while Vulpian described a rarefaction of the posterior and external portion of the spinal cord, but the opinion, first advanced by Charcot and Vulpian, that the essential change is to be found in the peripheral nerves, is now gaining ground with pathologists to the exclusion of all other views. In kaki or beri-beri, the peripheral nerves have been found diseased by Raelz and Scheube.

Now, upon the question of the nature of the lesion, whether it is inflammatory or simply degenerative, a parenchymatous or an interstitial neuritis, I do not feel myself competent to speak. Eichhorst and Hadden believe the changes observed by them to have indicated a perineuritis, while most other authors believe that the disease begins in the nerve-fibres themselves. In one of the cases reported by Dr. Grainger Stewart, which was most probably an example of alcoholic paralysis, the changes observed in the nerves were regarded by Professor Hamilton, now of Aberdeen, to have indicated a simple degenerative neuritis, and the same view has been adopted by Moeli and others.

In some reported cases, the affected nerves have been found much swollen, and, in a case recorded by Desnos and Pierret, the posterior tibial nerve is said to have been nearly as thick as the little finger, and such a condition could hardly have been produced by a simple degenerative neuritis. It is hardly probable that an active parenchymatous neuritis would lead to such a remarkable degree of swelling of the nerve. That a parenchymatous neuritis has been met with in some cases of this affection scarcely admits of doubt. By inducing chronic lead-poisoning in guinea-pigs, Gambault produced inflammatory changes in the nerves of such a character as to lead him to name the lesion segmental periaxillary neuritis. In this condition, a segment lying between two of the nodes of Ranvier is found diseased, while those on the other side of these nodes remain healthy, and several segments may be affected in the course of one fibre. Dr. Dreschfeld met with this condition in the nerves in a case of alcoholic paralysis, and it may be assumed that it indicates a parenchymatous neuritis. A very valuable observation has just been made by Gambault in cases of alcoholic paralysis. At a certain elevation of the diseased nerve, he found the endoneurium thickened, and the nuclei of the endoneurium and of the sheath of Schwann much increased in number, while the medullary sheaths and the axis-cylinders were simply attenuated, probably by compression. Below this level, the changes observed indicated simply the ordinary Wallerian degeneration. It is therefore possible that, at a certain level of the nerve, a perineuritis may be observed, and that the fibres below this level, being separated from the trophic centres, undergo the usual descending degeneration.

With regard to the localisation of the lesion, this much is certain; the paralysis, which is present in these diseases, being of the atrophic variety, the lesion must implicate the ganglion cells of the anterior horns of the spinal cord, the fibres which connect them with the muscles, or the muscles themselves. The sensory disorders which are present, render it certain that the disease is not a purely muscular one, and, consequently, the lesion must be situated in the grey matter of the spinal cord, or in the fibres connecting the motor ganglion cells with the muscles. We have just seen that morbid anatomists are declaring in favour of the opinion that the disease is situated in the peripheral nerves rather than in the spinal cord.

Let us now see whether the clinical phenomena can be explained on this view, as well as, or better than on the assumption of the disease being of spinal origin. Some pathologists find great difficulty in adopting the neural theory, because the motor paralysis is often, as in the usual form of lead-paralysis, unaccompanied by any sensory disturbances. But, it is a general law that inflammation and injuries of mixed nerves give rise to a degree of motor paralysis which is out of all proportion with the sensory phenomena. In disease of the musculo-spiral nerve, caused by compression and exposure of its trunk as it winds round the humerus, the extensors and supinators of the forearm remain paralysed for four or five months; but the anaesthesia which accompanies motor paralysis is only slight or transitory. We often speak as if the spinal nerves of the body were all mixed nerves throughout their whole course, and it seems to be forgotten that, for nearly half their length, they are either purely sensory or purely motor. In the musculo-spiral nerve itself, the sensory branch—the radial—is given off above the elbow; and, as in the ordinary form of lead-paralysis, the supinator longus is always spared, it is clear that the lesion does not attain to the level at which the sensory branch would become affected. Similar remarks apply, although not to the full extent, to the nerves which supply the extensors of the leg. The common extensors of the toes and the peroneal group, and the tibialis anticus, may be separately paralysed without the sensory branch being much implicated; but, when all the anterior muscles of the

leg are implicated, the lesion has most probably reached the level of the external popliteal nerve, and in a case of lead-paralysis under me, in which all these muscles were implicated, the external surface of the legs and the dorsum of each foot were more or less anaesthetic.

