TRAUMA AND THE NERVOUS SYSTEM *

With Particular Reference to Compensation and the Difficulties of Interpreting the Facts

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Introduction.—For many years in hospital and private practice one has become increasingly conscious of the importance of this subject to the patient and to his doctor, to the specialist and to the lawyer all in their different roles. During the war experience in many respects became more crystallised, and it is my purpose to explain some of the problems with which one has been confronted in the hope thereby of stimulating interest.

How much is it given to anyone to know what is in another man's mind? This question obtrudes itself repeatedly in one form or another in the experience of all clinicians when faced with the evaluation of an illness, or confronted in court by the opposing counsel. So many of our views are purely a matter of opinion, but fortunate indeed that it is so since, if medicine was an exact science, it would be an unpleasant if not intolerable profession. Facts there are in abundance, indisputable and defensible although frequently open to a variety of interpretations, or more correctly misinterpretations, a circumstance that makes us beloved if not necessarily admired by the lawyer and the journalist, with the one for the joy of litigation, his *raison d'être*, with the other for the constant supply of spicey copy, his bread of life. It is pertinent to ask ourselves up to what point we do practice an exact science and beyond that mark how do we handle the art. Clearly there are many sets of circumstances which are capable of only one interpretation, in each all must be agreed, a patient has a cold in the nose, a cyclist falls and breaks his leg, a person is blind or deaf. These things are obvious to the tyro no less than to the expert, but a more difficult elucidation requires knowledge and experience, factors which come with interest and opportunity, a sliding scale of value that by no means depends only on exertion. It involves the quality of competence. A sharpened wit may rapidly furnish an explanation for obscure aspects of the case which convince some but perplex others. Who are convinced and who are not? Is it a matter of preference only?—or is it preference based on reasonableness, a reasoned preference for what is attractive? The process of thought when broken up into its components leaves nothing to be explained. This is, it seems, the crux of the matter. The expert witness by accepted usage is one whose expertness is in direct ratio to his knowledge and

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the facility with which he expounds his facts and avoids confusion with theories. Thus always there are degrees, the element which brings out the personal equation and confounds the critics by its very persuasiveness. This, however, is subject to a test—the test of time, a salutary experience for those who approach their task conscientiously, but too often overlooked by others who refuse obstinately to learn their lessons. Would we do and say the same again is a recurring question, but too often the complacent answer is affirmative, bringing consolation to the doubting mind. Is it surprising that we grope so much in the darkness when the technique of satisfaction brings its inevitable reward of solace ?

Few of us have much training in court work. It is avoided by the young doctor through fear, and shunned by the older on the plea of insufficient time, and yet in the mind of both is the haunting thought that his ignorance may be exposed to ridicule, a healthy sign in so far as it indicates a realisation of limitation, but none the less it is a serious omission since each appearance is a yardstick by which can be measured the ability of clear thinking and honest deduction, an opportunity which no other part of our work affords. In the last resort a situation should be created for each problem with which we are confronted in order that zeal and prejudice do not mask the facts of the case. Such an attitude of mind at once separates the honest practitioner from the unscrupulous quack and charlatan who have far too long enjoyed a vogue and the doubtful advantage of material gain at the expense of a credulous and unwary public. That it cannot continue is obvious to anyone who stops to ponder the trend of nationalisation and its effects upon those who exploit the mental and physical weakness of their fellow men. Let us beware lest we ever stoop to imitate this undesirable character.

Patients inevitably feel a bias in favour of trauma as the important factor in explaining the causation of any ailment which is not readily accountable for otherwise ; and understandably nervous manifestations fall prominently into this category, since even now many of them are imperfectly understood let alone satisfactorily accounted for in lay language. An infection may seem clear cut, a meningitis, a poliomyelitis ; yet so often an attendant fall seems inseparable to the patient's friends from the sequence of events, indeed so convincing a factor that often skilful arguments leave a doubt. A doubt in part due to our own uncertainty in many instances. Need we be so uncertain ?—or is it merely a desire to be scrupulously fair, fair that is to the individual for whom a favourable medical opinion may mean a reward in cash, commonly called compensation. A study of nervous infection is pertinent now that more is known of the mechanism whereby a virus acts, and the realisation that this variety of disease at least is self limiting. For example in poliomyelitis during an epidemic only a proportion of the victims develop the disease in an indisputable form ; others, although undoubtedly affected by inference

from all the circumstances, yet recover completely owing to some unrecognised resistance which allows a general reaction to stop short of calamitous paralysis. This must be a process dependent upon inherent factors of constitution and well-being favourable and unfavourable, but also influenced by matters external of which who can say that trauma is not one. While the virus is exerting its evil influence and the patient is little if at all affected, there must be a point at which the outcome is in doubt and at which the outside effects are all important for good or bad. Thus if we imagine these points in the process at any one of which an accident, less or more, may occur, its results manifestly will differ according to the timing—little or nothing if early or late but overwhelming should it happen when the natural process is in doubt. We cannot escape the complexity of this problem, and nothing but added confusion accrues from attempting to simplify it. One solution would be to allow that any accident at any time during the accepted incubation period was held responsible, but that could never be allowed since clearly it would open the door to a greater degree of unscrupulous evidence than exists at present. A fair and more honest attitude is to state the facts clearly, a matter by no means as easy and simple as it sounds, and to leave it to others to make the judgment.

The illustration of poliomyelitis is not peculiar, the principles implied being equally applicable to a wide range of conditions, many much more common if less dramatic, including almost all forms of neuritis, brachial, sciatica and the rest.

