C J Mieny, U Mennen: Principles of Surgical Patient Care - Volume II

Chapter 4: Neurosurgery

Chapter 4.1: Head Injuries

M J van Rensburg

Introduction

In the UK more than one per thousand of the population are admitted to hospital every year because of head injuries, and 70% of fatal road accident victims have had a head injury.

With a disease of this magnitude it is obviously impossible for every patient with a head injury to be under the care of a neurosurgeon. A great many injuries occur far from a neurosurgical unit, and many head injuries constitute the lesser component of a patient with multiple injuries. It is therefore important for every doctor in a country practice and for every surgeon, irrespective of his specialty, to be familiar with the essentials of head injury care. Injuries which demand admission to a neurosurgical unit are in the minority, and complications necessitating transfer to a neurosurgical unit are not very common if the basic care of the patient is good. Some complications, such as most extradural haematomas, may have to be dealt with at the admitting hospital as the delay inherent in transfer may be intolerable.

It is cause for concern that head injuries, which are probably the commonest cause of death in the young, are generally managed so poorly. This leads to unnecessary loss of life as well as to additional functional curtailment in the survivors. The pity is the greater because meticulous care of the head injured patient is not usually difficult and is not shrouded in neurosurgical mysticism.

Classification

In order to have a perspective of the problem, it is necessary briefly to review head injuries and their major complications. There are different waysx of classifying head injuries, but the following one, based on whether the injury is open or closed, is useful:

Closed Head Injuries

- without closed depressed skull fractures
- with closed depressed skull fractures.

Open Head Injuries

- compound depressed fractures of the skull
- stab wounds and other low-velocity penetrating injuries of the brain
- missile injuries
- communication established with the exterior through the middle ear, cribriform plate, or a paranasal air sinus as the result of a basal skull fracture with tearing of the underlying dura mater.

Surgical Pathology of Head Injuries

Closed Head Injury

Impact Damage

At, and following the moment of impact, the brain undergoes injury as a result of local deformation of the skull at the point of impact as well as from subsequent linear and rotational acceleration and deceleration forces. The injuries which result fall into one or more of the following categories:

- concussion
- contusions and lacerations
- diffuse white matter injury.

Concussion

Cerebral concussion is usually described as a condition characterized by brief loss of consciousness, followed by recovery without any localizing neurological signs.

It was thought in the past that there was no structural basis for the condition and it was assumed to be functional in nature. Considerable work has been done in the recent past without producing a single final answer. It seems likely that in the end the common path for traumatic lesions that cause transient unconsciousness is inactivation of a sufficient part of the cerebral cortex, possibly as a result of shearing lesions with tearing of a few axons and reversible stretch of many. Other mechanisms of ultra-structural type such as temporary synaptic disruption may be postulated, and alterations in neural mitochondria have been described. Immediate persisting coma is probably due to widespread wide matter damage resulting in disconnection of large areas of the cerebral cortex.

Contusions and Lacerations

Predictably, contusions and lacerations tend to affect the summits of the gyri, but may extend into the underlying white matter. There is almost invariably extravasation of blood and at times this is so marked that it becomes a matter of semantics whether such a lesion is called a contusion or a haematoma.

Contusions and lacerations may occur under the point of impact and are frequently responsible for the focal neurological deficit that may be present when they underlie a depressed fracture of the skull. The commonest sites for such lesions are, however, on each side of the stem of the sylvian fissure where it accommodates the sharp edge of the lesser wing of the sphenoid bone. They thus straddle the sylvian fissure and occur in saddle-fashion over the posterior part of the orbital surface of the frontal lobe, the temporal lip, and adjacent undersurface of the temporal lobe. From here they may extend deeply into the brain substance.

Wherever the impact has been, contusions tend to be most marked in the frontal and temporal lobes. They are usually more marked on the same side of the brain as a skull

fracture, but bilateral contusions are common, and the major damage may be contrecoup and thus on the opposite side to a fracture or the point of impact.

Contusions as seen on computerized tomographic (CT) scans may be surprisingly extensive without the injury having led to loss of consciousness. They may, however, be followed by swelling and delayed haemorrhage which may cause brain shifts and a depression of consciousness.

Diffuse White-Matter Lesions

In 1956 Strich described extensive white matter damage in patients surviving for several months in the vegetative state. She attributed these lesions to tearing of nerve fibres at the time of impact.

Support for Strich's work has come from Adams et al who found that patients showing this type of lesion were always deeply unconscious from the outset. A fracture of the skull was much less common in these patients than in other types of fatal head injury. Contusions were few but were constant in the *corpus callosum* and superior cerebellar peduncle. The brains appeared misleadingly normal on external examination. Microscopic examination, however, showed signs of extensive axonal damage, the histological picture varying with the age of the lesion.

In their post-mortem examination of 151 patients who died from head injury, they could not find a single patient in whom signs of injury were restricted to the brain stem. They are thus in agreement with other authors that the clinical picture of coma and decerebrate rigidity which often leads to a diagnosis of "brain stem injury" has as its neuropathological basis diffuse bilateral hemispheral injury extending down as far as the upper brain stem, with the lesions being of lesser severity at the lower levels. Such lesions may functionally disconnect the brain stem from higher influences and give rise to the same neurophysiological consequences as midbrain transection.

The medical attendant clearly has no control over the impact that has been described above. He can, however, do much to lessen the incidence and severity of the unwanted epiphenomena and complications which may follow and lead to what has aptly been described as the "second head injury".

Secondary Brain Damage

Raised Intracranial Pressure

If intracranial pressure (ICP) is raised without causing brain shifts, as occurs when the cerebrospinal fluid (CSF) volume is artificially augmented, very high levels of ICP are tolerated without the production of symptoms. Under these circumstances it is only when cerebral perfusion starts diminishing that symptoms of brain ischaemia arise. Conversely brain shifts and hernias may occur without intracranial hypertension having been sustained, although obviously pressure gradients within brain tissue must have occurred. Usually, however, such brain shifts are accompanied by raised intracranial pressure (RICP), and intracranial pressure

monitoring is a useful guide in assessing the threat which an intracranial mass lesion proses to the patient.

During the first few hours and days after head injury, we are concerned with the development of brain swelling and haematomas which cause brain shifts, occasionally in the presence of only a modest rise of ICP. The pathological hallmarks of brain shifts in the supratentorial compartments are:

- a line of brain necrosis in the cingulate gyrus along the edge of the *falx cerebri* as the result of a subfalcine hernia

- a similar line of necrosis in the parahippocampal gyrus along the tentorial edge, signifying transtentorial herniation

- sometimes infarction of the medial occipital cortex due to compression of the posterior cerebral artery across the tentorial edge.

The oculomotor nerve may show evidence of compression in its passage across the petroclinoid ligament, and haemorrhage may occur into it here. The midbrain may be compressed from side to side and elongated in its anterior-posterior diameter. Haemorrhage and infarction may occur in it as a result of distortion and disruption of its blood vessels. Displacement against the opposite tentorial edge may cause grooving of the cerebral peduncle, producing the so-called Kernohan-Woltman notch, and this is undoubtedly the cause of the paresis which may occur ipsilateral to the haematoma causing the shift.

Space-occupying lesions of the posterior fossa may cause downward herniation of the cerebellar tonsils through the foramen magnum. Some degree of cerebellar tonsillar ectopia is a not uncommon normal finding so that care must be exercised in accepting this as evidence of a hernia unless the rim of the foramen magnum has produced a line of pressure necrosis on them.

As may be imagined, brain shifts producing compression, stretching, and displacement of cerebral tissue, with traction on, and disruption of blood vessels, may profoundly disturb brain function and destroy neural tissue. These shifts are the common cause of death in patients who survive the impact injury, and could be demonstrated in 83% of fatal head injuries in the Glasgow series.

Brain Swelling

Brain swelling is common after head injury, and it is at this that our efforts at treatment, other than surgical, are directed. The swelling is due either to oedema or to vascular engorgement.

Oedema, termed vasogenic and due to disruption of the blood-brain barrier, occurs in and around contusions and round haematomas, and may be recognized on the CT scan as increased lucency of the white matter.

The so-called cytotoxic oedema which follows on ischaemic cellular damage is less easily detected on the CT scan, as it initially affects the grey matter, and tends to erase the normal difference in density between white and grey matter on the scan.

In children, particularly, diffuse brain swelling may occur as a result of vascular engorgement and, in these young patients, may follow relatively trivial injuries which have not caused immediate loss of consciousness. This reaction to head injury was found by Bruce et al in 29% of 214 children and adolescents up to 18 year old studied with CT scanning, and in 41% of those admitted with a Glasgow Coma Scale (GCS) score of 8 or less.

Ischaemic Brain Damage

Considering the condition of many patients after head injury, and especially after multiple injuries, it is not surprising that anoxic and ischaemic brain damage are common. The ICP may be raised with an equivalent reduction in cerebral perfusion pressure; cerebral vasospasm may be present; and there may be hypotension from shock, anaemia from blood loss, and hypoxaemia from ventilatory insufficiency. This is a formidable array of problems for the brain to cope with, particularly as the autoregulatory ability of its blood vessels is often defective after trauma.

Excepting infarction of the medial occipital cortex due to occlusion of the posterior cerebral artery in patients with transtentorial herniation, the most common sites for lesions are in the peripheral zones of supply of the major cerebral arteries. This is not unexpected and leads to the so-called border zone, or water-shed, infarcts in the boundary zones between the areas of distribution of the major vessels.

Infarction within the territories of distribution of the anterior and middle cerebral arteries is also common and is often bilateral. Multiple foci of ischaemic cortical damage may occur, and cortical ischaemia is usually associated with ischaemic lesions in the basal ganglia, and often in the cerebellum.

The frequency and danger of ischaemic brain damage makes it mandatory to maintain an adequate cerebral perfusion pressure by attempting to keep both intracranial pressure and systemic blood pressure within normal limits. It is also important to maintain the oxygen-carrying capacity of the blood at optimal levels.

Intracranial Haematomas

Extradural Haematomas

Extradural haematomas may occur anywhere between the dura mater and bone, including the skull base, but are most common in the anterior temporal region in relation to the main trunk of the middle meningeal artery, and less common more posteriorly in a temporo-parietal position in relation to the posterior branch of the middle meningeal artery.

Fractures are present in up to 90% of adults and in about 70% of children, probably because the greater elasticity of the child's skull allows teraing of the dural vessels without fracturing of the bone. The haematomas are of arterial origin in over half the cases, in 40%

the origin is venous from dural and diploic veins, and in 10% there is diffuse oozing from the dura mater.

Extradural haematomas are usually surgical emergencies with delay in evacuation adding significantly to the mortality rate.

Subdural Haematomas

Significant subdural haematomas may develop under two very different sets of circumstances.

The more common variety of acute subdural haematoma is part of a significant head injury which has rendered the patient immediately unconscious. The bleeding may come from a bridging vein or surface artery but more commonly it is an extension of an intracerebral haematoma through a brain laceration. The deterioration that commonly occurs a few days after injury is usually due to swelling of the brain, although further delayed haemorrhage may occur. The long held view that the mortality rate in these patients is over 90% and that operation is hardly indicated cannot be condoned. Our experience is similar to that of Seelig et al who found that the mortality rate in patients who underwent evacuation of the haematoma within four hours was 30%, which was of the same order as the mortality rate in patients with severe head injury, but without haematoma. They also found that decerebrate rigidity was of very poor prognostic import, and that only 29% of such patients had a functional recovery.

Rather than the exhibition of a nihilistic attitude the approach should therefore be one of the greatest urgency.

The second type of subdural haematoma is that which occurs with little or no brain damage and often presents with the clinical picture of a lucid interval so commonly regarded as the hallmark of the extradural haematoma. The bleeding occurs either from a subarachnoid artery, often one in which a lateral hole has been torn by the avulsion of a side branch which bridges the subdural space, or by tearing of bridging veins during movements of the brain within the skull. This type of haematoma is particularly common following non-vehicular trauma such as assaults and domestic accidents. In the series consisting mainly of such patients and reported by Shenkin, over 60% of these haematomas were due to the rupture of a cortical artery.

The potential for recovery is obviously greater than in the first variety, and the need for haste as great as with an extradural haematoma.

Many subdural haematomas are either not detected or not treated in the acute stage and present later as expanding intracranial lesions, often without a history of antecedent trauma. They are sometimes arbitrarily divided into subacute, and chronic, a haematoma which becomes clinically manifest between the second day and second week after trauma often being referred to as subacute.

