On fibroid degeneration and allied lesions of the heart, and their association with disease of the coronary arteries / by John Lindsay Steven.

ON

FIBROID DEGENERATION

AND

ALLIED LESIONS OF THE HEART,

AND THEIR ASSOCIATION WITH DISEASE OF THE CORONARY ARTERIES.

BY

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ON

FIBROID DEGENERATION.

LECTURE I.

INTRODUCTORY.

THE condition of the heart wall, the consideration of which is to form the main object of the following remarks (viz., a development of what appears to the naked eye to be fibrous tissue in the midst of the muscle substance), is one which most English authorities would probably class under the term "chronic interstitial myocarditis," but which is frequently, though not invariably, to be referred to a cause quite different from that indicated by this term, implying as it does inflammatory action of a chronic kind. This fibrous transformation is most frequently met with in the left ventricle, may not be at all apparent either on the external or internal surface of the cardiac wall, and generally, though not invariably, occurs in association with widespread degenerative changes of the arterial system, obviously involving in most instances the coronary arteries of the heart. Besides the change just referred to, a more acute process of the nature of softening is also frequently met with in association with the same degenerative arterial changes or with embolism; and although the appearances of this acute condition are very different from those of fibrous change, yet the operating cause is so obviously similar that the two must frequently be regarded as different stages of the same affection, or as differing from one another only in degree. The details of the cardiac lesions thus briefly referred to, and of fatty degeneration as associated with arterial disease, will be described in due course, but in the meantime it will be necessary to give some account of our previous knowledge of fibroid disease of the heart.

HISTORICAL AND CRITICAL ACCOUNT.

The conditions of the heart wall (especially fibrous transformation[1]), which form the subject of these lectures, have long been described by special observers, but it is only of recent years that what appears to be, in most instances, their true pathological significance has been hinted at, and that much more on the part of continental than of English physicians. From the time of Morgagni downwards frequent references are to be found in medical literature to a fibrous condition of the heart-wall, apart from the lesions met with in connexion with the valvular apparatus. It is to be noted, however, that in many of the earlier writings reference is made to those conditions only where the fibrous change had advanced so far as to be visible on the surface, without requiring any special dissection of the muscular tissue for its exhibition. Thus Morgagni,[2] in his twenty-seventh letter, which treats of "Sudden Death from a Disorder of the Heart," article 17, after giving illustrations of calcareous plates in the substance of the heart, writes as follows: "For it is certain that the fleshy fibres of the heart themselves sometimes degenerate into a tendinous hardness. Albertini, therefore, saw in some bodies 'that the substance of the heart from the basis to more than half its extent had become tendinous, as it were, both in consistence and colour; which kind of metamorphosis I have it in my power to confirm, if there were any occasion, by an observation of my own.'" It will thus be seen that the condition has been recognised from the earliest days of pathological anatomy, and it is interesting in the light of modern research to observe that the term "degenerate" is used in the description of the change. It is not to be supposed, however, that the term necessarily meant the same to Morgagni that it does to us.

Towards the end of the last and the beginning of the present century medical literature abounds in communications on the subject of aneurysm of the heart, a condition

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[1] I use the terms "fibroid degeneration" and "fibrous transformation" as synonymous, and I think the latter is more scientifically correct. It is a real transformation into fibrous tissue with which we have to deal, so that the term "fibro'd" seems rather cumbrous.

[2] The Seats and Causes of Diseases investigated by Anatomy, in Five Books. Translated from John Baptist Morgagni, by B. Alexander, M.D. Vol. i., p. 845. London: A. Millar, 1769.

which is very apt to follow upon the morbid condition at present under review. These records are therefore of great interest in the present investigation, and a few of the more important may be shortly referred to. Dr. Matthew Baillie[3] in his work on Morbid Anatomy describes a case of aneurysm of the left ventricle, and refers to the rarity of the condition. He explained the occurrence of the aneurysm by the muscular tissue at the apex being thin and weak, and so unable to resist the impulse of the blood against it. In 1817 Portal[4] published a long article on the subject of cardiac aneurysm, mainly from an etiological point of view. Among the different causes which he states as giving rise to aneurysms of the heart, the following may be mentioned: (1) the formation of false membranes inside and outside of the heart; (2) engorgement of the coronary arteries and veins; and (3) serous and purulent infiltrations. In the second of these it would seem as if we had a hint of the relationship which exists between a diseased condition of the coronary arteries and the development of cardiac aneurysm, but, so far as I can gather, the author simply refers to a dilatation of these vessels as one origin of aneurysm, and not to any nutritive change in the muscular tissue. About the same time important observations were published in France by Breschet[5] and Reynaud.[6]

Another very important paper in connexion with this subject from an historical point of view is that of Mr. John Thurnam on Aneurysm of the Heart.[7] Although, like the others just referred to, not directly bearing on the subject of fibrous transformation of the heart, the essay is very important from the fact that many of the cases were found to be associated with what is called "fibrous degeneration" of the heart-wall. With regard to the causation of the "fibrous degeneration" itself, the views adopted are pericardial adhesions and rheumatic inflammations. But nowhere is there found any allusion to the state of the coronary arteries as

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[3] Morbid Anatomy, by M. Baillie, M.D., London, 1793, p. 17.; see also his Plates (1799), Plate iii., Fig. 1.

[4] Sur les Aneurysmes du Cœur.

[5] Recherches et Observations sur l'Aneurysme faux consécutif du Cœur. Répertoire d'Anatomie et de Physiologie Pathologique, 1827, vol. iii.

[6] Reynaud, Journ. Hebd. de Médecine. 1829, vol. ii., p. 363.

[7] Medico-Chirurgical Transactions, 1838, vol. xxi. p. 187.

an etiological factor, nor are there any references to fibrous change as a morbid state of the heart-wall *sui generis*, and apart from aneurysm. Although in several of the cases the coronary arteries are mentioned as ossified, no opinion is expressed as to the probable relationship of this to the aneurysmal state. Interesting statistics are also given by Thurnam as to the situation of the aneurysm. Out of 74 cases, 58 were situated in some part of the left ventricle; and "the 67 aneurysms which occurred in the 58 cases, omitting one case in which this is not mentioned, may as regards situation be thus distributed: at or near the apex of the ventricle, 27; in different parts of the base, 21; in intermediate portions of the lateral walls, 15; in the interventricular septum, 3. ..... To conclude these observations on the situation of the aneurysms of the left ventricle, the only general conclusion that we can come to appears to be that the thinnest parts of its walls, or the apex and highest part of the base, are those which are much more frequently than any others the seat of the disease." This in general agrees with the observations of more recent writers and also with my own. It will thus be seen that in the record of Thurnam's cases we have a most important contribution to the literature of this subject from a pathological as well as from a historical point of view. In 9 cases the muscular tissue was the seat of more or less extensive "fibro-cellular degeneration," caused by inflammation it was supposed. If the disease was present to such an extent as to cause aneurysm in so many of the cases, we have strong reason for supposing that in its slighter manifestations, which are easily overlooked, it may occur much more frequently. The fibro-cellular degeneration, however, does not seem to have been regarded by Thurnam himself or by others as an independent morbid state; and one of the objects which I have at present in view is an attempt to establish that in fibrous transformation of the heart wall we have a diseased condition which has as much right, from a clinical as well as from a pathological point of view, to be considered an independent affection of the heart as fatty degeneration, or even as certain valvular lesions.

Another reference in regard to this subject, important from an historical point of view, may be found in the work

of Dr. Ramadge on the Heart.[8] In the chapter on Carditis he refers to a condition called "induration of the heart," said to be one of the results of inflammation attacking the muscular or intermuscular cellular tissue. "Sometimes," he adds, "the consistence of the organ is fibrous or tendinous; sometimes cartilaginous." Similar brief references to the condition may be found in most other text-books on heart disease, up till the present time.

Coming now to more recent times, it is found that this condition of the heart-wall has been abundantly observed and described by English physicians and pathologists, although, so far as I can find, the relationship of the change to obstructed coronary circulation has been almost entirely overlooked, and, as a general rule, it has been attributed to chronic myocarditis, probably not without truth, in a certain number of cases. In 1872 the attention of the profession in England was strongly directed to this subject by the Lumleian Lectures of Dr. Richard Quain on the Diseases of the Muscular Walls of the Heart.[9] Under the name "connective tissue hypertrophy," Dr. Quain enters into a full consideration of this affection of the myocardium. No one can read these lectures without seeing that the author was very strongly of opinion that the morbid state originated in an inflammatory action. "The origin," says he, "of the disease is unquestionably to be sought for in a chronic interstitial inflammation or hyperplasia." But he does not seem to recognise any influence on the part of the coronary arteries in bringing about fibrous transformation, although, in describing fatty heart, he points out that "there are no communications between the two coronary arteries; and hence in obstruction of one no collateral circulation can be set up." The strength with which the inflammatory theory is inculcated is further seen in the remarks on treatment: "In connective tissue hypertrophy, if we could diagnose it in its early stage, remedies likely to subdue the inflammatory state in which it takes its origin might have a beneficial influence." Here and there, too, statements which seem to contradict one another are met with, as if, on the whole, the true signifi-

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[8] Asthma, and on Diseases of the Heart, by F. H. Ramadge, M.D. Second Edition. London: Longmans, Brown, Green, and Longmans. 1847. Page 359. [9] THE LANCET, vol. i. 1872, pp. 391, 426, 459.

cance of the condition were not perfectly clear to the mind of the lecturer. Thus: "In connective-tissue hypertrophy the freedom of contraction of the fibres is restrained by the surrounding new tissue, and such a heart is dynamically weak. It would seem, so far as present observation goes, less likely to dilate than the simply degenerated heart"; whereas in the section on aneurysm of the heart he speaks of "the production of a fibroid cirrhotic state which yields to the internal pressure."

During the twenty years which preceded the delivery of Quain's Lumleian Lectures numerous cases of this transformation of the heart wall had been recorded in the current medical literature, to a few of which I may refer in some detail, in order to give some indication of the predominating opinion in the profession concerning the lesion. In 1854 Dr. W. T. Gairdner[10] reported a case of ossification of the coronary arteries, with tendinous degeneration of the heart; and I may add that this is one of the few records that I have been able to consult in which any mention is made of the coronary arteries. In the great majority of cases recorded the state of the coronary arteries is not mentioned, and therefore one is justified in coming to the conclusion that they had not been specially examined. In the Transactions of the Pathological Society of London is contained a rich collection of cases, a full reference to all of which is to be found in Dr. Hilton Fagge's communication, to be immediately referred to. Dr. Bristowe,[11] in recording a case of what is undoubtedly fibrous transformation, states that the cause in all probability was pericardial adhesion, and explains the production of the fibrous material by supposing that a deposit of lymph capable of organisation had been deposited between the fibres, the condition being thus comparable to a cirrhosis. No mention is made of the state of the coronary arteries. Again, Dr. T. Henry Green[12] records a case of sudden death, in which a patchy deposit of fibroid tissue was found in the left ventricle. The aorta and cerebral arteries were atheromatous, but the state of the coronary arteries is not described. The view expressed by Green in

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10 Monthly Journal of Medical Science, Edinburgh and London, 1854, vol. xix., p. 79.

11 Pathological Transactions, vol. vi., p. 148.

12 Ibid., vol. xxv., p. 47.

his Pathology as to the mode of production is that it is secondary to an inflammatory process, commencing either in the pericardium or endocardium. Interesting cases, from our present point of view, are also recorded by Bence Jones, J. W. Ogle, Whipham, Payne, Wilks, and Moxon.[13] Some of these cases, however, described by the contributors as fibrinous masses in the heart wall, I would regard as examples of the acute process—i.e., of infarction of the heart, which, as I have already indicated, is closely related from an etiological point of view to fibrous change. Moxon, however, in recording a case,[14] regards the fibrinous mass in the heart wall as coagulated blood, which had been effused into a partial rupture, and he distinctly recognises the state of the diseased coronary artery as a cause of the rupture. With the exception of Dr. Moxon's case, the state of the coronary arteries is not described in those referred to above; and although the relationship between diseased arteries and localised patches of extreme "fatty" change (as it was considered) in the myocardium had been made abundantly clear by Quain and others, it does not seem (judging from the records) to have struck many of the observers that there was any necessary connexion between arterial disease and the morbid state at present under review.