Sometimes, indeed often, one despairs of litigation. It is endless and often unsatisfactory, but so too are examination tests which have been in vogue for a much longer time and always had their critics, yet remained unreplaced for want of a better system. So it is with litigation, the real pity being that it has become increasingly wrapped up with finance. One reflects with wonder at the consternation of the legislators *circa* 1897 could they but have had a peep of the outcome through half a century of the laws they were formulating. It is appropriate to consider briefly some aspects of the Workman's Compensation Act in its evolution and what it has come to imply in medicine to-day. Familiarity has led to indiffernece, and most of us are ignorant of how far the Acts have been extended and are still extending. The present position certainly affords material for reflection. Quoting Sir John Collie,[3] it was calculated by the Home Office, in affording information as to the probable effect of the passing of the Act of 1897, that 150,000 accidents a year would fall within its scope. But in the seven principal industries for which accurate statistics are obtainable, the number of cases of accident in which compensation under the present Act was paid in 1930 amounted to 461,130; in addition there were 2303 uninsured employers who reported that they were paying compensation in respect of accidents sustained by their employees.

Making due allowance for the extensions made by the subsequent Acts, and the possible increase in the numbers employed, the disparity between anticipation and realisation is striking. It leads to the conclusion, either that the framers of the estimate had been unduly optimistic and badly informed, or that they had failed to attach sufficient importance to the psychological and other effects of the passing of such legislation. Given in figures the comparison between then and now is not a question of an increase of thousands of cases but of hundreds of thousands, and not a question of tens of thousands of pounds but of millions.

What are the reasons for the large number of cases now coming under the Workman's Compensation Act? An undoubted one, pertinent to my subject, is that whereas in the original Act no consideration was given to disease, a constant chain has been linked ever since by the accumulated results of Court cases which pass into law an ever increasing range of possibilities. Altered conditions too are playing their part; for instance there has been a great increase in mechanisation, and processes have been considerably speeded up, which might be alleged to cause more casualties. On the other hand, of 113,249 accidents recorded some years ago before the war, the majority were due to some failure in the human element and not to the machinery. There have been increased Statutory and other provisions framed to avoid accidents, and many operations are now safely performed by machinery which in the past were done by manual labour and put an enormous strain on the workmen engaged, rendering them liable to hurt. Probably, too, some of the increased number of accidents are due to the carelessness and slackness which some observers have noticed among many of the younger generation of workers. The door of compensation being once opened it has since received so many pushes from the Legislator and the Bench in interpreting what is understood to be the intention of the Acts, that it is now almost as wide open as even the extremists could desire.

Trauma in Organic Nervous Disease

The approach to the organic side of this subject is perhaps best made by selecting in series a few of the more familiar conditions and bringing out the essential points as they arise.

Disseminated Sclerosis.—During the hundred years or more since disseminated sclerosis was first recognised as a pathological entity, the question of trauma keeps cropping up among the etiological factors as indeed it must always do in any organic disease whose elucidation proves difficult. Even before Charcot's wonderful description of the symptomatology and pathology of this condition an interesting paper by Leyden in 1863 emphasised the causal importance of trauma while linking to it damp and cold, mental stress and possible preceding infections. But by the start of this century the opinion was established that the principal change was a neuroglial

hyperplasia, and when pathological research was aided by new staining methods revealing more precisely neuroglial reactions it became customary to regard disseminated sclerosis as an infective disease. However, in spite of the enormous volume of work done in the hunt for a causal agent, no proof has been forthcoming, and this notwithstanding our greatly increased knowledge of virus and other infective processes which show many clinical features of a similar kind.

All through this long time theories of the relationship of accident have had their adherents, slightly so in the matter of causation, but more persistently in regard to aggravation, a question of prime importance in a malady which is prone to run so erratic a course of ups and downs, remissions and exacerbations. Quite obviously the distribution and incidence which has been carefully worked out statistically reveals an overall feature in geographical and racial incidence which precludes so chance a cause as accident as a prime factor. For instance, the disease is very common in Switzerland, whereas in the United States it is much lower than in Britain. Indeed outside Europe it seems to be rare. The geographical distribution can be carried further and in many instances pinned down to actual towns and localities. I remember well many years ago in the out-patient department of Professor Bing in Basle marvelling at the enormous number of these cases and learning from him that 3 or 4 out of every 5 patients with organic nervous trouble suffered from disseminated sclerosis. Again at one time the belief was widely held in this country and elsewhere that farm workers were especially prone. Such an idea, long disproved, is merely mentioned to show that environment and occupation in addition to racial distribution have attracted attention of workers in this perplexing field ; also examples of familial tendency are fairly numerous in the literature and is now unquestioned.

These reasons, very briefly mentioned, are perhaps chief among those which account for so little attention being given nowadays to trauma as a predisposing cause of disseminated sclerosis. No one aware of the facts would be likely to claim that trauma was essential or even common, but equally it is certain that in an individual case its consideration demands the most careful scrutiny and application of reason to all the known facts, clinical and otherwise. It would be rash to deny the possibility of a causal relationship between trauma and disseminated sclerosis and perhaps equally rash to assert it, as witness this example :—

Female (40). Fell seven years ago, damaging right hip but breaking no bones. Never walked without stick and great difficulty thereafter. Always assumed by herself and everybody else to be due to accident, but examination only recently showed indisputable signs of disseminated sclerosis including pale discs, nystagmus, and an upgoing toe on both sides, the opposite as well as the injured. The injured leg muscles extremely stiff, but great limitation of movement at all joints (disuse).

