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ON THE

RARITY OF PERICARDIAL ADHESION

IN COMPARISON WITH THE

FREQUENCY OF PERICARDITIS.

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Old deposits of lymph, of whatever size or amount, and whether consisting of false membranes or organised adhesions, when found in the pleural or peritoneal cavities, have long been ascribed to their right cause, namely, a by-gone attack of inflammation. But when occurring in the pericardium it has been customary, until very recently, to refer to an inflammatory origin the adhesions only, not the false membranes, or *white spots*, as they have been termed: these latter have, by some writers, been viewed as inexplicable in their origin, while others have hesitatingly attributed them to some partial or inflammatory process. By the satisfactory proofs of their inflammatory origin furnished by Mr. Paget, in the 23d volume of the Medico-Chirurgical Transactions, all doubt on the subject seems, however, to have been removed. I need not, therefore, recapitulate the various points of evidence by which this correct view of their nature has been established; but, assuming as an admitted fact that the deposits are analogous to other false membranes, and therefore have their origin in lymph effused during an inflammatory process, I will endeavour to show that such inflammatory process, instead of being either partial in extent, or chronic in degree or duration, as has been supposed, may, at least in many cases, have possessed all the characters of an acute and general attack of pericarditis.

I found this opinion almost entirely on the varieties in *number*, *size*, and *thickness*, presented by the deposits: for these varieties seem to furnish a satisfactory clue to the changes which in course of time the deposits undergo. The simplest form in which we meet with traces of old lymph on the pericardium is that of opaque white lines on the surface of the heart, usually following the course of the coronary vessels, and their principal divisions: co-existent with these, or even independent of them, we sometimes notice fringes of old lymph on and about the borders of the right auricle, also several small, firm, slightly-elevated granules, rather firmly adherent to the roots of the great vessels at the base. It may seem that such trivial changes as these are scarcely worthy of notice, and perhaps we should not be justified in ascribing to them any relation to a previous inflammatory process, were it not that in other cases they are frequently accompanied, as shown by Mr. Paget, by fine bands or threads of old adhesion between the great vessels, and by various sized patches of false membrane on different parts of the heart.

The patches of false membrane, or white spots, present, as already stated, considerable diversity in different cases. Sometimes we see one or two pale or whitish deposits on the anterior surface of the heart, thin, smooth, and of no great breadth, and firmly ad-

herent to the tissue on which they are placed. In other cases they are more in number, perhaps broader, often thicker, and even separable into laminæ, and now, perhaps, no longer confined to one part of the heart, but scattered irregularly over the upper portions of the organ. They may still be smooth and polished on the surface, though not unfrequently they are rough and uneven, occasionally soft and villous, or even presenting a shaggy tufted appearance: in the latter case a band of adhesion may sometimes be found connecting the mass of lymph with the opposite point of the parietal pericardium; but this is rare. In other cases, again, still more extensive deposits may be found, nearly the whole surface of the heart being not unfrequently coated over with irregular patches of whitish lymph, while considerable masses of similar deposit may be found at the base of the organ, accumulated in the spaces between the roots of the great vessels, to the walls of which they closely adhere.

