An Address

ON

TRAUMA IN RELATION TO DISEASE OF THE NERVOUS SYSTEM.

Presidential Address to the Manchester Medical Society.

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It is not an uncommon thing for patients who suffer from symptoms indicating serious disease of the nervous system to attribute the onset of their illness to some antecedent external injury. Thus patients who suffer from fits, from mental incapacity, or from some form of paralysis may state that these symptoms followed an injury to the head, to the back, or to one of the limbs. They assure the doctor that they were quite well before the accident, and that the fits, the mental incapacity, or the paralysis began some days, weeks, or months after the accident. Unless the injury has been severe, and unless a very direct connexion can be traced between the accident and the symptoms of the nervous disorder, the doctor in his superior wisdom is apt to make light, at least in his own mind, of the traumatic influence, and prefers to regard the apparent close relation between the two events—namely, external violence and nervous disease—as a coincidence. For, as a rule, he finds it impossible to trace any pathological sequence. His scientific training has taught him to beware of the dangerous doctrine *post hoc ergo propter hoc*, and for surer footing he falls back on the well-worn rock of inherited predisposition to disease of the nervous system, or to the modern favourite hypothesis that unknown microbes have entered the body, where they have manufactured toxins which have exerted a deleterious influence on certain parts of the nervous system, the selection of the part attacked varying partly with the nature of the toxin and partly with inherited weakness of structure. Such hypotheses appear to him the most likely explanation of the development of lesions which underlie well-marked types of brain and cord disease.

In many cases the views of our imaginary doctor are doubtless correct, either in whole or in part. Nevertheless, there are reasons for believing that trauma, apart from fracture or disease of bone, may initiate changes in nerve tissue which progress and lead to symptoms of permanent serious disease. At any rate, with our present ignorance regarding the etiology of disease, it behoves us to keep our minds open and prepared to admit the possible influence of any important antecedent to the onset of symptoms indicating bodily disease.

With these considerations in mind, it occurred to me that it would be profitable to consider to what extent trauma might be regarded as an exciting cause of serious nervous disease. At the outset it is desirable to indicate the limitations of my inquiry, and for this no single heading is quite appropriate. Thus the word trauma implies any external violence producing bodily injury. Now for my present purpose a term implying a narrower range of inquiry is needed. Concussion in the sense of a violent shock or shaking is more to the purpose, but I wish also to speak of the effects of injuries to the peripheral nerves. My purpose, in fact, is to consider briefly some of the effects of concussion of the brain and spinal cord apart from the results of injury or disease of their bony envelopes as well as some of the results of injury to the peripheral nerves.

CONCUSSION OF THE BRAIN.

In surgical works this heading is used for a definite clinical condition which is characterized by a more or less complete suspension of the functions of the brain. I propose to use it in a sense which is wider and in accordance with its original meaning—namely, a violent shaking or agitation—and to consider some of the results that may follow a shaking or concussion of the intracranial contents as produced by a blow or a fall on the head.

The anatomical effects of such concussion are manifold; they may be slight and temporary, as when limited to a sudden displacement of the cerebro-spinal fluid with ensuing or concomitant changes in the quantity of blood in the cerebral vessels, or they may be severe and more or less permanent and demonstrable at necropsies. Such conspicuous pathological results may be classified as follows:

Haemorrhage	{	Meningeal	{	Subcranial.
				Subdural.
				Cerebral from rupture of a vessel.
Inflammatory changes	{	(a)	{	Subcranial abscess.
		(b)		Abscess in the brain.
		(c)		Pachymeningitis.
				Meningitis { Acute.
				Chronic.
- Laceration of brain—associated with ecchymoses or extravasations of blood into the cerebral substance.
- New growths—started by molecular changes arising in connexion with some of the above lesions or independently of them.

The symptoms and results of these various lesions are to be found in the textbooks, and it would be out of place to attempt their systematic description to-night. My object is rather to bring before you types of cases which have impressed me as of great practical importance, and especially those which illustrate some of the later effects of head injuries.

