Thesis for Degree of Doctor of Medicine

"On adherent Pericardium of Rheumatic origin. With Cases:

by

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It is a somewhat remarkable circumstance that though most modern writers upon Systematic Medicine, and those who have turned their attention especially to Cardiac pathology, give more or less place to the lesion of which this paper treats, very few, if any, have been able to give a definite opinion of the part, if any, played by adhesion of the pericardium to the heart surface in the production of serious and, as I believe, frequently fatal Cardiac disease.

Dr Wilks[1], the most recent I believe of English writers upon this subject, if one leaves out of mind the late Dr Sibson's article on Adherent pericardium in Reynolds' System of Medicine, remarks that it is curious that the disease in question has not attracted more attention, since it does, as he gives cases to show, produce under certain conditions irretrievable damage to the central organ of the circulatory system. That it is only under certain conditions, many of which at present are not well understood, that obliteration of the pericardial sac becomes of serious import, is obvious from the very diverse and vague opinions expressed upon the subject by writers, both at home and abroad.

Dr. Hope, whose work upon diseases of the heart is still deservedly quoted, seems to have strongly inclined to the view that adhesion of the pericardium

(1) Guy's Hospital reports. Vol XVI.

ought to be regarded as a definite & not constant cause of cardiac disease, and this in a special form — namely of dilatation and hypertrophy and this apparently whether the pericarditis be of rheumatic origin or chronic from the outset, as in some rare cases it seems to be. Since Hope's time many writers and many cases have appeared both supporting and weakening his position; and it is somewhat unfortunate for the point he wished to Establish that in the cases given by him in illustration, one was not fatal, in another was marked disease of of the aortic valves existed, and in the third there was an aneurism of the aorta with deformity of the aortic valves.

In 1851 and again in 1858. Professor Gairdner[2] published some important observations on the Subject; on the whole unfavorable to Dr Hope's views Dr Gairdner's cases were, indeed so far as I can see, cases tending conclusively to show that adhesion of the pericardium Either local or universal, does not by any means constantly produce any serious secondary Effects upon the heart. It is worthy of note that in his series of cases there was not one of known rheumatic origin. In many of the cases the adhesion was only partial, and in one or two instances one cannot Escape the doubt that

(2) Edinburgh Journal.

other causes were at work than those specially noted
For example, in case 14, the man had been hemiplegic
and had given other evidence in the shape of
albuminous urine, & there probably being some
chronic renal change, which I believe at the time
Dr Gairdner was writing was not clearly recognised
as a cause of Cardiac Hypertrophy.

To recur to Dr Wells' Paper. Of six cases in which
General and firm adhesion of the pericardium
was found in only one instance was a history of
acute rheumatism noted. and throughout the
series the changes in the heart chambers were
by no means uniform; in some the walls were
natural, in others thinned. and in one cases the
auricles alone were seriously affected.

Dr Sibson's Extreme Elaboration of detail renders
it difficult to grasp with any clearness his General
conclusion, and tends rather to obscure than Elucidate
the point raised in this paper - namely, that Simple
General adhesion of the pericardium of comparatively
recent rheumatic Origin, without Valvular complication
is frequently if not constantly followed by a fatal
Ectatic condition of the Cardiac chambers.

Dr Powers[3] discusses the question fully in his article
on dilatation of the Heart and quotes, Hope, Gairdner
Kennedy and others to show that pericardial
adhesion causes Enlargement of the heart in
one-third of the cases: but at the same time Very

(3) Reynolds System of Medicine Vol IV

justly remarks that more evidence is required as to the condition of the heart muscle and adhesion of the pericardium to surrounding structures. In regard to the condition of the cardiac muscle I shall have something to say further on: but in regard to the second point I may here say that it was absent in all the cases given; and I cannot help thinking, from the great parity with which it has been noted by observers, that this condition, so much relied upon by some who have attempted to lay down rules for the correct diagnosis of the lesion, has found place in the books rather as the result of a priori reasoning than of accurate post-mortem observation.

The weak points in Dr Kennedy's paper were long ago indicated by Dr Gairdner and I need not refer to it here except to say that no details are given of the cases and that external adhesion of the pericardium is spoken of as having been observed in but an extremely small number of the series.