This case brings out the following points: (*a*) Did the patient have disseminated sclerosis at the time of the accident? (*b*) Was the accident a predisposing factor? (*c*) Was it a coincidence? Clearly from the facts at least it can be assumed that one was merged with the other, since the accident was wrongly presumed to have caused so much incapacity. But equally it is thought to be inconceivable that peripheral trauma could by itself bring about such widespread central nervous change. Then did it "light up" a latent tendency? Here it is that we come to the core of the problem in this important disease.

In this connection the following case is of interest:—

J. F. (34). Diagnosed as disseminated sclerosis in 1936, quiescent until 1943 when an attack of dysentry (Sonne) aggravated symptoms for a time but never sufficiently to prevent him travelling to and from his work as a clerk each day, until January 1945 when a bus he was mounting started unexpectedly, throwing him to the ground. He was dazed and bruised around one eye but managed with aid to mount the stair to his house. After a day or two complete paralysis of the legs and much incapacity of the arms with general aggravation of all signs was manifest to the extent that he became completely incapacitated and is unlikely to leave his house again let alone follow a useful occupation.

In this case the relationship of trauma to the original illness cannot be in dispute, the onset having preceded the accident by many years, but obviously it is more than a coincidence that complete incapacity resulted at once so strikingly that a jury must be convinced of the connection. Indeed in this case the substantial claim was settled out of court. But we are interested more than merely in the application of lay reasoning to circumstantial evidence. What did happen to stimulate the pathological process in this man? Could it be that the disturbance broke down in some way the defensive immunity he had acquired by altering his state of nervous tension to the extent that the process became irreversible? or was it the actual bang of his fall that directly damaged nervous tissue? While preferring the former choice one is yet left unsure.

For over a hundred years a great deal has been written about the traumatic etiology of disseminated sclerosis. There can be no question that a number of cases have developed shortly after trauma. Wilson [8] states that the concept of a blow on the arm causing multiple plaques is untenable. But if emboli were proved to be the cause of the sclerotic plaques the statement would no longer be valid.

J. M. Neilson [7] quotes the case, similar to the one above, of a fireman who slipped down a long flight of stairs in a sitting posture, receiving thus fourteen bumps which jarred the spine. He appeared two weeks later with a fully developed picture of disseminated sclerosis, and averred that he was perfectly well before the accident.

The interpretation of this case as of nearly all, if not all, cases of related trauma and multiple sclerosis, is that the jarring may crystallise

or precipitate the disease but cannot actually cause it. Some authorities state that any acute disorder seems capable of starting the disease, but others deny the relationship. Until the cause of disseminated sclerosis is known and the pathogenesis understood, such discussion is largely devoid of meaning.

The medico-legal aspects of this difficult question can only be judged in each individual case. Russell Brain [2] enumerated the following possibilities for consideration :—

1. That the association is a coincidence.
2. That traumatic lesions of the nervous system may be mistaken for disseminated sclerosis.
3. That trauma may induce changes in the neighbourhood of pre-existing, but hitherto latent, plaques of disseminated sclerosis and so lead to the appearance of symptoms.
4. That a patient, after spending some time in bed as a result of the trauma, may manifest symptoms because he has lost the power to compensate for a defect, such an incoordination of the lower limbs due to a previously acquired disseminated sclerosis.
5. That the trauma (*e.g.* a fall) may be the result of pre-existing symptoms of disseminated sclerosis (*e.g.* incoordination).
6. That the trauma may produce a lesion of the nervous system (*e.g.* contusion) which may afford a locus minoris resistentiæ for the development of the virus of the disease, hitherto latent.

War experience might have been expected to afford some light, especially since the age group involved coincides closely with those most liable. During one period when the medical admissions to my hospital numbered upwards of 20,000 only 8 cases were diagnosed as disseminated sclerosis, and in only two of these was the question of trauma sufficiently obvious to be considered, although even then their experience was no worse than that of their immediate associates. And in one a pale disc proved that the incident occurred during the course of a previously existing disease. The subject was discussed at a clinical meeting attended by over seventy medical officers when no expression of opinion shed any very different light. A possible factor but not a cause.

EPILEPSY.—Of all conditions in which the role of trauma has been contested most hotly this is the one *par excellence*. Here cause and effect appear to be related most significantly in time and in space, a view-point which inevitably will impress almost all juries, especially those members who most fancy their ability to make logical conclusions. A young man hits his head or is struck ; the degree of violence varying from trivial to serious, and at a later date, after often months or even years, takes his first convulsion. The two are linked together and thereafter the label traumatic epilepsy is affixed with the result that

doctor and patient alike have the smug consolation and poor comfort that the condition has been diagnosed. Yet it is remarkable that relatively so few head injuries end in this way, and perhaps even more noteworthy that the more serious seem even less liable.

It is interesting to look back upon the observations of half a century ago and read in the clear and precise style of Sir William Gowers [4] what he has to say on this subject when analysing his impressive total of 3000 cases.

" Important among the exciting causes of epilepsy are traumatic influences, blows and falls on the head. To these, after the elimination of doubtful instances, 108 cases were due ; a third occurred under ten years, rather more than a third between ten and twenty. Males are afflicted by this cause more than females—63 to 45. This is clearly due to the risks of occupation, for in the cases commencing before ten the females were in excess. Between ten and forty the male cases in each decennium were rather more than twice the number of females. Over forty the only cases were in men.

