THE LUMLEIAN LECTURES ON BRIGHT'S DISEASE.

Delivered before the College of Physicians.

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LECTURE III.—PART II.

THE HYPERTROPHY OF THE HEART IN BRIGHT'S DISEASE.

Bamberger's statistics show that hypertrophy of the heart was encountered by him in 344 out of 807 cases of primary Bright's disease, or in 42.6 per cent. He does not tell us that he excluded amyloid cases from the category; and, from his figures alone and our own experience, we should infer that the proportional occurrence of this complication would have appeared higher if he had done so.

The special form of the hypertrophy, and its incidence and relative occurrence in different stages or forms of the disease, are shown by the following table.

Form of Bright's Disease.	Excentric Hypertro- phy of whole Heart.	Eccentric Hypertro- phy of Left Ven- tricle.	Simple Hypertro- phy of whole Heart.	Simple Hypertro- phy of Left Ven- tricle.	Dilatation of whole Heart.	Dilatation of Left Ventricle.	To- tals.
Acute.....	3	4	2	2	4	0	15
Chronic....	51	33	3	24	5	1	122
Atrophic....	88	65	6	39	7	2	207
Totals ..	142	107	11	65	16	3	344

It will be perceived that, in 41 per cent. of the entire cases of excentric hypertrophy, both sides of the heart were affected; but Bamberger informs us that in 48 out of these 142—one-third, that is, of the cases—notable emphysema of the lungs existed to help to explain it. Excentric hypertrophy of the left ventricle alone befel 31 per cent. of all the cases. Why both sides of the heart should undergo muscular hypertrophy, when it is pleaded that the left ventricle only has an extra task to fulfil, is not at first very apparent. Pulmonary obstacle doubtless is the chief explanation of hypertrophy of the right heart; and emphysema and bronchitis and pleuritic mischief are the commonest incidents to the history of Bright's disease. More than this, however, may be said upon the subject. My own experience teaches me that the cases in which the renal disease has lasted longest, and has been longest maintained at a period of standstill at comparative health, are those in which both sides of the heart have been most hypertrophied; and I am, therefore, inclined to explain the occurrence upon gradual establishment of the hyper-nutritive changes in the heart's structure, which would affect both sets of chambers pretty uniformly. The theories which have been broached to explain this hypertrophy offer no inconsiderable interest.

Dr. Bright's Theory.—The altered state of the blood (he means its impurification, not its hydræmia) exerts an unwonted stimulus upon the muscle of the heart; and it also offers an obstacle to the easy transit of the blood through the smaller blood-vessels, and therefore compels greater exertion upon the left ventricle to effect its circulation. Nothing, apparently, could be clearer than this explanation; but it fails to grapple with many of the facts of which later observations upon the disease have informed us.

This hypertrophy of the heart, as Bright correctly noticed, especially occurred with the contracted and indurated kidney. Later observations have proved that, in this particular form, excess of water passes through the system; and the task of urinary depuration is effected in a method unusual to what it occurs in healthy bodies; the kidneys requiring flushing through with much more than normal water, to enable them to carry off the requisite solids and effete materials. Still they do so; for the blood is maintained pure as in health. So long as the heart works well, what remains of the kidney fulfils the whole task; and not merely is there no diminution of the excretion of urea *per diem* according to the individual's mean average allowance, estimated by weight and nutriment, but what occurs is that actually some-

times the excretion is in excess of what is normal. Dr. Grainger Stewart, at page 218 of his work, in his chapter on Atrophic Kidney, relates such a case. "The amount of urea excreted", he writes, "was nearly as high as the maximum of the tables given by Dr. Parkes." The same fact is well brought out by Bartels's observations; so that, when no maintained impurity of the blood can be assumed to have existed, to have stimulated the heart or obstructed the passage of the blood through the vessels, the greatest amount of hypertrophy is found to exist. Further, against the theory of impurity of the blood, which must be vouchsafed to exist in other forms of Bright's disease indisputably associated with an impure blood, stands the fact that, in many other acute and chronic diseases besides Bright's disease, the blood is very impure, or maintained abnormal, and yet these do not bring about hypertrophy of the heart.