Two points seem to be agreed upon by most writers, first that adhesions of limited extent, and perhaps of certain length, have little deleterious effect; and secondly, that a certain chronicity & firmness of the adhesion is essential to the production of the changes here described. Wilks and Moxon,(4) for instance, insist strongly upon this latter point, saying that while they believe simple pericardial adhesion produces no untoward

(4) Pathological Anatomy 2nd Ed.

consequences, the heart is always found dilated and hypertrophied when the adhesion is by means of ~~means of~~ a thick layer of fibro-vascular material which cannot be stripped from the surface of the heart without lacerating the muscle. Dr Wilks's cases before alluded to go to support this view. It is upon this latter point, that I wish to say a few words by the light of the cases I am allowed to make use of below. It seems to have been clearly determined by the above writers that such conditions as those following upon acute rheumatic pericarditis even though of some standing, have had according to their experience little to do in producing an ectatic condition of the cardiac chambers. I believe that further experience will shew that rheumatic pericarditis, universal in extent, and of well marked intensity is frequently, if not as a rule soon followed by the lesions found in the cases before us.

Non-rheumatic pericarditis is not, under present methods of treatment at any rate, by any means a common disorder. I am inclined to think that the frequency of its occurrence has been somewhat overestimated in the minds of many. We have a tendency to assume a familiarity, though we have it not, with a disease of such well marked and definite character, as acute inflammation of the pericardium. For instance in the two years

ending April 1881. during which time I acted as House Physician to the Leeds Infirmary Seventy cases of acute rheumatism were admitted into the medical wards. but in only three of the number was a friction sound noted. During the same interval of time the four cases of adherent pericardium given below were observed by myself in the post-mortem room. It would undoubtedly be rash to assume from this that every case of pericarditis eventuates in adherent pericardium Still it is a somewhat strange coincidence that in a hospital where the experience varies relatively little, the cases of adherent pericardium should equal and exceed in number those of acute pericarditis. Again, if, as some pathologists teach and apparently with good reason, there is no such thing as the restitution of a once inflamed serous surface to its former condition of health - that every inflammatory effusion must always leave behind some, little though it be, lowly organised tissue, then such a condition at any rate of obliteration of the pericardial sac would seem to be the inevitable result of every pericardial inflammation of equal extent. I do not for one moment insist upon the statistics of the Infirmary in support of this; the cases are too few for any conclusion of the kind to be justly arrived at

It must not be lost sight of that I am claiming only universal and intense pericarditis as a cause of cardiac dilatation. Many pericardial adhesions of limited extent produce obviously no change in the heart of importance. I wish, if I can, to make clear that a universal pericardial adhesion of even a comparatively soft nature, quite distinct from the firm fibrous material spoken of by Wilks and Moxon and others, is capable of producing and does produce dilatation with more or less hypertrophy of the cardiac chambers, especially the ventricles.

I suppose we may take it as true - indeed it would be curious if it were otherwise - that there is a relationship of the nicest equilibrium existing between the heart power and the work or resistance to be overcome. Or, if I may so speak, the heart is not in a position to treat with indifference any increase of resistance in front or hinderance to its action behind or externally - at any rate within the very narrowest limits.

It must not be supposed that I for a moment lose sight of or ignore the fact that in every case of acute pericarditis more or less inflammation of the heart muscle exists. On the other hand I am inclined to assign to it a most important if not essential rôle in the production of the further changes in the ventricles. In the initial

stage of the process, before whatever inflammatory effusion there may be has attained to any density, the myocarditic condition will be. and undoubtedly is, the principal factor in producing weakness of the parietes and consequent dilatation; but it is not a persistent lesion, in this instance tending gradually to diminish. and probably only placing the outermost zone of the heart muscle hors de combat for the time being. The condition we have to deal with is one the maximum intensity of which is reached in and about the pericardium itself.

