CLINICAL LECTURE

ON A

CASE ILLUSTRATIVE OF THE CIRRHOTIC FORM OF BRIGHT'S DISEASE.

By T. McCALL ANDERSON, M.D.,

Professor of Clinical Medicine in the University of Glasgow, etc.

In the investigation and in the treatment of patients, it is of the utmost importance that we should carefully discriminate between the affection and the disease. The difference between the two will be at once apparent if I give you an illustration. Two patients are labouring under painful affections of the joints; but in one case the pains are due to the presence of an excess of uric acid in the blood, while in the other they are dependent upon a syphilitic taint. In each, the lesion of the joints is the affection; while in the one the disease is gout, and in the other syphilis.

The patient whose case is the subject of to-day's lecture was labouring under a variety of affections; but we shall see how beautifully they all fit into and harmonise with one another, and are dependent upon one and the same disease. He was forty-one years of age, a shoemaker, unmarried, and was admitted into Ward 2, Bed 2, on January 5th, 1876, complaining of palpitation with pain in the præcordial region of eighteen months; of loss of flesh, irritability of stomach, giddiness, pain in the head, and dimness of vision, of eleven weeks; and of cough, with expectoration, of two weeks' duration.

The family history could not be satisfactorily ascertained; but his father died at the age of seventy-seven, and his mother at sixty; while, of eight brothers and sisters, only two survived.

At the age of fourteen, he had scarlet fever; at seventeen, gastric fever; at nineteen, typhus; and at twenty, rheumatic fever. About fifteen years prior to admission, he had a gonorrhœa, which soon disappeared; and he seems never to have had any other form of venereal disease. He was a man of extremely irregular habits, sometimes drinking for a week at a time; but more recently he limited himself to a "break out" on Saturdays. He also smoked to great excess, sometimes using more than a quarter of a pound of tobacco in a week; but of late he had been more moderate in his dissipations.

About eighteen months before admission, he began to complain of palpitation, associated with uneasiness in the præcordial region, especially on exertion; and, some months afterwards, to these symptoms was added dyspnoea. About thirteen weeks before I first saw him, while undressing at night, he had a severe attack of vomiting; and a few minutes afterwards, having gone to bed, his breathing became stertorous, and he could not be roused. Along with the insensibility, which continued until six o'clock the following evening, the whole of the left side became completely paralysed. The paralysis was decidedly less by the following evening, and, on his admission, had in great measure disappeared. About the same time, he first noticed that he was passing a great deal of urine; and, several weeks afterwards, slight dimness of vision set in, which in a day or two suddenly increased, so much so that he was unable to recognise faces, although he could count the number of fingers held up before him. From that time, his vision did not further deteriorate. Along with the impairment of sight, irritability of the stomach came on, to such an extent that he could seldom retain food for any length of time, especially if he got up and walked after a meal. About an hour after food, he complained of heartburn and brought up sour mouthfuls; and this terminated in vomiting the contents of the stomach. Latterly, he suffered from pain in the head during the day, particularly in the occipital region; and from giddiness. To these symptoms must be added pallor, increasing emaciation, and debility.

In this case, there was hardly a single organ of the body whose functions were not more or less interfered with; and this, as we shall see, was the result of a wide-spread tendency to alteration or degeneration of tissue.

Let me first of all direct your attention to the eye-affection, which began three months before we saw him, with, as the report says, slight dimness of vision, which in a day or two suddenly increased, so much so that he was unable to recognise faces, although he could count the number of fingers held up before him. On his admission into the Eye Infirmary, he was barely able to read No. 20 (Jäger); the right eye being, however, a little better than the left. Dr. Thomas Reid,

under whose care he then was, made the following report. "Pupils dilate only partially, though regularly, with atropine. Ophthalmoscopic examination: Fundus of each eye occupied by the characteristic white deposit of albuminuric retinitis, interfering with the definition of the optic disc, which appeared to be somewhat atrophied. Retinal vessels greatly reduced in calibre. In the left eye, a considerable portion of the choroid below and outside the disc had an atrophied look; and scattered pigment-granules were seen in this situation." In a letter which Dr. Reid kindly sent me, he thus faithfully describes the characters of retinitis albuminurica.

1. Almost all cases of this disease are chronic, dimness of vision being generally the first symptom complained of.

2. The essential cause of the dimness of vision is fatty degeneration of the retina in the neighbourhood of the optic nerve-entrance and macula lutea: parts of the retina most used in direct vision.

