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CONCERNING INJURIES OF THE SPINAL CORD*

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It is most fitting that we should discuss within the precincts of this university the question of spinal injuries and the accompanying injuries of the cord, that we should pool our experiences in this town where Sir Charles Sherrington has for so many years conducted his researches into spinal nervous function. If we know much less about the damaged human spinal cord than we do of the cat's it is because accident inflicts crude and clumsy injuries at its own time and in its own way. Moreover, the unwieldy mass of the human subject renders it difficult material to handle, and, apart from humaner considerations, its very proper demand for a high level of comfort makes it in the acuter stages of injury difficult of quiet observation. These thoughts are induced by reflection on the great debt which science owes to this school, for the brilliance of its researches, for the great ingenuity with which experiments have been planned to give understandable answers.

While this discussion is limited by its terms of reference to spinal cord damage, it is obvious that we cannot completely deny ourselves any allusion to the vertebral injuries which accompany it. We must contribute something, if we can, to the very important problem of treatment of the injury, which entails a consideration of the damage as a whole, both neural and osseous. We shall be led thus to consider methods of treatment and to pronounce, if the facts allow it, a preference for one method over another.

Elective Sites

My own records are of seventy-five cases of cord or root injury, of which thirty-nine are cervical injuries, twenty-two are lumbar, and fourteen are thoracic injuries.

In previous papers I have commented on the frequency of damage at certain levels rather than at others, and have concluded that the injuries occur at the points of greatest mobility in the spinal column, and not, as was previously believed, at the springer points, where a relatively immobile portion joins with a more freely moving segment. The special proneness to damage of the fourth, fifth, and sixth cervical vertebrae, and of the twelfth dorsal and first lumbar, would make it probable that cord injury is commoner at those levels than elsewhere—it is so in my series. On the other hand, the relative narrowness of the thoracic spine and the fact that it is only injured by force of a particular kind and direction makes concurrent cord injury more uniformly present with these relatively rarer injuries than is the case at the two sites of election. At these two latter places vertebral injury is by no means uncommonly unaccompanied by neural damage. The result is that the graph of incidence of cord injury, while still showing cervical and lumbar peaks, is a little higher in the thoracic region for cord injuries than it is when uncomplicated bone injury is charted. One may well meet with three or four compression fractures without cord complication for every one that has it.

Mechanism of Injury

The mechanism of vertebral injury is so well understood to-day that there is no need to dwell on it. I have described it very fully before.[6] Damage to the cord occurs most particularly when there is displacement as well as compression of a vertebral body, but it can also take place during excessive flexion alone. I am inclined to think that the train of events in the cervical and lumbar regions is rather different. In the former excessive forward flexion of the head leads eventually to rupture not only of the zygapophyseal joint capsules, but also of the intervertebral disk. In this instance not only does the upper fragment (the head and upper vertebrae) move freely forward and injure the cord, but it allows of easy replacement. Thus comes about the interesting though useless fact that replacement of the dislocated vertebrae is easier in cases with severe cord damage than in those where it is slight. One other point of interest arises also, and that is that this same mobility and ease of recoil explains why in some cases of haematomyelia in the cervical region no sign of vertebral displacement can be disclosed by the most careful of x-ray examinations. In the lumbar region displacement and recoil can rarely occur, and here I incline to the view that rupture of the intervertebral disk with backward displacement of the liquid contents of nucleus pulposus contributes something of a nocuous kind to the injury, but it does not do so invariably.

Effects of Cord Damage

It is well accepted that loss of function following a spinal injury is due to neural injury inflicted at the instant of primary deformity. It would be untrue to follow this up with the statement that the cord is very rarely compressed by bone fragments, because we know from experience that sometimes it is so inconvenienced, and is then usually irreparably damaged. It is generally agreed that central haemorrhages occur into the well-vascularized substantia grisea of the cord, and it is assumed that this is the cause of the neural disturbances whether vertebral injury is clearly manifest or not. Every surgeon accustomed to operating on the spinal cord knows how tough is its pial covering, and it is easy to visualize pulping of the contents of this strong sheath and to predicate central haemorrhage. It comes, therefore, as

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something of a surprise to find that the cervical cord in cases of fatal injury often fails to show bleeding. It cannot be stated that haemorrhage never arises, for it is necessarily present when the cord is actually torn ; but it is not a feature in those common instances when the cord is fatally contused without any notable alteration in its external appearance save swelling.