It is thus probable that in the earliest stages of pericarditis a condition of more or less "parietal debility" is established and consequently slight dilatation prone to ensue. At the same time the plastic material is increasing in quantity and in density (I assume that fluid is absent throughout the case or having been present has disappeared), the whole thickness of the pericardium softening from extension of the inflammation and thus less able to resist the accumulating effusion and dilated heart. The surface of the heart is now covered by a more or less thick layer of spongy, fibro-gelatinous material, an inelastic non-contractile body, closely applied and adherent with more or less firmness to it; it is ~~but~~ in fact in the condition of a distended Elastic ball

with an inelastic material evenly and carefully, but by no means of necessity firmly, applies to its outer surface. It is the tenacity of the adhesion that I am inclined to regard as of little importance so long as it is sufficiently strong to maintain the material closely applied to the surface during all movements. It will fulfil, I believe, every condition necessary for the increase and perpetuation of dilatation when once that dilatation is commenced.

It would scarcely be necessary to point out that the pericardium does not follow all the movements of the heart had not the contrary been stated by some authors. Wishing to show that adhesion of the pericardial surfaces could produce no change in the heart, and so the adhesion be able to impair from the first, to some extent, the completeness of the Systolic Emptying.

The adhesion of the pericardium to the chest wall was, as stated above, absent in all the cases and consequently I have not found it necessary to discuss it further, as I believe it to be of no importance - at any rate not Essential for the production of Cardiac disease.

I am of necessity compelled to enter at greater length into the question whether the myocarditis which ever accompanies inflammation of the pericardium is not in itself quite sufficient to explain the dilated condition of the Ventricles. Now in all the cases

there was considerable and even great increase in the weight of the organ and that increase was obviously due almost entirely to the great amount of tissue in the hearts' wall. This must clearly have been the result either of a true hypertrophy of the muscle or of the effusion of inflammatory material in some form or other. We must assume that whatever inflammatory material was present was thrown out in the early stages of the disease. and had gone on to conditions of more or less organisation The cases were all of many months duration; and consequently if there had been much new tissue of inflammatory origin present the muscle structure must have suffered considerably ~~in some~~ by its long-continued influence. Though in only one case unfortunately was a microscopic examination ~~made~~. of the structure made, it never occured to me that the muscle presented an appearance that all would not admit to be healthy or nearly so. I need scarcely say that in the one case the microscope confirmed the opinion; - in short it scarcely admits of doubt that conditions of hypertrophy and inflammation of muscle are entirely antagonistic. I cannot but conclude that whatever else might have to do with the causation of the ectatic condition, the myocarditis had nothing but the production to a greater or less degree of a primary and acute dilatation of the ventricle.

Although lesions of the Valves were found in Every case. they were so minute in Extent and obviously of Such Short duration that it could be admitted at once that they could have played not part whatever in the process. The Opaque condition of the Endocardium and Valve-folds I am inclined to attribute to the long continued dilatation ~~as~~ in the Same manner as pointed out by Dr Goodhart in cases of Chlorosis.

Case I Elizabeth W..... aged 16 years. was admitted into the Leeds Infirmary under Dr Clifford Allbutts' care on Sept 26th 1879. She had been in the Hospital 8 months before for an attack of acute rheumatism and had Suffered from dyspnoea Ever since. She was at that time Supposed to be the Subject of double aortic disease. When admitted on Sept 26th 1879. She was Suffering from Slight rheumatic inflammation of one or two large joints and Great dyspnoea with intense oedema of the lower Parts of the body. There was a much increased area of Cardiac dulness. with intense thumping action of the heart so that the whole body was Shaken with Each Systole. Bruits pointing to double aortic disease and mitral regurgitation were present The radial pulse was most markedly regurgitant in character. Under Treatment She improved somewhat but was always worse when taking

digitalis. Alternately both pleural and the peritoneal cavities became the Seat. of Effusion On Nov 7th She jumped out of bed with a Shriek and fell dead upon the floor.