" In four-sevenths of the cases the injury was a fall on the head ; in three-sevenths it was a blow. In most of the cases the patient was stunned for a time but in only a fifth did the first fit occur immediately. In the rest an interval elapsed ; in one-third of the cases the interval between the injury and the fit was more than a day and less than a week ; in another fifth the interval was between a week and a month, and in about the same proportion the interval was more than a month. It is certain that a blow or a fall may excite fits without causing any visible lesion of the brain, and all cases were excluded in which the present or past symptoms or the mode of onset of the fits made it probable that ' coarse ' changes had been produced."

What have we learned since this was written to modify or support the interpretations given ? For one thing a medical opinion and a legal one expressed about the same set of facts is by no means the same thing. The reason why this is so is fairly obvious. Law demands justice irrespective of the qualities of the individual and the concern. The doctor, on the other hand, finds it hard to be impersonal ; his vocation interests him in mankind *qua* man, but he may err either in his observations or in marshalling his facts.

A young man of 23 saw me recently in hospital on account of a major convulsive attack he had had a few days previously. Those who saw him were able to prove that it had been an epileptic seizure, the first in his life. Physical examination revealed no abnormality but his history was interesting. Three months previously while in the army he was on duty in Trieste during a riot when he had been struck on the side of the head by a bottle, momentarily losing consciousness and requiring a few stitches for a scalp wound but recovering so quickly that he was only detained in hospital for three days. A month or so later his normal release occurred and he returned home, having been discharged in Category A1, nothing untoward happening until the convulsion. There was no history obtained suggesting any family tendency.

At once we know from experience of numerous similar cases that this young man's claim for a pension will receive sympathetic consideration just as it would have done had it been an industrial accident. The two episodes hang together in reasonable relationship in a person with an unblemished medical record. But what in fact is the etiological relationship between the bottle and the fit? If it was an invariable sequel the clue would be simpler, but it is the exception rather than the rule or the epileptic population would be tragically numerous. This young man's endowment lacks something that others have; something that has made him more susceptible. We must visualise a group of "silent" epileptics who may slip through life with no fits but who are at the mercy of a predisposing cause. Sometimes they are easy to detect, as when the family history is strong, but more often they are unsuspected. Hurst's percentage of peptic ulcer subjects is an analagous story.

Let us look for a moment at what is now known about the actual nature of an epileptic attack. So many different morbid conditions have been observed in persons subject to seizures that many clinicians have declared that epilepsy is only a symptom; a convulsion, like a headache, is a red light signalling the dysfunctioning of some somatic mechanism. However, generations of physicians have realised that in the majority of cases no significant dysfunction could be found; the cause of seizures was inscrutable. Hence epilepsies have been divided into two groups: the first, the inscrutable (idiopathic or essential); and second, the scrutable symptomatic, in which an apparent cause had been discovered, some disorder of body or brain. Deeper study and the electroencephalograph now permit the use of more decisive terms, namely, genetic (for essential) and acquired (for symptomatic) epilepsy. However, these groups are not mutually exclusive. Some degree of inherent susceptibility may be present in persons who suffer "traumatic" epilepsy; some undiscovered acquired stimulus may be the trigger which discharges a genetic epilepsy.

Of the two main causes of epilepsy, the genetic and the acquired, the former is the more important. Lennox,[6] who is probably the greatest living authority on this subject, states that of patients who are subject to seizures approximately three-fourths give no history or, on examination, present no evidence of significant brain injury or bodily dysfunction. Further he states that among the near relatives of unselected epileptics, both epilepsy and cerebral dysrhythmia occur approximately five times more frequently than in the general population. Among identical twins only one of whom has epilepsy and anticedent brain injury, the normal twin nevertheless usually possesses an hereditary dysrhythmia of brain waves. In a given patient the relative importance of a genetic factor is greater if evidence of brain injury is lacking, if there is a family history of epilepsy or migraine and, most important of all, if one or both parents have cortical dysrhythmia

on electroencephalographic examination. The practical aspects of this in connection with prevention are fairly obvious.

Proof of Hughlings Jackson's definition of epilepsy as a sudden excessive unruly discharge of neuronal cells had to await the patient labour of Hans Berger who, in 1929, published the first electro-encephalograms. Much useful work has since been done chiefly in comparing brain records of patients with supposedly healthy persons and the Gibbs have produced a valuable classification of electro-encephalograms which make it possible to envisage a time when the use of this apparatus will be demanded in all disputes concerning cause and effect in epilepsy. It may never offset the hard facts of legal logic completely, but it should and will influence the amount of compensation awarded.

In my view one may summarise the situation by stating categorically that trauma can only cause symptomatic epilepsy by creating gross demonstrable cerebral damage, and even thus it is quite probable that newer methods will lay bare some inherent genetic fault, otherwise how is one to explain that in a series of over 200 head wounds consecutively observed by myself in only two did convulsions occur within the first two weeks of wounding. Those not susceptible may have no fits although it is well known that their behaviour in other respects may be greatly altered and their use of even moderate quantities of alcohol may be pathetically grim in its results.

PARKINSONISM.—The spate of literature which accumulated after the advent of epidemic encephalitis contains a sprinkling of references to the possible association of trauma to the production especially of the common sequel of Parkinsonism. There were advocates both for and against, but always doubts persisted, which were expressed by Kulkov [5] in the question: "Could it really be the fact that a cranial trauma by way of destruction of tissues or in consequence of hæmorrhages in the region of paleostriatum (globus pallidus) or adjacent apparatus, could evoke a vivid picture of Parkinsonian appearances?"

