REMARKS

ON THE

SYSTEMIC INDICATIONS OF CHRONIC
BRIGHT'S DISEASE.*By J. MILNER FOTHERGILL, M.D., M.R.C.P.L.,
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THE importance of the recognition of chronic renal disease will be admitted by everyone: it may, then, not be out of place to bring the matter before the notice of this influential gathering of medical men. The very able paper of Dr. Grainger Stewart, read at our last annual meeting, and the work of Dr. George Johnson recently published, have given to this subject a prominence which it distinctly merited. But, in a matter so complex, with sequels varying so much, according to the peculiarities of individuals, with manifestations so widely different, it is not easy to grasp the subject without applied and sustained attention to it; and, as many blows are required to fell an oak, so the vigorous reiteration of numerous individuals is necessary in order to place the subject of the consequences of chronic renal disease, commonly called "suppressed gout", before the medical public in an intelligible and comprehensible manner.

To-day, I propose to exclude from our consideration the effects of chronic renal disease upon the urine itself, designedly to avoid the questions of albuminuria and tube-casts. On these matters, we all know more or less, and have our own opinions. I would rather draw attention to the effects produced upon the system generally by renal inadequacy, by imperfect kidney-action—of a blood laden with the products of retrograde tissue-metamorphosis, or of peptones which have never been converted into tissue. What I aspire to do, is to furnish the material for the formation of a strong presumptive diagnosis of the presence of renal disease when it is not possible, or not convenient, to make a thorough examination of the renal secretion. It is obvious that there are many circumstances under which such examination is impracticable; and no other than a thorough examination is trustworthy. It also happens that albumen is not uncommonly absent even in marked renal disease, and especially in the commonest of all forms, that in which we are now most interested, namely, the granular kidney; while tube-casts, especially in this form, are only to be found after patient, elaborate, and painstaking examination of the urine; and such investigation is simply impossible in the majority of the cases attending our out-patients' rooms, and even in a large proportion of the cases which the general practitioner is called upon to treat in his every-day practice.

Are there, then, any general indications, any ready means, of forming a presumptive diagnosis at least, of these cases, which the medical man can have ever at hand, and can apply to every case with which he is brought into contact? I am fully persuaded that there are. But do not mistake me: the matter is but yet in its infancy, there are also sources of fallacy; and what is put before you to-day is no more than what I have been able so far to gather. A further series of observations may, and probably will, add new facts, will winnow and sift what has been already collected. If each gentleman who forms a part of my audience to-day will only keep these cautions clearly before his mental vision, it will be easier for me to say what I have to tell.

In the first place, we may review the changes inaugurated in the circulation by chronic renal disease, not so much as consequences as almost a part of the disease. We might as well leave the effects upon the throat out of the consideration of scarlatina, as the effects upon the circulation out of the consideration of Bright's disease. Without alluding to any controversies, I will confine myself to the enumeration of the changes so induced as given by George Johnson and Traube. When the blood is imperfectly depurated by the kidneys, and the aid given by the skin and other auxiliaries is not sufficient to supplement that imperfect action of the kidneys, the products of tissue-waste and of superfluous peptones accumulate in the blood. The effect of the excess of waste products in the blood is to induce spasm of the muscular coat of the tiny arterioles of the system generally. This produces an obstruction to the flow forward of the arterial blood, and a rise in the blood-pressure. This was well shown in a paper which recently appeared in

the BRITISH MEDICAL JOURNAL, by Mr. Mahomed of the London Fever Hospital. The heightened blood-pressure, or increased arterial tension, offers a great resistance to the flow forward of the blood in the contraction of the left ventricle; and, as a consequence, hypertrophy of the left ventricle follows. We must dismiss from our minds any idea of antagonism betwixt the heart and the arterioles; such an idea would but obscure the subject, and create artificial difficulties. The excess of waste products induces spasm of the arterioles; this obstructs the blood-flow, and obstruction to the blood-flow, no matter how produced, induces hypertrophy of the heart. The matter in itself is clear enough, if not darkened by unfortunate explanations, which rather tend to shroud than illustrate the subject. The sustained spasm of the muscular coat of the arterioles eventuates in hypertrophy; so that we have an hypertrophied condition of the two muscular ends of the arterial system—the central muscular mass the heart, and the peripheral muscular tubes the arterioles. Betwixt these two hypertrophied muscular ends, the connecting tubes, the elastic arteries, are highly distended.

