THE BRADSHAWE LECTURE

OR

MORBID ARTERIAL TENSION.

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It falls to the lot of the Bradshawe lecturer of to-day to deal with circumstances that are sadly peculiar, and which—young being the days of the foundation—no one has hitherto been called upon to handle. He is here to-day, not in his own right, so to speak, but to fulfil the duty, to reap the pleasures—in this instance but sad ones—which are some of the obligations of friendship. In happier fate, we should have listened to one of the most distinguished of the younger Fellows of this College, as he pointed, as assuredly he would have done, from the honoured and brilliant Past to the hopes of a robust and seething Present, and then, perhaps, on to the

"What Now discovers not, Hereafter knows,"

and the boundless capacity of the Future—fit subject of discourse by one whose enthusiasm seemed as limitless as it was prodigal.

But Mahomed's destiny was not this; his presence has gone on-wards, and we who remember its individuality and its vigour may well make it our business to inquire what of the essence—

"The Living Power, if that which lived
True Life, live on"

--of that lost individuality is here.

But, although thus fettered, as I think, in the choice of a subject upon which to lecture—for there are other matters upon which, as being more conversant with them, I would rather dwell—when I come to examine them, my bonds are those not of restraint, but of freedom. For, on the one hand, the memory of the most illustrious Past, although not dead, yet sleepeth, and the task of wakening may most gently and naturally come by Time's substitutions and the more vivid impressions of its latest finished effort, the more recent Dead. And, on the other, in this particular instance, my subject happily embraces the work, not of Mahomed only—not of him first, far from it, though changeful fortune gives him now a courteous, sad, respectful precedence—but of such distinguished Fellows of the College, past and present, as Bright, Sibson, George Johnson, Owen Rees, Sanderson, Gull, Sutton, Dickinson, Broadbent, Galabin, and others. Indeed, when once the enumeration is begun, I know not where one is to stop; so many, according to my idea of what arterial tension embraces, have worked at some branch or other of it.

The subject, therefore, is appropriate, whether for a Bradshawe lecture in ordinary, or for one delivered under the—let us hope—long peculiar circumstances which attach to that of 1885, or for a lecture emanating from this College; for it concerns itself with a matter which, by the work of many of its Fellows, has been made, and still is, one of the leading pathological and clinical questions of the day.

I could not, of course, attempt in one short hour to review the whole history of arterial tension—a history practically concurrent with that of the circulation of the blood. Nor is anything of that soporific kind my purpose in this the middle of August. I am only concerned with the latest developments of arterial tension, the application of the idea to what, for want of a better term, I shall call the household explanation of disease. By that, I mean an explanation which can be made to apply to such multiform conditions that, from one point of view, it is a morbid agency the importance of which can hardly be overrated: from another, it bears more the aspect of a faded Cambridge blue, so little of depth is there in the colour it contains.

These two alternatives will, I believe, fairly represent the value set upon the idea, or the fact, by practical men in the present day. I do not mean the conception of its value in the mind of the absorbed pathologist: he looks at things with an æsthetic severity of conviction that may chance to arrive at conclusions which are beside and

beyond our everyday realities. He works for to-morrow rather than to-day. His mood must often seem to be:

"Yet still shall I speed
On my way without heed,
Nor mourn for the wreck that is doing
For my calm, cold light
Is my own delight,
And I smile on the ashes of ruin."

The stress of the fight comes not on him; the upheavals produced by his diæta come upon the sons of men, and theirs is the stress in clambering the rugged crevasses of discovery.

Now, arterial tension and Bright's disease have danced so much together of recent years, that it is necessary to occupy much of the time with the aspect of the question derived from their association. Bright, in his day, noticed the hypertrophy of the left ventricle that occurs in chronic renal disease, and attributed it to the impurity of the blood, and to the extra work thus thrown on the heart, in consequence of the refusal of the tissues to allow a free passage to material of which they disapproved.

His words are these: "Either the altered quality of the blood affords irregular and unwonted stimulus to the organ immediately, or it so affects the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system." Dr. George Johnson, in his earlier writings, maintained much the same view, but abandoned it, in deference to the teaching of modern physiologists, for what is now well known as the stopcock-theory, which supposes, and no doubt justly—so, that the function of the muscular coat of the arterioles is a controlling one; and that, when blood is ill adapted to the tissues, control will be increased, and the blood shut off more or less from the tissues. This must necessarily create obstruction behind, throw increased work upon the heart, and thus account for the hypertrophy of the left ventricle. Fully examined, there is not much objection to that way of stating the case; but there is just a touch of fancy in the stopcock simile, which provokes the criticism that it smacks too much of a purposive volition. The arteries might seem to say to the heart, "You shan't," and the heart to retort, "You shall;" or, as Dr. Dickinson writes, "the heart and arteries are thus represented as acting in antagonism, and hypertrophied by the efforts each makes to get the better of the other; the arteries contracting beyond their wont in the endeavour to shut the blood out of the tissues, the heart using increased efforts in the attempt to overpower their resistance and drive it in; and both, like conflicting athletes, are increased in muscle by the exercise." Dr. Johnson takes exception to that way of stating the case, but it is a clear way of putting the issue raised by a too dogmatic insistence on the stopcock analogy. In that light, the theory does not seem to me unphysiological. There is no strife in Nature; and, if the hypertrophy of the vessels exists, as it surely does, then both it and the increase of the muscular tissue of the heart must be part of one common process—one purpose, if you will—which is consistently and adequately explained by the effort of the entire muscular element of the circulatory system to forward a fluid to which the absorptive or appropriative powers of the tissues are ill adapted. I must say I find myself in complete accord with Dr. Dickinson, and Bright was probably well advised in confining himself to the facts. The bare supposition that the heart is large because it has more work to do, is quite unassailable. Nevertheless, Dr. Johnson advanced us an important step by insisting upon the thickening of the vessels, because so little attention had been paid to it. Bright, however, had noticed it, and you will see at once that the knowledge of that time implied its existence. It must be so. An athlete uses certain muscles for a certain purpose, and he perhaps doubles their size. We do not question the cause or the nature of the hypertrophy; the heart does double duty, owing to some impediment in one or other of its valves. Who questions the reason of its increase in size? If the stomach or the intestine have to overcome obstructions, and we find them thickened, again, who disputes the nature of the thickening? It goes without saying. In all these instances we see simple illustrations of a common physiological principle admitted by all. What reason, then, is there to discuss whether the muscle of the arterial coat hypertrophies? Of course it does, when excessive work is demanded of it, just as organic or any other muscular fibre elsewhere. Therefore, to the question, Is the muscular coat of the arterioles thickened in chronic renal disease? I reply not so much by a demonstration of the fact, which is, perhaps, not so easy as might at first hand appear, but by an appeal to the entirely *a priori* ground that it must be so. And I do this with intention, in the conviction that the appeal to demonstration, and to demonstration only, in the present day, is too absolute. No doubt it is, on the whole, the safest ground to tread; but appearances admit often of various interpretations; while, within