The next argument brought against the theory of peripheral neuritis is that the muscles are paralysed in groups, according as they are associated in their actions, and not according to the peripheral distribution of the nerves. But the extensors of the forearm—which are the muscles usually attacked in lead-paralysis—form a functional group, which is innervated by one nerve, namely, the musculo-spiral, and consequently disease of that nerve would explain the paralysis as readily as disease of the spinal centre.

But what Remak calls the upper arm type of lead-paralysis, in which the deltoid, biceps, brachialis anticus and the supinator longus are attacked, is more difficult to explain on the peripheral neuritis theory, inasmuch as these muscles are innervated by three nerves, namely, the circumflex, the musculo-cutaneous, and branches from the musculo-spiral. It is now known that these muscles are paralysed by injury or disease of the fifth cervical root just before it joins the brachial plexus. This form of paralysis was first described by Erb, and, in a case observed by me a short time ago, the motor affection was accompanied by relative anaesthesia of the skin of the outer surface of the arm, and of the external border of the forearm and hand as far as the metacarpo-phalangeal joints of the thumb and index finger. Now in a case of the upper arm type of lead-paralysis recently under me, the paralysis was also accompanied by a considerable degree of anaesthesia on the outer surface of the upper arm, and even although it could not be traced below the elbow, the combination of symptoms present could be better explained on the supposition of a neuritis localised in the fifth cervical root immediately above its point of junction with the brachial-plexus than by a lesion in the grey matter of the cord. These considerations help to remove the main objections which have been advanced against the neuritic theory, but time will not allow me to enter upon the positive objections which might be urged against the spinal theory.

SOME POINTS IN THE PATHOLOGY OF MULTIPLE NEURITIS.¹

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MODERN observation has been rewarded by the discovery of an increasing number of cases in which more or less pronounced symptoms of paralysis have been found to be dependent upon changes in the peripheral nerves, the cerebro-spinal centres being usually free from any sign of lesion. In speaking of an "increasing" number of such cases, it is not implied that many more examples of the kind occur now than formerly, but that, as observation is on the alert, a larger number of cases is differentiated from the category of central organic lesions with which these instances were formerly confounded. The writer has so recently published a work upon this subject,² that he does not consider it necessary on the present occasion to do more than refer in the briefest and most general manner to the clinical and pathological aspects of multiple neuritis, but proposes to present for consideration a few remarks bearing chiefly upon the nature and pathogenesis of the disorder.

Multiple neuritis has been observed in connection with many conditions which are provisionally assumed to be associated with its etiology. Amongst these may be mentioned certain toxic influences, lead, arsenic, alcohol, infective disorders, as leprosy, diphtheria, syphilis, beri-beri, variola, enteric, recurrent and intermittent fever, tubercle, whilst other examples are associated with the not easily classed conditions of cold, rheumatism, diabetes. But besides these there would seem to be a somewhat large number of cases in which it is not possible at present to discover any antecedent which can properly be considered a cause. I shall recur presently to this very interesting part of the subject.

It is not intended to assert that the various influences mentioned produce examples of disease exclusively affecting the periphery of the cerebro-spinal nerves, the great centres themselves being invariably intact. Such is manifestly not the case. Although, for example, a large number of observations has shown disease limited to the peripheral nerves in the course of leprosy and of lead-poisoning, yet examples of these disorders have occurred in which the spinal cord itself

¹ Read in the Section of Pathology at the Annual Meeting of the British Medical Association in Brighton, introductory to a discussion on Peripheral Neuritis.

