MEDICAL PRACTICE

Occasional Review

Changing attitudes to the management of severe head injuries*

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British Medical Journal, 1976, 2, 1234-1239

Just 100 years ago this term young Victor Horsley entered University College, London, as a medical student. He was a founder of British neurosurgery. Some of his other interests have a contemporary ring. Thus he helped to provide the pathological proof of an outbreak of rabies among the deer in Richmond Park in 1888, while his friendship with Pasteur and his vigorous advocacy led to the muzzling of dogs and finally the quarantine restrictions which helped stamp out the disease from Britain at that time. We find him in 1912¹ reporting on the forcible feeding of suffrage prisoners and urging its reconsideration, echoed in October 1975 by the World Medical Association in the "Declaration of Tokyo," which set out the role of the doctor in relation to these and other practices in a manner which I believe Sir Victor would have approved. In his medicopolitical activities Sir Victor made strenuous efforts to change the constitution of the BMA with the establishment of the Representative Body proper, of which he was its first Chairman in 1903. All these contributions to medicine and society Horsley pursued with energy, concentration, and almost apostolic fervour.

Horsley made little direct contribution to the subject of cerebral concussion or the management of head injury, though he did study the mechanics of cerebral compression. Together with Kramer he also showed that intracranial pressure rose immediately after head injury and that the primary cause of death was due not to heart failure but to arrest of respiratory

centre function; thus death could frequently be prevented by artificial respiration.²⁶

Head injury. A dynamic problem

This emphasis on the role of an impeccable airway was revived in the early years after the second world war, dramatically demonstrated by the introduction of tracheostomy for some of these patients.

The fall in mortality rate, and the longer survival for other severely injured patients that followed these and other measures, led to a more critical examination of the clinical and pathological correlates of head injury. Several secondary changes—the so-called "second head injury"—which might be set in motion after injury were identified, while the recognition of major metabolic disorders after head injury and hormonal effects after injury to the pituitary body or its stalk or other parts of the hypothalamus followed. Injury to major intracranial vessels with subsequent infarction had been suspected for some time, but with the development of angiography speculation could be replaced by proof.

CEREBRAL OEDEMA

Cerebral oedema came under close scrutiny. Focal oedema around a laceration or contusion may develop, so that together with the area of injury it gives rise to cerebral compression, for which medical or surgical decompression may be needed. It became apparent that the dramatic and frightening generalised brain swelling that may occur shortly after injury and lead to death had, at least in part, a different explanation from the more gradual development of brain swelling some 24 hours or later after injury, secondary either to infarction or to multiple areas of injury. Langfitt *et al*² showed that immediate oedema

*The 16th Victor Horsley memorial lecture delivered at the Royal College of Surgeons of England on 11 December 1975.

was related to the enormous expansion of the vascular bed after vasoparesis, itself often due to hypoxia and able to be alleviated by restoring the blood gases to normal by artificial ventilation. Later brain oedema, on the other hand, was either true oedema of the brain, with an increase of extracellular fluid, or was due in part to an increase of intracellular fluid, for which hypertonic osmotic agents might be effective.

CEREBRAL CONCUSSION

The changes just described illustrate the dynamic process after head injury and that from the time of impact a series of changes may take place, some of which may be prevented by appropriate measures and others corrected. Central to this theme is the nature of cerebral concussion itself, and recent studies have made the older concept that cerebral concussion was essentially a transient state of unconsciousness without structural change and followed by complete recovery no longer tenable. Thus Windle *et al*³ showed experimentally that after mild concussion chromatolysis was present in the brain stem, while more recently in man Oppenheimer⁴ has demonstrated, in addition to anoxic cell changes and capillary haemorrhages, foci of clusters of microglial cells. This reaction, which is essentially a phagocytic response to trauma, has been demonstrated throughout the brain, even in patients who have sustained mild concussion and died later from other causes. At the other end of the scale, neurosurgeons have been aware for years of some patients who sustain a severe head injury and remain unconscious for weeks or months until death. At necropsy the brain may be macroscopically virtually normal; nevertheless, microscopically—as Strich⁵ has shown—there is diffuse degeneration throughout the white matter, and the total effect of such damage may be far worse than multiple areas of macroscopic focal contusion.

