A LECTURE ON DISEASE OF THE VALVES OF THE HEART.

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The Anatomical Results of Chronic Valvular Disease upon the Walls and Cavities of the Heart.—The Physical Signs and Diagnosis of Disease affecting the different Valves.—Valvular Disease without Murmur.—Reduplication of the Heart's Sounds.—Bellows-Sound without Organic Disease.—Inorganic Murmurs.

GENTLEMEN,—In my lecture to-day, I propose to describe to you first the effect of chronic valvular disease upon the walls and cavities of the heart—in other words, the anatomical results of valvular disease; we shall then pass on to the consideration of the physical signs and diagnosis of each form of valvular disease. The effect of valvular disease upon the walls and cavities of the heart depends greatly upon the situation and the character of the disease. An almost constant result of all forms of valvular disease is to impede the onward movement of the blood; and this result occurs whether the valvular disease be what is called obstructive or regurgitant; whether it be such as to cause narrowing of one of the orifices, and so a direct impediment to the onward passage of blood; or whether the valve, being rendered incompetent to close its orifice, permit a reflux or regurgitation of blood. The tendency of valvular disease is to cause dilatation of those cavities which lie behind the seat of disease. Disease of the aortic valves tends to cause dilatation of the left ventricle; and this dilating effect upon the ventricle is much greater in cases of regurgitant disease at the aortic orifice than when the disease is simply obstructive. The influence of incompetence of the aortic valves in causing dilatation of the left ventricle is intelligible if you bear in mind that in these cases, during the diastole of the ventricle—that is, while the muscular walls are relaxed, the cavity becomes rapidly filled and distended, partly by the onward stream of blood through the mitral orifice, but chiefly by a reflux current forcibly driven through the unclosed aortic orifice by the elastic resiliency of the arterial walls. This unnatural and forcible distension of the relaxed ventricle tends gradually to dilate its cavity. The dilatation is a destructive process; it tends to weaken the heart's walls, and it may so widen the mitral orifice as gradually to render its valve incompetent to close it. The tendency to dilatation is checked by a compensatory and conservative process of hypertrophy of the muscular walls of the cavity. The muscular fibres, stimulated to increased exertion by the excessive pressure of blood, grow firmer and thicker, and this conservative hypertrophy of the muscular walls tends to check the destructive dilatation of the cavity.

We shall find, when we come to study the symptoms of valvular disease, that for a period which varies in different cases, hypertrophy of the heart's walls may entirely counteract the injurious effect of a narrowed orifice or a defective valve: by degrees, however, the result of valvular disease is to cause structural changes in those cavities, which, in the order of the circulation, lie behind the seat of disease. Thus disease of the aortic orifice tends to dilate the left ventricle, so as sometimes to render the mitral valve incompetent, and then to dilate the auricle. Disease of the mitral valve, whether obstructive or regurgitant, acts directly upon the left auricle, and then retards the pulmonary circulation. Obstruction at either the aortic or the mitral orifice tends gradually to act backwards through the lungs upon the right cavities, and to dilate them: thus the ultimate result of a defective aortic or mitral valve may be to dilate the right ventricle and auricle, and to render the tricuspid valve incompetent.

The general tendency of valvular disease, then, is to cause hypertrophy with dilatation, combined in various degrees. When with hypertrophy of the muscular tissue the thickness of the walls and the size of the heart's cavities retain their normal relative proportions, the case is said to be one of *simple hypertrophy*. It is spoken of as *eccentric* or *dilated hypertrophy* when, with thickening of the muscular walls, there is a disproportionate dilatation of the cavity, such as occurs in the left ventricle as the ultimate result of incompetence of the aortic valves. On the other hand, the term *concentric* or *contracted hypertrophy* is applied

to those cases in which thickening of the walls is associated with contraction of the cavity. This may be seen in the left ventricle in some cases of mitral disease. In cases of simple hypertrophy, the form of the heart remains unchanged; but the effect of dilated hypertrophy is to render the heart somewhat globular in form; and in some instances its transverse exceeds its longitudinal diameter. The size and the weight of the heart are also greatly increased. In cases of dilated hypertrophy, the result of aortic valve incompetence and consequent regurgitation, the weight of the heart may be three and even four times the normal weight—the excess of weight being a measure of the increased growth of muscular tissue.

