THE RÔLE OF TRAUMA IN THE ETIOLOGY OF ORGANIC AND FUNCTIONAL NERVOUS DISEASE.*

BY S. A. KINNIER WILSON, LONDON.

INTRODUCTION.

THE subject of the possible causal relation of trauma to the development of organic and functional nervous disease is thorny and difficult, yet of first and ever-increasing importance. It concerns the general practitioner fully as much as the specialist, if not, indeed, more. There must be scarcely any medical men who have had experience of the question of trauma in compensation cases but are able to quote decisions in law which have been admittedly unsatisfactory from a medical and scientific point of view. The reason is that medicine is not an exact science, at least by no means wholly so, hence we must expect (1) great variety of opinion, and (2) occasional conflict of medical evidence. I say occasional, yet when, in reality, has a case involving the question of trauma come before the courts in which medical men have not appeared to be diametrically opposed to each other? One can understand differences in opinion and in interpretation, but how can we reconcile differences in observed facts? Too often has this medical conflict provided material for the cynic; too frequently have the representatives of our profession allowed themselves to be influenced, not by the objective medical facts of a case in dispute, but by the standpoint of the particular side on which they happen to be called. A man famous in the Edinburgh medical school of the middle of last century was Sir Robert Christison, the medical jurist, and of him Lord President Inglis, the greatest Scottish judge of the same period, said: "The Professor went into the witness-box not in the spirit of a partisan, but in his proper office as a medical jurist, to aid the court and the jury in the elucidation of truth, and in securing the ends of justice." Were this, the ideal attitude of the medical and scientific witness, adopted more generally, there would be fewer unseemly disagreements on the part of those of us who are asked to express medical opinion in court cases.

In fairness, it must be admitted that the absence of authoritative

* Read at the Annual Meeting of the American Medical Association, San Francisco, June, 1923, and published here by permission of the Association.

medical pronouncements in the matter of trauma is greatly to be deplored. There is no body or compendium of medical doctrine to assist the medical witness and, through him, the court. There is no unanimity in respect of terminology and definition. The least experienced and least knowledgeable medical man may find the same weight attached by the court to his evidence as to that of others much better able to express considered views. For the guidance of the profession, for the attainment of some semblance of accord, opinion expressed by a commission of accepted experts is the first desideratum. Contradictory statements cannot be avoided unless and until the physician has a thesaurus of neurological doctrine to which he may turn and from which he may learn.

The extreme importance of the subject is evidenced by the enormous amount of litigation to which it gives rise. The total of money involved is almost incredible. According to figures recently given in the *Lancet*, quoted from a book by J. L. Cohen on 'Workmen's Compensation in Great Britain,' the "total cost to the country of accidents and industrial diseases is from 36 to 48 million pounds a year," a colossal if in any way approximate figure. Litigation is incessant. Cases are constantly being taken up as a speculation by the less reputable kind of legal man, while it need scarcely be said, as long as human nature is as it is, efforts will be directed to making capital out of infirmities. Erichsen, whose right to speak with authority none ever denied, declared that "an extensive experience in railway accidents will probably impress the observer more with the ingenuity than the honesty of mankind." As we shall see, and as I shall be able to prove by figures, the greater the chance of remuneration the higher the percentage of 'accident' cases.

WHAT CONSTITUTES AN ACCIDENT?

Trauma has been defined as "an abnormal condition of the body caused by external injury"; an accident, as "injury by some unexpected and external event." The original (English) Workmen's Compensation Act of 1897 was silent as to disease; the cases coming under it were defined as constituted by "personal accident arising out of and in the course of the employment" of the individual concerned. The Amending Act of 1906, however, included certain scheduled diseases arising out of the employment, such as lead, mercury, and phosphorus poisoning, miners' nystagmus, telegraphists' cramp, and many other ailments. These are all now regarded as morbid conditions for which compensation is due. From the legal point of view, therefore, in respect of workmen's compensation, the distinction between accident and disease is vanishing. I need scarcely indicate how unsatisfactory this is from the medical point of view, and how it tends to etiological confusion. A few legal decisions may be quoted by

