Address delivered at the opening of the section of medicine at the annual meeting of the British Medical Association in Bath, August 1878 ; On tendon reflex: a clinical lecture delivered in the Royal Infirmary, January 1878 ; On a case of epileptiform neuralgia treated by nerve-stretching / by T. Grainger Stewart.

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Dr Angus Fraser
with the author's kind regard

ADDRESS delivered at the **Opening** of the **Section** of **Medicine** at the **Annual Meeting** of the **British Medical Association** in **Bath, August** 1878.

On **TENDON REFLEX. A Clinical Lecture** delivered in the **Royal Infirmary, January** 1878.

On a **Case** of **EPILEPTIFORM NEURALGIA** treated by **Nerve-Stretching.**

By T. GRAINGER STEWART, M.D.,

Professor of the Practice of Physic in the University of Edinburgh.

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., President of the Section.*

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 this 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 Pathologischer Anatomie*, Band i, s. 645) as occurring in some cases of post-scarlatinal 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' general description I have no doubt; for my friend Mr David J. 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 in former years 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 those of 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 dis-

eases. 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 had become superadded to other affections; another in which the waxy change is the primary, and other lesions are supperadded to it. Professor Bartels (whose untimely death the profession has such cause to regret), again, says (Diseases of the Kidney in Von Ziemssen's *Cyclopedia*, 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, &c.*, page 432)—"There are three steps in the disablement 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 supperadded 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 fur-

ther stage was reached. Cases of this 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 had 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*, *p.* 18), 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 tappings. 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 the 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 become 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 diarrhœa, which is so common a symptom of waxy degeneration of the intestine; and sometimes its absence is accounted for by a combination of circumstances, as is illustrated by one of Dr Litten's cases. A boy aged 17 was admitted to hospital suffering from phthisis, enlarged liver and spleen, and some diarrhœa; 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 diarrhœa 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 lxxxi.) 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, considering the importance of the subject which I have ventured to bring before you, I may be held not to have trespassed unduly upon your time.

On TENDON REFLEX. A Clinical Lecture delivered in the Royal Infirmary, January 1878, *by T. Grainger Stewart, M.D., Professor of the Practice of Physic in the University of Edinburgh.*

GENTLEMEN,—I purpose to bring under your notice to-day a series of cases illustrative of a phenomenon of much interest, and probably of very considerable clinical importance. The phenomenon is one which has been long known to schoolboys, but whose physiological and pathological import was first brought under the notice of the profession only three years ago. It was simultaneously described by two independent observers—Professor Erb of Heidelberg, (*a*) and Professor Westphal of Berlin. (*b*) The former applied to it the name "tendon-reflex," which, as the shorter and more satisfactory of the two names employed, we shall adopt; the latter described separately different manifestations of it under the names "foot phenomenon" and "knee phenomenon." It is best studied in the knee, and most of you will at once understand what it is when I recall to you a favourite trick of boys—viz., sharply tapping on the ligamentum patellæ, and thereby producing a jerking forwards of the foot. This jerk is best seen when the leg is bent at about right angle, and the foot unsupported, as when one is sitting with one knee crossed over the other, or upon a high seat with the feet dangling. It is a result of sudden contraction of the quadriceps extensor femoris, manifestly produced by reflex action. Brief observation of the muscle and of the movement will convince you that it is not a mere mechanical result of a momentary indentation of the tendon, for the jerk follows the tap after an appreciable interval, and is always associated with contraction of the muscle. It is best produced by sharply tapping with the finger or with

(*a*) "Archiv für Psychiatrie und Nervenkrankheiten." Band v. 1875.
(*b*) *Eod. doc.*

a percussion hammer. All parts of the tendon are not equally sensitive, and it is necessary sometimes to try different parts before the result is obtained. It is invariably present in healthy people, but its intensity varies in different individuals. I shall show you, first, a patient in whom it exists to an ordinary extent, and, second, one in whom it is very slighly manifested.

You see in this first patient that when I place him on the table with his feet dangling, and strike the ligamentum patellæ near its upper part, his foot jerks forward several inches, and then instantly falls back. You see that the movement is as readily produced in the one leg as in the other, and that the two legs are equally susceptible. This I present to you as a normal standard. In contrast with it, notice the phenomenon as exhibited by this soldier. His long legs dangle from the table, and you perceive that the stroke must be sharp and well planted in order to produce the movement, and that the movement at the best is slight. This deficiency may be due to the inordinate development of the inhibitory functions in the course of his military training, or, more probably, to innate nervous stolidity.