such limits as its present use, I believe my argument to be unassailable.

Some particular feature will, I say, admit of various interpretations. Thus, Sir William Gull and Dr. Sutton write: "As to the hypertrophy of arterioles we may say that, though the muscle in some of the larger arterioles especially seemed to be increased, yet we are still sensible of the difficulty of giving a true interpretation to such an appearance." They lay stress upon the possibility that irregular and unusual contraction of the vessels, and a want of constancy in the natural thickness of the layer in different arteries of equal calibre, may simulate disease; and those of us who are familiar with the pitfalls which abound, even for the wary, in latter-day microscopy, may well be ready to admit that these are no mere captious reservations of the hostile critic. I, however, agree with Dr. George Johnson, Dr. Dickinson, Dr. Galabin, and others, that a comparison of comparable specimens is not so difficult as to throw doubt upon the reality of the hypertrophy.

As regards the existence or not of spasm, Dr. Johnson's position is to my mind inadequate, by just so much as he rejects the function of the muscular coat of the arterioles as an auxiliary force in forwarding the circulation. I am aware of the difficulty of this question; that physiologists as a body are in favour of his position; and that demonstrable evidence against it is either absent or indecisive. Nevertheless, I cannot conceive the evolution of a circulatory system upon lines which, whatever name be applied to them, must be antagonistic. Dr. Johnson makes use of the term "an antagonism of forces." I would rather believe that, developed for a common purpose, and by a gradual evolution, heart and arteries so work together under natural circumstances that we cannot detect their harmony; and that, when it is possible to study the action of the muscular coat, it approximates to the conditions of disease. We say, for instance, that non-striated muscular fibre acts slowly and persistently, and that such an action is inconsistent with the lightning-wave which would be necessary, if the muscular arterioles were a propulsive power. But muscle, organic or other, acts as it is taught to do; the pupil, the œsophagus, the intestine, the uterus, the bladder, none of them act alike; and whether the aid to the circulation be vermicular, or what not, it cannot be but that the aid is positive. Some years ago, Dr. Charlewood Turner gathered the evidence on this head into a very able paper, his conclusion being entirely in favour of a considerable propulsive force being exerted by the muscular arterioles.¹ Dr. Dickinson also takes this view; and, in withstanding the increased blood-pressure, it surely must count for something, if it be but the pigmy to the giant.

A pure control-theory seems to me to bristle with difficulties in the direction I have indicated; but take the one case of digitalis, which will appeal as much as any to our practical interests in the question. Digitalis is said to increase the strength of action of the muscular wall of the heart on the one hand, of the arteries on the other.

If the muscular coat of the arterioles control and retard the circulation, the undoubted diuretic action of the drug is not easy to explain; and if, as the control-theory supposes, the stopcock-action be considerable, it would always be a question whether it would do more good by its action upon the heart than harm by its action upon the vessels. At any rate, the increased action of the latter might very conceivably, in a failing heart, bring about sudden dissolution.

It cannot be said with any certainty that this is one of the risks of its administration. The drug is not without its risks, but they are primary and central to the heart itself; and it would seem that the action of digitalis teaches that the muscular coat of the arterioles makes for propulsion rather than for retardation of the circulation.

But there can be no muscle, and no physiological muscular action, without the risk of, probably, under such circumstances as these, the frequent exhibition of morbid muscular action or spasm. It is clear that a muscular action which leads to hypertrophy might easily pass into spasm under conditions similar to, but in excess of, those which have provoked the increased action. And, indeed, the assumption of some such spasm, if not necessary, is, at any rate, agreeable to many of the phenomena of Bright's disease. I may instance the headache and the renal asthma, both which are sometimes much relieved by nitrite of amyl and nitro-glycerine, drugs which unquestionably relax spasm, and promote dilatation of the peripheral vessels.

Thus far I have devoted myself to the opinions of Dr. Johnson—first, because they preceded others; and, secondly, because by the effective presentation and vigorous advocacy of their author they have done much to promote our advanced knowledge, and to widen the borders of their own field of interest. It requires, indeed, but little imagina-

tion to believe that, without this stage of its growth, the present conception of arterial tension would yet have to be developed. But be this as it may, the subject now expands in two noteworthy directions: by the hypothesis that chronic Bright's disease is an arterio-capillary fibrosis, or generalised tissue-change, on the one hand, and by the use of the sphygmograph on the other; and it is remarkable how these two lines of observation tend to support each other. As a direct recoil from Dr. Johnson's teaching, we may take the former first, although both proceeded independently and concurrently. I said a direct recoil from the teaching of Dr. Johnson, but it was not that; in point of time, and in the position it assumed in the discussion, it seemed to be; but it was rather the outcome of an approach of the question of the nature of chronic Bright's disease from the clinical side, or from a general rather than a local standpoint. Epigrammatically stated from the paper of Sir William Gull and Dr. Sutton, it is this: "Old age is not an entity, but a set of conditions predisposing to that state which is called chronic Bright's disease." And while to most this comes in natural order when the prime of life is run, yet to some old age is no matter of years and averages, but the running down of a spring set for an individual. It comes, at times, even to children. In the tints of summer, yes, even in those of spring, you can read old age if you will. And although chronic Bright's disease is in many cases associated with renal disease, it is not essentially a matter of organs; the generalised change in the tissues of the whole body is the essential, and to some this comes not by kidney chiefly, but by lung chiefly, by brain, by heart, and so on.