Traube's Theory.—Traube offered a double explanation. First, he attributed the hypertrophy to obstruction of the peripheral circulation at one spot—the kidneys themselves—and excess of water in the blood. He urges the impermeability and destruction of so many blood-vessels as takes place in renal disease as the obstacle offered to the blood-passage; and that, like the shutting of a sluice-gate upon a stream, this obstruction must lead to increased pressure upon the channels and banks above it, and especially to the accumulation of excess of water in the blood, since this cannot run off. According to him, the order of events runs thus: obstructed circulation, increased arterial tension, supplemented or not by abnormal hydræmia, lead to dilatation and hypertrophy of the heart. He places, as Bartels writes, "the consequences of renal contraction in the same category with the results which valvular deficiency of the mitral valve and certain diseases of the lung indisputably exert upon the right chamber of the heart."

We cannot enter at this time and in this place into the relative merits of the objectors, upon one score or another, to Traube's theory. It has received and deserved ample discussion at the hands of Bamberger and von Buhl most recently; and by Campana, Erichson, Ewald, Gull and Sutton, Lebert, Rosenstein, Schrötter, and Senator, in the last few years.

The matter is summed up by Bamberger (*loc. cit.*, p. 1554) with great justice. He writes that in its totality it is untenable on three grounds. 1. This hypertrophy by no means accompanies the kidney which presents greatest destruction of its vascular channels, to the exclusion of all other forms of Bright's disease. 2. It occurs when there can be no question of any obstacle to the amount of the circulation through the kidney; for injection-fluids pass readily enough, as in the small contracted kidney they undoubtedly do. 3. Mere obstruction to the passage of the blood will not suffice to explain it, either situated at the Malpighian tufts, because this is quickly remedied by collateral blood-direction in the kidney itself through the arteriole rectæ; or placed anywhere else in the body by tying an artery, because of the ample room offered to collateral blood-flux. Cutting off a limb, which is surely suppression of a large vascular area, will not produce it; and, as Rosenstein showed, a dog, one of whose kidneys was removed, presented, many months afterwards, no hypertrophy of the heart. 4. Traube's theory, if it throw any light upon the hypertrophy of the left heart, throws none upon the hypertrophy of the right heart that frequently accompanies it.

Dr. G. Johnson's Theory.—In his early writings, was very much the same as Bright's—impurity of the blood; but later on he referred the hypertrophy of the heart to the alterations that were brought about throughout the arterioles generally; their stopcock action stimulated by the noxious fluid in the blood; their hypertrophy; and the greater and greater resistance which they come to offer to the passage of the blood. (*Med.-Chir. Trans.*, vol. li, 1858, p. 60.) According to him, a noxious quality of blood is so disagreeable to the capillaries, that they, through the nervous system, urge the arterioles to forbid its passage. Incapable of saying "No" themselves to it, they bid the regulators of the blood-supply—the arterioles—resist it; and this resistance stirs or works up the left ventricle to pump with redoubled vigour. Let us, however, quote Dr. Johnson's own words. At p. 61, he writes: "In proportion to the destruction of the renal gland-cells, and the consequent diminution of the secretory power of the kidney, there is less demand for blood to be acted upon by the gland; the small arteries consequently contract upon their contents so as to maintain the balance between the blood-supply and the diminished secretory action of the kidney. This continued overaction of the small arteries in antagonism to the heart results in hypertrophy of their muscular walls." Again, p. 65: "Action and reaction are equal; force must be met by an equivalent amount of force; and we shall probably find that the greater the hypertrophy of the left ventricle of the heart, the greater and the more general is the hypertrophy of the arterial walls in various tissues and organs throughout the body." At p. 62, he states:

"Although the muscular walls of the arteries are hypertrophied in the advanced stages of all forms of Bright's disease, the purest specimens of hypertrophy are found in the small granular kidneys." And here, as he truly remarks, there is least impediment to the circulation through the kidney itself.

