my case, he also had been equally happy in thus dealing with an ulnar nerve.

Thus, gentlemen, aiming discursively at much and accomplishing only, I fear, too little, I have wandered over the field of more recent surgical advancement, and it only now remains for me to thank you for the kind and patient indulgence with which you have listened to the feeble effort I have made to acquit myself of the very onerous and responsible duty it has been the pleasure of your Committee of Council to impose upon me.

AN ADDRESS

DELIVERED AT THE OPENING OF

THE SECTION OF MEDICINE,

*At the Annual Meeting of the British Medical Association,
in Bath, August 1878.*

By T. GRAINGER STEWART, M.D., F.R.C.P.E., F.R.S.E.,
Professor of the Practice of Physic and of Clinical Medicine in the
University of Edinburgh; President of the Section.

ON CERTAIN MORBID STATES OF THE KIDNEY.

GENTLEMEN,—My first word in rising to address you must be one of thanks to the Council, for the great honour they have done me in appointing me to preside, on this occasion, over the Medicine Section of the British Medical Association. I feel that it is an honour of no ordinary kind, when I consider the long line of eminent physicians that have preceded me, culminating in the distinguished President of last year, Sir William Jenner. It is also a gratification to me to be associated in office with my friends Dr. Foster and Dr. Clifford Allbutt, men known and esteemed wherever clinical medicine is zealously cultivated.

We may, I think, congratulate ourselves on the prospects of our meeting. We cannot look over the list of our papers without a feeling of gratification that communications on so many important and interesting subjects are to be brought before us by respected members of the profession; and I hope that, following upon those communications, we shall have a frank interchange of opinion and comparison of experiences. I beg you to notice that it has been arranged that, at one of our meetings, a discussion on the diagnosis and treatment of intestinal obstruction shall take the place of the ordinary reading of papers; and I congratulate you on the fact that that discussion is to be opened by Mr. Jonathan Hutchinson and Dr. Long Fox. I may be permitted to bespeak your attention also to the paper by Professor Rutherford, in which he embodies the outcome of his admirable researches into the action of cholagogue remedies on the dog. We in Edinburgh are proud of that research, as a piece of genuine scientific work, and especially as a valuable contribution to the relief of human suffering.

Gentlemen, I have formed the opinion that, while the President of the Association and the readers of the addresses in their several departments may appropriately deal with great general questions, the President of a Section may, in his opening address, best fulfil his task by confining his remarks to some individual topic with which he is familiar. By adopting such a plan, he may give prominence to any lately discovered facts which he deems important; he may make known any changes in his opinions; he may attempt to remove misconceptions as to his statements previously published, and to vindicate the correctness of his views. In accordance with this conception of my duty, I venture to offer a few remarks upon some questions connected with diseases of the kidneys, which have lately attracted my attention. Even after thus limiting my range, I cannot pretend to consider all the topics which invite discussion. I dare not, for example, detain you during the time which would be required for the adequate consideration of the doctrine of arterio-capillary fibrosis, inviting though it may be.

The first subject to which I ask your attention is the disease known as glomerulo-nephritis, which was first described by Professor Klebs of Prague (*Handbuch der Pathologische Anatomie*, Band i, s. 645), as occurring in some cases of postscarlatinal dropsy. It is a form of interstitial nephritis, which affects only the interstitial tissue of the glomeruli. A normal Malpighian body consists, as you remember, of a group of vascular loops, surrounded by a capsule; between the capsule and the tuft, there are epithelial cells; between the loops, there is connective tissue. The affected kidneys present to the naked eye no abnormality beyond congestion of the tissues generally, and pallor and