Without doubt one of the most important monographs on this subject is that of the late Dr. Hilton Fagge on Fibroid Degeneration of the Heart,[15] which may be said to contain the sum of British experience of, and teaching on, this subject up till the year of its publication—1874. The paper contains the results of much labour and thought, and in many places is to be regarded as a forecast of still more modern views on the subject; but a most striking feature is that in the midst of the most cogent reasoning on the pathology of the condition, not a word should have been said as to the etiological significance of obstructed coronary circulation. The first part of the paper gives the record in a very perfect way of eleven cases of the affection, whilst in the latter part the author states his opinions of the pathology of the condition, a few of which may be briefly referred to in order to

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[13] Ibid., vol. iii., p. 281; vol. vii., p. 167; vol. viii., pp. 116, 118, and 139; vol. xv., p. 87; vol. xxi., pp. 103 and 115; vol. xvii., p. 70.

[14] Ibid., vol. xvii., p. 70.

[15] Ibid., 1874, vol. xxv., p. 64.

give an idea of their wide scope. "For it is to be observed," says he, at page 89, "that this affection never attacks the whole heart at once, nor even the whole of a single chamber. It is always more or less localised, and sometimes invades only a very small tract." Such a statement seems to me to be a very strong argument in favour of the circulatory theory of its origin. At page 90 he says, "In the early stage, however, it sometimes seems that a retrograde change in the muscular fibres of the heart advances more rapidly than the growth of the fibrous tissue that should accompany it"; and he goes on to show how a softening, not unlike a pyæmic abscess in appearance, may be produced in some cases. It is very strange that he should have argued in this way, and yet not have suggested any connexion between obstruction of the coronary arteries and the lesion in question. We must, however, regard these statements of Dr. Hilton Fagge as forecasts of the still more modern views and observations of Weigert and Huber on this subject. He goes on to speak of the frequency with which aneurysm is due to this affection, and to point out how rupture of the heart, so frequent in local fatty degeneration of the muscular fibre, very rarely occurs in this condition. I think, however, that this statement must be received with some hesitancy, for, as I pointed out about two years ago in a paper on Rupture of the Heart, there is good reason for believing that many cases are not due to localised fatty change at all, but to infarction. "With regard to the *causes* of fibroid disease of the heart," says he, "we seemto be at present completely in the dark." He discusses in detail the influence of pericarditis, syphilis, &c., and points out that fibroid change of the heart wall may be a cause of sudden death.

As I have already indicated, the relationship existing between diseased coronary arteries and localised extreme fatty degeneration (leading to rupture in some cases) had been insisted on by Quain and others many years ago; but to show that the possibility of disturbed coronary circulation setting up other morbid states of the cardiac wall (more nearly allied if one may judge from the descriptions given to the conditions forming the subject matter of the present remarks) was an idea which occurred to one of the greatest living masters of our art in the earlier years of his work,

I may here briefly refer to Sir William Jenner's paper on Congestion of the Heart.[16] In this communication, which was made to the Royal Medical and Chirurgical Society of London, he writes: "The principal object of this paper is to call the attention of the Fellows of the Society to the occurrence of congestion of the muscular tissue of the heart; to the most common and direct consequences of that congestion—viz., induration, toughening, and thickening of the walls of the heart; and to the influence which those changes of texture exercise as predisposing causes on the development of permanent dilatation of the heart." The chief interest and importance of this paper are clinical, for it seemed to me, in reading it, that the pathological details were somewhat vague and indefinite, and my main object for referring to it at this point is to show, on the authority of a great clinical observer, the necessity of considering the condition of the coronary circulation in all affections of the heart.

In 1881 a most important paper on Fibroid Degeneration of the Heart was read by Dr. Charlewood Turner at the London meeting of the International Medical Congress.[17] In this paper he gives a minute description of the appearances, naked eye and microscopic, of three cases which he had the opportunity of carefully studying. In this article the influence of obstructed coronary circulation as an etiological factor in the production of fibrous transformation is, so far as I have been able to examine, for the first time categorically mentioned in English medical literature. "Other facts," he says, "suggest the possibility of the origination of localised fibroid degeneration as a result of arterial obstruction, or of venous thrombosis from injury to the vessel from overstraining the walls of the ventricle." And he further shows how some of Dr. Hilton Fagge's cases point to the same thing. From all points of view Dr. Turner's article must be regarded as a most valuable and original contribution to the literature of the subject. In the latter portion of his observations he deals with the generalised form of fibroid degeneration which is met with in dilated hearts, and which may contribute to cardiac failure, but which is a subject beyond the scope of my present investigation.

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[16] On Congestion of the Heart and its Local Consequences, by William Jenner, M.D. (Med. Chir. Trans.) London, vol. xliii., p. 199.

[17] Transactions of the International Medical Congress, Seventh Session, vol. i., p. 427. London: J. W. Kolckmann. 1881.

Having so far dealt, in as complete a manner as has been possible for me, with the work of the more recent English observers, it now becomes necessary that I should give some short account of the foreign literature of this subject which I have been able to consult. In the writings of two German observers—viz., Professor Weigert[18] and Dr. Carl Huber,[19] I have met, for the first time, with what appears to me to be an accurate and tenable explanation of the relationship existing between obstruction of the coronary arteries and the formation of fibrous patches in the walls of the heart, and I do not think I can do better than give a brief abstract of the views expressed by these gentlemen. My friend Professor Weigert, in his observations on the infarction of the heart, has shown that, where the circulation is slowly interfered with by sclerosis of the arteries, atrophy, with destruction of the muscular fibres, takes place without injury to the connective tissue. The shrunken fibres are thus set off by the stringy connective tissue, and this process, he considers, really constitutes the so-called chronic myocarditis. In such cases he has good ground for believing that the vascular change caused the destruction of the muscular fibres as a primary factor. If, however, the arrest of the circulation is more sudden in its onset, then yellow dry masses similar to coagulated fibrine make their appearance. Often the tissue appears quite normal (sometimes the transverse markings being quite apparent), but the muscular fibres and the connective tissue show no nuclei—a necrosis has occurred. Around these masses, as a reactionary phenomenon, abundant round and spindle cells are found.

Weigert's observations, which I have just noticed, appeared in the course of a long paper on the general subject of the Occurrence of Coagulation in the Tissues, and were followed by those of Huber in 1882. In Huber's paper the whole question of the so-called chronic myocarditis is discussed in a broadly scientific spirit, and his facts and conclusions are so convincing that it is difficult, after a careful perusal of the article, to arrive at any other opinion than that the

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[18] Ueber die pathologischen Gerinnungsvorgänge. Von Prof. Dr. Carl Weigert (Leipzig). Virchow's Archiv (1880), Band lxxix., p. 106.

[19] Ueber den Einfluss der Kranzarterien-erkrankungen auf das Herz und die chronische Myocarditis. Von Dr. Karl Huber. Virchow's Archiv (1882), Band lxxxix., p. 236.

condition of the coronary arteries must be regarded as one of the most important etiological elements in the production of the morbid changes at present under consideration. He bases his opinions on eighteen cases given in the paper, and a number of others which he thought it unnecessary to record, in all of which the fibrous changes associated with distinct vascular disease were well marked ; and, in addition, he gives brief details of four or five others, in which the acute affection or infarction of the heart wall was distinctly present. He describes the lesion as the occurrence in the heart-wall of lines or streaks of firm, tendinous, glancing tissue running parallel to the muscular fibres, and somewhat depressed beneath the level of the cut surface of the surrounding muscle. It is mostly observed in the median layers of the cardiac parietes, and is often not to be seen beneath the endocardium or pericardium. This is precisely the condition that I have observed in my own cases. He further remarks that the process, as it presents itself anatomically, is what is most commonly regarded as chronic myocarditis, and that until quite recently syphilis, rheumatism, or inflammation spreading either from the endo- or peri-cardium was regarded as its cause. He then proceeds to show by very cogent reasoning, as it appears to me, that in a very large proportion of cases indeed the lesion must be looked upon as primarily a vascular one. It therefore becomes at once evident that, from a strictly scientific point of view, the terms "chronic myocarditis," "indurative interstitial myocarditis," &c., are, as has been mentioned at the outset, in a large majority of cases misleading.

Since the foregoing remarks were originally penned, the splendid work on Medicine by Dr. Hilton Fagge has appeared, and one cannot help referring to the universal feeling of sorrow experienced by the entire profession when the gifted author was so suddenly called away before seeing the end of his labours. In the article on Fibroid Disease of the Heart,[20] it will be found that Dr. Fagge had fully appreciated the value of Weigert and Huber's researches, and saw in them an explanation of many of his own carefully recorded cases. He refers to myocarditis,

[20] The Principles and Practice of Medicine. By the late C. Hilton Fagge, M.D., F.R.C.P. Vol. ii., p. 32. London: J. and A. Churchill. 1886.

syphilis, and thrombi as causes, but obviously places most importance on the relationship existing between fibroid change and disease of the coronary arteries; and, referring to two of his own cases of infarction of the heart, he writes the following sentence: "Thinking over these two cases, I have long seen clearly that the peculiar change in the muscular tissue must be the primary affection, and that the fibroid development must be secondary." He then proceeds to speak of the light thrown upon the condition by the researches of Weigert and Huber.

Ziegler[21] has also very fully described acute necrosis of the heart-wall under the name of "myomylacea cordis," and has clearly related this change to obstructed coronary circulation; and the connexion is also insisted upon by Birch-Hirschfeld[22] in his large work on Pathological Anatomy.

The subject of interstitial myocarditis has also excited considerable attention on the part of French physicians, and in 1880 an account of a case was published by M. Déjérine.[23] In looking through his paper, I find that the case was one of anæmia complicated by aphasia &c.. At the post-mortem, the heart was found to be the seat of interstitial myocarditis, which was also stated to be primitive. As the consequence of the myocarditis, which he supposes to have been due to the anæmia, clots were formed in the cavities of the heart, which explained the occurrence of the aphasia. Holding the opinions which I have already indicated as to the pathology of fibrous transformation of the heart wall, I would rather be inclined to regard the interstitial myocarditis as also the result of embolism, and the description of the morbid appearances is in some places distinctly suggestive of infarction.

Dr. Wickham Legg's Bradshawe Lecture on Cardiac Aneurysm[24] is a most valuable contribution to our medical

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21 Lehrbuch der allgemeinen und speciellen pathologischen Anatomie. Vol. i., p. 408. Jena: Gustav Fischer. 1881.

22 Lehrbuch der pathologischen Anatomie. Von Dr. F. V. Birch-Hirschfeld. Zweite Auflage, Zweiter Band, 1 Lieferung. Leipzig: F. C. W. Vogel. 1883. Page 86.

23 Note sur un Cas de Myocardite Interstitielle Primitive. Le Progrès Médical, 1880, No. 39, p. 781.

24 Some account of Cardiac Aneurysms. Being the Bradshawe Lecture, read Aug. 18th, 1883, by Wickham Legg. London: J. and A. Churchill. 1884.

literature, and one of very considerable importance as regards my present subject, because in discussing the etiology of "fibroid disease" he differs from the views of Huber and Cohnheim. He makes no mention of Weigert's paper on Coagulation Necrosis, in which the theory was first promulgated, and which was published the year before Cohnheim's on the Results of Obstructing the Coronary Arteries of the Heart. His first objection is as to the age of Huber's patients, and he mentions several young persons who had the disease, and in whom the coronary arteries were reported healthy. Now, in going over cases of this disease, I have been struck by the fact that in very many the state of the coronary arteries is not mentioned at all, and unless there be a specific statement at least in the earlier recorded cases, I think we are justified in concluding they were not specially examined. His next objection is that in a case of Greenfield's the orifices of the arteries were obstructed without producing any result, except generalised molecular fatty change. Now, I am strongly of opinion that a distinction must be drawn between cases where the orifice is obstructed and cases where individual branches are occluded. In the former class of case, I think, resulting fatty degeneration is the rule. His next objection is as to the anastomosis of the cardiac coronary arteries, and this objection I will examine further on. As has been observed, the paper is a most valuable one, and the views of so careful an observer must be thoroughly taken into account, in order to arrive at any conclusion as to the true nature and cause of this interesting disease.