Having now witnessed the phenomenon, you are ready to consider the mechanism of its production. Admitting that the jerk is of reflex origin, the irritation might be propagated from the nerves of the skin, from the joint, or from the tendon. That it is not from the skin seems proved by the fact that only firm percussion induces it; no mere touching of the surface suffices, but this may be better demonstrated by the production of the phenomenon after the skin has been anæsthetised. That it is not from the articular structures is certain, because we may tap all round the joint without producing any movement excepting when we touch the sensitive part of the quadriceps tendon. By process of exclusion, then, as well as by direct observation, we must conclude that the tendon is the starting-point of the irritation; and as that structure is supplied with nerve-filaments, we can be at no loss to understand how the movements are brought about.

The first alteration of this function which I shall demonstrate to you is its absence in cases of locomotor ataxy. The patient whom I now introduce has that disease in a marked degree, and has suffered from it for years. He has, as you observe, spinal myosis, which indicates affection of the ciliospinal region, and you perceive that when standing he plants his feet apart, so as to widen his base; that he walks with a peculiar ataxic gait; turns round awkwardly and with difficulty; and when his eyes are closed, sways about unsteadily and threatens to fall. When I place this man upon a table with his feet hanging over the edge, and tap on his ligamentum patellæ, as I did in the other cases, you perceive that there is no response. The legs remain absolutely quiescent as before; not the slightest quiver appears in the muscle, and this notwithstanding the fact that when I tickle his soles, the ordinary reflex movements are at once produced.

The next patient is also the subject of locomotor ataxia, probably associated with multiple sclerosis in the cord and base of brain. In him also you notice the characteristics of locomotor ataxia, and in him you perceive that tapping the tendon is followed by no result. I have not thought it desirable to bring into the theatre that very advanced case of locomotor ataxia of years' duration, in which you have at the bedside studied many of the features of the disease, but I have satisfied myself that in him also there is complete abrogation of the "tendon reflex."

All the cases that I have had an opportunity of examining corroborate the statement made by Westphal in his original paper, and by Erb and Berger, that in the fully-developed grey degeneration of the posterior columns of the cord the "tendon-reflex" is lost.

But in a lecture published in Berlin(*c*) in the beginning of the present month (January), Westphal has raised the important question whether the "tendon-reflex" does not disappear even before the development of the acknowledged

(*c*) "Ueber ein frühes Symptom der Tabes Dorsalis" (*Berliner Klinische Wochenschrift*, January 7).

symptoms of locomotor ataxia; and whether, therefore, its absence or presence may enable us to pronounce positively as to the nature of some spinal cases otherwise obscure. In connexion with neuralgic pains in the limbs, white atrophy of the optic nerves, and such-like conditions frequently but not exclusively associated with locomotor ataxia, he has found the presence or absence of the "tendon reflex" afford valuable indications as to the nature of particular cases. I have not yet had any occasion to test this in my own practice, but I can recall cases in which it might have proved very helpful. I shall presently show you two patients whose symptoms in some degree resemble those of locomotor ataxia, and we shall see what light the "tendon-reflex" throws upon them. Before bringing in the first of them, I shall tell you a few facts as to his history. His case has been dwelt on repeatedly at the bedside, and has been selected as the subject of clinical studies (*d*) by several of the senior students. He is a joiner, twenty-three years of age, and he married before he was twenty-two. Five months after marriage he began to feel weakness in the right leg—numbness, a tendency to jerking of the muscles when at rest, and unsteadiness of gait, particularly in the dark. These symptoms gradually increased, and the left leg became affected. He never had any darting pains in the limbs, nor girdle pains, nor abnormality of micturition, nor squinting, ptosis, or other temporary paralysis; and thus in respect of history there was nothing excepting the staggering gait aggravated by darkness which could suggest locomotor ataxia. I now bring him under your notice, and you observe that he makes no complaint of abnormal sensations, for the headache which once troubled him has disappeared, and the burning feeling and numbness in the legs are not distressing. You perceive that he feels tickling of the soles, and that his sensibility to pain is unaffected, but that in both legs his

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(*d*) Clinical studies are short essays upon obscure or otherwise specially interesting cases, which the senior students attending in the wards are invited to write and submit to the Professor for inspection and criticism.

sense of touch is impaired, and he fails to distinguish be tween a hot and a cold application. The muscular sense appears also diminished. The sight is rather dim; and while, as you see, there is no myosis, Dr Argyll Robertson tells us that he finds distinct commencing atrophy of the right optic nerve. The sensory functions are otherwise natural. Reflex movements are, you see, readily produced by tickling the soles. There is a certain feebleness in the muscles of the legs, and as he walks his movements are abrupt and ill-regulated. He turns round in an awkward way; when he stands he plants his feet somewhat apart, and is even then unsteady, but as soon as his eyes are closed he begins to totter. The muscles of his legs are well nourished, but he suffers much from spasmodic cramp-like contractions when he is lying in bed or sitting. When I place him on the table and test the "tendon-reflex," you observe that the movements are exaggerated, that a slight tap produces a very marked jerk, and that that is followed by a sudden drawing backwards as if by contraction of the flexors.