prominence of the Malpighian bodies. Under the microscope, one sees little or no change in the stroma or in the tubules, but marked alteration in the glomeruli. They appear prominent and opaque; but the opacity is not due to accumulation of epithelium between the layers of the capsules, but to an extraordinary hyperplastic growth of angular nuclei among granular substance within the tuft. This change leads to compression of the vascular loops, and consequent anæmia. It may be readily understood how this might induce great diminution or actual suppression of urine, and how it should lead to uræmia, and, in many cases, to speedy death. Of the correctness of Klebs's general description, I have no doubt; for my friend Mr. David I. Hamilton, Pathologist to the Royal Infirmary and Demonstrator of Morbid Anatomy in the University of Edinburgh, has afforded me ample opportunity of studying the pathological characteristics of the disease. On looking back, I am satisfied that I have met with examples of this lesion. I recollect that the peculiar appearance of pallor and prominence of the Malpighian tufts amid the congested tissues attracted attention, and that I concluded that an increase of the epithelium of the capsule accounted for the appearance. The admirable methods of preparing specimens now in use—methods which have been brought to great perfection in the pathological department of the University of Edinburgh—render it clear that this impression was erroneous; that the change is really in the fibrous tissue, glomeruli, and capsules. The discovery of this special form of lesion does not interfere with our acknowledgment of other changes which have been long recognised as associated with post-scarlatinal dropsy, and with which glomerulo-nephritis may be found associated in greater or less proportion. Its clinical history is believed to correspond to severe and rapidly fatal forms of post-scarlatinal dropsy.

The second new condition to which I wish to draw attention is the remarkable lesion described at the April meeting of the Medico-Chirurgical Society of Edinburgh, by Dr. Angus Macdonald (*Edinburgh Medical Journal*, April 1878), as having been found by him and Mr. Hamilton in a case of fatal puerperal eclampsia. The kidney presented, to the naked eye, such an appearance as is ordinarily seen in the inflammatory affections of the tubes; but the microscope revealed a very different condition. The tubules did not appear opaque, nor the epithelium swollen and granular. These cells, on the contrary, were small, and their nuclei abnormally distinct. The capsule was normal. The most superficial convoluted tubes were wide; their epithelium was quite distinct. In many of them, the lumen was blocked with a peculiar hyaline or colloid material. This material was very abundant in certain districts. In the medulla, many of the tubules were completely occluded, and the observers were able to satisfy themselves that the material had flowed down the tubes, and, by occluding them, had led to secondary dilatation above. It appeared that, at many points, the epithelial periplast contained drops of this clear homogeneous substance, and the conjunction of numerous drops formed the mass. Mr. Hamilton tells me that he has met with this lesion in at least one other case of puerperal renal disease. By his kindness, I have had the opportunity of examining specimens, which satisfy me of the correctness of the description given. It is remarkable that, in Dr. Macdonald's case, the symptoms corresponded precisely with those met with in ordinary examples of puerperal eclampsia when associated with the usual inflammatory condition of the tubules.

The next particular to which I wish to refer is one which may appear of little importance—viz., as to the true nature of the cirrhotic process. I formerly was unable to satisfy myself of the inflammatory nature of this disease, but regarded it rather as a result of excessive growth of the renal connective tissue. My view was thus opposed to that of Dickinson and other eminent authorities; but an examination of microscopic specimens prepared by Mr. Hamilton has now satisfied me that the process, although so slow, must be regarded as inflammatory. The modern methods of staining render distinct the cell-proliferation, abundantly present where the disease is advancing; and this proliferation indicates the inflammatory character of the lesion. But, with this difference, there appears to be no reason for altering the opinions hitherto held as to the character, the progress, and the result of the cirrhotic process. This, as I have said, may not seem a very important change, but it is one which has a bearing, not only upon the individual disease, cirrhosis, but upon other varieties of Bright's diseases. I purpose, however, to retain the term cirrhotic kidney, because it is now well understood and in general use.

