not uniform layer of fibrous tissue was present a short distance

from the endocardium. In this fibrous tissue, which was for the most part of very dense character, there were visible merely the pigmented remains of muscle fibres and a few blood vessels,—arteries and wide veins. Fibrous tissue was seen at places spreading inwards irregularly and diffusely between the degenerated muscle fibres to the endocardium, and also running outwards along the intermuscular planes as more distinct bands which also invaded the muscle bundles. This fibrous overgrowth reached its maximum at the anterior branch of the coronary artery (mentioned above), where the dense layer came right up to the surface of the heart, so that at this part the wall was fibrous in its whole thickness, with the exception of a layer of degenerated muscle on the inner aspect (Plate XI., Fig. 1). Here also the wall was thinnest.

The most marked degenerative changes were found in the innermost part of the wall, *i.e.* internal to the dense fibrous layer. In fresh sections the muscle fibres there showed extensive granular degeneration and disintegration, some of which was fatty in nature, and at places, amongst the fibrous tissue, appeared simply as rows of granules or small globules. The degeneration was also marked in the papillary muscles, which also showed fibrous thickening here and there on their surface. These parts, in sections cut in paraffin, showed shrivelled muscle fibres, which appeared as if they had lost their sarcous substance, only a sort of envelope remaining, and which, with the rubin and orange stain, were coloured only a faint pink instead of the usual deep orange (Plate XI., Fig. 1, *d*). Immediately superficial to the fibrous planes, muscle fibres could be seen of nearly normal appearance, but, at places, of extreme tenuity, with thin fibrous bands spreading between them (Plate XI., Fig. 2). But an interesting fact is, that at some places it was quite evident that an extreme atrophy of the fibres preceded and was not caused by the fibrous overgrowth. At no place was there any collection of necrosed fibres, such as are seen in infarcts in the heart wall. Beneath the epicardium the muscle appeared fairly healthy, some of the fibres, however, appearing rather hypertrophied and their nuclei large and somewhat vacuolated. (Plate XI., Fig. 1, *b*).

The condition of the arteries, always an important point

in such cases, was the following. The large branch on the anterior wall and its branches beyond showing very slight periarteritic and endarteritic thickening at some places, were practically normal. At no place was there any distinct encroachment of their lumen, or any thrombosis. In the dense fibrous tissue, as one would expect, some of the capillaries and small arteries were lying compressed and closed, but even there many of the arterioles had quite normal walls, with wide lumen. In short, there was no evidence of there being any primary lesion of the *branches* of the coronary arteries of importance in relation to the other changes.

Clinical Remarks.—The most important points of clinical interest to be drawn from the study of the case which we have described, are connected with the physical signs. There is one subjective symptom, however, which must not be passed over without comment. The presence of præcordial pain has been noted in most of the cases of this nature which have been placed on record. It was, unfortunately, impossible to trace out the exact distribution of the pain in the present instance, and we must therefore content ourselves with the simple mention of the symptom. As regards the physical signs which were observed, the first point worthy of notice is the fact that the pulse was throughout absolutely regular. In cases of cardiac failure, irregularity of the heart's action is extremely common, and it is a matter of interest to observe that in this patient there was no approach to arhythmia. The only remaining aspect of the case requiring remark from the clinical point of view is the entire absence during life of physical signs indicating aortic regurgitation. There was nothing resembling the water-hammer pulse of aortic incompetence, and no diastolic murmur could be heard. The reason for the absence of both phenomena is probably to be sought in the pronounced condition of cardiac failure.