Autopsy Oedema of legs. Considerable Effusion in peritoneal cavity. Both pleural cavities contained a fair amount of fluid. Heart: The pericardium was universally and tolerably firmly adherent to the surface of the heart. The pericardial cavity was completely obliterated. The membrane could be entirely separated with a moderate amount of force leaving the Surface of the heart covered with Shaggy fibrinous material. The heart when stripped weighed 31 ounces and was much enlarged. The Enlargement was mainly due to hypertrophy and dilatation of the Ventricles especially the left. The cavity of the left Ventricle was large enough to lay one's fist in with Ease. The musculi papillares were enormously increased. The Ventricular wall was twice its natural thickness even in this greatly dilated condition The mitral orifice admitted five fingers Easily The mitral Valve was thickened and opaque to some extent but otherwise Shewed no Signs of disease. The left auricle was much dilated and its walls thickened The aortic valve on removal of the heart from the body proved to be quite competent to the ordinary water test. The Segments of the Valve were much Stretched downwards towards

the cavity of the Ventricle, and like the mitral Valve although not distinctly diseased were much thickened and opaque. The right Ventricle was not so much dilated as the left. The right Valves were healthy. The aorta and pulmonary artery were healthy. The liver, Kidneys, and Spleen were enlarged and indurated as is usually seen in long standing cases of mitral disease.

This patient presented the General condition of one suffering from well established mitral disease and such indeed appeared to have been the case, but not as an ordinary case of mitral Valvulitis. The condition was undoubtedly one of dilatation of the mitral orifice secondary to a dilated condition of the left Ventricle. The absence of the ordinary Evidences of Valvulitis go to support the truth of this Explanation.

The competency of the aortic Valve to the ordinary post-mortem water test is not matter for Surprise. Though an aortic regurgitant murmur was heard during life. The test is I believe entirely unreliable on account of the Extremely Easy manner in which the relations of parts can be disturbed post mortem.

Case 2 Alice W.... age 20 years. was admitted under the care of the late Dr Hilton on March 23rd 1880. She had been in Hospital 18 months previously with an attack of acute rheumatism lasting Eight weeks

followed by dyspnea on exertion. Six weeks before admission on March 23rd the present attack of acute rheumatism had commenced. On admission the right knee and right wrist were swelled and painful. Temperature 102°. Heart Visible pulsation in fourth and fifth left interspaces in mammary line with some heaving of chest wall. Apex beats full and strong over an increased area. ~~full and S~~ in 5th space. four and a half inches from middle line Area of cardiac dulness increased Especially laterally towards Right Side. Cardiac action regular but rapid. At apex a distinct systolic bruit was heard with other indeterminate sounds. at the base a marked double aortic bruit Pulse markedly regurgitant in character and audible 3 inches from Ear. She died quite suddenly on April 21st. There was great dyspnea with edema throughout. Digitalis was administered in fairly large doses without any beneficial Effect.

Autopsy 24 hours after death On removing Sternum there was no adhesion of under Surface to pericardium. The pericardium was seen to occupy a much greater area than usual. Especially in transverse diameter. On incision it was found to be universally adherent to the Surface of the heart. (It was at this point only that the Question arose of there having been any recent pericarditis, the main if not the Whole, lesion being obviously due to the previous attack of rheumatism) The

surface of the heart had a ragged bruised appearance
Heart weighed 31 ounces and was much enlarged.
All cavities were dilated Right Ventricle hypertrophied
to twice its natural thickness. Endocardium
opaque in parts. Pulmonary and tricuspid
valves healthy. Left Ventricle much dilated and
walls twice their natural thickness if not more.
Muscule papillares much enlarged. Aortic Valve
permitted free flow of water into Ventricle from Aorta
The Cusps of the Valve were thickened and opaque
Especially along their borders. and had the appearance
of having been stretched downwards into the
Ventricle There was no Evidence of recent Valvulitis
Mitral orifice much dilated sufficiently so to
admit five fingers Easily. The mitral Valve.
thickened and opaque but otherwise normal.
Aorta and pulmonary artery healthy.
Solid Viscera Shewed usual changes of old mitral
disease.

Case 3 Elizabeth. J— aged 16 years was admitted
under Dr Chuntor's care. on July 22nd 1880. She had
had Rheumatic fever. some few years previously.
Admitted pale, breathless and edematous and typical
Symptoms of Severe mitral disease. The Physical
Signs were anomalous throughout, but Generally
Speaking were rather those of mitral than of
Aortic disease, a diastolic thrill with a double
apex murmur being tolerably constant. She

continued to get worse up to Sept 22nd when she died somewhat suddenly.