Now, here comes a difficulty in dealing with the case. May I say that this is a magnificent scheme of decay, to discredit which would be to do for this line of thought what I understand is meant by the expression "taking the romance out of life?" Still, life has the equivalent of romance attaching to it, which to many of us is most attractive, suggestive, propulsive. We often hear it said in the present day, this or that is mere sentiment, as if no solid worth attached to it; but I should like to know whether life would be worth living with all that is comprehended in the term "sentiment" cut out of it. What progress would be made in its absence? And I make bold to say before the Fellows of a College the reputation of which is based upon a firm foundation of fact laid by many generations of distinguished men, that pathology would lose much of its interest, much of the energy which characterises it and makes it so indispensably and helpfully the groundwork of medicine—physiology complete—if it chained us too subserviently to the march of facts. There are some, yes, many happily, who are absorbed upon the foundations, but they do not comprise the whole body of workers, and we cannot and would not do without the soul-inspiring idea, the fond imagining of the more precise knowledge of the future, forecast and sketched by the hand of genius.

This is a digression which will be pardoned, in that it helps me to convey what is, I think, the strength and the weakness of the argument of Sir William Gull and Dr. Sutton. The idea is a suggestive one, and it is largely true. Pathology is distinctly the richer for it; but it derives its strength less from the labours of the histologist than from the bird's-eye view, the intuition, may I call it so, of experience. There is ground in plenty for the argument that we wear out by tissues or systems; there is, I think, a fair amount of evidence admitting the opinion that chronic Bright's disease, with contracted kidney, is a generalised degeneration; but to base it upon the non-existence or unimportance of muscular hypertrophy in the arterioles, and to ascribe the thickening which exists to an essential hyalin-fibroid degeneration, is equivalent to the unskillful selection of a battle-field.

How far, then, do I think it true? Perhaps an answer to that question may best be arrived at by continuing upon the other, how far it is not true. And going to the other end of the circuit, there are groups of cases which admit no doubt, in which the disease in the kidney is certainly the cause of the changes in the heart and arteries. One of the first steps in the direction of proof of this kind was made by Dr. Galabin, when he showed that the heart and arteries are hypertrophied in a large proportion of cases of chronic parenchymatous nephritis (which, as a group, I may, perhaps, still call the large white kidney group, although the kidney is often contracted), and which are indubitably primarily renal. I have taken up the cases where he left off, and have collected from the *post mortem* records of Guy's Hospital all the cases of chronic parenchymatous nephritis that occurred in the ten years 1873 to 1882, amounting in all to 191. At the lowest estimate, taking the average of the healthy heart at twelve ounces, which is full high, 103 were above this and 88 below it. But the proportion claimed by hypertrophy is considerably higher than this; for, in the minority are fifteen children under ten, and seven more between ten years and fifteen, and others in which the left ventricle is stated to be thick or

¹ On the Functions of the Vessels in relation to the Circulation of the Blood. (St. Thomas's Hospital Reports, New Series, Vol. vi, 1873.)

markedly hypertrophied, although the actual weights would not indicate this. I think the preponderance may fairly be put at two to one. It has been said that hypertrophy is less frequent in these cases than in the granular kidney. But figures do not show this well. I have tabulated also the cases of granular kidney, recorded in our *Reports*, a total of 342 cases; and, taking the average again as twelve ounces, 103 are below that line and 226 above it. But it is not easy to come to a positive opinion in this way. I think Dr. Dickinson makes the observation that some of the cases of parenchymatous nephritis are primarily cases of granular kidney; and, in looking over a large number of cases like this, one cannot but be confirmed in the opinion, derived from clinical study, that it is quite impossible to separate too arbitrarily between the one form of disease and the other. The more acute form of disease is often far more insidious than is usually taught; the granular kidney frequently winds up with tubal or parenchymatous changes.

Perhaps I may remark, in passing, that a large series of observations of this kind may possibly throw some light upon the moot point, the rate at which the heart can put on muscle; for the heart has been found large in some cases in which there was every probability of the renal disease having existed but a few weeks.

Such differences as do exist between the two classes of cases are of more practical moment than of worth as distinctions. For, in the first place, it may be said that the failure of health and of nutrition will not unfrequently combine, so to reduce the arterial tension, as to deprive the circulatory system of any excuse for hypertrophy. The damage, too, in these cases is much more severe at any one time than the granular kidney can show; the left ventricle has, in consequence, more tendency to dilate, and thus to relieve the excessive tension and forestall the hypertrophy by the death of the patient. I put it thus pointedly as a fact of importance. In proportion as the renal disease is sudden and severe, so is the risk of dilatation of the heart. The more insidious it is, the more likely is the cardiac hypertrophy to be present in greatest perfection. Serious acute renal disease means sudden increase of arterial tension, and the imposition of greatly increased labour on the heart, and rapid dilatation of its wall, becomes a serious risk.

I speak with some diffidence before those of larger experience than my own; but surely this is corroborated by clinical observation. The most acute and sudden form of renal disease with which we are acquainted is undoubtedly that following scarlatina, and it is just here that acute dilatation of the heart occurs most typically. That coming next in severity is the chronic parenchymatous nephritis, in which dilatation is common; and, although it is more or less combined, it also closely competes with the hypertrophy. The granular kidney, which has small and inappreciable beginnings, and which drags its slow course through many years, while it cannot be said by any means to be ignorant of dilatation is, nevertheless, the disease in which the well known simple hypertrophy is most often found, and to a degree which is perhaps never reached under other circumstances.

