

ADHERENT PERICARDIUM AS A CAUSE OF FATAL  
ENLARGEMENT OF THE HEART.

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THE term "adherent pericardium" is used here somewhat arbitrarily to signify complete obliteration of the pericardial sac by organized tissue. Extensive yet not universal adhesion is of less frequent post-mortem occurrence, and in dealing with a number of cases it is desirable that upon this one point they should conform to an exact description.

Adherent pericardium, as is well known, is not necessarily associated with either appreciable alteration of size or embarrassment of the heart. Rarely the heart is atrophied or arrested in growth, apparently strangulated by its fibrous encasement. In most cases the heart is enlarged; but often, besides the adhesion, there is some disease of the cardiac valves or muscle, or of the lungs or kidneys, to which, partly at any rate, the enlargement might be reasonably attributed. In a certain number, however, the adhesion is associated with great hypertrophy and dilatation of the heart for which there is no such possible explanation.

What I have to say concerns the last condition only, and is founded upon a series of nine fatal cases which came under my observation at St. George's Hospital during a period of three years when I was responsible for the clinical records. In seven the post-mortem examination was performed by Dr. Rolleston, to whose careful and valuable notes I am greatly indebted; in the remaining two by myself.

Youth is common to all the nine, the ages ranging from eight to twenty-one years, with an average of slightly over fifteen years.

During the same three years adherent pericardium was found after death in the bodies of twenty-eight other patients dead from various causes, in no instance with great enlargement of the heart referable simply to adhesion. Their ages were mostly over fifteen years, and averaged upward of thirty-six years. In these cases it was not always possible to determine at what age the adhesion had been contracted. In twelve, however, the history is reliable as to the initial mischief; that is to say, the pericarditis having occurred at a later age than puberty. In one of these the myocardium was extensively invaded by fibrous tissue, but in the other eleven there is no reason to suppose that life was materially shortened by the adhesion.

In five the pericarditis occurred in childhood. Four of these five had advanced valvular disease and died at the early ages of thirteen, sixteen, eighteen, and twenty-two years, respectively, with great enlargement of the heart, for which evidently the adhesion was partly

responsible. The fifth is the one solitary case in which adhesion certainly contracted in childhood proved ultimately harmless.

Conceding the possibility that fifteen is too late for its average fatality, it is clear that the condition in question presents itself chiefly in later childhood and during the period of adolescence, and that afterward it does not show. Further, pericardial adhesion contracted in adult life is comparatively unimportant, while in children it generally proves fatal at no distant time in association with great enlargement of the heart.

The explanation must be sought in the special character and attendant circumstances of the pericarditis of childhood.

Pericarditis in children, if the very young be excluded, is almost entirely rheumatic, and in this relation a far more serious disease than in adults. It has, in fact, a considerable immediate mortality. It is always accompanied by endocarditis, which, however, is unimportant mechanically—at any rate, for the time—as is also the pericarditis, much fluid effusion being exceptional. In fatal cases, it is true, the pericardial surfaces are stuck together, but rather as the result of the act of dying than the cause of death. The gravity of the affection consists essentially in its profound effect upon the heart itself, and death, when it occurs, is due to failure of the cardiac muscle and dilatation of the ventricles. All this and much more was set forth clearly and for the first time by the late Dr. Sturges in the Lisleian Lectures for 1894. The condition which he describes as active or acute carditis was found to include the great majority of cases of fatal heart-disease in children between the ages of six and twelve years at the hospital in Great Ormond Street. It occurs with lessening frequency during the subsequent years up to the time of puberty, and in adults scarcely at all.

Now in those patients that recover—that is, do not die in the acute stage—the enfeeblement and dilatation of the heart are often severe and prolonged, kept up, it may be, by the recurrent attacks of carditis peculiar to childhood. Meanwhile the pericardial surfaces, already stuck by lymph, become united by organized tissue; and although ultimately the nutrition and vigor of the muscle are completely restored, the heart is fixed in the state of dilatation. Hypertrophy follows, but under circumstances of disadvantage, and compensation is never attained.

