AN ADDRESS ON SOME CLINICAL ASPECTS OF CHRONIC BRIGHT'S DISEASE.

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IN speaking of chronic Bright's disease I speak from a purely clinical standpoint, so as to avoid the confusion which so readily arises, and never more readily than in the case of chronic Bright's disease, when pathological terms are used to denote clinical states.

The distinctions which the pathologist draws between the different forms of chronic Bright's disease have, in my opinion, little clinical value. There is no practical utility from a therapeutic or prognostic point of view in distinguishing between a large white and a small red kidney even if we could do so with any certainty. The one point it seems to me in this connection which requires determination is—is the condition a chronic or an acute condition? For upon its not being the former and being the latter depend such chances as the patient has of recovery.

The orthodox statements as to the duration of life and prevalence of this or that special group of symptoms in the different forms of chronic renal disease have long ceased to have any weight with me in estimating the dangers and determining the management of any given case. The indications for treatment and prognosis depend entirely, I believe, upon the effects which the disease has had and is having upon the general health and vital functions rather than upon the particular pathological process in action. The kidney is a vital organ, and whether its destruction is accomplished by an interstitial or a parenchymatous inflammation makes no essential difference in the character, and very little in the march, of the clinical effects which follow upon it. The prognosis and the treatment are determined by the conditions existing at the moment—the extent to which the kidneys have proved themselves inadequate to their function—and not upon the peculiar features of the pathological process to which their inadequacy is due.

ETIOLOGY.

The circumstances which directly call Bright's disease into being have always afforded a theme fruitful of speculation and discussion. It has always seemed to me that the direct causative relation of acute to chronic Bright's disease is extremely doubtful. Cases which begin as acute nephritis always end as such, either in recovery or in death. I mean, I rarely see a case of chronic Bright's disease in which there is a history first of acute nephritis, then of an interval more or less prolonged in which the symptoms fall into abeyance, and then of a chronic persistent condition lasting for years, it may be, and ending in death. I have for a long time held and taught that acute nephritis has much the same definite march, though usually over a much longer period of time, as acute pneumonia, and ends in complete recovery or death in due course. The present teaching of pathology would lead us to expect, if in any case we ought to expect it, that the commonest form of acute nephritis, namely, that due to scarlet fever, would, if it ended in a chronic condition, end in a chronic interstitial change; and cases have now and again been recorded in which a small, red, contracted kidney has been traced, and not too remotely, to an attack of scarlatinal nephritis. Still I think the great majority of cases of chronic Bright's disease which come before us give no history whatever of anything that could be regarded as an acute attack. Sir Samuel Wilks used to say, "People have a great dread of acute diseases becoming chronic, whereas the great danger is that chronic diseases should become acute"; and I think it would be true to say that we much more frequently see acute nephritis associated with long-standing latent renal disease than we see chronic nephritis directly traced to an acute attack. How, then, does chronic renal disease arise? I have always felt that this is one of the most difficult questions in medicine. We see in chronic renal

disease the result of a steady, progressive, destructive, inflammatory process. Such a process occurring in almost any other organ, the lung for example, would at once suggest an infective process. Tuberculosis of the kidney we know, but we do not call it Bright's disease, although many details in the process of renal tuberculosis must be indistinguishable from the processes of Bright's disease. Yet an infection has not, so far as I know, been invoked in chronic Bright's disease. Lead as a cause of chronic nephritis we can understand, but does the inflammation of the kidney come to an end when that poison ceases to be supplied to the organ? Alcohol, I believe, has little to do with chronic nephritis, and the comparative rarity with which hepatic and renal cirrhosis are seen together is a matter of common knowledge. Certainly any form of albuminuria is quite uncommon in alcoholic cirrhosis unless cardiac failure is also present.