There is another fact of similar bearing. It is this: that while granular kidney often terminates in cerebral hæmorrhage (86 times in 117 cases of apoplexy—*Guy's Hospital Post Mortem Records*, 1873 to 1882), the subject of chronic parenchymatous nephritis rarely dies by that means. Bartels states that he has never known death to occur in such cases from apopleptic effusion, and it is certainly a rare occurrence.²

At first sight, this might seem to be agreeable to such as maintain that the disease of the vessels is a peculiar one, and independent of the nephritis; for here is a severe form of renal disease, and the large heart in most cases, yet seldom apoplexy. But the better explanation is probably that I have given; it is obvious that dilatation of the heart is unfavourable to the occurrence of apoplexy: it relaxes the tension at the centre which would otherwise be put on at the periphery, and death comes about by cardiac failure, and not by peripheral hæmorrhage.

I have only time to add on this point that it applies, of course, not only to parenchymatous nephritis, but to all cases, more or less, in which there happens to be dilatation of the left ventricle. It is worth remembering, for it cannot be unimportant, whether the risks of an individual are those of apoplexy or of the more gradual process of cardiac failure; and the undoubted value of a drug such as digitalis in many of these cases, is a ready criterion of the importance of the point in the matter of treatment.

I have still something to add concerning the pathology of the arterial changes. Enough has been said about the reality of the mus-

cular hypertrophy; but what about the excess of hyalin-fibroid material, and the atrophy of the muscular coat in the arteries, that have been observed? I have no doubt of the correctness of the observation, and very little difficulty in explaining it. There is a general demand for extra labour, and a physiological response represented by hypertrophy; but, in the process, not only does the muscle thicken, but the other constituents of the arterial coats must thicken too, and thus the excess of hyalin-fibroid—of the connective material of the coats of the vessels—is natural to the circumstances. But, further, when once a growth is started which operates impartially upon several varieties of tissue, high and low, if the conditions for obtaining supplies be in any way prohibitory, as may be concluded is the case with the arterial coats, to judge from the frequency with which, even in otherwise sound bodies, atheromatous changes are met with, the least specialised, or less dainty feeders, are likely to have the best of it and to supplant the more highly specialised. There are plenty of instances in pathology of the ease with which embryonic or structureless tissue gets a living by the process which is popularly known as "sponging." There could not indeed be a more apt term, for these lowly organised tissues seem to live by a process of simple soaking. Take an *ante mortem* polypus in the heart, or even those larger masses of thrombus which sometimes form in the apex of the left ventricle. In either case, there are considerable tracts of clot which must have maintained themselves in this manner; and if you tell me that these things are dead, then so are we in great part (as has indeed been contended), for they are no more so than several of the parts of the living body; and I would have it that they thus live. But perhaps it may be said that the life of a clot is no fair evidence that definite structures will live after such a menial fashion. If so, there is a well worn case of the dissecting aneurysm, with its false channel as good as the natural one, as far as its lining membrane is concerned; or there is the still more ancient question of the nature, true or false, of the coats of an aneurysmal sac. The largest sac we can take, and that is no mean limit, will show as irreproachable a lining membrane, even to its reproduction of disease, as the vessel from which it springs; so that, brushing aside the cobwebs of the intellectual tarantula, it is impossible but that new coat has formed. Perhaps some may contend that it is formed by means of the legitimate blood-supply procured from the vasa vasorum. That may be allowed to be possible; but not the least of the advantages of holding such an office as this is, that one becomes entitled for one short hour to an opinion, and consequently I venture to adhere to the view (not by any means a novel one), that the internal coat of the vessel or sac, as the case may be, is derived directly from the blood-stream, by what may, perhaps, be described as a natural asphalted process, the blood being asphalt, roller, and clerk of the works all in one. I dare not presume too much, or I should be inclined to investigate the mode of life of foreign bodies in the peritoneum and in the joints; of our natural cartilages and intervertebral substance, and even of tendon, notwithstanding the noteworthy observations of Dr. Mitchell Bruce upon its system of canals. I must, however, content myself with the statement that physiological and morbid phenomena alike appear to show that a perfectly natural tissue may not only live, but be the best of its kind in this easy way, while more highly strung tissues, such as muscle, give way and disappear. Therefore, I see no contradiction in the fact that, in the tissues and vessels of chronic renal disease, the muscle may certainly be thick in some parts, wasted in others; or that the vessels can be recognised as well by the hyalin-fibroid thickening of the coats as by the muscle.

But stronger, and perhaps less fanciful ground in favour of the existence of degenerative changes in this disease, may be found in what no one now questions, namely, that strain or overwork leads to disease. Nay, more, bearing in mind the observations of Dr. Moxon, upon ulceration of the aorta by friction, and on the nature of atheroma, it may be said that wear and tear lead to inflammation; and if there may be a question about this in the aorta, though I do not think there can be, there can be no question whatever in the pulmonary artery, where, when we meet with atheroma, there will generally be plenty of evidence that there has been obstruction to the passage of the blood through the lungs. What is true for the larger vessels is still more so for the smaller, for inflammatory processes are certainly more luxuriant in them than in the traveling products which constitute atheroma. Hence, as the direct outcome of the overwork, we might expect more or less excessive nucleation of the coats of the arterioles, and ultimate fibrosis. This is a point of importance, very properly insisted upon by Dr. Dickinson; and, I may add, not only in regard to the changes attaching to chronic renal disease, but also in dealing with the question of the nature of disease in the smaller vessels.

² "Eight times in 101 cases, of which four admit of question, being not improbably cases of granular kidney with an acute parenchymatous change superadded. Cerebral hæmorrhage is still rarer in lardaceous disease, as Dickinson notes."