The notion that a sudden squeeze sets up a massive haemorrhage and that the pressure and possible disruptive effects of that are the cause of the resulting dysfunctions must be accepted with caution. It is doubtful whether such a state as " haematomyelia " actually exists. It is more than probable that the clinical syndrome which we call, and no doubt shall continue to call, haematomyelia is due to local neural disruption, to relatively bloodless contusion, rather than to intramedullary clot. The damage which does in fact occur is in the nature of rupture of myelin sheaths, fracture of axons and nerve fibrils, contusion of nerve cells in the central grey mass, a distortion of the general cord pattern in which sliding of neural elements on the fibrous mesodermal partitions plays a considerable part. In most cases there are evidences of oedema due to the disturbance of humoral barrier mechanisms in the intraspinal capillaries. The appearances of the cord in some fatalities leaves the impression that the patient might have recovered if he could have survived the initial period of profound depression of function. We still lack information as to the extent to which axonal rupture takes place in the long projection paths. It may be that injury to the myelin sheaths is not of primary importance, for the axon will still conduct when this is damaged. It is my impression that we have a sure clinical indication of axonal rupture in the state of sensation in the patient with an injured cord, and a less sure indication in the degree of paralysis. This brings us to the difficult subject of spinal shock, for we know that if a patient is for a time severely paralysed by an injury and yet recovers motor power only one conclusion is possible—that little true anatomical damage had been inflicted, so delicate a reflector of injury is the cord. Indeed, it is this extreme sensitiveness that introduces all the difficulties of spinal shock.

Spinal Shock

In the fatal cases we can discover the extent to which the cord has been damaged, and we infer that there has been a lesion of like, but less, kind in the cords of patients who recover. This takes us but a short way in the solution of the immediate clinical problem presented by a patient just injured. We assume cord damage when we are faced with traumatic paralysis ; we judge its degree by the extent and also by the quality of that paralysis. We recognize that the paralysis is compounded of both anatomical alteration (which may be small) and of abrogated physiological function (which may be large), two interdependent but separable entities, of which one does not appear without the other. At first the functional derangement overshadows the structural to such an extent as to make it difficult to know how much or little the other is, to know precisely what damage has been inflicted on the central grey mass, on the pyramidal and other long projection tracts. As we shall see, the sensory changes are a more reliable index of the extent of the lesion than are the motor. We shall inquire why this should be, as the problem has never before been stated. If the patient is unable to move his lower limbs are we to assume that his pyramidal pathways have been torn? Clearly not, for complete recovery may yet take place, as is proved not only by traumatic cases but by collateral observations on the effects of removal of spinal tumours. The paralysis is due to shock, to oedema, and in compression cases to local anaemia.

" Spinal shock " is a term introduced long ago by Marshall Hall as an explanation of the loss of reflex activities that follows spinal section. But as Sherrington soon showed that the condition affects only that part of the cord which is distal to the injury, and stated that it lasted only a few days, there is evidently something peculiar about it. The fact that it lasts as long as a few days makes it unlike " shock " in its ordinary surgical sense, and in actuality no one has ever ventured to define its exact duration. Every clinician knows that the state of reflex activity of the lower limbs alters over a long period, and voluntary movement (which is not included in the original pure physiological conception of spinal shock as studied in complete spinal sections) over longer periods still. In order to avoid the use of the word " shock " von Monakov introduced " diaschisis " to indicate depression or loss of synaptic function without any dogma as to its precise cause. It is known that other factors—cold, discomfort, pain, bleeding, low temperature, low blood pressure, infection—can all depress these activities without implying irrevocable damage to cell structure and connexions.

The problem has been further studied in the Oxford laboratories by E. G. T. Liddell,[8] whose work I have drawn on because it contains many very suggestive facts and speculations. Liddell divides shock into two parts: (1) true shock, a matter of hours or days at most ; (2) " isolation-alteration " in Munk's term. It is chiefly this latter which Liddell has studied, the slow rise in excitability of the previously inert cord. It is often assumed that if a nerve cell is in connexion with an excitor fibre (such as one from the pyramidal tract) it will discharge on volition. Liddell stresses the importance of tonus impulses, the stream of subliminal impulses from varying sources (notably the vestibular nuclei) which are requisite for the maintenance of readiness to discharge. In the absence of these and of equally valuable " fringe effects " from other cells in the motor neurone pool a normal cell is not excitable.