Cerebral concussion therefore has a pathological substrate, and the microscopical changes after the shearing strains induced can result in unconsciousness extending from minutes to weeks or months. Under this definition prolonged unconsciousness can be explained on severe concussion alone and does not necessarily imply the presence of macroscopic contusions or lacerations. Conversely there are many instances of brain contusion without any loss of consciousness, well illustrated in some penetrating missile wounds of the brain.

The significance of these findings has to be realised. For many years the “punch drunk” boxer syndrome has been recognised and has been well reviewed by Roberts.⁶ The finding of pathological changes even in mild cases of concussion must mean that successive head injuries may have an additive effect and that the patient will be susceptible to more serious effects after a further and apparently mild injury. So the implication for other sports such as horse riding and football, which carry especial risk of repeated head injury, is apparent.

To summarise, around the central core of cerebral concussion with its pathological changes are secondary effects of injury that may develop at various times after injury. In this setting I should now like to consider some of the present attitudes to the management of severe head injury.

Severe head injury

The definition we have adopted of a severe head injury includes those patients who are unconscious at the outset and remain so until death and, among the survivors from the early days, those who have a post-traumatic amnesia (PTA) of one week or more. Both in the Oxford and the Cambridge series our policy has been to admit all patients with concussion to hospital under neurosurgical care. In the former series of 7000 consecutive cases the mortality in the first thousand completed in 1952 was 9%, which over the succeeding years fell gradually to just under 4%. In the Cambridge series since 1961, of just under 10 000 head injuries, the overall mortality rate has remained

steady at about 3.7%. These figures emphasise that a head injury as such is an eminently recoverable lesion. But since policies for admission vary from hospital to hospital they may hide the incidence of severe head injuries as defined, which comprise some 6.5% of the total, and the much more limited outlook for those patients with prolonged unconsciousness of a month or more.

The attitude to the treatment of severe head injury may be illustrated by a brief reference to three major killing factors after head injury: intracranial haematoma, cerebral hypoxia, and increase of brain bulk.

INTRACRANIAL HAEMATOMA

The incidence of intracranial haematoma of all types in an unselected series is 6–7%. Untreated the mortality is extremely high, and major clots are fatal. Over the years the diagnosis has been essentially clinical and, indeed, save for a few, reliable. Nevertheless, a limit has been reached along these lines. Clearly if the mortality rate from this cause is to be lowered the diagnosis has to be made earlier. The clinical diagnosis—which rests on physiological changes in blood pressure, pulse, and respiration, lapse into coma, and particularly dilatation of the pupils—is based on the signs of tentorial impaction and severe brain stem compression. Hence at present treatment is usually undertaken late and is the main limiting factor in further improvement in the mortality rate. Clearly adjuncts are required to diagnose this further complication in patients with an already severe head injury. In recent years angiography, ultrasonics, and gamma encephalography have all been employed. Nevertheless, I believe that, apart from the use of angiography in the diagnosis of vascular injuries, these methods will be discarded in favour of transaxial computerised tomography (EMI-scanner). Here at last is a safe non-invasive technique which has the tremendous advantage that it can be repeated in the early days after injury and show reliably not only that a change has taken place but exactly where a clot is situated. Once the machines are generally available this use will surely become routine in the early diagnosis of severe head injury.

In addition, as Strang and Isherwood⁷ have pointed out, the EMI-scanner can demonstrate even small areas of contusion and thus provide further information on the sites of injury in the brain. Possibly as the technique improves further it may throw further light on the mechanics of head injury itself. If, as seems likely, this technique will demonstrate intracranial clots at a stage before disastrous brain stem compression and displacement has taken place, and lead to earlier surgery, undoubtedly not only will the mortality rate fall but the quality of survival will improve.