Since Parkinsonism is a syndrome having various etiologies (such as encephalitis, syphilis, arterio-sclerosis) we can take it for granted that the disease may appear after a trauma which calls forth some very subtle and elective hæmorrhage around which later secondary symptoms of neuroglial sclerosis would arise.

According to Jelliffe and White, "trauma may be a sufficiently exciting cause to bring the symptoms of a slumbering paralysis to the surface, or those of a mild case rapidly to a severe stage." Emotional disturbances undoubtedly play a large role in causing the arterio-sclerotic foundation. The direct relation of the striatum to emotional activities should be borne in mind. One patient in whom I have watched the slowly advancing development of paralysis agitans for upwards of ten years believes even now that her incapacity originated as a result of a painful venesection for transfusion during a debilitated

state after operation. In this case as in others one has seen it happen that tremor commenced in the injured limb. On the face of it even the lay imagination has to be much stretched to accept this association ; moreover the claimant in law would have a poor chance of establishing a case. Yet are we really so sure of its impossibility ? Bing [1] coined a phrase, " Commotional encephalos," to suggest a disturbance of function, short of a demonstrable pathological lesion, that would be sufficient to set the stage in a debilitated and frightened person for rapid advancement of a latent arteriosclerotic process. How much more understandable the " lighting up " of a dormant encephalitis, since it is well known that only a proportion (possibly 25 per cent.) of those who used to be seen with acute encephalitis proceeded to the Parkinson state after a very variable interval, often indeed only those in whom the acute phase was only detected in retrospect ; the remainder apparently making a complete recovery.

Continental authors on the whole have been much more particular in mustering their evidence than we are, to the extent of often laying down rules by which a given case must be measured if the relationship with trauma is to be accepted. For instance Bing advances the following points in order to establish a causal connection between the trauma and paralysis agitans :—

(1) The trauma must be severe enough to be able to cause some cranial lesion or at least call forth symptoms of a *commotio cerebri*.
(2) The patient who suffered from the trauma must not have been previously subject to any cerebral symptoms.
(3) The Parkinsonian symptoms must not follow immediately from the trauma ; their development ought to be preceded by some prodromal symptoms (cerebral symptoms) which would then gradually develop a characteristic picture.

Trauma in Proven Conditions

There is no difficulty in selecting the symptom-complex of herniated intervertebral disc as a condition in which the link between injury and resulting neurological state is indisputable, differing only in degree and kind from a hemiplegia resulting from gross damage to a part of the brain, the essential difference being that the violence (often even trivial) is indirect. This fact may be important since it introduces the principle that the outward effect of trauma may be invisible and established solely from the inference drawn from history and physical examination. Twenty years ago, before the herniated disc complex had been explained and accepted, it is inconceivable that a simple jerk or stubbing of the foot would ever have been invoked to explain a sciatic pain, yet obviously many victims must have continued in ignorance of the role trauma had played in their malady, content to believe that a mysterious neuritis accounted for their pain

and suffer the indignity of teeth extraction and other measures designed to eradicate septic foci. Unhappily now authors try to oversimplify this problem by attempting to use this simple pathology to explain all manner of cases. But nature is not interested in simplicity, and in this condition our real challenge remains, which is to discover the true relationship between disc pressure and neuritis, since it is already agreed that in many instances the two exist side by side. Progress has naturally been slow in revealing the pathologic appearance of painful nerves as seldom is material available for examination, but now very many cases have undergone operation when it is frequently found that the compressed roots are thickened, congested and inflamed. An association that requires no septic or toxic theory to explain. We must beware of being too dogmatic lest we open up too attractive a field for litigation, and yet the implications of all this are far reaching in connection not only with sciatica but of " neuritis " in many other parts of the body, a subject which for long has been in an unsatisfactory state, dearly loved by the quack who finds in it a rich and safe field for his universal technique.

The multiplicity of pathological lesions which affect the spinal cord itself are all well recognised entities whose behaviour conforms in each case to a definite process whether it be inflammatory, vascular, or neoplastic, and yet it is the experience of all practitioners no less than specialists that violence less or more is presented by the victim or his friends as the alleged explanation. Numerous illustrations of this leap to one's mind.

A middle-aged lady with chronic lumbago secretly visited a chiropracter and was submitted to the common if unorthodox procedure of powerful blows on the spinal column while lying prone on a table, the middle portion of which was missing. Returning home with difficulty she retired to bed and never rose again. Fearful of her doctor's censure she lay for some weeks before obtaining help, but soon, by the ordered methods of diagnosis, it was found that a comminuted fracture had occurred at the site of a metastatic growth.

This illustrates quite vividly that things are not always what they seem to be, and so in every instance of spinal cord disease pathology must go relentlessly on, its existence brought to light by injury or violence, often aggravated but surely never initiated. But now the law of compensation has reached the point through a long series of litigation claims when the chances of success in law are strong if it can be proved that on a given date immediately before an accident the patient was symptomless and normal on examination ; that violence was indisputable even if variable in degree, and preferably shown by the same medical witness that physical signs began and progressed from a brief time after the injury.

A recent instance of this happened to a gamekeeper aged 60 who was penned between a backing car and a brick wall, bruising but not fracturing his pelvis. He had been active and well before this but during the few days

while recovering from his bruises, one foot, that on the injured side, was found to be weak. From this moment the clinical picture has developed into the easily recognised one of amyotrophic lateral sclerosis.