The acute haematomas are hyperdense on CT scanning usually for the first week, after which they become less dense and eventually isodense with the adjacent brain at some period

between nine days and six weeks. Following this they become progressively more hypodense. The reasons for the enlargement of the haematomas are still debated. Repeated haemorrhages certainly occur into some, but the demonstration of isotonicity between venous blood, CSF, and haematoma in our opinion in no way invalidates the theory that fluid is drawn into the haematoma because of osmotic forces, generated mainly by the breakdown of red cells with liberation of haemoglobin and its products. Any osmotic difference would, of course, be immediately corrected by diffusion of water into the haematoma.

Intracerebral Haematomas

Following the introduction of CT scanning it was found that traumatic intracerebral haematomas, which were previously considered to be relatively uncommon, are in fact common lesions.

The mechanism producing these haematomas is probably mass movement with shearing stresses causing disruption of blood vessels. Deep contusions may lead to softening and necrosis of the brain, with haemorrhage. This is presumably an important etiological factor in the development of the delayed haematomas which usually occur within 48 hours and which were originally described by Bollinger in 1891. Gudeman et al thought that "dysautoregulation" in damaged brain led to increased intravascular pressure in these vessels with consequent rupture.

Posttraumatic intracerebral haematomas may be single or multiple, superficial or deep. The superficial haematomas may arise from a cortical contusion or they may be subcortical. Intracerebral haematomas may be surprisingly silent or demand speedy evacuation.

Traumatic haematomas in the region of the basal ganglia appear to have the same poor prognostic import as does diffuse white-matter injury, or for that matter diffuse brain injury as manifested by extensive petechial haemorrhages. McPherson et al considered that basal ganglia haematomas are due to angular acceleration/deceleration forces with shearing of blood vessels. In a third of the patients they found radiological evidence of diffuse white-matter injury as evidence by a haematoma in the *corpus callosum* or superior cerebellar peduncle, or intraventricular blood in the absence of a juxtaventricular haematoma. On the basis of their experience they concluded that basal ganglia haematomas, which are rarely of surgically significant size, are evidence of severe primary brain damage and are of poor prognostic import.

Open Head Injuries

Compound Depressed Fracture of the Skull

Depressed fractures not due to high-velocity injuries are usually associated with only focal brain damage and many patients do not lose consciousness at the moment of impact as there is often no significant acceleration-deceleration force in operation. Focal neurological deficit is dependent on the depression overlying an eloquent area of the brain. Dural tearing is frequent, as is an underlying contusion or laceration, and intracerebral haematoma, with or without an associated subdural haematoma, is not unusual. Significant epidural clots are surprisingly infrequent.

Stab Wounds and Other Low-Velocity Penetrating Injuries

If the knife, screw-driver, or other puncturing agent has been removed, the wound may be very deceptive due to the minor external evidence of its passage. Even if there is no neurological deficit, the possibility of vascular injury, or of the development of intracranial infection, must be kept in mind and the patient managed accordingly. The slot-fracture that is often produced by a stab wound of the skull may be easily overlooked on a plain X-ray film.

Missile Injuries

Missile injuries vary from those inflicted by the pellet of an air gun through the common civilian injuries inflicted by the low-velocity bullets of hand guns, to high velocity injuries consequent on wounding by hunting and military rifles and projectiles.

The damage inflicted thus varies widely. A low-velocity bullet may do little more damage than a stabbing instrument, while a high-velocity missile may produce extensive brain damage in its passage and scatter fragments of bone and metal well beyond its tract.

Basal Skull Fractures and Dural Tears

A CSF leak from the nose or external auditory meatus is incontrovertible evidence that communication between the sub-arachnoid space and the exterior has been established. Its cessation does not necessarily indicate healing of the fistula, but usually only insecure plugging of it by brain tissue. Similarly its absence with basal skull fracture does not imply that such a communication does not exist, only that it is not clinically obvious.

Meningitis may occur in a patient who has never leaked and in one in whom the leak has stopped. The important diagnosis is therefore that of basal skull fracture with dural tear, not that of CSF rhinorrhoea or otorrhoea, which are merely symptoms of the former.

The Early Management of the Head Injured Patient

Respiration

The airway must be open ands respiration must be adequate. This is the first and great commandment.

It would be only a slight exaggeration to state that the management of the head injured patient consists of the maintenance of adequate cardio-respiratory function. Many patients with relatively minor head injuries die of asphyxia before reaching hospital, and unfortunately 40% of unconscious patients develop pulmonary complications within 12 hours, and respiratory failure is indeed often present on admission to hospital, particularly when there have also been thoracic and abdominal injuries.

Every unconscious patient is continuously exposed to the danger of respiratory obstruction, be it from his tongue or dentures dropping back, or from aspirating gastric contents or accumulated pharyngeal secretions. As a result he may die, sustain anoxic brain

damage, or initiate pulmonary complications which may eventually kill him. But it is common knowledge that the organism needs oxygen to survive.

What is often less clearly understood is the influence of intrathoracic events on intracranial pressure (ICP).

The volume of intracranial blood is under normal circumstances the most important factor in the determination of ICP, and the only one over which we have some voluntary control. Oversimplifying a little, it can be stated that intracranial blood volume depends on two mechanisms, namely the arterial input and the venous drainage. And both these factors are profoundly dependent on what happens in the thorax.

An increase of intra-thoracic, and therefore of central venous pressure (CVP), may produce a tremendous rise of ICP as a result of venous congestion of the brain. This sequence of events occurs with each cough and episode of restlessness and, in a sustained way, with respiratory obstruction. It is hardly necessary to add that venous congestion is likely to increase oedema and may lead to haemorrhage from damaged intracranial veins.

An increase of intracranial blood volume occurs as a result of increased input due to cerebral vasodilation when the PaO_2 falls, and particularly when the $PaCO_2$ rises. Cerebral blood flow changes by about 1.8 mL/100 g brain/1 mm Hg change in $PaCO_2$ and cerebral blood volume by about 0.04 mL/100 g brain in the normal person. Apart from any anoxic brain damage it may cause, respiratory insufficiency thus produces a rise of ICP. The deduction is, of course, that a patient must not be exposed to even transient respiratory obstruction or insufficiency. This causes cerebral anoxia, congestion and oedema, and may lead to cerebral haemorrhage, particularly in a contused brain.

To prevent this sequence of events from occurring, every patient who cannot maintain the patency of his own airway must be subjected either to naso-tracheal intubation or, if necessary, to tracheostomy. The patients who should be intubated are therefore those who are, by definition, in coma. These are patients who do not utter recognizable words and who do not obey commands. Bruce advises intubation of patients with a Glasgow Coma Scale (GCS) score of 7 or less, which usually means much the same thing.

If spontaneous ventilation is inadequate, mechanical ventilation must be instituted forthwith.

Respiratory obstruction can usually temporarily be dealt with easily by cleaning the pharynx and inserting a pharyngeal airway. The latter is a temporary measure because it does not separate the vocal cords, does stimulate secretions, and almost completely inhibits the swallowing reflex. The inevitable result of this is aspiration of accumulated pharyngeal content. The head must not be rotated as this produces a marked rise of ICP, probably due to kinking of the vertebral venous plexus. In addition there is almost always the possibility of a cervical spinal injury being present. If it is imperative, such as when there is rapid haemorrhage into the pharynx, the patient may be temporarily placed in a semiprone position.

It is advisable to view X-rays of the cervical spine before extending the neck during intubation. Intubation is therefore usually performed after the radiological investigations have

been done. However, if the procedure is mandatory to ensure airway patency, it should not be postponed. If there are fractures of the facial skeleton or an injury of the cervical spine it may be necessary to perform an early tracheostomy if the introduction of an endotracheal tube is thought to be undesirable or is found to be difficult.

Circulation

The clinical picture of surgical shock with hypotension and tachycardia is not produced by primary brain damage or intracranial haemorrhage. In such patients a careful search should be made for the source of haemorrhage, keeping in mind that a scalp laceration may have bled profusely. An intracranial haemorrhage may, however, produce clinical signs of blood loss in an infant with its small blood volume and expandable skull. As is discussed in the chapter on the management of raised ICP, surgical shock is often a prelude to intracranial hypertension as the maximally vasodilated cerebral vessels become perfused by blood at normal pressure in the resuscitated patient. This is particularly likely to occur in children and warrants the early placing of an ICP monitor in such patients.

Associated Injuries

Fractures, particularly of the facial skeleton and upper limbs, will be present in a high percentage of patients. Injuries to the thorax and the rupture of abdominal organs such as the spleen, liver and kidneys are common and must always be kept in mind. Injuries to the bladder and urethra must be excluded. An indwelling urethral catheter will yield a sample of urine for analysis and will serve to monitor urinary output.

Neurological Status

In the unconscious patient the neurological examination is perforce basic, and the level of consciousness and possible presence of limb weakness or abnormal posture can be determined within a few minutes. The establishment of sucha baseline is not only of value in estimating the severity of the brain injury but is also of great importance in the recognition of complications.

A number of charts have been devised for this purpose, but the one which has found wide international acceptance is the Glasgow Coma Scale (table 4.1.1) devised by Teasdale and Jennett. The Glasgow Coma Scale (GCS) is widely used in the neurosurgical literature and has the merit that it lends itself to the reasonable standardization of clinical material by defining the severity of the injury.

Table 4.1.1. Glasgow Coma Scale and Score

Eyes open	Score
- spontaneously	4
- to speech	3
- to pain	2
- never	1
Best verbal response	
- orientated	5
- confused	4
- inappropriate words	3
- incomprehensible sounds	2
- none	1
Best motor response	
- obeys commands	6
- localizes pain	5
- withdraws limbs	4
- abnormal flexion	3
- extensor response	2
- none	1

The maximum score assignable is 15 and the minimum is 3.

It is not applicable to infants and toddlers and for them other scales have been devised such as the Neonatal Arousal Scale proposed by Duncan and Ment and the less complicated Children's Coma Score (CCS) devised by Raimondi and Hirschauer (table 4.1.2).

Table 4.1.2. Children's Coma Score

Ocular response	Score
- pursuit	4
- extra-ocular muscles (EOM) intact, reactive pupils	3
- fixed pupils or EOM impaired	2
- fixed pupils and EOM paralysed	1
Verbal response	
- cries	3
- spontaneous respiration	2
- apnoeic	1
Motor response	
- flexes and extends	4
- withdraws from painful stimuli	3
- hypertonic	2
- flaccid	1

The maximum score assignable is 11, and the minimum is 3.

In all these scales numbers have been allocated to each neurological observation. The lowest score that can be obtained in each of these scales is 3, but the highest in the GCS is 15 and in the CCS 11. The scores of 12-15 in the GCS measure higher integrative functions which, as pointed out by Raimondi and Hirschauer, cannot be measured in the infant. In their scale more points are allocated to the grading of brain-stem functions than in the GCS.

After establishing the level of consciousness the size of the pupils is noted (in millimetres) and their reaction to light evaluated. Small pupils which react to light are indicative of pontine damage while midposition pupils which do not react to light are found in midbrain damage. A large pupil on one side may be the result of trauma to the eye or to damage to the third cranial nerve, either at the time of impact or as a result of brain shift and transtentorial herniation.

Signs of focal brain dysfunction are noted. The head and eyes deviate to the side of a frontal lesion and away from a unilateral pontine lesion. The presence of pareses of the face and limbs is noted as are abnormal postural responses. The strength or posture of each limb is noted as follows (in the terminology of the Glasgow Observation Chart):

- Normal power
- Mild weakness
- Severe weakness
- Spastic flexion (applicable to upper limb only)
- Extension
- No response.

Injuries to the Head

The presence of scalp lacerations and of open depressed fractures is noted. Bilateral orbital haematomas (raccoon eyes), bruising in the region of the mastoid process, and bleeding from the ear suggest fractures of the skull base. Cerebrospinal fluid rhinorrhoea or otorrhoea are proof of such a diagnosis.

Lacerations of the scalp must be cleaned and foreign material, such as hair, removed. A careful finger in a scalp wound will detect most depressed fractures but is not a substitute for X-rays of the skull. Wounds must be debrided, irrigated and sutured. A depressed fracture may be elevated up to 24 or 36 hours after injury without significantly increasing the risk of infection. Penetrating wounds should be covered with a sterile dressing after haemostasis of the scalp is obtained. If and when the patient is stable he should be transferred to a neurosurgical unit if it appears likely that he will survive the onslaught. Of the penetrating injuries only high-velocity missile wounds usually require urgent operation because of the extensive damage which is so often inflicted.

Patients who present with the clinical picture of an extradural haematoma must usually be operated on without delay at the hospital of admission.

Radiological Investigations

After the patient has been fully assessed clinically, and resuscitated if necessary, X-rays of the skull, cervical spine and chest are obtained as a routine, with additional investigations as dictated by the clinical examination. It is appropriate to subject patients in coma to CT scanning at this time, and in them skull X-rays may be reasonably omitted.