² *On Some Forms of Paralysis from Peripheral Neuritis*. Churchill. 1886.

has also proved to be affected. Quite recently, Oppenheim has published a case of lead-palsy in which the grey matter of the anterior cornua was diseased almost throughout the cord, the principal intensity being marked in the cervical and lumbar enlargements. There was atrophy of ganglion-cells, with increase and thickening of neuroglia, multiplication and enlargement of blood-vessels—a condition, indeed, of sclerosis. Nor have certain examples of lepra failed to show changes, though of a comparatively slight description, in the substance of the spinal cord. The same may be said in reference to alcohol. But the broad fact must, I think, be generally admitted, that a large number of cases is now on record in which the periphery of the cerebro-spinal nerves, more or less of them, is the seat of morbid change, whilst at the same time there is no discoverable lesion in the roots of the nerves or in the spinal cord.

The changes in the nerve-fibres are characterised by segmentation of the myelin, with more or less marked multiplication of the nerve-corpuscles, and disappearance of the axis-cylinder. With these there is often proliferation of the nuclei of the endoneurium, and the walls of the minute vessels are stuffed with cells,—evidence of interstitial neuritis. The lesions require the microscope for their recognition, and even then a doubt may arise as to their nature. In some, the appearances recall those characteristic of Wallerian degeneration—the alteration which takes place in the periphery of a nerve after section or destruction of its trophic centre. These are changes of a degenerative character to which it seems difficult to apply a term implying inflammation. In most examples, however, the proliferation of nerve corpuscles affords signs of inflammatory action sufficient to justify the application of the term parenchymatous neuritis to the condition.

The fact that, in most examples of multiple neuritis which come to a necropsy, the spinal cord and roots of nerves are perfectly free from pathological change, which is confined to a greater or less extent of the periphery of the nerves, is very startling and difficult of explanation. One cannot help feeling that, in spite of the absence of recognisable lesion, there must probably be some change in the central nervous system to bring about the almost simultaneous affection of the periphery of so many nerves. It was first suggested by Duchenne (of Boulogne) as an explanation of the absence of naked-eye changes in the spinal cord in a case of general subacute paralysis, that the spinal lesion might exist dynamically, if not organically. In the case in question, our present knowledge would lead us to think that there must have been lesion of peripheral nerves. In regard to cases of lead-palsy, where limitation of disease to the peripheral nerve so often occurs, De Watteville, advancing a step beyond Duchenne's suggestion, inquires whether, in such circumstances, there is not a functional lesion of the cord, which interferes with the trophic influence normally exerted by it upon the motor nerve-fibres, and thus gives rise to degeneration. More recently, Erb has suggested a similar explanation for the same paradoxical condition obtaining in cases of multiple neuritis generally, and as an alternative asks whether there may not be a pathological process in the ganglion-cells of the cord, which is short of destructive lesion, and is yet sufficient to produce degeneration in the periphery of the motor nerve-fibres connected with them. There is something attractive in this hypothesis when we remember the anatomical relation of a motor fibre to a ganglion-cell of the cord. The ganglion-cells of the anterior horn have, each of them, an unbranching process, which is called the axis-cylinder process of Deiters. This becomes invested with a medullary sheath, and then represents a medullary nerve-fibre entering into the formation of an anterior root of a spinal nerve. It is evident that the axis-cylinder of this fibre, even when it is continued to the remotest periphery of the distribution of the nerve, is yet still a portion—a long-drawn-out portion—of a large ganglion-cell of the anterior horn. But one would not expect, if this hypothesis were correct, that the lesion of nerve-fibres would continue, as it sometimes does even after a lengthy illness, strictly confined to the periphery. Were the functional activity of the cord to be long suspended, the atrophy might well be looked for throughout the length of the motor fibres. Moreover, if recorded observations be correct, it is not in the long-drawn-out process of the ganglion-cell that the pathological change commences in multiple neuritis, but in the myelin which invests it.