The great dilatation which may occur in pericarditis, the opportunity thus afforded for adhesion, and the consequent persistence of the dilatation have been well pointed out by Dr. John Broadbent. In many cases the continuity of the sequence can be established from direct observation. In others, of longer duration, and which have not been observed from the first, the importance of the adhesion is overwhelmingly obvious at the post-mortem examination; but there is no doubt that here also the original mischief was dilatation, the adhesion playing a

secondary though necessary part. There is abundant evidence that the mere fact of obliteration of the pericardial cavity around a normal heart is a trivial impediment to its action and excites neither hypertrophy nor dilatation. Thus it is that adhesion following upon non-rheumatic pericarditis, which even in children is attended by little enfeeblement of the heart, proves generally harmless.

Hope was the first to recognize the importance of adherent pericardium as a cause of fatal enlargement of the heart. Of the nature and fatality of the association he was for long the sole, and is even at the present day the leading, exponent. He justly laid stress upon hypertrophy as the result of what he termed the shackling of the heart, and is generally quoted as having taught that the morbid processes took place in the reverse order to that given above—first adhesion, last dilatation. It appears, however, upon attentive perusal of his work that he was aware of the essential fact that the heart is enlarged *ab initio*. The first enlargement was described as hypertrophy, but inflammation was one of the causes of hypertrophy, and the word is not used in a favorable sense, for the muscular substance is spoken of as having been softened by inflammation, dilatation thus taking place the more readily.

Upon this point Hope is not explicit, but he expressly observed that "cases of adhesion terminating in enlargement often hurry to their fatal conclusion with more rapidity than almost any other organic affection of the heart;" and again, "adhesion of the pericardium, which rarely fails to produce hypertrophy with dilatation, is an extremely formidable complication of this malady. It greatly aggravates all the symptoms and accelerates the fatal event. It is not unusual for this to take place within the period of a year, and I have known it occur in nine months." The truth of these observations, so far as they go, is not to be disputed.

A hard and fast distinction is sometimes drawn between external and internal pericardial adhesion—necessary to explain the frequent absence of certain physical signs, but somewhat artificial as regards the circumstances we are considering. Internal adhesions alone around a dilated heart must seriously impede its return to the normal size. The supple and elastic envelope is changed into one of considerable thickness and rigidity which opposes more passive resistance than the heart can exert active force when the muscle begins to recover, and the relaxation of the fibre due to feebleness passes into permanent elongation.

Be this as it may, acute inflammation of the pericardium in children is seldom confined to its inner surface. The outer participating, contracts adhesions to some or all of the parts with which, in the distended state of the sac, it is in contact—the sternum, chest-wall, lungs, structures in the posterior mediastinum, and, perhaps most important of all, a larger surface of the diaphragm than that which forms the normal pericardial floor. Of these, lateral adhesions to the lungs are probably

the least embarrassing to the heart, unless, as often happens, the lungs adhere to the chest-wall also. Such matters do not escape the notice of the pathologist, but it may be otherwise with morbid adhesions in front of the heart. Normally the pericardium is loosely attached to the sternum and fifth and sixth left costal cartilages. Pathologically the intimacy of this attachment is a question of degree, and the less degrees are liable to be overlooked under the ordinary post-mortem procedure of removing the sternum before the connections of the heart are examined.

The same procedure is ill calculated to display the full extent of any adhesions there may be of the pleuro-pericardium to the front of the chest, when the lungs being pushed aside the folds of the pleural cavity which overlap the heart are obliterated.

In most of the cases which form the subject of this paper external adhesions were a prominent feature. In some they are not mentioned; but for the reasons given it is probable that they were never entirely absent, and that in general they were more extensive than would appear from the bare records.

Adherent pericardium in children has recently formed the subject of a valuable article by Dr. Theodore Fisher, who concludes that the enlargement of the heart must be referred to the specially virulent qualities of rheumatic pericarditis and not merely to the adhesion, for pericarditis of local origin, as in phthisis or pleurisy, results in adhesion without enlargement. It is certainly true that enlargement seldom results when inflammation has invaded the pericardial sac from without, but the statement requires some reservation. When the pericardium is adherent in connection with chronic pleurisy or the condition known as indurative mediastinitis, it is the rule to find that a certain amount of dilatation and hypertrophy, not confined to the right ventricle, has been induced more or less mechanically by the contracting tissue. Such cases, however, are essentially chronic and have little in common with those under consideration in which enlargement of the heart is the predominant feature throughout.