Dare we say that the accident had nothing to do with the subsequent condition? I think not, and for this reason; the sequence of events reach a logical conclusion especially in the lay mind, and moreover we are dealing with a malady whose etiology is unknown, differing in that respect from the surer ground of established causes as in meningitis, poliomyelitis and the rest, where the reasoning even allows for a prodromal phase of several days, and each case follows a recognisable behaviour pattern.

How different is the picture in the young boy who was admitted a few weeks ago with severe headache and vomiting after hitting his head sharply against an iron girder. This youth was in the second stage of tuberculous meningitis and died eight days later, showing that the accident was an unimportant incident.

Carrying the argument further, it is seen that when the whole course of the disease is known as an entity we are medically strong, and conversely when much is still doubtful or uncertain we are weak. None the less in spite of this appeal to reasonableness and the hunting down of simple but significant facts, it is safe to assume that much time and energy will be spent by the unwary in making impossible claims; a kind of projection of theory over fact from whose occasional success springs continuing hope. Too often an ignorant doctor with a plausible manner gives an opinion that will override the more cautious utterances of the scientific specialist.

Since early times the association between brain tumour and injury has occupied attention for reasons that are too obvious to require emphasis. Before the advent of neurosurgery and the experience clinically and pathologically of two world wars, it was a matter that once suggested must have been extremely difficult to disprove. However, nowadays the most that can be said is that head injury may be an incident in new growths within the skull just as indeed it may not be. An early case showing this remains in my mind after many years.

The patient, a young man of about 30, was under observation for epilepsy which had developed after a car accident. No fracture or obvious damage was found in the skull and no organic signs, but it was noticed that the fits had a Jacksonian quality. However, they were infrequent and he returned home to take the usual sedatives. He was admitted to the same ward some five years later, having fallen unconscious in the street. Death followed and at the post-mortem two gliomata were found, one small and slow growing, in the motor area corresponding to the fits, the other near the corpus collosum into which a large hæmorrhage had occurred. Even if it was allowed that the accident had caused the one glioma, it is inconceivable that the second could have had any association. Therefore if one had not, why should the other?

Again reciting war experience, it is highly probable that if trauma had any true relationship with cerebral tumour such an association would have emerged in an incontroversial way among the many head wounds and injuries of every variety and degree. All evidence points entirely the other way even now when much follow-up material is available. The proportion of brain tumours in the army was small in all campaigns and in my own experience the contribution of the front line soldier was infinitesimal. I cannot give figures in an overall sense, but in a concentrated sample one remains impressed with the complete absence of any hint that neoplastic pathology was influenced by violence—gliosis there may have been, but glioma never, one being compatible with continuing life, the other not.

On the other hand vascular disturbance in the skull is common, with its consequent effect on nerve function. Personality changes following severe concussion are regular examples of this, also every variety of catastrophe from a ruptured aneurysm to a delayed subdural hæmatoma is familiar in war as in civilian practice, but here it is important to recognise that violence creates a situation by itself and does not play a part in initiating a pathological process that is understood. An analogy with cardiological knowledge is appropriate since in this field the effect of blows on the chest have been studied with great care. It has been shown convincingly that even in the absence of external bruising or fractured ribs the myocardium may be profoundly disturbed and results including coronary thrombosis, arrhythmia, ventricular rupture and angina pectoris are accepted on occasion as due to this cause. The phrase "inefficient heart" is sometimes used to express the result accruing from a compression or blow over the precordium. Litigation is incessant in these matters with its tedious argument, but luckily up till now I am unaware of any claimant having the temerity to press his "inefficient brain," unaided by substantial additional signs, as the sole result of injury. Yet if allowed in one organ, why not in the other?—the important point being that the effect of trauma is vascular and in that sense not a pathological process.

Leaving this subject to pursue a much more controversial question with a vascular slant, I should like to touch for a moment on these difficult and little understood cases of "burning pain" or causalgia. The wretched victims of this suffer agony which is often both mental and physical, due to the condition sometimes being unrecognised for what it is with insulting psychological treatment employed and the fact that the pain is very real.

An elderly miner had his left foot pinned by a fall of heavy stone. The skin was unbroken but extensive bruising resulted, although no bones were damaged as shown by X-ray. Very shortly even the signs of bruising disappeared, but gradually he complained of increasing pain on certain movements of the foot and in time he stated that he could not bear the weight of the bedclothes or a soft slipper unless the foot was swathed in wool. When

I saw him six months later this condition was unchanged and he was indignant at what he felt were the insinuations implied by the treatment he had received. This man was a good type and an excellent witness, giving his story in a convincing way to the extent of volunteering the statement that he felt if his foot was removed he would be as fit and happy as ever he had been. Most of the foot was tender to touch but there were trigger points which caused excruciating pain and a strenuous attempt to draw the foot away.

Numerous similar cases are familiar to anyone who has had the opportunity of studying the diverse effects of injury to a limb.

Since the sixties of last century this subject has intrigued neurologists and physiologists. In more modern times the names of Henry Head and Sharpey Schafer are identified with personal experiment in attempting its elucidation. Even now it is not fully understood, although the relationship between cause and effect is never in dispute, only the exact mechanism is in doubt. The answer, when worked out as it will be, may well give a far reaching clue to the role of trauma.

If we allow, as we should, that trauma of a peripheral nerve may disturb capillary circulation, allowing the vasomotor disturbances which take place in cutaneous areas deprived of sensation, then we are entering on an unpredictable phase of values. But it seems especially probable that the obscure inter-relationship of the vascular and nervous systems may be unravelled to the extent that injury has a precise and definable position among the etiological causes.

