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TWO HARVEIAN LECTURES

ON

BRIGHT'S DISEASE AND ITS TREATMENT:

CONSIDERED MAINLY IN RELATION WITH ARTERIAL
TENSION FROM BLOOD-CONTAMINATION.

Delivered before the Harveian Society of London, November 1875.

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LECTURE I.

MR. PRESIDENT,—When you so kindly conveyed to me the invitation of the Council of this Society to be its first Harveian lecturer, I felt that this great honour was, if possible, increased by being conveyed through yourself, my dear and frequent fellow-worker in the wards of St. Mary's Hospital.*

The illustrious name and labours of Harvey have been so richly illustrated by the long roll of distinguished Harveian orators at the Royal College of Physicians, that I need not occupy your time by speaking of this great physician, whom we all honour as one of the two or three greatest advancers of medicine in England; but I shall make the subject of my lectures the relation of Bright's disease to those organs, the true physiology of which Harvey did so much to establish.

Before I begin with the subject of these lectures, I would pay my sincere homage to the young school of medicine, of which school you, sir, are so eminent an example, that is labouring in this country and abroad to enlarge and fix our knowledge of disease and of the action of remedies; and that, not by the pursuit of a vague and superficial routine, but by rigid and scientific investigation. When I spoke, Mr. President, of the young school of medicine, I turned to them from the great name of Harvey; and I regard them as the lineal descendants and heirs of his labours, and of the labours of all true workers in our profession. The workers of the present day stand upon the platform that has been erected by the labourers of the past, and they work upwards from that platform. They have thus this immense advantage that, while they receive all the knowledge and traditions of their predecessors, they advance forwards into new fields of knowledge; and, in doing so, they test and accept the truths and they test and reject the errors of the past; and the results of every-day experience are jotted by observation, science, and reason.

Bright's disease, whatever its force, acute or chronic, fatty, granular, or lardaceous, by interfering with the function of the kidney, lessens excretion of the broken-up tissues, and, by causing an undue accumulation of that *débris*, contaminates the whole volume of the blood circulating in the body. The escape or the non-escape of the albumen with the urine is not, I need scarcely say, the essential part of the disease. The weeping of the albumen in the acute fatty and lardaceous forms of the affections leads to disintegration of the blood and wasting and softening of the tissues; and the non-escape of the albumen, in cases of granular kidney, saves the blood and the tissues; but in both sets of cases a contaminated fluid circulates through every part of the body; affecting on the one hand the nervous system, and all the structures; on the other, rendering the flow of blood difficult through the smaller vessels. The blood is, therefore, unduly retained in the arteries, and they become tense. The quantity of blood may be great, as it often is in granular kidney; or it may be small, as it often is in fatty or lardaceous kidney; but whether the blood be increased or diminished in quantity, the arteries, be their diameter increased or diminished, contract firmly on their fluid contents, and are tense.

* These lectures were delivered during the presidency of Dr. Broadbent.

The influence of this tense state of the arterial system bears backward upon the heart, inducing enlargement and hypertrophy of the left ventricle, forwards on the capillaries, and immediately on the whole arterial tree, from the root of the aorta to the finest arterioles.

I shall examine in this lecture the physical evidence of arterial tension in Bright's disease and its influence on the heart and arteries; and in my next lecture I shall consider the nature of the sphygmographic tracings characteristic of this disease, and those cases that present remarkable arrest and accumulation of those secondary influences that tend to lessen arterial tension, and then pass to the treatment of the disease.