The most striking change is hypertrophy. The muscle is usually firm, red, and free from fibroid invasion; the weight of the heart always immensely increased, seldom to less than twice, sometimes to more than thrice the normal.

The dilatation, however, though less conspicuous, is the essential mischief. With rare exceptions all the cavities are dilated; but the change is most marked in the ventricles where it begins, and the auricles may practically be disregarded.

To take the simplest case of a child dying early in acute carditis, the condition of the muscle is merely functional failure, unexplained by gross or even microscopic change, and the thin-walled right ventricle

suffers most. When the acute stage has been prolonged the myocardium on both sides of the heart is found swollen and infiltrated with inflammatory elements, a state of things from which the thick-walled left ventricle is naturally the last to recover. So it happens that sometimes the one ventricle, sometimes the other, is taken at a greater disadvantage by the adhesion. With the returning vigor of the heart the stronger left with its wider range of movement recovers its freedom to a greater extent than the other; and, upon the whole, no doubt adhesion is more disastrous to the right.

The relative dilatation of the two ventricles is not always exactly apparent from post-mortem descriptions of the heart itself, and allowance has to be made for the final dilatation of the left ventricle which sometimes sets in upon the approach of death, and from which the right, by the very reason of its close attachment to the diaphragm and chest-wall, is in great measure protected.

A better method of estimating the relative efficiency with which the two ventricles have been accustomed to act is to compare the state of the lungs with that of the general venous system. Submitting my nine cases to this examination dropsy was present in all but two, and in these two the liver was nutmegged; that is to say, in every case the right side of the heart had given way. In five this was not due to backward pressure from the left side, for the lungs were free from any sign of chronic congestion; while in four there was either pulmonary apoplexy or well-marked chronic congestion, from which it may be inferred that the chief and probably the primary fault was on the left.

We may yet hesitate to ascribe the failure of the left side entirely to pericardial adhesion, for a complication has to be reckoned with in the invariable presence of some degree of valvular disease. As previously stated, the rheumatic pericarditis of children is always accompanied by endocarditis. The aortic valve usually escapes, the mitral never; and so it necessarily happens that in each of my cases the mitral showed an appreciable departure from health.

The change was of that limited and gradual kind which, if free to pursue its natural course, would end in stenosis. In such cases the valve was particularly described as being shaped like a button-hole or like a funnel. In no instance, however, was the orifice actually stenosed; and in some it was wider than normal. Clinically the blowing murmur of mitral regurgitation was also present. Frequently, also, there was a so-called "presystolic murmur," more or less loud, long, and rough, and otherwise resembling that which occurs in mitral stenosis. There are good reasons for believing that this murmur may correspond to the systole of the ventricle and so to regurgitation, but here it suffices to point out, what has been observed before, that the murmur does not necessarily indicate stenosis.

Regurgitation, and not obstruction, was in every case what occurred, and due more to the pericardial adhesion than to the proper disease of the valve.

The two lesions, in fact, are mutually antagonistic. On the one hand, the development of actual stenosis is prevented by the dilatation of the ventricle and consequent stretching of the mitral orifice; on the other, the free regurgitation which would result from stretching of a healthy valve is moderated by the tendency to stenosis.

As for the diagnosis of adherent pericardium, the only certain physical signs depend upon the heart being so closely connected with the chest-wall or diaphragm that with systole the præcordial parts, or, as Broadbent finds, the lower ribs on either side are pulled inward. Such direct indications are seldom available, but cases of the kind which have been discussed are sufficiently recognizable from a number of circumstances and phenomena, none of which taken alone are conclusive of adhesion.

A history of pericardial friction or of recurrent rheumatism in childhood is highly suggestive, and still more so is the development of immense enlargement of the heart within a short time from the first illness. The præcordium is often unduly prominent and occupied by an impulse which, though both wide and strong, is more particularly characterized by the impression it gives of a curbed and struggling heart. Dropsy, especially in the form of ascites, is of early occurrence; and in spite of the invariable presence of one or more mitral murmurs, the lungs are often entirely unembarrassed. The complexion ranges from a faintly bluish pallor, when the right heart only is at fault, to a leaden but hardly cyanotic hue when the left also fails. Last and most characteristic of all are the rapid progress of the case to its fatal end and the signal failure of all known means of relief in heart-disease.