I now desire to draw your attention to the fact that a general consensus of opinion is beginning to exist as to the occurrence of three stages of transformation of the variety of Bright's disease, which has as its main feature inflammatory affection of the tubules. The fact that fatty degeneration follows upon inflammation of the tubes, acute or chronic, has of course been long universally recognised; but the further truth

that atrophy results if the patient lives long enough and is not cured, was at one time doubted by many authorities. In what exact way the atrophy comes about is, however, open to question; some observers holding that it is a result of an interstitial inflammatory or cirrhotic process, superadded to the inflammation of tubes, others thinking that it is entirely a consequence of changes within the tubules. Here, as in all the other renal atrophies, there is at least a relative increase of fibrous tissue, but there is not, as a rule, that adherence of the capsule, nor that extraordinary increase of the stroma which occurs in cirrhosis; and there is evidence that the process occurs in those regions of the kidney in which the tubular changes are most marked, and in which blocked tubules may be seen in all stages of destruction. In illustration, however, of the atrophy, I may refer to one of Dr. Dickinson's cases, in which the patient lived for eleven years after an attack of post scarlatinal dropsy, and after death the kidneys were found to be much atrophied. As another illustration, I may cite a case briefly described in the *St. Bartholomew's Hospital Reports*, by Dr. Moore, in which, within three years of the commencement of the symptoms, atrophy had occurred.

With regard to the stages of the waxy kidney, a similar consensus of opinion can scarcely be said to exist. German observers have not yet been satisfied with the accuracy of the description which has been given in this country. Dr. Litten (*Berliner Klinische Wochenschrift*, June 3, 1878) explicitly objects to this description, and recognises one form of waxy kidney in which that lesion has become superadded to other affections; another in which the waxy change is the primary, and other lesions are superadded to it. Professor Bartels (whose untimely death the profession has such cause to regret) again says (*Diseases of the Kidney in Von Ziemssen's Cyclopædia*, page 518), "I do not indeed doubt that, under the influence of amyloid degeneration alone, an anæmic necrosis and destruction of the epithelium within the tubuli may take place, and in this way a considerable reduction may be effected in the size of the previously swollen organ; but, from all that I have hitherto seen myself, I must declare my adherence to the opinion entertained by Virchow and Klebs, that the cases thus described by Grainger Stewart were not simple cases of amyloid kidney, but were cases of amyloid degeneration, with simultaneous, or more likely with a previously existing contracting disease (the genuine or secondary contracting process). I, at least, have never seen a kidney that was smaller than normal, which was the seat of amyloid degeneration alone, and did not present at the same time distinct evidences of connective tissue hyperplasia."

On the other hand, it is satisfactory to notice that Dr. Dickinson indicates particularly in his last edition that he recognises something analogous to the three stages, for he says (*Diseases of the Kidneys, etc.*, page 432): "There are three steps in the dismemberment of the kidney by this disease—first, an alteration in the walls of the blood-vessels; second, an effusion through them into the tissues and cavities of the gland; third, consequent changes, both in the interstitial tissue and the tubes;" and Mr. Hamilton informs me that he has been able to trace the three conditions referred to in different examples of the disease, although he inclines to explain the series of changes in a way different from that which I suggested. To me it seems that the clinical evidence is strongly opposed to the explanation which Virchow, Klebs, and Bartels suggest regarding the extremely atrophic condition. Cases fitted to throw light upon such a point do not occur frequently in practice, but I may refer to two which afford strong evidence. In a very typical case, which was under my observation for more than nine years, the organs were found after death to weigh two and a half ounces each, the tubes were wasted, the Malpighian bodies were large and waxy, and closely grouped together, the fibrous tissue was certainly relatively more abundant than in the healthy organ, but the capsules were at all points readily separable, and the organs did not present the appearances characteristic of cirrhosis. During the years in which I watched the case, the bulk of the liver underwent a diminution corresponding to that which I believe to have taken place in the kidney, but the liver showed no increase of fibrous tissue in its substance. Moreover, the clinical history of the case seems incompatible with either of the suggestions thrown out by the German authorities. If, on the one hand, we assume that cirrhosis became superadded to the waxy degeneration, it is singular that absolutely no change of symptoms was produced, but that the case went on, scarcely varying in its features till within a week or two of death. On the other hand, if we assume that the cirrhosis existed from the first with the waxy degeneration, the symptoms during all the years would be difficult to explain, nor could we have expected the course of the disease to be so prolonged as it was. Take again another instance. In a patient who was under observation for about four years, the organs were found somewhat contracted, the vessels markedly waxy, many of the tubules