Let me begin by trying to draw a clinical outline of the sequences of cerebral concussion. A man receives a blow on the head which, without seriously damaging the skull, is severe enough to give a violent shake to the intracranial contents. He may or may not lose consciousness, and in either case he may make a complete recovery, or become the subject of symptoms which are either temporary or permanent. The individual symptoms are numerous: they comprise headache, vertigo, convulsions, mental disorders, paralysis, anaesthesia, and, indeed, almost any symptom that may result from disease of the brain occurring independently of trauma. They may, however, be arranged into definite groups constituting clinical conditions which for the most part are well recognized. Of these I would especially refer:

1. To the early group of symptoms which indicate the presence of meningeal haemorrhage.
2. To symptoms, which although they may develop soon after the accident, are not usually conspicuous until some weeks later. Under this heading we have:
 - (a) Varieties of so-called neurasthenia.
 - (b) Some forms of insanity.
 - (c) Symptoms of brain tumour.
3. To symptoms associated with the condition known as "late apoplexy."

Meningeal Haemorrhage.

Of the immediate effects of head concussion none is more serious than meningeal haemorrhage. In typical cases the diagnosis is easy, but we must remember how variable the clinical course may be, and how easily the condition may be overlooked—a mistake which is responsible for many deaths as well as for many cases of permanent physical and mental incapacity.

The haemorrhage may occur either between the dura mater and the bone or between the dura mater and the cerebral cortex. The former is called subcranial, the latter subdural, haemorrhage. In a typical case of subcranial haemorrhage three clinical stages may be distinguished: An initial stage in which there is partial or complete loss of consciousness as an immediate result of the blow to the head. Then consciousness is regained, and the patient passes into the second stage, known as the "lucid interval," which lasts from a few hours to a couple of days. During this period the patient may feel and appear to be pretty well, and sometimes he is able to resume his work. Sooner or later, however, according to the quantity of blood which is separating the dura mater from the bone, consciousness is again lost, and stupor deepens into fatal coma. During this third stage spasms, accompanied or followed by paralysis, may be observed on the side of the body opposite to that of the blood clot.

The subdural is distinguished from the subcranial variety of haemorrhage by the absence of a lucid interval and by the more rapid onset and development of compression symptoms, which, too, are more general in distribution. To these rules there are, however, many exceptions, and I will now describe a case of subdural haemorrhage in which not only was there a comparatively lucid interval, but one also of considerable length.

CASE I.

A man aged 30 fell off a lorry, probably on to his back. He was stunned but quickly recovered, carried a hamper for about half a mile, and then walked back to his place of business another half-mile. He told his employers that he had had a fall and sprained his wrist, but he made light of the accident. The same night he felt giddy, and is said to have lost consciousness for a time. During the next day he felt pretty well, but in the evening he had two fits in which the right hand worked. During the following week he felt well, and eight days after

the accident he had a chat with his manager, who did not notice anything wrong with him.

Four days later, that is, twelve days after the accident, I saw him in consultation with Dr. Hopps. The patient was lying in bed in a state of stupor, and could be roused with difficulty to answer questions. Twitchings of the right thumb and first finger were observed; there was no paralysis. The knee-jerks were exaggerated. The pupils were small, but reacted to light. The tongue was large and thickly coated. The urine contained a considerable quantity of albumen but no casts; there were signs of hypertrophy of the left ventricle. Dr. Hopps informed me that the man had followed his work regularly for the last seven years without any illness, and had never suffered from headache, vomiting, or drowsy. It is interesting to note that he suffered from violent headache for the first time on the seventh day after the accident.

I was unable to form a positive diagnosis. The condition of the heart and urine pointed to chronic nephritis, and suggested that the nervous symptoms might be expressions of a uraemic condition. On the other hand, the history of good health prior to the accident was a strong point in favour of the view that the stupor and twitching were the results of an intracranial lesion produced by the fall.

In order to be ready for a possible operation we decided to send him to the infirmary, where he came under my care. At this time, just a fortnight after the accident, he could not be roused to answer questions. No injury to the skull could be detected. There was ptosis greater on the left than the right side, and the left pupil was smaller than the right one. When supported he could walk a little; the left limbs were weaker than the right limbs. The knee-jerks were exaggerated. A week later he was less drowsy, could answer questions, and said he remembered falling off the lorry. Two days later—that is, three and a-half weeks after the accident—he lapsed again into stupor and had a convulsive attack on the right side. I then asked Mr. Thorburn to see him. After a careful examination Mr. Thorburn decided that Bright's disease might be a sufficient explanation of the man's condition, and that he did not feel justified in opening the skull.