way of illustration. A wool-sorter contracted anthrax, and this was held to be an 'accident' arising out of employment; the 'accidental alighting of the bacillus in the eye' was taken to be equivalent to the accidental squirting of molten metal or some poisonous liquid into the eye. Owing to the breakdown of a pump a miner had to stand in icy cold water for some hours; pneumonia supervened and led to death, and this illness was considered an 'accident' arising out of employment. On the other hand, a fever hospital attendant developed scarlet fever, but failed in his claim for compensation due for this 'accident,' because he could not prove a definite association of his illness with his employment. Again, a workman inhaled sewer gas and developed enteritis; he claimed that his inhalation of the bacillus was an 'accident,' but the court held that sewer gas was not unexpected in a sewer, and the claim failed. I know of a case in which a man fell off a ladder when at work and developed a hemiplegia; medical evidence showed without any question the case was in reality one of encephalitis with hemiplegia, yet it was held to be an 'accident' that the 'germ' of encephalitis attacked the man while he was working.

If, then, these and similar illnesses are to be accounted 'accidents' within the scope of the Act, etiology becomes meaningless. It can always be maintained, apparently, that the arrival of pathogenic organisms in the tissues of the body is an 'accident' arising out of external injury, and our ideas on causation are, at a stroke of the legal pen, rendered obsolete.

WHAT IS MEANT BY THE CAUSE OF A DISEASE OR MORBID CONDITION?

As medical men, we believe in the doctrine of specific causes. We know the rules laid down by Koch in regard to the bacteriological origin of certain diseases. It may be taken as a good general rule—the more apparent causes for a disease, the less likely is any one of them to be specific. As a single illustration, take disseminated sclerosis. Because we do not yet know its actual cause, etiological speculation has ranged, as a fact, from the inhalation of pollen to an *affaire du cœur*. Once the cause is discovered—we may be near it at last—these varying hypotheses will all go by the board.

The whole tendency of modern research is oriented in the direction of specific causes, both in nervous and mental disease; hence it is shown from time to time that many 'causes' are clearly spurious. No one now but believes that syphilis is the essential and adequate cause of tabes and of general paralysis. Nevertheless, we continue to suppose that some factors, not truly causative, are contributory. We suppose that the mere arrival of the spirochæte in the neuraxis is not sufficient; we are taught that strain, stress, exposure, chill, other

infections, trauma, etc., cause the specific organism to 'light up' by reducing resistance, but such views are often unsatisfactory and always more or less speculative. In any case, it introduces an element of confusion, in my opinion, to claim such factors as contributory causes, since in innumerable instances the diseases mentioned develop without any such additional etiological elements. Neurosyphilis is no more and no less an infective condition than typhoid, pneumonia, diphtheria, dysentery, etc., but I am not aware that—to take the factor that now concerns us—typhoid or dysentery is ever considered traumatic in the sense in which the term has been applied to cases of neurosyphilis. For none of these infections will we consider trauma a cause, in the real meaning of the word, and there is no reason why the same argument should not apply to an infection of the nervous system.

In the case, therefore, of organic and of functional nervous disease, we ask ourselves whether the *rôle* supposedly played by trauma has not been greatly exaggerated, whether there is not a true cause for tumours and degenerations as there is for the infections to which the nervous system is liable, and whether there can be any real differences, etiological speaking, between nervous disease and that of any other of the somatic systems. Do we hear as much of a blow on the chest producing bronchitis, of a blow on the abdomen producing appendicitis or malignant gastric disease, as we do of a knock on the head causing a stroke, epilepsy, cerebral tumour, or what not?

I have never been able to understand why it is alleged so persistently of the nervous system in particular that trauma initiates morbid neural processes, and can only imagine that our ignorance of much of nervous etiology gives rein to speculation in a way scarcely applicable in respect of other parts of the body.

I do not, of course, attempt to deny for one moment that accidents, such as a fracture of the skull, a fracture-dislocation of the spine, wounds and penetrating injuries of many different kinds, will give rise directly to organic nervous symptoms; I am on the present occasion restricting myself to nervous disease in the ordinary acceptance of the term, and I maintain that, in proportion as etiological knowledge augments, such disease will be ascribed with less and less frequency to trauma as a *vera causa*.

TRAUMA IN ORGANIC NERVOUS DISEASE.