I have thus endeavoured to give some account of the history of our knowledge of fibrous transformation of the heart. In addition to the articles noticed above, several important papers, some of them of great clinical interest, have recently appeared, but space forbids me to do more than simply mention them. Thus we are indebted to O. Rosenbach,[25] E. Leyden,[26] Albert Robin,[27] C. Paul,[28] and others.

In Dr. Byrom Bramwell's large recently published work

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[25] Centralblatt für die medicinischen Wissenschaften, 1886, No. 38, p. 678.

[26] Ibid., 1884, p. 651.

[27] Sur les Ruptures du Cœur. Gaz. Hebdom. de Méd. et de Chirurg., Dec. 18th, 1885, No. 51, p. 829.

[28] Aneurysme du Cœur. Gaz. Hébdom. de Méd. et de Chirurg., March 6th, 1885, No. 10, p. 165.

on Diseases of the Heart, the subject of fibroid degeneration is included in the chapter on chronic myocarditis, "which," he says, "for practical purposes may be considered as synonymous with fibroid degeneration." In the long account of the etiology and pathology of the condition, not a hint is given of the possible direct connexion which this lesion may have with disease of the coronary arteries or obstruction of these vessels. The author points out, however, that long-continued venous engorgement of the heart-wall may have an influence in its production, and this statement is of importance in connexion with Sir William Jenner's paper, to which I have already referred. The absence of any reference to the causal influence of the coronary arteries in so large and valuable a work shows that the true etiology of a large section of such cases has not as yet been fully comprehended. I do not wish to assert that an abnormal condition of the coronary circulation is the cause of all fibroid conditions of the heart. Far from it; but so far as my experience and that of many others go, it is directly responsible for a very large number, in which anything of the nature of an inflammatory course of events cannot come into court. It is also further obvious that to many cases the term "chronic interstitial myocarditis" cannot logically or scientifically be applied. I am decidedly of opinion that the influence of the coronary arteries in producing many of the well-known lesions of the myocardium has not been sufficiently recognised, and if the publication of the present lectures has the effect of directing greater attention to these vessels among English physicians and pathologists my purpose will have been amply fulfilled. In Dr. Bramwell's article on "Aneurysm of the Heart" its association with fibroid change is fully recognised; and in the account of the author's case of rupture of the heart it is pointed out that the branch going to the ruptured area was obstructed. It is therefore somewhat strange that the great importance of the coronary arteries in all affections of myocardium should not have been more clearly stated and strongly insisted upon.

LECTURE II.

DISTRIBUTION OF THE CORONARY ARTERIES OF THE HEART.

As has been gathered from the foregoing considerations, the blood supply of the heart is intimately associated with the causation of the lesions at present under review, and, therefore, before going further it will be necessary briefly to refer to the different views which have been held as to the distribution and anastomosis of the cardiac arteries. Very great differences of opinion still exist as to whether the coronary arteries anastomose or not—some observers holding that the anastomosis between the two vessels is quite abundant and free, others maintaining that it is exceedingly limited or even absent altogether. Amongst the former are to be placed the names of Norman Chevers, Wickham Legg, Samuel West, Krause, and Langer; and amongst the latter those of Hyrtl, Cohnheim, and Joseph Swan. Chevers founds his opinion on a water injection; I think that this is clearly insufficient, and that no reliable conclusion can be drawn from it.[1] Wickham Legg says: "A number of human hearts were collected, and the arteries injected with a blue-coloured size. Nothing was easier than to fill one artery by injecting the other, and communicating branches could be seen passing over the apex of the ventricles....."[2] Samuel West says "The fact, then, of the very free and complete anastomosis of the coronary arteries is established."[3] It will be admitted that these opinions are most authoritative, and contain the certain convictions of the writers. On the other side of the question, however, the following sentences must be quoted. Joseph Swan says: "Although the coronary arteries communicate at the ring at the base of the heart and at the apex, the communication is not very free; but each can do little more than supply its

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[1] Guy's Hospital Reports, 1843., p. 109. [2] Loc. cit., pp. 19 and 20.
[3] The Anastomoses of the Coronary Arteries. THE LANCET, vol. i 1883, p. 945; also Path. Trans., vol. xxxv., p. 66.

respective region."[4] Hyrtl seems to have modified his original view that the arteries do not anastomose, and he now admits a capillary anastomosis. Cohnheim held that the arteries were end-arteries, and, with his usual minute care and scrupulous accuracy, has shown that, before coming to any conclusion as to the result of injection experiments, we must take carefully into account the character of the injection fluid used. He found that if a thin fluid, capable of passing easily through the capillaries, were employed, it returned by the opposite artery; but if a thicker fluid, incapable of so passing, were used, it did not.[5] It should be recollected, however, that Cohnheim's experiments were made on dogs, and that, in one of the many canine hearts he examined, he did find a small anastomosing branch. It will be seen, then, that on this question we have on each side a pretty equally balanced consensus of opinion; and, not so much with the hope of finally settling the question as with the object of possibly finding out which side was most likely to be right, I undertook a few injection experiments of the human heart, of which the following is an account. I made in all three careful arterial injections of healthy human hearts, after which they were examined and dissected, and the conditions found were noted at length at the time of experiment or of examination.

Injection 1.—This experiment was carried out on Jan. 17th, 1885; the fluid used was the ordinary histological carmine gelatine mixture, the continuous pressure apparatus being employed, and a maximum pressure of from eight to nine centimetres of mercury was used. The vessels before injection were washed out with salt solution; the mass was thrown into the left coronary artery, and after a short time it flowed from the right coronary in an oozing fashion. The tissue of the left ventricle and auricle was fully injected, but that of the septum and right side scarcely at all. By tracing out some of the branches it became obvious that the main point of communication between the two vessels had been in the apical region.

Injection 2.—In this case an exceedingly careful anatomical

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[4] On the Bloodvessels of the Nerves of the Heart. The London Medical Gazette, new series, vol. vii., 1848, p. 750.

[5] Virchow's Archiv, vol. lxxxv., p. 508.

injection of both arteries was made, in order that by subsequent examination and dissection any anastomosing branches might be discovered. In this and the succeeding injection, I had the valuable assistance of my friend Mr. A. E. Maylard, B.S. Lond., formerly of Guy's Hospital, and the method employed was that of Mr. H. G. Howse, described in Guy's Hospital Reports. The glycerine and arsenic preserving fluid was thrown into both vessels by the constant pressure apparatus at a pressure of eight centimetres of mercury, and it was particularly noted that there was no return by either artery during the process. The glycerine fluid was of the consistence of mucus. On the following day red lead mixture was thrown into both vessels by a powerful anatomical injection syringe. The vessels to their minutest ramifications filled out well, but there was no return by either artery. A careful dissection was made, and full notes of all the appearances observed were kept. Between the two coronary arteries three very fine and delicate anastomosing branches were discovered, all of them, however, so exceedingly small that a lens was required to trace them—viz.: (1) between the transverse branches on the posterior surface of the auricular ventricular sulcus there were two hair-like connecting vessels; (2) one hair-like vessel passed behind the aorta to connect the auricular branches; (3) a branch connecting the two vessels, which was verified by a lens, was found near the apex on the surface of the right ventricle. Between branches of the same artery only two very small anastomosing vessels were discovered, and both were in connexion with the left coronary. Not one of these connecting branches deserves to be considered as much more than a capillary branch, and had there been any larger communicating vessels it is not easy to see how they could have escaped detection.

Injection 3.—Both vessels were filled with glycerine solution under a pressure of from five to eight centimetres of mercury. The red paint solution was then thrown into the right coronary; the vessel filled out to its smallest branch. A careful dissection failed to find any communicating branch, although there must have been one or two small anastomoses, as in one of the descending branches of the left coronary a minute fragment of the red injection mass was found; and at the time of the glycerine injection one branch of the right

coronary was found to be slightly filled while the left was being injected.

I think, therefore, it must be admitted that inoscultations do exist between the coronary arteries of the heart, but that these are anything like so large and numerous or of so much importance in maintaining the vitality of the heart when a branch is plugged, as one would gather from the statements of Wickham Legg and West, is, I think, much to be doubted. So far as my experience goes, I would say that the arterial inosculations are so trifling and so far apart from one another that they are quite unable to compensate for vascular obstructions, except in their immediate neighbourhood, and even there only after very considerable destruction of cardiac tissue has occurred. To be of any service in the way of restoring circulation after obstruction the anastomoses would need to take place through the medium of primary and secondary branches, and Struthers has pointed out that Hyrtl only said that the coronary arteries did not anastomose by such branches, a statement with which I perfectly agree. In two of the cases of obstruction of the coronary arteries to be afterwards recorded an injection of soluble Prussian blue was made by Dr. Joseph Coats in order to throw light upon the question of anastomosis. It was seen that the communication between the two vessels was pretty free, but, taken by itself and in the absence of any careful dissection, such an experiment would only prove a capillary, or, if we may so speak, an *arteriolar* anastomosis, the existence of which, I think, must be admitted, but the service of which in compensating for any serious obstruction may fairly enough be doubted.

LESIONS OF THE HEART-WALL, WHICH MAY BE ASSOCIATED WITH DISEASE OF THE CORONARY ARTERIES.

The lesions of the cardiac muscle which are most likely to result from obstructed coronary circulation are—(1) *Fibroid Degeneration;* (2) *Fatty Degeneration;* (3) *Infarction of the Heart*, associated not uncommonly with spontaneous rupture of the ventricular wall. The lesions have been named in what I think to be the order of their frequency and importance, but for certain etiological reasons, which will become apparent afterwards, I prefer, in considering them

in detail, to place Fatty Degeneration last. These morbid states, though very frequently occurring quite independently of each other, may be combined in the same heart, as is illustrated by some of the cases to be afterwards recorded.

Fibroid Degeneration.

In the course of the critical review which I have given of the literature of this affection it has been shown that in the minds of most English observers fibroid degeneration and chronic inflammatory change of the myocardium are regarded as practically synonymous terms (at least so it is stated by Bramwell). That this is not so, it is, as I have already said one of the purposes of these lectures to prove. In a very large number of cases fibroid change is associated with disease of the coronary arteries of the heart, and if this be so then we have to deal with an affection of the myocardium, which may logically be placed in a category *per se*, and which clinically and pathologically has as much right to be regarded as nosologically a separate and distinct affection of the heart as, let us say, fatty heart. We have, I believe, in fibroid degeneration of the heart a morbid condition, the possibility of the presence of which should always be borne in mind by the careful clinical observer as a frequent source of cardiac weakness, and even of fatal syncope.

Fibroid degeneration is generally found in the wall of the left ventricle, and the extent of the affection may vary within very wide limits. Thus in some cases we may not find more than a patch or two the size of a split-pea or an almond, or, on the contrary, we may find the entire thickness of a very considerable area of the ventricular wall converted into a dense white fibrous tissue, as is perhaps most strikingly seen in cases of aneurysm of the heart. It is not at all infrequent to find the musculi papillares converted into tough fibrous tissue, and in some cases this may be the starting-point of the disease as regards other regions of the myocardium. In cases where the muscular tissue is extensively changed, as in cardiac aneurysm or fibroid disease affecting the entire thickness of the ventricular wall (see Cases 9 and 10 of the series to be afterwards given), there is not the slightest difficulty in at once recognising the morbid state.