The patient died a few days later, and at the necropsy a large partially-organized haematoma was found under the dura mater on the left side; it covered the whole motor area, and extended backwards over part of the parietal and forwards over part of the frontal lobe. The kidneys were small and granular, and the heart was hypertrophied.

In this case the presence of Bright's disease made it difficult to decide whether there was an intracranial lesion or not. Moreover, even if the existence of meningeal haemorrhage had been certainly known it was not easy to locate it, for while clonic spasms were observed on the right side paresis affected the left side of the body. It is noteworthy that although a varying degree of stupor was present during the first three weeks, actual coma did not come on till three or four days before death. Other cases might be mentioned in which the onset of compression symptoms was still longer delayed. A remarkable case in this respect was recently operated on by Mr. Collier.

CASE II.

A man, aged 46, was crossing the street when he slipped and fell on the back of his head. He went home by tram, then fainted, and went to bed for three days. On getting up he felt so weak and dazed that he returned to bed, where he remained for six weeks. During this time he suffered from severe headache. About ten months after the accident he was admitted under Dr. Steell at the infirmary. He was then suffering from paresis of the left arm and leg. After three weeks at the infirmary and another three weeks at the Cheadle Convalescent Hospital he returned home, where he had an attack of unconsciousness lasting two days. He remained in a weak state of health suffering much from headache, till he came under Mr. Collier's care about sixteen months after the accident. At this time he had persistent headache, and fell at times into partial stupor. His memory was bad, and his speech was often indistinct and aphasic; constant twitchings of the flexors of the left wrist and fingers were observed, and the left arm was partially paralysed. About a fortnight later Mr. Collier opened the skull on the right side and removed from beneath the dura mater a membranous looking clot of yellowish colour. The man died the next day from collapse.

The cases just related show what a long time subdural haemorrhage may exist without giving rise to decided signs of compression, its presence being associated with symptoms which indicate disturbance rather than abolition of the functions of the cortex. We may now ask, Are these cerebral symptoms due to the surface haemorrhage or to associated cortical lesions, both being immediate results of the trauma? We know that haemorrhage over the surface of an infant's brain produced by obstruction to the circulation during a difficult labour will hinder the development of the brain, and lead to atrophy and sclerosis of the cortex, lesions which are expressed clinically, by spastic limbs, convulsive attacks, and a varying degree of dementia. At least this is the usually-accepted pathology, for it is only fair to state that recently evidence has been brought forward to throw doubts on such a pathological sequence, and in support of the view that the cortical degeneration is primary, being possibly started by some toxin, and not due to the pressure effects of haemor-

rhage. Certainly in the adult it is probable that a blow on the head severe enough to produce meningeal bleeding will also cause laceration, with extravasation of blood into cortical tissue, and that many symptoms, such as headache, vertigo, mental disability, are due to a bruised brain, rather than to a clot on its surface, the latter lesion being responsible for pronounced stupor or coma. Nor can there be any doubt that the former group of symptoms is frequently associated with a bruised cortex in the absence of meningeal haemorrhage. To this group of symptoms the term "neurasthenia" is often loosely applied, and on this subject I would now make a few remarks.

Traumatic Neurasthenia.

The symptoms of typical neurasthenia, whether the result of trauma, of prolonged mental strain, or of other causes, are well known, and constitute a clinical condition which as a rule is readily diagnosed. In all probability the condition depends on molecular changes in certain groups of cortical cells, changes which are closely allied to those which have been shown to follow great fatigue or prolonged experimental stimulation.

The subject of traumatic neuroses is a large one; it has been admirably studied by Page, Oppenheim, my colleague Mr. Thorburn, and many others, and to-night I will only allude to a small portion of the subject. I will omit all reference to the diagnosis of malingering, to traumatic hysteria, and to the transient forms of neurasthenia which, even apart from bodily injury, are met with after railway accidents, as results partly of the terror or horror incident to the catastrophe, and partly of the mental anxiety associated with litigation. This, together with repeated medical examinations and suggestions from friends and lawyers, leads the patient to conscious or unconscious exaggeration of his symptoms, and frequently to the simulation of serious disease.

