CLINICAL LECTURE

ON

CHRONIC BRIGHT'S DISEASE, WITH CONTRACTED KIDNEY

(ARTERIO-CAPILLARY FIBROSIS).

Delivered at Guy's Hospital, June 6th, 1872.

By SIR WILLIAM GULL, BART., M.D., D.C.L.,

Physician Extraordinary to the Queen; Consulting Physician to Guy's Hospital.

[From Notes by MR. HENRY CLARKE.]

THE subject I have chosen for your attention to-day is one for which the clinical wards of Guy's Hospital are particularly famous. To use an expression of our German friends, Guy's Hospital is the "fatherland" of this disease. It was here that Dr. Bright pursued those investigations which have made his name immortal. "Bright's disease" is, however, but a generic expression, and appertains to many affections of the kidney, which, though more or less allied by the character of their morbid anatomy, so far as the kidneys themselves are concerned, are probably distinct in their cause and clinical history. It has long been felt that the term "Bright's disease" must either be discontinued, or be more strictly limited to one given pathological state.

The object which I have in view on the present occasion is to make some remarks on that general condition which is perhaps more properly Bright's disease, and which is associated with one form of fibroid contraction of the kidneys. And here it should be observed that the kidney may undergo fibroid contraction and disorganisation, and cause death by uræmia, without being associated with that change in the heart and arteries upon which I desire to discourse in this lecture. In such cases, the renal disease is probably due to some hereditary or congenital defect in the kidneys themselves, and is, more strictly, a local affection; and although the results, so far as the kidneys are concerned, may be very much like, or even apparently identical with, the more morbid conditions arising under other circumstances, what constitutional accompaniments there are are widely different from such as occur in the disease now spoken of.

The form of chronic Bright's disease, with contracted kidney, now under discussion, is, it would seem, a more general affection: it occurs chiefly at or after the middle period of life. Some of its various phases were, no doubt, recognised by the physicians of former times, and vaguely assigned, even so early as the Greek physicians, to special epochs of life, denominated by them "climacterics". It is chiefly between what they would have termed the seventh climacteric and the ninth—that is, between the forty-ninth and sixty-third years—that this morbid condition occurs. I do not quote these terms to give any support to the idea that there are climacteric epochs; but I may affirm that the state of which I am speaking is a mode of decline from the climax of health and vigour of a special kind.

As the facts of disease came to be more and more the objects of inquiry over mere symptoms at the bedside, and as morbid anatomy in support of such clinical observation was pursued more accurately after death, there would naturally be at first a tendency, with increased precision, to a somewhat narrow pathology: for instance, the investigations of the intestines in typhoid fever led Broussais and his followers, as we know, to maintain that fever was a local intestinal affection. A similar error seems to have been committed in respect of this form of chronic Bright's disease. The common change in the urine during life, and the morbid state of the kidneys after death, have so closely confined the attention of physicians, that they have often looked no further than the kidneys and their failing function for the grounds of their pathology, and their pathological views have been limited to the line thus indicated. Now, though no one will deny the almost paramount importance of keeping fully in view the results on different parts of the organism of defective renal excretion, and the tendency to death by uræmia, still we believe it can be no less maintained that the fundamental clinical facts of the disease in question are not included in such limited causation, and cannot be understood by the conditions of a merely renal pathology.

The constitutional form of chronic Bright's disease, with contracted

kidney, or, as Dr. Sutton and myself have desired to designate it, "arterio-capillary fibrosis", is, it would appear, a primary and general change in the capillaries and arterioles of the body, and corresponds, therefore, in its seat in the adult to the "area vasculosa" in the embryo. And as it is the object of the embryologist to elucidate the changes through which the ovum proceeds upwards, to the perfect development of its tissues and organs, so it may be broadly stated to be the object of the pathologist to set forth the steps which lead downwards to death. There are occasions which make it advantageous for the pathologist to enlarge the scope of his considerations by admitting this comparison; for it is obvious that what is generally expressed by the term "vis formativa", whose resultant changes we see in development, is represented by the "vis conservatrix" in the adult, which failing, we have senility and death.