Pathological Remarks.—The special points of interest pathologically in this case are the marked stenosis of the left coronary artery, the degenerative changes in the wall of the left ventricle, and the process of sclerosis in the situations described. The importance of lesions of the coronary arteries in relation to the nutrition of the heart wall has long been

recognised, but it is only within comparatively recent years that the relationship of certain of these lesions to one form of so-called interstitial myocarditis has been established. Weigert, Huber, and Ziegler in Germany, Lindsay Steven in this country, and others, have been chiefly instrumental in establishing this relationship (*vide* references). When the branches are the seat of chronic endarteritic change, they may become obliterated or they may be thrombosed, and infarcts result in the heart wall,—somewhat soft necrosed areas, which form the condition known as *myomalacia cordis*. As a sequel, interstitial change occurs around these areas, and in this way there result fibrous patches scattered in the myocardium. We have recently had an opportunity of examining a heart in which all the stages of this process could quite easily be followed. Apart, however, from actual closure of the vessels, and the formation of infarcts, fibrous myocarditis occurs in association with disease of the branches of the coronary arteries, as both Huber and Lindsay Steven state. The former says that owing to the impairment of the nutrition a molecular necrosis may result, which is followed by fibrous change,—a statement which, if correct, has an important bearing on this case.

Now, it is of importance to distinguish the changes associated with diseases of the branches from the effects produced by a stenosis of the commencement of a coronary artery. The latter may occur from atheroma at the beginning of the aorta, either the soft form, as in this case, or that associated with marked calcareous change, whilst the coronary arteries and their branches are quite healthy, and conversely the latter may be atheromatous, whilst the aorta is free. On examining accounts of cases of stenosis of the artery at its commencement, we find that the changes observed in the various cases are: degenerative change, partly fatty, in the wall of the ventricle, though in some this has been absent; dilatation of the cavity, with, in some cases, a certain degree of atrophy, in others hypertrophy of the wall. No doubt the result will vary according to the amount and duration of the stenosis, the condition of general nutrition, etc. In a few cases fibroid patches have been recorded, but

we have not been able to find any description of an extensive fibroid change of the same character as in this case.

The most reasonable explanation of the fibroid condition is, that the impaired nutrition due to the small supply of blood produces gradual degenerative changes, which excite or are followed by interstitial overgrowth. We have already pointed out that the area of greatest change corresponded with the distribution of the anterior division of the left coronary artery, and that there was no sufficient disease in the latter or its branches to account for it. It may be that the results were more marked in this position, because the slight anastomosis with the right coronary artery assisted the posterior part of the ventricle more than the anterior. Of course the interstitial change might possibly have preceded and been independent of the stenosis of the artery, but there is no evidence of this being the case.

The marked thinning of the wall of the left ventricle in front, without any aneurismal dilatation, is also worthy of note; and the case well illustrates one mode in which a lesion in the aorta may affect the left ventricle, and produce mitral incompetence with its accompaniments.

Literature.

The following list contains a number of papers bearing on this subject:—

Dickinson, *Trans. Path. Soc.* 1866, p. 53; Greenhow, *ibid.* 1867, p. 80; Greenfield, *ibid.* 1877, p. 157; S. West, *ibid.* 1883, p. 66; Samuelson, *Virchow's Archiv*, Bd. lxxxvi. s. 539; Percy Kidd, *Trans. Path. Soc.* 1886, p. 197; Sainsbury, *ibid.* 1887, p. 117 (Cases of Stenosis of Coronary Arteries); Weigert, "Die pathologische Gerinnungsvorgänge, *Virchow's Archiv*, Bd. lxxix. s. 106; Huber, "Ueber den Einfluss der Kranzarterienerkrankungen auf das Herz," *ibid.* Bd. lxxxix. s. 236; Ziegler, "Ueber Myomalacia Cordis," *ibid.* Bd. xc. s. 211; Leyden, "Ueber die Sclerose der Coronar-Arterien," *Zeitsch. f. klin. Med.* 1884, ss. 459, 539; Lindsay Steven, "On Fibroid Myocarditis," Lectures in *Lancet*, 1887, ii., *Journ. of Path.* 1893, vol. ii. p. 190; Budor, "Obliteration des artères cardiaques," Paris, 1888; Nicolle, "Les grandes scléroses cardiaques," Paris,

1890; Verhand. d. zehnten internationalen medicinischen Congresses, Bd. ii. Abtheil. iii. s. 67, 1891.

