of the inner side of the left thigh was a large, hardish, ill-defined tumour, not painful, irregular in shape, and lobulated on its surface, which was deeply imbedded in the muscles, but not connected with the bone. It had been first noticed some months previously as a small, hard, deeply-seated lump, which for awhile had remained pretty stationary; but of late it had been growing rapidly. Several enlarged glands existed in the groin. There had been no accident or injury of any kind. Divers opinions had been given as to the nature of this tumour, and a hint had been thrown out as to its perhaps being malignant. On further examination, the right groin also proved to be full of enlarged glands, amygdaloid glands as they are called now-a-days; and then came the question as to syphilis, but of this, either as primary or as secondary, there was no history whatever, and the patient had no traces of hereditary syphilis about him. Nevertheless, with these amygdaloid glands in the groins, it was thought advisable to treat the case as one of tertiary; and when I last saw the patient the tumour had all but disappeared, there being merely a slight trace of thickening at the part where it had existed; and the patient himself was now a strong and healthy looking man.

As surgeons, it is in such a case as this that it especially behoves us to be on our guard. When several tumours of this kind exist in various parts, attention may, by their multiplicity, be directed to the cause of their existence; but with one tumour only an error in diagnosis may very easily occur. A short time ago, at a consultation at which I was present, concerning the removal of a tumour from the back, a question arose as to its perchance being of a syphilitic nature; amygdaloid glands were found in the groins, and a distinct history of syphilis of a few years standing was obtained; the case was treated as one of tertiary, and in due time the tumour disappeared. But patients have, in such cases, sometimes undergone formidable operations. In one case, a large portion of the scapula was removed; and in another, in which the whole tongue had been cut out for cancer, there were good reasons for believing that the disease was of a syphilitic nature.

As far back as the year 1845, Mr. Tatum put into my hands a paper on "Tumours of Muscles cured by Iodide of Potassium"; and, as in two of the cases, at any rate, there were distinct evidences of constitutional syphilis, I advised him to send the paper to the Royal Medical and Chirurgical Society, where it was read; but it was not thought fit for publication in the *Transactions*. In the present day, Mr. Tatum's paper would naturally have found a place in the records of the Clinical Society; and here I think lies especially the value of such a Society, dealing as it does with short practical papers, which may apparently be unimportant at the time, but which in after years become of value.

UTERINE CANCER.—At a meeting of the Berlin Obstetrical Society on November 26th, Herr Ponfick showed two specimens of cancer of the female reproductive organs. In one, the ovaries were converted into hard nodulated masses, and there were cancerous deposits in the neighbourhood of the uterus, especially near the bladder and rectum, and cancerous induration of the mesenteric glands. The uterus was not enlarged. In the second case, the whole body of the uterus was enlarged and infiltrated with cancer; its vaginal portion was destroyed; and the vagina, which was enormously wide, contained cancerous nodules. The parts near the uterus were free; but cancerous degeneration was found in the glands in the abdomen, chest, and neck, and in the thyroid body. The body of the second lumbar vertebra and the left pleura were also affected. Herr Ponfick referred to the affection of the cervical glands as of rare occurrence in uterine cancer.—*Berliner Klin. Wochensh.* February 3rd, 1873.

EFFECTS OF QUININE UPON THE UTERUS.—Dr. F. K. Bailey of Knoxville, Tennessee (*Philadelphia Medical and Surgical Reporter*, November 9th, 1872), gives the result of his experience in a malarious district. "It was very common to find a pregnant woman suffering from uterine pain during a fit of ague, and abortion or premature labour often occurred in cases where timely antiperiodic treatment could not be carried out. In all such cases it was my practice, as well as that of others, to administer quinine promptly and freely. There was no other way to carry a patient over such a period of danger, except to suspend the periodical tendency. Again, the question is often asked, Is quinine admissible or ever indicated during labour, to regulate the expulsive efforts of the uterus? In answer to this, I can say that, in very many instances during labour occurring in the summer or autumnal months, I have given quinine with the most happy effects. It was often the case that a natural labour at term was ushered in by a chill; and the depressing effects of malaria would render the pains feeble and without expulsive power. At this juncture, a full dose of quinine would equalise the circulation and render contractions more forcible."