In this case a diagnosis of locomotor ataxia is suggested by the mode of walking, the unsteadiness in the dark, and the white atrophy of the optic nerve. But certainly it is not established, for such a gait as this occurs in other spinal diseases; the unsteadiness in the dark is also not distinctive; and the white atrophy occurs in other spinal maladies as well as in cerebral affections and independently. These considerations satisfied me that we were not entitled to diagnose locomotor ataxia, and the soundness of this opinion is attested by the application of Westphal's principle. I am inclined to think that here we have an irritative affection of the cord, not confined to the posterior columns, and manifesting itself by interference with the sensory and motor functions, and markedly by the jerking of the muscles and the exaggerated reflex irritability.

I shall now bring in another patient whose case closely resembles that just described. She is a healthy-looking young women of twenty, a milliner, and has suffered from some degree of weakness of the limbs for two years. She

has at times a good deal of pain in the legs, and the sensibility is diminished. As she walks, you notice that her gait is peculiar and her mode of turning vaccillating. She stands unsteadily, and when she closes her eyes the unsteadiness is increased. Ordinary reflex movements are readily produced by tickling the soles, and I have in the ward tested the "tendon-reflex" and found it exaggerated in the same way as in the last patient. I incline to think that in this case also there is an abnormal irritability of the cord.

The last case that I have to bring before you to-day is that of a little boy who is suffering from hemiplegia. This hemiplegia has existed for about a year, and is attended by some degree of aphasia. In the left leg you perceive that the "tendon-reflex" is normal; in the right it is exaggerated, but in addition it is followed by a period of tonic contraction of the muscle, so that by repeating the tapping at short intervals we can keep the leg almost extended—a result which we cannot by any effort produce in the left leg. This is, moreover, associated with a condition which was pointed out to me by the House-Physician, Dr Strang—a distinct resistance to sudden passive movement. Thus you observe that when I rapidly flex and extend the left arm or leg of this patient these movements are not resisted, but when I make corresponding movements of the right arm or leg there is a certain amount of resistance, due to instantaneous contraction of the opposing muscle. Moreover, it appears that just as the boy is awaking from sleep his arm and leg are seen to move about more freely than they do when he makes a voluntary effort when fully awake.

It is obvious that the "tendon-reflex" function as manifested here is very different from what we have seen in any of the cases hitherto studied. In the first two abnormal cases it was completely lost; in the second two it was exaggerated; in this case it is also exaggerated, but it is followed by a degree of tonic contraction. There is no evidence of such irritation of the cord as existed in the second two cases, and I should think it not improbable that (as was suggested by one of my colleagues to whom I was showing these phe-

nomena the other day) it may be best explained by a diminution or abrogation of the inhibitory influence of the brain. This hypothesis has the advantage that it would well accord with the obvious explanation of the comparative vigour of the automatic movements on awakening, viz., that the lesion inducing the hemiplegia interferes with conduction between the voluntary and automatic centres.

You will then, gentlemen, remark that the "tendon-reflex" is quite independent of and distinct from the ordinary reflex movements produced by tickling the sole, for you have seen in one of our cases that "tendon-reflex" is entirely abolished, while "skin-reflex" is normal. Also that it is reasonable to conclude that the ordinary action of muscles is in some way promoted by sensory impressions derived from the tendons. I mean, that in all probability the motor function of muscles is subserved by impressions derived from sensory structures situated in the tendons, as well as by those derived from the sensory structures in the muscles and the skin. I must confess that this interesting physiological consideration had not occurred to me before I read the papers of Westphal and Erb.

But you must not fall into the error of supposing that the awkward movements of ataxic patients are due to those changes in the muscular mechanism which produce the loss of "tendon-reflex," for, on the other hand, we have it on the authority of Westphal that he has seen the "tendon-reflex' absent in cases where there was no ataxia, and to-day you have seen that a gait like the ataxic may be present when the "tendon-reflex" is healthy or exaggerated.