DESCRIPTION OF PLATE XI.

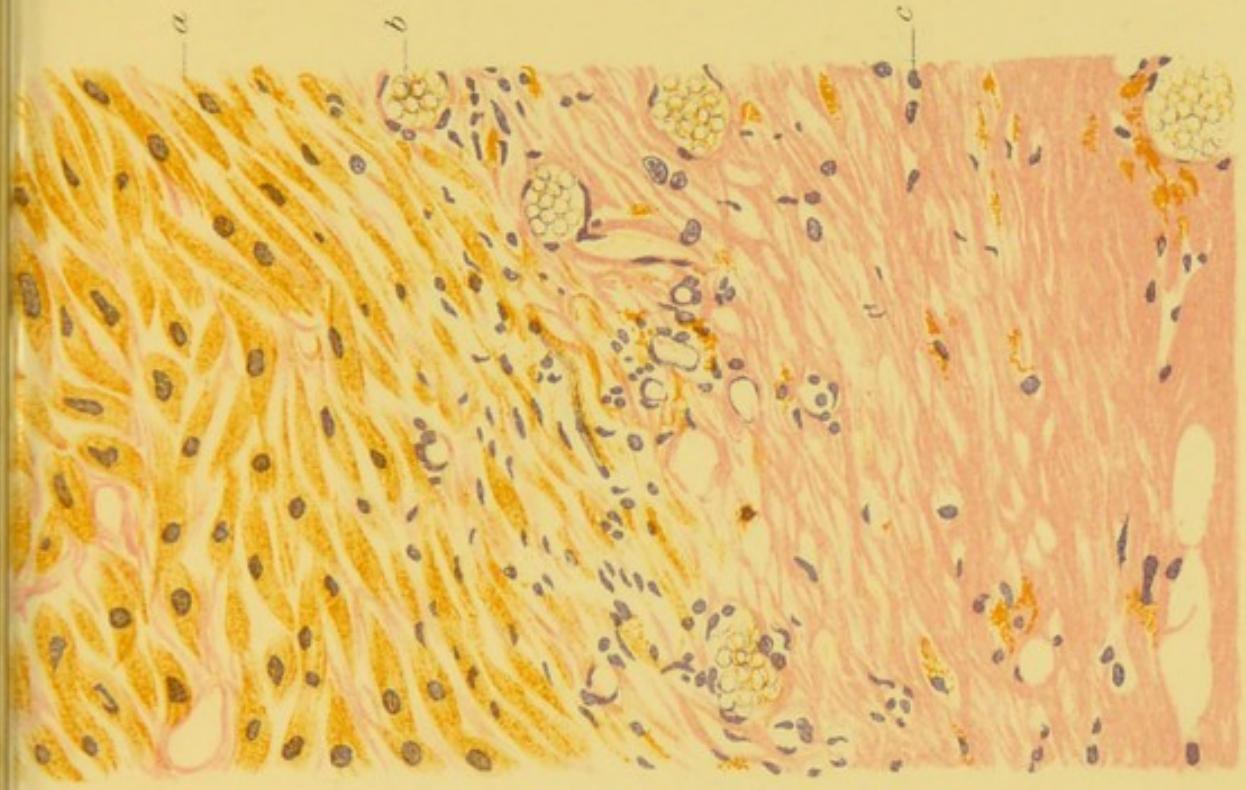
FIG. 1. Low-power view of section of heart wall, close to the position of greatest fibrous change. × 50.

a. Epicardium with dilated blood vessels.
b. Layer of comparatively healthy muscular tissue under epicardium.
c. Plane of dense fibrous tissue.
d. Layer of degenerated and partly fatty muscle fibres next to endocardium.