Of such temporary cases I am tempted to give one example, because it affords an amusing illustration of the mixture of neurasthenia, hysteria, and simulation met with in many cases that go to trial.

CASE III.

A young married man, after a blow on the back from the sudden closing of a railway carriage door, suffered from weakness of the legs, left hemi-anaesthesia, and general debility of both mind and body. One symptom on which he laid particular stress was complete impotence. After the trial, at which each side was well represented by the medical profession, he received satisfactory compensation for his ailments, and soon regained his health. About a year later I met his doctor, who said, "You will be interested to hear that the wife of our patient who complained of impotence was safely delivered of well-developed twins exactly nine months from the time when his symptoms were most pronounced."

Passing now to more permanent forms of neurasthenia, it is, I think, open to question whether they have yet received adequate recognition. I allude to cases which, whether the result of railway or other accidents involving compensation for damages received do not recover after the cessation of legal irritation, and after claims for loss of work and health have been duly met.

Two groups may be distinguished. The one group comprises cases which clinically closely resemble those of non-traumatic neurasthenia. As a rule, there is inherited predisposition to neurosis—that is, the intimate structure of the cortical centres is easily deranged, and molecular changes started by head injuries tend to pathological increase, while the chemical products of cell disintegration may set up sclerosis of the surrounding tissue.

I have said that clinically these cases resemble those of ordinary neurasthenia. No doubt this is so at first; but as time goes on the tendency to exaggeration of individual symptoms gives way to mental dullness and to other signs of cortical deterioration. The other group comprises cases where from the first there are signs of cortical lesions, these occurring either beneath the injured spot or on the side of the brain opposite to the injury as a result of *contrecoup*. The lesions are various, including minute lacerations, punctiform haemorrhages, and other changes which collectively constitute contusion of brain tissue. In severe cases these lesions, as already stated, may be associated with meningeal haemorrhage. Now, the symptoms of a bruised brain may closely resemble those of the later stages of the first group of cases which are dependent on molecular changes and slight degrees of cortical sclerosis, hence a certain diagnosis of the pathological condition can only be attempted soon after the accident, when notable differences may be observed between the symptoms of a bruised brain and those of ordinary neurasthenia.

Instead of an uncontrollable tendency to discuss their sym-

ptoms in detail, sufferers from a bruised brain are dull and apathetic; mental action, instead of being acute and introspective, is carried on with difficulty, as if a thick mist enveloped the faculties. Insomnia, a prominent feature in traumatic neurasthenia, is less common in cases of bruised brain, in which, on the contrary, there is a tendency to drowsiness and stupor.

The neurasthenic complains of pains in the back, which often radiate to the head and to other parts of the body. In addition to pain there is pronounced hyperaesthesia; the spine and adjacent parts become exceedingly sensitive not only to deep pressure but also to a light touch, and the hyperaesthesia may extend to the special senses. Dyspepsia, palpitation, and other visceral disorders are also common.

The above symptoms are absent or inconspicuous in cases of bruised brain. The neurasthenic is able to commence work, either physical or mental, and to do it accurately for a short time, but he soon tires; he lacks concentration, finds his memory unreliable, and exhibits indecision in all his actions. He is constantly restless, while anxiety about his condition fills his thoughts, and thus constitutes another hindrance to reliable work.

The patient who has a bruised brain is quite unfit to follow any employment; he is too apathetic as a rule to exhibit anxiety or to worry, and when his cortical lesion is severe and extensive apathy is often associated with profound loss of memory and other signs of considerable mental deterioration. In both groups a marked intolerance to alcoholic drinks is frequently observed.

Now the distinctions above mentioned, although practical ones, are not always prominent, and tend to fade as time goes on. Nor need we be surprised if unable to make a diagnosis between molecular changes in the cortex and bruising of its tissue, for both morbid processes may disorder the same neurons, when the symptoms of the changes will be similar or identical.

But in whatever way we look at the matter, one thing stands out clearly, namely, the permanent physical and mental incapacity that so often follows concussion of the brain, and even when the injury to the head appeared to be a slight one. I would lay particular stress on the minor degrees of mental disturbance which are apt to be ignored, and yet seriously interfere with the patient's wage-earning capacity.