CEREBRAL HYPOXIA

Perhaps the commonest complication after head injury is respiratory insufficiency with resultant cerebral hypoxia. This may imperil injured but potentially recoverable cerebral tissue and affect adversely the quality of recovery; it will result in brain swelling with rise of intracranial pressure, infarction, and death. The insistence of an impeccable airway at all times and the development of tracheostomy for some cases was a significant advance in the active care of head injuries in the early 'fifties and mainly responsible for the fall in mortality rate seen at that time. Recent developments have stemmed from the observation that controlled ventilation lowers intracranial pressure. Rapid methods of determining the arterial O₂ and CO₂ saturations now make it possible to check whether respiratory exchange is adequate and to apply corrective measures at an earlier stage before irreversible damage has taken place.

Controlled ventilation

In practice, routine measures including endotracheal intubation and tracheostomy suffice for the majority, but some patients are left with arterial hypoxaemia even with the administration of oxygen. Since 1959 controlled ventilation has been used in such cases, at first where there was an accompanying chest injury and more recently with head injury alone, both to produce a higher oxygen supply to the brain and to reduce intracranial pressure. At Cambridge Dr Jean Horton has recently reviewed a series of 91 patients in which we have adopted this method of treatment. Previously the combination of a severe head

injury and a major chest injury (a not uncommon combination in motor car accidents) carried a high mortality rate. Now controlled ventilation is the treatment of choice for these patients: among 29 such patients 15 survived, of whom 12 made a good recovery. Severe head injuries along with respiratory insufficiency unrelieved by other means, may also benefit from this treatment: of 31 such patients, 15 survived, 11 of whom made a good recovery.

Controlled ventilation has its complications and should not be used routinely even in severe head injuries, where simpler measures usually suffice. On the basis of our results we have suggested criteria for its use: (1) intracranial pressure above 30 mm of mercury, and an inadequate response to the usual treatment; (2) uncontrolled convulsions and decerebrate spasms; (3) P_{aO_2} below 70 mm of mercury not responding to an increased concentration of inspired oxygen; and (4) a patient who is hyperventilating and has a P_{aCO_2} below 25 mm of mercury. If used, the method should be applied early and rapid blood gas analysis should be available in the admission room.

Hypothermia

A note on the use of hypothermia in the management of severe head injury may be made here. At the therapeutic level of hypothermia recommended—32°C (90°F)—the cerebral oxygen consumption falls by 25%. This lessens the risks of hypoxia, while the decrease of muscular tone and drying up of respiratory secretion aid the airway. Intracranial pressure also falls moderately as the temperature goes down. The technique involves the maintenance of this low temperature for an average of six days and calls for skilled nursing and medical supervision.* Our experience of this method in 121 cases suggested it has helped to lower the overall mortality: for example, in patients under 30 years of age in coma with decerebration but without gross increase of intracranial pressure, the use of hypothermia was associated with a recovery rate of 75%. This method of treatment, however, has not proved popular in many clinics, and probably with the development of controlled ventilation, patients will in future be treated mainly by controlled ventilation, particularly when they have raised intracranial pressure. Nevertheless, the possibilities of combining both hypothermia and controlled ventilation have still to be fully explored.

INCREASE OF BRAIN BULK

This is a broad descriptive term which avoids defining detailed pathological changes that are still imperfectly understood. It includes acute brain swelling from vasoparesis; brain oedema secondary to cerebral contusion or a haematoma or diffuse microscopical injury; and oedema after cerebral infarction resulting from hypoxaemia or direct vascular injury.

Besides preventing or correcting this complication by controlled ventilation or hypothermia as outlined, the hope for the future is that with the correct and early application of these methods it may be possible to prevent cerebral infarction, which is probably the most disabling complication that so limits the quality of survival.

Drug treatment

Drug treatment has a place. Osmotic agents, such as magnesium sulphate, to lower intracranial pressure have a time-honoured place in neurosurgical practice, but the introduction by Javid⁹ of intravenous urea heralded the development of a further range of drugs with similar action. Intravenous urea still remains the most reliable method in acute brain swelling and is effective for eight hours. Mannitol has fewer complications, but its action is shorter (four to five hours) and its effect not as dramatic in the severe case. Glycerol has the same disadvantages and given by mouth may lead to vomiting; we gave it up in the management of head injuries some years ago.