But not only do these cases of chronic parenchymatous nephritis testify to the potency of renal disease in causing hypertrophy of the heart and arteries; there are instances of granular kidney in children and adolescents which are to be traced directly to scarlatinal nephritis, and occasionally to poisoning by lead. Degenerative changes of any kind, although not unknown, are not common at these periods of existence, and it is therefore by so much the more certain that the renal disease initiates the circulatory changes. The same may be said of the occasional cases where the kidneys are spoiled by chronic obstruction to the passages; and finally, there are the experiments of Gravit and Israel upon the lower animals, in which it has been found that an inflammation of the kidney can be produced by the temporary ligature of the renal artery on the one side, which, if the animal live long enough, is followed in course of time by contraction of the kidney, and sometimes by hypertrophy of the heart also.³

The arguments that have been used against the renal origin of the cardiac and vascular changes, although based upon admitted facts, are by no means conclusive; for instance, it is said that the kidney may undergo extreme degeneration without being attended by any such ulterior effects, and this is about equally true as regards the heart, as has been shown for both forms of disease, the granular and the parenchymatous. But the argument which is held to explain the same fact in relation to lardaceous disease, will apply to these forms of renal affection also. It is only in cases where the excretory area is disproportionately small for the body-weight, that the effect must be sought. If the body waste and the heart fail, the kidney may be small indeed, yet sufficient for its day, just as the chronic phthisic could not possibly have lived on, had not the progressive wasting compensated the balance. It is also true that the general changes may be extreme, and the kidneys only a little granular or coarse; but this may mean much less than it appears to do, for very aberrant organs may look most meek to the naked eye.

These constitute the main points adverse to the contention of Sir William Gull and Dr. Sutton in its entirety. They show conclusively that renal disease will produce hypertrophy of the heart and of the smaller arteries; and this is indeed admitted by most pathologists, of whom I will specially mention Dr. Dickinson as having, as long ago as 1877, said all that one can possibly say now.

But there is another aspect of the question, and this it is which provides ground for a belief in the existence of diffused degenerative changes such as have been described.

I suppose that, ever since Bright's time, it has been felt by all that albuminuria must often be but a rough and a late test of disease—that there are conditions preceding it which, if we could recognise them, would tell us more; and one of the earliest of my medical experiences was the work of Dr. Owen Rees in the wards at Guy's Hospital, upon the detection by tincture of galls of morbid conditions of the urine which could not be reached by the nitric acid test. That line of research has been developed of recent years by tests of greater delicacy, and we now hear talk of physiological albuminuria, peptonuria, and so on. So far as my own experience has gone, I have not met with much encouragement in the field of pre-albuminuric precipitates, and all the less because it holds out hopes of great things as regards the early detection of disease. Peptonuria and physiological albuminuria do not seem to me at present to have shed much light on anything, not even on gout or indigestion, which are ever ready to coquette with novelty; yet ideas of this kind are beginning to colour our thoughts and our practice. Only the other day I saw a lady suffering much from headache, and with a variable but often copious albuminuria of seven or eight weeks' standing to our knowledge. I need not go into details of the case; it will suffice to say that, although it was admittedly obscure, I thought that some serious organic disease of the kidney must exist. It was arranged that another opinion should be obtained, and one for which assuredly I am not wanting in respect. This resulted in the verdict that the headache was megrim (which was quite possible), and that the albuminuria was a functional one dependent upon malnutrition.

But I must not pursue this question. I have no wish to prejudice its issue, only at present its promise hangs back a little, and we have hitherto learnt more from sphymographic observation, and it is here that Mahomed entered the field. The way being prepared by Burdon Sanderson, he improved upon Marey's instrument, and soon settled

down to an elaborate investigation of the pulse-tracings of disease, and he, more than any other, has familiarised us with the characteristics of the pulse of high tension. If others have treated the subject with a more philosophic delicacy of perception of the fallacies which strew the path of this method of investigation, as perhaps they have, it was a matter of earlier training and culture, not of spirit, and he was too enthusiastic to be self-contained.

I say this intentionally, because, looking back upon the history of the sphymograph—if it can be said as yet to have a history—I doubt whether the observations of Mahomed have received quite their due meed of estimation. Yet his was the work which kept the subject going, and of which our present knowledge is, in great part, the development.

If I were to choose from Mahomed's many papers that which has always seemed to me the most valuable, I should select one published in the *Transactions of the Royal Medical and Chirurgical Society for 1874*, on the *Etiology of Bright's Disease and the Pre-albuminuric Stage*. It bears directly upon the present part of our subject. In it was shown precisely that morbid conditions of the pulse (high tension) precede any evidence of disease in the kidney; and he draws the conclusion, from a number of observations, that the vascular condition is the cause of the albuminuria, and not the converse, as had been generally supposed.

His observations and conclusions seem to be particularly worthy of credence, because they no more than state, in terms of greater precision, what had been long known. There was nothing new in the knowledge that the pulse is hard under certain circumstances; let the early and long day of venesection attest that. There is no novelty in the assertion that careful purgation is the best means of preventing or combating the onset of scarlatinal dropsy; let the sheet-anchor of the treatment of Bright's disease attest that. The sphymograph but told us in one sense what we knew before, but it told its tale with a graphic interest which had much of the merit of a new discovery. It did not educate the finger, but it reduced the composite of the impressions derived from touch; and this account of high tension, demonstrated by the sphymograph, but not unknown before, leans obviously towards the hypothesis advanced and supported by Dr. Walshe⁴ as early as 1849, that Bright's disease is not renal, but primarily a blood-disease. It is fair to suppose that the blood is, under such conditions as those of acute gout, lead-poisoning, blood-poisoning of some other kinds, some states of anæmia, and so on; in a condition which renders it repulsive to the tissues, and that the kidney only suffers in common with the other members; and it is quite possible—nay, there are grounds for believing—that under circumstances which apply to and are imposed upon all, it may suffer more than the rest by reason of some disability inherent in its anatomical structure, for example; or an undue readiness of its glandular elements to undergo degenerative changes; or a disproportionate activity of function, for it is hardly to be supposed that there is no maid-of-all-work in a household so economically conducted as ours. At any rate, it is well known that diseased conditions of the kidney—of the epithelium chiefly, but not only—are common enough in the *post mortem* room in all sorts of cases, which could not by any means be primarily renal; and such changes are probably far more common than allied ones in the liver.