Insanity.

With regard to actual insanity after injuries to the head, there is abundant evidence that it does occur, and sometimes apparently without any evidence of hereditary predisposition. It may develop out of the prolonged depression and irritability so often associated with bruising of the brain, and especially if there is a predisposition to insanity, either inherited or acquired, in consequence of the cerebral changes which result from syphilis or from prolonged indulgence in alcohol. As to forms of insanity which ensue in cases of depressed fracture and of surface haemorrhage, these are doubtless becoming rarer owing to the activity and skill of our surgeons.

Sometimes a mental condition develops after concussion which presents a close resemblance to that of general paralysis, but as a rule dementia is less marked, while the pupil phenomenon and other paralytic symptoms are usually absent. One such case I saw some years ago.

Cerebral Tumour.

That tumours of the brain may owe their origin to nutritive changes produced by concussion is now generally admitted. In the *Medical Chronicle* for 1893 I have recorded a case in which symptoms of a brain tumour developed a few weeks after a blow on the head from the fall of a scaffold pole. The patient died a year after the accident, and at the necropsy a vascular sarcoma was found in the upper part of the right frontal lobe and immediately beneath the part injured.

Late Apoplexy.

The last sequel of head injuries to which I will refer is that known as late apoplexy. Some weeks after a blow on the head the patient becomes suddenly unconscious and dies in a state of coma, or he regains consciousness when it is found that he has hemiplegia. Cerebral haemorrhage from rupture of an artery is found at the necropsy. The character of the apoplectic attack and the morbid anatomy do not present any obvious differences from those of cerebral haemorrhage occurring independently of trauma. Neverthe-

less, evidence is forthcoming in some cases that the accident was responsible for the arterial disease. Thus the patient was under 40, and had no signs of heart disease or of arterial degeneration, while the influence of syphilis, of alcoholism, and of nephritis could be excluded. Moreover, symptoms of vascular disease developed soon after the accident, and the apoplectic attack occurred within a few weeks. A voluminous literature, especially in Germany, has gathered round these so-called late apoplexies, the pathology of which has excited much discussion. Bollinger, who was the first to use the term "traumatic late apoplexy," states that a necrotic softening is set up in the injured portion of brain, and that this is combined with changes in the walls of the vessels, which, together with alterations in the relations of resistance and pressure lead to haemorrhage.

The possibility of arterial disease being set up by concussion is a valuable suggestion, but as to the correctness of the pathological views just related I am unable to offer an opinion.

CONCUSSION OF THE SPINAL CORD.

Using the term concussion in the sense already explained, we omit from consideration all cases of injury to the cord from direct wounds or from fractures and dislocations of the vertebral column, and our inquiry is limited to cases which seem to result from a violent shaking of the elements of the cord. Such concussion may follow a blow to the back, or a fall on the back, the head, the feet, or the buttocks.

Indications of cord disease may come on immediately after the concussion or be delayed for a few days or weeks. In the former case the lesion is usually a severe one. Occasionally the cord is lacerated when the vertebrae are uninjured. Sometimes haemorrhage occurs either outside or inside the dura mater, or into the substance of the cord itself. A case remarkable as regards the probable origin of the haemorrhage is now in my wards at the Royal Infirmary.

CASE IV.

The patient is a coachman, aged 42, who four years ago was thrown from a carriage and sustained a fracture of the base of the skull which kept him in hospital for fourteen months. After the accident he was unconscious for nearly a month. On regaining consciousness he was unable to move his arms and legs; the paralysis remained absolute for three months, then power began to return and has gradually improved ever since. At the present time he can walk slowly in a stiff fashion, the legs being rigid and their movements weaker than normal. The knee-jerks are exaggerated, and both ankle-clonus and Babinski's reflex are well marked. The left arm is weak, the right is subject to coarse tremor on voluntary movement. The wrist-jerks and elbow-jerks are much increased.

This condition of spastic paresis indicates a slight affection of the pyramidal tracts, in all probability due to a compression myelitis in the cervical region set up by haemorrhagic effusions, which drained down to the top of the cord from the destructive lesion at the base of the skull.

