formidable and more difficult to cure than the hiatus in the hard palate. It frequently happens, in successful operations for cleft of both hard and soft palate, that a small opening is left at the part where the two operations have met each other; but, in most of the cases which we have seen, the inconvenience produced by this has been so trifling that the patient has not been inclined to submit to the ordeal of a third operation for what is so easily remedied by mechanical means. Sometimes the opening will granulate up and heal spontaneously.

Original Communications.

THE PATHOLOGY, DIAGNOSIS, AND TREATMENT OF CARDIAC DISEASES.

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II.—PATHOLOGICAL ANATOMY OF PERICARDITIS: PERICARDIAL ADHESIONS: THEIR CONSEQUENCES.

It is most important that we should keep the fact already referred to constantly in view whilst studying the pathological anatomy of pericarditis, viz.: That pericarditis is not merely a local inflammation. Excepting in those rare cases in which it is the result of traumatic injury, or is excited by contiguous disease, pericarditis is to be regarded as one of the consequences of some exciting cause operating generally in the body.

The anatomical changes which represent the disease to us, it should be remembered, are not, essentially, the disease. What may be more truly called so, viz., the exciting cause of the local disorders, exists prior to them. The changes are but the consequences of the unseen, but pre-existing, causes which are working in the system. And these causes again, though pathology cannot in all cases clearly define their nature, are various; and they attack the vigorous and they attack the weak; and certain of them operate in particular at one period of life, and some at another; and they fall upon other organs and parts of the body besides the pericardium. The same cause, for instance, which produces the rheumatic pericarditis is doubtless that which produces the rheumatic joints, and which produces the endocarditis, and the pneumonia, and the pleurisy, which so often accompany pericarditis; and so again, the cause which, in disease of the kidney, excites inflammation of the pericardium, is the same which gives occasion to inflammation of other serous membranes and parts and organs of the body. To look upon pericarditis in such case as the disease, is surely much the same as if we were to designate typhus as a pneumonia or a cerebritis, whenever the lungs in the one case, or the brain in the other, happen to be the organs on which the essential element of these diseases has, in an especial manner, exerted its noxious influence. In pericarditis, certainly, as in the pneumonia and cerebritis of typhus, the local inflammation demands especial consideration; but in pericarditis, as in typhus, we must remember that the disease is general, and that, therefore, in the general character of the disease, and in the general bodily condition of the patient, we must still seek our main guides to direct us in the management of the local disease.

Pericarditis, anatomically considered, consists in an inflammation of the whole or a part of the serous membrane which lines the pericardial sac. The anatomical changes effected by the inflammation are the same as those which are observed during the progress of inflammation in other serous membranes of the body. We have to note the stages of congestion, of exudation, of new growth, of organisation, of absorption, and adhesion; or of degeneration of the products exuded.

In the early periods of the inflammation, the superficial vessels of the subserous tissue become visibly injected with blood, and form tuft-like arborescent patches, frequently having little ecchymoses scattered between them. The surface of the serous membrane, at the same time, loses its naturally smooth, shining, and semi-transparent appearance, in consequence of serum being infiltrated into it, and into the subserous cellular tissue. The membrane, in this condition, is softer than natural, and consequently is more easily torn, and more readily separated from its attachments.

As the inflammation progresses, exudations are poured out into the cavity of the sac, and pseudo-membranous growths form upon its surface. The exudations vary greatly, both in quantity and in quality. Sometimes only a few ounces, and sometimes several pints of fluid, are effused into the sac. The fluid has even been found in quantity so large as to distend the pericardium in a diagonal direction, from the sternal end of the first rib on the right side, down to the eighth rib on the left side, expanding the costal cartilages on either side of the sternum, and compressing backwards the lower lobes of both lungs. In one case which I lately observed, the distended pericardium reached twelve inches across the thorax, and contained three and a quarter pints of serum.

In quality the exudation varies much; it may be of a serous, a fibrinous, or a purulent nature, and is not unfrequently tinged with blood. It collects chiefly about the base of the heart and in front of it. The heart, being specifically heavier than the fluid, sinks beneath it when the patient lies in the recumbent position.

The new growth upon the surface of the membrane presents great varieties in appearance. At one time it is smooth and thin; or again, and as we generally observe it, uneven, flocculent, villous, or honeycombed. When very rough and villous over the heart, the organ takes on that appearance to which older authors gave the name of *cor villosum*, *hirsutum*, *hispidum*, *tomentosum*. The new growth, again, varies considerably in thickness and in colour; it is generally most developed on the convex surface, and about the base of the heart. Its variation in colour results from its being accidentally stained with blood effused from the newly formed blood-vessels, which at this period of their formation are very readily ruptured.