FIG. 2. High-power view of portion of same. × 250.

a. Comparatively healthy muscle fibres.
b. Atrophied muscle fibres with fibrous bands spreading between them.
c. Fibrous tissue with scattered collections of pigment, etc.

Hæmatoxylin, rubin, and orange.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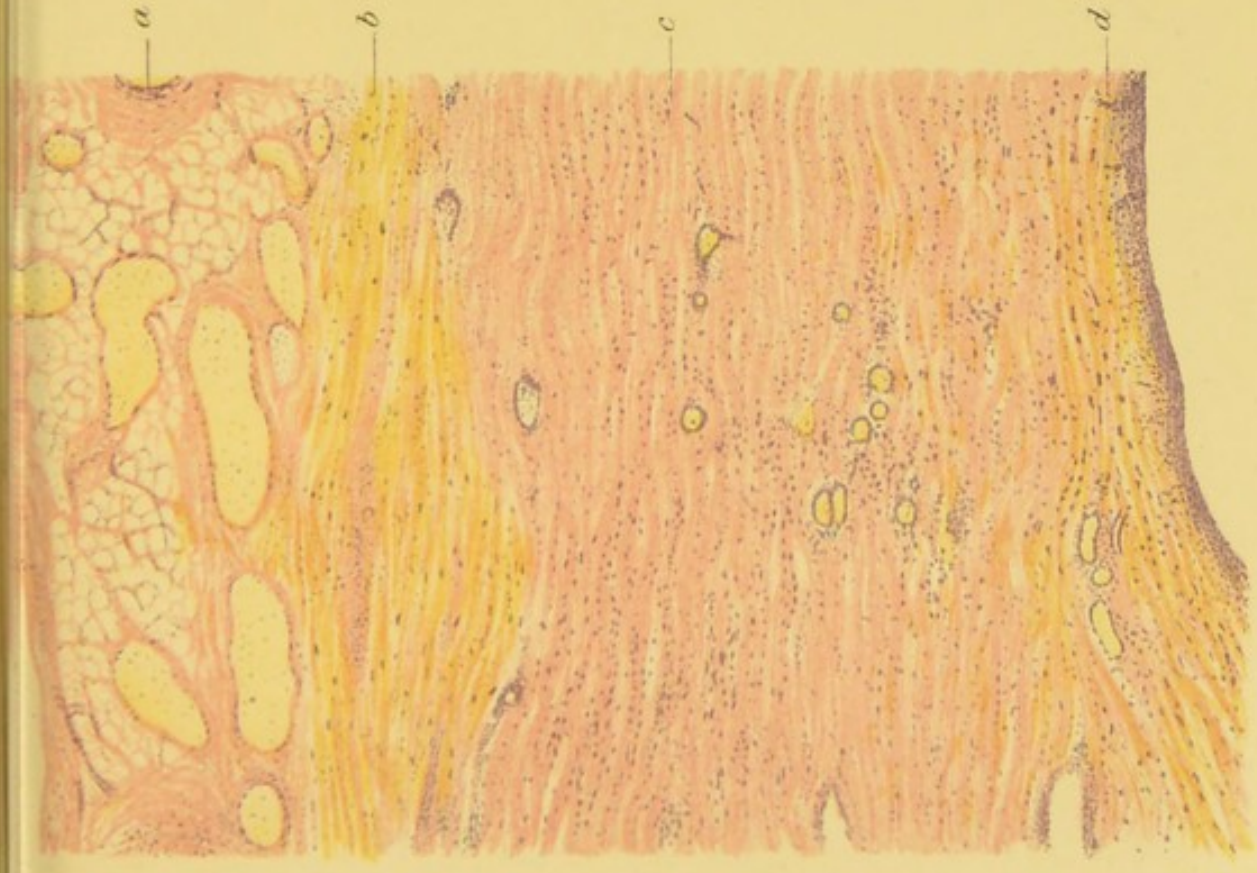
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