The causative relation of gout to chronic renal disease, and especially to the so-called gouty kidney has been very generally accepted; yet I venture to doubt whether the relation is so frequently causative as is commonly believed. Of this I am certain, that we see many cases of the most pronounced and prolonged articular gout, extending over years, in which albuminuria is never present, and in which there are no manifestations of any kind which would suggest a destructive kidney disorder. I have in my mind at this moment one case in which acute gout proved fatal, after having existed for many years, from cardiac failure, and in which the urine was always free from albumen and of good specific gravity, and two other cases of the most colossal uratic deposits in the joints and elsewhere in which there was no sign of kidney disorder. I am indeed almost bold enough to say that I believe that the relation of gout to renal disease is frequently quite the other way about, and that when renal disease and gout are associated the renal disease is the cause of the gout, and not the gout the cause of the renal disease. Dr. Clifford Allbutt once told me that in his experience men who suffered from acute, overt articular gout rarely had the visceral complications which are ascribed to it. My experience is that gout leads much more commonly to glycosuria than to albuminuria. Syphilis, which is so readily accepted on all hands as a cause of chronic inflammatory processes in other organs, rarely affects the kidney directly.

Pathologists are becoming, I believe, less and less inclined to accept a purely mechanical or chemical irritant as a cause of progressive inflammatory processes, and the idea of a bacterial origin of such diseases as have hitherto been regarded as not due to microbic infection is frequently suggested. By the blood stream and by the urinary tract the kidney is as accessible to infection as any other organ similarly placed, and we should probably be wrong in altogether denying the possibility of a bacterial origin for some of the chronic inflammatory destructions of the kidney which we place in the category of chronic Bright's disease.

DIAGNOSIS.

The recognition of acute renal disease is usually a matter of no great difficulty, and perhaps the diagnosis of chronic renal disease is not so exceedingly intricate as some who, if I may say so, are anxious to discover it, are inclined to think. To put the question of diagnosis in legal phraseology—On what evidence ought a patient to be convicted of chronic Bright's disease? What reasonable doubts ought to secure his acquittal? Unfortunately in the great majority of cases the verdict we should give is only too obvious. Still there is a tendency, it seems to me, in some quarters to convict on insufficient evidence. There is only one point in this regard to which I shall call attention, and that is the diagnosis of chronic Bright's disease when albuminuria is not present. I may state my own view of this question quite frankly, and say that I am strongly of opinion that a diagnosis of chronic Bright's disease in the absence of albuminuria cannot safely be made. I have never seen a patient in any way ill from chronic Bright's disease, which could be recognised as such, unless there has been albuminuria. I have never seen a patient with albuminuric retinitis, the only distinctive physical sign outside the urine on which reliance might be placed, without albuminuria. I have never seen the characteristic and severe toxæmic manifestations which are recog-

nised as due to renal disease without albuminuria. Yet this condition, namely chronic Bright's disease without albuminuria, has been frequently spoken of, and experiences differ. That renal changes, Bright's disease in a pathological sense, may exist, and the urine be free from albumen, I do not deny. All I am saying is that it cannot, with such certainty as we have a right to demand in so serious a matter, be recognised during life, unless such a characteristic lesion as albuminuric retinitis be present, and that, as I have said, I have never seen without albuminuria. After careful consideration and observation I am constrained to believe that the statement that chronic Bright's disease exists clinically without albuminuria has originated in the *post-mortem* room. Dr. Mahomed, I believe, went so far as to say that the great majority of cases of granular kidney had not albuminuria (Dr. Mahomed, when he wrote on Bright's disease, was largely engaged in *post-mortem* work). My point is that we cannot recognise chronic Bright's disease during life unless there is albuminuria. When chronic Bright's disease gives rise to symptoms it gives rise to albuminuria, otherwise we cannot recognise it clinically. Let us ask ourselves whether we could, from the symptoms apart from albuminuria, show that the kidney was the organ at fault. There is nothing, I take it, so characteristic about the headache, the dropsy, the shortness of breath, the vomiting, the cachexia, etc., that we could say at once that it is due to renal disease unless we found albumen in the urine. (There is one symptom of the late stages of renal disease which I think might alone lead us to a diagnosis of Bright's disease, and that is the horrible fœtor of the breath which comes on when the toxæmia is nearing a fatal termination, but this I have never observed apart from albuminuria).