LECTURES

ON THE

PATHOLOGY, DIAGNOSIS, AND TREATMENT OF BRIGHT'S DISEASE.

By GEORGE JOHNSON, M.D., F.R.S.,

Physician to King's College Hospital; Professor of Medicine in King's College, London; etc.

LECTURE III.—CHRONIC BRIGHT'S DISEASE (*concluded*).

Local and General Symptoms of Contracted Granular Kidney: Frequent Micturition; Pain in the Back.—Dyspepsia as a Cause and a Consequence of Renal Disease.—Dropsy.—Hypertrophy of the Heart.—Inflammation of Serous Membranes.—Hæmorrhage from various Surfaces.—Cerebral Hæmorrhage.—Impairment of Vision.—Cerebral Symptoms the Result of Uremia.—Theory of Uremia.—Nervous Dyspnoea.—Disease of the Liver.—Diagnosis.—Prognosis.

The Local and General Symptoms of Contracted Granular Kidney.—There are few diseases equally serious, whose progress is so insidious as that of the disease which we are now considering; yet there are few maladies whose presence is indicated by more unequivocal signs, if only they be diligently and intelligently sought for.

One of the earliest symptoms, in the majority of cases, is *increased frequency of micturition*, and especially during the night. The more frequent call to empty the bladder may be a result of a more copious secretion of urine and consequent distension of the bladder, or it may be due to irritation of the bladder by some abnormal quality of the secretion. This symptom is sometimes absent, and it may result from other causes than renal disease. When present, it serves to direct attention to the urinary organs; and it is a symptom which should never be neglected.

Pain in the back is not a frequent or an important symptom. In many cases, it is entirely absent; and often it is not spoken of until the patient's attention has been directed to the subject. When present, it is more frequently muscular than renal—an aching pain in fatigued and feeble lumbar muscles, and often complained of by debilitated patients who have no renal disease. In numerous instances, a patient in the advanced stage of incurable degeneration of the kidney has said, "I cannot understand how my kidneys can be diseased, since I have never had pain in them."

Dyspepsia is frequently associated with this form of disease, sometimes as a cause, sometimes as a consequence. You may often learn that a patient of strictly temperate habits has for months or years suffered from pain or uneasiness after food, flatulent distension of stomach and bowels, occasional nausea and vomiting, habitual looseness or irregularity of bowels, constipation and diarrhoea alternately. With this, there is often turbidity of the urine, which is high coloured, excessively acid, and deposits urates abundantly. After a time, the urine, which had been scanty, becomes more copious, of pale colour, of low specific gravity, and is found to contain albumen and granular casts. In such a case, probably renal degeneration is a consequence of the long continued elimination of products of faulty digestion through the kidneys. I have seen this sequence of events so frequently, that I have no doubt as to their causative relationship. Dyspeptic symptoms such as I have described, and consequent renal degeneration, are in some cases excited or greatly aggravated by habitual excess of alcohol—less frequently, perhaps, by excessive smoking of tobacco.

In other cases, dyspepsia is a *consequence* of advanced renal degeneration. Urea and other urinary products are vicariously excreted by the mucous membrane of the stomach and bowels, in consequence of the defective action of disorganised kidneys. The gastric secretions are deranged; the digestive functions are disordered; and nausea, vomiting, and diarrhoea, are amongst the results of this secondary renal dyspepsia.

The chronic degeneration of the kidney which we are now considering is often preceded and accompanied by such symptoms as the following: a gradual loss of energy, with emaciation to a variable extent; unusual fatigue after exertion, with a tendency to rheumatic pains and cramp in the feeble and fatigued muscles; defective perspiration, with a dry and harsh state of the skin; a peculiar pallid or sallow complexion; and a watery condition of the conjunctiva, or of the connective tissue beneath it. Pallor is not a constant symptom; there is sometimes a florid complexion even in the advanced stages of this form of degeneration. The tongue is sometimes dry; at other times, moist

and pale. There is often thirst, with loss of appetite and some of the dyspeptic symptoms before mentioned. Not unfrequently there is pain or a sense of weight in the head; sometimes a tendency to drowsiness, and occasional dimness of sight.