3. The fatty degeneration is said to be preceded, and in some cases is certainly accompanied, by congestion and hæmorrhage from rupture of the arterioles of the retina. When the congestion and extravasation are considerable, there may be loss of vision from this cause in the early stages of the disease; but the vision improves as the congestion diminishes and the blood is absorbed. The sight is never perfectly restored in chronic cases, but the amount retained is determined by the extent and position of the fatty deposit. In no case is the vision entirely lost.

4. In the advanced stages of the disease, and probably also during the whole course of the more chronic forms, congestion and hæmorrhage are never present. The fatty deposit occurs primarily in the retina, but may also involve the choroid, as would appear from the thinning of this membrane observed when it is partially absorbed.

5. The origin and course of the disease are obscure, but are evidently connected with the blood-vessels, being due either to some condition of the system affecting the retina and kidneys equally, or to the diseased condition of the kidneys which, by altering the character of the humours, initiates the pathological changes observed in the blood-vessels.

6. The patients, in the chronic cases, never recover, but may live for a year or two after the first symptoms have been recognised.

(See Figs. 1, 2, and 3, showing (1) the healthy retina, (2) the disease in its early stage from the case of a patient who, at the time, was in the Infirmary, and (3) in its advanced stage from the case under consideration.)

The state of the eyes, then, as a matter of course, led us carefully to investigate the condition of the kidneys. As you are aware, there are three forms of chronic Bright's disease: 1. The chronic inflammatory form, that which specially affects the uriniferous tubules; 2. The amyloid, which, in the first instance, attacks the blood-vessels; and 3. The contracted, granular, cirrhotic, or gouty form, which specially involves the interstitial tissue. Any one of these may be complicated with albuminuric retinitis, but the last with much greater frequency than the others. Let us, therefore, run over the main features of it, and see whether they correspond with those observed in our patient.

It is for the most part a disease of adult males. It is not uncommon between twenty and thirty (Grainger Stewart), but is more frequently met with in older persons, as is apparent from the statistics of Dickinson, who found that the average age of 250 patients examined by him was 50.2 years. Our patient was a male aged 41.

The subjects of it gradually lose flesh and strength, become pallid, and frequently complain of headache and of giddiness just as our patient did. The cause of the headache has been variously stated: many hold that it is due to defective elimination by the kidneys and consequent accumulation of poisonous excrementitious matters in the blood; while others suppose that it is dependent upon the anæmia, and point, in corroboration, to the relief sometimes experienced by the administration of ferruginous preparations. In all probability, sometimes the one condition, sometimes the other, gives rise to it. For similar reasons, they are very liable to catch cold, and more or less bronchitis is a pretty uniform accompaniment; so that it was not surprising to note, in the case under consideration, the presence of cough with mucous expectoration and slight bronchitic râles, especially at the base of the lungs. You have learned, no doubt, that one of the most striking symptoms to look for in cases of Bright's disease is dropsy, but in the contracted form it is absent in from one-quarter to one-half of the cases, and, when present, it is slight and usually transient; so true is this, that if you meet with a patient labouring under the cirrhotic kidney who is markedly dropsical, you may be pretty sure that it results from some complication. Our patient has had no dropsy at all, and his urine presented the characters which we might have expected. It was passed in large quantity (over one hundred ounces in twenty-four hours), was pale, its specific gravity was low (1009), and it contained a fair amount

of albumen (one-third). On leaving it to stand, a scanty deposit was thrown down, in which the microscope detected a few structureless and finely-granular tube-casts.

Again, he had stomach-symptoms such as frequently occur in connection with the contracted kidney. On referring to the history of the case, we find it stated that, "along with the impairment of sight, irritability of the stomach came on, and to such an extent that he could seldom retain food for any length of time, especially if he got up and walked after a meal. About an hour after food, he complained of heartburn, and brought up sour mouthfuls, and this terminated in vomiting the contents of his stomach." This irritability of the stomach is sometimes indicative, as *post mortem* examinations have proved, of chronic gastritis, but it often, I believe, results solely from defective excretion by the kidneys, and consequent retention of poisonous ingredients in the blood—is, in fact, uræmic; and it is right you should know that for long it may be the most striking symptom called forth by the disease of the kidneys. Some years ago, a medical man, himself a distinguished teacher of medicine, began to complain of sickness which always set in if he fasted for more than two hours. This symptom continued for months without either himself or his medical advisers suspecting its cause, but at last his urine was examined and found to be albuminous, and about two years afterwards he died with all the symptoms of uræmic poisoning.