The idea of chronic Bright's disease without albuminuria is based largely, if not entirely, upon *post-mortem* room experience. Those who have done much *post-mortem* room work know how common it is to find kidneys which do not conform in every particular to normal standards; the capsule strips with a little difficulty, the organ is a little deformed, a little under weight, a little tough, the cortex a little thin, and so on; and the pathologist says, "This is not a good kidney; there are early interstitial changes," and then the clinical record is referred to and the urine is found to have been free from albumen and any other indications of renal disease, and above all the patient has had no symptoms during life which pointed to renal disease. A surgeon performs some serious abdominal operation; the patient dies, apparently as the result of the operation, and the only explanation of death which the pathologist can detect is such changes in the kidney as I have mentioned, and he is tempted to say, "It is a mistake to operate on people with granular kidney." But the surgeon, after using every care to detect renal disease, and being fully alive to the dangers of operating on patients whose kidneys are not sound, could find nothing of the kind, or he would not have operated.

I am far from saying that the kidney condition is not answerable for the patient's death, for large as the reserve of kidney tissue is, it is not always so large as to provide for the safe conduct of a patient through the stress of a serious operation. What I wish to insist upon is that such a condition is not Bright's disease in the strict and clinical sense of the term; it is not a malady from which the patient is suffering, and it cannot by any means at our disposal be recognised during life. There could, I think it may safely be said, be no proof that a case of Bright's disease without albuminuria was really a case of Bright's disease except by *post-mortem* examination. To make a diagnosis of Bright's disease, then, I maintain that albuminuria must be present, and persistently present more or less.

Whilst the presence or absence of albumen is the all-important point, I am quite sure that the quantitative estimation of albumen is of no importance whatever. The estimation of urea in cases suspected of Bright's disease, and where albuminuria is not present, is not, I am inclined to think after much experience of various methods, of any great value. We constantly practise it in the wards, and our results are frequently of a contradictory nature. We not uncommonly find in cases where there can be no doubt about the presence of renal disease the urea record quite normal or nearly so, while it may be found to be reduced by 40 or 50 per cent. in many conditions in no way associated with renal disease.

The fact is that our clinical methods of estimating urea are at present of a very coarse and faulty character.

Urine of persistently low specific gravity without albuminuria are said, I know, to indicate chronic Bright's disease, yet I venture to think that this is not a reliable indication; but I will not pursue this point further than to remind you that urines of low specific gravity are seen frequently, and especially in women, where no sign or symptom of Bright's disease exists. I come then to this position, that the question of Bright's disease is not raised during life unless albuminuria is present; when albuminuria is present it is always raised, and has to be settled by careful consideration of all the attendant circumstances, such as its persistency, the quantity of the urine and its specific gravity, and above all the presence or absence of symptoms due to renal insufficiency. I only refer to the question of tube casts in non-albuminous urines to enable me to say that no urine need be searched for them which does not contain albumen.

CARDIO-VASCULAR CHANGES.

The cardio-vascular changes are not so uniformly inevitable or so common as the familiar manner in which they are spoken of would indicate. Speaking from an extensive experience in the *post-mortem* room, I am sure there is great uncertainty in the association of renal with circulatory disease. Extreme granular kidneys are not infrequently seen without atheroma or fibrosis of the arteries, and clinically many cases run on to a fatal termination without any cardiac symptoms or vascular accidents. High arterial tension is not nearly so much talked about as it used to be, and we are generally beginning to recognise the fact that the estimation of the human blood pressure either by the unaided finger or by means of instruments is no easy matter. In my experience anything which by the finger or the sphygmograph could be recognised as an increase of arterial tension above the normal is exceptional. Most cases of chronic renal disease which come under treatment have frequent and soft, if not intermittent or irregular, pulses, all indications of a falling blood pressure, even in the presence of cardiac hypertrophy; and the truth and importance of Sir William Broadbent's observation that a falling, and not an increasing, blood pressure is the danger of chronic renal disease cannot be too frequently enforced both for purposes of treatment and prognosis. Arterio-sclerosis, altogether independent of renal disease, is a far more common cause of cardiac hypertrophy than Bright's disease; and vascular disease of this nature is probably a cause as well as a result of renal disease.

CEREBRAL HÆMORRHAGE.

Chronic renal disease has not nearly such a common association with cerebral hæmorrhage as has been supposed. Pure, primary arterio-sclerosis explains the majority of the cases which I see. The fact is that in such a serious disorder as chronic renal disease there is no need to invoke the presence of any secondary changes in the circulation or elsewhere to explain its disasters. Toxæmia, the inevitable result of progressive destructive disease of a vital excretory organ, is the direct and only cause of death in the vast majority of cases.