Hoff's[4] estimation that there are 300 to 350 synapses (*boutons terminaux*) in relation to a single anterior horn cell in the cat (the number in the human is not at present known) emphasizes the influences to which a cell is subjected. It is easy, and no doubt correct, to assume damage to the long conduction paths in the spinal cord in a paralysed patient. But the point which must be made is this, that the inability to move may be due to temporary injury of other pathways besides, or other than, the pyramidal, and that because of the suppression of impulses carried by them the anterior horn cells at a lower level may fail to behave normally, either to willed movement or reflexly to stimuli. Thus a very profound atonic paralysis with absence of knee-jerks points to severe damage to the cord and complete abolition of the torrent of impulses from higher levels which normally pour into the cells of the central grey columns. In other cases reflex excitability is not abolished, and even slight voluntary movements may be possible from the first, with sensation also retained in some degree. In these cases the anatomical damage is probably slight and a good prognosis is justifiable. It will be seen that the state of paralysis alone is a difficult basis for estimation of damage.

The situation is different as regards sensation, which must next receive attention. Since the primary cell stations of sensation are outside the cord in the posterior root ganglia, spinal shock is unable to affect it adversely in anything like the same degree. If there is dense anaesthesia below the lesion there must be severe damage to all the sensory pathways and recovery will be rare, because this implies injury to the whole width of the cord. In less complete injuries from the first the sense of touch may be retained, though perhaps impaired, because there

are no intramedullary cell stations in its path that can be affected by shock. Touch uses long fibres coursing up the posterior columns and will be interfered with only by oedema causing pressure or by axonal fibril fractures. Pain and temperature senses are more apt to be interfered with owing to the more complicated synaptic nature of their transmission. Thus it comes about that a patient with a partial lesion may have very little power of movement, because of the complex nature of the maintenance of the normal excitatory state in the spinal nerve cells, and yet have relatively good sensation in the paralysed limbs, sensation being less susceptible to the phenomena of "isolation-alteration." This anomaly is explained by the facts laid down above and explains why sensory tests are the more reliable in deducing the extent of the lesion, and therefore the prognosis. The value of sensation as a criterion is well brought out in C. P. Symonds's contribution to expectancy of recovery.

Cervical Injuries

In none of my own cases of fracture of the atlas (seven clinical examples) has there been any cord injury, and in only two of the cases of fracture of the odontoid process (nine cases); nor has there been cord involvement in any of those curious and interesting vertical fractures of the axis which I described some years ago. The cervical group, excluding these two, comprises thirty-six patients, of whom seventeen died. It is important to particularize what differences there were in the neurological state of the recoveries when first seen in contrast with those where death followed.

Of the recoveries, in six there were cervical dislocations with root injuries only, and, as might be expected, all did well as concerns life, and all but one neglected case as regards function. In three patients there was "haematomyelia" without demonstrable vertebral injury—all recovered. In addition, there were ten patients with more or less severe injury to the cord who eventually regained a considerable or well-nigh complete use of their limbs. What was there in the immediate condition of these patients to differentiate them from the others who died?

In all there had been an immediate virtual quadriplegia, numbness of the whole trunk and limbs, but not a complete anaesthesia, usually with a feeling of "pins and needles" in the hands and arms; but in all there was some return of function within the first twenty-four hours, Often it concerned one leg, the other leg and both arms recovering to a lesser degree or, at first, not at all. In several there was no urinary retention. The patient, therefore, though badly damaged, is at the end of a day, or less, in possession again of some of the powers that he had lost, and when that is so the prognosis should be good. However, four of the fatal cases were severely but incompletely paralysed and none the less died within the next two or three days. Increase in the severity of the lesion can therefore occur, and is due, so far as one can dogmatize on something incapable of proof, to increase of oedema in the injured segments and surrounding tissues. Since all of these cases were fixed in one way or another, it is unlikely that further trauma was the reason for their death. Although in all of my own cases it has been the fact that a heavy and deep injury has always been fatal, it cannot be assumed that this will invariably be so. Information on this head is difficult to obtain. A. S. Taylor[10] has reported one case which was as completely paralysed as any of mine and anaesthetic below the sixth cervical segment and yet recovered; the exact state of sensation is not described. Similar experiences may have befallen others. More authenticated descriptions of such cases are required.

Case Reports

A résumé of the recoveries follows. The nature of the sensory loss in each is interesting.