In the nature of things, attempted explanations of this kind are inconclusive, but they serve a useful purpose in pointing out paths of inquiry, and I would venture to refer to another which has been already alluded to by me in another place. Is it possible to explain the limitation to the periphery by the hypothesis that an influence of some kind is exerted upon vaso-motor centres in the bulb and cord? The effect of this would be to give rise to some alteration in the calibre of minute arteries, those in which the muscular element is relatively

most developed. These belong to the periphery of the arterial system, and in general terms correspond with the periphery of the distribution of nerves. It is conceivable, therefore, that the supply of blood to the periphery of nerves might, in this way, be modified by an irritative influence exerted upon vaso-motor centres in the bulb and cord.

Quoting from Michael Foster, "The tone of any given vascular area may be altered positively in the direction of augmentation (constriction), or negatively in the way of inhibition (dilation), quite independently of what is going on in other areas. The change may be brought about . . . by stimuli (chemical, blood stimuli) acting directly on the central nervous system." Notable diminution of blood-supply, sufficiently long continued, would occasion degeneration of the elements of nerve-fibre, whilst arterial dilation might be expected to cause blood-stasis and inflammation.

A very evident objection presents itself to this hypothesis. It will be naturally urged that marked signs of modified circulation ought to be observed in the course of multiple neuritis if this view is to receive support. Without attempting to refute adequately this objection, I would merely suggest the probability that the vaso-motor apparatus is highly differentiated in its function, and a limitation of its action to the blood-supply of one tissue is therefore quite conceivable.

A case which occurred in the practice of Mr. Hyde in the General Infirmary at Worcester, and was reported in the *BRITISH MEDICAL JOURNAL* of January 30th, 1886, by Dr. Allman Powell, has a very interesting bearing on this question. It was that of a man who suffered from diphtheria, and three weeks later was attacked with symptoms of Raynaud's disease (symmetrical gangrene) involving progressively the nose, ears, four fingers of the right hand, and two of the left, as well as two of the toes of the right foot. In the sequel the gangrenous fingers were amputated. Dr. Powell remarks that in this case "the vaso-motor, or trophic lesions, which occasioned the gangrene, would appear to have taken the place of post-diphtheritic paralysis." It is peculiarly interesting that the patient presented no symptoms of paralysis motor or sensory, and this fact is, to my mind, suggestive, so far as it goes, of the differentiation of function of vaso-motor centres to which I have alluded. It is well known that Raynaud is disposed to refer the symmetrical gangrene, which is associated with his name, to an influence exerted upon the vaso-motor centres of the bulb and cord. His view is contested by Pitres and Vaillard, who are inclined to see in these cases of gangrene the results of a primary peripheral neuritis. This opinion is based upon the constant presence, in such cases, of certain signs known to be commonly associated with neuritis, *e.g.*, the exquisite pains which may long precede the appearance of sphacelus—the phlyctenæ, followed by ulceration, developed on the extremities which are the seat of the pains—the altered state of the skin, with falling off or disordered nutrition of the nails. Unfortunately, as they remark, in none of the cases of symmetrical gangrene reported by Raynaud has any regular microscopical examination of the nerves been recorded. It will be observed that the view which I take is in some respects the converse of that advanced by Pitres and Vaillard. I should regard the peripheral neuritis as secondary to the altered state of circulation in the blood-vessels, and should refer the latter to an action upon vaso-motor centres by some stimulus contained in the blood. At the same time, I must disclaim any intention of bringing forward the hypothesis as more than a suggestion which appears to deserve a little consideration. It might be worth while to observe whether symmetrical gangrene—of the cause of which we are at present quite ignorant—is at all commonly associated with the antecedent occurrence of diphtheria, or some other infectious disease. The subject is one which is evidently of extreme difficulty, and explanations at present must be something but little removed from guess-work. I cannot help feeling, however, that for an explanation to be possessed of any force, it should not only shed light on the occurrence of the localised changes of nerve-fibres in an acute disease like multiple neuritis, but should be large enough to embrace also those instances which are apt to occur in the course of chronic affections of the spinal cord, as well as in diabetes. This is provided for in the hypothesis which I have advanced.

The occurrence of various trophic lesions in the course of tabes, is very familiar to us, and Pitres and Vaillard have found profound alterations in the nerves distributed to the parts involved. There is a not unfrequent association of perforating ulcer of the foot with arthropathy, and, in such circumstances, the same observers have disclosed evidences of parenchymatous neuritis in the nerves supplying the affected joints. The observations are in accord with the view long ago expressed by me, of the nutrition of bones and joints being dependent upon a centre—possibly vaso-motor—in the medulla oblongata.