1. **Cerebral Tumours.**—Cerebral tumours are often attributed to blows on or other injury to the head. In his monograph on the subject, Mendel cites four personal cases and two from other sources, in which the question of a possible connection between a head injury and the development of an intracranial neoplasm arose. None of them is entirely convincing, though they are quoted as fulfilling the rules laid down by

that author as follows : (a) the individual concerned must have had no symptoms before the accident ; (b) other etiological factors must be wanting ; (c) the injury must have involved the head itself ; (d) a certain relation in time (not specified) must exist between the injury and the commencement of the tumour symptoms, which subsequent operation or necropsy must not prove to have been erroneous.

Fortunately, the recent war has provided us with an experiment on a colossal scale, since in its course head injuries have occurred by the thousand. It is already nine years since gunshot wounds of the head began to come under my professional notice, and I have been seeing such cases ever since, as have many others. Abundant time has elapsed for the appearance of consecutive intracranial neoplasms, yet out of very many scores of personal cases I have never seen a single instance. The only practical conclusion is that the assertion that trauma may originate cerebral tumours is unjustified and obsolete.

A recent case from civil life may now be given :

A male adult, a labourer by trade, one morning fell some 2 feet only on a scaffolding, and landed across a plank, which bruised the inside of his left thigh at its upper part. He was shaken, but resumed work an hour or two later, though in the afternoon he felt sick. The same evening he had two fits, which were definitely right-sided and Jacksonian in character, beginning in the right side of the face, and temporarily interfering with speech. Progressive weakness of the right side ensued some weeks later, and after about three months he was admitted to hospital under my care, when the general symptoms of cerebral tumour, and the localising ones pointing to a left fronto-rolandic site, were definitely present. Operation was undertaken, but the patient's condition was not improved, and he died shortly afterwards. At the necropsy a left frontal glioma was found, occupying mainly the posterior ends of the second and third frontal gyri.

The widow sued the employers for compensation, asserting that the tumour was caused by the fall of 2 feet on to the crutch. In court two medical 'experts' declared that a fall on the coccyx might by concussion and dispersion of spinal fluid cause or initiate a morbid neoplastic process in the brain, but were unable to cite any experience objectively proving their speculation. On behalf of the employers I gave the argument from war experience as indicated above, and had the satisfaction of finding that the court appreciated the force of the contention.

2. Disseminated Sclerosis.—Trauma is not infrequently stated to be one cause of disseminated sclerosis, but since all the present available evidence suggests that the disease is infective in origin it cannot possibly be assigned to trauma, in the sense of external injury. It is gratifying to find in the recent investigation of this affection by the Association for Research in Nervous and Mental Diseases that Dr. Lewellys F. Barker excludes trauma as a true cause, though he says injury may on

occasion 'light up' a latent case, or exacerbate one that is already manifest.

The evidence of the war, further, is quite opposed to any traumatic etiology. At Fulham Military Hospital, of some 900 consecutive neurological cases seen by me from 1916 to 1918, only two were examples of disseminated sclerosis, and in neither had the patient been wounded, or blown up or gassed, or undergone any other injury. The relative rarity of cases of the disease during the war has already been commented on by more than one observer, yet nervous trauma was extremely common. Since 1919 I have had charge of a pensioners' clinic at the National Hospital, Queen Square, London, and hundreds of cases of war nervous disease have passed through my hands. Among these have been only six cases of undoubted disseminated sclerosis, and of the six the following are the only two where a possible connection with trauma comes up for consideration.

F. R., fighting at Cambrai in 1917, was blown up and gassed at the same time. He had no mask on at the moment, was badly burned over the back, and was unconscious for a brief period. Three months later, when in a military hospital in England, he began to notice unsteadiness in his legs, and to have some slight defect in articulation. The legs became numb and cold for a time, but he had no bladder difficulty. When he came under observation in 1923 he showed the usual symptoms of disseminated sclerosis in moderate degree.

While there is no history of any previous symptom, and no evidence of the action of any other factor than that of trauma, it is clearly inadmissible to hold that trauma caused the disease; the most to be said is that it may have 'lit up' a latent morbid process. In hundreds of instances of identical trauma (gassing and concussion) no such organic disease has made its appearance.