Acute Bright's disease from cold, when it attacks, as it usually does, those who are young and free from disease of the heart or arteries, presents us with albuminuria, from affection of the kidney, in its most simple form; and the clinical study of that form of Bright's disease is, therefore, more easy and free from complication than any other. Of nine fatal cases of acute Bright's disease, not due to scarlet fever, in three the heart was large and in four it was very large, the left ventricle being usually hypertrophied. There was no complication in any of these cases of general disease primary to and preceding, or secondary to or following, the acute Bright's disease. There was no other influence, besides the actual hæmorrhage and weeping of albumen from the kidneys, to cause wasting of the tissues or destruction as well as direct loss of the red corpuscles. The wasting influences of the loss of red blood-corpuscles and albumen, and of the saturation of the tissues with serum, exercised, however, a strong influence tending to cause wasting of the heart as well of all the tissues. In spite of these wasting influences, however, the heart was enlarged in all those cases, and was greatly enlarged and hypertrophied in more than half of them; and there was, therefore, demonstratively some powerful cause at work, overriding the wasting influence of the disease and causing great enlargement of the heart. The two cases of acute Bright's disease not from scarlet fever, in which the heart was natural in size, would seem to say at first sight that the wasting influence of the disease is *per se* sufficient to balance the heart-enlarging effect of the disease; but the clinical history of those two cases removes them altogether from the cases of primary acute Bright's disease, and places them, like those from scarlet fever, among the secondary forms of the disease, for, in one of them, the affection of the kidney followed an attack of small-pox, which occurred some weeks previously; and in the other case, the acute disease of the kidney came on during an attack of acute rheumatism, and was accompanied by purpura with fatal hæmorrhage from and into the stomach and bowels.

The accompanying valuable table of fatal cases of acute Bright's disease, drawn up by my friend Dr. Greenfield, who kindly supplied me with it for my Lumleian lectures, gives an important illustration of the principles just stated. Dr. Greenfield gives nine cases of acute Bright's disease, in which there had been no scarlet fever. In three of them, the heart was rather large; in five of them, it was very large; and in one, its size is not noted. In seven of them, the left ventricle was hypertrophied, and in two its thickness was not noted; but, in one of these two, the heart weighed twenty-seven ounces, and, in the other one, twelve ounces. Two of his cases had ascites, while one of these two had pleural hæmorrhage; and in one of these the heart weighed eleven ounces, and in the other twelve ounces, so that the tendency of the heart to increase in size, owing to the acute Bright's disease, was manifestly held in check, not only by the exhausting loss of blood due to the disease itself, but also by the secondary or other affections implanted upon the disease.

The St. Mary's Hospital cases of acute Bright's disease from scarlet fever number only five; and in two of these the size of the heart was natural, in three it was rather large, and in none of them was it very large. Dr. Greenfield's cases of this class amount to seven; and in them, as in those in my tables, the heart is much less frequently enlarged than in his cases of primary acute Bright's disease. Thus the heart was small in one of his cases, was of natural size in two, was large in three of them; while in one its size was not noted, but as in that one the left ventricle was of moderate size, it may be inferred that

the dimensions of the heart were natural. The left ventricle was hypertrophied in three of Dr. Greenfield's cases. These cases afford clinical evidence that, in acute Bright's disease from scarlet fever, the wasting effect of the primary disease, added to the wasting effect of the drain of red corpuscles and albumen, tend to counterbalance the natural influence of the disease of the kidney, to cause hypertrophy of the left ventricle and enlargement of the heart. Such influence is, however, actively at work, since, but for the power that is exercised, the heart, like the rest of the body, would have dwindled in size, whereas it was larger in one-half of the cases.

The cases of Bright's disease with fatty or large white kidney, in which the heart was rather or very large, which amount to two-fifths of the whole number of those cases (eight and fourteen respectively, or twenty-two in fifty-four) give an exact repetition of the clinical lesson afforded by the cases of acute Bright's disease, from which disease, indeed, the fatty kidney springs. Thus, among the whole of those cases, in only one instance was a secondary wasting disease, in this case phthisis, added to the primary and continuous waste of albumen, due to the kidney affection. In none of these cases was there notable emaciation of the body; the true wasting of the tissues being marked in many of them by the general dropsy.