The greater frequency of the occurrence of lardaceous disease in the kidney might also be an instance in point. It must, however, be said that the apparent precedence of high tension does not necessarily carry the conclusion that the kidney follows the lead of the blood. It may still be that the excess of tension is compassed, if not by albuminuria, yet by some milder form of renal inadequacy, to adopt Sir Andrew Clark's happy, if somewhat gossamer term, as the initial fault. The pathologist has no right to be contemptuous of function. It cannot be but that it sometimes fails first; it cannot be but that it often fails before any appreciable structural change is manifest; so that in scarlatina, gout, and such like conditions, it may so be that the tension is responsive to the visceral failure.

But if this importance attaches to function, as it surely must, it may be contended, on the other hand, that it supports the probability of the production of an arterio-capillary fibrosis by means other than renal defect. Can it be, with other excretory organs of probably no less importance, namely, the skin, the liver, and the intestine, that the kidney monopolises the power of thus rendering the blood distasteful to the tissues? Surely some of the many disorders of a huge viscous like the liver must be efficient, even if gout be discredited for being a trimmer; and it is possible enough, it is probable enough

³ In looking over our *post mortem* record for the purposes of this lecture, I have noted one case of one-sided disease, the other kidney being only hypertrophied, in which the heart was enlarged. Dr. Galabin had previously noted two others in earlier years, and there are several in which one kidney having been long destroyed or substantially crippled, the other having undergone inflammatory changes in addition to the hypertrophy, characteristic disturbances have followed.

⁴ Bright's Disease not Essentially a Renal Disease, but Essentially and Primarily a Blood-Disease (*Lancet*, 1849, vol. ii, p. 29).

from the clinical side of the case, that certain morbid states of the nervous system, "worry," for example, when expressed in terms of nervous action, by their influence on the peripheral circulation, will bring about the same result. Indeed, Dr. Clifford Allbutt, in one of his many attractive and suggestive papers, has taken up this very question, and has narrated cases, if I mistake not, of granular kidney which have seemed to date their origin from prolonged anxiety; and Dr. Broadbent has alluded⁵ to the increase of tension that may be observed in states of nervous excitement in hysteria and other diseases of the brain and spinal cord.

A good deal has sometimes been made of the occurrence of a general arterial disease with hypertrophy of the heart, without renal disease, in young people, but it is very exceptional. More common, but still rare, is the same combination in older people. Of the former class, I cannot say that I have seen a single example, although for twelve years I have been on the look out in our *post mortem* room; of the latter, I can count fourteen in the ten years collated. But I must say that, holding, as I do, the probability of the existence of a general arterio-capillary fibrosis, I am for ever wondering at the small amount of tangible evidence that is to be obtained from the deadhouse. As a matter of practice, a thick sudden appearance of the arteries but rarely points wrongly to chronic renal disease. Some years ago, MM. Bouchard and Charcot published some observations⁶ upon the dependence of cerebral hæmorrhage upon the presence of miliary aneurysms on the cerebral arteries. Heschl, Meynert, Bastian, Douglas Powell, and Charlewood Turner, have since then met with a similar condition. MM. Bouchard and Charcot consider the disease to be a sclerosis of the outer coat of the vessels leading to atrophy of the muscular coat, and thence to weakening of the walls and aneurysm. One cannot but suppose that here is the same change as that described by Sir W. Gull and Dr. Sutton, to the accuracy of whose observations independent testimony is thus afforded. They also contend for the special nature of the disease, although they would confine the sphere of its action to the vessels of the brain; but this lands us in a dilemma, for, on the one hand, it would seem to traverse the observations, too numerous to admit of the least doubt, that renal disease is the common associate of apoplexy (63 cases are given, a large proportion of which must have been, and obviously were, accompanied by renal disease and hypertrophy of the heart); and, on the other, if it is, as it must be, the change with which we have been so much occupied, it cannot be simply local, since so many other observers unite in saying that it is a generalised one, even though they differ as to the nature of the precise change.

But scant time remains to allude to what is perhaps the strongest evidence in favour of the generalised nature of the changes which include chronic Bright's disease. It is referred to by Walshe; Sir William Gull and Dr. Sutton depend much upon it; and it forms the subject of some of Mahomed's latest work in a paper on Chronic Bright's Disease without Albuminuria (*Guy's Hospital Reports*, Series iii, vol. xxv). I may say that I reject such a nomenclature as this. Bright's disease, if it mean anything, means nephritis; and if it be true that it is but a part of a much larger subject, some more expansive title, arterio-capillary fibrosis, for instance, must be found to incorporate it.

It is certainly a remarkable fact in the disease, if, being an affection of one organ, it should frequently present symptoms apparently so remote that it is not difficult to overlook the primary disease altogether. There is no doubt that the urine is for long free from albumen in some of these cases (although I think myself that that feature has been rather strained); and perhaps the only tangible abnormality pointing to the kidney will be the low specific gravity of the fluid. Some die of apoplexy, some of asthma, some of cardiac failure, some of pneumonia. Of 100 cases given by Mahomed,⁷ 74 presented the symptoms of diseases other than renal.

I will also make use of another fact, which seems to me to point in the same direction; it is the great frequency of granular changes in the kidney in the *post mortem* room. There have been as many as seventy cases in one year out of a total of 490, or in a seventh of all the cases, and 463 cases in ten years; a number doubtless under the mark, as the slightest degrees are so common as sometimes to escape attention. This does, I think, tend to confirm the suggestion I have already put forward, that the life-history of the kidney is one of early senility, and that it runs in this respect with baldness, grey hair, and the

atheromatous aorta, any of which may be the symptoms of essential disease, but which are more often milestones; and milestones, happily, with their faces blurred.