Apart from accidental disturbances in the course of life, we might, perhaps, with due knowledge of the inherited forces, be able to predicate somewhat of the course of their failure. The curve of the ascent and the curve of the decline would, in other words, have some more or less exact correspondence, and we should be able to realise fully the truth that there is not only "a time to be born" and "a time to die", but death might be predicted for a given individual to come in a certain time—say of any particular organ or of any particular system: for instance, we might be able to foretell that the failure would first appear in the nervous, or the vascular, or the secreting system. The form of chronic Bright's disease, with contracted kidney, is, it seems to us, one example of such (senile?) decadence.

Its clinical history varies in every case, according to the organ which suffers most and earliest. In one case, sleeplessness, headache, and other slight brain-disturbances, may be the first symptoms complained of; in another, the altered condition of the heart may first draw attention; in a third, loss of appetite, dyspepsia, sickness, and failing digestion, are most prominent; in a fourth, diuresis; in a fifth, a liability to bronchitis, with attendant emphysema; in a sixth, a state of general *malaise*, loss of strength, some emaciation, etc.

These various phases, separately or in combination, constitute the earliest ailments for which the physician is commonly consulted. They are for the most part vaguely referred to a gouty condition; and it must be admitted that the general morbid state of which we are speaking, though distinct from gout, is especially common in those whose ancestors have been gouty. It is often more erroneously referred to suppressed gout. This is less appropriate, since, except the vague flying pains to which such patients are liable, and the occasional deposit of uric acid or urates in the urine, there have often been no phenomena which could properly be termed gouty.

In a large number of cases which come daily before the physician, no doubt the disturbances in question are no more than a mere variation in the general equilibrium of nutrition, which, by due care as to diet and exercise, and the other general conditions of health, are easily corrected; but in a considerable number of persons they are the early signs of a general change in the vascular area, of which disease of the kidneys may or may not be an early and prominent part.

And here I may refer to the evidence supplied by the urine at the outset of chronic Bright's disease, with contracted kidney. Hitherto it has been the habit of the clinical physician to take the specific gravity of the urine, and to test it for the presence of albumen: should the specific gravity be normal (1020 to 1024), and should no change in the fluid be produced by heat and nitric acid, whatever ailments have been present, they have not been referred to the early stages of Bright's disease, but have been explained, as I have said above, by reference to some gouty condition, or to some limited change—functional or otherwise—in the part complained of: for instance, if in the stomach, to the food taken; if in the chest, to catarrh from cold. If flying pains have been complained of, they have been referred to neuralgia or rheumatism; if headache, to overwork, etc. But, assuming that we have to do with a case of coming Bright's disease in so early a stage, the following may be considered as not uncommon clinical conditions. The heart may be even already thickened, so far as to produce an increased impulse over an increased area. These signs of hypertrophy are somewhat more obvious, as already there may be a loss of subcutaneous fat, and, although not very marked, a further distinct loss of weight.

In a case that lately came before me, the patient, a gentleman aged 53, although he made no mention of emaciation, yet, when questioned, said his weight had declined within a year from fourteen stone eleven pounds to thirteen stone eleven pounds. Cardiac hypertrophy under these conditions may have no connection with valvular disease. Indeed, at an early stage both the loss of weight and the hypertrophy of the heart may easily be overlooked; the hypertrophy especially, as the

cardiac sounds may have undergone no change, or only a slight loss of distinctness in the first sound.