W. S. was in the R.A.F. during four years of the war. He had crossed the English Channel some 200 times in the course of his flying experiences. Later, towards the end of the war, he was shot down in air-fighting on three separate occasions, sustained numerous minor injuries apart from the shock of crashing, but was never rendered unconscious. Four months after the last of these, he first complained of double vision and of tremor in the limbs. On his coming under observation in 1923, the symptoms and signs of disseminated sclerosis were exhibited in moderate degree.

Here, again, it must be allowed that the injuries, such as they were, can at the most have done no more than possibly accelerate the development of the disease; it might, indeed, be contended with perfect justice that they played no part at all in its evolution.

In view of the rarity of the disease amid the welter of war injury, one is naturally more than ever disinclined to allow its association with

trauma in civilian life. Nevertheless, as every neurologist must know, such cases make their appearance at intervals. Two personal instances may be selected by way of illustration.

N. B., a young woman in domestic service, was cleaning a large and heavy mirror over a mantelpiece when it somehow became detached and fell on her right forearm, breaking as it fell. She felt a good deal of pain in the arm, which was bruised but not cut. In the course of a day or two numbness followed the pain, and weakness of the muscles of the hand and forearm. The case was diagnosed as hysterical dropwrist by the doctor under whose care she came, but as the condition, though improving, did not clear up entirely, I saw her some six months after the accident. I was then struck with the fact that, on grasping, the wrist deviated unmistakably to the radial side, indicating relative weakness of the ulnar groups—an organic symptom. The patient was put on steady treatment and kept under observation. Some six months later an extensor response was obtained on the right side, and three months afterwards on the left. In a word, the subsequent course of the case proved the disease to be disseminated sclerosis.

I will admit candidly that in this case I have always felt hesitation in excluding the *rôle* of the initial trauma, for the condition continued without a break to the development of definite organic signs, yet here also it is impossible to believe that a single trauma of the description given can by itself cause a progressive nervous, disseminated disease. That scattered islets of sclerosis of a quite peculiar kind in the neuraxis can be *caused* by a blow on the forearm is a proposition which cannot be entertained. Nevertheless, if we dismiss the hypothesis of mere coincidence, we are compelled, in fairness, to suppose that a latent organic nervous disease was 'precipitated' by a relatively severe peripheral injury, incapable though we may be of framing any reasonable theory as to how such an injury might act.

I was consulted not long ago in the case of another young woman who happened to be sitting in a public restaurant when part of the metal framework of a revolving electric fan on a shelf above her became detached and fell on her head from a height of about 6 feet. She undoubtedly received a severe shock and fright, was dazed and faint, and later on in the day was sick. She was at no time unconscious, but complained of much headache and giddiness. Her hat had kept her head from being cut, and there was no obvious evidence of direct injury.

When she was seen in consultation some weeks later she presented unequivocal signs of disseminated sclerosis! This was surprising enough. The case, fortunately, was settled out of court, for as the girl declared she had been perfectly well up to the time of the accident, it would have been impracticable to dissociate it and the symptoms she complained of when examined. Nevertheless, medically and scientifically speaking, it would be impossible to prove that, in the absence of other etiological factors, the injury caused the disease.

This latter case aptly illustrates the difficulty confronting the physician, since as the patient was not examined by a neurologist before the accident we do not in fact know that objective signs could not then have been found ; further, when monetary compensation looms in view, it is a trait in human nature to suppress anything which might tend to reduce the amount ; and again, as spontaneous remissions occur in the disease under discussion, so do spontaneous exacerbations.

3. Neurosyphilis, including Tabes and General Paralysis.—For years controversy has raged on the question of tabes and general paralysis arising from or originated by trauma, but not, I venture to think, quite so much now as in the years before the discovery of the spirochæte in the cortex and the spinal fluid of neurosyphilitic cases. Various cases are on record, however, where an apparent connection between trauma and the appearance of symptoms of general paralysis, for example, is sufficiently impressive.

In the absence of spirochætal infection no one, I suppose, will now admit that trauma *per se* can cause neurosyphilis in any of its manifestations. Can it actually initiate a morbid process on the part of the spirochæte, in the sense that the latter otherwise would have remained for ever latent and innocuous ? Put thus, the question can scarcely be answered in the affirmative, yet who shall say that a direct negative represents the only possibility ? It is the sort of question difficult either to prove or to disprove. Take a recent case.