CASE I

A. B., aged 16, after a diving accident was unable to move; there was numbness and loss of function in the hands. In a few hours he could move the left leg a little; the right leg and both arms were paralysed, apart from slight power of closure of both hands. The sense of touch was reduced over the left side of the trunk and the left lower limb. Five days later joint sense was good in both lower limbs, but pin-prick and temperature sensations were impaired below the eighth dorsal segment on the left side. Touch was recognized everywhere except on the hands in the eighth cervical segments, but less on the left than on the right below the eighth dorsal segment.

Comment.—There was local damage to the eighth cervical segment, with the right pyramidal tract involved temporarily after an initial period of complete suppression of all movement due to spinal shock. The right spinothalamic tract was injured incompletely, and probably also the left posterior column. This patient made a complete recovery, and six years later was able to do anything save for slight residual weakness of the right hand.

CASE II

C. D., aged 43, fell downstairs and dislocated the fifth on the sixth cervical vertebra. There was severe pain in the neck radiating down on to the shoulders with immediate complete paralysis, but in a few hours the left leg could be moved and the right a little. The bilateral paralysis of the arms was associated with anaesthesia down the inner sides of the arms to the little fingers. Below the lesion touch and pin-prick were both felt; hot and cold were not distinguished, both felt warm on the left side below the second dorsal segment. The tendon reflexes were present and there was no incontinence.

Comment.—This patient twelve years later was ambulant, but the injury to the grey matter at the eighth cervical segment left him with permanent dysfunction of a mild grade of both hands. The right leg remained weaker than the left. There was evidence of a partial impairment of temperature and pain sense on the left (Brown-Séquard), but the posterior columns were normal.

CASE III

D. E., aged 55, fell eight feet and sustained a crush fracture of the third and fourth cervical vertebrae. Immediate quadriplegia developed, and he was numb from the neck downwards. Sensation of pins and needles appeared within an hour and a flicker of movement returned to the left leg and, in a few hours more, to the left arm. At the end of three days the movements of the left leg were vigorous, and were present but less active in the left arm, except for the fingers. The right arm and leg were still paralysed; there was no incontinence. Cotton-wool was felt over both lower limbs; joint sense was present on the left, absent on the right, side. Pin-prick sensation was sharper on the right side and dulled on the left below the seventh dorsal segment.

Comment.—This patient made an excellent recovery, and five years later can walk ten miles. The right hand is still slightly weak. Once again there is evidence of local damage to the anterior horn cells of the seventh and eighth cervical segments (in spite of the high level of the vertebral injury), and to the spinothalamic tract of one side, and posterior column. The pyramidal lesion cleared up, leaving as its only trace slight overactivity of the tendon reflexes on the right side.

Space does not allow of fuller details of these or of an analysis of other cases. In general plan all were the same: there was evidence of local contusion of the cervical enlargement; paralysis of the legs with rapid recovery of one, slower of the other; retention of crude sensation on both sides but a definite clean-cut sensory level, often several segments lower, below which pin-prick and temperature sense, together or dissociated, were impaired or

lost. This bears out perfectly the suggestions made in the section on spinal shock as to what would be likely to happen in incomplete lesions of the cord. On the other hand, in the fatal cases there was a profound paralysis below the level of the lesion, with flaccid paralysis of the legs and to a varying extent of the arms ; diaphragmatic breathing only ; retention of urine ; and sometimes priapism. The worst sign of all is a dense anaesthesia up to the level of injury. Bilateral miosis was present in many, and is a bad sign, because the intramedullary cervical sympathetic is deeply placed, therefore a Horner's syndrome points to extensive central damage. The level of the vertebral never corresponds accurately with the neurological injury, but root pain usually indicates the highest level. Thus in a dislocation of the fifth on the sixth cervical vertebra the sixth roots are squeezed in the intervertebral foramina and pain radiates down into the radial borders of the forearms. At times the neurological level is distinctly higher than the vertebral, a fact due not only to the spinal segmental levels being from above increasingly higher than the vertebral, but also to spreading oedema in the cord. Two points make it difficult to give a standard neurological picture for an injury at a fixed and recurring level: (1) the muscles of the arm are represented in the cord as vertical columns of nerve cells which overlap several segments and our commonly accepted segmental supplies are averages which we use, and satisfactorily as it happens, to cover up the difficulties of the actual physiological arrangement ; (2) the cord contusion is inclined itself to be irregular.