In conclusion, although there is nothing to say about the treatment of adherent pericardium, a word may be hazarded about that of pericarditis so far as concerns the dilatation of the heart upon which the gravity of adhesion depends. The best authorities are agreed that in the acute stage of pericarditis digitalis is worse than useless, but it does not follow that the heart will not respond to a simpler stimulus. Gaskell, in his classical experiments upon the tonicity of the heart and arteries, found that minute quantities of alkali produce a tonic contraction of normal cardiac muscle indistinguishable from that which occurs with digitalin, and it would seem that this discovery is not without its clinical application. I have watched many cases of pericarditis under alkaline treatment given for a different purpose, and have often observed the heart's action to become calmer and more efficient, and a mitral regurgitant murmur, probably of dynamic more than organic origin, to disappear.

CASE I.—A child, named Charlotte D., had a first attack of rheumatism in 1880, and a second, much more severe attack in October, 1885, when she was fifteen years old. In August of the following year she began to suffer from dropsy, and in April, 1887, she was admitted into the hospital for the first time.

The face was pale, the lips faintly cyanotic, and, although the legs were oedematous, the chief dropsy was in the abdomen. The heart was greatly enlarged to the right, with much epigastric pulsation but little leftward displacement of the apex. A blowing systolic murmur was heard on both sides of the chest, and a short "presystolic" murmur in the apical region. She improved under treatment, which included tapping the abdomen; but during the next three years and a half was readmitted often and with decreasing intervals of relief, always with the same characters of complexion and dropsy. The abdomen was tapped altogether twenty-six times. The heart underwent considerable further enlargement, as shown by the gradual displacement of the apex to a point in the sixth interspace, two inches outside the nipple-line, the murmurs remaining much as before. In October, 1890, pericardial friction was audible for a few days, after which she sank, having now a little bronchitis for the first time, and died in December, at the age of twenty-one.

*Post-mortem:* The pericardium was adherent externally to both lungs, the chest-wall, diaphragm, and posterior mediastinum. The sac was completely obliterated by tissue, which was mostly old and fibrous, though comparatively recent over a limited area posteriorly. The heart weighed thirty-six ounces. The right ventricle was both greatly dilated and hypertrophied, the left described as slightly hypertrophied only. The pulmonary and aortic valves were healthy, the tricuspid dilated and slightly thickened. The segments of the mitral were thickened and rigid, the chordæ tendinæ retracted, the orifices not constricted but shaped like a button-hole. The whole of the left pleural cavity and the back of the right were occupied by old adhesions. The lungs were small, otherwise normal. In the abdomen the most important morbid appearance was thickening of the capsule of the liver, which organ was nutmegged and enlarged, weighing sixty-four ounces.

CASE II.—Amelia C., previously healthy, was attacked by rheumatism at the age of fourteen, in June, 1892. Pericarditis developed in the course of July, and was followed by obstinate vomiting. She was admitted into the hospital toward the end of November, complaining chiefly of the vomiting and not yet conscious of any trouble about the heart. The heart, however, was already much enlarged, the apex being in the sixth left interspace, outside the nipple-line, and the area of pulsation extending to the epigastrium. A blowing systolic murmur, preceded at the apex by a knocking first sound, was heard in the mitral area, and a blowing diastolic internal to the nipple. The pulsation grew rapidly more prominent and labored; great oedema of the legs and ascites set in, and at last swelling of the trunks from thrombosis of the subclavian veins. She died at the end of December.

*Post-mortem:* The pericardium was adherent externally to both lungs and firmly to the tissues in the superior mediastinum, but not to the sternum or ribs. The two layers were completely united. The heart weighed twenty-five ounces. The left ventricle was both dilated and hypertrophied, the right dilated only. Both tricuspid and mitral valves

were dilated. The edges and chordæ tendinæ of the latter were thickened, but the condition was clearly more stretching of the orifice than disease of the valvular structures. The nortic valve showed a few recent vegetations, but appeared competent. The myocardium was healthy. The right lung was slightly cedematous, the left compressed by pleural effusion; no pulmonary apoplexy. Liver nutmegged; spleen and kidneys hard and congested.