A guard on one of our English railways was apparently in normal health, in so far as ability to perform his duties was concerned, when his train, standing on the line, was run into by a second train, and he was knocked from one end of his van to the other. There were no visible signs of injury of any kind when he was examined the same evening, complaining of 'shock.' He resumed his duties very shortly after, yet within a month was found to be making mistakes, to be waving his flag wrongly, to be getting confused and incapable of explaining his erratic behaviour. Further precise investigation revealed the early symptoms of general paralysis, and within six weeks of the 'shock' he was under certificate in an asylum.

I submit that in this case we cannot say that the injury did anything whatever by way of aggravation of an already existing, but unrecognized condition, or by way of 'lighting up' a morbid process which was in abeyance. In view of the innumerable instances of the development of general paralysis without any trauma, we cannot suppose the speculation to have any degree of likelihood. In view, further, of the known remissions and exacerbations of the malady, the onus should rest on those representing the patient to prove, in a compensation case, that the condition was not due to an ordinary exacerbation from within. The modifications arising in the course of general paralysis are the result of intrinsic, not extrinsic, factors.

The so-called time factor is, doubtless, a somewhat elastic element in an alleged traumatic case. My contention is that unless it can be proven that a commencement of the symptoms of general paralysis, or of tabes, actually arises within forty-eight hours, say, of an accident, or that they are augmented within the same period, on the evidence of the same physician as has seen the patient on previous occasions, it is illegitimate to argue for a causal relation between accident and symptoms. In genuine trauma of the nervous system, do we not agree that the symptoms arise practically at once? Even in the case of delayed traumatic apoplexy, on which so much has been written of a contentious nature, is it not agreed that there is continuity of symptoms, however slight they be, from the very time of the injury? Since there must be some limit to the interval of time elapsing after an alleged injury ere symptoms appear, I suggest that in the case of organic nervous disease it should be restricted at the widest to one week. The neuropathologist is well aware that morbid histological processes make their appearance at a much shorter interval after a genuine trauma than one week.

4. *Other Organic Nervous Diseases.*—Time will not allow me to deal with other organic nervous conditions such as syringomyelia, progressive muscular atrophy, ascending neuritis, etc., in various clinical examples of which it has at one or other time been claimed that trauma has originated the process. The same claim has been made during the war in respect of traumatic dementia præcox and other forms of mental disease.

My general standpoint is that I am unable to understand how a single trauma can cause a progressive neural degeneration or abiotrophy, still less, of course, a progressive neural toxi-degeneration. The former appears to me a physical impossibility. We are ignorant, it is true, of what the 'span of life' of a neuron system is in terms of biochemistry; we do not know under what circumstances the spinal degeneration of a Friedreich's case makes its appearance, but I am convinced we should seek the solution in the biochemical field of intrinsic neural 'life and death,' and not glibly assign progressive degenerative processes to the action of a 'shock'; even assuming a concussion so bad as to produce, on a small scale, fragmentation of myelin, we know, as a histological fact, that scavenging takes place very promptly and that neural regeneration is equally sure. This being so, the view that trauma may on occasion cause neural abiotrophy of a progressive character is in my opinion opposed to the facts of neuropathology.

TRAUMA IN EPILEPSY.

It has been asserted almost universally that trauma may cause epilepsy; I have never been able to understand why. Nothing is more

common than for the physician to be told that the epilepsy of his patient is the result of a fall from a perambulator or of some other blow on the head. Were this in actual fact the real sequence of events, then every child should be epileptic, for which of them has not at one time or another had a knock on the head? The persistence with which epilepsy is attributed by parents to a fall or blow on the head—often trifling enough, too—is amazing, in face, it may be, of an overwhelming history of family nervous instability.

War is the great experimenter. In a recent communication Aldren Turner has given figures derived from the Ministry of Pensions in England. Of 18,000 cases of gunshot wounds or other injuries of the head occurring in warfare, some 800 patients have subsequently developed epilepsy, i.e., rather less than 5 per cent. The extreme importance of these figures will at once strike the reader. The percentage is approximately the same, according to Turner, as was observed in the Franco-Prussian War. Another set of figures is given by Holmes and Sargent; of 610 cases of gunshot wound of the head, observed from two to eighteen months after the injury, thirty-seven, or 6 per cent., developed epilepsy. This percentage, without doubt, is much less than might have been expected if trauma *per se* has in reality the effect too readily attributed to it by parents and, indeed, by many medical men.