The condition of heightened pressure in the elastic arteries leads to various sequelæ, not only pathologically interesting, but diagnostically useful. Thus, we get an accentuation of the second sound of the heart, or, to be more exact, of the sound produced by the closure of the aortic valves. These valves are closed by the recoil of the elastic arteries, and especially of the aorta. Now, elastic bodies recoil in direct proportion to the amount of distension to which they have been subjected. Consequently, the arteries, over-distended in this condition, recoil with proportionate force, and so close the aortic valves with much violence, producing a louder note. This accentuation of the aortic second sound is highly estimated in Germany as a physical sign of Bright's disease. It is really of much importance.

We are all aware that the filtration of fluid from the blood into the tubules of the kidney is very much affected by the blood-pressure; the greater the pressure of the blood the faster the flow, as seen in the rapid accumulation of urine after the free imbibition of fluids; the lower the blood-pressure, the slower the filtration through the thin-walled vessels of the glomeruli of the kidney, and the less the bulk of urine; as seen in the scanty urine of advanced heart-disease. In chronic Bright's disease there is a persistent condition of high arterial tension, and consequently there ought to be a large flow of urine. Clinically, we know that this is the actual state of matters. A large flow of pale-coloured urine of low specific gravity is one of the best marked evidences of chronic renal disease.

This is all simple enough, surely; but, nevertheless, the absence of a large flow of urine does not negative the presence of renal disease. In some patients, and especially in women, the effects upon the circulation vary from what has just been described, and, instead of hypertrophy of the left ventricle being induced by the obstruction offered to the blood-flow, dilatation is produced from the yielding of the heart-structure; consequently, there is not the increased arterial tension and the results thereof. Notably, there is not the increased bulk of urine which is found along with hypertrophy of the left ventricle. There is an absence of the hard incompressible pulse of high arterial tension, while palpitation is often present, indicating the inability of the ventricle to overcome the obstruction offered by the spasmodically contracted arterioles. Mistakes are commonly made in the diagnosis of Bright's disease in women, from these variations in the resultant vascular changes of that condition.

But what is more important still, the hypertrophy of the left ventricle, ordinarily found, in the course of time undergoes structural degeneration. The increased arterial tension leads to over-distension of the elastic arteries, and this over-distension (*Ueberspannung*) is the commonest cause of that change in the arterial coats known as atheroma. This change, called atheroma, is a parenchymatous inflammation of the arterial walls, which gradually alters their structure, and causes them to become less and less elastic. The aorta especially is affected; and, as the aortic recoil is the propelling power which drives the blood into the coronary arteries—the nutrient arteries of the heart itself—when that recoil is diminished by growing inelasticity, the blood-supply to the heart becomes insufficient, and the muscular structure undergoes fatty degeneration, or necrosis. This decay of the primitive muscular fibres cuts down the power of the heart, and with it the sustained high arterial tension. What then follows? The large flow of urine, characteristic of the earlier stages, gradually and silently passes into the small bulk of urine, indicative of heart-failure, as the condition of cardiac debility becomes superimposed upon the primitive condition of renal cirrhosis. This change in the bulk of urine is most lucidly pointed out by Sir William Jenner in some lectures published in 1865. I know no more valuable contribution to our knowledge of this matter than those lectures, which should be read, and carefully re-read, by all thinking practitioners. In time, then, as the changes in

* Read before the Medical Section at the Annual Meeting of the British Medical Association in Norwich, August 1874.

the heart-structures proceed, the large bulk of urine merges, by insensible gradations, into the scanty flow of heart-failure. This change is one that must never be forgotten, as misapprehensions may readily arise if this change be overlooked.