Grainger Stewart, in 1881, suggested that the localised muscular paralysis often seen in tabes was due to peripheral neuritis. Déjerine, more recently, has expressed the same opinion. In one case of bilateral paralysis of the levator palpebre muscle, he found degeneration of the branch of the oculo-motor nerve to that muscle, whilst the main trunk of the nerve was unchanged. He sees in these an analogy with the degeneration of peripheral nerves of sensory function observed in tabes. The suggestion is an important one. The fact that these paralyzes are more often than not of transitory character, lasting, however, several months, is quite consistent with the notion. There is just now a woman under my care in hospital suffering from typical tabes. Upwards of a year ago, she had complete paralysis of the anterior tibial group of muscles in each leg, causing "foot-drop," and accompanied by anæsthesia in the corresponding nervous district. She has gradually recovered from this condition, and can now perform dorsal flexion of each foot with considerable force, the sensory disturbance having also correspondingly cleared off. She has manifestly suffered from peripheral neuritis of the external popliteal nerves.

My impression is strong that it is by an attack upon the vaso-motor centres in the bulb and cord by the disease process in tabes, that we must explain the various trophic disturbances, including those of bones and joints, perforating ulcer, falling of nails and teeth, and skin affections, as well as the localised paralyzes observed in the course of tabes dorsalis. They are associated, with remarkable frequency, with the occurrence of gastric and laryngeal crises. These are dependent, as I have shown, upon the irritative influence of sclerosis invading the immediate neighbourhood of the vagal nucleus, which is closely adjacent to the principal vaso-motor centre in the medulla oblongata. The peripheral neuritis occurring in diabetes may be conceived to depend upon an analogous association.

Time will not admit of further prosecution of this very interesting subject. Before concluding what I fear is a somewhat discursive paper, I shall be glad to say a few words respecting a form of multiple neuritis, to which allusion has already been made—that form in which none of the ordinary antecedents are discoverable on the closest inquiry.

I saw recently, with Mr. Howard Barrett, a young lady who was gradually recovering from what had evidently been an attack of multiple neuritis, which dated from November last. After symptoms of so-called "rheumatic" character, without any history of exposure to cold, the gait became ataxic in December; the knee-jerks disappeared; there were pains in her limbs, followed by wasting of muscles and loss of faradic excitability, together with cutaneous anæsthesia. No muscles of the trunk were apparently affected, except the rhomboid group, which were much atrophied when I examined the patient in July. She was then greatly emaciated, and the arms showed much muscular atrophy, the muscles, however, having begun to respond to induced currents. In the lower extremities, there was no power whatever of dorsal flexion of the feet, and the muscles on the front of the legs gave no response to faradism. Nor was there any increase, but rather a diminution, to the action of a slowly interrupted galvanic current. This reaction corresponded with what I have already noted in other cases published by me. The skin of the feet and legs was anæsthetic; the knee-jerks were still absent.¹

There was nothing in the history of this girl to explain the attack, except the symptoms styled "rheumatic"—pain and swelling below the knees—with which it is described as having commenced. Such etiological factors as alcohol, lead, syphilis, and diabetes, could be confidently put aside.

In March last I first saw, in consultation with Dr. Douglas Phillips and Dr. Baldock, a gentleman aged 66, who was unable to stand or walk without support. With help he could move about, and his gait was then markedly ataxic. Without entering now upon details, which must be reserved for future publication, I will only say that he suffered from inability to swallow (so that he had to be fed by stomach-pump), diplopia, ptosis of one eyelid, facial anæsthesia, some lingual paralysis, anæsthesia of the lower extremities, and of the right arm. In the left arm a cold object felt warm, and, indeed, throughout the left half of the body cold was felt as "quite warm." The knee-jerks were absent, and only slight plantar reflex occurred in one foot. The uvula was drawn to the left side. The voice was husky. Dr. de Havilland Hall, who examined the larynx at my request, reported the right vocal cord in cadaveric position, the left transgressing the median line. The patient, whom I have since seen on several occasions, has recovered to a very great extent, being able to eat, drink and walk about. His symptoms were clearly