CASE III.—Florence H., aged ten years, who first suffered from rheumatism in the summer of 1890, was admitted into the hospital in the following September with acute rheumatism and slight chorea. There was already a blowing mitral murmur, and almost immediately a sharp attack of pericarditis developed, from which she recovered without extreme enlargement of the heart. Subsequently she had much rheumatism and was often readmitted, the heart undergoing progressive enlargement. The blowing murmur was constantly present; sometimes also a so-called "presystolic murmur and apical thrill." Her complexion was always of a leaden color, the veins being evidently full. Dropsy set in in May, 1893, and she died in July, at the age of fourteen, with extreme œdem of all the subcutaneous tissues and much ascites.

*Post mortem:* The heart occupied an enormous part of the left side of the chest, but did not extend far to the right. The pericardium was closely adherent to the sternum by tough fibrous tissue, and firm hands passed from near the apex of the heart to the chest-wall. Between the pleuro-pericardium and each lung were firm bands which appeared to have become stretched since their formation. The two pericardial layers were everywhere closely united and could only be separated by forcible tearing. The heart with the adherent pericardium weighed twenty-one ounces. Both ventricles were greatly hypertrophied, the left dilated also. The aortic valve and those of the right side were healthy. The mitral was funnel-shaped and somewhat thickened, but the orifice was not stenosed, admitting just two fingers. Both lungs were compressed by fluid in the pleuræ; no pulmonary apoplexy. Liver nutmegged, spleen and kidneys cardiac, pancreas extremely indurated.

CASE IV.—John O'B., died in June, 1890, at the age of thirteen years. His illness dated from an attack of rheumatism attended by præcordial pain six months before. He was under observation for the last five weeks of his life, the chief clinical phenomena being a large, laboring heart with a loud systolic blowing murmur; ascites, for which he was tapped; and latterly bronchitis, with duskeness of face but not cyanosis.

*Post mortem:* The pericardium was firmly adherent externally to the trachea and aorta, and internally to the heart. The heart weighed fifteen ounces. The right ventricle was dilated, the left hypertrophied. The mitral orifice was rather large, admitting two fingers loosely; the edges of the valve thickened, retracted, and beaded; the chordæ tendinæ shortened. The muscle of both ventricles was unusually firm, but showed nothing abnormal under the microscope beyond slight increase of fibrous tissue around the bloodvessels. Pleural adhesions on right side; no engorgement of lungs. Liver nutmegged.

CASE V.—Joseph J., previously healthy, had rheumatism in February, 1890, and became dyspnoic and œdematous in October of the same year. He died in November, at the age of eleven years, shortly after admission into the hospital. There was much pulmonary distress,



with hæmoptysis, blueness of lips, galloping action of the heart, and bellows mitral murmur.

*Post-mortem*: The pericardium was slightly adherent to the chest-wall and everywhere firmly to the heart. The heart weighed seventeen ounces. Both ventricles were hypertrophied—the right slightly, the left markedly dilated. The mitral edge and chordæ tendinæ were thickened, the orifice hardly dilated. In the septum between the ventricles was a minute perforation, apparently congenital and obviously unimportant, though of interest in connection with the fact that the patient's complexion had always been high colored. Extensive bronchopneumonia and several pulmonary apoplexies. Much dropsy.

CASE VI.—Collin B., died in June, 1891, at the age of nineteen years. His health had been very good until November, 1890, when he was attacked by rheumatic fever which confined him to bed for five weeks. The immediate cause of death was pneumonia, but he had suffered from præcordial distress and dyspnoea for two months previously, and the essential disease was undoubtedly that of the heart.

The main cardiac phenomena were a cantering rhythm, knocking first-sound, and blowing apical systolic and rumbling diastolic or presystolic murmurs. At the end the heart's action was very tumultuous. The complexion was conspicuously pallid.