blocked with hyaline material. Such cases as these may be appealed to as evidence of the third stage—that of ultimate atrophy. In many cases, again, in which the symptoms have lasted a shorter time, the organ is found enlarged and pale, with smooth surface, and many of its tubules blocked with hyaline material—a material quite different from that which is seen in tubular inflammations. It is this condition which seems to warrant the description of the term, the second stage. In other cases, where the symptoms were only of recent origin, I have found no change beyond the vessels, the patient having died of some other disease before a further stage was reached. Cases of the kind seem to vindicate the first stage.

As to the diagnosis of the waxy disease, opinions are still discrepant; indeed, the most recent German writers on the subject seem to imply that in many cases it is impossible to establish a diagnosis. Dr. Litten (*loc. cit.*), for example, after describing four most remarkable cases of waxy degeneration without albuminuria, says that, among the complex group of symptoms associated with Bright's disease, none are more variable than those of the waxy degeneration, and that no lesion varies more than it in respect of the secretion, its quality, its chemical composition, the nature of the sediments deposited, and the presence and distribution of the dropsy. Such statements, gentlemen, by no means accord with my experience. How are we to explain this discrepancy?

The explanation seems to be that many physicians fail to distinguish cases of purely waxy degeneration from those in which that lesion is associated with inflammatory disease of the tubules or with other maladies. When the disease is simple and uncomplicated, I have invariably found the symptoms distinct and uniform, an absence complete, or almost complete, of dropsy throughout the whole course, the presence of polyuria from the beginning to the end of the case, the very rare absence of albumen, the scantiness of tube-casts, the history of wasting disease, and the absence, or comparative absence, of cardiac changes.

Now, with regard to dropsy, it is true that it is often met with in people suffering from waxy disease. I have seen it in cases in which probably the waxy degeneration became superadded to a chronic inflammatory affection of the tubules. I have frequently seen it persistent and very intractable in cases in which inflammatory disease had become superadded to the waxy degeneration; indeed, I have in my hospital practice been in the habit of drawing the attention of the students to this peculiarity—that, when in cases of chronic inflammatory disease under the influence of diuretics, the quantity of urine has risen to the normal standard, or even higher, and yet the dropsy persists, there is strong reason to suspect the previous existence of waxy degeneration. I have, by inquiry in such cases, elicited the fact that, before dropsy had ever appeared, there had been polyuria, and repeated nocturnal calls to micturition, and have found on examination after death the old chronic waxy underlying the inflammatory disease. I have seen a patient much reduced with exhausting disease associated with simple waxy degeneration become dropsical during the last days or weeks of life; but that was due either to extreme anæmia and debility, corresponding to what we see in so many cases of phthisis unattended by renal change, or to secondary and inflammatory changes in the renal tubules. In some cases, again, I have seen marked ascites associated with waxy degeneration, and no other or little other manifestation of dropsy, but that ascites was not due to renal disease, but to a waxy or a syphilitic affection of the liver, or, perhaps, partly to hepatic disease, partly to slight affections of the renal tubules. I have seen these cases go on to a fatal result (see *Medical Times and Gazette*), but also sometimes have witnessed great improvement, and have even seen the symptoms, both renal and hepatic, entirely disappear under persistent constitutional treatment and repeated tapings. We have thus certainly associated with waxy degeneration varieties and degrees of dropsy, but the dropsy is not due to the degeneration.