SUMMARY OF ORGANIC ASPECTS.—The examples I have chosen, although by no means exhausting the possibilities, do at least illustrate vividly how troublesome is the ground in attempting with any degree of exactness to evaluate the role of trauma in the etiology of organic nervous disease. One recognises only too clearly the difficulties of offering a convincing argument for or against in a given case, but none the less certain principles do shine throughout, not the simplest of which is the need for exactness of observation, which precept embraces the rule that the medical witness, be he expert or other, must never lower his standard to that of a partisan. The obviously ill-considered and biassed opinion does infinitely greater harm than mere ignorance to injure the age long and mutual respect of our sister profession—the law. As medical men we believe in the doctrine of specific causes, but with trauma one has to endeavour to get some general principles on which to go.

TRAUMA IN FUNCTIONAL NERVOUS DISEASE

There will be those who consider that this part of my subject should have been dealt with first, in that it is obviously the most difficult and most chaotic. I was tempted to do so but refrained on finding myself bewildered in the effort to find a single satisfactory line of approach. It is a crowded, tricky field to survey without

proper aid from accepted terminology, and with the years comes the realisation that experience is fallacious and judgment difficult, leading us to recognise that in medicine the area in which confident knowledge is at present possible is but a limited one. In the neuroses, especially the traumatic ones, final and absolute truth is not attainable, only working truth. To elaborate this a little further, one of the commonest mining accidents sheds much light. A fall of stone from the roof on to the working-man's back is, sadly enough, a very usual experience as I have good reason to know after a long association with the Scottish Mine Owners' Defence Association. The results of such an accident may best be thought of as a scale, at one end of which is the man whose spinal cord is irreparably damaged with all the attendant motor, sensory, reflex, and bladder changes. At the other end of the scale is the lucky man who by good fortune escapes with minor bruising from which he rapidly recovers. Between these two is every degree of symptom-sign combination. There comes a point on this scale when no sign, as we understand the term, is found and yet complaint is made. It is reasonable to argue that very slightly more damage would have produced some organic indication on examination. Let us admit at once that a limited amount of spinal cord disturbance may be impossible to detect clinically, but in what category is such a case to be considered ? Is it a slight spinal concussion or a traumatic neurosis ? From my experience with miners and the more concentrated opportunities of making observation during the war, I believe that the correct designation only becomes apparent at some interval after the event. A slight spinal concussion will recover completely in a short time, whereas a traumatic spinal neurosis will be perpetuated and possibly increased for many months depending on other factors both conscious and unconscious in the individual. It is these factors that I wish now to consider.

To turn to the dictionary is almost a digression, but in this connection it is helpful if the derivation is backed by acceptance and usage. The word " neurosis " in Greek means " nerve." In such a reputable publication as the American *Illustrated Medical Dictionary*, edited by Newman Dorland, an accepted authority, this simple word is given a choice of definitions : 1. A nervous disease ; more especially a functional disorder of the nervous system. 2. In psychiatry, a relatively minor disorder of the psychic constitution ; in contrast with the psychosis, it is less incapacitating, and in it the personality remains more or less intact. Sometimes called psychoneurosis. Thereafter, among a long list, one can read that accident neurosis is a neurosis with hysterical symptoms caused by accident or injury, and that traumatic neurosis is one which results from an injury. It would weary you to amplify, but what in fact does it all mean ? No one will deny the good faith of these important authors, only their helpfulness in this subject is disputed, since the unwary are too often betrayed by an exchange of words for knowledge. This, in effect, is the very

essence of the problem, since so much of our understanding is bemused by vague thinking which passes for learning let alone knowledge.

The effect of trauma on the mind is conditioned by conscious thought which in turn is under the influence of subconscious processes. This involves diathesis and character, together with an incalculable something that we regard as personality, all adding up in an individual to a state for which at present we have no reliable yardstick or measure. These factors are egocentric, but when an incident occurs a further consideration arises due to suggestion accruing from the circumstances of the accident ; the aggregation of all these resulting in the behaviour of the person during the weeks and months following the injury.

INTERPRETATION OF DELAYED NEUROSIS FOLLOWING ACCIDENT.—

T. B., aged 35, a shaftsman in the mine ceased work on 3rd April 1946, claiming to be unfit on account of " an injured leg and shock " which he attributes to be the result of an injury he sustained in September 1943. At that time apparently he was engaged at the installation of some new plant in the shaft and was in the act of transferring a pipe from one rope to another. The signal had been sent to the winding engineman to lower the cage but instead of doing so the cage was pulled back and the man was thrown over a beam and had to climb part of the shaft. I saw him on 25th May 1946, when in addition to these details he told me that he was not unconscious and the bruising of the leg only necessitated his remaining at home for five days. After this he did light work for six weeks and then obtained other occupation of ordinary work in the mine. Later, in about six months, he resumed his usual work but in another pit ; however, at the end of February this year he went to his original post in the pit where the accident occurred. After five weeks of this he was unable to continue because of increasing weakness in his back and a feeling of nervous strain. In answer to questioning he alleges that these sensations have been present in varying degree since the accident, sufficiently on one or two occasions to keep him off work for a week or so at a time. He admitted that the original shaft haunted his thought and that, while able to continue at other work, he had a horror of his original job, and always opposed the suggestion that he return until he finally did so with, what he termed, the inevitable result. Since being idle he alleges that his symptoms have become steadily worse.

In appearance the man had a good colour and was well nourished. He moved carefully and slowly, holding himself stiffly. Hands were tremulous and reflexes lively but there were no signs of injury and no indication of organic disease in the nervous system or elsewhere, but the pulse rate was rapid at 100 per minute.