*Post-mortem*: The pericardium was not unduly adherent to the sternum or chest-wall. There were fairly recent adhesions to the right lung. The two layers were completely united by firm fibrous tissue. The heart weighed twenty-eight ounces. The left ventricle was dilated and considerably hypertrophied, the right slightly dilated. All the valves showed bead-like vegetations. The mitral segments and anterior chordæ tendinæ were thickened, but the orifice was not contracted. Some of the columnæ carneæ were affected with fatty degeneration; the rest of the muscle, though pale, showed no fatty or fibroid change under the microscope, and most of the fibres retained their striation. The upper lobe of the right lung was in a state of gray hepatization; the rest of the lungs œdematous. Liver nutmegged; no dropsy.

CASE VII.—Ellen J. died in February, 1892, at the age of eight years. There was no history of rheumatism, but one of chorea and left hemiplegia (probably embolic) in the summer of 1891. She recovered from both, but only to fail again from shortness of breath at the end of the year. She was now admitted into the hospital with progressive cardiac embarrassment which proved fatal without dropsy. There was extensive pulsation, with a blowing mitral systolic murmur preceded in the apical region by a short rumbling sound and an ill-defined thrill. The complexion was a sickly bluish pallor.

*Post-mortem*: The pericardium was adherent to the sternum and both lungs by firm fibrous tissue. Filamentous fibrous bands existed also between the base of the heart and the upper lobe of the right lung. Internally the pericardium was universally adherent to the heart, for the most part by fibrous tissue, though on the left side was some sanious lymph. The heart weighed sixteen ounces. The right ventricle was dilated, the left both dilated and hypertrophied. The mitral segments were thickened and retracted and the chordæ tendinæ shortened, but the orifice was of normal size. A few vegetations on aortic valve. Both pleural cavities extensively occupied by old adhesions. In lower lobe of right lung a firm patch of congestion resembling an apoplexy

in process of absorptioa. Lower lobe of left lung collapsed. Liver nutmegged.

CASE VIII.—Annie B. first had rheumatism at the age of three years, from which time she was known to have heart-disease. A second and very proloaged attack occurred when she was about twelve, and a third three years later. In August, 1890, being now sixteen, she was under treatment for subacute rheumatism; and in November she was readmitted into the hospital, severe præcordial distress and much œdema having developed suddenly. The heart was immensely enlarged, especially on the left, the apex being in the sixth interspace two inches outside the nipple-line. Blowing systolic murmurs were heard in both mitral and tricuspid areas, and occasionally a so-called "presystolic" murmur at the apex, where there was a marked diastolic thrill. She became rapidly worse and died in January, 1891.

*Post-mortem:* The pericardium was considerably thickened and universally adherent to the heart. The left ventricle was much dilated and also considerably hypertrophied, especially the columnæ carneæ; the right very slightly dilated only. The mitral valve was much thickened; it was somewhat wider than natural, admitting three fingers barely, but was stenosed in relation to the size of the ventricle. Right lung engorged; apoplexy at base of left lung.

CASE IX.—Ada C., aged twenty years, was admitted in December, 1892. Her health had been good until the end of 1890, when she had an illness which she called "influenza," but which was probably rheumatism. Since then she had suffered from palpitation of the heart. Œdema of the legs first appeared in July, 1892. She was pale, cyanotic, and moderately dropsical, with an extensive cardiac impulse and turbulent action. At the apex, which was in the sixth space and far outside the nipple-line, was an early rough murmur with a thrill ending in a snap and followed by a blowing systolic murmur. She improved and was sent to the Convalescent Hospital at Wimbledoa; but the dropsy soon returned, surpassing its former degree, and she was readmitted at St. George's in February, to die a few days later.

*Post-mortem:* The pericardium was universally attached to the heart by organized but not very strong adhesions, and there were filamentous adhesions to the chest-wall. The heart was very large: the left ventricle greatly dilated, the right slightly hypertrophied. The myocardium was healthy. The mitral orifice fairly admitted two fingers, but the edges and chordæ tendinæ were thickened and the segment pulled down into the ventricle, the valve being thus somewhat funnel-shaped. The lower lobe of the left lung was in a state of red hepatization, and the rest of the lungs showed signs of chronic congestion.

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