His theory, then, places the cause of the hypertrophy of the heart in the hypertrophy and resistance offered by the general systemic arteries, not by those of the kidney alone. But, as may be seen by reference to what we have already quoted in discussing the hypertrophy of the coats of the arterioles, Dr. Johnson had already constituted the hypertrophy of the heart the cause of the hypertrophy of the arterioles. But surely it is more likely, or at least as likely, that the hypertrophy of the heart is provoked by increase of blood-tension independently of spasm of the arterioles; and that the alteration in these small arteries is, as it obviously is in aortic regurgitant disease, secondary to the hypertrophy of the heart; and, as Bamberger justly objects, it fails to throw any light on the hypertrophy of the right heart.

Von Buhl's theory (*Mittheilungen aus der Pathol. Institut. in München, 1878*) is that the affection of the heart is due to an inflammatory process taking place in the heart, and stimulating it to overaction. He thinks that the granular kidney and the hypertrophy of the heart occur coincidentally, but wholly independently of each other, and own some common source of origin, and that this is some subacute wide-spread inflammatory action. He found evidence of such past inflammatory changes in the endocardium or the valves, in the heart's muscular substance, and in the pericardium, in 65.7 per cent. of all cases of granular atrophy of the kidney. He also found relative constriction of the aorta, or an abnormal ratio between the circumference of the aorta and the length of the left ventricle, which he regarded an accessory stimulator of the hypertrophy of the heart. But then he leaves wholly unexplained a great many cases of hypertrophy of the heart, in which he can find no traces of past inflammatory action, and refers them to fatty degeneration.

Sir W. W. Gull and Dr. Sutton's theory refers the hypertrophy of the heart to a change in the blood-vessels of the heart itself, similar to that which they observed and described as occurring in various parts of the body, their arterio-capillary fibrosis; a change which, occurring in the kidney, led to granular atrophy; in the dura mater, brain, and spinal cord, to various manifestations of sclerosis. Their publication appeared before that of Von Buhl, and perhaps suggested in some measure the lines of his inquiries and the opinions at which he arrived. It certainly serves to explain the occasional hypertrophy of both sides of the heart, and many of the wide-spread pathological lesions, bronchitic, pneumonic, hepatic, and cerebro-spinal symptoms and complications sometimes manifested. Further, that a wide-spread increase of nuclear interstitial tissue in the capillary meshwork is one of the most manifest pathological changes that occur in chronic Bright's disease, and especially in the very chronic forms of atrophied kidney, we can entirely confirm by our own experience, and prove by abundant microscopical specimens in our possession. But, at the same time, we are bound to state our conviction that the muscular hypertrophy of the arterioles is an equally well-established fact, which Gull and Sutton cannot be supposed to have either overlooked or denied, and is a matter wholly independent of the thickening of the capillaries and of the external tunic of the artery, which it usually, but not invariably, attends. Why it was not more prominently alluded to by them, we cannot tell. But we have seen marked thickening of the capillary walls in the kidney when little or no noticeable hypertrophy of the muscular coat of the arterioles existed in chronic renal disease, and believe it, therefore, to be the most important factor in the production of the increased blood-pressure and hypertrophy of the heart; in saying which it must be observed that we hold Ewald's views, and had done so for some time, quite independently of having read them. Ewald thus propounds his opinion in Virchow's *Archiv*, Band lxxi, p. 453. 1. In point of events (and in causation), he believes in the existence of the kidney-affection; 2. The blood-deterioration ensues; 3. The altered blood, he says, offers an obstacle to the capillary circulation everywhere. And we may digress for a moment to dilate upon the nature of this obstacle more than Ewald does. The impure blood fails to favour the circulation as purer blood would and does; the nutritional changes, which doubtless are in health a blood-moving force, take place less easily and less perfectly when the capillaries are thickened and their walls hypertrophied from chronic inflammation, first in the kidney itself, later on, however, through the capillaries in various parts of the body in which the current would run sluggishly—because the blood is impure—if it were not driven onwards by greater pressure *à tergo* by the heart; and still, as life is prolonged, the capillaries far and wide suffer in their nutrition, and thicken interstitially as the blood becomes more impure (just as they do in any passive hyperæmia), until, as Ewald continues, the fourth stage is reached,

when the general arterial tension is increased and maintained by a compensative hypertrophy of the left heart; next, and sixthly, he says the right heart becomes hypertrophied; and, seventhly, the arterioles generally; all in the same endeavour striving to push the blood forwards through the capillaries.