Fluid and Electrolyte Balance

The high incidence of the SIADH has been discussed in relation to the water and sodium requirements of the patient during the first few days following admission.

The later onset of the SIADH is, however, much commoner than the early syndrome. It becomes manifest during the second and third weeks after trauma, and most commonly on the ninth day. Besides the brain injury there are often other factors which favour the release of ADH. Intermittent positive pressure ventilation, presumably by reducing the transmural pressure in the atria of the heart, is a potent stimulus. Pain, prolonged immobilization in a supine position, increased room temperature, and certain drugs, such as barbiturates, may play a role in the liberation of ADH.

The best treatment is fluid restriction, but in established cases the induction of diuresis with mannitol or furosemide, and the administration of hypertonic saline, may be necessary.

Diphenylhydantoin inhibits the secretion of ADH but we do not have experience of its use for this purpose.

Obviously many different forms of fluid and electrolyte disturbance may occur in patients with head injury, but it would not be productive to discuss them all. One of them, namely diabetes insipidus, does, however, merit brief discussion.

Diabetes insipidus is probably about one tenth as common as the SIADH in patients with head injury, occurring in something like 3% of cases, and is often associated with basal skull fractures. Whenever the urine output exceeds the fluid intake by 250 mL in one hour and more significantly by 1000 mL in a four-hour period, and if the urine SG is less than 1,005, diabetes insipidus is suspected. If the serum osmolality is greater than 300 mOsm/L and urine osmolality is less than 200 mOsm/L, and if the serum sodium is greater than 145 mOsm/L, the diagnosis of diabetes insipidus is made. If the diabetes insipidus is mild, with a urine output which does not exceed fluid intake by 1000 mL in 24 hours, the shortfall can be made good by additional oral or intravenous fluids. Otherwise vasopressin (ADH) or desmopressin acetate (DDAVP, Rorer Pharmaceuticals) administration greatly simplifies the maintenance of fluid balance. We have found the latter product, usually by intranasal administration, very useful.

While keeping the common abnormalities of fluid and electrolyte disturbance in mind, the maintenance of the patient in as near a perfect balance as possible is no different in head-injured patients than in other surgical patients.

Nutrition

It has in the past been common practice to provide nutritional support for the head-injured patient only by the enteral route. Feeding is then delayed until the return of gastrointestinal function, commonly a week or more after injury. This practice is clearly not in the patient's best interest. Rapp et al compared the results of total parenteral nutrition (TPN) commencing within 48 hours of admission, to those of standard enteral nutrition (SEN). Of the 18 patients in the SEN group eight (44%) died during the 18 days of data collection, whereas none of the 20 patients in the TPN group died during this period. There was thus a highly significant difference in survival at 18 days in this well-controlled prospective randomized study. The authors felt that the basis for the favourable effects of TPN may have been the enhanced immunological response to infection by the nutritionally supported patients. They found that the serum transferitin, serum albumin, and total lymphocyte count declined in both groups but to a lesser degree in the TPN group. One cannot fault their conclusion that TPN prevented early death in their patients and provided the opportunity for neurological recovery in a high proportion of cases. In a recent publication the same group found that these patients had made a significantly better neurological recovery by day 18 and at three months, but not at six months and one year. They feel that their daya supported the contention that neurological recovery during the first three months following head injury is more rapid in patients with better nutritional support.

The magnitude of the metabolic response to trauma in head-injured patients is not widely appreciated. They may excrete massive amounts of nitrogen in the urine of similar magnitude to patients with burns over 20-40% of their body surface. Nitrogen excretion of up to 35 gm/day was found by Clifton et al and the high rates of excretion were shown to persist through the fourth week in patients who remain comatose. These patients received steroids (dexamethasone 24 mg/day) but the authors cosidered that this probably did not substantially alter nitrogen excretion. This view was vindicated by the study of Young et al in patients who did not receive steroids and who showed an almost identical metabolic expenditure and nitrogen excretion.

Resting metabolic expenditure (RME) in the patients (unsedated) of Clifton et al was variably raised to 250% of normal. In most patients the protein caloric contribution exceeded 20%, so that considerably more calories were derived from protein catabolism than is usual in trauma to other body regions. This was thought to be possibly related to muscle tension as gross motor activity was present during measurement. Values of caloric consumption of 5000 to 12000 Kcal per 24 hours were measured when patients spontaneously postured. In three patients who were either heavily sedated, paralysed, or in barbiturate coma, the RME was approximately 90% of normal. Although nitrogen excretion in normal man is closely linked to RME, this may not be the case in head injury. Clifton et al found large nitrogen excretion in some patients with low RME, particularly those who had been paralysed and sedated, which suggests that RME and nitrogen excretion may be influenced by different factors.

The findings of Gadisseux et al were largely in aggreement with those of Clifton et al. They found the mean nitrogen output to be 199 mg/kg/day in patients who did not receive any nutritional support, and during nutritional therapy it rose to 357 mg/kg/day. In the 23 patients studied a positive nitrogen balance was achieved for one day in one patient only,

despite administration of up to 496 mg/kg/day of nitrogen. Values of caloric expenditure ranged from 42.7% to 234% of BMR. They found significant hypometabolism in patients who received barbiturates or neuromuscular blocking agents, and in brain-dead patients. When these patients were excluded, they observed a rise of caloric expenditure from 114% of BMR on the first day to a mean peak value of 170% of BMR between days five and eleven. They concluded that:

- nitrogen loss, when steroids are not administered, is similar to that occurring after skeletal trauma

- hypometabolism follows the administration of barbiturates and neuromuscular blocking agents

- energy expenditure increases up to a peak of 170% of BMR during the 12 days after injury.

In severely but variably hypermetabolic and hypercatabolic head-injured patients our information is still too inadequate to lay down dogmatic nutritional guidelines. Hyperalimentation with the object of meeting caloric expenditure and achieving positive nitrogen balance is not usually successful in achieving this, and it is not without its own problems. Thus the administration of large amounts of glucose may aggravate the hyperglycemia which is often present. It may also lead to increased oxygen consumption and carbon dioxide production, and therefore energy expenditure. This causes respiratory stress and may impede weaning from ventilation. With parenteral hyperalimentation, fluid overload must be guarded against, and infection remains a constant risk. It must, however, be kept in mind that infection is not confined to patients with intravenous catheters. Deutschman et al reported that 11 of 43 patgients on nasotracheal intubation, each also with a nasogastric tube, developed paranasal sinusitis necessitating removal of the transnasal tubes.

To optimally meet caloric expenditure and nitrogen loss without overfeeding would mean the regular measurement of RME and urinary nitrogen excretion. This is not a practical routine in most units. The intolerance of most patients to enteral feeds of more than 2.5 to 3 litres places a limit on enteral feeding and the problems of solute loading hypertonicity must also be guarded against. Nevertheless Clifton et al were able to achieve the provision of 150% of the resting metabolic expenditure in most of their patients by enteral feeding without great difficulty. The risk of excessive fluid administration similarly limits parenteral feeding.

Clifton et al advise that enteral feeding by continuous administration be commenced, if possible, within 48 hours of injury with the goal of providing at least 0.24 gmnitrogen/kg body mass/day. If enteral feeding is not tolerated within 48 to 72 hours they advise intravenous hyperalimentation as such patients usually do not tolerate enteral feeding for many days. Standard nutritional supplementation is aimed for, again providing about 0.25 gm nitrogen/kg body mass/day.

We are in agreement with these recommendations.

The RME may be modified by the administration of beta adrenergic blocking agents, sedation, neuromuscular blocking agents and anti-pyretics. The excessive muscular activity

which occurs with decerebrate or decorticate posturing should be avoided or minimized by sedation and reduction of stimulation or, optimally, by endotracheal intubation with paralysis and ventilation.

Respiratory Care

It is not essential to provide assisted ventilation for a head-injured patient if his own efforts are adequate to maintain his PaO_2 and $PaCO_2$ at acceptable levels. Outside the confines of an intensive care unit spontaneous ventilation is undoubtedly safer, and assisted ventilation should be used only for very specific indications. The employment of hyperventilation to treat raised intracranial pressure is considered in the appropriate chapter of this book.

There is no arbitrary time limit beyond which an endotracheal tube should be replaced by a tracheostomy, but if it is obvious that the patient will require prolonged ventilation, tracheostomy may as well be done early. Tracheal toilet is much easier to do through a tracheostomy, and if secretions are a problem, a tracheostomy can produce a dramatic improvement in a patient's condition. If an obtunded patient's jaws are wired together, a tracheostomy is a wise precaution in providing an avenue for tracheal suctioning in the event of aspiration.

Urinary Bladder

A catheter is introduced initially to monitor urinary excretion and to prevent distension of the bladder. Later a condom catheter is usually preferred in men.

Rectum

Both diarrhoea and faecal impaction must be guarded against.

Skin

Pressure sores must be prevented by regular turning of the patient without rotation of the head, as this may precipitously raise intracranial pressure.

Eyes

Drying of the cornea must be prevented by keeping the lids closed and by introducing an eye ointment.

Restlessness

A source of discomfort such as a full bladder (or a developing intracranial haematoma) must be excluded. Valium is useful for sedating patients, and if pain appears to be responsible, a cocktail of pethidine, chlorpromazine and promethazine, 12.5 to 25 mg of each intramuscularly, is effective.

The effect of muscular activity on the metabolic expenditure of the patients has been discussed and it has been shown to be responsible for the high caloric requirements of these patients.

Raised Intracranial Pressure

The treatment of raised intracranial pressure, and especially that caused by head injury, is considered in a following chapter.

Prophylactic Anticonvulsants and the Treatment of Seizures

I am in agreement with Young et al that there is little to support the use of prophylactic anticonvulsants following head injury and that phenytoin should be used only after a seizure has occurred. It should be promptly administered intravenously in order to prevent additional seizures and a dose of 18 mg/kg body mass given over a 30 minute period would seem appropriate. A daily maintenance dose of about 5 mg/kg should be instituted to maintain the blood level between 10 and 20 micrograms/mL, although it is the experience of Young et al that blood levels as high as 25-30 micrograms may be required to control early seizures.

The Management of the Injured Brain and Its Coverings

Closed Head Injuries

Without Depressed Fracture of the Skull

Most of these patients will require no more than the general supportive care that has been described.

A certain number of patients will be shown to harbour significant intracranial haematomas or large space-occupying contusions on admission, and many of these will require urgent evacuation. In other patients haematomas will enlarge in the few days following admission, or new ones may appear. These problems are best dealt with by the neurosurgeon, and their speed of evolution is usually such that the patient who has been carefully monitored by means of the Glasgow Coma Scale may be transferred safely to a neurosurgical unit.

The role of the surgeon, other than a neurosurgeon, in the operative management of a closed head injury is confined to the evacuation of a haematoma manifesting with a speed of evolution which clearly prohibits transfer to a neurosurgical unit. Haematomas which dramatically declare themselves are usually situated extradurally. Acute subdural haematomas may, however, give rise to the same clinical picture, particularly those which are not associated with severe brain damage.

The clinical indication for the removal of an intracranial haematoma is found in a patient who has not sustained an overwhelming intrinsic brain injury, and who subsequently deteriorates. The implication of this is that the neurological status of the patient, and in particular his level of consciousness, must be carefully documented on admission, and as

carefully monitored thereafter. It also presupposes that the metabolic and respiratory care of the patient has been meticulous, as hypoxia, hypercapnia, electrolyte disturbances, and brain swelling are more common causes of deterioration, although not usually with the dramatic speed of the developing haematoma. The presence of an extradural haematoma is thus suspected in a patiet:

- who was not unconscious after injury or

- whose conscious level had improved after injury or

- who was still stable for a period after injury and who then deteriorates with the exhibition of one or more of the following features:

  - a diminishing level of consciousness

  - the development of a hemiparesis

  - the enlargement of a pupil

  - the development of decerebrate rigidity

  - and possibly an inconstant lowering of heart rate and rise of blood pressure.

The most important early observation is a depression of the level of consciousness which indicates that displacement of the brain stem has already occurred, with impairment of the reticular activating system. If intracranial pressure has been monitored, a rise of pressure will usually precede clinical deterioration by a considerable period and it serves as a valuable early warning signal. The appearance of a dilated pupil and of decerebrate rigidity are signs of severe brain shift and of transtentorial herniation, and they are indications of impending death. Compression must then be relieved with the greatest urgency. The outcome in patients with extradural haematomas is closely related to the rapidity of treatment.

The most common situation of an extradural haematoma is in the anterior temporal region due to injury of the main trunk of the middle meningeal artery, but an injury of the posterior branch of this vessel will commonly produce a posterior temporal haematoma above and behind the ear. Haematomas may of course occur anywhere, and frontal and parietal situations are not unusual. Bleeding is then often from bone, a dural sinus or vein, or from a peripheral branch of a meningeal artery.