The cases of lardaceous kidney and of suppurative disease of that organ, due to affections of the bladder and urethra, with enlargement, need not occupy us here; for they are few, and are given in the table in your hands, and correspond in general tendency with the cases of the same kind with fatty kidney.

The valuable clinical labours of Dr. G. Johnson, Dr. Dickinson, and many other observers, and everyday work at the bedside, have made us all familiar with the great vital differences between the two important forms of Bright's disease—the fatty kidney, with scanty highly albuminous dense urine and general dropsy; and the granular kidney, with abundant watery urine containing traces of albumen, and with little or no dropsy. The kidneys refuse to do their proper work in both forms of the disease, so that in both of them the blood becomes poisoned with the retained and accumulated *debris* of the tissues, and the arteries are therefore rendered tense. While, however, the granular kidney parts freely with the water of the blood, and the body, being thus drained, is free from dropsy, the fatty kidney sets free the albumen of the blood, retaining its water, which, escaping from the tense arteries, saturates the structure with general dropsy. The tissues are thus rounded and softened in cases with fatty kidney, the heart being enlarged, as we have just seen, in only two-fifths of them; while the tissues remain firm and are not wasted in cases with granular kidney, the heart being enlarged in three-fourths of the cases.

The frequent occurrence of hypertrophy of the left ventricle in Bright's disease, and especially in cases with granular kidney, is so well established that it is sufficient if I here refer to it as the heading of this section of my lecture. The frequency of the occurrence of great enlargement of the heart in the cases of Bright's disease, amounting to 277, examined after death in St. Mary's Hospital, during the first nineteen years of its existence, and the relative size of the heart, in 248 of those cases in which the size of the heart is stated, is exhibited in the table which I place in your hands.

The heart was very large, the left ventricle being almost always hypertrophied, in two-fifths of those 248 cases in which the size of the heart was stated (107); it was large in one-fifth of them (47); it was of natural size in more than one-fourth of them (67); and it was small in one-ninth of those cases (27).

There were some cases in which the heart was very large, some in which it was of the natural size, and some in which it was small (except in acute Bright's disease), in each of the different forms of kidney disease that are rightly grouped under the common name of Bright's disease. The proportion, however, in which the heart was affected depended very much on the different forms of Bright's disease, as will be seen from the following brief comparative analysis of those cases.

The heart was very large in nearly three-fifths (60 in 106), large in nearly one-fifth (20 in 106), of the cases with contracted granular kidney; while it was of natural size in a little over one-fifth (25 in 106), and was small in only one in twenty-six (4 in 106) of those cases; the comparison being necessarily limited in this and in the other groups of cases to those instances in which the size of the heart is stated. The proportions are very different in cases of fatty or large white kidney, following upon acute Bright's disease, since among these the heart was small in more than one-fourth of the cases (15 in 54), and was of natural size in nearly one-third of them (17 in 54); while it was very large, the left ventricle being nearly always hypertrophied, in nearly one-fourth of the cases (14 in 54), and was large in fully one-seventh of them.

Cases of lardaceous disease of the kidney, and those with disease of that organ, chiefly suppurative, due to stricture of the urethra or disease

of the bladder or prostate, rank nearly with cases of fatty kidney as regards the size of the heart. Thus the heart was small in nearly one-fourth (3 in 14 and 3 in 13), and of natural size in one-half (7 in 14 and 7 in 13 respectively), of each of those forms of Bright's disease; while it was very large in nearly one-fourth (3 in 14), and large in one of the cases with lardaceous disease of the kidney; and it was very large in only one and large in two of the thirteen cases with suppurative disease of the kidney from stricture or affection of the bladder or prostate.

Acute Bright's disease stands alone in not presenting every variety of size of the heart, since, while in every other form of the disease we are considering the heart was small in a greater or less proportion, in the acute form the heart was not small in one instance; whilst it was very large in fully one-fourth of those cases (four in fourteen), it was large in nearly one-half of them (six in fourteen), and was of natural size in but a little over one-fourth of them (four in fourteen).