Our present practice in the presence of brain oedema is to give urea initially and thereafter to use small doses of mannitol monitored by intracranial pressure recordings.

The results of our efforts since 1949 to control cerebral oedema by the use of steroids—first with cortisone acetate, then prednisolone, and in recent years dexamethasone—have been disappointing. In this respect the poor response to dexamethasone stands in sharp contrast to its undoubted beneficial effect in cases of local oedema around brain abscesses and brain tumour. The added complications of steroid treatment also have to be remembered, so that we do not employ it routinely in the care of head injuries.

INTRACRANIAL PRESSURE MONITORING

In the majority of patients with severe head injury we make exploratory frontal burrholes, not only to exclude the presence of a haematoma but to determine the intracranial pressure as a guide to treatment. In the future probably haematoma will be diagnosed and checked again if necessary by the EMI-scanner. But the need to measure the intracranial pressure remains. Reliance on clinical observation alone is unwise when methods of treatment such as hypothermia and controlled ventilation are used. The neurosurgeon must know not only whether the intracranial pressure is raised or not at the time of exploration but the response to treatment in the succeeding days. For these reasons, continuous intracranial pressure monitoring was introduced, and we owe a debt to Lundberg¹⁰ for his work.

From our own experience with intracranial monitoring since 1970 we believe that it is a useful adjunct to treatment, particularly when special methods of treatment (all of which have their own complications) are being used. We use a subdural transducer in many instances since so often the very cases in which more help is required and the brain swelling is severe are those where it is difficult to place a ventricular catheter safely. Monitoring enables controlled ventilation to be adjusted to achieve a more satisfactory level of intracranial pressure. Small repeated doses of hypertonic agents can be strikingly effective and allows these drugs to be used more rationally.

But what are we measuring? We know, for example, that when measured simultaneously the ventricular pressure and the subdural pressure are about the same, but it should not be assumed that we are necessarily measuring the intracranial pressure throughout the head. There may be striking differences not only between the supra- and infra-tentorial compartments but also between one side and the other. Moreover, even if the intracranial pressure is normal, there may yet be some significant brain oedema present. Within these limitations, however, the technique is a help to a more logical application of some of the specialised methods of treatment.

External hydrocephalus

The dynamic nature of head injury is not only confined to the acute period. One example of a late effect which we have been concerned with is the development of external hydrocephalus after severe head injury. In many of these patients ventricular dilatation can be demonstrated by air encephalography, and in some, such dilatation may be accompanied by obstruction to the circulation of the CSF from the basal cisterns over the cortex. The development of shunting procedures for other causes of hydrocephalus and the description by Adams *et al*¹¹ of dementia accompanying normal pressure hydrocephalus led to consideration whether further failure to improve in some cases of severe head injury might be related to this cause. This is in fact so. In a study of just under 100 such patients who were still unconscious or in post-traumatic amnesia a month or more after head injury, by air encephalography and in some by RISA (radio-iodinated serum albumin) cisternography as well we found that a quarter of them had some external hydrocephalus present, severe in some.

So far 26 patients surviving after severe head injury have undergone ventriculoatrial shunting.¹² In nine of these the indication was obvious, since clearly the external hydrocephalus was producing raised intracranial pressure. By the same token the operation was effective and led to dramatic improvement with recovery in four, and improvement also in the other five, although not unexpectedly limited by the severity of the pre-existing underlying brain injury. We have recognised five other patients where deterioration after head injury, occurring from one to nine years later, was shown to be due to external hydrocephalus and improved by this operation. What, however, is more interesting for our present purpose is the response in two other groups. In seven cases where external hydrocephalus was present without increased intracranial pressure and the patients remained unconscious ("persistent vegetative state") for a month or more, dramatic improvement was seen in only one. Hence we believe at present that even if this complication is identified its correction is unlikely to help such patients and operation is probably indicated only in those few patients who have shown some improvement in the hope that one can further improve the quality of the result. A similar consideration applies to the last group of five patients, where clearly not only were the patients going to survive but significant improvement had taken place, only apparently to be halted between two and sixteen months after injury. It could be said that the further recovery that occurred in this group after operation would have happened in any event, but the unexpected improvement and the rate of improvement

in two of these patients left no doubt about the beneficial effect of operation.