Another cause of severe damage to the cord is a blow in the vicinity of the vertebral column. Cases of gunshot wounds were long ago described by Weir Mitchell, in which immediate and complete paraplegia followed the impact of a bullet near the spine, the cord being softened at the spot while the bones were uninjured. A case of this kind was recently under my care.

CASE V.

A man was kicked by a horse between the left scapula and the spine. During the next two days he suffered from severe pain, associated with numbness in the legs. On the third day his legs were paralysed. When seen a fortnight later the paraplegia was absolute and was accompanied by other symptoms indicating destruction of the functions of the cord at the level of the fourth dorsal segment. The patient died at home a few months later. No necropsy was obtained.

In connexion with the immediate effects of concussion it is of the greatest interest to note that a few cases of complete paralysis have been recorded in which no lesion of the cord was found either with the naked eye or the microscope a few days after the injury. The assumption is that the molecular nutrition of the nerve elements was abolished. Now whether minute changes were overlooked or not, these fatal cases without obvious lesions afford a valuable suggestion in regard to the development of symptoms of cord disease a long time after an accident. With such cases in mind, it would not, indeed, be foolish to suggest that more diseases of the cord owe their origin to a long-forgotten trauma than has hitherto been admitted. But without dwelling on this speculation, and without attempting to give complete pathological explanations, one may admit that such evidence as we have bearing on the development of cord disease some months after concussion is worth considering and is often convincing.

Before referring to these later effects of trauma, mention may be made of an intermediate group of cases in which initial symptoms are absent or slight and grave symptoms come on a few days or weeks after the injury, having the character and course of a subacute myelitis.

Thus Lohrlich³ reports the case of a man, aged 58, who, after a fall on his back, suffered for a fortnight from violent pains on attempting to move his legs. During the next fortnight the pains were less severe, but on the twenty-ninth day after the accident there quickly developed a complete motor and sensory paralysis of both legs together with paralysis of the bladder. At the necropsy a softened focus was found in the dorsal region.

Gowers,⁴ too, mentions the case of a lady who was severely shaken in a railway collision. Immediately after the accident she seemed to have suffered no injury, but in a few days paraplegia developed, and from its consequences she died six weeks after the accident. Throughout the dorsal portion of the cord indications of a subacute myelitis were found, chiefly in the white columns.

These cases bridge over the interval between the early severe changes, whether molecular or coarse, and later changes, more or less degenerative in character, which underlie the symptoms of locomotor ataxia, disseminated sclerosis, chronic myelitis, and other slowly progressive diseases of the cord. The symptoms of these disorders may not appear until several months after an accident, and their dependence on morbid changes produced by concussion can only be inferred after the exclusion of other causes.

On going over cases of spinal disease which have been under my care during the last fifteen years, the collection of hospital cases having been kindly summarized by a former house physician, Dr. Charles O'Neill, I find that a relation to trauma can be traced more frequently in disseminated sclerosis than in locomotor ataxia. The following are two examples:

CASE VI.

A woman, aged 30, previously healthy, fell through a trap door into a cellar, a distance of 5 ft., and dropped on to a stone slab in a sitting posture. A few days later she complained of numbness on the left side of her face and back. In about five months she began to have a difficulty in walking; this increased, and spastic paraplegia slowly developed. This was followed by nystagmus and weakness of the arms. When seen at a later period the symptoms were unmistakably those of disseminated sclerosis.

CASE VII.

A man, aged 27, fell over a banister on to his back. He was unconscious for four days. After a week he went to work, but could not stand properly. The weakness in the legs increased, and when examined two years after the accident he had well-marked signs of disseminated sclerosis.

Much has been written on the subject of traumatic tabes and in a few cases it seems probable that concussion has started the changes in the posterior root fibres. A patient of my own stated that his ailment dated from a fall three months before he came to see me. He slipped down some thirty stone steps, jogging the lower part of his back on each step. He walked home and went to bed. Two days later he began to stagger, and had pains in the soles of his feet. On examination the patient showed a moderate degree of ataxia. The knee-jerks were absent. The pupils were unequal, and did not react to light. Micturition was delayed.

Lichte,⁵ who has written a thesis on traumatic tabes, gives a full account of a case in which a severe sprain of the left arm was followed in a few months by signs of tabes. Syphilis and other possible causes could be excluded.