On a Case of EPILEPTIFORM NEURALGIA treated by Nerve-Stretching, by T. Grainger Stewart, M.D., Professor of the Practice of Physic in the University of Edinburgh.

[Read before the Border Counties Branch of the British Medical Association, May 22, 1879.]

On returning from my autumn holiday last year I found among the patients under treatment in my department in the Royal Infirmary one who had been sent up from the Surgical Wards suffering from Epileptiform Neuralgia.

He was a man of 70 years, and was employed as a station-master on one of the railways of Cumberland. There was no evidence of hereditary predisposition to nervous disease; he was a temperate man; and his surroundings had been for the most part favourable. In his railway work he had naturally been somewhat exposed to the weather, and a good deal to draughts, but never in any extraordinary degree. He had been perfectly healthy till the year 1862, when he was seized with facial neuralgia. At first the pain was of a burning character, and it gradually increased in severity—the paroxysms becoming as time went on more frequent and intense, until at last his life was almost intolerable to him. Indeed, had it not been for the remissions during which the pain was easier, and the periods of immunity during which he was entirely well, it would have been so. These periods of immunity varied in length—sometimes six weeks, sometimes three months, and on one occasion a whole year; but sooner or later the attacks returned, and for six or eight weeks he had little freedom from agony, and never a moment's feeling of security. The attack from which he was suffering at the time of his admission to my ward had lasted from the end of April, and showed no signs of abate-

ment up to the time I saw him. He was a short man, rather thin, but not emaciated—said he had lost during the past year about a couple of stone weight. Still there was nothing wrong with him excepting the neuralgia, and often that was not severe.

When a paroxysm occurred his face would suddenly change, twitching of its muscles on the right side would set in, leading to the strangest grimaces. The agony had begun simultaneously with the movement, and in the lines of distribution of the middle branch of the fifth nerve on the right side was most intense. The patient would seize his head with his hands and press the painful part with the utmost violence, would drive his knuckles into the space beneath the malar bones, would slap his face, tear his hair, twist his body in all directions, and sometimes lose all self control and shout out in his agony. This would continue for a few seconds, or perhaps a minute or two, then the pain and other symptoms would subside. The paroxysm might recur almost immediately, or not for hours; generally they were most severe in the evening and during the night. They were induced easily by touching the skin or pulling the hairs of any part of the area of distribution of the affected nerve, or by touching the gums or tongue. Mastication had thus become impossible, and all food had to be taken in the liquid form, and no effort was spared, by the use of tubes or other contrivance, to smuggle it past the sensitive region. Nine of the teeth had been extracted in the hope of obtaining relief, but without benefit.

It was clear that the case afforded a typical example of the malady which Trousseau has described as Epileptiform Neuralgia. No doubt many of you are familiar with that classical description, and will remember that it includes two varieties—the more common one, in which there is pain without spasm; the more rare, in which pain and spasm co-exist. A few years of practice had sufficed to satisfy Trousseau that the disease was quite different from ordinary Neuralgia in the face, and one of the features was its utter incurability. This feature, along with its suddenness of ap-

pearance and disappearance, led him to associate it with Epilepsy, and to employ the name now in general use.

He sketches several cases with his wonted vividness, of which the following may serve as an example:—

"This poor patient had for many years been subject to the convulsive form of Neuralgia. His paroxysm lasted sometimes a few seconds only, and sometimes a minute; they recurred whenever he spoke, drank, or ate, or whenever one touched with the tip of a finger the few teeth which he had left. The pain was seated in all the branches of the trifacial nerve of one side, but chiefly in the infra-orbital division. Several of the nerve trunks had been divided already, but the relief had only been temporary, and the pain had always obstinately returned after an interval of from a few weeks to a few months. The extraction of his last remaining teeth gave him no relief. Prolonged applications of a solution of cyanide of potassium did some good; but the pain still returning as awful and as unbearable as ever, I decided upon dividing the infra-orbital branch. Bonnet performed the operation with great skill; the patient was relieved instantly, and remained free from pain for several months. The following year I saw him again, suffering in the same way in the course of another nerve of the face, and with the same convulsions. Professor Roux, as far as I can remember, again divided several nerves. Lastly, in 1841, Dr Piedagnel saw in his wards at La Pitie this same individual, whom he had known thirty years previously when House-Physician at the St Antoine Hospital. The poor man's face was scarred from the surgical operations which he had undergone, for whenever the pain became intolerable he implored the help of the knife, for this at least gave him relief for a few days, and sometimes a few months."