A haematoma which merits special mention occurs under an occipital fracture as the result of a tear in the transverse sinus or a tributary vein. Although the haemorrhage is of venous origin, the evolution of symptoms may be very rapid, with early impairment of vital functions as a result of brain-stem compression, and without the development of hemiparesis, a dilated pupil, or decerebrate rigidity.

Where such facilities are available, surgery is usually preceded by CT scanning or cerebral angiography to confirm the presence of a haematoma and to demonstrate its situation. However, even in a neurosurgical unit, the speed of deterioration may very occasionally be

so rapid that a burrhole may have to be placed under unsterile conditions in the ward. The only procedure which takes precedence over such an operation is the intubation of the patient.

If there are no facilities for special investigations, or if the condition of the patient demands immediate operation, the situation should be managed as follows:

If time allows the whole head is shaved and the position of bruises, abrasions and swelling noted. The likely side of the haematoma is:

- Virtually always (94%) on the side of the dilated pupil or of the pupil which dilated first.

- Usually (82%) on the side opposite a hemiparesis or the most abnormal motor response.

- Commonly (66%) on the side of, and usually under, a skull fracture.

- On the side of visual evidence of trauma to the head.

After a decision has been made concerning the probable side of the haematoma its likely situation is estimated as follows:

- Where a fracture line crosses a meningeal vascular groove or dural venous sinus.

- Under a swelling, bruise, or abrasion of the scalp, particularly when this corresponds with the above.

If there is no clue as to the situation of the haematoma, burrholes are placed as follows until it is found:

- Over the main trunk of the middle meningeal artery about 2 cm above the zygomatic arch and behind the temporal hair line. About 75% of extradural haematomas will be found here.

- Over the posterior branch of the middle meningeal artery about 2 cm behind, and just above, the tip of the ear. This is the second most common location of an extradural haematoma.

- About 4 cm from the midline and immediately behind the position of the normal frontal hairline, anterior to the coronal suture.

- About 4 cm from the midline in the parietal region, about 10 cm above the inion.

- Symmetrically on the contralateral side after the possibility of a haematoma over the transverse or sagittal sinus has been considered.

These burrholes are diagnostic and the skin incisions are placed so that they can be connected to form a skin flap, if required. If no haematoma is found, there is no reason for

self-recrimination; if no negative explorations are ever done, some haematomas will surely be missed.

If an extradural haematoma is found, it is usually necessary to enlarge the burrhole with a bone rongeur. The evacuation of the haematoma is simple and haemostasis is not usually difficult.

If an extradural haematoma is not found, the dura mater must be inspected for the purplish discoloration often imparted by an underlying subdural haematoma. The finding of a thin layer of subdural haematoma on incising the dura is common and does not require further attention. A larger subdural haematoma can usually be evacuated at least partly before transferring the patient to a neurosurgical unit if his condition warrants it. Apart from space-occupying contusions and intracranial haematomas there are other indications for neurosurgical intervention in these closed head injuries (such as hydrocephalus) but these fall clearly within the ambit of the neurosurgeon. Many of the patients will, of course, develop raised intracranial pressure from brain swelling, without the presence of a surgically significant haematoma. Their detection and management is considered in the chapter on the treatment of raised intracranial pressure.

Closed Depression Fractures of the Skull

Not all closed depressed fractures of the skull require elevation. The incidence of epilepsy is not reduced by doing so. However, if the fracture is so severely depressed as to constitute a space-occupying lesion, or if it is associated with a neurological deficit, it is of course advisable to elevate it. Even in the latter event it is likely that an underlying cerebral contusion is etiologically of more importance, as focal signs often start improving before the depression has been relieved. Cosmetic considerations may be important, such as when a depression is situated over the forehead.

It is good policy to seek neurosurgical opinion if it appears advisable to elevate a closed depressed fracture.

Open Head Injuries

Compound Depressed Fractures of the Skull

The surgical treatment of most compound depressed fractures of the skull should fall within the ambit of competence of general surgeons and of those general practitioners who practise operative surgery. There are not enough neurosurgeons available to manage them all. If the surgical practitioner is unwilling to operate on an uncomplicated depressed fracture of the skull, it is unlikely that he will accept the responsibility for evacuating an acute extradural haematoma, with predictably tragic consequences.

Depressed fractures which involve the frontal air sinus or which are situated over the major venous sinuses are best managed by neurosurgeons. In most cases of fractures situated over the venous sinuses, if he can be sure that the soft-tissue injury has received proper care, the neurosurgeon will elect to leave well alone.

Compund depressed fractures are treated in accordance with basic surgical principles, with debridement of all the layers of traumatized tissue. Careful dural closure is obtained, employing a graft harvested from the adjacent periosteum, if necessary.

As with all open injuries, the incidence of infection rises with the passage of time before closure but not significantly so before 36 or even 48 hours have passed. During this period the replacement of the larger bone fragments within the skull defect, even in contaminated wounds, do not appear to increase the risk of infection. This obviates the need for cranioplasty in virtually all the patients.

Antibiotics are usually prescribed only if infection is suspected or if debridement has to be delayed for some particular reason, usually one dictated by associated injuries.

Stab Wounds of the Skull

The patient who gives the history of having been stabbed in the skull must be evaluated with great care, as must the patient whose scalp or facial injury has the appearance of a stab wound. The slot fracture of a knife wound of the skull may not be visible on X-rays, not does a transorbital stab wound orindarily leave radiological evidence of its occurrence.

Such patients are preferably managed in a neurosurgical unit because of the danger of injury to major cerebral vessels, particularly with transorbital stab wounds, and also to guard against the development of cerebral abscesses and haematomas. All patients with suspected stab wounds of the skull undergo computerized tomographic scanning on admission, followed by cerebral angiography if a haematoma requires surgical evacuation. We have found an aneurysm of the distal anterior cerebral artery in association with a haematoma within hours of the injury. Appropriate angiography is in any event carried out at the beginning of the second week to exclude the development of a traumatic aneurysm, which must be surgically treated if found.

Missile Injuries

Unless the prognosis is clearly hopeless, these injuries should be treated in a neurosurgical unit.

It has been reasonably suggested by Nagib et al that patients with bihemisphere injuries and with a GCS score of 3 to 5 on arrival, particularly with a scattering of bone and metal fragments away from the bullet track, should not be operated on. A similar approach was suggested for comatose patients with unilateral multilobar injuries when there was scattering of bone and metal fragments. They felt that all other patients should be vigorously treated.

All patients in whom the decision to treat has been taken, should undergo computerized tomographic scanning. Cerebral angiography is done to clarify specific problems such as suspected major vessel injury. The principles of surgery remain conventional with debridement of injured tissue, evacuation of necrotic bran and of haematoma, and removal of bone and metal fragments when accessible. Firm dural closure is effected, if necessary with the help of a cranial periosteal graft. Must has been made of the necessity for the removal of

all retained bone fragments to reduce the incidence of infection. On the basis of the American experience in Vietnam, Meirowsky advised reoperation for the removal of a single retained fragment. We would concur if the retained fragments are large, but small scattered fragments must perforce be left behind and have not in our experience frequently led to abscess formation. This would seem to be in accord with the experience of Nagib et al and is supported by Cooper in his "Comments" on this article.

The mortality of missile wounds is high, and in the case of civilian gunshot wound to the head it is in the region of 66%.

Basal Skull Fracture

As has been said, the detection of a traumatic CSF leak is merely evidence of the presence of a basal skull fracture with the establishment of a communication between the subarachnoid space and the exterior. The finding of intracranial air on radiological examination or the occurrence of meningitis have the same meaning. And clinical indications that a basal skull fracture has occurred, such as haemorrhage from the external auditory meatus, bruising over the mastoid process, and bilateral orbital haematomas, should alert one to the possibility that a leak may have been established. The absence of an over CSF leak does not mean that it is not present, as a certain tempo of leaking is necessary for it to become clinically obvious. Nor does its cessation mean that healing of the skull fracture has taken place. Studies which we have done in such patients have shown the appearance of a radio-isotope (Tc99m) in the nasopharynx of the great majority in whom it had been introduced into the lumbar subarachnoid space. In our experience a nasal leak rarely really ceases. In contrast, CSF otorrhoea virtually always safely stops, although it must be kept in mind that healing of the tympanic membrane may convert otorrhoea into rhinorrhoea.

Conventional wisdom dictates that CSF leaks which persist for longer than 10 to 14 days, or are of delayed onset, should be repaired. Persistence of intracranial air or the occurrence of an attack of meningitis are similarly regarded as indications for operation. Certain authors recognize additional indications such as extensive frontal skull fractures involving the sinuses, a broad fracture line, and an angulated spicule of bone.

We accept all the above indications, but remain suspicious of a CSF rhinorrhoea which has ceased, even if there is no detectable injury to the paranasal sinuses on CT scanning. It has been mentioned that isotope studies have shown the persistence of a leak in most such cases seen by us. We now use this study broaden our indications for surgery with the result that most patients who have leaked are subjected to repair. I feel that Lewin is probably correct when he states that all patients who have had CSF rhinorrhoea should be operated on whether the leak has clinically ceased or not.

In the setting of the intensive care unit, and before the repair of the fistula becomes a consideration, the question of antibiotic cover in an attempt to prevent meningitis usually arises. Not surprisingly authors differ greatly in the adive they offer. Thus Bakay and Glasauer consider prophylactic antibiotic administration mandatory and suggest broad spectrum cover. Jennett and Teasdale consider penicillin to be the most rational choice for prophylactic cover as the usually sensitive pneumococcus is the commonest infecting organism. They feel that the threat of haemophilus may justify ampicillin in children, but that

broadspectrum antibiotics should be avoided in general, because of the risk of breeding resistant organisms. They think it wise to take nasal or aural swabs in order to be able to predict the infecting organism.

In a prospective study employing penicillin (20 million units daily) and a placebo Klastersky et al could find no evidence to support or rehect the use of penicillin in these circumstances. They felt that available retrospective studies indicated that no conclusion could be drawn regarding the use of prophylactic antibiotics in acute traumatic cerebrospinal fluid fistula.

Ignelzi and VanderArk in a study of 129 patients with basilar skull fractures came to the conclusion that there was no evidence that prophylactic broadspectrum antibiotics prevented meningitis, but that they may alter the nasopharyngeal flora to potentially more invasive organisms. The study of McGee et al on their own patients did not yield statistically significant results, but the results of their review of the literature came down fairly heavily against the use of prophylactic antibiotics.

The use of penicillin, as suggested by Jennett and Teasdale, appears to be harmless and may doubtfully be of value in reducing the incidence of pneumococcal meningitis without significantly changing the nasopharyngeal flora.

On the whole, however, there is much to support Spetzler and Wilson's forthright rejection of prophylactic antibiotics on the grounds that they have been shown to be ineffective in both a retrospective and prospective study, their potential for causing meningitis by an unusual pathogen, and the development of pneumococcal meningitis despite prophylactic therapy with appropriate drugs.

Conclusion

The prognosis of a patient with a head injury obviously depends on many factors, such as the age of the patient, the severity of the injury, the occurrence of intracranial and other complications and, not least, the skill with which he is cared for. The outcome is usually evaluated according to the scale proposed by Jennett and Bond with the best outcome designated Grade 1 and the poorest Grade 5.

- Grade 1: Good recovery. This implies resumption of the previous pattern of life even though there may be minor neurological and psychological deficits.

- Grade 2: Moderate disability. These patients are disabled but independent, and can work in sheltered employment.

- Grade 3: Severely disabled. These are patients who are dependent for daily support because of mental or physical disability, and usually a combination of both. They may be able to maintain self-care within a room or house.

- Grade 4: Persistent vegetative state.

- Grade 5: Dead.

The severity of a head injury is difficult to quantify, but the GCS score does allow for an approximation, as it is fairly closely related to outcome. In their vigorously treated patients Young et al found that 95% of patients with initial GCS score of higher than 7 had a favourable outcome and 95% with initial scores of less than 5 had an unfavourable outcome. In the intermediate groups with GCS scores of 5, 6 or 7 prognosticating was more complex and they found that accurate predictions could be made only when the score later changed to greater than 7 or less than 5. When deterioration below 5 occurred no patient of theirs survived. The sooner the score improved to above 7, within the first 24 hours, the more likely was a favourable outcome, and the longer a patient remained in the intermediate catergory, the more likely was an unfavourable outcome.

Other factors which are related to the severity of injury such as the reactivity of the pupils to light, the nature of eye movement, the speed and pattern of respiration, the level of blood pressure and temperature, are not considered in computing the GCS score. Their consideration does not, however, appear significantly to improve predictive ability.