These cases, however, are exceptional, and a study of the literature, as well as one's own experience, leads to the conclusion that although concussion may initiate the development of characteristic signs of tabes it rarely acts as the sole cause of this disease. Frequently it will be found on careful inquiry that the tabetic patient was not quite free from symptoms before the accident. No doubt trauma often co-operates with other causes, especially syphilis, and tends to light up disease which was pre-existent, or for which the ground was duly prepared.

INJURY TO THE PERIPHERY OF THE BODY.

Passing now to the periphery I will consider only the question whether disease of the spinal cord may result from injury to the peripheral nerves. A good starting point, because the condition is well known, is arthritic muscular atrophy. Articular inflammation, whether produced by injury or disease, is almost invariably followed by rapid wasting of the muscles that move the joint, the extensor muscles being predominantly affected. Thus, if the shoulder is injured the deltoid chiefly wastes; if the elbow the triceps; if the hip the glutei; if the knee the quadriceps. The atrophy comes on quickly so that in case, for example, of injury to the

knee a difference of an inch between the girth of the thighs might be noticed at the end of a week or ten days. As to the explanation of this condition, the evidence both experimental and pathological gives support to the reflex theory held by Paget, Vulpian, and Charcot, which is that the muscular atrophy depends on a damaged nutrition of the motor cells of the cord, this derangement being the effect of morbid impulses from the irritated nerves of the injured joint. The correctness of this view is practically proved by the experiments of Raymond, who found that a previous division of the posterior spinal roots prevented the wasting of the muscles.

Further light has been thrown on the subject by the investigations of a former Manchester student, Dr. Warrington, who has shown that actual changes in the motor cells may be detected after lesions of the posterior roots.

There can indeed be no doubt that healthy sensory impulses are essential for the proper nutrition of motor neurons, and that morbid sensory impulses will lead to detectable disease. Do the morbid changes pass away when the cause—say articular inflammation—is removed, or do they ever progress and lead to atrophy and destruction of the motor cells? In other words, may progressive muscular atrophy result from peripheral irritation? The answer is that occasionally the muscular atrophy resulting from arthritis becomes permanent, and that many cases of progressive muscular atrophy appear to originate from a severe sprain or other injury to a limb. I have notes of several instances of this relation. A few years ago I showed a patient at a meeting of this Society in which the sequence was striking.

CASE VIII.

The patient, a powerfully built policeman, aged 36 years, was admitted in the infirmary with characteristic signs of progressive muscular atrophy, the muscles of the right arm being principally affected. He attributed the onset of his ailment to a violent struggle with a maniacal person in which he strained the upper part of the right arm. About a month later he noticed that the arm was getting weak and thin. The weakness and wasting increased and were marked when he came under my care twenty months after the injury.

The boundary line between cases of arthritic muscular atrophy and chronic anterior poliomyelitis is not always easy to define, and the differential diagnosis may present great difficulties.

In conclusion, I would draw attention to the need for further study of the after-effects of trauma, and especially as regards their prognosis. Traumatic neurasthenia and hysteria have received ample consideration, but there is room, I think, for a fuller recognition of the prolonged and sometimes permanent incapacity for work that may result from concussion of the head or the body.

Adequate recognition of the possibility of disease leads to increased care in preventive treatment, and to mention only one thing, I believe that the simple prescription of a longer rest in bed after injury than is usually obtained in hospital practice would do much to reduce the number of sufferers from chronic diseases of the nervous system.

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THE EXAMINATION OF THE TISSUES OF THE CASE OF SLEEPING SICKNESS IN A EUROPEAN.

[WITH SPECIAL PLATE.]

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In October, 1902, Sir Patrick Manson was consulted by the wife of a missionary from the Congo suffering from chronic pyrexia and enlargement of the spleen. After an examination he came to the conclusion that the disease was probably trypanosomiasis, and sent the patient to the wards of the London School of Tropical Medicine, where Dr. Daniels confirmed the diagnosis by finding trypanosomes in the blood. A full account of the case was published in the *BRITISH MEDICAL JOURNAL* of May 30th, 1903, by Manson and Daniels, and the termination and results were described later by Manson in the same journal, December 5th, 1903.

The symptoms that were present when the patient was