Whenever symptoms such as I have described are complained of, the urine should be carefully examined. I need not repeat what I said in the earlier part of this lecture of the indications afforded by the urine from the earliest to the latest stage of this form of renal degeneration.

Dropsy, as I have before told you, is not a prominent symptom in this form of disease. In the majority of cases, it is absent throughout the whole progress of the malady. Excluding those cases in which there is the complication of valvular disease of the heart, I found that, of thirty-three fatal cases of contracted kidney, there had been dropsy in only fourteen, the proportion being 42 per cent.; and in most of these fourteen cases the dropsy was only slight and partial, coming on towards the close of the illness. (See a paper on the Forms and Stages of Bright's Disease, *Med.-Chir. Trans.*, vol. xlii.) The comparative infrequency of dropsy is explained by the free and often copious secretion of urine, which, as a rule, is not highly albuminous. There is not so great a deficiency of the normal blood-constituents in this form of disease, as in most acute cases and in other forms of chronic disease. The specific gravity of the blood-serum is less reduced, and the proportion of water to solids less excessive. An excess of urea, however, is often found in the blood, especially in the later stages, when the secretion of urine becomes scanty.

Hypertrophy of the heart occurs in a large proportion of cases of contracted kidney when the disease has reached an advanced stage. In some cases, valvular disease, in others, atheromatous and calcareous degeneration of the walls of the large arteries, suffices to explain the hypertrophy; but in other cases, as Dr. Bright pointed out more than thirty years ago, there is no such obvious explanation of the hypertrophy, which affects chiefly the left ventricle; and he suggested, as a probable explanation, that "the altered quality of blood so affects the minute and capillary circulation as to render greater action (of the left ventricle) necessary to force the blood through the distant subdivisions of the vascular system." (*Guy's Hospital Reports*, vol. i.) About six years ago, it occurred to me that the hypertrophy of the left ventricle of the heart in cases of contracted kidney might be a result of increased contraction of the small arteries throughout the body, this contraction being excited by the abnormal quality of the blood; and I went on to argue that, if this were so, we should find evidence of the fact in the existence of hypertrophy of the muscular walls of the minute arteries in various tissues. And we have found this hypertrophy not only in the arteries of the kidney, but also in those of the skin, the intestines, the muscles, and the pia mater. It probably exists in the arteries of other tissues which we have not examined.

The probable explanation of the hypertrophied left ventricle in the advanced stage of contracted kidney, then, appears to be this. In consequence of degeneration of the kidney, the blood is morbidly changed. It contains urinary excreta, and it is deficient in some of its own normal constituents. It is, therefore, more or less unsuited to nourish the tissues, and probably more or less noxious to them. The minute arteries throughout the body resist the passage of this abnormal blood, and in consequence the left ventricle beats with increased force to carry on the circulation. The result of this antagonism of forces is, that the muscular walls of the arteries, and those of the left ventricle of the heart, become simultaneously hypertrophied.

Now I wish to direct your attention to the fact that hypertrophy of the left ventricle, indicated by the apex beating below and external to its normal position, with a strong heaving impulse, and the second sound accentuated over the aortic valves, without signs of valvular disease or senile degeneration of the arterial walls, but with a full resisting radial pulse and high arterial tension, may be taken as evidence that the renal disease is not only chronic, but also in an advanced stage. These physical signs, therefore, will assist you in forming a prognosis.

Both the investing and the lining membrane of the heart are liable to become inflamed, as a result of blood-contamination during the progress of the renal degeneration. This complication will be indicated by the local, general, and physical signs of pericarditis or endocarditis, or, it may be, of both combined. Other serous membranes sometimes become inflamed—the pleura more frequently than the peritoneum. Œdema of the lungs and bronchitis are frequent complications. Pneumonia is comparatively rare, but it does sometimes occur.

Hæmorrhage.—In the advanced stages of the disease, hæmorrhage from different mucous surfaces is a common and often a troublesome and alarming symptom. Epistaxis is the most common form of hæmorrhage; but I have seen it occur from the stomach and intestines, from

the lungs, the bladder, and from the uterus in the form of menorrhagia. Amenorrhœa is, however, according to my experience, a more frequent result of advanced Bright's disease than menorrhagia.