We have just seen that, in each form of Bright's disease, except the acute, though much less frequently when the kidney is granular, the heart in certain cases is unduly small; and that, in each form of Bright's disease, though much more frequently when the kidney is granular, the heart in certain cases is unduly large; and I shall now inquire whether, in those different forms of the disease, there are any points in common that may account respectively, on the one hand, for the diminution, and, on the other, for the increase of the size of the heart.

There are two influences at work that appear, from clinical evidence, to modify the size of the heart; one of these is wasting disease or emaciation, whether induced by the great weeping of albumen and the disintegration of the blood, or by the escape of serum into the tissues; the other is, on the one hand, incompetence of the mitral or aortic valve, whether from intrinsic affection of the valve, from enlargement of the left ventricle, or dilatation of the aorta; and, on the other hand, disease of the aorta and the arteries.

It will be seen that these two influences, which are themselves usually caused by the kidney-disease, exercise an important and opposite effect on the size of the heart, and especially of the left ventricle; and that, while the wasting disease and emaciation tend to lessen the size of the heart, valvular disease of the heart and disease of the aorta and arteries tend to increase the size of the heart, and especially the left ventricle, the right ventricle being also implicated when the mitral orifice is incompetent.

I shall first inquire into the influence of the large weeping of albumen from the blood, disintegration and imperfect repair of the red corpuscles, wasting disease and emaciation, and dropsy, on the size of the heart; and I shall first examine those instances in which the heart was small and of natural size, and then those in which the heart was enlarged, the left ventricle being hypertrophied.

The heart, which was small in one case in nine of the whole number (27 in 248), was so in only one in thirty of the cases with granular kidney (4 in 128); but in one in four of all cases of fatty (15 in 54), lardaceous (3 in 14), and suppurative (3 in 13) kidney, and kidney with calculus (2 in 8). In all but two of these cases, there was general disease of an exhausting character, such as phthisis (12), low form of inflammation (2), empyema and diarrhoea (3), or ascites (1), or caries of vertebrae (1), or abscess of the kidney itself, and in all but one or two there was emaciation. Thus, taking it for granted that the natural influence of the poisoned blood and arterial tension was to cause enlargement of the heart, there was in these cases an exhausting influence tending to emaciate the body and to lessen the size of the heart in general disease, added to the constant drain of albumen in all the cases, except the four with granular kidney.

The heart was of natural size in one in four of the whole of the cases of Bright's disease (63 in 248), in one in four of those with granular kidney (26 in 128), in one-third of those with fatty kidney (17 in 54), and with acute Bright's disease (4 in 14), and in one-half of those with lardaceous and suppurative kidney. About three-fourths of these cases (46 in 63) presented cerebral diseases or affections of the heart and arteries, which constitute the second influence that apparently tends to modify the size of the heart in cases of Bright's disease. This influence, looked at as a question of simple statistics, according to the evidence supplied by the table of fatal cases examined in St. Mary's Hospital, seems to exert the exactly opposite effect to that first influence of which I have just spoken—wasting disease and emaciation.

If we look, for instance, at the fifty-four cases with fatty kidney, we find that, in the group of fifteen in which the heart was small, in none of these was there valvular incompetence; in two of them, there was slight atheroma of a valve; and that in all the rest the heart was healthy; and not one with partial pericarditis; while, in these cases, the number was exactly increased in relation to the influence of wasting

disease, or thirteen, or all but two of them, were thus affected or were emaciated. If we now turn to the other end of the scale, we find that, among the fourteen cases in which the heart was enlarged, often to a great degree, there was wasting disease or emaciation in no single instance, while there was valvular incompetence in six, and organic pericardial adhesions in one, of the cases; there was atheroma of the valves or the aorta in four instances, and in only three was the heart healthy. Putting aside the great increase of size, we thus see that, as regards these two influences, there is an exactly opposite polarity.