I had intended to have taken up, more fully than I can do now, another and somewhat neglected aspect of my subject: the frequency and practical importance of the effects of pulmonary arterial tension. I am, of course, well aware of Dr. George Johnson's work in this direction also; and some of my hearers must have listened to or read his Luncheon lectures delivered here in 1877. But my own attention was called to it more particularly by a case that came under the care of Dr. Moxon, in Guy's Hospital, two or three years ago, and which for some time was under my own observation. It must suffice to say of it that the only symptom of much obtrusiveness was a peculiar *bruit* in the pulmonary area, which upon the whole we thought not unlikely to be due to an aortic aneurysm, with possibly some communication with the pulmonary artery. It eventually proved to be an enormous dilatation of the pulmonary artery, dependent upon a fibroid state of lung. The preparation is on the table before you. Since then, I have been surprised at the frequency with which dilatation of the pulmonary artery, not of course always in this excess, has come under my notice. Now, all cases of chronic bronchitis and of mitral disease are more or less cases of pulmonary tension, and the indications of its presence in the alteration of the character of the second sound are to be found in every text-book. To such cases I am not alluding, but to a pathological and clinical extension of the subject which on both sides requires development.

Those of us who have been in the way of making numerous inspections will, I think, assent when I say that every now and then a puzzling case of this kind is met with: the symptoms during life have been those of severe bronchitis and emphysema, and yet the bronchial tubes are practically dry, and the emphysema, if it cannot be said not to exist, is, at any rate, not more demonstrably present than the fatty heart which is so often made to satisfy the unenlightened conscience of a coroner's jury. But the pulmonary artery is dilated and perhaps atheromatous. It is difficult to say what the origin of these cases is; I only know that the lung has sometimes seemed practically sound, and I have been in the habit of explaining them by supposing that, if some of us are born with large hearts, others are born with little ones, and some in like manner are provided at the outset with lungs which are inadequate to their requirements. But, however this may be, something has happened which has left the lung comparatively sound, and made dilatation of the pulmonary artery the main disease.

It has often happened to me in the *post mortem* room to have to ask the question—in many cases, alas, without answering it—Which has been the more to blame, hypertrophy or dilatation of the heart and arteries? It is, indeed, impossible to mete out the relative share of each in a given result; but it is quite clear that the relative effects of the two cannot be unimportant. Extensive dilatation, for instance, of any of the cavities of the heart, while on the whole lethal, certainly has some compensatory aspects; while extreme hypertrophy, on the whole conservative and protective, as certainly hurries some lives on to a catastrophe. But, in this instance, observation tends to exalt the importance of dilatation; and I may remind you that there is *post mortem* evidence that, even in so common an ailment as anæmia, the pulmonary artery may undergo dilatation (Russell and Bramwell; *Bramwell on Diseases of the Heart*, page 197). I think, then, it is fair to conclude that the pulmonary artery sometimes dilates under certain transient conditions of pulmonary disturbance, and afterwards the dilatation remains permanently as the substantive disease.

I can only add that the clinical importance of these cases is neither small nor uninteresting. There is no need to intrude upon you the well worn discussion of the nature of the hæmic murmur, but you will see that the ready dilatation of the pulmonary artery, if it be a fact, revitalises this question, and gives it fresh prominence and purpose.

This, thus hastily sketched, is the subject, such are the scenes, which occupied one of the hours of Mahomed's short but busy day. I know that they possess far more importance for some minds than for others. Some of us never think in terms of arterial tension at the bedside. With some, it is descent; with some, the wear and tear of life; with some, nervous action; with some, blood; with some, our environment lit by Professor Tyndall's beam that forms the more suggestive pose of the mobile study, whose servants we in common are, but whose masters we reverently all might wish to be. Each of us has his ruling idea, which lights his path and guides his hand in ways innumerable helpful, but unknown to any save himself. Yet, though we must in this sense be negative to lines of thought not obviously in harmony with our own, we can always gauge their value justly by studying that line which best serves us.

⁵ On the Causes and Consequences of Undue Arterial Tension (BRITISH MEDICAL JOURNAL, vol. ii, 1883, p. 357).

⁶ Nouvelles Recherches sur la Pathogénie de l'Hémorrhagie Cérébrale (*Archives de Physiologie*, 1868).

⁷ The Clinical Aspects of Chronic Bright's Disease. (*Guy's Hospital Reports*, Series iii, vol. xxiv.)

It was said in my hearing the other day by one who, saying it, made it well worth the saying, that he did not feel altogether satisfied with his life's work. It was not enough for him to have been in the van of scientific medicine; he would have liked to have been able to say, "This is my discovery." In more general way, I have sometimes thought that medicine is less happy than painting or sculpture, which can have all a life's work visibly before it, and point with satisfaction to its numerous creations. There is something in the idea, but it wants in depth. One may be well content to have been in the van; it is a truer evidence of worth than the one discovery. Wherever I go I hear of Addison spoken of by his pupils, not as the discoverer of a disease, but as a master teacher, whose hold still bends them to an almost reverential admiration. I hear of Bright, not so much as a name attaching to a disease, as that of a man whose observations are worth attention, whatever be their subject. I hear of Latham and of Murchison in like manner; we shall hear of those we may not mention in their presence. Such as these influence their fellows by a power quite independent of their additions to our knowledge.

Mahomed's name will not live upon discovery; dying unfortunately so young, it would be but a flimsy conceit of the hour to suppose that it will live in any sense beyond the lives of his own friends and the precincts of his school. Progress is as relentless as death, and, although it is a glorious resurrection, it is none the less a grave. Even the gentle Emerson says: "What care we who sing this or that, it is we at last who sing?" All the more, then, may we dwell upon a name to-day which, could one depict on canvas or in the history of scientific medicine the individual elements in the wave of progress, during the last ten years, whether by the work itself, by the stimulus to thought which that work has proved, or by the *sympathy* of personal contact, would certainly be largely represented.

Only this more. Pardon the personal tribute:

Then notes came pouring through the wicker bars,
Climbed half a rapid arc of song, and stopped;
I turned and passed along;
But Time and Death, Eternity and Change,
Talked with me ever, and the climbing song
Rose in my hearing, beautiful and strange.