Cerebral Hæmorrhage.—The most serious and by no means the least frequent form of hæmorrhage is that which takes place within the cranium. In a large proportion, probably half, of the fatal cases of sanguineous apoplexy, the kidneys are found more or less diseased; and the granular degeneration which we are now discussing is the form of disease which is most frequently associated with cerebral hæmorrhage. The explanation of this common and too often fatal accident is not difficult. The minute cerebral arteries resist the passage of the abnormal blood, while the hypertrophied left ventricle is forcibly driving it onwards. Meanwhile, the walls of some of the intermediate arteries undergo atheromatous degeneration—partly, perhaps, in consequence of the circulation of morbid blood, partly a result of the unusual strain and pressure to which they are subjected. At length, in the struggle between the propelling left ventricle and the resisting muscular arteries, a brittle artery gives way, and a fatal hæmorrhage occurs.

Impairment of vision is one of the most serious results of granular contraction of the kidney. It occurs in two distinct forms. 1. The impairment of vision may be so sudden in its onset, that in a few minutes or hours there is complete blindness, which usually passes away as suddenly as it came. The attacks of sudden and transient blindness may recur again and again. In these cases, ophthalmoscopic examination discovers no structural change within the eye. This form of amblyopia is believed to be of uræmic origin, and is designated uræmic amaurosis. It is usually associated with other symptoms of uræmia, and I shall presently have something more to say of its pathology. 2. In the second form of impaired vision, the dimness of sight comes on more slowly, and is more durable. One eye alone may be affected, but both are often implicated simultaneously or in quick succession. The ophthalmoscope reveals peculiar structural changes in the eye, the result of which is called *retinitis albuminurica*. You will find these appearances fully described and depicted in works on diseases of the eye, amongst which I may especially mention the elaborate and excellent treatise of my colleague Soelberg Wells. Dr. Clifford Allbutt, too, in his able and instructive book on the *Ophthalmoscope*, has an interesting chapter on the Retinitis associated with Albuminuria. The most characteristic ophthalmoscopic appearances are a broad glistening white mound surrounding the optic disc, the result of sclerosis of the optic nerve fibres and fatty degeneration of the connective tissue elements. The extreme margin of the white mound is broken up into small irregular patches, which assume in the neighbourhood of the yellow spot a peculiar stellate arrangement. The retinal arteries are diminished both in size and number, while the veins are dilated and tortuous. Blood-extravasations, varying in number and in size, sometimes both numerous and large, occur here and there, chiefly in the internal layers of the retina, but sometimes in the external layers, or between the retina and the choroid. The coats of the blood-vessels are sometimes found in a state of sclerosis or fatty degeneration. These structural changes appear to be of an inflammatory and degenerative character. They are associated more commonly with the contracted kidney than with other forms of chronic Bright's disease. So characteristic are the appearances in the retina, and so insidious is the disease in the kidney, that an ophthalmoscopic examination for determining the cause of dimness of sight has in many instances led to the discovery of an unsuspected renal disease. It may be well to mention here that the two forms of impaired vision which I have described may occur together or in succession in the same subject. Uræmic amaurosis may in time be succeeded by albuminuric retinitis; and the dimness of vision which results from the latter may be temporarily much increased by uræmic amaurosis. The hæmorrhage into the retina may be explained partly by the injecting force of the hypertrophied ventricle, partly by degeneration of the walls of the retinal vessels, and partly by venous engorgement consequent on pressure upon the veins by inflammatory exudation.

Cerebral Symptoms.—In the advanced stages of contracted kidney, various forms of nervous disorder occur with so great frequency, that the disease may be said to have a natural tendency to terminate with symptoms referable to the brain. These nervous symptoms are very variable. In some cases, epileptiform convulsions or profound coma may occur suddenly, without premonitory symptoms. Much more frequently these formidable symptoms are preceded for a variable period by other indications of brain-disturbance. Amongst the commonest of these are headache more or less severe and constant, sudden transient vertigo, equally sudden and transient loss of sight or hearing, temporary inability to speak, or the speech for a time is imperfect and stammering; numbness or neuralgic pains, cramps, chorea-like twitchings or transient loss of power may occur in one arm or leg, or in both the