There can be no doubt, then, that our patient was suffering from the effects of granular degeneration of the kidneys; but, if further proof be wanting, it is to be found in the discovery of lesions of the circulatory and nervous systems. On reference again to the history of his illness, we note that "about eighteen months before admission he began to complain of palpitation associated with uneasiness in the præcordial region, especially on exertion, and some months afterwards to these symptoms was added dyspnoea".

On making a physical examination of the heart, we found that there was slight fulness in the præcordial region; the apex-beat was displaced somewhat downwards, and carried decidedly to the left (half an inch to the left of the nipple-line); its area of visible impulse was preternaturally great, and it was strong and heaving in character. On percussion, too, there was an increased area of dulness in a downward direction and to the left, and the sounds of the heart, though pure, were unusually loud and strong. All this shows that the left ventricle was the seat of hypertrophy. Now, why should such a condition arise in connection with the granular kidney? The explanation is obvious. The blood being poisoned by the excrementitious matter which ought to be excreted by the kidneys, the minute arteries throughout the system, arterioles as they are termed, are irritated and contract, and their muscular coat, as pointed out by Dr. George Johnson, becomes hypertrophied. There is thus an obstacle to the onward flow of arterial blood to the capillaries, to overcome which the left ventricle contracts with unusual vigour, and, as a consequence, it becomes hypertrophied. This excessive action of the heart on the one hand, and the state of contraction of the arterioles on the other, produces, as was noted in this case, a full hard prolonged pulse, a pulse of high arterial tension. This is well shown by the sphygmographic tracings which were kindly taken by Dr. McVail; the first being the normal tracing from the radial artery of our esteemed resident medical officer Dr. Sewell, the second from the patient whose case we are considering.

Another feature in this case—a very common one it is, and one which in part explains the attack to which I shall immediately refer—was, that the superficial arteries pulsated very visibly, were tortuous, and felt like firm cords; that is to say, they were atheromatous, leading one to suspect that the delicate vessels in the brain were atheromatous and brittle too. And now let me refer once more to that part of the history wherein it states that one night, while undressing, "he had a severe attack of vomiting, and a few minutes afterwards, having gone to bed, his breathing became stertorous, and he could not be roused. Along with the insensibility, which continued until six o'clock the following evening, the whole of the left side became completely paralysed. The paralysis was decidedly less by the following evening, and on his admission had in great measure disappeared". The paralysis was supposed to result from the rupture of a cerebral vessel in the neighbourhood of the right corpus striatum, for there was everything to favour such a lesion. This will be apparent when I remind you that, on the one hand, the hypertrophied ventricle was driving the blood with violence into the cerebral vessels, while, on the other, the arterioles were in a state of contraction, and obstructed the onward flow of the blood; an extra strain was thus put upon the larger cerebral vessels, which we suspected to be brittle and atheromatous, and nothing could be more likely than that one of them should give way.

These cases of contracted kidney are very insidious in their onset and course, and are extremely apt to be overlooked both by the pa-

tient and medical attendant, because often there is no very prominent symptom for a long time, only a gradual loss of flesh and strength, with more or less pallor. On this account, medical advice may not be sought until the disease is far advanced, and when some striking disturbance of function has supervened, such as obstinate irritability of the stomach, failure of vision, an attack of convulsions, or a paralytic seizure. In our patient's case, advice was only asked for when the eyesight became impaired, and yet the disease must have been going on for long, because albuminuric retinitis only occurs in an advanced stage of the disease, and because the history points to the existence of hypertrophy of the left ventricle eighteen months before the vision became dim, and hypertrophy itself does not ensue until the disease has made some progress, and has induced long continued contraction of the arterioles from the poisoned blood passing through them.

This patient was sure to die, and that at no very distant period. I need not dwell upon the treatment, further than to say that we gave him whatever light nourishment he could take, and, along with it, a course of iron in effervescence,* as it is then more readily tolerated, and, in combination with hydrocyanic acid and bismuth, to soothe, if possible, the irritability of the stomach.

For some days there was some improvement in this patient's state; but soon the irritability of the stomach reappeared and became uncontrollable. Suppression of urine gradually set in, followed by coma and death, on January 20th, fifteen days after admission.

The *post mortem* examination was made by Dr. Joseph Coats, Pathologist to the Infirmary, and the microscopic examination of the eyes and kidneys by Dr. Thomas Reid, with the following result.