Autopsy 18 hours after death Body generally edematous abdomen distended; deep Excavated Sores on each instep the result of ~~#~~ Sloughing induced by the introduction of Southey's trocar at this point. On Opening the chest the pericardium was seen much distended occupying the greater part of anterior view. Lungs Shrunken. Pericardium closely and firmly adherent to heart: Pericardial cavity completely obliterated membrane much thickened but with some difficulty could be separated from the walls of the heart, leaving them ~~coated~~ covered with a coating of edematous, fibro-gelatinous looking tissue Especially thick about the auricles and roots of great Vessels. Pericardium itself much thickened and edematous.

Heart much enlarged weighing 24 ounces; all cavities flaccid and dilated. Right Ventricle dilated and flaccid; walls in this condition do not appear to be hypertrophied: all Valves healthy and competent. Left Ventricle dilated. Walls hypertrophied to twice their natural thickness. Endocardium thick and opaque; papillary muscles large and fibrous at their apices. Mitral Valve competent; admits three fingers only; flaps thickened and indurated along free margins but quite smooth. (thickening in all

probability not sufficient to hamper movements); aortic valves thickened and opaque but competent to water test.; aorta pulmonary and coronary arteries healthy.

Case 4 Joseph B— aged 14 years admitted under Dr Eddison's care on March 11th 1881. He had had three attacks of acute rheumatism, the first not earlier than 2 years ago. For the last attack which occured in December 1880 he was an inmate of the Hospital. At this time no signs of pericarditis were observed, but there were evidences of some serious cardiac lesion of an obscure nature. On admission on March 11th the cardiac impulse was diffuse and increased in force. Apex beat was not found. On auscultation a loud rasping systolic bruit was heard over usual site of apex and could be traced to the axilla but not to the angle of the Scapula behind. He died somewhat suddenly on March 22nd.

Autopsy 30 hours after death Surface of body pale. Legs edematous to a moderate degree. Both pleural cavities contained a small amount of serum. The distended pericardium occupied quite half of anterior view of the chest and measured at its broadest part 6 inches External surface of pericardium had an opaque reddish injected appearance and was coarse in texture. There

were a few adhesions to visceral pleura about root of large vessels. One or two much enlarged lymphatic glands were adherent to anterior surface. There was no pathological adhesion to anterior chest wall. The pericardial cavity was practically obliterated by adhesions of varying consistence, most of them soft enough to break down easily under the fingers, but leaving the adjacent surfaces much roughened and altogether unnatural. Some small amount of blood stained fluid was confined in interstices of tissue. The pericardium itself was much thickened, and in parts blood stained but still soft and pliable. The heart (stripped) weighed 21 ounces; was much enlarged by dilatation of all cavities; walls of ventricles bore natural relation to each other as regards thickness. There was no marked hypertrophy to be observed in their extremely dilated condition but probably the amount of muscular tissue was above the average. (Considerable increase in weight would be accounted for by adventitious tissue attached to surface). Muscle a little soft but good in appearance: no fibroid or fatty change visible to naked eye. The Valves: There was a little delicate fringe of granulations along each cusp of aortic valve, but in no way likely to impair its function; the anterior flap of mitral valve was opaque.

I have avoided reproducing in detail the clinical reports of the cases, as to do so would only increase the length of the paper without commensurate advantage. I had at one time thought that the cases would enable me to give some help in the diagnosis of this very striking but complex condition but further experience has led me to abandon my earlier views, and so far as the clinical phenomena are concerned I have but one observation to make. In my experience I have rarely seen a case of ordinary valvular disease, either aortic or mitral in which great benefit has not followed the administration of digitalis; in the cases here given it signally failed to improve the symptoms; indeed it seemed rather to aggravate them. I have sometimes thought that it may have been from the observation of such cases, that the very curious doctrine, still, I believe, taught by some, of the dangers of digitalis in aortic disease came into existence.

In conclusion, I have to thank the Physicians to the Leeds Infirmary for their great kindness in permitting me to make use of their cases.