There are indications furnished by these changes in the arterial system which are diagnostically valuable. The overstretching of the elastic arteries, which occurs in chronic Bright's disease, is the cause of the atheromatous condition of the arteries generally found along with renal disease. This over-distension, as the cause of atheromatous changes, has been pointed out by Tzostakowski in Germany, and by Moxon, Clifford Allbutt, and myself in this country ("Strain, in its Relation to the Circulatory Organs." BRITISH MEDICAL JOURNAL, March 15th, 1873). Consequently, arterial degeneration is very common in chronic Bright's disease; and the elongation and tortuosity of the arteries produced by atheroma, or endarteritis deformans, are readily seen in the serpentine temporal arteries, and felt in the radial pulse. Hard, thickened, elongated and tortuous arteries, are very common in chronic Bright's disease, and ought always to put the practitioner on his guard as to his diagnosis.

The spasm of the arterioles, which, as we have seen, is one of the first links of the pathological chain which we have here been describing, is pronounced in the vessels of the skin. Consequently, the skin is dry, inactive, and does not readily perspire. The spasm, and consequent hypertrophy of the cutaneous arterioles, render the action of diaphoretics in ordinary doses quite ineffective. Such a condition of the skin is very common amongst the subjects of chronic Bright's disease, and especially in men, and in women where the effects upon the circulation are pronounced. At other times, chiefly in those women in whom hypertrophy of the left ventricle is not so distinct, the skin presents the appearance of water being effused into it, underneath it rather, and into the meshes of the subcutaneous areolar tissue. It is very commonly apparent in fulness of the lower eyelids, and in the pale oedematous hands, which look as if they would fester if scratched. This condition is found in the adynamic forms of renal disease, chiefly in women with dilated hearts; and still more, if they drink more alcohol than is desirable.

At this point, we may leave the changes in the circulation. A word, however, as to the frequency of apoplexy in the subjects of Bright's disease. The high arterial tension maintained by the hypertrophied heart and arterioles commonly eventuates in the rupture of an artery in the head; especially if there be already existing arterial degeneration.

We will now review the abnormal changes inaugurated or instituted by renal inadequacy through the blood upon different organs, especially the eliminant organs. Experiment has established the presence of urea and of the earlier products of histolysis in the blood after ligation of the ureters, or extirpation of the kidneys (Oppler, Schottin, Perls, and Zalesky); and Garrod has demonstrated the presence of uric acid in the blood of the gouty. The presence of these products in the blood in excess produces various results. Among the commonest are changes in the hair. If the hair be fine, it usually falls from the vertex, and the circle left in time often grows grey at the temples. If the hair be strong and coarse, it does not fall out, but turns grey early and completely. At other times, very white hairs will be found scattered amidst hair of natural hue. Scores of observations made in the great dead-house of Vienna enable me to speak with much confidence as to the coexistence of these changes in the hair along with renal disease.

The eyes are very commonly affected; but the changes revealed by the ophthalmoscope do not come within the scope of this paper. The sight is often impaired, or the patient may see double. There is often, too, an arcus senilis, not rarely partial, and found only under the upper eyelid. It is not so much the blue sharply defined arcus of green old age as the yellow arcus with indistinct margins and a cloudy cornea—cloudy from fat-granules being scattered through its substance. This last is very commonly found with early and marked senile degeneration. The ear is worth observing. In young gouty subjects, it is usually high-coloured, swollen, and shining; sometimes the lobe is red, and full to glistening, so tense is the skin. At other times, the development of connective tissue is more sparing, and the ear, especially the lobe, is wrinkled and withered-looking, and the helix is studded with chalk stones.

The *tout ensemble* of each case is marked and readily recognisable by the eye which has learned to note and register these changes. In one man, there are a high-coloured face, grey hair, and glistening earlobes, with a fair *embonpoint*; in another, there are a wrinkled ear, a similar condition of the skin of the face, an arcus, a bald crown, and general leanness: in both, there will be found a distinctly tortuous temporal artery. In a third, you will find pallor, isolated grey hairs, a watery-looking eyelid, a hand bearing similar watery characteristics, muscular

tremulousness, and a wavering unsteady eye, with a morbid self-consciousness, and a suspicious readiness to inveigh against stimulants. Just look about among your lady patients over middle age; you will soon find the person I have last described. These are typical forms; but there are others besides these three.