arm and leg on one side; there may be confusion of thought, impairment of memory, and an indescribable nervous dread, with a feeling of utter prostration. After one or more of these symptoms have continued for a variable period, or recurred more or less frequently, the secretion of urine being usually scanty, and vomiting of frequent occurrence, the patient perhaps becomes drowsy, with more or less delirium; the tongue is brown and dry; the breath has a most characteristic sour and fetid odour; the drowsiness gradually increases and deepens into coma; the pupils being natural or equally dilated, and the breathing more hurried than in ordinary cases of sanguineous apoplexy. And so death occurs either with or without convulsions. In some cases, a single attack of violent convulsion is immediately fatal; in others, the convulsions recur again and again for several hours before the fatal termination. The brain after death is usually found extremely pale and anæmic, with some serous effusion beneath the arachnoid and into the ventricles. These are cases of so-called "serous apoplexy"; but the amount of serous effusion is insufficient to compress the brain, and so to explain the symptoms.

Theory of Uræmia.—In attempting to explain these nervous symptoms, I assume it to be indisputable that they are the result of the blood being deteriorated, partly by diminution of its normal constituents, but chiefly by retention and accumulation of urinary excreta. There are two ways in which it is probable that the brain and its functions may be injuriously affected by this blood-deterioration. First, the cerebral tissues, fed with poor and poisoned blood, may have their nutrition impaired, and may in various parts undergo structural changes analogous to those which are often demonstrable in the texture of the retina. Second, it is probable that some of the cerebral symptoms, more especially those which come on and pass away suddenly, are directly due to temporary interruptions or hindrances of the circulation through certain regions of the brain, consequent on excessive contraction of the minute arteries. In my lecture on the Pathology of Epilepsy (published in the BRITISH MEDICAL JOURNAL, March 21st, 1868), I adduced many facts and arguments in support of the theory that the immediate cause of an ordinary epileptic convulsion is sudden and extreme anæmia of the brain, the result of excessive contraction of the minute cerebral arteries.

Our increasing experience of the various forms of nervous disorder which may result from so purely mechanical a cause as embolism, in vessels of various sizes and in different regions of the brain, gives additional support and probability to the theory, that many of the cerebral symptoms resulting from uræmia may be explained by a defective blood-supply to certain parts of the brain, consequent on arterial contraction. An arrest of the circulation through a portion of the brain involves immediate suspension of function in that part, with perhaps a disorderly action in subordinate and correlated parts. Thus amongst other symptoms of nervous disorder, maniacal delirium and acute chorea have sometimes been found associated with, and probably have been directly caused by, mechanical plugging of minute cerebral vessels; the plugging being a result of embolic particles of fibrine detached from the so-called warty vegetations on a damaged mitral or aortic valve. Again, sudden and complete blindness in one eye may result from embolism of the arteria centralis retinae; partial and patchy blindness from embolism in one of its branches. It is in a high degree probable that uræmic vertigo, amaurosis, delirium, convulsions, and even coma, may in some cases be explained by partial or general cerebral anæmia, the result of excessive arterial contraction excited by the presence of impure blood. I do not ask you to adopt this as a complete and final explanation of the phenomena, but suggest it as a theory to be tested by the results of further observation and research.

Let me add that in some cases, notwithstanding the scantiness and ultimately the almost complete suppression of urine, uræmic symptoms are almost entirely wanting, and consciousness remains until death occurs from exhaustion. In some at least of these cases the uræmic symptoms are probably prevented by the occurrence of incessant vomiting or purging, which, while it rapidly exhausts the patient, favours the escape of noxious impurities from the blood. The cessation of the discharges is sometimes quickly followed by symptoms of uræmia.

Nervous Dyspnoea.—A common and very distressing symptom in the advanced stages of the disease is a peculiar form of dyspnoea. I am not now referring to the persistent dyspnoea which results from the oedema of the lungs, from hydrothorax, or hydropericardium, but to dyspnoea coming on in paroxysms, and especially at night. In some cases the attack resembles asthma, and there are loud sibilant sounds, apparently the result of bronchial spasm; while in other cases the heart's action is rapid and feeble, and the breathing hurried and laborious, with loud puerile respiration over the lungs. There is evidently no want of moving air in the lungs, and the disturbed circulation and breathing appear to result from some morbid influence of the poisoned blood upon the nerv-

ous centres. This distressing form of dyspnoea, which recurs in paroxysms night after night, is, in fact, a form of uræmia.