As may be expected statistics of outcome vary but the overall results of Marshall et al are representative of those units which treat head injuries vigorously. They analysed 100 consecutive patients who complied with the definition of coma on admission or at some time thereafter. All patients who were alive on admission were included. They found that 60% of their patients made a good recovery or were moderately disabled, that 4% were severely disabled, 8% vegetative, and that 28% died.

From this study and others like it, it is clear that the vigorous treatment of patients with severe head injuries does not result in a large percentage of vegetative survivors. And there can be few experiences in surgery more gratifying than the prompt recovery following operation of a patient dying of an intracranial haematoma.

Chapter 4.2: Delirium, Coma and Brain Death

C H van der Meyden

Delirium

Delirium is the Latin word for furrow, having the figurative meaning of being deranged, crazy, or out of one's wits. Other terms with the same meaning include "confusional state", "acute organic brain syndrome" and "everyman's psychosis". Delirium has become the dominant term since Lipowski's work and monograph on the subject. It refers to the clinical condition in which there is an acute, diffuse, transient dysfunction of the content of consciousness (attention, perception, memory, learning and thought). The anatomical substrate involved includes the reticular activating system, the limbic system, the basal ganglia and the cerebral hemispheares. In metabolic, concussion, ictal and drug-induced forms of delirium the latter may well form a transitional state between alertness and coma.

Predisposing factors for the development of delirium include:

- age over 60 which reflects a clinical state of lower metabolism and excretion of drugs and a lower reserve of organ function

- alcohol and drug addiction

- previous brain damage

- other more relative factors such as

    - psychological stress

    - sleep deprivation

    - sensory deprivation and overload

    - immobilization.

It has been suggested that a cholinergic deficiency may be the central common denominator in the development of delirium. Many drugs cause delirium by their anticholinergic properties. Cholinergic mechanisms are involved in the states of wakefulness and arousal. In hypoxic delirium the psychological dysfunction precedes the failure of neuronal energy production and probably reflects a neurotransmitter dysfunction in which acetylcholine may be playing a key role. The progressive electroencephalogram slowing in delirium probably reflects an underlying reduction in cerebral metabolism.

Clinical Definition and Assessment of Delirium

The following acute though usually transient symptoms develop:

- Disorientation: which is tested by the patient's ability to orientate for time, day, week, month, year and person.

- Lack of attention: which can be assessed by the serial subtraction test of sevens or threes.

- Short-term memory loss: can be tested by the patient's inability to recall five things or objects after five minutes.

- Perception disturbance: with the development of delusions, illusions and/or hallucinations.

- Disturbance of the sleep/wake cycle: manifested by the development of insomnia or excessive daytime sleepiness.

The Etiology of Delirium

The incidence of delirium in the surgical wards and intensive care unit is approximately 8% and 20-30% respectively. Although metabolic and drug causes are the most common, epilepsy, meningitis, encephalitis, subarachnoid haemorrhage, hydrocephalus, and a unilateral cerebral mass lesion should be considered.

Drugs play an increasingly important role as a cause of delirium (see table 4.2.1). The chances of a drug causing the delirium is high when the history, the physical examination and the emergency biochemical investigations fail to yield an etiological clue. On the suspicion of intoxication the appropriate samples of blood, urine and gastric contents are sent for screening tests.

Table 4.2.1. Drugs Causing Delirium

Alcohol

Anticholinergics (and drugs with such action)

- Tricyclic antidepressants
- Antihistamines
- Antiemetics
- Antispasmodics
- Ophthalmic preparations
- Most neuroleptics
- Belladona alkaloids

Dopamine Antagonists

- Levodopa
- Bromocriptine
- Alpha methyldopa

Anti-Tuberculosis Drugs

- Isoniasid
- Rifampicin
- Ethionamide

Propanolol and Digitalis

Oncological Drugs

- Methotrexate

- Cyclophosphamide

- 5-Fluorouracil

- Asparaginase

Otherrs

- Indomethasine

- Cimetidine

- Lithium.

Patients with cancer frequently present with delirium. Predisposing factors include:

- age over sixty

- cerebral metastases or malignant meningitis

- infiltration and destruction of the liver, the lungs, kidneys (or other organs)

- associated infections

- chemotherapeutic agents

- radiotherapy

- remote effects of malignancy and anaemia.

Always consider hypothyroidism and porphyria. Hypothyroidism may be associated with hypothermia, hypotension, hypoventilation, dilutional hyponatraemia and conincidental adrenal insufficiency.

Post-Operative Delirium

Post-anaesthetic emergence delirium occurs in approximately 5% of patients. The anaesthetic agents associated with delirium include cyclohexane, methohexital, methoxy flurane, halothane and the anticholinergics. Halothane increases cerebrospinal fluid pressure and cerebral blood flow and decreases cerebral oxygen consumption and it does not protect the ischemic brain. Withdrawal delirium, mostly due to alcohol, may accout for approximately 10% of cases. Post-operative convulsions are associated with hypertensive encephalopathy,

the use of cyclohexane, halothane and flurane anaesthetics and with the transurethral prostatectomy procedure. The elderly patient is especially sensitive to the development of post-operative pneumonia and bacteraemia secondary to catheterization.

Major catastrophes during anaesthesia are frequently associated with the occurrence of cerebral hypoxia secondary to oesophageal intubation, aspiration and hypotension.

High-Risk Associations with Delirium

- Cardiac bypass surgery: This has an approximately 10% incidence of post-operative delirium. Possible contributing factors include:

    - duration and severity of heart disease

    - duration of cardiopulmonary bypass time

    - hypotension

    - hypothermia

    - hypocapnia

    - hypoxia

    - microembolisation

    - arrhythmias

    - anticholinergics

    - penicillin encephalopathy.

- Cataract surgery: The factors of old age dehydration, anticholinergics and sensory deprivation are of importance.

- Transurethral resection of the prostate: Water intoxication may occur when non-electrolyte irrigating solutions are mistakenly used.

- Burns: The surface area involved; infection and electrolyte disturbances play a role.

Hypoxia and Hypoglycaemia

Oxygen and glucose are essential substrates for cerebral metabolism. The brain has no capacity to store oxygen and very limited reserves of glucose. A deficiency of oxygen or glucose or both will lead to delirium, coma and death within seconds to several minutes (< 4 minutes). In hypoxic hypoxia there is a deficiency of oxygen at alveolar level and it can be caused by pneumonia and pulmonary embolism. In anaemic hypoxia there is too little haemoglobin available to carry the oxygen and it is caused by severe anaemia and carbon

monoxide poisoning. Ischaemic hypoxia is decreased cerebral perfusion with a resultant deficiency of both oxygen and glucose and it is caused by oligaemia, hypotension, severe pulmonary embolism and cardiac arrhythmias. With hypoglycaemia there is too little "fuel" to utilize the oxygen. Histotoxic anoxia secondary to cyanide poisoning occurs rarely. The cerebral cortex, especially the occipital and parietal regions, the basal ganglia and the cerebellum, are selectively more sensitive to hypoxia.

Reactive hypoglycaemia is rarely associated with delirium and other neurologic symptoms and should not be regarded etiologically important unless:

- a clear association between the symptoms and a low glucose level exists

- relief of symptoms occurs by glucose administration and

- there is evidence of an associated stress response (anxiety, tachycardia, sweating).

Surgical causes of hypoglycaemia include:

- post-gastrectomy hypoglycaemia

- retroperitoneal mesenchymal malignancies

- pancreatic islet cell tumour hypopituitarism and hypoadrenalism.

Electrolyte Disturbances and Delirium

- Hyponatraemia (less than 134 mEq/L)

- Hypernatraemia (more than 150 mEq/L)

The normal osmolarity of the blood is 280 ± 5 mOsm/L. This can be calculated roughly by doubling the serum sodium level and adding the mOsm of glucose, urea and other solutes (triglycerides, mannitol and glyceral).

The approach to the hyponatraemic patient includes the assessment of the hydration state which can be divided into:

- normal hydration, usually due to inappropriate secretion of antidiuretic hormone

- overhydration and oedema associated with excessive intravenous hypotonic fluids

- renal failure

- cardiac failure

- cirrhosis and the nephrotic syndrome

- dehydration and hypotension associated with diuretics

- diarrhoea

- vomiting

- burns and tissue trauma.

Major clinical abnormalities are unusual with a serum sodium above 120 mEq/L. The measurement of the serum and urine osmolality and the urinary excretion of sodium per day assist in making the correct etiological diagnosis.

The patient with the inappropriate secretion of antidiuretic hormone has normal hydration, low serum osmolality, high urine osmolality and excretes more than 20 mEq of urinary sodium daily. The correct diagnosis is essential to prevent overloading the patient with saline when fluid restriction is the appropriate treatment. Drugs associated with the induction of this syndrome include:

- oxytocin

- vasopressin

- chlorpropamide

- clofibrate

- thiazides

- vincristine

- cyclophosphamide

- phenothiazines

- tricyclinc antidepressants.

Other causes include a variety of central nervous system and chest disorders and neoplasms of the bronchus, duodenum and pancreas.

Clinically, hypernatraemia is associated with delirium, convulsions and coma, pathologically, with brain haemorrhages (including subdural haematoma) and venous and sinus thromboses. The more common causes include:

- diabetes insipidus

- diabetes mellitus

- osmotic diuretic drugs

- excess fluid loss associated with diarrhoea, fever and burns

- administration of corticosteroids

- hypertonic fluids and excessive amounts of $HaHCO_3$

- and rarely, neurogenic hypernatraemia secondary to hypothalamic or pituitary disease.

Hypophosphataemia

Hypophosphataemia usually occurs on a background of a chronic wasting disease such as chronic alcoholism, diabetes mellitus, intestinal dysfunction, anorexia nervosa and cancer. Decreased phosphate intake is further complicated by long-term intravenous feeding, hyperalimentation, and glucose and insulin therapy. Clinically hypophosphataemia can cause muscular weakness, delirium, seizures and coma.

Hypo- and Hypercalcaemia

Clinically hypocalcaemia manifests with tetany, seizures, delirium and movement disturbances. Its causes include:

- chronic renal disease

- gastrointestinal disturbances such as malabsorption, pancreatitis and liver disease

- hypoparathyroidism.

Hypercalcaemia shows the clinical picture of depression, delirium, stupor and coma. Its causes include:

- hyperparathyroidism

- hypervitaminosis D

- sarcoidosis tumous such as myeloma, metastatic carcinoma and paraneoplastic states

- thiazide diuretics.

Acid-Base Disturbance and Delirium

Acidosis is more likely to be associated with the development of delirium than alkalosis. Hyperventilation usually points to an underlying metabolic disturbance and is associated with lactic acidosis, hypoxia or liver encephalopathy. The calculation of the anion gap and the determination of blood gases is useful in approaching the diagnosis of metabolic acidosis. The anion gap is the difference beteen the serum sodium and the total chloride and bicarbonate:

$$\mathbf{Na - (Cl + bicarbonate) = 10 \pm 4\ mEaq}$$

Causes of metabolic acidosis with a high anion gap include disorders such as:

- lactic acidosis

- diabetic ketoacidosis

- uraemia.

Causes with a normal anion gap include:

- ureteroenterostomy

- gastrointestinal bicarbonate loss as with diarrhoea and pancreatic fistula

- hyperalimentation

- renal tubular acidosis.

Respiratory acidosis is associated with:

- drugs

- depressing respiration

- neuromuscular disease

- pulmonary disease.

Liver Failure and Delirium

The clinical picture of hepatocellular failure manifests with the development of delirium and coma. The latter are directly related to the rapidity of onset and the degree of severity of the liver failure. Special parameters of the liver delirium include the rise in the blood ammonia level, a rise in the cerebrospinal glutamine level and a progressive slowing of the electroencephalogram with the development of triphasic waves.

The severity of the hepatic encephalopathy is aggravated by the following complicating factors:

- gastrointestinal haemorrhage

- infections

- surgery

- sedatives such as opiates, benzodiazepines and neuroleptics to which the brain is especially sensitized

- high protein diet

- hypotension

- hypoxia.

The pathogenesis of hepatic encephalopathy appears to be complex and multi-factorial.

Pancreatitis can be associated with the development of delirium which is associated with increased levels of lipases in the cerebrospinal fluid. The hypothesis of cerebral demyelination secondary to release the lipasesw into the blood stream from the diseased pancreas seems a likely explanation.

Renal Failure and Delirium

Uraemia causes delirium by its electrolyte imbalance, metabolic acidosis and vitamin deficiency (among others). Dialysis disequilibrium worsens mental function post-dialysis and is possibly related to the retention of molecules of middle molecular weight and other osmotically active substances causing brain oedema. Dialysis encephalopathy is a chronic progressive disorder presenting with dysarthria and dysphasia followed by delirium, seizures and dementia. It has a distinctive paroxysmal electroencephalographic picture.