In the large and important class of cases with granular disease of the kidney, including those in which the kidney was enlarged with cysts, was natural in size, and was contracted, we still find the two contrasted influences, each exercising its proper forces at, so to speak, the opposite pole. Thus, among the four cases with small hearts, none had affection of the heart and arteries, and all had wasting disease; and, among twenty-nine with a heart of natural size, three had valvular incompetence and eight had simple atheroma of the valves or aorta; while three-fourths of these, or twenty-two, had wasting disease or emaciation. These relative numbers are nearly reversed, but not so closely so as in the cases with fatty heart; among the seventy-five patients in whom the heart was large or very large, one-third of these, or twenty-four, had valvular incompetence or organic disease of the heart, not including pericarditis; one-third, or twenty-six, had atheroma without valvular incompetence of the valves, aorta, or coronary artery; and in one-third, or twenty-four, the heart was quite healthy, except as regards its size.

These records afford conclusive clinical and after-death evidence that, when the heart is greatly enlarged and the left ventricle is hypertrophied in cases of Bright's disease, the heart and its valves, and the aorta, are much more frequently diseased than when the heart is of natural size or small. I have spoken of this diseased condition of the heart and its valves and the arteries as an influence tending, in conjunction with Bright's disease, to enlarge the heart. The threefold question must here present itself to us, if we would make a scientific estimate of these facts, first, Do the valvular and other diseases of the heart, and the affection of the aorta, added to the Bright's disease, tend to increase the size of the heart? or (secondly) does the proved power of Bright's disease to cause enlargement of the heart, also cause, firstly, valvular incompetence from the increase of the size of the heart and the aorta; secondly, cause fibroid and fatty degeneration and atheroma of the walls and valves of the heart and of the aorta; and, thirdly, inflammation of the interior and the exterior of the heart? or (thirdly) are these three classes of conditions—the occurrence of the Bright's disease, the enlargement, namely, of the heart, on the one hand, and the disease of the heart and its valves and that of the aorta, on the other—the coinciding affection due to a common cause? I believe that an affirmative answer must be given to each of these questions in different cases. I shall take it for granted that the occurrence of disease of the valves of the heart and of the heart itself had an influence in many cases, in concurrence with the Bright's disease itself, to induce enlargement of the heart; for we know that, when there is no Bright's disease, those morbid changes directly tend to induce enlargement of the heart; and we see, as I have shown in these tables, the frequency of the occurrence of valvular and aortic disease in the cases of enlarged heart, and their rare occurrence when the heart is small or of natural size.

The second question—Does the Bright's disease ever, or generally, cause the affection of the valves of the heart and of the aorta?—is a very important one, and brings me face to face with a subject that is now causing much discussion.

In certain cases, with great enlargement of the heart, the valves are incompetent, although their structure is healthy, and these cases may occur in every form of established Bright's disease. Thus, in a patient of Sir James Alderson affected with fatty or lardaceous kidney, the right kidney weighed ten ounces, the left eleven ounces; the heart weighed twenty-one ounces; the walls of the right auricle and ventricle were exceedingly thin; the valve-flaps were healthy, but the valve was incompetent, owing to the size of the aperture. In another case, under my care, a man aged 47, with fatty disease of the kidney, the heart weighed twenty-three ounces; the mitral valve was only slightly thickened; the left ventricle was large, and its walls were very thin; and the mitral valve would nearly close under the influence of the pressure of water.

The aortic valve, like the mitral and bicuspid, sometimes becomes incompetent under the influence of the dilatation of the aorta, owing to the great arterial tension induced by the contamination of the blood in Bright's disease, and the consequent obstruction to the flow of blood through the smaller vessels. In one case that I watched for years, an aortic regurgitant murmur came into play towards the end

of the case. The regurgitation was not great, and, when the tension and size of the aorta lessened, it disappeared for a time to reappear when the aortic tension again increased. Thus the occurrence of the incompetence, by relieving the man, again restored the competence.