Without feeling so sure as he is upon the later events, which are of minor importance so long as the great primary ones are placed rightly, we may pause to dwell a moment here to insist upon what Bartels and Cohnheim have ably advocated, the truly compensative, not degenerative, nature of the hypertrophy of the heart and muscular wall of the artery. The best evidence of this is to be derived from observation: first, of the pulse when its tension is at its best marked, when, as Galabin describes it by a sphygmographic tracing, the pulse-wave is bold, and high-peaked, and square-topped, like a heavy Atlantic swell after a storm, an event which takes place before the walls of the artery are much hypertrophied. This secondary hypertrophy of the arterial walls has corresponded, in our experience, with the surely sooner or later commencing cardiac degeneration. This stage of increased blood-tension is that during which the heart is vigorous, and when, in the granular atrophy, the patient passes excess of urinary water, and urea enough for ordinary purposes, with albumen in small quantity occasionally, and always most after overexertion. In cases of chronic hæmorrhagic nephritis, we find the same hypertrophy, and a pulse of high tension, but not so high; here, too, the albumen varies in amount with the blood-pressure, and is less, just as the urine is more copious, when this is maintained high.

Ewald found that in every case of interstitial nephritis (kidney-cirrhosis), the arteries were hypertrophied as well as the heart; that, in the mixed forms of kidney-disease—interstitial and parenchymatous (or, as we should prefer to call them, more chronic forms of hæmorrhagic nephritis, mottled kidneys becoming granular, etc.), two-thirds of the cases presented hypertrophy of the heart and of the walls of the vessels, while one-third presented hypertrophy of the heart only; whereas, in the pure parenchymatous cases—*i. e.*, the most acute forms of nephritis (those with smallest discharge of urine and greatest dropsy, and shortest course), the heart was only found hypertrophied to the extent of about thirty-three per cent., and the arterioles were not affected at all.

Bamberger's theory is that an increased quantity of blood or heavier column of fluid in the systemic circulation, caused by a perpetually maintained excess of water in the blood, and itself due to the kidney-disease, is the source of increased blood-pressure. This increased blood-pressure must have one of three results—dropsy, dilatation of the heart, or hypertrophy. The argument stands thus: normally, as after drinking, the passage of excess of water into the blood raises the blood-tension in the systemic vessels; when, presto! open fly the flood-gates of the kidneys, and out flows the excess of water; but if its passage outwards be partially obstructed, the pressure must tell backwards everywhere upon the systemic capillaries, and will show itself earliest and most of all by anasarca in those parts where the capillaries are least supported by muscular fibres and the connective tissue is loosest; such local anasarca temporarily relieves the circulatory channels.

Now, if the individual be in a state of bad or impaired general nutrition, or of lax fibre, or the subject of other tissue-wasting disease, like phthisis, the heart and blood-channels yield to the increased pressure forced upon them, and dilate; whereas, if the subject be otherwise vigorous, and his digestion well maintained, and the increased strain not too suddenly forced upon his vascular system, hypertrophy of the muscular walls of the heart and of the arterioles follows. Next, a *circulus vitiosus* is reached by habit; the heart and blood-vessels have had their channels enlarged; they hold more than was their wont; neither can they ever go back again to normal dimensions. The water-balance in the system is conformed to a different standard, in which the blood always presents a larger than normal water-content. Thirst is habitual; there are an abnormal absorption, an abnormal maintenance, and an abnormal excretion of water. Increased pressure in the arterial, and increased quantity of fluid in the general circulatory channels, soon shows itself, however, in the veins also, by dilatation of these; and at length by dilatation and hypertrophy of the right side of the heart, as well as of the left.