Possibility of Survival

Two of the patients died in hyperthermia with temperatures of 107° F., the precise meaning of which is not known, but it suggests the release of temperature control by the cutting of autonomic pathways as surely as does hyperthermia following a pontine haemorrhage. In general the temperature in severe cord injuries is low, in fact it may be the lowest ever recorded in the human subject in these climes. Gordon Holmes[6] observed several patients with gunshot wounds of the spine whose temperatures were 80° F. or lower, and at the same time found a remarkable associated bradycardia and mental apathy ; in one of his cases the pulse rate was 22, in another it was 32 when the mouth temperature was 80.6° F., 102 when the temperature rose to 98.8° F. There are likely to be many more factors bringing about the death of the patient than we are yet aware of, upsets of visceral circulation and metabolism of direct or indirect neurological origin that make recovery impossible, quite apart from the more obvious difficulties of pulmonary ventilation arising from the partial paralysis of respiration. The oliguria noted by Holmes points in the same direction. The majority of these patients die within the first three days, sometimes from associated injuries, more often for the reasons just given. Occasionally a longer survival is seen ; two women lived, respectively, two and thirteen months completely paralysed below the seventh cervical segment and without the smallest sign of recovery. An even more astonishing survival was that in a case seen with G. R. Girdlestone—a woman of 30, with complete cord injury at the same level who had survived for six and a half years. She had had urinary infection, stone formation, spontaneous rupture of a peri-renal abscess, then a urinary fistula in the loin a year after the accident, but had got over all that, and but for troublesome pain in the radial parts of her hands was as well as a completely paraplegic patient could be. As we are often asked to give a prognosis in the first few hours it is well that we should be aware of the possibility of such survival, useless and distressing though it is.

Subjective Feelings in the Limbs

Interesting points emerge from questioning patients with complete cord lesions on the feelings experienced below the level of injury. It might be imagined that they felt nothing at all below the lesion, as if the body had been amputated. But the use of that word will at once raise speculation, for we know that " phantom limbs " are the rule. Do not " phantom bodies " also occur? Certainly they do, for the subjects are well aware of their lower limbs, and often feel them to be drawn up at the knees when actually they are flat on the bed. These limbs may feel to be hot or cramped, no doubt from impulses arising in the damaged area of the cord. The grey matter in the root entry zone can excite pain, and equally so, perhaps, other parts of the substantia grisea. The sensations felt in the lower limbs can confuse the patient when he is being tested clinically, so care must be taken to discount errors that may arise in this way.

Thoracic Injuries

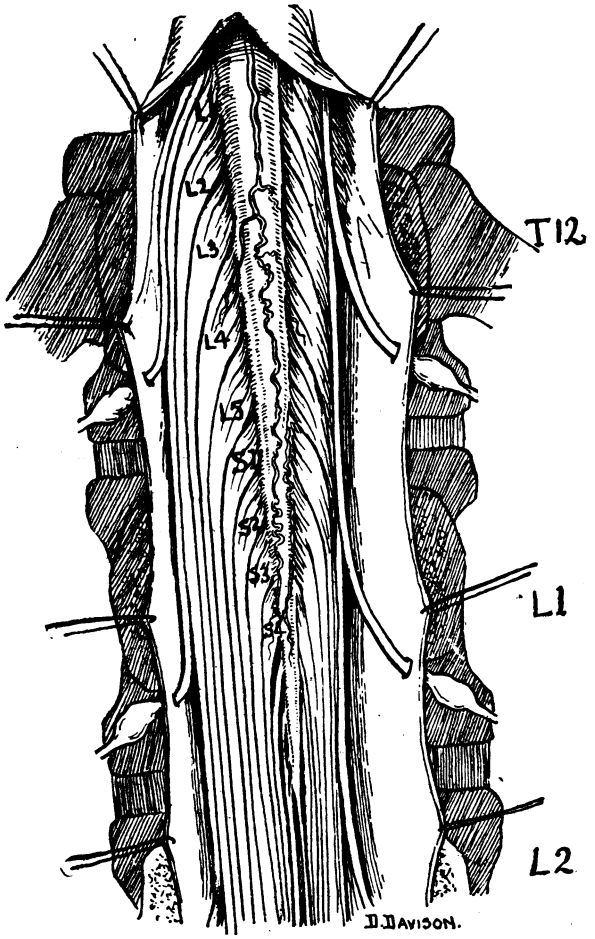
Of these injuries, fourteen in number, of the thoracic spine between the first and the tenth dorsal vertebrae, I shall say little. Three died within the first ten days. In every case there was a complete lesion except in one ; that patient had a crush fracture of the fifth thoracic vertebra and a cord lesion, incomplete from the first, rapidly recovering. The vertebral injury was reduced by the Watson Jones[7] method, and the patient made an admirable recovery. Of the rest some died months later, while one is known to be alive and totally paralysed below the eighth dorsal segment seven years after the accident.