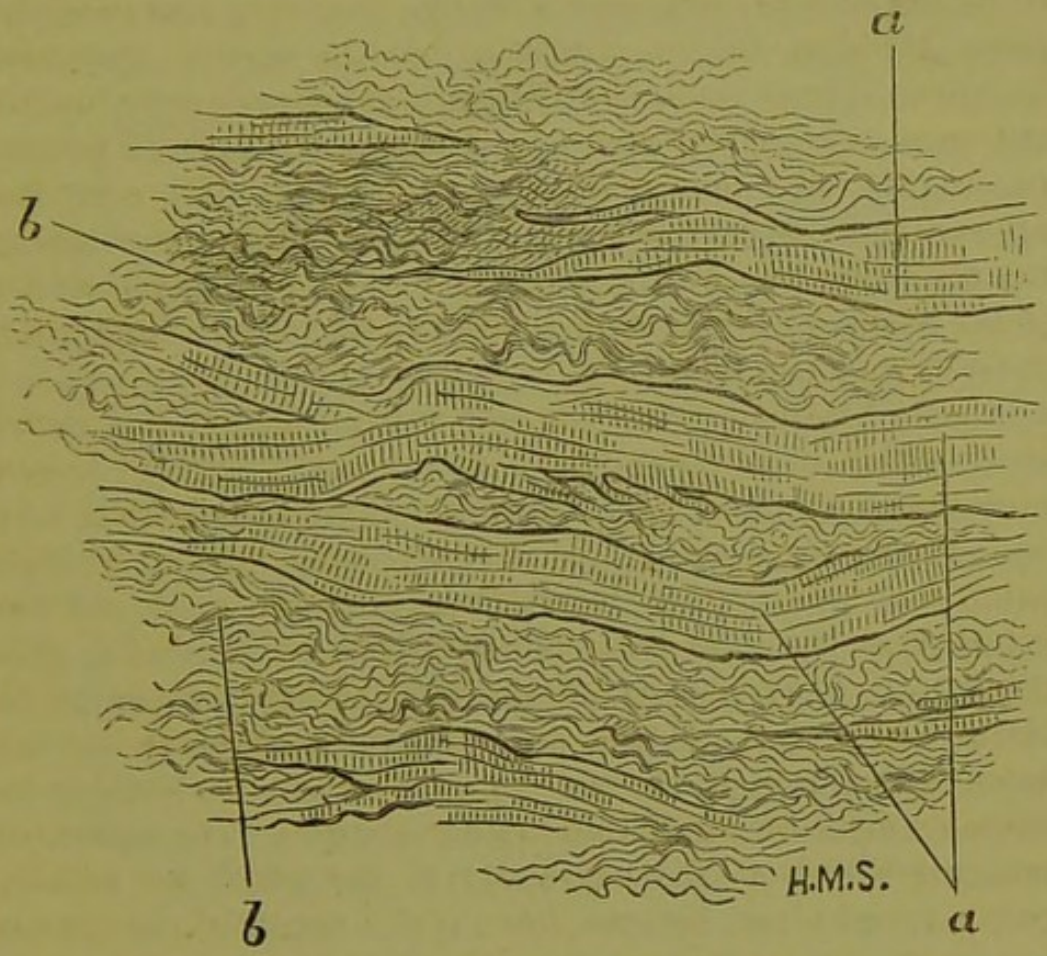
In a very large number of the slighter cases, however, unless the lesion is carefully searched for it is almost certain to be overlooked, and under these circumstances it is only after slicing through the muscle in sections parallel to the surface that the morbid state can be detected. When thus present and discovered in the substance of the muscular tissue, fibroid degeneration presents the following naked-eye characters, which are typical and easily recognised. It occurs as a bright, white, glistening patch of irregular size and shape, and of tendinous character; the cut surface of the patch is usually depressed below the level of the surrounding muscle, and has a streaky, fibrous appearance, the fibres running in the direction of the normal muscular bundles. There is no sharply defined line of demarcation between the healthy and the diseased tissue, the normal passing into the abnormal quite irregularly. These are the appearances as described by Huber, by whom, at Leipzig, my attention was first drawn to the subject, and they are those which I have always seen in cases which I have since examined in this country.

When examined under the microscope the patch is found to be entirely composed of delicate wavy fibrous tissue, often presenting a very finely granular appearance. In well-marked cases—i.e., of fibroid degeneration as distinguished from chronic interstitial myocarditis—no cellular infiltration is to be seen, nor anything in the least suggestive of inflammatory action. The margin of the patch is quite irregular, and the adjoining muscular bundles are more or less broken up and separated from one another by encroachment of the fibroid metamorphosis. The separated muscular fibres at the extremity of the patch are usually pointed, and often become finer and finer until the fibre is lost in the fibroid tissue. Sometimes the muscle-cell seems to be prolonged into a wavy fibre, an appearance very suggestive of the gradual wasting of true muscle substance, only the connective tissue remaining to form part of the morbid area. It is also frequently seen that the muscular fibre itself has a wavy character very similar to that presented by the strands of fibrous tissue. In the midst of the patches isolated and much atrophied muscular fibres are frequently seen, and all the appearances evidently point to a gradual loss of true muscle substance, and to its place being

taken by the wavy fibrous tissue. (See Fig. 1.) Minute arterial branches may often be made out in or in the neighbourhood of the patches, and these often give evidence of not being strictly normal, the chief abnormalities observed being a very decided hypertrophy of the muscular coat, and sometimes perhaps a slight thickening of the internal.

Etiological considerations. — In the foregoing remarks enough has been said to indicate pretty clearly my decided opinion that in true fibroid disease (perhaps it might be

FIG. 1.



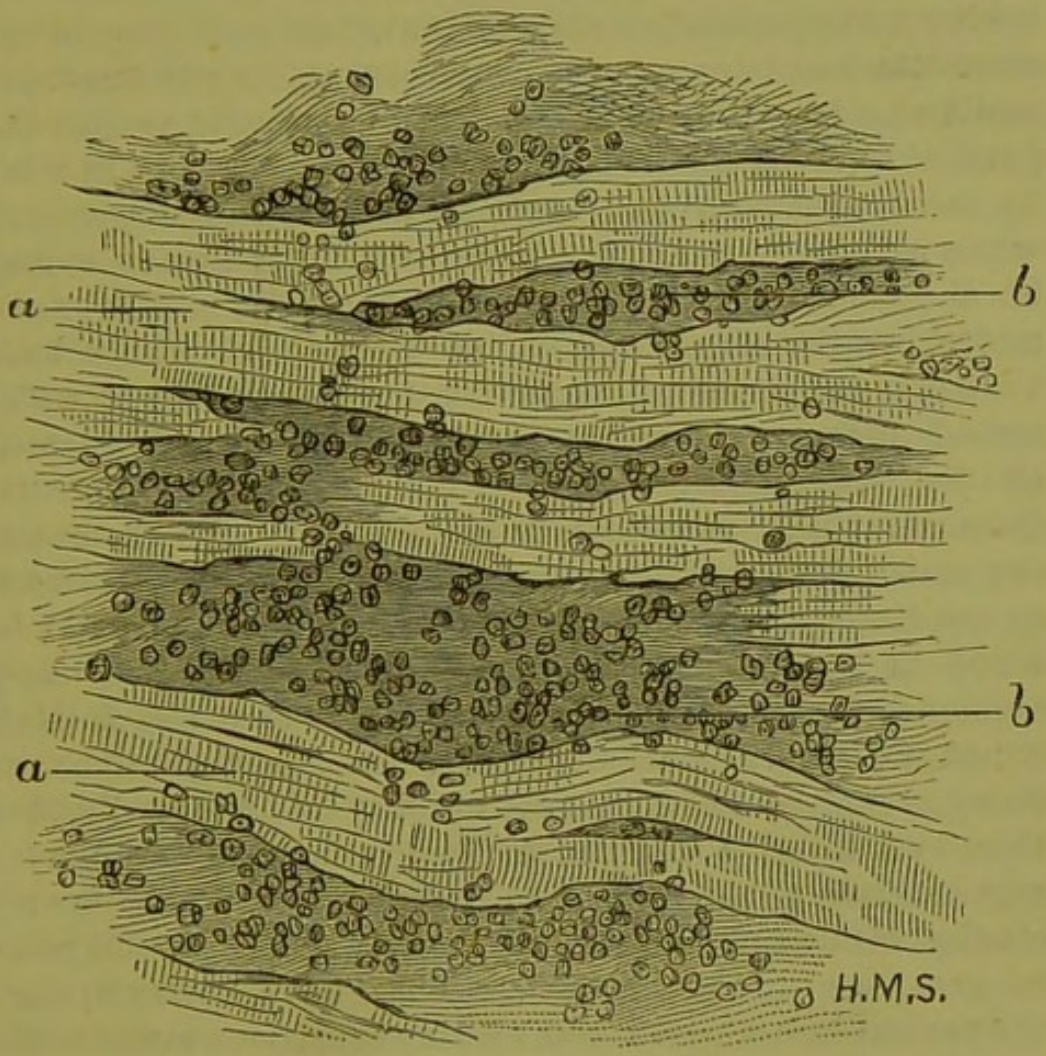
Showing the microscopical characters observed near the margin of a patch of fibroid degeneration. (Zeiss D. Eye-piece No. 3. × 350.) The drawing was made from a section taken from Case 3. *a*, Muscular fibres. *b*, Wavy fibrous tissue.

more correctly termed, as I have already pointed out, fibrous transformation) of the heart we have to deal with an atrophic process going on in the affected areas, and that this atrophy is directly to be related, in the great majority of cases, to a diseased condition of, and consequent insufficient

nutrient supply from, the coronary arteries. Of the very frequent association of fibroid degeneration with diseased or obstructed coronary arteries, the cases given by Huber, and also those to be afterwards recorded in the appendix, leave, I think, no very reasonable doubt. There is also but little difficulty in understanding the relationship existing between the two conditions and in tracing the sequence of events. Owing to the gradual obliteration of a particular branch of the diseased artery, and to the difficulty of establishing collateral circulation, a certain area of muscular tissue is slowly deprived of its necessary blood supply. Under these circumstances the connective tissue, requiring less nourishment and more tenacious of life than the muscle substance, is able to subsist on the diminished nutrient supply, whilst the more delicately and highly organised muscle readily succumbs. The muscular fibres slowly atrophy and disappear, and a fibroid patch is left. The patchy character of the disease is due, I believe, to the fact that all the branches of the coronary artery are not equally affected or obstructed. Fibroid degeneration is thus, in the first instance at least, always a localised affection, and never involves the entire myocardium simultaneously, although neighbouring patches may coalesce, and so give rise to extensive areas. It may be associated with fatty degeneration, which, though patchy or mottled in distribution, is generalised and dependent, as I shall endeavour to show, on a general as distinguished from a local deprivation of blood supply. In cases where the arteries are apparently healthy to the naked eye, abnormalities may be discovered with the microscope (see Cases 2 and 3); and in cases where the arteries are actually healthy, branches may be obstructed by emboli, and give rise to the affection in the manner soon to be adverted to (see Cases 4 and 5). Fibroid degeneration of the heart is, I believe, most frequently caused in the manner already described; but there can be no doubt that in a certain proportion of cases we may have fibrous patches in the myocardium which are inflammatory in origin—i.e., we may have a true interstitial myocarditis as opposed to a true atrophic fibroid degeneration.. Case 10 seems to be an illustration of this form of disease. In many cases associated with pericardial adhesions the origin of the affection is inflammatory, and probably an ex-

tension from the inflamed serous membrane. In such cases numerous leucocytes are found mingled with the fibrous tissue and lying between the muscular fibres, a condition of matters which is hardly ever found in pure atrophic fibroid degeneration. In cases of syphilis, too, fibroid patches, which have probably resulted from the presence of gummata, have been found in the heart wall; and

FIG. 2.