DIET IN CHRONIC BRIGHT'S DISEASE.

I propose now to make a very few general observations on the management of patients suffering from chronic Bright's disease, and I will take the question of diet first. In laying down a dietary for a patient suffering from chronic renal disease I think we are too apt to forget that he is entitled, like any other sufferer, to such food as will keep him as far as possible in comfortable use and enjoyment of so much of life as may be left to him. Chronic Bright's disease is a very chronic disease in many cases, and is not, when properly managed, incompatible with much usefulness and not a little enjoyment of life; and we ought to exercise every care that such usefulness and enjoyment are not unnecessarily outtailed by anything we may do. All mortal disorders of the body, even phthisis and cancer, have their natural level of health and strength, and our care should be that our patients should not fall below that level as the result of any treatment which we may advise. We do not keep all patients suffering from chronic Bright's disease in bed to the end of their days, neither, in my opinion, should they be kept on continuously restricted diets.

I have no doubt that our practice in this matter in renal disease has a more scientific basis than in many other instances. To limit the input of proteid material so as to bring the work of the diseased kidney within its capacity is to act on scientific lines, but we know so little of the more remote stages of nitrogenous metabolism that beyond this very general statement of our object we cannot safely go. Yet there is no disease in which the tyranny of diet has been so recklessly practised. To live for weeks and weeks on a purely milk diet is not, I am sure, in many cases, necessary, and in some cases it is distinctly harmful, and there is no clear evidence that it meets the scientific indication. Some personal experiments made by Dr. Engelbert Taylor in the Pepper Laboratory of Clinical Medicine¹ showed that the total output of nitrogen was greater on a purely milk diet than on an ordinary mixed diet. I only quote this to show that it can be demonstrated that so far as the urea output is concerned there is no advantage in a purely milk diet. Dr. Hale White has shown the same thing clinically in chronic Bright's disease. My own practice in the matter of diet is simplicity itself, and I believe in the great majority of cases it is advantageous for the patient. My rule is that if the bowels are acting freely the patient may live on such ordinary mixed diet, including meat, as he has an appetite for and can digest. Patients who are confined to bed and suffering from uræmic vomiting or diarrhoea, and are therefore getting towards the end of the disease, cannot of course have any appetite, and the difficulty is to contrive food of any kind for them.

In 1897 there was in the Leeds Infirmary for the whole year a man suffering from large white kidney (that was the clinical diagnosis). Dropsy was his chief and almost only distress. His abdomen was tapped for ascites no fewer than eleven times. At the end of twelve months his dropsy had entirely disappeared, and he was practically well so far as the symptoms were concerned. He returned to his work and continued at it for six months, then came back to the infirmary with dropsy as before, and was tapped again several times. During the whole time he was in the infirmary he lived on ordinary diet, and I may say he also lived upon the "white mixture," for that was his only medicine.

The distinctions which we draw between the different meats in dieting cases of renal disease and gout are to my mind ridiculous. A patient may eat chicken and fish, and not mutton; mutton and not beef, and so on. I am sure there is no reason for this. Pork which is white, and in the case of Alexis St. Martin, was shown to be the most digestible of meats, is usually entirely forbidden. Van Noorden² mentions the case of a patient with chronic parenchymatous nephritis, who took half-a-pound of poultry daily for five days, excreted the same amount of nitrogen, and a trifle more albumen than he did in the next five days, in which, instead of poultry he took an equivalent amount of nitrogen in beef. Water and other fluid sustenance should be freely taken. Alcohol is perhaps as well avoided if cardiac failure does not require it. Climatic treatment, when at the disposal of the patient, is an important aid, but I do not propose to discuss it in detail.

DRUGS IN CHRONIC BRIGHT'S DISEASE.