Our poor patient had, like Trousseau's, submitted to many plans of treatment, but with a like want of relief. He had had many teeth extracted, as we have seen; had opium by the mouth, and morphia subcutaneously; had croton-chloral, and other sedatives, quinine and iron, all without result, and after a further trial of many of these the question

arose whether we should dismiss him as incurable or try yet other remedies. Experience seemed to show that Trousseau's gloomy prognosis was better warranted by facts than the brighter one maintained by Dr Austie, and we certainly could not look with hopefulness to any of the ordinary methods. Section of the nerve or neurotomy might have been tried, but the advantage obtained in former cases had been merely temporary, the pain reappearing as soon as the nerve had healed, or even sooner, or at the best not being long deferred. Excision of a piece of the nerve or neurectomy had not produced results conspicuously better. Neither of these methods therefore commended themselves for adoption here, but it seemed to me possible that by introducing Nussbaum's plan of nerve stretching, which had proved so markedly useful in some similar conditions, might be tried with advantage. My first personal experience of that plan of treatment was in 1876, when a patient was admitted to my wards complaining of various nervous symptoms, and above all of very agonising pain in the line of the sciatic nerve. As none of the sedatives usually helpful afforded any relief, I thought that Nussbaum's plan might be tried; and, after consultation with Professor Lister, it was arranged that he should operate. He did so with the usual antiseptic precautions, and the operation was followed by extraordinary relief. Since that time it has taken its place in Edinburgh, and has been successfully performed by Mr Chiene and several others of our surgeons.

In the absence of Professor Annandale, Dr Bishop, who was in charge of the clinical surgical wards, proceeded on the 22d October to operate. With the usual antiseptic precautions, he cut down upon the infra-orbital nerve at its point of emergence from the bone, and having isolated the nerve, stretched it as vigorously as its size seemed to warrant. In the course of that day there were several severe attacks, and for sometime the pain occasionally recurred, but it speedily abated, and for a month thereafter there was almost complete immunity. At the end of that time paroxysms recurred, and on November 28th another attempt

was made to stretch the nerve. In consequence of the matting of the tissues in the cicatrix, the nerve was cut through, and the parts became anæsthetic for the time. Still the pain continued, and it was soon clear that little or nothing had been gained by the second operation. However, on examining the patient closely, I found that the points of origin and of maximum intensity of the pain were different from what they had been at first; that the pain now mainly originated in the mental branch of the third division of the fifth instead of in the labial branch of the middle division, and I then regretted that I had not had the mental nerve stretched as well as the infra-orbital.

On December 18th Dr Bishop proceeded to operate upon it also. The operation afforded instantaneous relief, and from that day to this there has been no return of the pain. Last week I received a letter from the patient stating that he had never had a twinge of pain since the last operation was performed.

Considering that the disease has hitherto yielded to no treatment, it seems that this case is of considerable value. It is true it is but one case, and the practice may not prove equally successful in others. It is also true that the relief may not prove permanent. It is only five months since the last operation was performed, and the patient has had at least one period of immunity as long during the course of his illness. But it cannot reasonably be doubted that the present immunity is due to the operation, and till evidence turn up to prove its failure, I think the treatment deserves a trial in every such case. Indeed, I would say that even should the pain recur, the plan of treatment is entitled to take the foremost place among the remedies for the disease.

With regard to the operation, there are two points on which I should like to insist—1st, That all the branches affected should be stretched, and not merely the one in which the disease is chiefly localised; and 2d, That tne nerve being grasped, not merely should traction be made upon the proximal part, but upon the distal also, the lip and cheek being seized and pulled downwards while the nerve is held at the point of emergence.

The case affords an illustration of the associated sympathetic or reflex pain. I think we may conclude that after the first operation there was no paroxysm originating in the infra-orbital nerve, but pain was felt there in association with the morbid action in the line of the third division. I have known of a member of the profession getting, as he said, the whole anatomy of the fifth nerve flashed upon his consciousness by the acute pain produced as he was getting a nasal polypus extracted, and as there pain was felt in all the branches when the one was irritated, so here the pain was irradiated along the nerves which had so long been the seat of morbid action.

As to the *modus operandi* of the procedure, it is impossible to speak positively, but there is apparently only one mechanical condition which such a procedure could relieve, viz., a shrinking or shortening of the nerve from thickening of its fibrous tissue. It is more probable that the explanation may be found in some more subtle molecular change, with which we are as yet unacquainted. But whatever be the correct explanation, the utility of the operation is in this case beyond question.