Atheroma, thickening, which may be either atheromatous or fibroid degeneration, and fibrinous concretions, usually from endocarditis affecting the mitral and aortic valves, and atheroma of the aorta, were present in a certain number of cases of Bright's disease, scattered through the whole of the former affection.

Atheroma and Thickening of Valves.—In one of the St. Mary's Hospital cases of acute Bright's disease, a man aged 47, with great enlargement of the heart (sixteen ounces), the mitral valve was thickened. In another of them, the aortic valve was fatty, the aorta being much dilated; but, as this patient was an old man aged 73, and as the acute attack was in this case evidently implanted upon old standing granular affection of the kidney, this case cannot here be classed with those of acute Bright's disease.

Among Dr. Greenfield's well observed nine cases of acute Bright's disease, not from scarlet fever, there is one patient (a girl aged 14) in whom there were concretions on the mitral valve, which was very thick, so that in this case there was evidently endocarditis affecting that valve and producing thickening of its flaps. Among Dr. Greenfield's seven cases of acute Bright's disease from scarlet fever, there is one patient (a girl aged 8) who presented, as in the instance just quoted, a small vegetation on the mitral valve, with thickening of its flaps; and in this case, as in the other, endocarditis had evidently produced these effects. In another patient of this group (a girl aged 13), there was opacity of the mitral valve and commencing atheroma of the aortic valve; and in this case the changes in those valves were, doubtless, due to endocarditis.

These three cases observed by Dr. Greenfield are especially important; for in them we can with precision attribute the inflammation, vegetations, thickening, and atheroma of those valves to their parent cause. We have already seen that, in acute Bright's disease, the heart is greatly enlarged, owing evidently to great arterial tension, due to the difficulty of sending the poisoned blood through the smallest vessels. It is the natural effect of the struggle of the heart to carry on its over-work.

Fibrinous Concretions.—The fatty or large kidney grows out of acute Bright's disease, being the chronic stage of the acute affection when it does not end in recovery. The fatty kidney is, therefore, like its parent affection, a primary kidney-disease. It is, indeed, more invariably a primary kidney-affection than acute Bright's disease, which sometimes attacks a granular kidney, and, when it does so, I need not say that the original disease remains unchanged after the acute attack has disappeared. We may, therefore, look to cases of fatty kidney to teach us the effect of the kidney-disease, in its simplest forms, in producing secondary affections of the valves of the heart. There were concretions on both the mitral and aortic valves in two, on the mitral valve alone in two, and on the aortic valve alone in one of the fifty-eight cases of fatty kidney; and we may, therefore, conclude that one in ten of those cases were affected with endocarditis. Again, the mitral and aortic valves were both thickened or atheromatous in two instances, the mitral valve alone in seven, and the aortic valve alone in one instance. We thus have an aggregate of fifteen cases in fifty-eight, or fully one in four of the whole, in which one or other, or both of the valves of the heart were thus affected in cases with fatty kidney. The heart was large or very large in eight of these cases; was rather large in three; was of natural size in one, and was small in two of those cases, and in one its size was doubtful. It is probable that, in the majority of these cases, the affection of the valve, probably, as a rule, inflammatory in its first start, was secondary to, and caused by, the Bright's disease. It is impossible, however, to say in what proportion of these cases the affection of the valve was secondary to, or caused by, the affection of the kidney; for the great majority of these patients were of mature age, and it is probable that, in a fair proportion of them, the affection of the valve was primary.

[To be continued.]

ALARMING reports were presented to the metropolitan asylums' managers on Saturday, showing the progress of small-pox in London. At Homerton, not only had the small-pox hospital three over its number, but the fever hospital has been pressed into the service, and 233 out of 236 beds were occupied. Of 826 beds provided, 813 were filled. Resolutions were adopted, requesting the Local Government Board to communicate with the Privy Council, with a view to steps being taken by the sanitary authorities to provide accommodation for small-pox patients not coming within the category of paupers.