By way of amplification, it should be remembered that thousands of individuals with severe war injuries to the head—with fractures of the skull, laceration of brain tissue, paralysis, and all the rest of it—have not developed epilepsy in any shape or form. I associate myself entirely with Turner when he insists that “it is difficult to avoid the conclusion that something more than local tissue alterations is requisite for the production of the seizures of traumatic epilepsy, and the determining agent, in my opinion, is an inherited or inborn constitutional predisposition to nervous instability and epilepsy.”

It seems difficult to come by statistics as to the family history and individual make-up of those soldiers who have suffered from traumatic epilepsy the result of war injuries; R. G. Gordon has obtained evidence of a neuropathic predisposition in 75 per cent. of his cases; in a series of my own, from the pensioners' clinic at the National Hospital, Queen Square, I have found similar evidence in 80 per cent.

We are not, therefore, justified in admitting more than that in a small minority of war cases of traumatic epilepsy the injury seems to have initiated the disorder in individuals previously healthy and unimpaired by heredity; in the great majority the existence of the constitutional factor cannot be ignored or explained away; finally, the percentage of epileptic production among the head-wound cases is itself so small that it is abundantly evident the predisposition is much more important than the head injury. So clear is this that we should

be chary of certifying that a head injury has 'caused' traumatic epilepsy in any single case; if the predisposition is obvious, its importance should be emphasized, and only when we have exhaustively scrutinized the family record with a negative result can we allow trauma *per se* to be the *causa causans* of the condition. In this respect I wish to direct attention to the frequency with which asthma is reported in family histories otherwise supposedly normal.

A young officer in the army consulted me recently because of three 'attacks' he had had in the last six months. From the description given it seemed certain they were epileptiform in character. I found no sign of organic nervous disorder or of rise of intracranial tension. The 'attacks' were attributed by the parents to two tosses the patient had taken when steeple-chasing; in the first instance, he had fallen on the back of the head and been unconscious for two or three minutes. An *x-ray* examination showed no definite changes in the cranial vault. I was assured confidently that the family history was in every way normal, but on further questioning I elicited the fact that the patient's father had suffered from asthma for years, and so had a paternal uncle.

To my way of thinking this strain of neurosis was much more significant than the trauma, such as it was, and I cannot say that the latter caused anything at all. The interval between the second 'toss' and the first fit was several months. While it would be equally difficult to prove that the head injury had no connection with the subsequent fits, the conclusion is that the experiences of war cases must lead us to attach prime causative significance to the constitutional make-up of the individual.

TRAUMA IN FUNCTIONAL NERVOUS DISEASE.

When we approach the subject of the rôle played by trauma in neurosis-formation we enter at once on thorny ground. We find ourselves lost in the confines of a vast and inchoate group of morbid affections variously described as traumatic neurasthenia, traumatic neurosis, traumatic hysteria, etc. There appears to be neither etiological, symptomatological, nor even terminological agreement in respect of this class.

As a single illustration, we may select the question of spinal concussion. In a fairly long experience I have seen all sorts of conditions classed as spinal concussion, or, alternatively, as 'spinal irritation,' whatever the term may mean. It is well that those who give certificates to state that their patient has sustained spinal concussion should have a real idea of what the symptoms are and to what extent, once more, the war has assisted us in this connection. Concussion cannot heighten function; it must reduce or inhibit it. In cases of genuine

spinal concussion we find diminution or loss of power in the legs, with diminution or loss of the deep reflexes, involvement in greater or less degree of the organic reflexes (rectal, vesical, sexual), but with little or no impairment of sensory conduction. The plantar reflexes may be also abolished for the time being or may be either in flexion or in extension.