Lumbar Enlargement

This is a very important group, and includes injuries of the eleventh dorsal to the first lumbar vertebrae, which have been admirably studied by G. Riddoch.[9] These are unquestionably the commonest forms of spinal injury in industrial districts and, as I have pointed out before, their incidence varies with different hospitals according to the character of the industries of the population surrounding it. For example, in 100 cases observed by Atha Thomas[11] the cervical spine was damaged in four, the lumbar in sixty. Thoraco-lumbar fracture is essentially a fracture of those engaged in heavy industries, especially in coal-pits and at docks. I have had twenty-two cases of injury of the thoraco-lumbar region with cord injury, of which only one died immediately. This was a case of flexion injury ; at necropsy the stomach, spleen, and intestine were found to be herniated into the left thorax through the torn diaphragm. Two others died in hospital: one hopeless paralytic of urinary sepsis, the other of embolism ; the remaining eighteen cases recovered. Some of these latter are since dead. Clearly the whole problem depends on the degree of neural damage, which can be a very variable factor with fractures at this level. In order to illustrate this I must speak of the relationship of the lumbar enlargement and conus to the spine. The particular point of importance is this: what are the structures which lie opposite the twelfth thoracic and first lumbar vertebrae, those most often injured? The lumbar enlargement begins above at the tenth thoracic vertebra and reaches its greatest diameter opposite the twelfth, from which point the cord tapers away to end at the lower border of the first or the upper border of the second lumbar vertebra. The structures opposite the first lumbar vertebra are the conus and all the sacral group of nerves arising from it. Therefore an injury limited to the first lumbar vertebra might produce anaesthesia in the classical saddle area of the perineum and gluteal regions (third and fourth sacral segments), with bands of anaesthesia down the legs to

the feet (second sacral segment), from contusion of the extreme end of the cord proper. The motor weakness would fall chiefly in the legs below the knees, and disturb also the control of the bladder and rectum. But alongside the conus and the nerves that arise from it are all the lumbar nerves downwards from the first lumbar nerve, which is emerging at the lower border of the vertebra. Thus with the most severe injuries to this vertebra damage to these roots is added to the medullary injury, and the lower extremities are put completely out of action. The first lumbar nerve, in spite of its theoretical vulnerability in fractures of that vertebra, often escapes injury, as is evidenced by a characteristic tongue-shaped area of normally sensitive skin on the upper end of the thigh, the lower limit of the territory of this nerve.

Opposite the twelfth thoracic vertebra lie the origins of the third, fourth, and fifth lumbar nerves. The



The neural structures opposite T.12 and L.1 vertebrae.

highest level of injury which can ordinarily be inflicted in fracture at this level is the twelfth thoracic, and since at this level the cord is considerably thicker the chances are that medullary damage will preponderate over root injury. This is well seen in the comparison of the actual results of lesions at the two levels, near together though they are. When the injury implicates the twelfth thoracic vertebra the paralysis which follows has commonly been lasting. Of seven cases only one made a good recovery, was ambulant, and lived for fourteen years. This is similar to the results of injuries at higher thoracic levels. When it is the first lumbar vertebra which is damaged the prognosis improves greatly, and of ten injuries at this level five did very well, three died, one, two, and seven and a half years later, while two died in hospital from complications.

Nowhere else in the cord does sensory loss give such precise information as in these lumbar injuries. In the worst cases the anaesthesia may be as high as the twelfth dorsal segment on the lower abdomen, but more often it leaves the first lumbar segment uninjured, and one gets the characteristic tongue-shaped areas of sensitive skin at the groins and down over three inches on to the thigh. As the injury is less and less serious so the anaesthesia is of smaller and smaller extent, and in the least serious examples one finds a small saddle area of anaesthesia on the perineum as the only neurological sign apart from disturbance of visceral function. In these last cases the knee-jerks will be present, but very likely the ankle-jerks will be absent. It is possible for a patient to retain extremely good movement in the lower limbs and yet have a neurological lesion of small size such as this, a lesion that may easily escape detection in its earlier stages, though sphincteric difficulties will soon draw attention to it. It is a gratifying experience to discover how well patients will, as time passes, accommodate themselves to these visceral difficulties and, in the end, overcome them.

