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THE EFFECTS OF RHEUMATIC FEVER ON THE HEART.

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EVER since it was first indicated by Bouillaud[1] and emphasised by Trousseau,[2] Graves,[3] and Stokes,[4] the fact has been generally acknowledged that in acute rheumatism cardiac may precede articular lesions. This consideration naturally leads to the conclusion that the relations of rheumatism as regards the heart and the joints are similar in nature if different in degree. In other words, the cardiac effects of rheumatism are no more to be regarded as complications than the articular. The importance of this consideration cannot be overlooked, and it furnishes the cue to the modern methods of treatment.

In the following short paper upon the effects of rheumatism it is my intention to deal as briefly as possible with the production and nature of the cardiac lesions of rheumatism, after which the rest of the paper will be devoted to those methods of treatment by which the implication of the heart may best be

obviated or minimised. The rheumatic poison may attack one or other of the serous membranes, or the muscular tissue of the heart. The pericardial effects, somewhat dimly apprehended by Pitcairn[5] in 1788, were more clearly grasped by Dundas,[6] and distinctly understood by Wells[7] and Latham.[8] The endocardial lesions of rheumatism were suspected by Baillie[9] and described by Kreisig,[10] but to Bouillaud[11] is due the honour of clearly formulating the connection between endocarditis and rheumatism. The myocardial changes of acute rheumatism have only been clearly comprehended in modern times. Although the association of myocarditis with rheumatism has, for some time, been recognised, as indeed is stated in my own work,[12] yet the frequency of rheumatic myocarditis has only in recent years been recognised, chiefly through the work of Poynton[13] as regards acute myocarditis, and Fisher[14] in respect of the more chronic forms.

It is highly probable that all cases of acute and sub-acute cardiac diseases are of microbic origin, and all may therefore be termed infective. We have, until recently, had to admit that cases arising in the course of acute rheumatism have not been definitely proved to have their origin in microbic infection, but the admirable work of Poynton and Paine[15] goes a long way to prove such a connection. Microbes have been found in all forms of pericardial and endocardial inflammation, and a large number of different organisms have been described in this relation. It seems probable that the diplococcus described by the observers mentioned will soon be accorded a place amongst them.

Many interesting questions arise with reference to the manner in which the micro-organisms attack the heart. There are no difficulties in regard to the pericardium, seeing that it is a highly vascular membrane, or to the myocardium, which is almost like a sponge. It must be remembered, however, in dealing with the endocardium, that the valves differ in respect of their vascularity; while the great venous valves of the heart are freely supplied with blood vessels, the cusps guarding the arterial orifices are destitute of them, unless some previous lesion has led to their development. It therefore follows that endocarditis, when occurring primarily on the aortic cusps, must be due to poisons circulating in the blood flowing over the surface of these cusps, and not brought to them by the nutrient blood

vessels. An important consideration bearing upon this subject is that advanced by Washbourn.[16] The endothelial cells of the vascular system are really phagocytes, capable of englobing bacteria passing along in the circulation. Washbourn quotes the statement of Metchnikoff, that these endothelial cells in fish have their origin in the motile cells on the surface of the yolk sac; hence it is no wonder that they preserve some traces of their ancient activity. That this is true has been clearly proved as regards the blood vessels, and there can be no doubt that the cells of the serous membranes of the heart are endowed with similar functions. While exercising their protective influence in destroying bacteria, these endothelial cells, however, may suffer and give rise to pericarditis or endocarditis.

Much work has been done in order to ascertain how organisms can produce endocarditis. Klebs[17] announced his belief in the infection of the endocardium by organisms circulating in the blood and acting directly upon the endothelial surface, while Köster[18] expressed his opinion that the organisms were conveyed to the blood vessels of the membrane, where they acted as emboli. This view does not account for a primary affection of the arterial valves, which are destitute of blood vessels. Wyssokowitsch[19] found that wounding valves by means of a sterilised instrument did not give rise to endocarditis, while the injection of pyogenic organisms into a vein after wounding the cardiac valves gave rise to acute endocarditis. Ribbert[20] went beyond this observation, and showed that the introduction of such pyogenic organisms by means of a vein, without previous injury to the cusps, was a sufficient cause of endocarditis. Since the publication of his results they have been frequently verified by other observers.