Treatment of Delirium

Determine the cause of the delirium and treat it promptly. Anxiety and fear can be relieved by keeping the patient informed about his progress and treatment. Hypoxia should be excluded as a cause of delirium before ordering sedation as it frequently presents with increasing restlessness. The electroencephalogram is a useful parameter of the severity of the delirium and aids in differentiating it from psychogenic or psychotic states and in diagnosing subclinical epilepsy.

Chlorodiazepoxide is the drug of choice in the treatment of delirium tremens and is usually administered in dosages of 25 mg to 100 mg six-hourly. Intravenous benzodiazepine therapy is best given and assessed by the physician as it tends to show large individual variation in response. The buterophenones: haloperidal (5-10 mg IMI six to eight-hourly) and clotiapine (40 mg IMI six to eight-hourly) are popular and relatively safe drugs. Frequent control of sedative effect by the physician himself is mandatory to prevent the adverse effects of over- and undersedation. The continuous infusion of drugs such as morphine, pethidine or ketamine may be effective and safe in the intensive care unit.

Coma

The word "coma" (see fig. 4.2.2) derives from the Greek word koimao and refers to a state of deep sleep. Coma differs from sleep in the depression of arousal, the disturbance of the electroencephalographic pattern and in the finding of a reduction of the cerebral metabolic rate for oxygen. The emhasis is on the decreased levels of arousal from sleepiness to stupor and coma. The anatomical structures subserving arousal include the ascending reticular activating system and the cerebral hemispheres. The classic monograph on coma by

Plum and Posner has greatly advanced our understanding and treatment of the comatose patient.

In the assessment of coma a comprehensive history and examination are essential in order to establish:

- the level of consciousness and its chronological course

- the clinical type of coma

- its etiology.

Immediate and Emergency Treatment

Urgent treatment of the comatose patient can prevent permanent brain damage. The following points are assessed:

- ariway and ventilation

- circulation and blood pressure

- anaemia and fever

- blood glucose level

- evidence of trauma.

Inadequate ventilation and uncontrollable secretions are indications for intubation and ventilation. Asymmetrical radial pulses suggest a dissecting aneurysm of the aorta. An electrocardiogram will confirm the presence of any arrhythmias. A blood sample of glucose is obtained before glucose is administered intravenously. In alcoholic and malnourished patients thiamin is added to the glucose infusion. This prevents the precipitation of an acute thiamin deficiency which can cause Wernicke encephalopathy, circulatory collapse and death. Evidence of trauma leads to the consideration of a fracture of the cervical spine and injury of the internal organs. Blood leaking from the external auditory meatus or from the nose points to a fracture of the base of the skull, orbital and mastoid haematomas indicate an underlying fracture of the orbital roof and mastoid bone respectively. The skull is palpated for bruising and tenderness.

Do not leave the patient unattended while obtaining a history from the family, friends, ambulance personnel and other relevant people. A sudden onset of coma indicates the possible presence of an intracranial haemorrhage, a brainstem stroke or epilepsy. A previous medical history elicits conditions such as epilepsy, alcoholism, diabetes mellitus, subarachnoid haemorrhage and previous head injury. The latter often leads to the diagnosis of a subdural haematoma. The presence of fever and a history of travelling may suggest malaria or tickbite fever. It is essential to enquire about the possible excessive intake of drugs and alcohol.

Assessing the Level and Evolution of Consciousness

The important principle of measurement in clinical medicine is exemplified in the Glasgow Coma Scale's assessment of the level of consciousness.

The degree of coma is assessed by the coma or responsiveness scale which may be quantified from 3 to 15 and is called the responsiveness score (EMV Sum). The lower the score, the deeper the level and the less responsive the patient.

The pupil test is added:

- pupil size and response to light

In clinical practice the application of sufficient pressure over the masseter muscles, the sternum or the nailbed acts as an adequate painful stimulus to elicit a response. With decorticate posturing the arms are in flexion and adduction and the legs in extension and internal rotation and with decerebrate posturing all four limbs are in extension and adduction. The internal stimuli of airway obstruction and bladder distention can cause spontaneous posturing. When no motor responses are obtainable, the possibilities of Guillan Barré and locked-in syndrome are considered.

The temporal profile of the level of consciousness is constructed starting with a history prior to admission and continuing by recording it hourly thereafter. The acute immediate onset of coma is strongly suggestive of:

- a rostral brain-stem infarction or haemorrhage

- a brain-stem injury

- intracerebral haemorrhage with transtentorial herniation

- hypoxia or hypoglycaemia.

The gradual progressive deterioration of the level of consciousness suggests a cerebral hemispheral mass lesion when cerebral hemispheral signs are followed by the development of rostral brain stem signs. Cerebellar haemorrhage demonstrates the clinical picture of occipital headache, gaze palsies, nystagmus, cerebellar ataxia or cerebellar hemispheral signs with unilateral limb incoordination and intention tremor accompanied by a decreasing level of consciousness. The latter may result from either direct brain-stem compression, hydrocephalus or both. The level of consciousness profile of deterioration, improvement and secondary deterioration is strongly suggestive of an underlying subdural or extradural haematoma.

Pathogenesis of Coma

The level of arousal and consciousness of a patient (figs. 4.2.1 and 4.2.3) is determined by the normal functioning of the ascending reticular activating system and the

cerebral hemispheres. Structural or functional disturbance of one or both of these systems can cause coma.

Involvement of the Ascending Reticular Activating System (ARAS)

The ascending reticular activating system is situated in the rostral pons and midbrain and projects to the cerebral hemispheres via the thalamus.

- Intrinsic rostral brain-stem lesions

Infarctions and haemorrhages of the rostral pons and midbrain are the cause of early or immediate onset of coma resulting from a direct structural disturbance of the reticular activating system.

- Unilateral cerebral hemispheral space-occupying lesion

Coma ensues when the mass effect leads to a significant displacement of midline structures (pineal gland or septum pellucidum) and with transtentorial herniation. Herniation of the parahippocampal gyrus between the tentorial edge and the midbrain causes a secondary compression and dysfunction of the reticular activating system. Pathological conditions causing transtentorial herniation include:

- cerebral hemispheral infarction

- intra-cerebral haemorrhage

- brain tumours

- brain abscess

- subdural haematoma.

The clinical picture of a transtentorial herniation includes:

- the development of a decreased level of consciousness

- an ipsilateral dilatation of the pupil

- a contralateral or ipsilateral development of upper motor neuron signs

- progressive respiratory depression.

- Posterior fossa space-occupying lesions

Posterior fossa mass lesions can depress the level of consciousness by causing direct compression of the reticular activating system in the rostral brain-stem or by cauing obstruction of the cerebrospinal fluid pathways with the secondary development of hydrocephalus. Hydrocephalus can cause a posterior transtentorial herniation with a type of

Parinaud syndrome, compressing the dorsal mid-brain causing a failure of upward gaze. Pathological conditions causing a mass effect in the posterior fossa include:

- cerebellar haemorrhage
- cerebellar infarction
- cerebellar tumours
- acoustic neurofibroma.

These may cause herniation of the cerebellar tonsils through the foramen magnum dorsally, compressing the medulla. The clinical picture of foramen magnum herniation includes:

- the development of neck stiffness
- sudden deterioration of respiration to that of ataxic breathing or apnea
- hypotension
- hypothermia.

- Metabolic and drug dysfunction of the ascending reticular activating system

The multiple synapses of the ascending reticular activating system can form the basis for the selective depression of the level of consciousness by some drugs and poisons. Metabolic conditions, hypoxia and hypoglycaemia probably affect the brain-stem and cerebral hemispheres diffusely.

Primary Cerebral Hemisphere Dysfunction

When cerebral hemisphere lesions are the primary cause of coma, the lesions are usually extensive, bilateral and diffuse and such causes include:

- hypoxia
- hepatic failure
- renal failure
- hypoglycaemia
- encephalitis
- advanced Alzheimer's disease.

The Clinical Syndrome or Type of Coma

The patient's clinical coma can be classified into one of two broad categories namely "metabolic and company coma" or "space-occupying or structural coma" according to the etiology and the clinical symptoms and signs.

Metabolic and Company Coma

The etiological conditions causing this type of coma include:

- renal failure

- hepatic failure

- electrolyte disturbances

- acid-base disturbances

- hypoglycaemia

- hypoxia

- drug overdose

- posioning

- subarachnoid haemorrhage

- encephalitis

- meningitis

- vitamin deficiencies

- hormonal disturbances.

The clinical hallmark of this type of coma is the absence of lateralizing signs and the preservation of the pupil reflexes.

Space-Occupying or Structural Coma

This type of coma is caused by:

- cerebral hemispheral structural or space-occupying lesions

- transtentorial herniation

- posterior fossa mass lesions

- intrinsic rostral brain-stem pathology.

The clinical picture of space-occupying or structural coma characteristically demonstrates the presence of lateralizing signs.

Clinical Picture or Syndrome of Metabolic and Company Coma

History

A history of coma and sudden onset suggests the presence of underlying hypoglycaemia, epilepsy, subarachnoid haemorrhage or a rostral brain-stem stroke. Hypoxia is strongly suspected as the cause of coma when a history is obtained of cardiac or respiratory arrest, circulatory collapse, status epilepticus and gassing. A history of diabetes mellitus, insulin administration and liver failure suggests hypoglycaemia. It is essential to enquire about drug and alcohol intake and the possibility of drug overdose and poisoning.

Depression of the Level of Consciousness

With increasing severity of the etiological condition the level of consciousness deteriorates from alertness to delirium and to unresponsive coma. The presence of eyelid blinking, a sign of reticular activation, suggests a lighter type of come.

The Absence of Lateralizing Signs

The pupils are characteristically equal in size and react normally to light. Opiates constrict the pupils and atropine and glutethimide dilate them. Fixed dilated pupils can (rarely) be caused by barbiturate and xylocaine overdosage and hypothermia. When the eyelids are lifted and inspected the eyes' position is usually conjugately forwards. Some degree of divergence may be present but this usually ceases with arousal. Roving conjugate side-to-side movements of the eyes are frequently observed and suggest suppression of hemispheral function and intactness of the brainstem. Doll's eyes or oculocephalic movements (not tested with suspected neck injury) are characteristically intact and indicate normal brain-stem function. On turning the head to the left the eyes will move conjugately to the right and on turning the head downwards the eyes will move conjugately upwards and vice versa. Drug overdose can cause an internuclear ophthalmoplegia and suppression of doll's eye movements. Caloric oculo-vestibular reflexes are characteristically normal and indicate a functionally intact brain-stem. A cold-water stimulus of 50 to a 100 mL (after checking the intactness of the ear drums) would elicit a conjugate deviation of the eyes to the ipsilateral side, nystagmus will characteristically be absent as the coma suppresses the cortical saccadic components. Drug overdose occasionally causes downward deviation of both eyes after unilateral cold-water stimulation.

The motor responses of the face and limbs are symmetrically disturbed with either decreased or increased tone. The arms drop at the same speed. Generalized flaccidity is the characteristic motor finding in acute severe metabolic encephalopathy. Occasionally symmetrical decorticate and decerebrate postures are found, either spontaenously or in response to stimulation. Although decerebrate rigidity is a common sign of transtentorial herniation or of a rostral intrinsic brain-stem lesion, it does also occur with the metabolic

encephalopathies of hypoglycaemia, hypoxia and hepatic failure. Lateralizing signs occur rarely with metabolic conditions such as uraemia, hepatic encephalopathy and hypoglycaemia and may take the form of an asymmetry in muscle tone or a unilateral lack of spontaneous movements of the limbs. Babinski responses appear with many causes which depress the level of consciousness including a variety of metabolic encephalopathies, drugs and the post ictal state.

Spontaneous Abnormal Movements

The presence of involuntary movements in the form of multiple wide-spread muscle fasciculations, myoclonic jerks and tremors usually points to an underlying metabolic encephalopathy. Widespread myoclonic jerks of low amplitude occur frequently with uraemic and post-anoxic coma. Asterixes (flapping tremor) is elicited with the arms stretched out before the patien, the palms facing forwards and the wrists held in extension. It occurs with hepatic failure, uraemia, hypercapnia, sedative drugs and subarachnoid haemorrhage. Unilateral asterixes usually point to an underlying structural lesion. Multifocal epilepsy occurs especially with renal failure and hyperosmolar diabetic coma.