referable to multiple neuritis. No causative antecedent was discoverable; alcohol, syphilis, diabetes, and other conditions could be excluded, and there was no history of any extraordinary exposure to cold. These two cases, in neither of which any of the ordinary causes was to be discovered, are still under observation, but I have also seen others. In the *Archives of Medicine*, August, 1884, there is an interesting paper by Dr. S. G. Webber, of Boston, in which he mentions having seen, during the fall and winter of 1883-84, no fewer than eighteen cases of multiple neuritis, in most of which no cause could be given for the disease. Some, however, appear to have been due to alcohol. Graves describes an epidemic of the disease which he had witnessed in Paris in the spring of 1828. "It began," he writes, "with sensations of pricking and severe pain in the integuments of the hands and feet, accompanied by so acute a degree of sensibility, that the patients could not bear these parts to be touched by the bed-clothes. After some time, a few days, or even a few hours, a diminution or even abolition of sensation took place in the affected members; they became incapable of distinguishing the shape, texture, or temperature of bodies, the power of motion declined, and finally they were observed to become altogether paralytic. The injury was not confined to the hands and feet alone, but, advancing with progressive pace, extended over the whole of both extremities. Persons lay in bed powerless and helpless, and continued in this state for weeks and even months. At last, at some period of the disease, motion and sensation gradually returned, and a recovery generally took place, although in some instances the paralysis was very capricious, vanishing, and again re-appearing. The French pathologists searched anxiously in the nervous centres for the cause of this strange disorder, but could find none; there was no evident lesion, functional or organic, discoverable in the brain, cerebellum, or spinal marrow."

Dr. Boissarie has described an epidemic of diphtheria, in which he was himself a sufferer (*Gaz. Hebdomadaire*, 1881, Nos. 20 and 21). It was remarkable, he states, in this, that paralysis in several cases occurred suddenly without angina or any diphtheritic affection of the skin or mucous membrane, and led to death in some hours or days. In other cases, the paralysis was followed by angina instead of being preceded by it, as ordinarily happens. In the midst of these exceptional cases, he observed others of characteristic diphtheritic throat, which were neither preceded nor followed by paralysis.

The disease termed kakké or beri-beri, which is endemic in Japan and the shores of Eastern Asia, is manifestly a form of multiple neuritis. Lacerda (according to Webber) claims to have discovered the bacillus of this disease. However this may be, the probability that beri-beri is dependent upon a specific bacillus appears to be very great. Specific bacteria are found in leprosy, and probably in syphilis and diphtheria.

Pitres and Vaillard have shown that, in the course of tuberculosis, as in other infective diseases, it is not rare for the peripheral nerves to show microscopical changes similar to those of degenerative neuritis, occurring independently of any recognisable changes in the nerve-centres, and attacking indifferently sensory, motor, and mixed nerves, involving, also, sometimes the cranial nerves, vagus, and phrenics.

The frequent known association of multiple neuritis with diseases characterised by the presence of specific bacteria, leads one to think it very probable that the cases to which I have referred as being without recognised antecedents, may owe their origin to a source of infection. It is evident that, if this be so, we know nothing of the path by which infection is introduced, nor of its nature. Is it possibly diphtheria introduced into the economy, through some part of the alimentary canal, which is beyond the opportunity of observation? This does not seem very probable, because the symptoms of these cases of multiple neuritis are, as a rule, more serious than those of the recognised diphtheritic form, in which it is exceptional to see examples of great severity. Pierson, of Dresden, has expressed the opinion that the "spontaneous" form of multiple neuritis seen in Europe is due to sporadic cases of the Oriental disease, beri-beri.

Even if time permitted, there would be but little use in speculating further upon this point, and I content myself therefore with suggesting for consideration and observation the question of the existence in this country of a specific disorder, which may occur without the anginous symptoms of diphtheria, and display itself in the form of a progressive multiple neuritis.