Disease of the Liver.—In a large proportion of fatal cases of contracted kidney, the liver is found more or less diseased, sometimes enlarged and indurated or fatty, more commonly cirrhotic and contracted. Alcoholic excess may, and often does, excite at the same time cirrhosis of the liver and granular contraction of the kidney. With the cirrhotic liver there is often ascites. When ascites exists without anasarca, or remains after the removal of anasarca, and so forms the prominent dropsical symptom, serious disorganisation of the liver may always be suspected.

Diagnosis.—In addition to what I have said of the symptoms and progress of the disease, I have yet some hints to give you on the subject of diagnosis. The state of uræmic stupor or drowsiness, with a dry tongue and sordes on the teeth, may be mistaken for typhus or enteric fever. The difficulty of diagnosis is increased by the fact, that in some cases of typhus and enteric fever, when there is much cerebral oppression, the urine is often scanty and albuminous, and it sometimes contains granular casts. A close attention to the entire history of the case, and a careful examination of the urine, will seldom leave you in doubt. The specific fever-eruption, when present, is decisive. The thermometer will assist you. The temperature is higher in fever than in uncomplicated uræmic poisoning. Bear in mind that a patient with chronic renal disease may, in addition, have a specific fever—a complication which is usually fatal. With regard to indications afforded by the urine, remember this, that although during the progress of typhus or typhoid fever there may be an acute and transient disintegration of the renal gland-cells, as indicated by the appearance of granular casts—not easily to be distinguished from those which occur in cases of chronic desquamative disease—yet there is this difference, that whereas in the advanced stages of chronic desquamative disease the urine is pale and of low specific gravity, the albuminous urine of fever is usually of deep colour and rather high specific gravity. It is important to bear in mind that granular casts, with albumen, may appear temporarily in the urine as a result of other blood-poisons than those of typhus and enteric fever. I have seen them in cases of pneumonia, erysipelas, and pyæmia. Once, in a case of pyæmia, I found granular and large hyaline cysts exactly like those represented in Fig. 15, but the urine was of deep brown colour and of normal specific gravity; and after death, which resulted from pyæmic abscesses in various parts, the only disease found in the kidney was a recent result of pyæmia. You see, then, that, although the observation of the various forms of tube-casts is of great practical value as an aid to diagnosis and prognosis, yet a too exclusive reliance upon this microscopic evidence may mislead you. When, after a careful inquiry into the history of a case, a doubt exists as to renal disease being recent or of long standing, the evidence of hypertrophy of the left ventricle of the heart without valvular disease, but with a full and firm radial pulse, points to chronic disease in an advanced stage.

I have seen several cases of subacute renal disease occurring in men about middle age as a result of overwork and anxiety, in which it was difficult to decide between acute and chronic disease. I have preserved the urinary sediment from three of these cases; and although the first case occurred nearly fifteen years ago, the tube-casts are as well seen as when the specimen was recent. You may see these specimens under the microscopes on the table, and, having carefully inspected them, you may recognise their like when you meet with them in practice. One case was that of a solicitor aged 40, another a merchant aged 56, another a clergyman aged 45. The symptoms and the condition of the urine were alike in all. There were great prostration, vomiting, bleeding at the nose, and in one case from the gums, no dropsy, ultimately a typhoid condition, and unconsciousness shortly before death. The urine was blood-tinged, the specific gravity from 1009 to 1017, moderately albuminous. A rather copious sediment was composed of dark granular and large hyaline casts, with scattered blood-discs. Some of the granular casts had a blood tinge, and it is probable that they were in part composed of disintegrated blood. After death, in the only case examined, the kidneys were found somewhat enlarged, soft, and congested. Some tubes were injected with extravasated blood, and others, opaque, with desquamated and disintegrated epithelium. In cases of this kind, although the prognosis is very unfavourable—in fact, all the cases that I have seen have died—yet the disease is not so inevitably fatal as chronic desquamative disease in an advanced stage, and therefore it is important to distinguish between them.