Respiration

Cheyne-Stokes breathing is frequently seen with metabolic depression of the cerebral hemispheres although it can occur in the elderly during sleep. It is characterized by cyclic periods of hyperventilation tapering gradually to apnea of variable duration and back to hyperventilation. The presence of hyperventilation usually points to the presence of an underlying metabolic disturbance rather than a structural lesion. It is seen with diabetic coma, renal failure, lactic acidosis, hypoxia, hepatic encephalopathy and poisonings. Depression of respiration with slow, shallow and ineffective breathing usually suggests drug induced depression of the respiratory centre.

Meningism

The presence of neck stiffness, Brudzinski and positive Kernig signs probably point to a disease of the subarachnoid space as meningitis or subarachnoid haemorrhage. Meningism with lateralizing signs may suggest the presence of an inctracerebral abscess or haemorrhage. With the tentative diagnosis of acute or chronic meningitis it is frequently useful to repeat the lumbar puncture twelve hours later to confirm the diagnosis.

Epilepsy

Grandmal epilepsy may be followed by delirium and coma although the latter seldom lasts longer than 30 minutes. The presence of post-ictal coma may suggest the presence of subclinical status epilepticus, encephalitis or subarachnoid haemorrhage. The presence of tongue and cheek injuries, urinary incontinence and gum hypertrophy may suggest underlying epilepsy.

Special Investigations in Metabolic and Company Coma

Further investigations are undertaken when the history and clinical examination fail to yield an adequate cause of the coma. Blood tests for urea and electrolytes, blood glucose, blood gases, serum calcium, phosphate and magnesium are sent off promptly. If the cause of the coma remains unclear or drug toxication of poisoning is suspected, samples of blood, urine and where safe gastric contents are sent off for pharmacological and toxicological screening.

A lumbar puncture is relatively safe in the absence of papilloedema and lateralizing signs. Patients suspeced of having a diagnosis of bacterial meningitis require a lumbar puncture and the urgent institution of appropriate therapy. If the suspected diagnosis of bacterial meningitis is associated with the presence of papilloedema or lateralizing signs suggesting the presence of a brain abscess or intracerebral haematoma, a computer axial tomography (CAT) scan of the brain becomes the primary procedure. In situations where the CAT scan facility is not readily available, it would be prudent to send off blood cultures and treat the suspected meningitis empirically to avoid the rapid deterioration which may occur within hours. The performance of a lumbar puncture in the presence of papilloedema or lateralizing signs may lead to transtentorial herniation and progressive deterioration over minutes, hours or days and requires urgent neurosurgical attention.

The electroencephalogram can reveal useful information in the comatose patients and is invariably abnormal in metabolic encephalopathy with the presence of slow wave activity parallelling the depth of the coma. It is normal in psychogenic coma, the locked-in syndrome and in patients receiving succinylcholine which may mimic a clinical picture of brain death. The electroencephalogram often detects the subclinical presence of status epilepticus. Periodic complexes are suggested of an underlying herpes simplex encephalitis. Triphasic waves are characteristic but not specific of liver coma. Myxoedema coma shows a characteristic depression of activity. Drug intoxication is reflected by the presence of beta activity. The electroencephalogram may show electrocerebral silence simulating brain death in hypothermia, hepatic encephalopathy and with barbiturate and xylocaine overdosage.

Patients remaining in a coma of unknown cause or patients demonstrating unexpected clinical deterioration, require a CAT scan of the brain to exclude a possible remedial lesion like a subdural haematoma, hydrocephalus or a midline tumour of the brain.

The Clinical Picture or Syndrome of Space-Occupying or Structural Coma

History

Loss of consciousness following on an immediate onset of headache is strongly suggestive of an intracranial haemorrhage. A space-occupying lesion is suspected when the loss of consciousness is preceded by headache, nausea and vomiting or by the progressive development of paralysis, ataxia or visual disturbances. The diagnosis of a subdural haematoma is suspected when a history of previous trauma or alcoholism is obtained.

Lateralizing Signs are Characteristically Present

The presence of pupillary inequality or unilateral dilatation in the comatose patient is of pathologic significance and an improvement sign of transtentorial herniation. Small reacting pupils are associated with the diencephalic phase of central tentorial herniation or with a pontine haemorrhage (interrupting sympathetic connections). Pontine haemorrhage is associated with the development of bilateral corticospinal tract signs, skew deviation (eyes dysconjugate in the vertical plane) and facial and abducens nerve palsies. Bilateral dilated non-reacting pupils are usually the result of irreversible structural mid-brain injury.

The eyes are frequently deviated away from the conjugate forwards position. In a cerebral hemispheral lesion the eyes deviate conjugately to the side of the lesion and the hemiparesis occurs contralaterally. With a pontine lesion the eyes deviate conjugately away from the lesion and the hemiparesis presents contralaterally to the pontine lesion. A thalamic haemorrhage causes the eyes to deviate conjugately downwards. Skew deviation of the eyes or disconjugation in the vertical plane points to a lesion of the pons or mid-brain region.

The oculocephalic and oculovestibular reflexes are very useful tests to assess the intactness of the brain-stem in comatose patients. The presence of an oculomotor or abducens nerve palsy or of an internuclear ophthalmoplegia can be detected with these tests.

Asymmetrical motor responses of the face and limbs occurring either spontaneously or in response to painful stimuli is a characteristic and important lateralizing sign and may point to a neurosurgical emergency. The only signs of an underlying hemiparesis may be the findings of asymmetry of tone and deep-tendon refleces or the presence of a Babinski response. The presence of a unilateral decerebrate response and a contralateral decorticate response will lateralize the signs to the side of the more primitive decerebrate response. With rostral caudal transtentorial herniation the motor signs may evolve from that of a hemiparesis to that of a decorticate and decerebrate response successively (fig. 4.2.5). Focal motor seizures which may be associated with post-ictal or Todd's paralysis, would be of clear localizing value. Corneal sensory testing can help to demonstrate an asymmetrical facial blink response. The presence of a truncal, gait or limb ataxia usually points to the presence of a cerebellar or posterior fossa lesion and the presence of nystagmus strengthens this suspicion.

Fundoscopy

The presence of papilloedema in the comatose patient indicates the presence of an underlying structural or mass lesion or the presence of hydrocephalus and constitutes a neurosurgical emergency. The sudden increase of intracranial pressure which occurs after a head injury or subarachnoid haemorrhage may be associated with the development of superficial round or semilunar (subhyaloid) preretinal haemorrhages.

Respiration

Death after transtentorial herniation or direct brain-stem involvement is usually a respiratory death. The rostral caudal progression of transtentorial herniation is associated with Cheyne-Stokes breathing during the diffuse hemispheral phase, with central neurogenic hyperventilation during the midbrain phase, apneustic respiration at pons level and ataxic

irregular breathing in rhythm and volume at medullary level which is followed by apnoea and death.

Special Investigations in Structural Coma

The presence of any lateralizing signs or papilloedema in a comatose patient constitutes a neurological emergency. Secondary brain damage is avoided by clearing the patient's airway and ensuring adequate ventilation. CAT scanning of the brain is usually the investigation of choice.

Differential Diagnosis of Coma

Psychogenic or Hysterical Coma

This diagnosis should be made only by an experienced observer. The paradox existing between the absence of any response to painful stimuli and the intactness of normal reflex activity is characteristic. Breathing, pupillary reactions, corneal and gag reflexes, deep tendon and plantar responses are normal. Forced closure and fluttering of the eyelids are frequently found. Oculocephalic reflexes cannot be elicited as the patient is awake and looks voluntarily in any direction, often upwards. Caloric oculovestibular cold water testing gives rise to phasic nystagmus to the contralateral side, indicating the patient's alerness. The voluntary saccadic nystagmus component of the test characteristically disappears with coma. Eye movements, when detectable, will often show a jerky saccadic or voluntary component. The encephalogram is normal and shows desynchronization to external stimuli. The coma usually does not last long.

Locked-In Syndrome

The term "locked-in syndrome" refers to a state of widespread motor paralysis of the lower cranial nerve and limb muscles but with blinking and vertical eye movements unaffected. The patient is often mistakenly considered to be in coma. He reacts to commands by moving his eyes voluntarily either up or downwards. The condition is usually caused by a basal pontine infarction or haemorrhage involving the corticospinal tracts and the centres for conjugate lateral gaze at that level. The patient's sleep/wake cycle and electroencephalogram are usually normal.

Vegetative State

The term "vegetative state" refers to the clinical condition following the evolution of subacute and chronic coma to wakefulness in which the cognitive functions are severely lacking or absent and the brain-stem and vegetative functions such as respiration and circulation are spared. This term is preferred to the other terms with similar meaning such as "apallic state" or "akinetic mutism". The term "persistent vegetative state" refers to the prolonged persistence of this state. Certain pathological conditions such as cerebral trauma, hypoxia, subarachnoid haemorrhage with diffuse cerebral vasospasm, hypoglycaemia, have a tendency to affect hemispheral neurons differentially more severely than the brain-stem neurons and to present with the vegetative state on recovery from coma which usually occurs within two to four weeks from the onset of coma. The incidence of the vegetative state

increases with the duration of coma. Although the electroencephalograms and CAT scans of the brains of patients suffering from the vegetative state are not distinctive, recent work suggests that the absence of the median nerve vertex referenced parietal somatosensory evoked potentials, with preservation of the brainstem auditory evoked potentials, may closely delineate the vegetative state. The outcome of the persistent vegetative state is extremely poor with a five-year mortality of 73% (adults). In children the prognosis for the vegetative state is poor although it seems prudent to keep the prognosis guarded until after three months. The development of decerebrate or decorticate rigidity, roving eye movements and spontaneous blinking may be early clues to its development. The vegetative state has been called a fate worse than death.

Prognostic Aspects of Coma

Levy et al studied the prognosis of non-traumatic coma in 500 consecutive patients admitted to New York and Newcastle upon Tyne hospitals. The prognosis of non-traumatic coma is influenced by the etiology, the depth and the duration of the coma and the development of individual clinical signs. The mortality was worst in the cerebrovascular etiology group, followed by the hypoxic-ischaemic and metabolic groups yielding mortality figures of 74, 58 and 47% respectively. The vegetative state developed in 20% of the hypoxic-ischaemic group and in the cerebrovascular and metabolic groups in 7% and 6% respectively. The ability of the patient to open his eyes, respond with a motor and a verbal reaction as elicited by the Glasgow Coma Scale was associated with a better prognosis. The absence of an eye-opening response, a motor response and a verbal reaction to command or pain was associated with death in approximately two-thirds, four-fifths and two-thirds of cases, respectively. The longer the duration of coma, the poorer the prognosis. The moderate to good outcome of coma dropped from 15% on admission to 12% after one day of coma, to 7% after three days, 6% after seven days and 2% after 14 days. The brainstem reflexes are by far the most important when assessing a patient in coma. The absence of the pupillary light reflex, the oculovestibular reflex and the corneal reflex is each associated with a very poor prognosis. When the absence of these reflexes occurs in combination they are associated with a uniformly poor prognosis. The spontaneous occurrence of eye movements, motor localizing signs, oculovestibular and inappropriate verbal responses after 24 hours is associated with a moderate to good outcome in 28%, 31%, 60% and 56% of patients respectively.

After cardiopulmonary arrest the absence of the pupillary and corneal reflexes at six hours indicated a very poor prognosis. Poor prognostic features after cardiac arrest include:

- absence of a localizing motor response to pain at the one-hour point

- dilated non-reactive pupils

- decerebrate and decorticate postures

- absence of oculovestibular, oculocephalic and corenal reflexes

- a systolic bloodpressures below 80 mm Hg

- the depth and duration of the coma (especially longer than 24 to 48 hours).

Open-heart surgery is associated with the development of a diffuse encephalopathy in 11.6% of patients and coronary bypass surgery has been associated with prolonged depression of the level of consciousness in 3% of patients. Cardiac transplantation has been complicated by multifactional metabolic encephalopathy.

The prognosis of a head injury is related to:

- the depth and duration of the coma

- the duration of the posttraumatic amnesia

- the presence of a fracture of the skull

- presence of focal neurological signs

- development of the complications of epilepsy, haematoma and meningitis.

Concussion refers to a loss of consciousness following on the occurrence of a head injury which usually lasts from a few seconds to minutes. Recovery takes place from the coma stage to that of confusion, amnesia and full wakefullness. More severe degrees of head injury recover more slowly but also through the same stages. The primary loss of consciousness after trauma is caused by a centrifugal force moving from the superficial hemispheral layers to the deeper structures involving the rostral brainstem. It follows that a loss of consciousness implies that the shearing force has caused tearing of the more superficial white matter fibres, often most severely at the junction between the white and grey matter.