The character of the exudation depends much upon the nature of the formative impulse which presides over the inflammation, upon the constitution, the idiosyncrasy, and the condition of the health of the individual attacked by the pericarditis. It is especially influenced by the nature of the disease with which the pericarditis happens to be associated.

Purulent exudations commonly arise during the progress of severe disorders of a low type, as in pyæmia, tuberculosis, and small pox, puerperal fever, and scarlatina. They are also observed in connection with chronic diseases; but are comparatively not of frequent occurrence. For the most part they have a fatal issue; sometimes, however, they appear to undergo partial absorption, as we may suppose to have happened in those cases in which cheesy, calcareous, and fatty matters are found sacculated between the layers of new formed pericardial adhesions. I have known small abscesses, thus situated in the pericardium, mistaken for purulent deposits in the walls of the heart. The new growth—the so-called fibrinous deposit upon the serous membrane—is generally slight in such cases.

Exudations have a natural tendency to undergo certain changes, under the influence of that ever presiding force, the *vis medicatrix nature*; the portions being absorbed, and the fibrinous deposit becoming organised, and converted into areolar tissue. In the purulent sort, as we have seen, hard or softish, or tubercular and cretaceous matters, may be left in the pericardium after the serous portion of the fluid has been absorbed. Laennec and other authors have described cases where the whole pericardium has been thus converted into a so-called bony shell. Instances in which portions of the pericardium are hardened into calcareous plates are not unfrequently met with. In other kinds of exudation the fluid portions of them may be entirely absorbed, so that the opposed surfaces of the new growth—the false membrane—are brought into immediate contact.

Under such circumstances, the surfaces gradually unite, and by their cohesion a perfectly organised and vascular union is at last established between the surfaces of the pericardium. The union thus formed may be general or partial. When the union is partial or slight, the connecting areolar tissue is sometimes long and loose; but when a large extent of surfaces is united, the union is for the most part intimate. A new vascular connection is established between the vessels of the heart itself, and the vessels of the parietal layer of the pericardium—a fact worthy of observation in a therapeutical point of view.

Thus far, the description of pericarditis is the description of the inflammation of serous membranes generally; but there are certain peculiar facts incidental to pericarditis which must be referred to. The immediate and the secondary effects of the inflammation are exercised upon an organ whose uninterrupted action is essential to existence; and hence it is that the smallest pericarditic friction-sounds excite such deep attention.

The action of the heart is interfered with, during the course of the inflammation, in several different ways. We may suppose that, in the onset, the local irritation—the cause provoking the local inflammation—acts as a stimulant to the heart's contractions; and that both the nutrition and innervation of the organ are impaired. Then, after a period of unusual excitement, reaction sets in, and the over-wrought muscular tissue suffers depression and diminution of its vital force, and becomes, so to say, partially paralysed. The same result, this, as happens in the advanced stages of pleurisy and peritonitis, when the intercostal muscles and the muscular coat of the intestines have been subjected to the influence of contiguous inflammation.

But this is not the only way in which the heart's movements are impeded. When the fluid exudation is considerable, it necessarily acts as a mechanical impediment to the motion of the heart. The pressure of the exudation upon the surface of the heart, when the pericardium is much distended, is very considerable; and as the pressure is equally distributed over the organ must act with greater effect upon the auricles than upon the ventricles. The solid fibrinous masses spread over the pericardial surfaces likewise interfere with the heart's action, especially when collected in masses about its base. I have seen cases in which the heart looked as if it had been flattened by the pressure of the fluid on it; the auricles being thickly coated with false growths, and crumpled down, and hardly to be distinguished from the ventricles. I have also seen the aorta apparently indented, from a like cause, at one point.* The pressure thus exercised on the heart interferes with the respiration, as well as the circulation, by impeding the ready flow of blood into the auricles from the pulmonary veins and the systemic veins.

Hence it appears that the pericardial inflammation injures the action of the heart and its great vessels in two ways; by the mechanical pressure of its exudations; and through the vital changes effected in the contractility and elasticity of the heart and its vessels by the interference with their nutrition which the inflammation occasions; the changes being either temporary or permanent.

Pericardial Adhesions. One of the important results of pericarditis is adhesion of the opposed pericardial surfaces, through the medium of the new growths. These adhesions are formed by a gradual conversion of the solid exudation into a completely organised areolar tissue. Inflammation or exudation upon one part—the visceral layer, for instance—of a serous membrane, seems almost invariably to excite inflammation in the opposed part of the parietal layer. I know not how we can account for this fact, except by supposing that the friction of the roughened surface of the part originally inflamed excites, by its mechanical action, inflammation in the opposed portion of the serous membrane.