Corroboration of this clinical picture has been obtained experimentally by various workers, notably by Alan Newton, who made a series of experiments with Sir Victor Horsley in 1913, on cats and apes. The spinal theca was exposed by preliminary laminectomy, and on it were dropped, from different heights, various weights. So sensitive is the spinal cord when no longer protected by the vertebræ, that the effect of the dropping of a 50-grm. weight from a height of only 1 cm. was sufficient to abolish conduction in the cord. It produced no visible effect on the surface of the cord, yet, pathologically, small hæmorrhages were to be found usually in the basal region of the posterior horns or in the posterior grey commissure, with scattered swellings of axis cylinders and disintegration of myelin sheaths. Newton was able to show experimentally that sensory conduction may remain after concussion that has quite abolished motor conduction. Compression for short periods (two minutes or more), produced by gently placing a 50-grm. weight on the cord of a cat, brought about the same clinical and pathological result.

When, therefore, we speak of spinal concussion in connection with accident cases, we can only use the term advisedly if the clinical picture is approximately as above described. I venture to say that in many of the traumatic cases of civilian life, facilely certified as spinal concussion, the diagnosis is erroneous.

A young man fell down a lift, some 15 or 20 feet. He was badly dazed but not unconscious, and by good luck no bones were broken. Taken to hospital, he was not detained; his reflexes there were noted as being present. He complained of pain in the back, at about the level of the last two dorsal vertebræ. Examined the same evening by his own doctor, and again the next day, his chief complaint was of this pain. Eventually he claimed compensation and was duly certified to have had spinal concussion and to be suffering from the effects of it—this was not less than about four or five months after the fall. When I examined him subsequently my *questionnaire* was directed to the points of importance outlined above, and I ascertained that there had been no reduction or abolition of power in the legs originally and no impairment of rectal, vesical, or sexual mechanisms. It was known, further, that the deep reflexes were active within an hour of the fall. In these circumstances I gave it as my opinion that there had been no spinal concussion and that therefore the patient could not be suffering from the effects of it.

If the injury is of so mild a character as not to give rise to recognizable signs of this character, we have at present no certain means of

detecting minimal degrees of commotion objectively. We do not know that they give rise to symptoms of a pathognomonic kind; we cannot point to any single, unequivocal, objective sign as of constant value. As far as my experience goes, I cannot distinguish an organic basis slight or minimal in degree from a non-organic basis in respect of motor weakness and changes in the reflexes, though it is usually simple enough to determine the hysterical or neurasthenic factors otherwise. Thus the statement that the knee-jerks, for instance, are 'exaggerated,' is valueless if it is thought to aid in differentiating between organic and functional cases.

There is, however, another aspect of the question which deserves fuller consideration than it commonly receives. Assuming a slight degree of spinal concussion or commotio, a moderate amount of myelin fragmentation, even minute hæmorrhages, we must not forget that within a comparatively short time this tissue-débris is scavenged away, while active regeneration is a normal reparative process. It follows as a definite histological fact that in slight or minimal degrees of spinal commotio any tissue-change in the organic sense that has occurred will vanish in due course and the cord be histologically normal again. But what of the patient's symptoms? What of the subjective complaints?

It stands to reason that the presumed effects of the spinal trauma having been countered by reparative processes, the cause of the symptoms is no longer in existence. Yet every clinician knows how months and years after a slight 'spinal shock' the patient will still complain of the same old pains, weakness, shakiness, and what not. It is obvious, then, that in many instances the condition changes definitely in the course of time; it is perpetuated by other pathological mechanisms than those originally implicated. If, to give the patient the 'benefit of the doubt,' we admit the possibility of mild spinal shock amounting to concussion, we are clearly prevented from allowing its effect to continue quite indefinitely. It is, in fact, actually the case that in innumerable instances the accident is recovered from, yet the symptoms remain. In Sir John Collie's words, such "claimants do not suffer from the accident but from the memory of it." It is the bounden duty of the medical examiner to insist that there is, in the cases of which I am now speaking, a time limit for the duration of any symptoms presumably due directly to minor spinal commotio. The conclusion to which consideration of all the circumstances drives us is that a neurosis supervenes, and that in the great majority of cases this is prolonged by (1) conscious and (2) unconscious motives.

That a process of this description usually occurs has long been recognized; the Germans speak of a 'Rentenkampfneurose'—a 'fight-for-compensation' neurosis. The war has shown us a thousand

times how the genuine effects of concussion, cranial or spinal, pass off eventually, with a complete return to the normal, but if they persist, in the absence of evidence of objective change, it may be taken as an infallible rule that the condition has ceased to be one of concussion.