The earliest anatomical change produced by acute rheumatism in the pericardium is cloudiness of the endothelium, through which shines a ruddy hue; there may also be arborescent blood vessels, and small extravasations may in addition be detected. On the surface the fibrinous deposit occurs, in some parts as a fine layer, in other regions heaped up like the sand of the shore left by the waves. A certain amount of fluid is, in most instances, also present. On microscopic examination, the fibrinous deposit is found to be composed of layers felted together like the structure of a schistose rock, and

between the strata are small round cells with leucocytes and hæmocytes. In many cases, besides, masses of organisms may be detected amongst the fibrinous web, and the myocardium may be invaded by the organisms and their results. It is probable that the earliest stage in such cases is an attempt on the part of the endothelial cells to enact a phagocytic rôle.

The terminations of acute pericarditis may be perfect resolution, but, in many instances, a thickening is left upon the epicardium, usually in patches, constituting the milk spots or maculæ tendineæ, while in a considerable number of instances some adhesion of the two pericardial surfaces takes place through organisation of the fibrinous exudation by a newly formed vascular system, derived both from pericardium and epicardium.

The first morbid appearance in the development of acute endocardial lesions is the invasion of the endothelial layers by the bacteria of the blood in the cardiac cavities, which, as has been already remarked, may be regarded as an example of phagocytic activity. Retrogressive changes occur in the cells thus invaded, leading to the deposition upon the affected surface of fibrin, corpuscles, and platelets. In this way the earliest phases of vegetations are brought about, but proliferation of the cells of the subendothelial layer also takes place, although somewhat later; while leucocytes from the neighbouring blood vessels pass into the affected tissues as well as into the thrombus. As already stated, there is considerable difference in degree between such changes in the venous and arterial valves of the heart. It is probable that such effects occur more or less in all instances of acute and subacute endocarditis, but in the former the cellular infiltration leads to disintegration and abscess or ulceration. In the latter, the newly-formed tissues become the seat of a formation of new blood vessels, and their ultimate result is a conversion into granulation tissue. The final result is simple fibrous tissue, which may, at length, show fatty or calcareous changes.

In acute myocarditis the alterations are of a somewhat variable kind, according to the cause of the lesion, but, speaking generally, in an early stage the tissues are thickened, softened, and deeply tinted. On microscopic examination in such a stage, the muscle fibres are large and swollen, the transverse striation is almost entirely obliterated, and the interstitial tissue contains

leucocytes, hæmocytes, and proliferating cells. At a later period the muscular tissue presents a paler appearance, and is even softer than at first. On examination with the microscope, the fibres are granular, and show dissolution into their component cells; the interstitial tissue may show much cellular invasion, or, on the other hand, reveal some newly-formed fibrous elements.

The more chronic forms of myocarditis result in some pallor in the colour of the heart, attended by a degree of hardness, while the microscopic appearances show atrophy of the fibres and increased interstitial tissue. The muscle cells are finely granular, or even distinctly fatty.

Starting with the conception, then, that cardiac lesions are part of the rheumatic infection, and knowing what results may be expected if the poison should attack the heart, we are in a position to consider how to prevent, if it be possible, or to lessen, if that alone be within our power, its effects. In attempting to do this, we must learn the lessons which nature teaches, studying her laws and trying to modify her processes in such a way as to lessen their untoward effects. Part of the lesions produced by rheumatism are defensive in their results. Newly formed material deposited upon the pericardial and endocardial surfaces is protective, as was long ago shown by Hilton[21] in his eloquent work:—"The result of the effusion of the inflammatory fluid is that the serous surfaces become defended immediately from direct and intimate surface-friction; thus, that possible source of irritation is removed. Thenceforward this acquired rest, or freedom from direct friction, enables the serous membrane to recover itself, and then to resume its important function of absorption." But, during the further processes which take place in the deposits, there is apt to be some adhesion or contraction by which the usefulness of the parts is impeded. Such a consideration leads to the reflection that, if it be possible, nature's own processes must be controlled.