Consequences of Pericardial Adhesions. Different authors differ much respecting the effects produced on the heart through adhesions of its pericardial surfaces. Hypertrophy, hypertrophy with dilatation, dilatation, and atrophy, each of these has been set down as the ordinary consequence of pericardial adhesions. There can be no doubt that each of these abnormal states of the heart has been observed coincidently with such adhesions, and as a consequence of pericarditis, therefore; but that one particular state—viz., hypertrophy with dilatation—is their most ordinary consequence. The discrepancies of opinions of authors seem to me to be capable of ready explanation, when we take into consideration the several coincident facts which are, one or other, associated with pericarditis. In the first place, the inflammation may have left the muscular structure of the heart unimpaired; or it may have so interfered with its nutrition as to have occasioned atrophy or degeneration of that structure. Then, again, with pericarditis is very frequently associated endocarditis, and with endocarditis injuries of the heart's valves; and from such injuries naturally spring hypertrophy and dilatation. Then, again, the nature and extent of the adhesions will modify the results occasioned by them. We must also look at the length of time which the adhesions have existed, when considering their influence upon the organ; i.e., the effects which they may have produced.

A consideration of these particulars would lead us to anticipate the occurrence of different conditions of the heart, according as one or other of the influences here mentioned is brought most prominently into action. When the nutrition of the

heart has been seriously interfered with, the muscular structure may have, to a greater or less extent, been permanently damaged; and, in such case, we should reasonably expect to find the organ atrophied, or that its tissues had undergone degeneration. But when the muscular structure of the heart has not been permanently affected by the inflammation, then the impediment to the heart's free action resulting from the adhesions, or coexisting valvular disease, would probably produce hypertrophy and dilatation. Thus, in cases of adherent pericardium, atrophy and dilatation of the heart would follow its impaired nutrition and its muscular degeneration; and hypertrophy, or hypertrophy with dilatation, when its muscular structure remained sound. It is right to add, that cases of completely adherent pericardium are occasionally met with, in which the heart appears to be healthy, and to have perfectly performed its functions during life.

Relation of Pericarditis and Endocarditis. Endocarditis very frequently arises during the progress of pericarditis; sometimes it precedes, and sometimes seems to arise simultaneously with, the pericarditis. It is only, however, in the rheumatic form of pericarditis that we notice this connexion. In such case, the endocarditis must be regarded as the product of the same exciting cause of disease as that which occasioned the pericarditis.

We have already seen that pericarditis may be the occasion of fatty degeneration of the heart, through the injury which it has caused to the nutrition of the organ.

When the pericarditis is considerable, it usually extends by contiguity to the parts external to and around the pericardium, producing local pleuritis. In consequence of this exopericardial inflammation, adhesions form between the pericardium and the lungs and the thoracic walls. Through such adhesions, the heart becomes directly attached to the thoracic walls, and the lungs are prevented gliding over the pericardium during inspiration—an important fact in diagnosis.

The pericardial effusion not only impedes respiration (as already described) by its pressure upon the heart and the great vessels, but it also, when considerable, mechanically compresses the lungs, and particularly the left lung, which is sometimes in this matter rendered in great part useless. We shall have occasion hereafter to speak of the many other effects which result from the congestions of the different organs of the body produced by the impeded respiration and circulation.

White Patches on the Heart are very frequently met with. They appear to be of two kinds, consisting of a fibrinous exudation poured out *within* the tissue of the pericardium, and of a fibrinous exudation *upon* its surface. They were once thought to be purely of inflammatory origin; but there is great difficulty in this regarding them. They are probably only quite exceptionally the results of inflammation, being, in the great majority of instances, merely the exudation of "unhealthy plasma", as described by Dr. Handfield Jones. In consequence of their usual position on the anterior part of the right ventricle, and on the right auricle, and sometimes on the posterior part of the heart, about the base of it, and over the coronary vessels, they have been attributed by some writers to the effects of friction produced by the heart's movements. "It is an appearance," said Dr. Baillie, "I believe, of no consequence whatever, and is so very common that it can hardly be considered as disease." This is exactly all that can be said at the present moment of these patches.

[To be continued.]

ACTION FOR RECOVERY OF FEES. An action was tried a few days ago in the Marylebone County Court, brought by Dr. A. B. Wall, of Bayswater, against Mr. T. B. Gordon, a retired wine-merchant. The plaintiff had attended the patient's wife, who had cut her throat; he made altogether sixty-three visits (sometimes seeing her twice a day), for which he charged five shillings a visit. The defence set up was, that the plaintiff was not entitled to charge for more than one visit a day; and that the plaintiff's treatment had been unsatisfactory. The judge decided that these grounds of defence were altogether set aside: the first, by the testimony of several medical witnesses; and the second, by the fact that the defendant had paid ten guineas into court. He was of opinion that in law a medical man could charge for every attendance; the public had their remedy, by first making a contract if they chose. The jury at once found a verdict for the full amount; and the judge allowed full costs, amounting to £5:4:0.

* Such a kind of pressure would probably excite an aortic bruit, it may be incidentally observed.