One of the most interesting and important ultimate results of valvular disease, is impaired nutrition of the muscular walls of the heart, consequent upon an impeded circulation through their tissue. The tendency of valvular disease of the heart is to lessen the blood-stream in the systemic arteries, and to increase, in a corresponding degree, the fullness of the systemic veins. The circulation through the walls of the heart is interfered with by a defective valve in the same way as the circulation through every other organ which receives its blood from the aortic system of vessels. Thus there is a defective supply of blood through the coronary arteries and an impeded return of blood by the coronary veins. Since every organ and tissue requires for its nutrition a free movement of blood through its capillary vessels, it is manifest that a defective blood-stream in the *afferent* arteries and an obstructed current in the *efferent* veins will equally, although differently, impair the nutrition of an organ. A defective mitral valve, whether causing a direct impediment to the onward flow of blood into the ventricle, or permitting a reflux from the ventricle into the auricle, tends to diminish the flow through the aortic orifice and the supply to the coronary arteries. Disease at the aortic orifice—more especially such as permits regurgitation from the aorta—has an equal tendency to lessen the supply of blood to the nutrient arteries of the heart. The influence of insufficiency of the aortic valves upon the circulation through the coronary arteries requires a few words of explanation. It is probable that the coronary arteries receive their blood-supply, not directly during the systole of the ventricle, but indirectly during the diastole. This is rendered probable by the retrograde course of the coronary arteries from the aorta towards the heart, and also by the fact that the contraction of the muscular walls of the ventricles during the systole compresses the vessels and prevents the entrance of blood. The muscular tissue in the exposed heart of a living animal is pale during the systole, but becomes dark red during the diastole. It is probable, therefore, that during the diastole of the ventricle, when the aortic valves are closed, the forcible elastic reaction of the aorta drives the blood through the coronary arteries into the muscular tissue of the heart. Now imperfect closure of the aortic valves must obviously influence the circulation through the coronary arteries; for, in direct proportion to the reflux of blood into the ventricle, the tension of the aorta and the consequent pressure of blood in the coronary arteries will be lessened. Then in process of time the continual over-distension of the aorta by the action of the dilated and hypertrophied left ventricle tends to dilate that vessel, to cause degeneration of its walls; and consequent impairment of their elasticity. Here, then, is another cause of lessened blood-supply to the coronary arteries. Thus the overworked left ventricle, receiving a diminished and a progressively diminishing supply of nutrient blood, begins to degenerate, becomes soft, pale, and fatty, and death occurs from a rapidly increasing failure of the circulation, or perhaps suddenly from syncope.

Thus far we have considered the effect of a diminished supply of arterial blood to the walls of the heart. The results of *venous* obstruction are equally striking and important. Sir William Jenner has directed attention to this subject in a paper published in the *Medico-Chirurgical Transactions* (vol. xliii). When the right cavities of the heart are overdistended in consequence of obstruction on the left side of the heart, the coronary veins are gorged, and the muscular tissue of the heart is kept in a state of passive congestion. The result is that, after a time, the walls of the heart become indurated, tough, and leather-like. When examined microscopically, the muscular striæ are generally found to be indistinct. Between the fibres there are innumerable fine granules, some of which, as well as the damaged muscular fibres, have undergone the oily transformation. When the congestion has been extreme and long continued, the coronary veins are often found dilated. Amongst the results of this passive venous engorgement are a considerable accumulation of dropsical serum in the sac of the pericardium, and minute hæmorrhagic spots beneath the serous membrane on the surface of the heart. Less frequently these specks of blood are found beneath the endocardium on the inner surface of the heart.

We will next consider the physical signs of chronic valvular disease. These, of course, vary according to the seat and the nature of the

valvular defect. [On a diagram the chief auscultatory signs of the various forms of valvular disease and the character of the pulse will be found briefly stated.]