My summary of this case was : that the immediate effects of the accident in September 1943 were slight, but gradually an anxiety has developed which is said to be increasing. It is a typical functional state and not associated with any signs of organic disease. In my view the condition is not due to the accident as such but is compatible with an attitude of mind that has developed and been elaborated by a subconscious dislike of his particular place of work : aggravated by suggestion coming from friends and others. Had the workman

not returned on February last to his original location, the present state of anxiety neurosis would probably never have arisen.

That, of course is my honest view reached by normal medical processes of deduction and assessment. One is convinced of its truth in fact, but this is an instance where the processes of medicine, perhaps aided by what the cynics call a hunch, come into conflict with the logic of the law whose representative will call dramatic attention to the far reaching emotional disturbance to the man of the grim possibilities inherent in the circumstances of the original accident and the present cruelty of endeavouring to establish that the claimant was work-shy. In this case a haunting fear was the dominant factor, to the exclusion of all others, including compensation. Yet how frequently in the attitude of to-day, pandering to the popular trend, would all attention have been drawn to the financial motive.

Two people of like age may receive a similar blow, the one at football, the other at work, neither really serious, yet while one recovers spontaneously, the other frequently undergoes a lengthy period of invalidism. Surely it is something more than the presence or absence of inducement. In a sense the subconscious process is the same, desire to exploit the situation, the footballer gaining prestige by an early return to the game, the workman escaping from an unpleasant routine to enjoy leisure and perhaps sympathy. There the matter ended in the days before the vast experiment originated by the Workman's Compensation Act. Since the advent of this, however, a new factor has arisen which has proved to be a testing ground both for the credulity and good faith of mankind. Other countries than ours have been confronted with interpreting and applying similar legislation, and against the justice which it has brought to men's lives must be offset the proven fact that the millions spent annually increase steadily, and with the outlay an enormous addition to the army of men off work. The cumbersome machinery itself involves endless delays during which the workman's symptoms, originally a " traumatic neurosis," become transformed into a " condition neurosis " in the sustained effort required in a fight for compensation.

No sane thinker to-day would ever deny the injured workman his due right to have the best available treatment and at the same time reasonable provision for himself and his family, but the greatest evil arises from the principle of lump sum settlement which provides a happy hunting ground for the less scrupulous type of lawyer and even more importantly a sustained mental collision for the victim.

To distinguish between neurosis and malingering is a relatively simple clinical procedure, and yet the medical literature on the neuroses following trauma is frequently tainted by an under current and by an unwarranted hostility and antagonism toward the neurotic, implying that he too is a simulator, thus losing sight of the fundamental nature of the condition. This attitude does much harm in its perpetual denouncing of the neurotic and in the condemnation of his inevitable

desire for compensation. Whereas so often it is not the victim himself who first thinks of the matter but more frequently his friends and advisers, the injured being stampeded by these influences when he comes to recognise the amazing therapeutic indifference of the many medical men who confront him as the case develops along the usual partisan lines. Then he begins to sort out those who are for him from those who are against and conditions himself accordingly. The physician who is there to alleviate human suffering often adds insult to injury by his manner, which undoubtedly traumatises the psyche of the injured, and does much to bring about secondary elaborations.

The " wish for compensation " explanation has been so publicised to the extent that it masks the vastly more complicated etiology of the psychoneurosis. This persistent notion has tended to oversimplify the whole problem, but one has seen too many cases where the measure of secondary gain from continued illness is insufficient to account for such lasting disability.

Not infrequently one has observed a workman with a highly developed sense of family responsibility who has continued at work after injury in spite of subjective symptoms such as dizziness, back pain and headache, who makes no claim for compensation and does not show secondary elaborations. The success of such an effort depends on a number of factors—intensity of the symptoms, character of the work, make up of the individual. Proper guidance and psychotherapy in the very beginning of the illness are important. Our best approach medically is to regard compensation as, at most, offering merely a secondary gain from illness.

Some years ago as a result of a disastrous mine explosion I had the opportunity of observing very closely the gradual development of this secondary elaboration in a group of over twenty men who had experienced relatively the same type of accident. At first each case had its own individuality and personal variation of symptoms, not untempered with relief that Providence had spared a worse fate, but slowly as time dragged on, with the inevitable group discussion and example, each of these men evolved an indistinguishable neurosis pattern that was pathetic to witness. Superficially it might be regarded as the herd instinct towards reward, but as one got to know these men the complexities of each problem became increasingly apparent.

The point I should like to make is this :—that a useful experiment might have been made by persuading every second man immediately after the accident to do a job within his capacity, probably much easier than his normal work, but at a similar wage. Then the future progress of each group could have been watched with a control for comparison at every stage.

CONCLUSION.—The most conflicting opinions concerning the organic effects of trauma on the nervous system are to be found in the vast amount of available literature. I have attempted within the limits of this paper to indicate the present position of our knowledge

as I see it, hoping to avoid the aspersion of being dogmatic, since such an attitude as yet has little to commend it. Nonetheless we must try thoughtfully and faithfully to see what light there is, expecting in time to build up the truth on a foundation of fact.

In the post-traumatic neurasthenias and post-traumatic hysterias, as I feel they should be called, one has laid emphasis on the intervening period of meditation and suggestion, while trying to relegate the question of compensation to a more subsidiary role than is given to it by many who are guided mainly by the fashion of the times and enjoy a simple explanation where indeed none exists.

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