Professor CHARCOT contended that the diseases grouped under the term peripheral neuritis were so different in nature, that to group them together only caused confusion. Thus, in alcoholic paralysis, the sensibility was affected, there were neuralgic pains, and the affected muscles were tender, whereas in cases of lead-paralysis these characters were absent. The term peripheral tabes was a misnomer,

¹ At the end of September I found the knee-jerks returned, though that of the right side was difficult to produce.

and probably all cases included under that name were examples of alcoholic paralysis. Peripheral neuritis was not a distinct disease, but a substratum underlying the symptoms of several very different affections.

Professor BROWN-SÉQUARD considered that the effect of vaso-motor influences in producing tissue-change was unduly exaggerated. He pointed out the necessity of remembering all the causes which operated in this direction: (1) changes in the chemical constitution of the part; (2) the blood-attracting influence of local tissue-change; (3) the temporary suspension of the ordinary relations between the blood and the tissues. Under some circumstances of injury, etc., the blood in the veins came to resemble that in the arteries. All these conditions were produced through the influence of the nervous system.

Dr. HYL A GREVES described the pathological changes found in two cases of alcoholic paraplegia occurring in the Liverpool Royal Infirmary, under the care of Dr. Glynn. In the first case, a woman aged 48, who had drunk largely of brandy for some time, had suffered from *delirium tremens*. When admitted, she was greatly emaciated, and had a large bed-sore over the sacrum. The nervous symptoms were paresis of the lower extremities, loss of knee-jerk, pain on pressure of muscles, cutaneous anæsthesia and analgesia, burning pains in the soles of the feet. She died from septicæmia following septic absorption from the bed-sore. On *post mortem* examination of the nervous system, the following changes were found. In the spinal cord, there was distinct ascending degeneration in the cervical region. The cord otherwise appeared normal. The peripheral nerves of the extremities were more or less affected, especially the cutaneous branches, the mixed nerves being affected in less extent. There was complete atrophy of a certain number of nerve-tubes in others and degeneration of the white substance of Schwann, which was split up into masses of myelin. There was also granular degeneration of the axis-cylinders. The ganglia in the posterior roots of the spinal nerves were also altered. Some of these cells were much degenerated, and the surrounding connective tissue was markedly increased; it was possible that this had something to do with the production of ascending degeneration in the cord. Similar changes were found in the second case.

Dr. WHITTLE said that the speculative problems of the subject were beyond the province of the general practitioner, but he proposed to deal with it in its clinical aspects. He had seen a case of subcoracoid dislocation which remained unreduced for about six hours, during which the patient travelled a distance of ten miles. After reduction, all went well for a day or two, when pain came on, starting in the shoulder, and radiating round the back of the arm in the course of the musculo-spiral nerve, which was distinctly tender. At the elbow, the pain became diffused, but subsequently was most intense in the hand. Atrophy of muscle commenced in the upper arm, but extended to the forearm. The ball of the thumb wasted extremely. Pain was very severe, and only relieved by hypodermic injections of morphine. Recovery took place in three months. In another case, a horny growth, causing pain disproportionate to physical evidence of disease, was removed; but the pain was intensified, and rapid atrophy of the muscles of the leg followed. After amputation of the toe, the pain was much relieved. The atrophy was arrested, and the patient slowly recovered.

CASE OF EPILEPSY OF SIX YEARS' DURATION. COMPLETE RECOVERY AFTER SURGICAL OPERATION ON THE SKULL AND BRAIN.