FORM OF DISEASE.	MORBID SOUND:		CHARACTER OF PULSE.
	When heard.	Where best heard.	
Aortic obstruction	Systolic.	Third right intercostal space.	Normal.
Aortic insufficiency or regurgitation	Diastolic.	Ditto.	Collapsing; locomotive.
Mitral insufficiency or regurgitation	Systolic.	At apex of heart and at back of chest.	Small; irregular.
Mitral obstruction	Presystolic.	Ditto.	Ditto.
Tricuspid regurgitation . . .	Systolic.	At bottom of sternum.	Normal.
Pulmonary obstruction . . .	Systolic.	Third left intercostal space.	Normal.
„ regurgitation	Diastolic.	Ditto.	Normal.

We will first consider the case of *Aortic Obstruction*, the result of narrowing of the aortic orifice, or rigidity of one or more of the semilunar valves. In these cases there is a systolic murmur heard most distinctly at the base, over the third intercostal space, close to the right edge of the sternum. The sound, when loud, may be heard along the arch of the aorta, at the top of the sternum, and again over the back, at the left vertebral groove opposite the second, third, and fourth dorsal vertebra, but rapidly ceasing downwards. The heart's impulse is increased through hypertrophy of the ventricle. The pulse may be normal in volume, force, and frequency.

Aortic Insufficiency or Regurgitation is indicated by very striking physical signs. The most constant is a diastolic murmur at the base, taking the place of the normal second sound. This murmur is often loud, having a cooing or a harsh sawing character. It is produced by the passage of a reflux current of blood over the free margins of the incompetent valves. In a large proportion of cases, there is some obstruction at the orifice as well as incompetence of the valves. In these cases there is a systolic as well as diastolic murmur at the base; but, although both sounds originate in the same orifice, the systolic murmur, passing onwards with the blood, will be most distinctly heard at a point somewhat higher than the diastolic sound, which appears, as it were, to be carried downwards with the regurgitating blood-stream. The sound is carried by the current of blood, just as the sound of a bell is conveyed by the wind so as to be heard louder and farther in the direction of the wind than in the opposite direction.

The left ventricle being much dilated and hypertrophied, a strong heaving impulse may be felt and seen over a large space. The extent of cardiac percussion-dulness is increased, and the apex beats below and to the left of its normal position, in the sixth or even in the seventh intercostal space.

The pulse is quite characteristic, and is called a *collapsing*, *jerking*, or a *locomotive* pulse. The cavity of the left ventricle, as we have already seen, becomes much dilated while the walls are hypertrophied. The large ventricle receives at each diastole not only the onward stream of blood from the auricle, but also a reflux current from the aorta. The large cavity, thus distended, empties itself forcibly into the aorta; the result is that, for a moment, the whole arterial system is distended, and the pulse feels full; then instantly, with the diastole of the ventricle, the regurgitant current lessens the tension of the arteries, and the pulse sinks away beneath the finger. The pulse, therefore, is well described as *collapsing* or *jerking*. The explanation of the term *locomotive*, as applied to this kind of pulse, is simply this: the superficial arteries, being alternately distended and elongated by the injecting force of the ventricle, and then rapidly contracting during the diastole, may plainly be seen to move beneath the skin. This is especially remarkable where a bend in an artery has its curve increased during the distended and elongated state of the vessel, returning to its former position when the distension ceases. Another peculiarity of the pulse is that, as felt at the wrist, it is in some cases notably behind the systole of the ventricle. The probable explanation of this retardation of the pulse is to be found in the fact, that at the commencement of the systole the artery wants the usual tension and fulness in consequence of the previous reflux of blood into the ventricle; the pulse-wave, therefore, is propagated more slowly than normal.

Mitral Insufficiency or Regurgitation is indicated by a systolic murmur heard most distinctly at the apex of the heart. The sound may also be heard at the back, near the angle of the left scapula, and somewhat lower than the point at which a systolic aortic murmur is best heard. A murmur in the mitral orifice is sometimes heard more distinctly over the back than at the apex of the heart in front. Still more rarely it may be heard at the back when it is quite inaudible in front. In some of these cases, owing to the distension of the pulmonary artery, on listening over the second and third left costal cartilages, the pulmonary valves may be heard to close with an intensified sound;

while the sound of the aortic valves to the right of the sternum is comparatively feeble, owing to the diminished tension of the aorta. The second sound is said to be *accentuated* over the pulmonary valves.