As we have already said, simple dilatation of the heart only can ensue in the presence of bad general nutrition, or of local difficulties in the condition of the heart itself, such as degeneration of its coronary arteries, damaged valves, adherent pericardium. But, apart from these, and an amyloid state of the blood-vessels, which facilitates, as Bartels says, anasarca, and so obviates blood-pressure, the natural consequence of an increase in the blood-mass is, for the channels to dilate, and the heart to hypertrophy and dilate, exhibiting excentric or simple hypertrophy.

Now, in granular contracting kidney, as Virchow and Thoma have more recently shown, certain remarkable alterations in the circulation through the kidney itself take place in the course and progress of the disease; and I may quote myself, for the fact is one I stated years ago, in 1865. First, as the blood's passage through so many Malpighian tufts is impeded by their wasting and gradual extinction, the topmost cortical layers become less vascular, the vasa ascenduntia dwindle at their tops, and the direct branches to the arteriolæ rectæ enlarge, by collateral blood-flux; and further, in many instances, especially in those larger Malpighian bodies, those placed nearest to the vasa recta, the vas afferens, can be seen passing straight on into the vas efferens, without capillarising in the glomerule at all, and the circumtubular networks here become dilated and highly blood-filled. This is the stage in which the heart is highly hypertrophied, and is a well-nourished muscle, with no trace of degeneration attaching to it; when the kidney is red and highly vascular, the blood-passage through it is short, the blood-current swift; and when the secretion of urine is very little abnormal, except in quantity, which is notably increased, and in specific gravity, which is preternaturally low. This is the stage of thirst and polyuria.

But, later on, the blood-mass sinks again, the kidneys' power of excretion gains upon the body's faculty of absorbing, or the blood's power of maintaining, so much water; then, as the blood-pressure subsides, the dilating cause disappears, the dilatation of the heart's cavities diminishes, but the hypertrophy does not stand still, it only becomes more concentric.

Then if we follow the life-history of the case of granular kidney further, we find that the impurity of the blood increases as the discharge of urine diminishes, the capillary obstacles from impeded nutritional peripheral interchanges increase, the friction is greater, and the heart has a harder and harder burden cast upon it, while its own nutrition is inadequately provided for; it degenerates, becomes unequal to drive the blood on, and the end comes quickly by uræmic symptoms or lung-œdema, or apoplexy.

Bamberger's theory is, as he admits, a modified Traube's theory; and it must be allowed to be the only one, except Von Buhl's and Gull and Sutton's, which at all faces the facts, and endeavours to explain the hypertrophy of both sides of the heart.

This historical sketch and critical review must end; and we must pronounce a judgment upon the evidence before us. The witnesses have been heard; the way in which they gave their evidence has been commented on with a bold hand; I trust, however, never with an impertinent one.

Bright's disease remains for us, at present, a good term under which to group together and study several very important clinical phenomena intimately connected with, and dependent on, some variously marked forms of inflammatory degenerations of the kidney, all of which still possess something in common. The symptoms entailed are mainly the consequence of a local disease.

We are unable to accept Gull and Sutton's idea, that interstitial atrophic kidney is a form so separate by its histology, or by the order of its secondary events, or by the intrinsic nature of its complications, as to justify us in constituting it a wholly distinct disease.

Arterio-capillary fibrosis appears to us a change wholly apart from senile degeneration, and from atheroma; it is a most real change, separate from the muscular hypertrophy which usually accompanies it, and involving the entire thickness of the capillaries and the walls of the small arterioles. Essentially, it is a chronic irritative or inflammatory change; it is the cause, perhaps, wholly of one form of renal degeneration; but it occurs in more or less degree in all. It is a change far more widely spread throughout the body than was at first suspected; it has a most important bearing upon those so-called accidents, secondary serous inflammations, liver-cirrhosis, lung-œdema, pneumonia, meningitis, cerebral and spinal complications, which were thought formerly to be due to the irritament of an impure blood. Its recognition is a great step in advance, and one which will, in due time, be appreciated; but it is still probably a part only of the whole truth, which subtends the wide subject of chronic inflammation. But we still reserve our opinion that Bright's disease is a real entity, and that, while granular atrophy may be a consequence of this in its most gradual and complete degree, it is the renal disease which gives the characteristic features to the wide-spread general phenomena.