Bladder and Rectal Disturbances

Recently this subject has been considerably illumined by the work of Denny-Brown and Graeme Robertson[2] and of Watkins.[12] In all severe spinal lesions, high or low, cord or cauda equina, the immediate result is always retention. It is only later that the fundamental differences become manifest. In cervical and upper thoracic lesions the emptying of the bladder regains, after days or weeks, qualities of massive emptying not unlike the normal, save that it is uncontrollable. In conus and cauda equina injuries detrusor action is feeble, and the bladder can only be emptied by straining—an entirely abnormal method, for normal micturition is initiated by an active inhibition of the muscles of the urinary passages, which sets going a steady contraction in the bladder wall. The alteration from normal is a useful one for the patient, as it enables him to keep himself dry. But before these stages are reached the patient must survive a danger period of retention. How is this to be managed? In the present series an indwelling catheter has been the routine method. It is true that some degree of infection is invariable when a catheter is installed. Catheterization, intermittent or continuous, is said to cause a high mortality in these injuries. This has not been so in these cases, and no patient who might otherwise have recovered has died of urinary infection. Connors and Nash[1] urge that the bladder should always be allowed to overflow, even if it needs thirty-six hours to do so ; they state that no bladder has ever ruptured spontaneously if left alone. This latter generalization is probably untrue, and the fact is that few patients will tolerate the pain of over-distension. Moreover, if once over-distension occurs it is impossible to express urine without risk. All will agree that catheterization ought to be avoided, and we all, probably, try to avoid it, but few of us, I feel sure, succeed.

As for the rectum, Denny-Brown and Robertson[3] showed that the external voluntary sphincter is not ordinarily in action, and is used for voluntary retention only under necessity. Normally the rectum is closed by the tonus of the internal sphincter and smooth muscle in the levator ani, which acts reciprocally with the rectal musculature, relaxing when the latter contracts. But as the rectum only normally voids at considerable intervals the patient with paralysis may get along quite comfortably (compare the colostomy patient). He lacks expulsive power if the abdominal muscles are unable to assist, and he may soil himself from loss of control of the striped muscle sphincter (including levator ani) if he gets loose stools by ill-advised purgation. A man may therefore

become ambulant after a pure conus or cauda equina injury, and yet be able to go about much as he desires when he has learned the precautions necessary to his state.

Treatment

The immediate treatment of all cases will be decided upon after a survey of the neurological signs, and of lateral radiographs of the spine. In cervical injuries good pictures of the lower end of the region are not easy to obtain, especially when there is a complete and high cord injury, because the shoulders are commonly elevated abnormally by the unopposed action of the trapezius and levator scapulae muscles. This can be overcome by the taking of stereoscopic oblique views of the thoracic inlet. Without exact information of the injury it is impossible to proceed. In general it is, of course, proper to reduce the dislocation, if one is present, but it must be observed that crush fractures without notable displacement sometimes occur in the cervical region as well as lower down, and call for nothing more than immobilization followed by plaster. As for the dislocation, some have been reduced by manipulation under anaesthesia, others by slow traction. I have not been favourably impressed by the method of reduction described by Taylor[10] of New York, although he got good results himself, in which great tractive power is developed by means of pulleys. I have reduced one case by this means, completely failed with another after two attempts, and in a third I believe the cord injury was made worse.

The difficulty in any method is to get the traction in precisely the right plane, without at the same time risking forward tilting of the upper fragment. The neck is so mobile at the point of dislocation, below the level as well as above it, that the lower fragment tends to be uncontrollable. Plain backward extension is equally not without danger. I feel sure that the safest plan is to try for slow reduction by fixing the head to the top of the bed and using the inclined body weight as the reduction force. I have no experience of the ice-tongs method advised by Coleman and by Mackenzie, applied after making small superficial burr holes in the skull. Whether this is less irksome than the collar encircling below the chin and occiput is debatable, but intrinsically it is a sound method. It is certain that complete reduction is not easily achieved, or once achieved is not readily maintained. Most of my own cases and those of others that I have seen have been imperfect, though perfect examples have been obtained.