The first and most important method of treatment, with a view to obviate rheumatic implication of the heart, is to enjoin rest in the highest degree available. It is clearly impossible to ensure absolute rest, however, to the pericardium, the endocardium, or the myocardium, seeing that the heart works steadily day and night; nevertheless, by securing complete bodily rest, the amount of work which the heart is compelled to perform

is reduced to a minimum. By the absence of all muscular exertion both the force and the frequency of the action of the heart may be greatly reduced. In this way the friction between the inflamed pericardial surfaces, the shock of the closure of the valvular cusps, and the strain upon the muscular fibres, may be enormously reduced. From the first moment, therefore, of an attack of acute rheumatism until some days after every symptom has finally disappeared, the patient should be compelled to retain a horizontal position.

Closely related to the question of rest is that of diet. While sufficiently nutritive to supply the demands of the diminished tissue changes which occur during rest, it should be of such a nature as not to produce very stimulating effects, or to introduce into the system too much animal proteid material. It must, moreover, contain abundance of fluid, so as to bathe the tissues thoroughly. During the earlier periods of acute rheumatism the best form of diet is accordingly composed mostly of milk. But, as time goes on, the nature of the diet may be judiciously extended by the introduction of soups, farinaceous foods, and the lighter forms of flesh.

With regard to the question of drugs, the specific remedies for acute rheumatism will naturally be in operation from the earliest moment. We are, at present, unable to answer the question whether salicin and its allies in themselves have any influence in lessening the tendency to cardiac implication. The results of statistics, unless spread over a lengthy period, and distributed over a wide field, are apt to be fallacious. There appears, however, to be good ground for the belief that the salicyl series of drugs considerably diminishes the cardiac effects of rheumatism. These drugs must be continued in full doses until every symptom has disappeared—until the pulse and temperature have become normal, and every appearance of joint affection has subsided. After that state of matters has been reached, it is well still to continue the specific remedies for some time. When all the general symptoms have disappeared for a few days, absorbents may be begun. Of these, the one which seems to me most satisfactory is iodide of sodium. It may be continued for some weeks, giving from 10 to 15 grains three times a day. If there should be any appearance of anæmia, the iodide of sodium may be combined with iodide of iron, or, at

the same time, some iron preparation may be administered with vegetable tonics. During all this period, no remedy that can stimulate the heart—such as digitalis or strophanthus—should be administered, the whole aim being to maintain the heart's action at a low level. After the disappearance, however, of every symptom, general or local, the employment of the cardiac tonics may be commenced. Throughout the whole course of the disease an occasional mercurial aperient should be administered every few days.

One other method of treatment remains for consideration, and one which, notwithstanding the recent expression of opinion by Mitchell Bruce,[24] seems to me of the highest value. This is counter-irritation. There are many difficulties in attempting to explain how counter-irritation can influence the course of disease in internal organs, but we may assume that the effect of the external stimulus causes some contraction of the afferent vessels, as suggested by Lauder Brunton.[23] This, no doubt, explains the action of counter-irritation as regards the pericardium and myocardium. It is much more difficult to give any rational theory to account for its effects on certain parts of the endocardium. Some such reasoning, doubtless, is applicable to the endocardium covering the mitral cusps, in which blood vessels are present. But it is not applicable to that of the aortic cusps, seeing that in them there are no blood vessels, unless there has been some previous lesion leading to vascularisation. This may be the reason why it is so much more difficult to control aortic than mitral endocarditis. For a good many years it has been my custom to employ counter-irritation about the præcordia in every case of acute rheumatism, and the results have been eminently satisfactory. The method has consisted in the application of small fly-blisters every night, or every second night, over the præcordia and their neighbourhood, and I am able most thoroughly to agree with Caton[24] as regards their utility. It is a pleasure, as it seems a duty, to accord to this author a hearty expression of appreciation of his labours.

Such are the means by which nature's processes, as regards the heart in acute rheumatism, may be modified and controlled. To do this successfully demands great patience on the part of physician, nurse, and patient. A case of the disease at present in my ward furnishes an excellent example of this fact. The

patient, who had never previously suffered from this or any other serious disease, was admitted at the outset of an acute rheumatic attack. He has been treated sedulously in accordance with the principles laid down, and yet, with every precaution, he has twice been threatened with relapse. Notwithstanding this tendency, however, he has escaped any cardiac implication other than some slight mitral and tricuspid regurgitation due to muscular relaxation, and even this is now passing away.

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