The examination of the urine at this stage gives varying results. For the most part, there is no discoverable change from health, or only the presence of a faint and doubtful opalescence, after boiling and the addition of nitric acid. Even this change may be absent altogether till later on in the disease; or, before it becomes constantly present, we may find albumen only in the urine passed after food. The presence of albumen may also alternate with the deposit of urates or uric acid. Albumen is often altogether absent when these deposits are present; but the relations of albumen to the deposits of uric acid and urates of soda and ammonia are by no means constant. These deposits and the albumen may occur together in the earlier stages of fibrosis of the kidney. It is by no means uncommon for even careful observers to overlook or lightly estimate the early indications which the urine thus affords of commencing fibroid cachexia.

Although the renal change is probably but part and parcel of a general state of morbid changes, the clinical value of the evidence afforded by the urine in the early stages in many cases is most important. It is desirable to examine the urine passed at different times of the day. The early morning urine may be normal in colour and weight (specific gravity, 1018-1022). That passed at noon may be still without deposits on cooling (1022-1024), with or without a faint opalescence after heat and nitric acid. That passed at 6 P.M. (assuming the dinner to be a late one) may be nearly the same as that at noon. That passed on going to bed may deposit urates, sometimes with, sometimes without, faint change from albumen by heat and nitric acid (specific gravity, 1024-1026). It is at this early stage that the symptoms are vaguely referred to disorder of the liver or stomach.

Those physicians whose minds are made up as to the cause of the cardiac hypertrophy in chronic "Bright's disease," may insist that even already there may be such a defective excretion from the kidneys as so far to interfere with the physiological relations of the blood to the tissues, that it cannot pass through the systemic capillaries without an increased impulse from the heart, and that the hypertrophy is so caused. But the assumption of such an altered state of the blood as a true cause of the hypertrophy is entirely unsupported by any observations on the blood itself, and is not supported by evidence on the side of the urine itself. The specific gravity, as stated above, may be normal; the quantity and colour may be normal; the cold urine treated with nitric acid may become as usual nearly solid with nitrate of urea; and the only change, and this but occasional, may be a faint opalescence, under heat and nitric acid, due to the presence of albumen. No doubt in a more advanced stage the uric acid and the urates may cease altogether to be deposited as the urine cools. The urine loses colour and specific gravity, and is considerably increased in quantity, though still in general free from all deposits of desquamated epithelium; as the albumen increases, it contains very transparent fibrinous casts, which are complementary of the albumen of the liquor sanguinis, and equally due with it to mere passive transudation. When these changes in the urine have become more marked, and the general cachexia is advanced, the renal affection is set down naturally, if the case is seen at this stage for the first time, as a primary cause of all the attendant changes, cardiac and capillary. But such a view seems to my colleague and myself to be an erroneous one, and to have arisen from a deficiency in our early clinical knowledge of such cases. Nor can this deficiency be easily supplied from hospital practice.

Poor men who are dependent upon bodily labour for their livelihood do not so often present themselves at hospitals in the early stages of disease. It is in private practice that we have to make good our wants in these particulars, and even here the scientific cultivation of medicine suffers by the readiness to explain away the causes of ailments down to the comprehension of the uninformed. It is, besides, to be noticed, that even when our hospitals afford evidence it is indistinct. In an early stage of fibroid cachexia, persons are apt to die of acute disease in the brain or lungs before the amount of renal change is enough to give it any recognised clinical significance. These early deaths take away from us the opportunity of observing the further course in such cases of the renal degeneration which, apart from such accidents, might soon have become a prominent clinical feature, though it escapes attention when less marked; and the subject is dismissed, with only this recognition, that the kidneys are coarse. I do not see how we could easily have escaped from the trammels of a narrow pathology, and, if I may be permitted to say so in respect of the matter under discussion, from its vicious influence on practice, if our observations had continued to be limited to the more prominent lesions of advanced "Bright's disease," as they present themselves at the bedside, and even on the *post mortem* table.

[To be continued.]

HIPPOCRATES: HIS LIFE AND WRITINGS.

An Inaugural Address delivered before the Royal Medical Society of Edinburgh on the 8th of November, 1872.

By J. WARBURTON BEGBIE, M.D., F.R.C.P.Ed.