I shall leave the discussion of the treatment of the thoracic and lumbar injuries in other hands. There is no doubt that the Watson Jones[7] method is admirable in its results on the vertebral injury. So much must at once be admitted. The point that we have to discuss is whether it is an equally good method when there is neural damage. Its advantages are that it will reduce the bony displacement provided that that is not of too extravagant a kind, and that it will be likely to relieve the cord of pressure. But no method, however skilfully applied or resolutely carried out, will restore function to a severely injured cord—which means to a patient with a densely anaesthetic body. The advantages of reduction are that if the patient recovers he will be freed from the painful back which so often disables the workman. Against reduction are two points: (1) the fear of pressure sores in anaesthetic areas, and (2) the plaster case compels catheterization whether we like it or not. The best plan, in my view, is the making of a plaster bed for the severer cases, to be followed in a week or two by a plaster case if the improvement warrants it. This obviates the dangers of immediately enclosing a paralysed and anaesthetic patient, and so preventing any variation in treatment or observation of alteration in sensory levels.

REFERENCES

[1] Connors, J. F., and Nash, I. E.: *Amer. Journ. Surg.*, 1934, xxvi, 159.
[2] Denny-Brown, D., and Robertson, E. G.: *Brain*, 1933, lvi, 397.
[3] Idem: Ibid., 1935, lviii, 256.
[4] Hoff, E. C.: *Proc. Roy. Soc.*, 1932, cxi, B, 175.
[5] Holmes, Gordon: *British Medical Journal*, 1915, ii, 769.
[6] Jefferson, Geoffrey: *Proc. Roy. Soc. Med.*, 1927–8, xxi, Part I, 625.
[7] Jones, Watson: *Journ. Bone and Joint Surg.*, 1934, xvi, 30.
[8] Liddell, E. G. T.: *Brain*, 1934, lvii, 386.
[9] Riddoch, George: *Proc. Roy. Soc. Med.*, 1927–8, xxi, Part I, 637.
[10] Taylor, A. S.: *Ann. of Surg.*, September, 1929, p. 321.
[11] Thomas, Atha: *Colorado Med.*, 1928, xxv, 19.
[12] Watkins, K. H.: *Brit. Journ. Surg.*, 1926, xxiii, 734.

PULMONARY TUBERCULOSIS IN YOUNG ADULTS*

SIGNIFICANCE OF CONTACT HISTORY

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The serious nature of pulmonary tuberculosis developing in young adult life was clearly shown by A. S. MacNalty in 1932 in his report on tuberculosis,[1] in which he stated that "towards the end of childhood and in young adult life . . . approximately one-third of the deaths from all causes in males, and almost one-half of the deaths from all causes among females, are due to tuberculosis, chiefly of the pulmonary form." The problem of young adult tuberculosis has been and is being attacked from many angles, and we, in our present investigation, have attempted to ascertain the significance of certain of the aetiological factors, with special reference to the history of contact with persons known to be suffering from pulmonary tuberculosis.

The histories of 1,000 patients between the ages of 15 and 25 years in whom a diagnosis of pulmonary tuberculosis had been made have been carefully examined. In the case of those with a history of contact with any person suffering from definite pulmonary tuberculosis we have noted whether this person was: (*a*) one of the family—parent, brother, or sister—or other relative; (*b*) a friend in the same household or not; and (*c*) a fellow-worker with whom there had been close association. So far as was possible information has been obtained of the details of the length and degree of exposure to the infection, the age of the patient when thus exposed, and the early symptoms which led to the diagnosis of the disease. Other points of possible aetiological significance have been noted—namely, type of occupation, hours of work, and mode of travel to work.

Of our series of 1,000 patients suffering from pulmonary tuberculosis 663 had tubercle bacilli in the sputum and the remainder had definite clinical and radiological evidence of the disease; no patients with a doubtful diagnosis or with pleurisy alone were included in the series. Apart from this there was no selection of material.

We found, in recording the family histories, that a number of patients were ignorant of the cause of death of even their near relatives, or else were content with such terms as "asthma," "bronchitis," "pleurisy," or "congestion of the lungs." Although some of the diagnoses were suggestive of pulmonary tuberculosis, we have included these patients in the "non-contact" group, and have also put in this group those (twenty-six) cases with a family history of meningitis or surgical tuberculosis only. In all those who were exposed to infection outside the household contact had been definite and repeated. The contact group, therefore, includes only those patients with

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