The pulse is small in proportion to the amount of regurgitation through the mitral orifice, and the consequent deficiency of blood in the systemic arteries. Not unfrequently, too, the pulse is irregular and intermitting.

Mitral Obstruction consequent upon narrowing of the orifice has the same effect upon the pulse and upon the relative intensity of sound produced by closure of the pulmonary and aortic valves, as regurgitant disease at the mitral orifice. The diagnostic physical sign of mitral obstruction is a *presystolic* murmur heard best at the apex. The murmur does not take the place of the normal second sound, as in cases of aortic regurgitation. It occurs earlier, and is, in fact, synchronous with the active contraction of the auricle which immediately precedes the systole of the ventricle. The murmur continues while the blood is being driven by the systole of the auricle through the narrowed mitral orifice. It ceases abruptly when the influx of blood into the ventricle is stopped by the ventricular systole. With reference to the auricle, then, the sound has been well designated by Dr. Gairdner *auriculo-systolic*. As regards the ventricle, it is *presystolic*. The sound is usually of a soft character, immediately preceding the systole of the ventricle, and closing abruptly with the systole. Without doubt, mitral obstruction is more common than this direct mitral murmur, although the murmur itself is by no means rare. There are two facts which help to explain the comparative infrequency of a direct mitral murmur. First, the blood, propelled only by the auricle, enters the ventricle in a somewhat feeble stream; and, second, during the diastole, the apex of the heart not being in contact with the wall of the chest, a feeble sound originating in the mitral orifice is less likely to reach the ear.

Although the cases in which there are both obstruction and regurgitation at the mitral orifice are sufficiently common, a *double mitral murmur* is comparatively rare. In a few cases, I have satisfied myself that such a murmur existed. The heart from one of these cases is on the table (case of Maria Johnson, K. C. H., ix, p. 89). It will be seen that, while there is extensive disease of the mitral valve, with a large fibrous coagulum on its auricular surface, there are some small fibrous fringes on the aortic valves, which would in no degree impair their efficiency or impede the blood-stream. The double sound heard during life must have originated at the mitral valve.

Hitherto we have been discussing the physical signs of disease affecting the valves on the *left* side of the heart—the aortic and the mitral valves. In the vast majority of cases, valvular disease occurs on the left side of the heart, and on the left side only. Of the two sets of valves on the right side, the tricuspid is much more frequently defective than the pulmonary. The curtains of the tricuspid valve are sometimes thickened by rheumatic inflammation, or by atheromatous degeneration. More frequently, however, the valve is rendered incompetent by dilatation of the auriculo-ventricular orifice; and this dilatation is usually a result either of valvular disease and obstruction on the left side of the heart, or of extensive emphysema of the lungs. Incompetence of the tricuspid valve may exist without audible sound to indicate its presence. We infer the existence of such incompetence when, at each contraction of the ventricle, a reflux wave is visible in the superficial jugular veins. The auscultatory sign of *Tricuspid Incompetence and Regurgitation* is a *systolic* murmur heard most distinctly at the *bottom of the sternum*, and becoming less distinct or inaudible at the apex of the heart. I have told you that dilatation of the tricuspid orifice, and consequent insufficiency of the valve, is a not uncommon result of valvular disease on the left side of the heart; accordingly, we sometimes find that, by careful auscultation, we can recognise the physical signs of aortic or mitral disease, together with those of tricuspid regurgitation, in the same patient.

Contraction of the tricuspid orifice is an occasional congenital defect, and sometimes a result of disease in after-life; but it is infinitely less common than dilatation; and I am not aware that I have ever heard a *diastolic*, or *presystolic*, or *direct tricuspid murmur*, the result of narrowing of the right auriculo-ventricular orifice.