Such, in outline, are the principal appearances presented by the deposits of old lymph on the heart,—appearances which seem to indicate that they have all originated from the same source, and that their varieties are due, not to diversities in their mode of origin, but partly to the different quantities in which the lymph was poured out, and partly also, most probably, to the different distances of time at which they are examined after the subsidence of the inflammatory attack. For, in regard to the latter point, when the amount of deposit is large, it seems not improbable that the period at which the inflammation giving rise to it existed was not far remote from that at which death ensued. Such supposition derives support from some of those not unfrequent cases in which, together with an abundant deposit of false membrane, the fluid in the pericardium is found turbid, and containing flakes of recent soft lymph, while scattered points of vascularity observed on various parts, especially about the base, favour still further the opinion that the pericardium had not long previously been the seat of inflammation. When, on the other hand, the false membranes are small in size and few in number, it does not seem necessary to conclude that the preceding inflammation had been small in degree, or limited in extent, or chronic in duration; for as well as being few and small they are generally also smooth and thin; appearances which seem to indicate gradual changes in the deposits, and lead us to think that a great length of time has probably elapsed since the inflammation giving rise to them ceased. For neither in the pericardium, nor in any other part of the body, does the amount of lymph found at a remote period bear any necessary proportion to the quantity originally effused in a by-gone attack of inflammation. There seems, indeed, to be scarcely a limit to the time within which the absorption of lymph, not yet organised, can take place. The process may extend over weeks, or months, and at length the inflammatory product be more or less completely removed. Instances of this are furnished in the slow and gradual, but often complete removal of lymph from the tissue of an iris or cornea which has been the seat of inflammation. Now, what occurs in other parts may surely occur in the pericardium; and there seems no other forcible argument against the belief that the deposits of lymph on the heart in the form of unorganised, non-vascalar false membranes, may, by the process of absorption, be gradually diminished in size, and in time perhaps ultimately removed.

Such being the principal evidence which seems to justify the conclusion that the white spots or patches found on the surface of the heart originate in a true attack of pericarditis, I proceed to notice some of the inferences naturally resulting from this conclusion. The principal of these seem to be—1st. That inflammation of the pericardium is a much more common disease than would be indicated by the infrequency of adhesion. 2dly. That the general opinion concerning adhesion of the pericardium as a consequence of pericarditis requires to be modified. 3dly. That the prognosis in cases of pericarditis is less unfavourable than it would be if adhesion invariably followed.

1. With respect to the first point, I may remark that hitherto it has been usual to regard as true pericarditis those cases only in which either distinct auscultatory evidence of the disease has been obtained during life, or, in fatal cases, anatomical changes resulting from recent inflammation, or more or

less complete obliteration of the sac of the pericardium by old adhesions, have been observed after death. Were pericarditis limited to cases such as these, it might indeed be considered a somewhat rare disease: but if we extend our range, and include (as we are justified in doing) among the anatomical proofs of its existence, not only deposits of *recent* lymph and organised adhesions, but also deposits of *old* lymph or false membranes, we are compelled to admit that it is a disease of not unusual occurrence. The opinion that the false membranes are the result of *partial* or *chronic*, rather than of *acute* and *general* inflammation, has very little evidence in its favour. There doubtless are *degrees* of inflammation in all parts, and the nature and quantity of the inflammatory products effused in any case are probably in great measure determined by the extent and intensity of the existing inflammation: so that when we see a part, such as the pericardium, immediately after it has passed through an inflammatory attack, we may form a moderately correct estimate of the intensity of the attack by the amount of the morbid products discovered. But if, as so much more commonly happens, we have no such opportunity of examining the part until long after the subsidence of the inflammation affecting it, we can, after such a lapse of time, form no correct opinion respecting the amount of mischief it underwent when inflamed. For, in the case of the pericardium, supposing adhesion to have taken place, most probably only a *portion* of the effused lymph has had part in the adhesive process, the remainder having been removed by absorption; while if no adhesion has ensued, then are we still less likely to obtain a correct knowledge of the intensity of the inflammatory process; for here also much of the effused lymph has doubtless been absorbed, while the remainder can indicate nothing more than that inflammation had at some period given rise to it. And had the pericardium been examined at an *earlier* period, probably more lymph would have been found; while if at a *later*, probably less, or even none at all, would have existed, owing to its more or less complete removal by absorption.