This raises the intriguing question of how far one should treat more aggressively traumatic external hydrocephalus after severe injury, even though the patient is making some improvement. Will its correction improve the quality of the result? One has to be cautious in ascribing improvement after operation to the procedure alone, for obstruction to the CSF pathways does resolve in some cases.¹³ Nevertheless, our results so far seem to justify further work for this small but important group of patients.

Results

The mortality rate has fallen over recent years, but the question arises whether in striving for a lower mortality rate a number of patients are left so severely disabled that it would have been kinder had they not survived. What is the ultimate result in those who survive prolonged periods of amnesia, and are often left with neurological disability such as ataxia or hemiplegia or aphasia? What is the quality of survival?

We chose to study the consecutive first series of 7000 civilian head injury cases treated at Oxford. A total of 468 survivors who had had a post-traumatic amnesia for one week or longer were identified.¹⁴ The surgeon took no part in the follow-up, which was supervised by Dr Roberts and a team that included a clinical psychologist and a psychiatric social worker. These patients were followed and examined by the group from 10 to 25 years after injury. It says much for the industry of the team that of the total series only 11 patients could not be traced or examined. Though the results are still being analysed, some broad conclusions may be drawn. Firstly, provided recovery is being made within the first month after injury, the tendency ultimately to reach a practical recovery is high, even though it may take some years. The best results are in younger patients and in those where the injury is uncomplicated by penetration of the brain or a haematoma. Even in these latter groups, however, the same pattern is seen, although recovery may take longer, provided that vascular infarction has not occurred. The real predictive factor is not just that some recovery has taken place within the first month, but the rate of that recovery. In the total series, no fewer than a quarter of the patients were finally left with no detectable neural lesion, and this improvement was maintained.

But factors interrupt this process, and end with severe disability to the patient. Surgeons are familiar with the syndrome of the unconscious patient whose neurological condition deteriorates within hours or a few days after injury, and in the absence of an obvious haematoma. The fresh neurological signs in some of these cases leave no doubt that vascular infarction has taken place. All the pathological evidence suggests that infarction is a major factor in the later deterioration of some patients. Of patients left severely disabled, no fewer than two-thirds of those with a brain stem syndrome had shown obvious deterioration after admission to hospital; the same applied to a third of patients described by Roberts as left with a dystonic pseudobulbar pattern of a central neural lesion.

This brief summary suggests that after severe head injury improvement can go on for far longer than has commonly been supposed; that in the young patient coma and decerebration can be followed by excellent recovery; that it is the rate of recovery which is an accurate predictor of ultimate disability, even though it may only reach the stage of severe confusion within one month of injury.

PROLONGED UNCONSCIOUSNESS

Many years ago clinical observation showed that a period of unconsciousness of over one month separated sharply the majority of patients in whom recovery might be expected from a much smaller group where the prognosis was very different. This division has been confirmed by the follow-up study, and I will now comment on 139 patients alive and dead who remained unconscious for more than one month after injury and who have been followed up. This is a selected group since it includes also the series of 10 000 head injuries at Addenbrooke's Hospital, Cambridge, and from the non-missile head injuries treated at the Army Neurosurgical Unit from 1948 to 68.

The definition of this clinical state has been under discussion recently, and I have used "prolonged unconsciousness" to include the time the patient is in coma or only reacting to pain, proceeding to a stage where the patient is mute, often tetraplegic, and requiring artificial feeding. This state has been usefully summarised again by Giovanelli *et al*¹⁵ as an incomplete apallic syndrome in which "there is

recovery of vigilance with circadian rhythm, possibility of spontaneous eye movements to follow moving objects, notwithstanding persistent tetraplegia, lack of contact with environment and mutism." Undoubtedly, the more active treatment given to the unconscious patient during the acute period has led to more patients surviving in this group, but what is their ultimate outlook?