Showing the microscopical characters observed at the margin of a patch of interstitial myocarditis. (Zeiss D. Eye-piece No. 3. × 350.) The drawing was made from a section taken from Case 10. *a*, Muscular fibres. *b*, Leucocytes and new fibrous tissue.

in Case 10 there was also evidence, especially in the condition of the liver, that the patient had suffered from that disease. In this regard a case of syphilitic disease of the heart reported by Mannino, which had been under the care

of L. Federici, is of some interest.[6] Under such circumstances, the development of fibrous tissue in the myocardium obeys the laws which regulate inflammatory new formations in general. To such cases only can the term "interstitial myocarditis" be logically applied. (See Fig. 2.)

Clinical suggestions.—With regard to the clinical aspects of fibroid degeneration of the heart it is much more difficult to speak in definite terms. There is no definite physical sign, so far as I know, by which we can diagnose its presence, and at best we can only surmise its existence by taking a comprehensive view of the whole pathology of the case. As has been seen, it constitutes a very distinct and easily recognised lesion of the cardiac wall, and as such the possibility of its occurrence should always be kept in mind by the careful physician; and this is all the more necessary when we reflect that, if the lesion is at all extensive, it may be a powerful factor in producing cardiac debility, and under certain circumstances, as Fagge and Huber have shown, it may lead to sudden death. In all cases of widespread disease of the arterial system (arterio-sclerosis of the Germans), of cardiac dilatation, and of valvular disease arising in middle life apart from the rheumatic diathesis, the possible influence of fibroid degeneration as one factor in the production of the symptoms should be borne in mind. The symptoms may probably be very similar to those usually ascribed to fatty heart, and I think it not at all unlikely that a number of cases likely to be set down as examples of this affection may be fibroid. Further than this I feel I cannot at present venture to go with regard to the clinical significance of this lesion, as so far my studies with regard to it have been pursued in the post-mortem room, and not at the bedside. I have, however, since the present investigation was undertaken, seen a considerable number of cases in hospital practice in which I strongly suspected the presence of fibroid degeneration, and, if clinical opportunities accumulate, these may perhaps at some future time be recorded in this connexion. With regard to the generalised form of the disease described by Dr. Charlewood Turner[7] I can at present say nothing, as I have hitherto had no experience of it, and no oppor-

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[6] Brit. Med. Jour., vol. i., 1882, p. 547. [7] Loc. cit.

tunity of examining it. There is only one symptom which should be specially mentioned in connexion with fibroid degeneration, and that is angina pectoris. The association between this and a morbid state of the coronary arteries has been pointed out long ago, and in many of my own cases angina pectoris was a prominent and most distressing symptom.

Infarction of the Heart Wall.

This is a lesion which is to be directly associated with a morbid condition of the coronary arteries, and which very frequently leads to spontaneous rupture of the ventricular wall. The naked-eye appearances of acute cardiac infarction (myomylacia cordis of Ziegler) are sufficiently striking. The area may vary very considerably in size, and the affected tissue is as a rule much softened. In the recent state, when cut into, it presents a brown colour, and its surface is depressed below the level of the surrounding tissue; in severe cases the affected part may be semi-fluid in consistence. The microscopic appearances have been described at length by several observers, and as I shall advert to them afterwards in briefly describing typical cases (see Cases 4 and 5), it is unnecessary to refer to them in detail in this place. The muscular tissue is more or less broken up, and in many places has undergone coagulation necrosis—the nuclei and transverse striæ having disappeared, and the fibres having assumed a glassy homogeneous appearance; there is often a dense infiltration of round cells, probably from inflammatory reaction; the neighbouring bloodvessels are liable to be engorged with blood; and red blood-corpuscles are usually discovered in large numbers lying in the interstices of the tissue.

Etiological and clinical considerations.—Acute softening of the myocardium is almost invariably caused by complete and sudden occlusion of the arterial branch supplying the affected area of the heart-wall. The size of the area and the liability to subsequent rupture are directly dependent upon the size of the obstructed branch, as is illustrated by the cases of rupture recorded in the appendix. As a general rule, cases of infarction leading to rupture are associated with extensive disease of the coronary arteries, where, in addition to the chronic deterioration of nutrient supply,

a sudden obliteration of the arterial trunk has occurred—e.g., by the sudden occurrence of thrombosis or embolism. But besides this very serious form of infarction there is also a variety of the affection (illustrated by Cases 4 and 5) in which the occluded vessels are small, and in which the resulting infarctions are also small and may be multiple; and it is necessary before going further that this form of lesion should be referred to at some length. Cases 4 and 5 are good examples of this condition, and a description of the appearances usually present will be given in describing them afterwards. Here also, just as in the slighter varieties of fibroid disease, the lesion, unless specially searched for, may be altogether overlooked, because there may be nothing either on the endocardial or pericardial surface to indicate its presence. In this condition the walls of the coronary arteries may be perfectly healthy, and probably the most frequent cause, as seen in Case 4, is severe endocarditis with abundant loose vegetations of the aortic curtains, giving rise to multiple embolism of the coronary branches. It was once believed that the mouths of the coronary arteries were closed by the aortic curtains flapping against them, and that the arteries were filled by the recoil of the blood after the systole was over and the curtains closed. We are, however, indebted to several observers for showing that the orifices of these vessels are open during the systole, and that the coronaries of the heart receive their blood-supply precisely like the other arteries of the body. Chief among these observers must be mentioned the names of Martin and Sedgwick,[8] and of Worm Müller and Sandborg.[9] There is thus not the slightest difficulty in understanding how under such circumstances embolism of the coronary arteries should be a not infrequent occurrence, and I think it not unlikely that we have in this morbid state a cause of cardiac debility and failure in severe aortic disease, and in other diseases of the heart as well, which has hitherto been overlooked on the part of clinical observers. Embolic lesions of this kind, small and multiple as they are, are not very likely ever to cause rupture of the organ; but it is quite possible to con-

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8 Journal of Physiology, 1880-82, vol. iii., p. 165.
9 Pflüger's Archiv, xxii., p. 408; also Medical Times and Gazette, vol. ii. 1880, p, 623.

ceive the likelihood of a large embolic occlusion suddenly leading to this fatal result. Such a lesion, too, might be suddenly fatal without rupture, and before any very serious change in naked-eye appearance of the myocardium had been produced; for Cohnheim[10] and others have shown, in the case of dogs, that occlusion of a large coronary branch leads to stoppage of the heart in from thirty seconds to a minute. Minute and multiple embolic lesions, however, may be regarded as intermediate between the slowly progressing and localised atrophy leading to fibroid degeneration, and the sudden suspension of vitality over a large area, terminating in sudden death or spontaneous rupture. In Case 4 I think I have direct evidence of the truth of this statement. In this case, in addition to the small, soft, brown infarctions, there were also numerous minute areas of fibroid degeneration—the explanation of this doubtless being that, after the degenerated muscular substance had been absorbed, a fibrous patch, practically a scar, remained behind. The clinical importance of a morbid process of this kind directly resulting from valvular disease of the heart can hardly, I think, be over-estimated. With regard to the subject of rupture of the heart and its association with obstruction of the coronary arteries it is unnecessary for me now to speak, because I have elsewhere fully discussed the question in all its bearings.[11] Among the cases to be recorded as illustrating the present remarks, three of spontaneous rupture, in which the dependence of the accident on an obstructed coronary branch was undoubted, will be found, and in connexion with these is described all that is necessary to be said with regard to the histology and pathological anatomy of the condition.

Fatty Degeneration.

Fatty degeneration of the heart may frequently be directly associated with a diseased condition of the coronary arteries, although, as is well known, there are very many other morbid states which may give rise to it. In the

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10 Ueber die Folgen der Kranzarterienverschliessung für das Herz, von Prof. Jul. Cohnheim und Dr. A. V. Schulthess-Rechberg. Virchow's Archiv, lxxxv., p. 503.

11 Glasg. Med. Jour., December, 1884.

present connexion it is well to bear in mind the distinction between fatty infiltration and fatty degeneration of the heart. In the former we have, practically, merely an exaggeration of the normal adipose tissue of the organ, in which the muscular bundles are pushed asunder, and caused to atrophy by the growth of the fatty tissue (fatty growth on the heart). In the latter we have a real conversion of the true muscle substance into fat, granular molecules of fat making their appearance in the muscle-cells, first of all in the neighbourhood of the nucleus, and ultimately filling up the whole cell. It is of the latter of the two conditions only that I am now speaking, as I have no evidence to show, nor have I seen it stated, that fatty infiltration of the heart is in any special way connected with disease of the coronary arteries. Fatty degeneration presents itself as yellow flecking or mottling, visible to the naked eye, on the endocardial surface, and generally most typically seen in the left ventricle. It is chiefly, if not solely, caused by any serious deterioration either in quantity or quality of the blood; thus it is almost a necessary concomitant of all profound anæmias, and, as showing the influence of deteriorated quality of the vital fluid, it is also found in connexion with certain forms of poisoning, a circumstance which has been frequently put to the test of experiment. This being so, one does not wonder that fatty degeneration should be frequently met with in cases of disease and obstruction of the coronary arteries, from the organ being thus deprived of its necessary nutrient supply. In several of the cases which form the basis of these remarks fatty degeneration was found along with the fibroid disease, but the fatty change was specially typical in the last two cases of the series—i.e., those in which a whole coronary artery was shut off from its blood supply by reason of complete obstruction at its point of origin. In Case 15 the generalised fatty degeneration was so pronounced as to produce a more than usually prominent naked-eye change; and there can be no doubt that the very serious interference with the coronary blood supply in these two cases was entirely to blame for the widespread fatty change in the muscular fibre. From what I have seen I would be inclined to formulate the following proposition, subject of course to the corroboration to be afforded by more extended observations—viz., in all cases

where the main coronary trunk is slowly obstructed, and at last completely occluded at its origin, a generalised fatty degeneration is likely to result, and the cause of this change is essentially similar to that operative in profound general anæmias or phosphorus poisoning—viz., a grave deterioration of the blood supply to the whole organ. When, however, the arteries, though diseased, are generally pervious, and only the smaller branches seriously obstructed, while slight fatty degeneration may be present, we are much more likely to have a localised patch of fibrous transformation resulting. The effect of sudden obstructions of the coronaries has already been considered in connexion with infarction of the heart. The amount of deprivation of nourishment which can be borne by the heart, if it be but slowly induced, is indeed wonderful. In two of my cases the patent artery was also very considerably narrowed, and Greenfield has recorded a case where both arteries were occluded, and in which he suggested that under such circumstances the heart might be partially nourished by imbibition.[12]

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[12] Med. Times and Gazette, vol. i. 1875, p. 297.

LECTURE III.

SUMMARY OF CONCLUSIONS.

THE somewhat exhaustive study which we have made of the varieties of heart disease described in the foregoing lectures naturally leads me to formulate the following conclusions, which I think will be fully borne out by the facts already passed in review:—1. That a very sharp distinction must always be drawn between true atrophic fibroid degeneration and interstitial myocarditis; that, though similar in their results, they are quite different in their mode of production, and that the former is a much more common lesion than the latter. 2. That we should not be satisfied with merely opening the heart and examining the endocardium and valves, as is too often the case at post-mortem examinations, but that in all cases the muscular tissue, by numerous incisions parallel to the surface, should be carefully investigated. 3. That the anastomosis of the coronary arteries is not so free as to be of any practical moment (especially when the vessels are diseased) in preventing the occurrence of fibroid degeneration or of infarction of the heart as a result of obstruction of their branches. 4. That the influence of a morbid state of the coronary arteries must be taken into account in considering all diseases of the heart, but especially those of the myocardium, and that no examination of the heart can be regarded as complete which does not include a careful investigation of the state of these vessels. 5. That, though admittedly difficult, the possibility of diagnosing fibroid degeneration and infarction of the heart should always be kept in sight, and that by carefully passing in review the whole symptomatology and pathology of a given case of cardiac disease, a correct opinion in this regard may probably be arrived at.

RECORD OF CASES OF FIBROID DEGENERATION, INFARCTION, AND FATTY DEGENERATION OF THE HEART ASSOCIATED WITH A DISEASED CONDITION OF THE CORONARY ARTERIES.