As to the quantity of urine, I have found it distinctly increased in the vast majority of true cases of waxy kidney. In one patient, whom I watched for nearly ten years, the urine continued copious throughout. When he first came under observation in February 1860, his urine varied from 90 to 102 oz. per day; in 1861, it averaged about 120 oz.; in 1864, it was usually above that amount; in 1868, it was about 100 oz.; and in 1869, when he came into the infirmary to die, it was from 110 to 120 oz.; and within a few days of his death it was still markedly above the normal, 110, 102, and 70 oz. It is quite true that polyuria may be absent. If the degeneration of vessels becomes superadded to pre-existing inflammatory disease of the tubules, the polyuria can never become marked. If the inflammation become superadded to the degeneration, the polyuria must necessarily disappear. I have often known polyuria prevented by the existence of profuse diarrhoea, which is so common a symptom of waxy degeneration of the intestine; and sometimes its absence is accounted for by a combination of circum-

stances, as is illustrated by one of Dr. Litten's cases. A boy, aged 14, was admitted to hospital suffering from phthisis, enlarged liver and spleen, and some diarrhoea; he had no dropsy and no albuminuria; and he passed only 35 or 36 oz. of clear transparent urine daily, of specific gravity 1011 to 1015. With symptoms of hectic fever and intercurrent pleurisy, he gradually became weaker, the quantity of urine diminished, and became darker in colour. *Post mortem* examination revealed, in addition to phthisis, pleurisy, and general miliary tuberculosis, waxy degeneration of the kidneys. With this group of symptoms, even in the absence of polyuria, one might have suspected the existence of renal degeneration, on account of the colour or specific gravity of the urine, taken along with the causal and concomitant complications. The absence of polyuria was explained by the diarrhoea and the fever, perhaps in some measure also by the renal tuberculosis. I am not positive that the last-named affection prevents the development of polyuria, but I have seen instances in which a syphilitic affection of the organ appeared to have the effect. The absence of albumen, in regard to which Dr. Litten's paper is so valuable, occurs sometimes, as he has shown, throughout the whole course of the case, and sometimes is not developed for a short period after the polyuria has appeared. The quantity of albumen varies greatly. As to urinary sediments in the cases of uncomplicated waxy kidney, there are scarcely any; tube-casts are by no means numerous. There sometimes occurs a deposit which resembles pus, but probably consists of wandered white corpuscles, which have made their way through the walls of the vessels, which are degenerated, and perhaps more permeable; red blood-corpuscles appear but very rarely. Whenever a deposit is found containing numerous epithelial, granular, and fatty casts, we may certainly conclude that we have no longer to do with an uncomplicated case of waxy disease, but that inflammation also exists. Attention to such considerations has often guided me in obscure and complicated cases. The importance of polyuria as an aid to diagnosis is well illustrated by a recent article in Virchow's *Archiv* (Band lxxxv) by Dr. Fürbringer of Heidelberg, who describes four cases which occurred in the practice of Professor Friedreich. In each case, waxy kidney was diagnosed, but in only one of the four did the necropsy justify the diagnosis. In all of them, there was albuminuria associated with enlargement of the liver and spleen, and phthisis or other exhausting disease; but, in three of them, the quantity of urine was not increased, and the specific gravity was never below the normal. With these conditions of the urine, the principles of diagnosis in which I have most confidence would have forbidden the expectation of waxy degeneration; whilst they would have led to the diagnosis in the remaining case, seeing that the urine, which had at first been scanty and dense, became, at the very end of life, more copious and of lower specific gravity.

I must apologise for the length to which these remarks have extended, and for the departure from the ordinary form of the sectional address. I trust that the importance of the subject which I have ventured to bring before you may be held not to have trespassed unduly upon your time.

AN ADDRESS

DELIVERED AT THE OPENING OF

THE SECTION OF SURGERY,

At the Annual Meeting of the British Medical Association,
in Bath, August 1878.

By GEORGE W. CALENDER, F.R.S.,

Surgeon to St. Bartholomew's Hospital; President of the Section; etc.

THE AVOIDANCE OF PAIN.

I KNOW of no alloy to the satisfaction a surgeon must feel in being allowed by your favour to preside over this section. His first words should give expression to his thanks for the honour which has been conferred upon him. Such words are mine.

Beyond this, however, it is his privilege to speak to you on the subject which interests us so deeply—the science and the art of surgery. In the age to which this Association has flourished, your President has an advantage over his earlier predecessors in that he can look back upon the work which has been done in connection with this section, and can reckon up many important advances in our knowledge of surgery which have been first made public under the auspices of the Association.