A murmur originating in the *Pulmonary Orifice* is the rarest of all cardiac sounds. The most frequent causes of such a murmur are, first, congenital malformation and narrowing of the pulmonary artery; second, aneurism at the origin of the aorta, pressing upon the pulmonary artery, and displacing its valves. Upon the table there is a preparation showing a small aneurism of the aorta which has displaced the pulmonary valves, so as evidently to have rendered them incompetent to close the artery. The late Dr. Hope, in his book on *Diseases of the Heart*, has given a drawing of a specimen exactly like the one on the table. In Dr. Hope's case there was a loud superficial diastolic murmur heard most distinctly to the left of the sternum, over the pul-

monary artery. This sound was not heard in the carotids or over the back. The pulse was normal. There was not the collapsing pulse which is so characteristic of aortic regurgitation.

In some recorded cases of malformation of the pulmonary artery, there has been a loud double murmur (systolic and diastolic), most distinctly heard to the left of the sternum, not conducted along the aortic vessels, and not associated with the collapsing pulse of aortic regurgitation.

Now, having discussed at sufficient length the physical signs of valvular disease, let me impress upon you the fact that serious *valvular disease may exist without abnormal sound over the heart*. In cases of old disease of the mitral valve with extreme narrowing of the orifice, it sometimes happens that, although the symptoms of impeded circulation are very manifest, there is no audible abnormal sound. Extreme narrowing of the orifice, with thickening of its rigid margins, and the very rapid action of the heart which usually accompanies extreme mitral contraction, are all unfavourable for the production of an audible murmur. On the table are two specimens of extreme narrowing of the mitral orifice. Both cases occurred in my hospital practice. In neither case was a murmur heard, although carefully sought for. When, with incompetence of the aortic valves, the margins of the valves become covered with fibrinous deposit, a regurgitant current may exist without audible murmur. Fibrinous warty vegetations are sometimes found on the surface of the mitral and aortic valves when no abnormal sound has been heard during life. Mitral regurgitation may so lessen the force and volume of the blood in the aorta as to prevent the generation of sound by an obstructive disease at the aortic orifice. Mitral regurgitation may also lessen or quite destroy the characteristic collapsing pulse of aortic regurgitation.

Reduplication of one or other of the heart's sounds, more commonly of the second sound, is sometimes heard. The probable explanation of this phenomenon is a want of synchronism in the action of the two sides of the heart. A narrow mitral orifice, by retarding the filling of the left ventricle, might lead to its contracting later than the right ventricle. In many cases, however, reduplication of the sound is not associated with signs of organic disease, and the want of synchronism is apparently a result of disordered innervation.

I have said that serious organic disease may exist without abnormal sound. I must now tell you *a murmur may exist without organic disease*. The most common and undoubted form of inorganic murmur is a soft systolic blowing at the base of the heart, which may be heard not unfrequently in cases of anæmia. The thin and watery blood passes through the aortic orifice with an audible blowing sound. The sound is more constant and louder when the heart's action is excited and violent; and, indeed, violent action of the heart, whether the result of muscular exertion or of emotional excitement, is not unfrequently attended with a systolic blowing at the base in persons who are not anæmic. I have frequently observed this in examining nervous candidates for life-insurance; and I find that, after waiting for a short time to allow the emotional excitement to pass away, the heart's action becomes quiet, and the blowing sound ceases.

In children with very flexible chest-walls, firm pressure on the stethoscope placed over the aorta will sometimes cause a temporary systolic blowing.

When a systolic blowing sound at the aortic orifice is the result simply of anæmia, a loud humming sound may often be heard when the stethoscope is placed over the veins at the root of the neck. This venous murmur has received the name of *bruit de diable*. It is a continuous murmur, with periodical intensifications of loudness, the intensified sound corresponding with the more rapid current of blood in the vein during the diastole of the auricle. A deep inspiration also increases the sound by sucking the venous blood more forcibly into the chest. An inorganic cardiac murmur is always systolic, and, according to my experience, always at the aortic orifice. Some writers have suggested that an inorganic systolic murmur may originate at the mitral orifice. I doubt this. A systolic murmur at the mitral orifice would indicate regurgitation through that orifice; and this must result from insufficiency of the valve. Such a murmur, therefore, would not be an inorganic murmur.