If pericarditis be so common as is here supposed, it may naturally be asked, why do we not more frequently witness the disease during life? The question, I admit, is rather difficult to answer, yet not so difficult as at first sight it appears. From the zeal with which auscultation is at the present day pursued, the disease is probably rarely overlooked in hospital practice; and the number of cases therefore in which it is detected perhaps fairly enough represents the proportional frequency of its occurrence, at least in the course of acute rheumatism. But do the records of hospital practice, or does the experience of any diligent attendant in the wards, for a twelvemonth, show that pericarditis is of unfrequent occurrence? Of 136 cases of acute rheumatism recorded by Dr. Latham,* the pericardium was inflamed at least 18 times—no inconsiderable number. But this analysis takes no account of cases of pneumonia, pleurisy, peritonitis, continued fever, scarlet fever, and the many other febrile and inflammatory diseases which are at all times sprinkled through the wards of a large hospital. It is true that acute rheumatism is, par excellence, the great source of pericarditis, yet abundant facts testify that the pericardium may become inflamed in the course of several other diseases besides this. The occurrence of this complication in continued fever has been repeatedly noticed; its supervention in the course of scarlet fever is a subject of still more frequent notice; while granular degeneration of the kidney is, next to acute rheumatism, perhaps the most fertile source of the disease. The tendency of serous membranes to participate in each other's diseases leads to the supposition that pericarditis may not unfrequently arise in cases of inflammation of the pleura or peritoneum, especially when the inflammation is due to a general rather than a local cause; while the analogy existing between all the exanthemata suggests that as the pericardium is apt to be inflamed in scarlet fever, so ought we more assiduously to watch the condition of this membrane in all other diseases of this class. Many facts show that pericarditis may arise without producing *any* symptoms referable to the heart; and I have no doubt the disease would sometimes be quite overlooked, even in acute rheumatism, were it not for the care and assiduity with which from day to day the cardiac region is

* Diseases of the Heart, vol. i. p. 143.

explored. Need we, then, be surprised if it be sometimes passed by in cases, where, from the nature of the disease, such as continued fever, we are not in the habit of paying that strict attention to the condition of the heart, which in other cases is found to be necessary to the detection of pericarditis?

But even granting the rarity of pericarditis in hospital practice, it must be remembered that of the patients under care in any public institution, the majority are seen for the first and only time in their lives: we know next to nothing of their former ailments; nothing whatever of what may happen to them hereafter. We know not how many may have had attacks of pericarditis in former illnesses, nor in how many it may come on ere they die. So that we should not hastily conclude about the frequency of the disease merely from what passes before us in hospital practice. The records of diseases of children might be expected to guide us to a sounder conclusion on the subject, but I have been unable to collect sufficient data for such purpose; for until recently this large field has been comparatively uncultivated, so that we can only hope to obtain hereafter the requisite amount of statistical information for this and other similar objects. One important fact, however, is established by the best writers on children's diseases—namely, that the false membranes on the heart are of not unusual occurrence even in very young subjects. Moreover, several fatal cases of recent acute inflammation of the pericardium are narrated by Dr. West, in his work on the Diseases of Children. Dr. West has also kindly informed me of a case which recently occurred in his practice, and in which there was abundant evidence of recent extensive inflammation of the pericardium, in a child only three months old, the heart being nearly covered by deposits of lymph.

II. If, then, it be true that pericarditis is a disease of not infrequent occurrence, the general opinion respecting the usual termination of this disease in the formation of permanent adhesion requires to be modified. Nearly all the highest authorities on this subject agree that inflammation of the pericardium, when proceeding far enough to cause effusion of lymph, terminates either in the death of the patient, or in the formation of adhesions whereby the cavity of the sac is more or less obliterated. In some of his expressions, Dr. Hope* certainly seems to imply that the effused lymph may in a few cases be absorbed, while in one important passage he remarks that "pericarditis sometimes leaves no other vestiges than opake white or milky spots, which," as he says further on, "possibly may be results of partial pericarditis, as supposed by Laennec; but there is no reason to suppose that they may not also be occasioned in some instances by universal pericarditis." (p. 151). The whole tenor of his previous and subsequent remarks on the subject, however, plainly indicates his belief that adhesion is by far the most common event of pericardial inflammation; indeed, that, next to resolution, it is "the most desirable termination which remains" (p. 148). It is difficult to ascertain the grounds on which such acute and experienced observers, as the late Dr. Hope, and many others well versed on the subject of cardiac disease, have founded their belief in the almost invariable termination of pericarditis in permanent adhesions; and I feel compelled, after a careful and attentive examination of the subject, to differ from the conclusion at which they have arrived.