MR. PRESIDENT AND GENTLEMEN,—I am going to ask your attention at this time to the life and labours of one of the greatest men connected with our profession in any age or clime. And, although his name is the very earliest name of distinction in Medicine, a careful consideration of his character and work, as these are unfolded to us in his writings, will satisfy all candid minds that we owe a great debt of gratitude to the "Father of Medicine," and that much in the way of instruction and encouragement is to be gathered by us from a diligent perusal of these writings.

In the four hundred and sixtieth year before the birth of Christ, according to very general belief, Hippocrates was born. Whether the date now given was the exact year of his birth or not, is not a matter of much importance. We know, on the authority of Aulus Gellius (who flourished in the second century), in the twenty-first chapter of the seventeenth book of the *Noctes Atticæ* (so called because the work was composed by Aulus Gellius in a country house near Athens during the long winter nights), that Hippocrates and the illustrious Socrates (although born later) were contemporaries. "Itaque inter hæc tempora nobiles celebresque erant Sophocles ac deinde Euripides tragici poete, et Hippocrates Medicus, et Democritus philosophus: quibus Socrates Atheniensis natu quidem posterior fuit; sed quibusdam temporibus iisdem vixerunt." That Hippocrates attained a very considerable age appears sufficiently ascertained, but in regard to the exact time of his decease there has arisen a difference of opinion. The years of his life have been fixed at 85 by some; at 90, 99, 104, and even as many as 108 by others. Which of these statements is correct, we have no proper grounds for determining. We know, however, that the period of the greatest activity of Hippocrates, when his reputation reached its greatest height, was the earlier part of the fourth century before the birth of Christ. Flourishing, therefore, in the time of Pericles, besides the greatest of Athenian statesmen, Hippocrates had for his contemporaries Socrates, Democritus, Herodotus, Thucydides, also Plato and Xenophon, the distinguished disciples of Socrates. Among poets too he had his birth; Æschylus, Sophocles, Euripides, Aristophanes, and Pindar flourished at the same time. Phidias, moreover, the "Sculptor of the Gods," the architect, at least superintendent of works, of the Parthenon, the sculptor of the Olympian Jove, was his contemporary. Hippocrates thus came into the world under circumstances which must have co-operated with his own remarkable genius in raising him to the extraordinary eminence which during his lifetime he attained, and which his name has in all subsequent ages retained. In the words of Celsus, as true now as when penned by him in the first century, that is, four hundred years after the death of Hippocrates, in alluding to his separation of the study of philosophy from medicine, "Hippocrates Cous, primus quidem ex omnibus memoriâ dignis, ab studio sapientiæ disciplinam hanc separavit, vir et arte et facundiâ insignis."

You have all heard of Æsculapius (*Ἀσκληπιός*, in Greek)—the god of the medical art, or of Medicine. Homer makes no mention of the origin of Æsculapius; but you will remember his sons Machaon and Podalirius, who were medical men as well as commanders of troops in the Greek army during the Trojan War. Although Homer does not refer to Æsculapius as a divinity, but merely as a physician—styling him "*ἰατὴρ ἀμύμων*" "the blameless physician"—he was worshipped as a god over the length and breadth of Greece. His temples (*Ἀσκληπεία*) were, however, not only places where the god was worshipped, but were resorted to by numbers of sick people, who received treatment in them, and were therefore to be compared to hospitals in modern times. Hippocrates has generally been regarded as the lineal descendant of Æsculapius. Putting aside the fabulous, there can be no question that he belonged to the family of the Asclepiade, the name given to the supposed descendants of the god. The genealogy of Hippocrates is given as follows by Tzetzes, the learned but self-conceited Greek grammarian of Constantinople in the twelfth century, after the manner of an infinitely more exalted genealogy which we have in a still more ancient book. "Æsculapius, who was the father of Podalirius (him of Troy), who was the father of Hippolochus, who was the father of Sostratus, who was the father of Dardanus,