Prognosis.—On the subject of prognosis, I have but little to add to what I have already said. Chronic desquamative disease, as a rule, tends gradually to a fatal termination. The rate of progress varies much in different cases and at different periods of the same case. You will remember what I said as to the evidence to be derived from the

amount as well as the character of the sediment in the urine. The most trustworthy prognostic indications are to be obtained by comparing the state of the urine with the general symptoms. When, with a condition of urine indicating advanced degeneration of the kidney, there is evidence of hypertrophy of the left ventricle, with an unspiring skin; when, with a diminishing secretion of urine, or even without a marked decrease, symptoms of uræmia begin to appear, the disease is generally not far from its fatal termination. You cannot be too cautious in giving a prognosis. The symptoms of chronic renal disease are sometimes much aggravated for a time by some imprudence in diet, by fatigue or anxiety, or exposure to cold. The patient may apparently be on the verge of uræmic coma, or he may have a fit of convulsions, yet, under appropriate treatment, these formidable results of his indiscretion or his misfortune, may pass away, and, in a few days, he may be apparently no worse than he was before the occurrence of this temporary disturbance. The uræmic symptoms which are not traceable to an obvious external exciting cause are, as a rule, more serious and intractable than those which result from influences capable to some extent of being removed or counteracted.

CHLOROFORM ACCIDENTS.*

BY AUGUSTIN PRICHARD, F.R.C.S.,

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I HAVE long wished and intended to lay before the Society some of my views on the subject of chloroform, and had already begun to write my paper, when Mr. Green introduced the subject at our meeting in October last. I have naturally, in the course of the last twenty years and more, had a good deal of experience of it; and I may state in the beginning, what you would discover as I proceeded, that my faith in it does not increase with years.

The late James Miller, the accomplished professor of surgery at Edinburgh, in the year 1848 introduced the subject of chloroform to his class with the utmost enthusiasm and eloquence; and I hope you will excuse my quoting some sentences of his lectures which treated the question of danger too lightly. While he helped to make the members of our profession blind to the risks of the agent, he introduced a most uncomfortable creed, the inconveniences of which meet us at every turn, and, by a public exhibition of one or two operations under its influence at the Edinburgh Infirmary, led the laity to think that it is a perfectly safe remedy.

On the 23rd December, 1846, Mr. Liston wrote to Mr. Miller on the new subject of anæsthesia. Mr. Miller says: "Its first sound had come from across the Atlantic; it fell on no dull or idle ears; it was taken up, tried, and speedily re-echoed, and in a few days it filled the island. Mr. Liston struck the key-note, and a pealing note it was." This of course had reference to the production of anæsthesia by ether. In October 1847, Dr. Simpson began his experiments with various other substances, and among them was chloroform. The following is Mr. Miller's narrative. Most of the experiments were performed after a long day's toil was over, and when the greater part of mankind was asleep. Each operator was provided with a tumbler, a finger-glass or saucer, into which the respirable substance was placed.

"Late one evening (November 4th, 1847), on returning home after a weary day's labour, Dr. Simpson, with his two friends and assistants, Dr. Keith and Dr. Matthews Duncan, sat down to their somewhat hazardous work in Dr. Simpson's dining-room. Having inhaled several substances, but without much effect, it occurred to Dr. Simpson to try a ponderous material, which had been set aside as of no likelihood whatever: that happened to be a small bottle of chloroform. It was searched for and recovered from beneath a heap of waste-paper; and, with each tumbler newly charged, the inhalers renewed their vocation. Immediately an unwonted hilarity seized the party; they became bright-eyed, very happy and very loquacious, expatiating on the delicious aroma of the new fluid. The conversation was of unusual intelligence, and quite charmed the listeners—some ladies of the family and a naval officer, brother-in-law of Dr. Simpson. But suddenly there was a talk of sounds being heard like those of a cotton-mill, louder and louder; a moment more, then all was quiet, and then a crash. On awaking, Dr. Simpson's first perception was mental. 'This is far stronger and better than ether,' said he to himself; his second, was to note that he was prostrate on the floor, and that among his friends about him there was both confusion and alarm. Hearing a noise, he turned round and

saw Dr. Duncan beneath a chair; his jaw dropped, his eyes staring, his head bent half under him, quite unconscious, and snoring in a most determined and alarming manner. More noise still, and much motion. Then his eyes overtook Dr. Keith's feet and legs, making valorous efforts to overturn the supper-table, or, more probably, to annihilate everything on it."