An earlier assessment¹⁶ suggested that after one month some two-thirds would survive the injury; that ultimately three-quarters of these could return to some work or run their homes, even though most were left with mental or physical disability, or both. Nevertheless, one-third of the survivors could make a recovery which was comparable to the preaccident state. This earlier estimate has been borne out in the longer and more detailed follow-up, although the years have added to the overall mortality. Of the 134 patients traced, 70 are now dead. With five exceptions where death was clearly due to other causes, it was decided that all the other fatalities should be assumed to have been related in some way to the head injury. The overall mortality after one month and within the first six months after injury of 33% had risen by the end of two years to 43%. This remained steady for some years—47% at five years—to reach the present mortality rate in this group at periods of six to twenty-four years later. The follow-up of the 64 survivors confirms that some patients can still make excellent recovery after this severe acute picture: 19 of these patients (30%) have ended up with only minimal physical disability and a further 29 have improved to the extent that they are at home or at work, although all patients have major disabilities either mental or physical or both. But together the two groups give a practical recovery rate of 75%. Only 16 of these patients remain totally dependent and unable to walk.

Much of the picture so far described seems to have emphasised the physical disability. Experience shows that the mental disability is as important. Indeed, it is the dominant factor that determines whether or not a patient can settle back in his home, or return to some form of work. This point has been studied again by Bond.¹⁷ The disabling patterns of mental impairment are mainly memory loss or frontal disinhibition or both, and in a few cases excessive irritability and rage. In the older survivors disabling levels of anxiety or depression may be seen. Nevertheless, considering mental and physical disablement together, we find that only 18 or 28% of the survivors still require constant care; 40% are gainfully employed; and in 16 (14%) the final disability could be called minimal and the recovery excellent.

These figures show the dangers of putting these patients into rigid groups without recognising, on the one hand, those patients in the group who can recover even though it takes time, return to work, and end with minimal disability; and, on the other, the need to search for predictive factors for identifying those patients who will remain unresponsive and demanding total care for months or years. It is essential to have an individual assessment and no blanket application of a predetermined formula.

So far as recovery is concerned the same two factors mentioned apply to this particular group of patients. Provided there is no added complication and the patient is young, a good recovery can still be made. As age increases the prospect of a good recovery diminishes. Indeed, in this series all the people with a good quality of survival were under 25 at the time of injury, while no patient over 45 who was initially comatose and decerebrate from the primary brain lesion (excluding haematoma) made a practical recovery (or even survived for a long period). The pattern during the first month can help, since failure to make any improvement whatsoever during this time carries a bad prognosis. So does fixity of the pupils (even though not dilated) for longer than the first 48 hours after injury. Persistent gross decerebration with tetraplegia after one month also carries an adverse prognosis.

This series reported here suggests that, although every year in Great Britain about 400 patients are admitted to hospital who remain unconscious for more than a month, the actual number who remain totally dependent for several years is small. It has been suggested that this is a useless life, unkind and unnecessary and even economically unjustifiable. I have always objected to the word "vegetable" because it carries the implication that these patients are not making any further contribution. I would say that their survival through the one standard of intensive care has meant a practical recovery for the majority. Those working in this field know that doctors, nurses, physiotherapists, and others attending these patients gain in their understanding of human life, and the same can be said for the relatives of these patients. In many instances they have been helped to adjust to life and understand sacrifice from the experience of an unconscious patient. No man is an island and whatever we do or not do

affects others in society. To very few is given the privilege of knowing that they have made a positive contribution to someone else. To suggest that one necessarily has to be conscious and speaking to make a contribution to others is an unwarranted assumption. There seems little justification for trying to measure the amount of care that should be given to these patients in terms of the economic health of the country. For without moral health there will be no economic health of any lasting duration. We should not lose sight of the fact that it is our responsibilities rather than our rights that are important to society, and this includes respect for the lives of others.

Finally, there are those patients who are put on to ventilators after respiratory arrest. There is a risk in the present controversy that the public may not be fully aware of the important role controlled ventilation now has in the treatment and recovery of patients. But there are those patients after head injury in whom spontaneous respiration stops and remains arrested together with fixed dilated pupils. Since 1949 we have normally put these patients on to ventilators. The decision is necessarily immediate and in the circumstances may be taken by a nurse or young doctor. In a few, such action may lead to corrective treatment for a previously undiagnosed condition. For most, however, it is the first stage in the death of the patient, but a ventilator is a far less harrowing experience than the other methods of artificial respiration, and both relatives and those attending the patient are assured that everything possible has been done.