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WITH REMARKS ON THE SURGICAL ASPECTS OF THE CASE

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THE interest which is being taken in the application of surgery to cerebral diseases induces me to place the following case on record. Although it is admitted that sufficient time has not yet elapsed to ensure certainty as to the permanence of the so far successful results of the operation, I think it advisable, without further delay, to add another contribution to a subject which, at the present time, is so much occupying the attention of the profession, as every additional fact cannot fail to be acceptable to an important practical inquiry which may be looked upon as still undecided. That frequently repeated epileptic attacks, which had been persistent for a period of no

less than six years, and so severe that the patient had been confined in a lunatic asylum for half that time, should by any procedure be suddenly arrested, and the satisfactory condition maintained for six months, is in itself encouraging regarding any proposed method of treatment, and, as such, worthy of note. In addition, however, this case presents other features of interest from a scientific and physiological point of view, which have been enhanced by the results of the operation, as it had been ascertained that each seizure was preceded by a visual aura, and followed by hallucinations, all of which have been entirely removed by trephining over the angular gyrus—a convulsion of the brain which recent researches have determined to be closely associated with the sense of sight.

W. A., aged 36, married, a stationer, with good family history, and without syphilitic antecedents, states that six years ago he received a violent blow on the right side of his head, by being forcibly thrown against a wall. At the time he was stunned, and was taken to the Middlesex Hospital, where it was found that he was suffering from a scalp wound, without apparent injury to the skull. In a few hours the patient regained consciousness, and, after his wound had been strapped up with plaster, he returned home, and, with the exception of headache and local soreness, was seemingly well. A few days after, the cut had healed, and he was in his usual good health. Six weeks later, he was suddenly seized with a convulsive attack, in which he lost consciousness, and it was observed that the movements of the limbs were confined to the left side. A fortnight afterwards there occurred a similar attack, and these have continued at irregular intervals ever since. He calculated that during the past six years there had been on an average one fit a week. His general health continued good; but after most of his attacks he was seized with violent mania, which rendered him dangerous to all around him, each of these seizures lasting many hours, and even days. He was, on this account, at various times an inmate of the Middlesex and St. Bartholomew's Hospitals, Highgate Infirmary, and other institutions; but in none of these did his condition improve. On the contrary, the violence after the attacks became such that, in February, 1882, he was sent to Camberwell Asylum, where he remained three years. In the report of his condition, kindly sent me by Dr. Schofield, his attacks are described as "of a very severe type; at those times he is most dangerous to those around him, and it takes four men to hold him down and prevent him injuring himself." Treatment of all kinds was tried without material benefit. He was admitted into the hospital under my care in May, 1886.

He was a tall, fair, somewhat delicate looking man, but otherwise in good health. All the organs and functions of the body were normal, and intelligence appeared to be unimpaired. Over the right parietal bone was a transverse cicatrix in the scalp. This was movable; no abnormality could be detected in the bone beneath it, and there was no tenderness on manipulation. On one or two occasions, hard pressure caused the sensation of a flash of light, followed by temporary loss of consciousness, lasting for a few seconds. The exact site of the centre of this scar was $3\frac{1}{2}$ inches from the longitudinal fissure, in a line drawn vertically $2\frac{1}{2}$ inches behind the external meatus of the ear. Nowhere was there any trace of paralysis or loss of sensation. The special senses were normal; the fundus of each eye was as in health, and the patient was in all other respects, physically and mentally, in sound condition.

The patient remained for nearly two months in the hospital under observation. During this time he had weekly epileptic attacks, none of which I myself had an opportunity of observing. The nurse, however, asserted that the attack was preceded by irritability of temper, and a tendency to let things fall. Then he somewhat suddenly lost consciousness, and had general convulsions, most marked on the left side, the whole seizure lasting from five to ten minutes. On recovering, he was only partially conscious and very violent, rushed about the ward, shouted, called out to and threatened imaginary people, and, when held, struggled and hit out with his fists. In from half an hour to an hour he gradually calmed, and fell asleep; after a few hours he woke with headache, and was dull and drowsy. Next day the patient was in his usual condition. On interrogation he asserted that immediately prior to the attack he saw a bright red light in front of his eyes, soon after which he lost his senses. He had no recollection of any hallucinations after the fits, but during them he certainly acted and spoke as if he saw people and other threatening objects.

The condition of the patient being very serious both to himself and his friends, as all ordinary methods of treatment had failed to give him any relief, and as he was anxious to adopt any means which offered the slightest chance of amelioration, I determined to have him trephined over the seat of the original injury to the scalp, in the hope that there might be a possible depressed fracture, or some other local or remov-