It now only remains for me to briefly record the cases which I have investigated, and upon the examination of which the opinions I have already expressed are mainly based. For the opportunity I have had of examining many of the following cases, and for liberty to make use of the pathological records in connexion with them, I have in this place to express my obligations to my friend Dr. Joseph Coats, lecturer on Pathology in the Western Infirmary of Glasgow.

CASE 1. *Slight fibroid and fatty degeneration of left ventricle, with atheroma of the coronary arteries.*—Duncan C——, aged fifty-two, was admitted to Dr. Anderson's wards of the Western Infirmary on Jan. 28th, 1885, complaining of a common cold of a week's duration, with a sense of uneasiness in the epigastrium, and several attacks of severe pain in that region. There was some tenderness and enlargement of the left lobe of the liver; and two days after his admission he was suddenly seized with severe pain of the nature of angina pectoris, and died in about ten minutes.

A post-mortem examination was made on Feb. 1st, 1885, of which the following is a summary: Flabby and dilated heart; passive hyperæmia of lungs and other organs. With regard to the heart, it was found to be somewhat enlarged, weighing 12¼ oz., and the right ventricle was considerably dilated. The cavities contained much fluid blood, and almost no clot; the mitral orifice admitted three fingers, the tricuspid six. The muscular tissue, especially that of the right ventricle, was pale, and to the touch it felt flabby. The valvular structures were normal. The internal coat of the aorta presented patches of fatty degeneration. On careful examination of the coronary arteries many small areas of atheroma were discovered, but the vessels were quite pervious. Throughout the muscular tissue of the left ventricle numerous small yellowish spots were discovered, and on its posterior wall near the apex there was a distinct fibrous area about the size of a threepenny-piece, with around it evidences of slighter degrees of the same thing.

There was also a generalised, but by no means extreme, fatty degeneration of the muscular tissue. The kidneys presented nothing remarkable. On microscopic examination of the fibrous area, it was found to consist of delicate wavy lines of connective tissue, with here and there a granular appearance as if nuclei were scattered through it. In the midst of the fibrous tissue remains of muscular fibres were present; these fibres became atrophied at their extremities, and were prolonged into delicate, wavy fibrillæ, which were ultimately lost in the surrounding connective tissue. The appearances were strikingly suggestive of an atrophy of the true muscle substance having taken place. On staining with picro-carmine, the affected patch stained of a deep-red colour.

Case 2. *Slight fibroid degeneration of left ventricle; coronary arteries pervious, but presenting evidence of hypertrophy of middle coat; granular kidneys.*—Alex. K——, aged fifty-three, a plumber, was treated in Dr. Joseph Coats' ward of the Western Infirmary. The clinical history was that of chronic Bright's disease, with two attacks of generalised œdema, one of which occurred during his residence in hospital, when fatal uræmic convulsions supervened.

The following is a summary of the post-mortem examination: Granular kidneys (atrophy of cortex); enlarged left ventricle of heart; dilatation of lateral ventricles of brain; œdema of membranes. The heart presented great hypertrophy of the left ventricle, and weighed 20 oz. The muscular tissue was of good colour and consistence. On careful examination a small area of fibroid change about the size of a split pea was discovered in the anterior wall of the left ventricle. The coronary arteries, so far as could be discovered by the naked eye, were quite healthy and pervious. A typical "soldier's spot" was present on the surface of the right ventricle, and it was also noted that the pericardium presented linear, opaque, white areas of thickening, which followed the course of, and seemed to surround, the branches of the coronary arteries. The fibroid patch and the pericardial thickenings were reserved for microscopic examination. The kidneys were extensively diseased; the cortex was much atrophied and infiltrated with round cells; uric acid infarctions were present in the pyramids. On microscopic examination the fibroid patch presented characters precisely similar to those observed in the last

case. In several of the sections minute arteries were observed, and these presented very obvious signs of disease. The middle coat was very greatly hypertrophied, but no sign of the so-called arterio-capillary fibrosis could be detected. In the thickened pericardium the arteries also showed distinct thickening of the middle coat, but no exaggeration of the external.

CASE 3. *Fibroid patch in left ventricle; widespread arteritis deformans; coronaries pervious, but hypertrophy of middle coat of arterioles.*—Mrs. M——, aged thirty-five, was admitted to Dr. Anderson's wards on Nov. 3rd, 1886, complaining of palpitation and dyspnœa. The symptoms had been present for eleven years, more or less. There was a double aortic murmur, with dulness over the manubrium sterni and purring tremor in the supra-sternal notch. Hæmoptysis occurred on several occasions, and dyspnœa was always distressing.

On post-mortem examination the case was found to be one of widespread arterio-sclerosis (arteritis deformans); but with the naked eye the coronary arteries were found to be flexible and healthy throughout, with not a trace of atheromatous deposit to be seen, even in the finer ramifications. The left ventricle was greatly hypertrophied, and in the apical region a very typical patch of fibroid change, about half-an-inch in diameter, was discovered. It was depressed, white, and glistening or tendinous in appearance. On microscopic examination the fibroid patch presented the characters already described. The arterioles, however, in and around the sclerosed area presented marked hypertrophy of the middle coat, and in some it was also thought that there was slight thickening of the internal coat as well.

CASE 4. *Multiple areas of fibroid disease and softening due to embolism of coronary arteries; extensive aortic disease.*—Mrs. S——, aged thirty-eight, was admitted to Dr. Anderson's wards on Feb. 6th, 1885, complaining of cough, dyspnœa, and profuse expectoration of about one month's duration. There was a history of bygone rheumatic pains in the joints. Six years previously she had suffered from similar symptoms after a confinement, and the fatal attack began after her last labour. There was passive congestion of all the organs, which continued till death, and well-marked signs of aortic disease.

The heart was enlarged, weighing 17 oz.; the hypertrophy was general, but was specially well marked in the left ventricle. The aortic valve was quite incompetent, and much disorganised—the curtains being greatly deformed and the seat of massive vegetations. In some places the segments of the valve were distinctly torn. Infarctions were present in the lungs, and other organs. On more minute examination of the heart, numerous small areas of fibroid change were found scattered throughout the substance of the wall of the left ventricle. Most of these areas had the typical shining and tendinous appearance, and were depressed beneath the level of the cut surface, and irregular in outline. At two places were observed more recent patches of a brownish or hæmorrhagic appearance, and distinctly soft in consistence. The characters observed in this case very decidedly suggested multiple embolism as the cause of the condition. On microscopic examination the distinctly fibroid areas presented features quite similar to those already described, but in the recent patches the histological characters were quite in keeping with the supposition that they had been caused by an embolic process—that is to say, in the recent areas the bundles of muscular fibres, while they preserved their bulk and general outline, had lost the opaque and striated appearance of the healthy bundles; the individual fibres looked as if they had run together more or less, and they had throughout a homogeneous, glistening, transparent character—coagulation necrosis. The bundles of coagulated muscular fibres were separated from one another, just as in the case of normal bundles, by strands of fibrous tissue. These appearances were observed in one of the patches which had been cut in transverse section, and on examination of another portion cut in longitudinal section precisely similar changes were made out. The fibres were evidently coagulated, and were in many places undergoing absorption. The absorbed fibres were replaced by very beautiful fibrous tissue, and so long as any necrosed tissue could be seen it presented the translucent, bright-yellow appearance already adverted to. The affected patch was quite irregular as regards its junction with the surrounding healthy tissue, and here and there isolated normal fibres might be seen in the midst of the necrosed area. At the margins of the areas in some of the sections numerous engorged veins were seen,

with slight exudation of round cells into the surrounding tissues. The muscular nuclei had disappeared from the coagulated fibres

This case seemed to me to be most important as showing the relationship existing between the acute infarction of the heart wall and the chronic fibroid change; and, clinically, it also points to a possible cause of rapid heart failure in cases of severe aortic disease.

Case 5. *Multiple areas of fibroid degeneration and softening, probably embolic; atheroma of coronary arteries.*—John C——, aged sixty-two, died in one of Dr. Tennent's wards on March 20th, 1885. He had been suffering from cough, spit, dyspnœa, and palpitation, with pain across the chest, and œdema of the ankles. On Feb. 24th there was complaint of pain and numbness in the left leg, with painful swelling of the left thigh. On Feb. 28th he was paralysed on the left side of the body and the right side of the face. On March 7th there was a second attack of facial paralysis, and on the 20th he died.

On examining the basal vessels of the brain, the right posterior cerebral was found to be cylindrical, being distended by a dark plug, which extended to its bifurcation, where the thrombus became rather pale. The obstructed vessel crossed over the right peduncle and the third nerve; and the peduncle was much softened. Embolic infarctions were also present in the kidneys and spleen. The heart was somewhat enlarged and dilated, weighing 13½ oz.; and there were thrombi in the apex of the left ventricle. The valvular structures presented opacities and occasional calcareous infiltration. On more particular examination, it was found that the organ presented patches of fibroid change, with a well-marked area of softening in the substance of the left ventricle near the apex: the morbid condition of the muscular tissue was in all probability due to embolism of the smaller branches of the coronary arteries. The area of softening at the apex of the left ventricle was about the size of a sixpence; and on being cut into was seen to be semifluid in consistence and brown in colour: the morbid area could be easily differentiated from the surrounding healthy tissue by being depressed below its level. About the middle of the posterior wall of the left ventricle and corresponding pretty nearly in

situation with the point of attachment of the musculi papillares of the posterior curtain of the mitral valve, there was an area about the size of a shilling of well-marked tendinous change, which was hard and tough on section, and which extended through the entire thickness of the ventricular wall. The coronary arteries were perhaps slightly dilated and tortuous, and presented here and there small patches of atheroma similar to those observed on the valvular curtains; but, as far as they could be traced, all the branches were pervious. The area on the posterior wall was in the region supplied by a terminal branch of the right coronary artery, that at the apex in the region of the descending branch of the left. Sections were made from several of the morbid areas in this heart, and on microscopic examination the appearances were found to be very similar to those described in connexion with Case 4—i.e., the softened areas presented well-marked signs of coagulation necrosis, and the fibroid patches showed the wavy strands of fibrous tissue. It was also noted that around the recent patches there was great dilatation with engorgement of the capillaries and veins; the arteries had thickened walls, were not engorged, and presented in their interiors a distinct fibrinous stroma, in the meshwork of which a few blood-corpuscles were entangled, this appearance being very suggestive of the presence of thrombosis in the smaller arterial branches. In addition the following features were also observed: (1) In some places a distinct cellular infiltration was observed, as if slight inflammatory reaction had been set up; (2) here and there large round cells not unlike compound granular corpuscles were seen, and it was thought that possibly these cells may have had some relationship to the muscular nuclei; and (3) in some sections, as well as the capillary engorgement, a distinct infiltration of the interstices of the tissues with red blood-corpuscles, a circumstance quite in keeping with the view that the condition was the result of embolic infarction, was noticed.

Case 6. *Extensive fibroid degeneration of left ventricle, with extreme calcareous disease of coronary arteries.*—Duncan G——, aged seventy-four, died in one of Dr. H. C. Cameron's wards on Feb. 25th, 1885. He had been admitted on Feb. 12th on account of difficulty of micturition, which had been

present in a more or less severe form for about eight years. On the 21st, on trying to get out of bed, he fell on the floor, and seemed to have lost the power of his right side to some extent; the left shoulder was also very painful. On the 24th breathing became stertorous; there seemed to be some loss of power in both arms and in the left cheek. In a few hours he died.

At the post-mortem, on examination of the head, no recent lesion could be discovered in the brain; but there was extreme atheroma of both middle cerebral arteries. The heart was considerably dilated, weighing 17¾ oz.; and on careful examination a most striking and extensive fibrous transformation of the muscular substance was found in the lower two-thirds of the posterior wall of the left ventricle. The greatest length of the patch was three inches and a half, and breadth one inch; its margin was irregular, and it stretched obliquely downwards from a point a little above the middle of the outer wall of the ventricle towards the apex. A calcareous plate causing constriction of the left coronary artery was discovered at its point of bifurcation into the descending and circumflex branch; the right coronary artery was also atheromatous.