In all forms, however, there will be found one characteristic: a mental one; viz., irritability—a symptom more constantly present than any other. Ask these different persons, when patients, if they feel themselves at times very irritable; easily put out by slight exciting causes; often with a distinct consciousness that there is an element of unreason present, which adds to their irritability; and they will all answer in the affirmative. The plethoric individual will be distinctly and markedly irascible; the second will be found irritable and anxious, and the third fidgetty, capricious, and desponding. We will linger a moment over these mental symptoms, so constant, so suggestive, and yet somehow so little understood. The plethoric individual has a well sustained flow of arterial blood to his brain-cells, and he is quick, hasty, impetuous, and commonly explosive. His cerebral cells are well supplied with blood holding in solution renal salts, which act as irritants. We are all familiar with the gouty temper, as well as the gouty temperament. But at times this person will suffer from unaccountable depression, and an anxiety he cannot dismiss. The explanation of this we shall find to lie in spasm of the arterioles of the pia mater and the grey matter of the convolutions; and the cerebral anæmia tints his thoughts with unhappiness. In the second and third forms, depression, gloomy presentiments, and dark forebodings, are the prominent characteristics, and the irascibility is veiled by an overwhelming condition of sadness. In the third form, this mental wretchedness is commonly the causal origin of the taste for alcohol; it is the relief it affords to this groundless mental pain or misery which forms its chief allurements, its essential attracting force.

In all these forms, there will be found a certain amount of sleeplessness. It does not arise from pain; but the patients cannot catch sleep. The thoughts are active, but they run in a circle. After long thought, or rather train of thought, the original point of departure is reached once more. This is exhausting and irritating. It is not till far into morning that they can drop off. When they awake, the imperfectly rested brain is irritable, lacking in grasp, and deficient in power of concentration and endurance.

One curious symptom you will find almost always present; and that is, the patient gets up at night to make water. One gets up at one o'clock; another not till five. Whatever the explanation of this phenomenon—and I have none to offer—it is most constant.

When, then, you find a patient, especially at or over middle age, presenting the physiognomical indications given above, put the following questions. "Are you irritable, and easily perturbed in mind? Are you often sleepless—not from pain; but you cannot catch sleep? Do you get up at night to make water?" If these questions be answered in the affirmative, then there exists good ground for a presumptive diagnosis of chronic renal disease. If the patient be old and the circulation failing, put the last question thus: "You do not make so much water as you used to do. Do you get up at nights still to make water?" An intelligent look, half of inquiry and half of suspicion, will tell you of your hit, before the speech of the patient assures you of the correctness of your aim. Here the failure of the circulation has produced that change in the bulk of urine previously described.

Such are the ordinary systemic indications of chronic Bright's disease; and when they are found, a thorough examination of the urine is indicated. In hospitals, it is also desirable to corroborate the diagnosis still further by the sphygmograph. The square-headed tracing is characteristic, and instructive to students.

When, then, we find patients presenting more or fewer of the characteristics given above, and at the same time suffering from ailments which are the common sequelæ of an imperfectly depurated blood, our suspicions ought at once to be aroused. The ailments which follow in the train of chronic Bright's disease are commonly affections of serous membranes, of mucous membranes, and of the skin. The mucous membranes and the skin are eliminant excretory organs which aid and supplement the action of the kidneys, especially when defective. Over-action or apparent disease upon their part is a common outcome of blood-impurity from renal inadequacy; and this over-action is a compensatory change, rather than actual disease. In particular, the bronchial lining membrane is liable to undergo changes in the form of chronic bronchitis, especially when the cold of winter arrests the activity of the skin, and so deprives the system of the aid furnished by cutaneous action in supplementing the imperfect action of the kidneys. The cold, too, acts as an irritant to the air-tubes; and between the two we find the explanation of much of our winter-cough.