SCARLATINIFORM RASH PRODUCED BY INTESTINAL ABSORPTION OF PTOMAINES.—A patient observed by MM. Lépine and Mollière (*Journ. de Méd. et de Chir. Prat.*, 1884) presented at first nothing abnormal, except an artificial anus, the consequence of a strangulated hernia. One day he was suddenly seized with violent delirium, and M. Mollière noticed a considerable dilatation of the pupils. The skin was covered with a scarlatiniform rash, but there was neither fever nor angina. Poisoning by belladonna was at first suspected; but, after a careful examination, M. Lépine came to the conclusion that the symptoms were due to the absorption by the intestine of ptomaine, acting like atropine, and probably similar to that obtained by Zuelzer and Sonnenschein from putrid substances. After a short time, acute conjunctivitis and fever supervened, and the patient died. At the *post mortem* examination, a highly offensive substance was found in the intestine below the artificial anus, so that the possibility of an acute auto-intoxication cannot be disputed.

CHRONIC ALCOHOLISM.—Messrs. Dujardin-Beaumetz and Audigé have communicated to the Institute the result of their researches on chronic alcoholism. From June 1879 to July 1883, 18 pigs were experimented upon, each of them with a different sort of alcohol, such as ethylic and methylic alcohol, alcohol prepared from corn, beet-root, and potatoes (pure and impure), absinthe, and tincture of absinthium. These were given daily with the food, in the dose of one to one and a half grammes of alcohol, two grammes of absinthe, and two centigrammes of the tincture absinthii for one kilogramme of the weight of the body. The symptoms of intoxication by alcohol were sleepiness and prostration, vomiting of bile and glairy mucus, diarrhoea, and sometimes intestinal hæmorrhage, dyspnoea, tremor, and incomplete paralysis of the hind legs. Some animals which were killed or died during the experiment were examined by Professor Cornil. He found congestion of the digestive tube, sometimes causing hæmorrhage; congestion and inflammation of the liver, but without cirrhosis; congestion of the lungs; and, finally, atheroma of the large blood-vessels, especially the aorta. The animals were not emaciated, but presented numerous extravasations of blood into the subcutaneous and muscular tissues. Impure alcohol had a much more rapid and deleterious influence than rectified alcohol. The symptoms caused by absinthe and tincture absinthii were excitement, and spasmodic contractions of the muscles and cutaneous hyperæsthesia, but true epilepsy was never noticed.

BRITISH MEDICAL ASSOCIATION.

FIFTY-THIRD ANNUAL MEETING.

PROCEEDINGS OF SECTIONS.

THE TREATMENT OF ACUTE RHEUMATISM.

Introduction to a Discussion in the Section of Medicine at the Annual Meeting of the British Medical Association in Cardiff.

By J. S. BRISTOWE, M.D., LL.D., F.R.S.,

Senior Physician to, and Lecturer on Medicine at, St. Thomas's Hospital.

MR. PRESIDENT AND GENTLEMEN,—Acute rheumatism is one of the commonest of diseases; it is one of the most distressful of diseases to those who are its victims; by the damage it inflicts on some of the internal organs, it spoils or shortens the lives of no inconsiderable percentage of the population; and, at any rate until recently, it has appeared to be but little influenced in its course by any kind of remedial treatment. For these reasons, acute rheumatism has ever been a subject of sorrowful and profound interest to medical practitioners; and nothing of greater importance than its curative treatment can well be discussed at a meeting of the British Medical Association.

In opening the conference on this subject on the present occasion, I shall not attempt to treat it exhaustively. I shall not even, in the short time placed at my disposal, pretend to treat it adequately. I shall simply place before the meeting the by no means original views I at present hold in regard to it, which are based partly on writings and researches that are now common property, and partly (but in a far less degree) on my own experience.

Like many others who are here this afternoon, I can in memory traverse a long series of years, and can call to mind many varieties of treatment of acute rheumatism, which, during that period, have been introduced with the flourish of trumpets, have been adopted more or less widely, have been largely regarded as curative, and most of which I have, at one time or another, tested upon my own patients. But, until within the last few years, no medicine that I have employed has seemed to me to have the least influence in shortening the progress of the disease, or in diminishing its liability to complications; and, one by one, I dropped them all, and trusted to careful nursing, to local applications for the relief of pain or inflammation, to medicines administered internally for the purpose of acting on the emunctories or procuring rest or ease, to the treatment of complications as they arose, and to the administration of tonics as soon as the promise of convalescence suggested their employment. In a word, my treatment of acute rheumatism, after a while, accorded in the main with the expectant plan which Sir William Gull and Dr. Sutton brought into prominence some years ago; and I as fully was satisfied with the efficacy of this as I had been with that of other kinds of treatment with more pretentious claims. I accepted the view that acute rheumatism was a disease for which there was no known specific remedy; and that, at any rate so far as treatment is concerned, it had close affinities with the infectious fevers and lobar pneumonia.

I may refer briefly to two or three of the methods of treatment which previously largely prevailed. I recollect that, when I was young in the profession, nitrate of potash, largely diluted and in quantities of an ounce or more daily, was much affected by some of my teachers and senior colleagues, and I had ample opportunities of watching its action. I never saw, as I thought, any benefit whatever from it. A little later, bicarbonate of potash, so much lauded by the late Dr. Fuller, came into general use. This, as is well known, was generally administered in doses of 20 grains or so every two or three hours, with the object of neutralising the excess of acid that was supposed to pervade the system, and of rendering and keeping the urine alkaline. I treated many cases on this plan, and watched them closely; but I was neither satisfied by my own observations, nor convinced by the statistical evidence adduced by Dr. Fuller and others, that the treatment had any real efficacy. The plan of blistering the inflamed joints was introduced into this country by the late Dr. Herbert Davies. He believed that the rheumatic poison abounded in the neighbourhood of the affected parts, and that it was eliminated with the serous fluid discharged from the blistered surfaces. He believed, moreover, that,