Brain.—There was considerable oedema of the pia mater all over the convexity, the sulci being filled up with a clear fluid. The ventricles did not contain an excess of fluid. In the left occipital lobe there was a pretty extensive softening of the brain-substance involving almost the entire bulk of three or four convolutions, the cavity caused by the softening being covered almost directly by the pia mater of the surface. The convolutions involved were situated on the external aspect, and at the extreme posterior portion of the hemisphere. The cavity contained a turbid fluid of a brownish-yellow colour, and the wall of the cavity had a yellow colour, and was composed of softened brain-substance. In the right corpus striatum there was a distinct cyst as large as a hazelnut. This was situated in the most external part of the corpus striatum, involving a portion of the most external part of the nucleus lenticularis, the external capsule, and nucleus tæniæformis. The cyst was pretty far back, its anterior margin nearly corresponding to the anterior margin of the thalamus opticus. The cyst was lined by a distinct vascular membrane, and had one or two septa running through it. It was separated from the neighbouring brain-substance by the membrane mentioned. The larger arteries of the brain were the seat of numerous patches of atheroma, this condition extending to vessels of the third or fourth order. On microscopic examination of the wall of the cysts of the corpus striatum, and of the cavity in the occipital lobe, there were found multitudes of compound granular corpuscles, as well as a few blood-crystals and granular pigment. The blood-pigment was not abundant, but still present in every part; otherwise the brain-substance appeared normal.

The woodcut (Fig. 5) shows the microscopical appearances of a section of the retina and choroid.

Chest.—The pericardium contained several ounces of a straw-coloured fluid. The heart was enormously enlarged, weighing twenty-three ounces. The enlargement involved chiefly the left ventricle, whose walls were very thick; the muscular tissue was rather pale. There was no thickening of the valves. The mitral orifice was slightly dilated, admitting three fingers. The lungs were slightly oedematous, and their margins were emphysematous.

Abdomen.—The liver was enlarged, and showed evidences of chronic congestion, viz., nutmeg markings. The spleen was also enlarged, being about double its usual size. The mucous membrane of the stomach was slightly thickened and irregular. The left kidney weighed two ounces and a quarter. The capsule was firmly adherent. The surface was finely granular, but there were no deep cicatrices. On section, the tissue was seen to have a generally red tint, but not dark red. The tissue was firm. The cortex was not distinctly thinner than usual as compared with the pyramids, but its normal markings were obscured.

The woodcut (Fig. 6) gives a better idea of the microscopical appearances of a section of the kidney than any verbal description can convey.

* R. Ferri citratis 3iss; acidi citrici 3vj; aquæ dest. ad 3vj.—R. Acidi hydrocyanici dil. ℥lxxii; potassæ bicarbonatis 3vj; liquoris bismuthi, syrupi aurantii, sing. ʒijj.—M. Sig. A dessertspoonful of the contents of each bottle in a glass of water thrice daily.

Fig. 1.—Normal appearance of the Fundus of the Eye, as seen with the Ophthalmoscope.

Fig. 2.—Fundus of the Eye in the early stage of Retinitis Albuminurica, showing hemorrhagic spots and a few spots of fatty exudation.

Fig. 3.—Fundus of the Eye in the advanced stage of Retinitis Albuminurica, showing well marked, diffused fatty degeneration.

Fig. 6.—Vertical Section of the Cortical Substance of the Kidney (magnified 120 diameters). (a) Artery of medium size, with hypertrophied walls. (b) Transverse Section of Uriniferous Tube, showing thickened wall. (c) Sections of Uriniferous Tubes in various stages of degeneration. (d) View of the Internal Surface of Uriniferous Tubules, showing conversion of their walls into connective tissue. (e) Sections of Atrophied Uriniferous Tubules, containing granules, etc. (f) Sections of the same, empty, and with no defined wall. (g) Malpighian Tuft, slightly atrophied, and enveloped in a hypertrophied capsule.

Fig. 5.—Section of the Retina and Choroid near the Macula Lutea. (a) Nerve-fibre layer undergoing fatty degeneration, with transverse section of an hypertrophied artery. (b) Ganglionic Cell Layer, the empty spaces representing cavities filled with hyaline substance. (c) Outer Granular Layer containing granular cells of a brownish tint, as in (b). (d) Layer of Rods and Cones replaced by granular exudation. (e) Hypertrophied Choroid, with diminution of pigment.