At other times, there is dyspepsia from the gastric juice being contaminated by the presence of waste products. Not unfrequently there is diarrhoea of an uræmic character. So common, indeed, are alterations in the intestinal canal in chronic Bright's disease, that Treitz found changes in the bowels in no fewer than one hundred and seventy-five out of two hundred and two cases examined. The bowel is affected quite as much as a compensating excretory organ, as a sequel, a secondary consequence, of the kidney-mischief.

The skin is very frequently the seat of changes under the circumstances of chronic Bright's disease; more so, however, in those cases where the skin is active in compensatory functional activity, than in those cases where it is dry and inactive. The chronic eruptions of advanced life are ordinarily the result of renal mischief; and this is especially true of eczema.

At other times, there are affections of the serous membranes. These lymph-sacs are often irritated, or even inflamed, by the waste-laden lymph flowing through them. At other times, there is what is called muscular rheumatism, from the presence of waste material in the muscles. Very commonly, we find the joints infiltrated with waste matter, especially in the form of uric acid. This is gout proper.

Such are the common outcomes of a condition of blood arising from imperfect depuration by kidneys no longer in their pristine integrity. When, then, such states are found along with the physiological indications given above, and the various questions above related are answered in the affirmative, a strong presumptive diagnosis is furnished as to the presence of renal disease. This species of examination is feasible in every case, and can form part of the stock knowledge of the busiest practitioner. Often and often will he find most valuable information so furnished; giving a direction to his treatment, and guiding his therapeutic measures, in cases where he may not have the opportunity of making a careful examination of the urine. Variations in the bulk of urine—at one time a large flow of limpid urine, at another a lessened flow of dark dense urine—give information almost as valuable as that furnished by the urinometer and the test-tube. Finally, to these general indications may be added headache, chiefly vertical. This last objective symptom is commonly present in the adynamic forms of renal disease.

There is a peculiarity about those diseases which are the consequences of chronic Bright's disease, and that is their tendency to recurrence. Not that necessarily the same form of ailment is always assumed; the different consequences are interchangeable. Thus gout is interchangeable with bronchitis, with a skin-eruption, or with a troublesome dyspepsia. The common origin of each is further evidenced by the readiness with which each form will yield to remedial measures directed to the causal renal condition, while stubbornly resisting the measures which ordinarily are found curative. One very illustrative case will point these remarks usefully. A friend of mine once put to me the case of a patient with an obstinate irritable ulcer, which had resisted for eighteen years the efforts of many leading men, both at home and abroad. The recognised association betwixt irritable ulcers and gouty individuals at once suggested a suspicion of "suppressed gout". The treatment was directed accordingly, and in three weeks the ulcer was well.

Such are the consequences of having therapeutic measures directed by a diagnosis which does not merely recognise the characters of a disease, and register its physical signs, but which penetrates into the conditions upon which that disease causally depends. The one is a mere intellectual feat, chiefly involving the mnemonic mechanism; the other involves a deeper insight, and becomes of actual utility to the patient.

Our knowledge of the chronicity of some forms of renal disease, and of the light which pathology now affords us of the slow insidious changes which precede marked and unmistakable kidney-disease, tells us that such renal mischief may exist with tolerable and even good general health for many years, the defective renal action being helped out, or supplemented, by compensating activity in other organs; that intervals of apparently perfect health may intervene betwixt these explosive cleansing processes; and yet that the condition of the kidneys upon which these affections causally depend is persistent, irremovable, and even unchangeable, except for the worse. It does not follow that these compensating actions or consequential ailments shall always be present; there may be, and commonly enough are, long periods or intervals of comparative health. It only follows that the individual is liable to have such maladies at any time; much in the same way that a soldier is not always being wounded even in a campaign, though he is liable at any time to be wounded—and that, too, sorely, even unto death; or a seaman is not always battling with storms, though he is liable to experience one at any time, and each storm may endanger his existence.