Bright's disease may be primary or secondary, acute or chronic; its consequences are best followed by recollecting what the functions of the kidneys are, and what their non-fulfilment must effect. These are, primarily, to maintain the blood at a proper specific gravity, by removing excess of water and excess of salts from it, as well as special nitrogenous excreta. The duty of the kidney is to regulate the quantity, as

well as the quality, of the blood—a matter sometimes lost sight of. If the blood-fluid be allowed, at any time, to swell like a flood, either dropsy, the transudation of its more watery part, must take place, or the blood-channels must dilate. If excess of salts accumulate in the blood, the specific gravity or weight of the blood-column must rise, and increase the friction through the capillaries; it comes, then, to strain first the channels in which it is confined, the chambers of the heart, and the blood-vessels, and to tax unduly the pump-power of the heart, and the muscular walls of the arteries. If nitrogenous excreta cannot escape by the kidneys, they accumulate in the blood, or must make a way out for themselves by vicarious channels; maintained in the blood, they interfere with the chemical nutritive changes, perhaps effect that general lowering of the body-temperature which is characteristic of the uræmic state; making their passage out by the intestines, skin, or serous membranes, they surely, sooner or later, provoke inflammatory changes. Just in so far as Bright's disease is held to comprehend all these phenomena of suddenly or gradually abrogated renal functions, we hold it to be a useful name.

But as to treatment, we fancy we hear some exclaim, Are you going to tell us nothing? In very truth, we can compress no more into our time. The treatment of disease is wisely pursued by those only who possess an accurate knowledge of it; and to an increase of this our entire efforts have been directed. It remains only for me to thank my audience for their attention.

ABSTRACT OF A CLINICAL LECTURE ON TWO CASES OF APEX-PNEUMONIA.

Delivered at the Middlesex Hospital.

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THERE have been recently, in the Hertford ward, two cases of acute pneumonia of the upper lobe of the lung, one terminating in recovery, the other in death. In this respect, the cases were typical, for in a large proportion of cases of apex-pneumonia in the adult, a fatal result ensues. Again, in one of the cases, delirium tremens was present as a complication; and in the other case, there was a decided history of alcoholic intemperance. In these respects, again, the cases were typical; for intemperance is the frequent precursor of apex-pneumonia.

CASE I.—An ex-soldier, aged 35, of very intemperate habits and bad character, marked on the left chest with a D, was admitted on December 30th, with the symptoms of an acute febrile disorder of undetermined kind. He had been feeling ill about a week; and five days before admission, had had several rigors, followed by cough, pain in the side, and vomiting of a greenish fluid. On admission, his temperature was 101.2°, pulse 120, respiration somewhat accelerated. He could give no further account of himself, save that he had, some years ago, had jungle fever and dysentery in India, and had lately been subject to chronic rheumatism. No definite physical signs could be discovered. The chest was symmetrical, with generally increased resonance; a few bronchial râles were apparent over the right base. The heart's apex was somewhat unduly covered, and its sounds muffled by intervening lung. There was no abdominal tenderness or enlargement. The tongue was coated with a white dryish fur; the breath offensive; and bowels confined. Towards the latter part of the day, the patient complained of chilliness, and the temperature rose to 105.2°; the pulse being quick and full, regular, and not compressible. The cough was now troublesome, and productive of a small quantity of sticky rust-coloured sputa. Cold compresses were applied to the chest, and a simple saline expectorant ordered. During the next day, the temperature, taken every four hours, ranged between 105° and 103°.

On January 1st (seventh day of illness), the sputa were more abundant and more stained; and in the upper scapular region in the right side, some fine crepitant râles were heard during inspiration, and some imperfect percussion dulness could be here elicited. Later in the day, the breath-sounds became tubular in character over this area, and similar inspiratory crepitus was audible below the clavicle, but there was here no dulness on percussion. The temperature through this day had ranged lower—between 103° and 101°—and the pulse 108.

On January 2nd, the temperature fell to 98.6°, rising towards mid-day to 101.4°; pulse 92.

On the 3rd, there was marked percussion-dulness, both in front and behind, over the upper third of the right lung, with tubular breath-