1. Conscious Motives.—Possibly, some have a natural hesitation in admitting that human nature is so constituted as to be prone to make financial profit out of personal trouble, yet the fact remains. Every experience points in this direction. Let me give the figures of three recent railway accidents on one of our biggest English railway systems.*

I. Accident, January, 1915. Number of passengers travelling in the two trains that collided, 693. Number of deaths, 14. Number who actually received visible and obvious injuries, e.g., wounds, fractures, true concussions, 54. Number claiming compensation for 'shock,' 'neurasthenia,' etc., who were medically examined, 275. Number who claimed similarly on account of 'shock,' and whose claims were settled without medical examination, 180.

Total claims, 523, out of 693 passengers; total claims for 'nerve shock,' almost 66 per cent.

II. Accident, October, 1922. Number of passengers travelling in train, 73. Number of deaths, *nil*. Number of actual and visible injuries, 3. Number who claimed compensation for 'shock,' 'neurasthenia,' etc., 41.

Total claims for 'nerve shock,' over 56 per cent.

III. Accident, November, 1922. Number of passengers travelling in the two trains involved, 183. Number of deaths, *nil*. Number of cases of visible and obvious injury, 27. Number of claims for 'shock,' 'neurasthenia,' etc., 87.

Total claims for 'nerve shock,' over 42 per cent.

Taking these together, the average of three railway accidents, only one of which was severe, gives a percentage of 55 of those involved claiming damages for 'neurasthenia,' i.e., one in every two passengers apparently suffered from traumatic functional nervous disease. Can we seriously believe that every other individual taken at random is neurotic? During the war, millions of soldiers were submitted to precisely the same warfare, the same stress and strain, the same shell bursts, the same bombardments; did 55 per cent. of them develop 'shell-shock'? Every one knows that the percentage was far less than that. The only conclusion we can draw, I submit, is that in many instances conscious motives connected with monetary compensation have a great deal to do with the simulation and exaggeration of symptoms almost invariably noticed among patients reputedly suffering from traumatic neurosis. The question of money to be obtained colours the whole situation, as the railway figures quoted prove up to the hilt.

2. Unconscious Motives.—We may take the facts as stated by

* For these figures I am indebted to my friend Dr. Grant MacMahon.

other observers, whose authority will not be questioned. In the matter of miners' nystagmus, the recent Report published in England, and contributed to by the late Dr. W. H. R. Rivers, proves conclusively how potent is the unconscious motive (if it is in reality unconscious). Since the introduction of compensation, we learn that the prevalence of disabilities from miners' nystagmus has vastly increased; monetary considerations (for what other factor is there?) have led to the production and to the prolongation of disability to work.* Since the passing of the Workmen's Compensation Acts in England, functional traumatic cases have been ever increasing. The victim of an industrial accident is now provided for by legislation; as Collie says, the accident is often regarded as a valid excuse for living at the expense of the former employer.

I might here cite case after case in which I have been personally concerned and in which simulation and exaggeration of symptoms have been grotesquely obvious. In each of these, medical certificates have been furnished by fellow members of the medical profession, substantiating the impossible claims of the individual concerned. This is the aspect of a serious situation that one must regard with concern. Through ignorance, or from press of work and shortness of time for examination, or because of personal friendliness with the claimant, the doctor gives a certificate to which exception may justifiably be taken, or continues to furnish them at intervals, even years after the genuine effects of the trauma have vanished. And let it not be imagined for one moment I am criticizing the general practitioner; on the contrary, I apply to myself the same comment on the urgent need for scrupulous care and faithful scrutiny of all the facts of every case. A certain individual, in connection with one of the railway accidents above referred to, went to a well-known consultant in London and stated he had been in the train and received a severe nervous shock. A certificate was given to the effect that he was suffering from 'traumatic neurasthenia,' and on the strength of this certificate he was awarded £200 compensation by the railway company. It subsequently transpired he had never been in the accident at all! The moral is plain enough.

* Since this paper was written, we learn from the Second Report of the Miners' Nystagmus Committee of the Medical Research Council that "not only the number of fresh claims but the average duration of incapacity has risen enormously, facts which . . . have no relation whatever to the actual prevalence of the disease, but solely to the facilities for obtaining compensation" (*Lancet*, 1923, ii, 293).