We must not, however, run away with the idea that the consequences or outcomes of this renal inadequacy based on organic disease, and

ordinarily termed "suppressed gout"—a happy term conveying at once the idea of latency and of the true pathology of the condition—are alike in different individuals. Far from it! Though, to a certain extent, its sequences are interchangeable, its different manifestations are directed by various conditions. In some families the changes are almost confined to the vascular system; apoplexy threatening during the period of hypertrophy of the left ventricle, and cardiac failure ultimately ensuing after degeneration has undermined the muscular structure of the heart. In others of a nervous diathesis, the consequences of suppressed gout fall chiefly upon the nervous system, especially in the form of those neuralgic of advanced life, so well described by Anstie. In women, there is often gouty dysmenorrhœa. In others, the manifestations are those of gout proper, especially in the phalanges of the extremities. In some, again, the tendency is to form masses of connective tissue in the valves of the heart, in the joints, or in the viscera. In one case, which has come under my notice, there is a dense patch of connective tissue in the palm of the left hand: the gentleman, a retired member of the profession, is an amateur geologist, and rests in his left palm the specimen he chips with his hammer. In agricultural labourers we usually find a dry inactive skin, rheumatic pains, fibrous thickening of the sheaths of tendons, and affections of the joints: chronic rheumatism, in fact, is the form assumed by chronic Bright's disease in them.

The diathesis, the occupation, the presence or absence of exposure to the weather, have much to do with the systemic indications of the primary renal disease. The peasant labourer never has gout in his great toe, or very rarely, and yet the affection is very common in portly butlers, who are heavy men, and who lean much upon their toes when waiting at table; though, in each, it may be found in the hands. The person of nervous diathesis does not suffer so severely from the changes in the vascular system to which some are most liable; while these latter, again, are not commonly subject to gouty neuralgia. I have at present under care a lady, who has suffered much from gouty neuralgia, complaining of gouty intermittency and palpitation, in whom the vascular system is almost free from structural change. Unlike, however, as are the different forms assumed, the origin is the same in each: and it is the recognition of that causal origin which is so important in giving the correct and rightful direction to the curative or palliative measures.

It is impossible to allude to the treatment of chronic Bright's disease here, or the measures appropriate to the management of its outcomes or sequels. These may well form the subject of another paper on some future occasion. To such as do not feel inclined to wait for this forthcoming occasion, I can refer them to the chapter on Combined Heart and Kidney Disease, in my work on the *Heart and its Diseases: and their Treatment*; where they will find the subject entertained at considerable length.

SCARLET FEVER DURING PREGNANCY.

MRS. B.'s daughter, aged 6 years, took scarlet fever on September 1st. In another week, two sisters and a brother were ill also; all eventually recovering. My anxiety from the commencement was about Mrs. B., who was eight months pregnant; but, in answer to my inquiries, she assured me that she had had scarlet fever and was not at all anxious. On September 22nd, she complained of sore-throat, and the rash was fully out on the 23rd. The same evening, vomiting came on, and continued incessantly until the 26th, accompanied by occasional darting pains in the right thigh; but, when this subsided, the rash had almost disappeared, and every dangerous symptom was gone, and it seemed as if the vomiting, which I had vainly attempted to check, had acted most beneficially. She steadily recovered and desquamated. On November 4th, she was delivered of a fine girl, whose skin was desquamating. On November 11th, erysipelas commenced at the infant's navel, spreading over the body from the arm-pits to the toes. This was treated with lead-lotion—the child being wrapped in linen soaked in the lotion, and then covered with gutta-percha. When the erysipelas subsided, an abscess formed on the dorsum of the right foot; then on the left; both of which were lanced, giving exit to a quantity of pus. Then, another abscess formed over the lumbar vertebrae with the same result; but, after this was lanced, vomiting of green and yellow bile (?) came on, and the child died on December 6th. Unfortunately, I did not examine the urine. This case is interesting as an example of a woman passing safely through a sharp attack of scarlet fever when so near her confinement. It shows also that the vomiting was evidently a successful effort of nature to eliminate the poison; and thirdly, it is remarkable that the child should have been affected *in utero* and desquamating when born.

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