We cannot but admire the courage of the experimenters; and my belief is, that at that period of the evening, after supper, and in an upright position, they ran as much risk of their lives as our soldiers did in the middle of the battle of Inkermann.

I had the misfortune to witness one fatal case in my own practice, where a man aged 49, healthy except for a diseased elbow brought on by injury, walked into the operation-room, and lay down on the table and died, after taking one or two inhalations of a drachm of chloroform, which had been poured on the sponge two or three minutes at least before. The case was published in the *BRITISH MEDICAL JOURNAL* for March 13th, 1858. Death was clearly the result of sudden stoppage of the heart's action. Since that time there have been three deaths at our infirmary; one since our last meeting in October; and, I believe, besides, there have been two or three others in Bristol—a small number, perhaps, in proportion to the vast number of cases where chloroform has been administered. There have been, however, in my own practice, and, I presume, in that of others, a number of marvellous escapes and terrible alarms, where the patients have ultimately recovered, but where the furious struggle made by the medical men present for the preservation of life, and the excessive anxiety of the time, have been almost unbearable. Some present here have been at such scenes with me and have aided in the fight; and, were it possible to weigh these dangers and calculate the risks, and gauge and compute their amount, and add the whole in due ratio to the deaths, we should have more to lament than even the dreadful and fatal score of one death per week in Great Britain, admitted recently by our *JOURNAL*.

I have already in some of my published cases given accounts of persons in whom the slightest inhalation of chloroform caused collapse, and almost death, and where I have had to perform extirpation of the eye or removal of the breast, or some other serious operation, without the use of the anæsthetic. My late colleague, Mr. Bernard, had a case requiring amputation in a boy, where for a similar reason he was obliged to put off the operation, and afterwards amputate the thigh with the aid of brandy and opium, without the evidence of much suffering. We must also admit the uncomfortable fact, that the *post mortem* examination in most instances gives no clue to the fatal result. When chloroform was first introduced, I had a bottle of it, and, in speaking of its properties, for about two seconds held it to the nose of a member of my own family, who was so weak and faint afterwards as to give me some alarm. If I were asked what cases, according to my own experience, were most likely to cause anxiety under chloroform, I should say those who were affected with organic disease of the brain, lungs, or heart. With your permission, I will narrate one or two of these cases, with the preface that no words of mine, however vivid, could ever represent faithfully the anxious and painful strain which accompanies this wrestling with death for the victim claimed by chloroform; and first I will give you, in the words of my friend Dr. Shingleton Smith, the excellent house-surgeon of the Bristol Royal Infirmary, an account of the fatal case which has happened there since our last meeting.

CASE I.—R. W., aged 51, a labourer, was admitted on November 4th, with Pott's fracture, caused by a fall in the street while drunk. His wife stated that he had been drinking excessively for six weeks. Dupuytren's splint was applied to the leg, and removed the next day and a weight attached to the foot. On November 6th, he was restless and talkative in the morning, and had not slept much during the night. The leg had become swollen and again displaced, with the heel drawn up, in consequence of his having moved about. At nine in the evening, he was talking incessantly, with muscular tremor, and resisted any interference. One third of a grain of morphia was injected. At thirty-five minutes past nine, the weight was to be removed and the limb placed on the MacIntyre splint. One drachm of pure chloroform was poured on the sponge and held over his mouth. He struggled violently. After two minutes, another half drachm was poured on the sponge and applied. Struggling soon ceased; the breathing and temporal pulse were regular; no stertor. The sponge was handed to the dresser, but not applied to the face, and attention was given by the house-surgeon to the leg: the apparatus was removed, and the other splint placed under the leg. His breathing was then noticed to be very full and slow, not stertorous; and the pulse could not be felt, nor could the heart's impulse be detected. The pupils were dilated. He continued to breathe at long intervals, and deeply; several efficient respirations occurred, but no heart's action could be induced. The wet

* Read before the Bath and Bristol Branch.