Of drugs, Cushny, in his *Pharmacology and Therapeutics*, says, "The period of constructive pharmacology has scarcely dawned; at present its chief function is destructive and critical, and it is due to the latter fact that the patient with Bright's disease in the next few years will have a much more endurable life."³ Still, of the many drugs which are in use in the treatment of Bright's disease, some are of great value. We have not, and we cannot have, any drug or other thing which can replace the kidney destroyed. We might have, and may have some day, some drug or something which will arrest the process of Bright's disease as distinguished from its results. Our therapeutic indications are at present entirely symptomatic. I have no great belief in what is known as the general treatment of Bright's disease by drugs, beyond the systematic use of purgatives to keep the bowels acting freely. Beyond that no drugs are necessary except for the treatment of special symptoms as they arise. I have no great faith in the so-called vicious actions of organs. The proper way out of the body for urea is the kidney, and neither the intestine nor the skin can be made to take its place efficiently. Diuretics such as digitalis are frequently of the greatest use,

and almost as frequently fail. Diaphoretic drugs—and of these pilocarpin is the only really active one—are sometimes unpleasant, not to say dangerous in their effects; and the hot bath and hot-air bath do all that can be done, and are strictly under our control. Vaso-dilators have very little, if any, diuretic action in chronic Bright's disease, but in dyspnoea, headache, and vomiting frequently give great relief. The nervous and respiratory disturbances of the more severe degree of renal toxæmia, which usually portend a not far distant death, exercise our therapeutic resources to the utmost. Bleeding, purging, and sweating are our usual methods of dealing with convulsions and coma, and I do not propose to refer to their use in detail.

The old practical rule—that in renal disease morphine or opium should not be given—is still one to be observed without hesitation in most cases; but there not infrequently arise respiratory and mental disturbances of the most distressing and painful kind, which morphine alone relieves, and in which it can be given with perfect safety. It is a very remarkable fact, and it has been frequently observed, that the manifestations of the uræmic state are singularly mutually exclusive. For instance, great dropsy may exist without any single toxæmic manifestation. (I think dropsy may in some sense be regarded as a toxæmic manifestation.) Albuminuric retinitis, causing absolute blindness, I have seen to be the only manifestation of ill-health apart from the urine. One patient becomes drowsy and comatose and dies; another becomes wildly maniacal—in both cases without a trace of dropsy. Dyspnoea, uræmic asthma of the toxæmic type—that is, unassociated with disease in the heart, lungs, or pleura—exists quite alone, sometimes for weeks together, a constant source of sleeplessness of the most distressing kind. It is in this wakeful, painful, non-soporific form of uræmia that morphine is of the greatest advantage, and may, when given with care, be used with perfect safety. I constantly use it in the treatment of this distressing dyspnoea and wakefulness, and no other drug relieves so completely and safely. The contraindication is that which common sense would suggest—namely, the presence of or tendency to coma. By stupor and coma produced by his own poisons the patient's sufferings ultimately come to an end, and there is no need for us then to administer a soporific in the shape of morphine.

We are much indebted to Dr. Stephen Mackenzie for pointing out the indications for breaking the old-established clinical rule—when they are clear we need not hesitate, when they are not we should never venture.

REFERENCES.

- ¹ *Amer. Jour. of Med. Sci.*, August, 1899, p. 141. ² *Munch. med. Woch.* No. 7, 1899, p. 214; and *Boston Medical and Surgical Journal*, November 16th, 1899, p. 498. ³ *Boston Medical and Surgical Journal*, November 16th, 1899, p. 498.

ON GENERALISED INFECTION IN GONORRHOEA.*

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THE thesis which I have to develop, and shall hope to justify step by step, is as follows. The gonococcus in its process of growth in the human body produces an irritating toxin. This toxin is the direct cause of all the symptoms of the disease. In all cases it is absorbed into the system, where its presence causes systemic degenerations of varying degrees of severity. Gonorrhoea is thus a general toxæmic affection; but the microbes which form the toxin are generally localised on or around a mucous tract. The microbic invasion may extend to the organs communicating with the infected tract, or it may penetrate into the tissues, either by direct extension, as in the invasion of the peritoneum through the uterus and Fallopian tubes, or by a process of growth through the mucous membrane affected. Thence the infection may invade the cellular tissues, the lymphatics and glands, and the vascular system. This invasion is rendered possible by the action of the absorbed toxin upon the leucocytes, which is of a paralysing nature, and prevents the encapsulation of the microbes by these cells. Having reached the circulation the

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