I prefer, for myself, to look forward and to endeavour, with your permission, to try to interest you in a subject for which you must for-

give me time. I seem to be in any way too earnest in my pleading; for I do feel respecting it most anxious that it should commend itself to your favourable consideration. From the wideness of its relations, it is, I think, a fitting one to be spoken to by your President, who should, I conceive, always endeavour to put before you matters of broad interest and of general professional concern.

Whilst engaged in the study of the treatment of wounds, one could not but be attracted to many side considerations. To one of these it is that I venture to ask your attention, and in doing so I preface my remarks by this statement.

An operation or an accident-wound can be so treated that the patient from first to last shall be free from pain; and, in the treatment of many other troubles, a great deal of pain may be avoided by forethought and care on the part of the surgeon.

Next to insuring the life-safety of our patients (and I think we may assert that the mortality after injuries and after operations is not a tithe of what it was, even in comparatively recent years), surely it is something to endeavour to attain to, that we may promise a patient, who can under an anæsthetic pass without suffering through an operation, that he may be sure of convalescing without an ache or a pain from the handling and dressing of his wound, and to feel confident that our promise will be borne out.

Now, what are the causes of pain? Why do patients make the varied complaints which we group together as those of pain? How various are those complaints! I often ask the sick to describe to me the sort of pain from which they are suffering. If we except certain odd and only occasional replies, I find that they adhere to the descriptions which from childhood they have been taught to associate with pain. There is tenderness to the touch, there is the sense of throbbing and of shooting or of darting, and there are dull aching pains, or there are pains likened to some chance experience of the past, a revival of some old association—the pain is like that which attended a cut, or a burn, or the rubbing of a blistered surface.

But it is not pain alone, that is, in the ordinary acceptation of the word, with which we have to deal, and it is not the patient's description of his kind of pain which is of importance. It is the cause of his discomfort or of his pain to which attention must be directed, if we are to learn how to prevent them by obviating the conditions out of which such troubles arise.

I say it is not pain alone. There are various sensations, not strictly of a painful nature, and yet causing restlessness, and hence causing pain. Such are the impressions after an amputation, which make the patient fancy that the hand or the foot cut off still remains part of his body, or still needs to be steadied by muscular action. As he dwells upon the sensation, or as the muscles assume their accustomed vigilance, the muscles which lie in the wound, and with which foot or hand movements are usually made, are apt to fidget and grow irritable; then they cramp or jerk and prevent rest, and so interfere with the process of repair. But this local condition begets general restlessness, and this is added to the muscular trouble, which later may induce what is termed jumping of the parts concerned in the wound, especially when the patient is falling off to sleep, so violent may be the muscular contractions and loosening.

This trouble arises from education, and from oversight in the treatment of a wound.

From education, if, for some time prior to the amputation, the muscles have been trained to keep a joint, such as the knee or ankle, in a state of rest. During such training, the muscles relax from the state of contraction when sleep begins, and then follows that jerking of the limb, causing pain in the jarred joint, and so arousing from sleep, which is so well known to surgeons as one of the distressing symptoms attending joint-diseases. It may be obviated by the use of weights so applied as to steady, under slight extension, the affected part, irrespective of muscular action. If it have not been so obviated, the training to which the muscles have been subjected will live in the stump, and the result will be that the muscular relaxation at the oncoming of sleep will shake the wound and cause severe pain, unless, indeed, the stump have been so supported as to rest easily and equally in its bearings, and free to swing with the chance movements of the body.

In cases in which this trouble, the starting of a limb, has been strongly marked before an operation, as it often is, more especially with disease of the knee-joint, I recommend that the nerves exposed on the face of the stump should be stretched so as to numb and for a time to quiet the muscles supplied by them or by nerves in association with them through a common plexus of origin; and if, in addition, the stump be properly secured and swung, the pain and unrest which might otherwise have followed the operation from the starting of muscles will be effectually prevented.