With respect to the *frequency* of pericardial adhesions, we have no means of determining *anything* with certainty, except by examinations after death. It is, I think, next to impossible to feel sure, during life, whether adhesion has or has not, in any given case, ensued. The great frequency with which pericarditis is accompanied by serious affection of the valves, vitiates all conclusions derived frem unnatural action or disordered functions of the heart; for with these the pericardial adhesion, supposing it to exist, may have less to do than the coincident changes in the valves. In support of such view may be mentioned those cases in which universal adhesion of the pericardium was found after death, while during life there has been nothing to give the least suspicion of its existence. Dr. Latham met with several such instances.† Compelled, then, to look to post-mortem statistics for information on the subject, let us inquire what is the evidence de-

* Diseases of the Heart, 3d edition.
† L. c. vol. i. p. 100.

rived from this source? As far as my own limited observation extends, an adherent pericardium is an extremely rare occurrence. With the exception of one case in which the pericardial sac was obliterated, and the heart surrounded by an adherent plate of bone, I have not met with a single instance of it during the last twelve months, although I have examined upwards of a hundred bodies. Surely this cannot be a mere accident, for cases of all kinds die and are examined, and many of them present other unmistakeable proofs of previous pericarditis.

I would here correct an error apt to arise respecting the *term* adhesion. The sense in which it is used here, and generally, in works on the subject, is that of an organized vascular structure of perfectly formed fibro-cellular tissue, more or less closely and compactly agglutinating together the contiguous surfaces of the pericardium, and thus more or less completely obliterating the cavity of the sac. An anatomical change of this kind indicates a state of things of long standing. The soft, easily-broken down adhesions found after death during a recent attack of pericarditis, must be carefully distinguished from the above: they consist simply in the sticking together of the opposed surfaces of the pericardium, by means of recent soft lymph effused between them.

The evidence against the frequency of organised pericardial adhesions which is afforded by post-mortem examinations closely coincides with what reason might lead us to expect. Permanent adhesion of the pericardium seems a thing of all others least likely to ensue: when we consider that the heart by its natural movements is constantly changing its position at the rate of seventy or eighty, or even more times, per minute; that the surfaces of the inflamed pericardium are usually separated by a greater or less quantity of serum; and that, when the disease is on the decline, and the system perhaps under the influence of mercury, absorption both of the lymph and of the serum is probably in full activity, we must admit that these are conditions not very favourable to a process which obviously requires rest, contact of surface, and persistence of the organizable material. The wonder is, how adhesion ever occurs,—how, in defiance of so many obstacles, the two surfaces of the pericardium can ever become permanently fixed together. That it *may* do so is sufficiently proved in that it *does*, though the phenomenon is difficult of explanation. I can imagine two conditions, either of which may perhaps lead to the process. First, it may occur in cases characterised by a large effusion of solid lymph, and a small effusion of serum. One can conceive in such a case that a moderately firm adhesion may have time to take place before the lymph in immediate contact with the absorbing surfaces can be removed, while the thickness of the mass will favour the process by rendering the movements of the heart less effective in impeding it. Once sufficiently strong to resist the fully restored action of the heart, the tendency of the lymph to contract and become organised would cement the adhesions for ever, and render the evil irremediable.

The other condition under which permanent adhesion may perhaps ensue was suggested by a case which recently occurred in St. Bartholomew's Hospital. A man, turned 50, was admitted with rheumatic inflammation of several large joints, accompanied with a distinct pericardial friction-sound. He was copiously bled from the arm, cupped, and treated largely with calomel and antimony, and kept under the influence of these remedies many days. His fever was completely subdued, the pericardial friction-sound entirely vanished, yet the patient got worse instead of better, and soon passed into a state of great exhaustion. From day to day he was observed to be deadly pale, bathed in profuse sweats, free from pain, but much distressed in breathing, sometimes quite gasping. His pulse was small, feeble, rapid, and irregular; the heart's action and sounds could scarcely be perceived; yet there was no unusual dulness on percussion to warrant the suspicion of copious pericardial effusion. After continuing in this state for upwards of a fortnight, he gradually sank and died. The examination of his body revealed much that was instructive. The kidneys were found degenerated. Here seemed one explanation why he never rallied, why he sank under the combined influence of an acute disease and the active remedies necessarily employed to combat it. Under other circumstances, these remedies might have