A murmur apparently originating in one of the heart's orifices may result from organic disease *outside the heart*. Thus, in one of my hospital patients, a cancerous growth in the left lung surrounded and compressed the pulmonary artery, and so caused a systolic blowing at the base of the heart. (Case of John Campbell, aged 41; admitted April 5th, died April 11th, 1862—Hospital Case-Book, xiv, p. 71.)

In my next lecture I shall speak of the general symptoms, the prognosis, and the treatment, of valvular disease and its consequences.

IS INSANITY ON THE INCREASE?*

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THE popular opinion undoubtedly is, that insanity is increasing greatly in this country. The necessity, year after year, of enlarging the existing county asylums; the erecting of a second, or even of a third asylum in some counties, and of special borough asylums for large boroughs; and the still continuing cry for more accommodation, are facts sufficient to account for, and give much show of probability to, the opinion. In the county of Middlesex there are already four vast asylums for pauper patients; and yet the Commissioners in Lunacy have for some time earnestly pressed upon the magistrates of the county, and upon the Home Secretary, the urgent necessity of taking immediate steps to build another large asylum. There can be no question that there are a great many more insane persons shut up in asylums in this country now than there ever were at any other period of its history, or, perhaps, in any country at any period of the world's history. For the last twenty years there has been an increase of upwards of a thousand a year in the registered insane population of England and Wales. Now, whether any, and, if any, what proportion of this great increase is owing to an actual increase in the proportion of lunacy to the population, are questions which may be more easily disputed than decided. There is always a large number of persons who are prompt enough to grumble at the age in which they live, and to believe readily any fault of it; we need not wonder, therefore, if the alleged increase of insanity is accepted without sufficient proof. Once, too, the opinion has gained acceptance, it is no hard matter to discover a multitude of plausible reasons why a greater number of persons should go mad now than in the days of our grandfathers. The fast pace of living, and the strain of eager competition at the present day, are sure to find out the weak members of the community, and to try them severely; the law of the success of the fittest pressing very hardly upon them. Still it seems to me that there is a tendency to overlook the countervailing advantages of modern life. By the exercise of a little ingenuity, it would be possible to adduce as many theoretical arguments to prove that insanity need not, as can be brought forward to prove that it must, increase. Let me give a few illustrations. If it be said, on the one side, that the excitement of financial speculation must injure the brain, it may be replied, on the other side, that the business of a man in the world is activity; and that as mental exercise is an indispensable condition of mental development, such activity is far better fitted than an indolent stagnation to preserve health of mind. Moreover, while the latter will not prevent a person who is exposed to an ordinary cause of insanity from going mad, a mental activity directed into channels outside himself sometimes will. Statistics do not say much in favour of agricultural immunity. In the county of Flint, on the 1st January, 1871, the ratio per 1,000 of the total number of pauper lunatics to the population was 4.64, while in the West Riding of Yorkshire the ratio was 1.42; in the county of Carmarthen the ratio was 3.04, while in the county of Lancaster it was 1.94; in Hereford it was 3.42; in Berks, 3.15; in Gloucester, 3.05; in Wilts, 3.22; while in Chester it was 1.79; in Surrey, 2.34; and even in Middlesex not more than 2.86.†

Again, it may be true, as is sometimes alleged, that the eager passions and interests arising out of the multiplied industries and complex relations of modern life are likely causes of insanity; but it is no less true that these passions are fundamentally the old simple passions inhabiting human nature, savage or civilised, which have undergone more special development; and they are presumably less dangerous, more divided in their energies, and more social in their aims, than when weighing heavily on the heart of the individual who has few or no interests outside himself.

It has been said that the rapid and frequent railway travelling of this age must be injurious to the brain, and predispose to disease of it; but those who say so, while assuming much of what they have no proof, do not sufficiently consider that railways have, by opening up remote parts of the country and facilitating intercommunication between the in-

* Read at the quarterly meeting of the Medico-Psychological Association, on December 6th, 1871.

† These figures are from the last Report of the Commissioners in Lunacy, which contains some valuable statistical tables.