There is no question that head-injured patients have prolonged survival because of ventilator treatment, as has been suggested in some places, confusing I believe different causes of unconsciousness. It has not been our normal policy to switch off ventilators, and the progress of these patients is that the blood pressure falls and most of them die within six days, with pupils fixed and dilated throughout this time, areflexia, no evidence of spontaneous respiration, and, finally, no circulation. Particularly in a few younger patients this period may extend up to 10 days. These findings derive from observations in several cases over the years; they should be emphasised, both to allay public anxiety and to alert them to the fact that by far the greater number of patients after head injury treated nowadays on a ventilator are undergoing care to prevent further complications and to ensure a good quality of recovery.

Pathology of survival

The recovery of patients with such severe brain injuries raises the question of what is happening in the brain to allow such recovery to take place. Cajal's statement is still true that "the regeneration of axones in the central nervous system of mammals is generally meagre and most abortive." Together with the observations that areas of brain tissue can be removed without obvious deficit to the patient this maxim suggests that recovery is due to compensation by other parts of the brain in a functional way only. In other words, true regeneration does not take place but functional reorganisation occurs. In most cases it is apparent that there is abundant reserve, and even with multiple diffuse microscopical injury throughout the brain the stage is not reached where pathways are completely disrupted. The observation that most anterior horn cells have to be destroyed in poliomyelitis before paralysis results is ample demonstration of the natural reserve in the central nervous system.

Recent studies suggest that there is another factor to be considered.

The development of neuromata after peripheral nerve section is well known. Similarly collateral sprouting from a divided spinal cord has been reported.¹⁸ Schneider¹⁹ has investigated neuronal growth after traumatic brain lesions in animals, and has found that the role of the dendrites is fundamental and their subsequent growth after injury seems determined not necessarily by a functional need but by specific factors that effect the growing of regenerating axones. The axones with their dendritic arbor compete with each other for the available space and like pruning a tree terminal injury can encourage sprouting from

further back. Thus in the newborn female Syrian hamster he has shown that after ablation of the superficial layers of the right superior colliculus anomalous connections of the right optic tract develop; while some axones grow into the damaged area to terminate, others grow across the midline into the undamaged opposite side. This at once raises the question whether such growth takes place in man after brain injury. An important factor seems to be the age of the animal, and this potential neuroplasticity seems more likely in the young. It is more than tempting to suggest that the better recovery after head injury in young children might be based on mechanisms of this kind. It is clearly speculative to suggest that here may be a physiological factor in the subsequent development of maladaptive function and behaviour.

Future developments

A further area of research concerns the effects of injury on neurotransmitter substances, transport systems, and other neurochemical changes. Lactic and pyruvic acid levels increase after brain damage, more so in the presence of oedema, and in the presence of anoxia. As long ago as 1926, Glaser²⁰ reported increased lactic acid levels in the CSF. More recently Kurze *et al*²¹ and others have attempted to use the ratio between the blood and CSF lactate levels as a prognostic factor in the outcome of brain injury. Acetylcholine levels in the CSF rise after injury and were described by Bornstein.²² This led Ward,²³ recognising the cholinergic component in the ascending reticular system and that acetylcholine can block synaptic transmission, to try the effect of large doses of atropine in an attempt to lighten the coma of these patients. Recently, studying ATP formation in brain mitochondria under ischaemic conditions, Ishii²⁴ has reported a sharp fall in the level and has also identified an endogenous inhibitor that accumulates rapidly in the brain under conditions of ischaemia. Injection of this inhibitor into the brain produces considerable oedema, and, more importantly, this effect can be blocked by serum albumin. In cats with cerebral compression he has reported improvement from coma with concomitant improvement in the EEG after the injection of a hydrolysate of egg albumin.