saved his life; but, with an organ like the kidney extensively diseased, they seemed but to accelerate his death. The next point of interest in the examination was an universal adhesion of the pericardium through the medium of a thin layer of moderately recent lymph. The adhesion readily admitted of separation, yet the force required for the purpose was probably greater than during life was exerted by the action of the exhausted heart,—an action scarcely sufficient to give rise to a sound in the cardiac region. So far had the process of adhesion extended, that, had the man recovered, and the healthy action of his heart been restored, the surfaces probably would never have separated, and the case would thus have terminated in permanent obliteration of the sac of the pericardium; and in this way probably arise many other cases of permanent pericardial adhesions,—the condition being that, for many days after the subsidence of the inflammation, the heart should remain with its action so enfeebled as to be unable to prevent agglutination of the two approximated surfaces of the lymph-besmeared pericardium, until the adhesion has become strong enough to resist its restored action.

In addition to what has been said respecting the rarity of permanent pericardial adhesion, it may be remarked that the very ingenuity displayed by Nature in the reparation of her damaged structures might reasonably lead to the supposition that, in repairing a mischief inflicted on the pericardium by inflammation, some more simple and less injurious method would be contrived than that of cementing the two surfaces together, and thereby irreparably crippling the movements and impeding the functions of one of the most important organs of the body. It is obvious that the formation of false membranes, instead of adhesions, is, next to complete absorption of the effused lymph, the most effectual method by which repair of the injury could be accomplished; for such false membranes, unlike organised adhesions, would probably afford but little obstacle to the ultimate removal of the greater part, or perhaps even the whole of the inflammatory products. Even supposing them to remain, as possibly they often do, they cannot be considered as very prejudicial to the functions of the heart, for we frequently see them passing into a perfectly harmless condition, becoming thin, blending with the natural investment of the heart, and developing a layer of epithelium on their exterior, whereby the smooth polished secreting surface belonging to the healthy pericardium is restored. Moreover, the heart does not seem to resent their presence; for their existence is not found attended by enlargement of this organ, unless there be coincident affection of the valves, or some other lesion, such as degenerated kidneys, whereby such enlargement may be explained.

III. On the last point little need be said; for, if what I have endeavoured to prove be true, that inflammation of the pericardium terminates far less frequently in the formation of permanent and organised adhesions than is usually supposed to be the case, the prognosis in this disease is evidently more favourable than if such results more commonly ensued. From the general opinion at present entertained on this subject, the prognosis in even the most favourable cases of acute pericarditis attended with effusion of lymph is gloomy enough; for most writers seem to agree with Dr. Hope, that "adhesion, so far from being a *perfect* reparation, gives rise to another form of organic disease, which, in a vast proportion of cases, ultimately proves destructive to the patient."*

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* Since the above remarks were communicated to the Abernethian Society, I have obtained what seems to be conclusive evidence in favour of the opinion I have ventured to express, namely, that inflammation of the pericardium, attended by effusion of lymph, does not necessarily terminate, as is commonly supposed, in the formation of permanent adhesions. A girl, aged 13, was admitted into St. Bartholomew's Hospital, about the middle of last October, for acute rheumatism, in the course of which both endocarditis and pericarditis ensued. The pericardial friction sound was distinctly audible for four or five days, and then disappeared, and the child left the hospital early in December. In less than three months, however, she was re-admitted in a state of great exhaustion, with œdematous legs, cough, palpitation, and pain in the cardiac region. She died within a fortnight; and, on examination of the body after death, the only evidence of the pericardial inflammation four months previously was the existence of numerous vascular tufts and fringes of fine cellular tissue about the base, and of several patches of equally fine and vascular tissue spread over the surface of the heart, especially in front. There was not a trace of adhesion between the opposed surfaces of the pericardium.