It is interesting to note that at the time of the post-mortem examination this patch was not discovered; and it was only on more detailed investigation, after the case had been demonstrated by Dr. Joseph Coats to his pathological class, that I found it to be present. Its presence could not have been guessed by simple inspection either of the endocardial or pericardial surfaces.

CASE 7. *Extensive fibroid degeneration of the left ventricle, with extreme disease of the coronary arteries.*—Robert W——, aged sixty, a traveller, was admitted to Dr. Tennent's wards on Feb. 13th, 1885, suffering from dyspnœa, hæmoptysis, and slight œdema of both lower extremities. There were distinct evidences of passive congestion of the lungs, and well-marked ventricular-systolic aortic and ventricular-systolic mitral murmurs were made out on auscultation, as well as accentuation of the second sound at the base. During a former residence in the hospital, December 1884, he had suffered severely from angina pectoris, which also caused much distress during his last illness. He was also occasionally seized with severe attacks of dyspnœa, during which the

whole body became very cold. He died on May 22nd, 1885.

The following is a summary of the post-mortem examination: Great atheroma of aorta, with thrombosis; atheroma of the coronary arteries; fibroid transformation and dilatation of the ventricles of the heart; plugs in the pulmonary artery; infarction of the kidney. The heart was enormously enlarged, but the weight of the organ has not been recorded. The left ventricle was greatly distended, as was also the right, but to a less extent. In the right auricle and ventricle were several globular vegetations—a large one in the auricle yielding a brown juice when cut into. The first part of the aorta was in an extreme state of atheroma, with calcareous infiltration almost continuous. The wall of the left ventricle presented in various places a remarkable atrophy, accompanied by a fibrous transformation of its muscular substance. Both coronary arteries were extensively diseased. On more detailed examination the following facts with regard to the fibroid degeneration were made out. The left or posterior coronary artery presented well-marked atheroma and calcareous change, especially in its descending branch. Just beyond the origin of this branch the lumen of the vessel was very considerably narrowed (being converted into a rigid calcified tube of fully an inch in length); and on laying open the artery, an adherent reddish clot, which caused additional obstruction, was discovered. The distal branches of this vessel were also considerably narrowed and calcified. In the ventricular septum in the area supplied by this vessel there was a large patch of typical fibroid degeneration, two inches long by one broad. The glistening tendinous character of the patch was most striking towards the centre, whilst at the margin the fibrous metamorphosis insinuated itself quite irregularly between the healthy muscular fibres; the change caused distinct loss of bulk, and the cut surface of the morbid area was distinctly concave. Here and there the morbid surface presented a reddish streak, as if due to an engorged vessel or a hæmorrhagic extravasation. The relationship to the diseased vessel was in this case exceedingly well marked. In the posterior wall of the left ventricle a long, ill-defined, and diffused strand of similar change, passing downwards towards the apex from the insertion of the musculi papillares, was discovered. A

similar patch was also present in the anterior wall. The right coronary artery was also extensively diseased and much dilated, but it contained no adherent clot, although there was plenty of loose débris. There was also extensive fatty infiltration of the organ.

Case 8. *Slight fibroid disease of left ventricle, with generalised fatty degeneration; serious disease and obstruction of coronary arteries.*—James S ——, aged forty-nine, was admitted to Dr. Gairdner's wards on March 13th, 1885, and died on June 18th of the same year. In December, 1884, he began to suffer from pain across the breast on any exertion, accompanied by attacks of shortness of breath. It was found to be a case of aortic regurgitation, with dilatation of the arch (possibly aneurysm). There was hypertrophy of the left ventricle, and symptoms of well-marked angina pectoris, with apparent extension of neuralgic sensations into the right lower limb, where distinct evidences of regular gout were observed. Ventricular-systolic and ventricular-diastolic murmurs were heard over the aortic area; the vessels were tortuous, and there was dull percussion over the manubrium sterni. Latterly there was a considerable amount of blood in the expectoration, with marked orthopnœa and acceleration of the pulse and respiration. There was no history of previous serious illness, but the patient had been a drinker.

Summary of post-mortem: Dilatation and hypertrophy of the left ventricle, with contraction of the aortic curtains and extreme atheroma of the aorta; passive congestion of the lungs; granular kidneys; deposits of urate of soda in the metatarso-phalangeal joints. The heart was very much enlarged, weighing 27¼ oz., and the arch of the aorta was somewhat dilated. The tricuspid orifice passed five, and the mitral three, fingers. The aortic valve was quite incompetent, from its curtains being involved in the atheromatous disease. The aorta presented an extreme state of atheromatous change, and the muscular tissue of the heart was very pale, and presented considerable fatty degeneration of the left ventricle. The coronary arteries were pervious, but much diseased in several places. In the first part of the descending branch of the left coronary a calcareous plate of stony hardness was discovered; it was half an inch long by one-eighth of an inch broad, and it emitted a ringing

sound on being struck with the probe; the orifice of the vessel was somewhat rigid and slightly narrowed. The orifice of the right coronary artery was greatly contracted, so that only a very fine probe could be passed through it. Beyond the obstructed orifice there was great dilatation and considerable tortuosity of the vessel; in its interior there was broken-down blood clot, at places slightly adherent to the vessel wall. In the posterior wall of the left ventricle slight traces of fibroid change, extending over an area of about the size of a shilling or more, and showing the usual microscopic characters, presented themselves; in many places, too, the muscular fibres showed great granularity, obviously due to fatty degeneration. This slightly fibroid area, the only one discovered, was in the area of distribution of the terminal branch of the right coronary artery.

Case 9. *Fibroid degeneration of the left ventricle; pericardial adhesions; probably caused by embolism of the coronary artery.*—The specimen from this case is in the museum of the Western Infirmary of Glasgow; and the following is the account contained in the museum catalogue:—

"The extreme left lateral part of the left ventricle was found adherent to the pericardium over a limited area by somewhat elongated fibrous connexions. This part of the wall of the ventricle is converted into fibrous tissue, the muscular substance being almost completely replaced. This transformation extends through the entire wall, and affects a bulky papillary muscle, which is almost entirely fibrous. The affected portion of the ventricle is distinctly bulged outwards. Examination of the coronary arteries showed no obstruction, but the capillaries in the affected region are greatly diminished, as shown by injection of soluble Prussian blue. The heart as a whole is much enlarged, weighing 22 oz., and there is a deep groove between the two ventricles, producing a double apex. The aortic valve is considerably thickened, and the auriculo-ventricular orifices dilated. There was thrombosis in the veins, and pulmonary infarctions, &c.

"The origin of the fibrous transformation here is not perfectly clear, but it has probably originated in embolism of a small branch of the coronary artery, the circulation having been restored by anastomoses after the muscular tissue had already suffered softening. This is rendered the

more probable by the fact that there were old embolic lesions in the spleen and kidneys, and the endocarditis of the aortic valve afforded a source of embolism when it was in the acute stage."

CASE 10. *Extensive fibroid degeneration of left ventricle, with pericardial adhesions; slight atheroma of left coronary artery; the lesion in this case inflammatory in origin (interstitial myocarditis).*—Janet B——, aged thirty-eight, died in Dr. James Finlayson's ward on June 2nd, 1887. On admission there was œdema of the ankles and eyelids, with considerable ascites. The urine was scanty, and contained abundant albumen and numerous granular tube-casts, but no blood. There was tenderness on pressure over the liver. The heart sounds were normal, and there was dulness with moist râles at both bases posteriorly. The illness had been of seventeen months' duration, and had been marked throughout by weakness and frequent inclination to faint. Swelling of the abdomen and ankles first supervened in October, 1886. The appetite had all along been bad, and vomiting occurred at intervals.

Summary of post-mortem: Considerable ascites; spleen firm, enlarged, weighing 8 oz.; deep cicatricial furrows on surface of liver, which on section present fatty areas; hepatic tissue generally fatty; organ weighs 3 lb. 7½ oz.; kidneys small, with adherent capsules granular surfaces, and atrophied cortices; both lungs firmly, adherent and œdematous. The heart is not greatly enlarged; the mitral orifice passes three, and the tricuspid four, fingers. On the anterior surface of the left ventricle near the apex, over an area of about two inches in diameter, there are firm fibrous pericardial adhesions. The muscular tissue immediately beneath the adherent surface is converted into a pearly white succulent tissue, which involves almost its entire thickness. On slicing the muscular tissue, similar patches, less extensive and evidently more recent, are found in the external and posterior walls of the left ventricle. An examination of the coronary arteries shows that, as a general rule, they are healthy and pervious; in the descending branch of the left, however, there is a very distinct patch of atheromatous thickening, which causes considerable narrowing of the lumen of the vessel. With this exception, however, no abnormality was discovered in the arteries. The wall of the

aorta is practically healthy. A careful microscopic examination of this case proves it to have been distinctly inflammatory in origin. The new fibrous tissue was abundantly infiltrated with leucocytes, and at the margins of the patches the muscular fibres were separated by numerous leucocytes. This, then, seems to be an undoubted case of interstitial myocarditis, which has probably extended from the pericardium. Here and there the infiltration of leucocytes was very dense, obscuring the muscular tissue. Evidences of syphilis were present in the liver, and the unhealthy coronary artery should also be borne in mind in thinking of etiology.

CASE 11. *Slight fibroid degeneration with generalised fatty degeneration; atheromatous disease of the coronary arteries, with complete obstruction of the left at its origin.*—John Y——, aged forty-eight, died in Dr. Finlayson's ward on June 6th, 1887. He complained of a dull aching pain in the chest, which varied in severity, and which set in with an angina-like attack eighteen months before admission. When serving in India he had suffered severely from ague, and up till six years ago he had been a heavy drinker and smoker. The apex beat was displaced to the left of the nipple line, and could be felt in the sixth, seventh, and eighth interspaces. At the apex there was a loud blowing murmur with the first sound. There were signs of passive congestion.

Summary of post-mortem: Dilated heart with fibroid change; obstruction of one coronary artery; atheroma of aorta. The pericardium contained a small, and both pleural cavities a considerable, quantity of clear fluid. The heart was much enlarged, weighing 19 oz., the enlargement preponderating in the right ventricle. The aortic and pulmonary valves were competent. The left ventricle was considerably dilated, and its walls were thin; its endocardium was obviously thickened, and the muscular tissue was interspersed with tendinous patches. There was atheroma of the right coronary artery, and the orifice of the left was completely obliterated, while that of the right was also somewhat narrowed. The valvular curtains were not much thickened.

In this case the fibroid change was not extensive, and the patches were tolerably recent. On microscopic examination the usual replacement of muscular fibre by fibrous

tissue was seen. There was also a slight but generalised molecular fatty degeneration of the muscular fibres. The left coronary artery was quite shut off from the aorta, but its walls were not seriously diseased. The right coronary artery was considerably dilated, even to its terminal branches, which largely supplied the posterior wall of the left ventricle. No arterial anastomosis between right and left could be discovered by tracing out the vessels.

Case 12. *Rupture of left ventricle; serious disease of coronary arteries, with occlusion of the branches of the left passing towards the seat of rupture.*—Richard M——, aged sixty, a boat-hirer from Dunoon, was admitted to Ward 5 of the Western Infirmary, suffering from eczema of fifteen months' duration, affecting the face, neck, and body. At the time of his death, which occurred as follows, the disease of the skin, as the result of treatment, was almost completely cured. On the morning of Sept. 14th, 1884, he had gone as usual into the bath-room of the ward with his pot of ointment to anoint the skin. He was absent from the ward for about half-an-hour, during which time no one had seen him, and he was found lying quite dead at the foot of the bath-room stairs, his pot of ointment standing on the last step, where he had evidently laid it on feeling faint. He had been in his usual health up till the time of death, and had never at any time made any complaint to lead to an examination of the state of the heart.