The role of RNA or substances that stimulate the synthesis of RNA in relation to memory merit further study. Looking at these patients after head injury with their multiple problems, one is frequently impressed with the adjustments many of them make to severe physical disabilities and the understanding shown by relatives and employers to personality disturbances. But so often it is the impairment of memory that is the telling factor which determines whether a patient can reintegrate himself into society once again. "Intelligence may be the pride, the towering distinction of man, emotion gives colour and force to his actions, but memory is the bastion of his being. Without memory there is no personal identity, there is no continuity to the days of his life. Memory provides the raw material for designs both small and great. Thus governed and enriched by memory all the enterprises of man go forward."²⁵

Conclusion

I have tried to show the dynamic nature of acute head injury, to describe some of the many problems that confront us, and to indicate the newer methods of investigation and treatment that should enable us to prevent the development of some complications and to correct others. The long-term follow-up of many patients with severe head injuries justifies this course and does not allow us to conclude that we have reached the limit in the management of these patients. Many of the advances will come from the meld of the basic disciplines of physiological experiment, pathology, and clinical observation. We recall today that these same disciplines were embodied with such success in one man when he was physician superintendent to the Brown Institute 90 years ago, and who is still remembered in the hot sun of Amara.

My thanks are due to Dr D Oppenheimer of Oxford, Professor I Isherwood of Manchester, and Dr Jean Horton of Cambridge for their help in supplying material; Mr E H Cornelius, librarian to the Royal College of Surgeons, and Mr F M Sutherland, librarian to the British Medical Association, for their help with the bibliography of Sir Victor Horsley; and to Miss Sheila Nicholson for secretarial help.

I wish to acknowledge my debt to the successive medical and nursing staffs of the accident services of the Radcliffe Infirmary, Oxford, Addenbrooke's Hospital, Cambridge, and the army neurosurgical unit at Wheatley and Colchester Military Hospitals who over the years helped in the care of the patients on whom this article is based.

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Letter from . . . Canada

The phantom crossroads

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British Medical Journal, 1976, **2**, 1239-1240

The acknowledged British genius for compromise is always in danger of degenerating into an attempt to have cake and to eat it at the same time. Dismally bewailing the loss of professional independence within the National Health Service, while at the same time extolling its virtues and enjoying its benefits, is an excellent example of this exercise.

A national health service as opposed to national health insurance is bound to have the advantages of security and the disadvantages of central control. Doctors cannot have it both ways.

Future of professional independence

The observer could not fail to be fascinated by the contrasts offered at the recent tripartite meeting of the BMA, CMA, and IMA in Dublin. Both British and Canadian speakers repeatedly voiced their concern about the future of professional independence. But the viewpoint, the philosophy, and the style were quite different. The British seniors, like so many well-groomed gannets, each with his own niche on the monolith of the National Health Service, comfortably ensconced on his merit award, and

ignoring the humble puffins at his feet, nictitated in mild disapproval at the raucous, disorderly flock of egalitarian Canadian gulls. The Canadians, guardedly satisfied with their comprehensive scheme developed in partnership with government, but fully aware of its faults, while discussing their efforts to control abuse, rather ingenuously exhibited only its seamier side. They were then quite taken aback by moralistic attacks on their method of charging fees only for service rendered; especially when these attacks issued from British colleagues, who, while enjoying the excellent Hibernian hospitality, were also drawing full pay. Their juniors were doing the work at home. Yet in the next breath these urbane and supremely competent men were bewailing their loss of freedom. The profession can't have it both ways. A disciplined hierarchy means control from above. A non-rigid system of piecework means some will try to abuse it. Both mean working with government, but in case of disagreement the Canadian doctor can opt out and bill his patient. After 25 years of salary and sessional and capitation payment the British feel that this is somehow "not quite cricket."

A common contemporary oratorical posture among the medical politicians of old Albion is to stand looking into the middle distance, the right arm extended and the left hand clasped over the heart while proclaiming: "British medicine is at the crossroads." Nonsense. There has been no crossroad since 1946, for the very good reason that a crossroads means that there is a viable alternative route available to the profession and, far more important, available to the patients. Since that date British medicine has been on the broad high-road towards a full-time salaried state service. Now rearguard action is becoming increasingly difficult without resorting to a withdrawal of services,

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