On Sept. 16th I conducted a post-mortem examination, of which the following is a careful record:—External appearances: The body is well-nourished and muscular, and almost the entire skin is discoloured by a tarry preparation which had been applied for a somewhat generalised eczema. On the right temporal region and right side of the forehead are several contusions. Chest: The lungs are highly œdematous, but otherwise not abnormal. Heart: On opening the pericardium a large quantity of blood-red serum escapes, and on evacuating this a large mass of clot, weighing about three-quarters of a pound, is found moulded on the surface of the heart. At the free margin of the left ventricle, midway between the auriculo-ventricular septum and the apex, is found a linear ragged aperture, measuring fully an inch in length, and lying in the long axis of the ventricle. Beneath the pericardium surrounding this opening, and extending

towards the front of the organ, there is considerable hæmorrhage; and on the anterior aspect of the left ventricle, about an inch from the septum and an inch from the apex is a distinct depression of a cicatricial appearance. The ragged aperture, before mentioned, communicates with the interior of the ventricle by a much smaller opening situated to the left of and behind one of the large fleshy masses, to which several of the tendons of the mitral curtains are attached. On section, the muscular tissue of the left ventricle in the neighbourhood of the rupture, and underneath that portion of the surface the seat of subpericardial hæmorrhage, is discovered to be exceedingly disintegrated. The tissue is soft, and presents a yellowish colour; so advanced is this in some situations that a condition of liquefaction is present. In some places, too, distinct cavities or channels exist in the heart wall, from which a brownish-coloured juice may be expressed in small quantity. The valvular structures of the left side of the heart are normal. The left coronary artery is highly calcareous, and is more or less completely occluded by atheromatous deposits. About one inch from its origin, and at a point beneath which it divides into two pretty large branches, is a distinct aneurysmal dilatation filled with soft clot. The branches leading from the aneurysm pass to supply the diseased portion of the ventricular wall, and are exceedingly hard and practically occluded. The right coronary artery is much dilated and tortuous, and here and there has a rigid feeling, but is nowhere obstructed. The external fat of the organ is generally somewhat exaggerated in amount, and encroaches very largely upon the tissue of the right ventricle. The valves of the right side, with the exception of some dilatation of the tricuspid orifice, are not abnormal. The whole organ is very soft and flabby, and weighs 15¾ oz. There is slight, but not at all extreme, atheroma of the aorta. The other features of the post-mortem are unimportant, and it only remains to note the condition of the kidneys. They are somewhat enlarged, and weigh respectively 6 oz. and 7½ oz. The surface of both is distinctly granular and lobulated, the capsule adhering with a moderate degree of firmness; and in some situations the demarcation between pyramid and cortex is not very clear.

Portions of the wall of the left ventricle were hardened

first in alcohol and then in chromic acid, after which they were subjected to a very careful microscopic examination—the sections having been cut with Schanze's (of Leipzig) microtome. The first point that strikes one in looking at the sections is the interpolation, if one may so speak, amongst and between the muscular fibres of a granular tissue, which is in many places distinctly associated with the course of the bloodvessels, and which often interrupts the continuity of the muscular fibres and breaks them up. In very many parts of the section the transverse markings of the muscle are distinctly preserved, but in others they are lost, and the muscular substance has apparently become hyaline or glassy. Here and there a very slight granular fatty condition of the fibre is observed, but nowhere is this at all decided or extreme. In some sections the fatty growth on the surface of the heart seems rather increased, but in no situation does it extend deeply into the cardiac tissue. Staining of the sections in a 2 per cent. solution of gentian violet in water brings out the appearances just described much more distinctly. It is then well seen that the new formation of granulation tissue is situated immediately external to the fibres—in the perimysial tissue,—and this is observed both in transverse and longitudinal section of the fibres. Where the cell proliferation is very abundant the fibres are largely destroyed, and, in addition, what appear to be the nuclei of the muscular cells are seen to be actively proliferating, as if they also were partaking in the process. Where the cell proliferation is abundant, too, the cells seem to be lying in a homogeneous transparent matrix, which is coloured neither by the chromic acid nor the gentian violet. In one of the stained sections the relationship of the new growth to obstructed vessels is distinctly observed. I refer to the appearance presented by a large artery, whose internal coat was so thickened as to leave only a very small lumen, and immediately outside of which the changes I have described were very extensive. The effect of the growth on the myocardium seems to be to cause the fibres to atrophy, to split up into longitudinal fibrillæ, and to lose their transverse striæ. Under the high power the cells comprising the interstitial new growths are, for the most part, seen to be of the nature of leucocytes, but where the change is very advanced much larger cells are

observed, and a careful examination of stained specimens indicates that these are in all probability nuclei of the muscle cells.

Case 13. *Rupture of left ventricle; extensive calcareous disease of coronary arteries; occlusion of the branch of the left passing towards the rupture.*—The specimen in this case was sent to the Western Infirmary museum by Dr. Yellowlees, of Gartnavel Lunatic Asylum, and was obtained from the body of a woman seventy years of age, who had been long insane. She was apparently in her usual state of health till she sank suddenly on the floor shortly after the usual weekly warm bath, and died almost immediately.

There is a large irregular aperture of a generally oval shape, and an inch long in diameter, situated in the anterior wall of the left venticle close to the septum. The wall of the heart is somewhat bulged around it, and the edges of the aperture are everted. There is no valvular disease. A careful dissection of the coronary arteries shows that both are the seat of very extreme calcareous disease, and that a large branch of the left going towards the rupture is quite impervious, being plugged by a thrombus. Under the microscope very distinct fatty degeneration of the muscular fibre is observed. But this change is by no means extreme, being entirely confined to the centre of the cell in the neighbourhood of the nucleus, and is certainly not sufficient of itself to have caused a spontaneous rupture of the muscular tissue; with this exception, however, the changes observed in this case are very similar to those described in the account of the last case, and so need not be dwelt upon at any length. It may be mentioned, however, that the main point of difference between this case and the last seems to be that the affection is more chronic—i.e., the cellular infiltration is not quite so abundant, and the development of fibroid tissue somewhat greater. Essentially, however, the changes in the two cases are the same.

Case 14. *Rupture of the left ventricle; serious disease of the coronary arteries, with great obstruction of branch supplying ruptured area.*—The specimen in this case was sent to me for examination by my friend Dr. John Love, of the Royal Asylum, Gartnavel. It was obtained from a man aged seventy-nine, who had been several times a patient in the asylum, and who was again convalescent from an attack of

acute mania, so that he was to have been discharged in a few days. He was seized with severe præcordial pain in the morning while asleep in bed. When seen he was cold and blue; the pulse was rapid, feeble, and irregular; and the cardiac sounds were very distant and feeble. The first attack abated somewhat, but about 5 P.M. of the same day he had another, and died in an hour and a half.

At the necropsy the pericardium was found filled with thin bloody serum, while around the heart itself was a layer of clot. A small rupture was found in the anterior wall of the left ventricle close to the septum, and about two inches above the apex. The fatty envelope of the organ was much increased, and the wall of the right ventricle was very thin. The wall of the aorta presented almost no trace of disease, but both coronary arteries were very seriously affected. The descending branch of the left coronary was rigid in almost its whole extent. About an inch and a half above the level of the rupture several transverse thickenings were observed, which slightly narrowed the lumen of the vessel. At the level of the rupture the calibre of the vessel was very considerably diminished by the presence of a thick calcareous concretion about one inch in length, which could be shelled out from between the coats of the vessel. The right coronary was also exceedingly calcareous. No detailed or microscopic examination of the muscular tissue of the organ was possible, as very considerable decomposition had set in before I received it.

Case 15. *Extreme fatty and slight fibroid degeneration: complete occlusion of right and partial of left coronary artery: extreme atheroma of the aorta.*—Arthur B —, aged sixty-three, a carter, was admitted to Dr. Finlayson's ward on Nov. 24th, 1886, with attacks of pain across the front of the chest and shooting to the back and down both arms, of twelve months' duration. During the last ten weeks the symptoms were more severe, and were accompanied by considerable flatulence. The heart sounds and the cardiac area were normal; there was no cough or spit; the respiratory murmur was fairly good; the temperature was normal; and there was no albuminuria. He suffered from diarrhœa and melæna six years before his death, and from slight rheumatism which never laid him up.

Summary of post-mortem : Extreme atheroma of the aorta; complete occlusion of the orifice of the right coronary artery; partial obstruction of the left; thickening and incompetency of the aortic curtains; calcification of the middle coat of the femoral artery. It is unnecessary to record the report by Dr. Coats in detail, but I may simply refer to one or two of the more important points. There was the most advanced fatty degeneration of the heart muscle; and the musculi papillares presented very distinct tendinous transformation. On microscopic examination there is seen to be a very extensive fatty degeneration of the muscular fibres; in addition, where the fatty condition is less distinct, there is a clear translucent appearance presented by the muscular fibres, which in these places are devoid of transverse striæ. On testing the anastomoses with soluble Prussian blue, it is found that the pervious coronary artery communicates with the impervious somewhat freely. This is determined in the case of a branch situated close to the obstructed end; this branch is distributed to the right auricle, and it communicates freely with a branch of the other artery distributed to the left auricle. It is also determined that the branches distributed to the ventricles communicate.

Case 16. *Generalised fatty degeneration; occlusion of one coronary artery, with narrowing of the other; atheroma of the aorta.*—John C——, aged thirty-six, a dock labourer, was admitted on Nov. 22nd, 1886, to Dr. Tennent's wards, complaining of shortness of breath and cough. Six months before he had been seized with a weight in the chest on exertion and palpitation, which were soon followed by cough and spit, tinged with blood. He died suffering from extreme orthopnœa, with œdema and dropsy. There were distinct ventricular-systolic and ventricular-diastolic murmurs over the sternum, with displacement of the apex downwards and to the left.

Summary of post-mortem: Atheroma of the aorta; complete occlusion of one coronary artery, narrowing of the other; thickening and partial destruction of aortic curtains; general venous hyperæmia. The general facts of the necropsy were similar to those recorded in connexion with the last case. Injection with soluble Prussian blue showed

pretty free communications between the two arteries, and the fluid did not return by the veins. Communications were determined for the auricles and ventricles. The cardiac muscle presented the typical mottled appearance of fatty degeneration, especially that of the right ventricle.

The injection experiments described in connexion with the last two cases were carried out by Dr. Joseph Coats at the time of the post-mortem examination. Soluble Prussian blue is practically as thin as water; and hence such an experiment is liable to the objection I have previously urged, that with a thin fluid we can fill the other artery by means of the capillary or arteriolar (?) anastomoses. Therefore, unless the anastomosing branches can be actually traced with the naked eye, such an experiment does not refute Hyrtl's assertion that the coronary arteries do not anastomose by means of their primary and secondary branches.

In the foregoing lectures I have endeavoured to demonstrate the intimate etiological relationship existing between disease or obstruction of the coronary arteries, and certain lesions of the myocardium, especially fibroid degeneration, and to make it plain that the terms "chronic interstitial myocarditis" and "fibroid degeneration" (pure and simple), are not synonymous terms. I have also in the course of the lectures shown that multiple embolic lesions of the heart-wall may play a most important part (which should never be overlooked) in determining the clinical course of certain cases of valvular disease of the heart. Since the publication of my first lecture, Dr. Quain has kindly directed my attention to the articles on Connective Tissue Hypertrophy and Fibroid Disease of the Heart in the Dictionary of Medicine. The subject matter of the former article, which is by Dr. Quain himself, has scarcely come under consideration, as it refers to a general increase of the connective tissue of the whole organ; but in reading it I observe that the inflammatory theory stated in the Lumleian Lectures has been departed from. "This, and similar cases," he writes, "exhibit no appearance of chronic inflammatory action, and thus differ altogether from examples of that form of fibroid degeneration which is described under a separate heading (see Heart, Fibroid Disease of)." In the latter article by Dr. Mitchell Bruce, we have a most careful exposition of the

subject of fibroid disease. The atrophic nature of the lesion and its association, in some instances, with degenerative changes in the vessels are pointed out. The term "fibroid disease," however, is still regarded as synonymous with chronic myocarditis; and the inflammatory theory of the affection seems, on the whole, to predominate in the writer's mind.