A study assessing the prognosis of severe head injury was carried out in Scotland, the Netherlands and the USA. Coma was defined as the clinical condition in which the patient could not give any verbal response, nor obey commands or open the eyes either spontaneously or to any stimulus. Only patients in a coma lasting longer than six hours were entered into the study. The Glasgow coma responsiveness scoring system was used. All patients with responsiveness scores of less than 7 were in coma, while 53% with a score of 8 were comatose and none with a score of more than 9. The lower the score, the worse the prognosis. The best score within 24 hours after coma onset was related to mortality, the development of the vegetative state, to moderate disability, and good recovery. Responsiveness scores of 3 to 4, 5 to 7, 8 to 10 and more than 11 were associated with a mortality or vegetative state in 87, 53, 27 and 12% respectively and with a moderate disability to good recovery in 7, 34, 68 and 82% respectively. Non-reactive pupils were associated with a mortality of 82% and in elderly patients this had an even worse prognosis.

The main advantage of the assessment of the duration of the posttraumatic amnesia (PTA) lies in the ability to assess it retrospectively. The duration of PTA has been categorized into:

- mild (less than 60 minutes)

- moderate (1-24 hours)

- severe (1-7 days)

- very severe (1-4 weeks)

- extremely severe (> 4 weeks).

Of the patients entered into the above study all survivors had a PTA of at least two days, 94% percent had a PTA for more than one week and 60% more than 4 weeks. The duration of coma was related to the duration of PTA. In patients with coma of less than one day a PTA of more than one month was present in less than 20% of survivors, while after one to two weeks of coma more than 62% of patients had a PTA of more than a month. The incidence of good recovery in patients after PTA of less than 7, 8 to 14, 15 to 28 and more than 28 days duration was 90, 80, 60 and 30% respectively. The duration of the PTA in the severely disabled group was between 14 and 28 days in 3% and more than 28 days in 97% of patients. In the patients with a moderate disability the duration of the PTA was less than 7, between 8 and 14, 14 to 28 and more than 28 days in 2, 8, 18 and 72%.

The orbital surface of the frontal lobes and the fronto-temporal regions are frequently injured with head injuries because of the irregular surface of the floor of the frontal fossa and the sharp prominences of the lesser wings of the sphenoid bone. The abilities to process new information rapidly and plan and organize knowhow are frequently affected after head injuries and may not reflect on formal psychometric testing. Disturbances of effect occur frequently and prominently along with irritability, loss of temper, decreased inhibition, and depression.

The electroencephalogram can be used as can index of cerebral cortical function but always in association with the brainstem reflexes which are of dominant importance. The course of the coma may be followed by performing serial electroencephalograms. Generally, the worsening condition is shown on the encephalogram by decreasing voltage, decreasing reactivity, and the appearance of isoelectric intervals. Unfavourable electroencephalographic signs are the development of a low voltage 2-10 microV, suppression bursts lasting longer than three days; slowing frequencies; continued low voltage and increasing isoelectric segments.

Brain Death

"Vex not his ghost: O! let him pass; he hates him that would upon the rack of this tough world stretch him out longer". Shakespeare

The advances of transplant surgery of the 1960s created a need for the death certification of the ventilated, non-responsive patient whose heart was still beating. The landmark report of the Harvard ad hoc committee on brain death of 1968 was followed by many authoritative papers crystallizing the current ideas on the subject. The concept that brain death equalled the death of a person and that brainstem death equalled brain death was developed and accepted (see fig. 4.2.6). The brainstem is essential for the maintenance of cortical functioning, breathing and blood pressure. Brainstem death invariably leads to cardiac arrest within 24 hours in over half of cases. The diagnosis of brainstem death can be assessed adequately on clinical criteria alone. The presence of spinal reflex activity reflects the status of the spinal cord and its circulation and does not exclude a diagnosis of brainstem death. The

diagnosis of brain death is usually certified by two doctors, one with consultant status and the other with five years or longer post-registration experience.

Definition of Brainstem Death

The term brainstem death comprises the presence of certain prerequisites which are inherent in the concept and include:

- Adequate resuscitation of the patient usually for a period longer than six hours.

- The exclusion of reversible causes such as sedative drug intoxication, hypothermia, metabolic and endocrine disturbances.

- The presence of an irreparable structural brain lesion.

Without all the above conditions, the patient has a diagnosis of coma. The presence of cerebral unresponsiveness, apnoea and the absence of brainstem reflexes is not sufficient for the diagnosis of brainstem death.

Many patients have a combination of structural and "metabolic and company" factors contributing to their coma. It is essential to restore an airway, treat hypotension and correct bloodgas and electrolyte abnormalities.

The assessment of the patient's sedative drug intake is neither easy nor free from error. The history is often unreliable and the analysis of the toxic agent and the interpretation of results difficult and time-consuming. With uncomplicated drug intoxication the signs miming brainstem death do not usually persist longer than 36 hours and the pupillary light relfex is usually the first to return. Electroencephalographic monitoring may be useful in demonstrating fast activity superimposed on general slowing. Although sedative drugs like barbiturates may cause electrocerebral silence, this activity usually lasts less than 48 hours if recovery is at all possible. A pupillary size smaller than five millimeters may suggest sedative drug effect. It is prudent to remember that neuromuscular blocking drugs may abolish brainstem reflexes and cause apnoea and miming the clinical picture of brainstem death.

The diagnosis of a known irreparable structural brainstem lesion may be obvious where the patient has had a head injury and a subarachnoid or intracerebral haemorrhage. A CAT scan of the brain often helps in confirming a structural brain lesion. In other patients the diagnosis may be more complicated and part of an evolving clinical picture, requiring continual monitoring and assessment.

Clinical Tests of Brainstem Function

Absence of Cerebral Responsiveness

No voluntary or autonomic reflexes to noxious stimuli.

The absence of ictal activity, decorticate and decerebrate postures and trismus are implicit.

Apnoea

The development of hypoxia during apnoea testing is prevented by preoxygenation the patient with 100% oxygen for ten minutes and by placing a catheter into the trachea delivering six litres of oxygen per minute. An apnoea period of ten minutes is recommended which will ensure that an adequate arterial carbon dioxide tension (more than 50 mm Hg) will develop to stimulate the respiratory centre. The patient may be given 5% carbon dioxide in 95% oxygen for a few minutes before disconnection when bloodgas determinations are not available.

Absence of Brainstem Reflexes

- pupillary light reflex

- corneal reflex

- oculocephalic and oculovestibular reflexes

- gag and tracheal reflexes.

The patients usually show hypotension and the secondary development of hypothermia.

Comatose Patient With Absent Brainstem Reflexes

Without irreparable structural brain lesion the patient's diagnosis is that of coma and he is investigated and treated as such. A history of hypoxia or hypoglycaemia usually implies a grave prognosis but warrants a more conservative approach and a longer period of observation. The patient without brainstem reflexes and with apnoea for a period of 72 hours can be declared dead on clinical criteria alone. The electroencephalogram is a useful prognostic procedure and reflects cortical hemispheral functioning. Electrocerebral silence without sedative drug intoxication implies a uniformly fatal outcome. The electroencephalographic procedure may shorten the period of observation in the hypoxic and metabolic causes of coma. Other diagnostic procedures of cerebral angiography and the measurement of cerebral metabolism are rarely used. The intracranial circulation ceases with brain death and demonstrates the no-flow phenomenon on angiography.

Chapter 4.3: Raised Intracranial Pressure

M J van Rensburg

Introduction

It would obviously not be meaningful to discuss under this heading the surgical treatment of space-occupying lesions such as brain tumours, cysts, haematomas, and hydrocephalus. We are concerned rather with the management of patients with raised intracranial pressure (RICP) as a result of lesions which cannot be surgically treated, such as Reye's syndrome, or with a surgically treatable lesions such as an intracranial haematoma in which an expectant policy has been decided on. The most common conditions which are

managed in this fashion are head injuries, Reye's syndrome, the anoxic states such as near drowning and cardiac arrest and, less commonly, cerebral haemorrhage and infarction. Neurosurgical patients in whom post-operative brain swelling is anticipated are often treated in similar fashion.

In the setting of an intenstive care unit, we are not usually concerned with the early diagnosis of RICP in a conscious patient. In such patients clinical criteria usually suffice, and in particular the charting of the level of consciousness on the Glasgow Coma Scale (GCS) or one of its paediatric equivalents is valuable in producing some standardization of clinical material and in monitoring patients whose levels of consciousness are not profoundly depressed.

In unconscious patients following head injury these coma scales are, however, of more value in assessing the degree of primary brain damage rather than the level of intracranial pressure (ICP). It is common knowledge that a deteriorating level of consciousness as judged by these scales is preceded, often for a considerable period of time, by RICP, whether caused by an accumulating haematoma or by brain swelling.

It is axiomatic that the greater the disturbance of brain function, from whatever cause, the more difficult it is to estimate ICP. Following severe head injury the neurological signs which have been traditionally related to RICP (such as bradycardia, motor posturing, and pupillary dilatation) may occur with normal ICP, or conversely may be absent in the presence of RICP. Clinical signs are therefore frequently not reliable indicators of the level of ICP and thus decisions regarding therapy cannot be based on the clinical examination alone.

In the comatose patient who exhibits abnormal motor posturing to painful stimuli, deterioration is signified by a loss of motor responses, usually accompanied by pupillary dilatation and apnoea. There is thus no safety margin and in this setting ICP monitoring may provide a valuable warning of impending catastrophe.

Although these considerations may constitute a theoretically good motivation for the monitoring of ICP in those unconscious patients in whom RICP is considered likely, the results must naturally be interpreted in the context of the clinical background. There is no linear relationship between the level of ICP and the clinical state of the patient. The cause of the rise of pressure is as important as its magnitude. If the pressure rise is, for example, due to the expansion of the cerebrospinal fluid (CSF) compartment without the presence of space-occupying masses or defects in cerebral vascular autoregulation, brain shifts and hernias do not occur and high levels of ICP can be tolerated. In these circumstances cerebral blood flow can be maintained until the perfusion pressure falls below 40 mm Hg. The perfusion pressure of the brain may be reasonably accurately estimated by calculating the difference between the mean arterial pressure and the intracranial pressure. Patients suffering from so-called benign intracranial hypertension, for instance, may exhibit an ICP of 60 or, albeit briefly, even 80 mm Hg without any disturbance of consciousness. But in other conditions in which brain shifts have occurred as aresult of space-occupying lesions, and when cerebral autoregulation is impaired, as after head injury and subarachnoid haemorrhage, a higher perfusion pressure is required to maintain cerebral circulation. There is also no predictable level of ICP at which brain shifts will lead to transtentorial or foramen magnum herniation. A trend of rising ICP is often of as much value as the absolute pressure is an indication ofimpending trouble.

On theoretical grounds as good a case can be made for measuring ICP in selected patients as for measuring the blood pressure. Blood pressure is, after all, monitored to ensure that the perfusion pressure to organs is maintained. When RICP is likely to be present, the adequacy of cerebral perfusion cannot be assumed from blood pressure readings alone.

There can be no doubt that the management of a patient is simplified by ICP monitoring if it is felt that he may suffer from, or is likely to develop, intracranial hypertension. The ICP of such a patient may of course not be raised and treatment may thus be avoided. It provides the only way to assess the effects of a particular method of treatment, and thus the only way to rationally tailor treatment to the level of the ICP.

Having said all this, it must be conceded than there is no clear statistical evidence that the monitoring of ICP improves outcome in the majority of the conditions in which it is employed. This is, in the case of head injuries, probably partly due to the difficulty of obtaining comparable series for statistical evaluation. Aficionados of ICP monitoring are reluctant to embark on studies which might produce statistically meaningful results as they are not willing to subject a considerable number of their patients to management without this facility. There is perhaps a message in this attitude. The best case in its favour can probably be made in Reye's syndrome and the worst in anoxic brain damage. We will briefly consider its place in these two conditions as well as in head injuries. Its employment must, however, be considered in any condition in which RICP is considered likely to adversely influence the course of an illness from which recovery is possible.

Reye's Syndrome

Reye's syndrome is a toxic encephalopathy associated with fatty infiltration of the liver. In severe cases the mortality is in the region of 75% or more.

With good intensive care and with the monitoring and control of ICP, the mortality may be substantially reduced. Thus Marshall et al reported seven cases, none of whom responded to deep pain, and all of whom had abnormal or absent oculocephalic responses, bilaterally unreactive pupils, and markedly irregular or absent respiration. With treatment all these terminally ill patients survived and six were neurologically intact. They thought it necessary to treat all of them with pentobarbital after vigorous treatment with hyperventilation, steroids, and mannitol (blood osmolality raised to at least 325 mOsm), and normalization of blood pressure and body temperature had not controlled RICP.

In a study from Yale, Shaywitz et al found that the aggressive treatment of RICP, including the use of barbiturates and CSF drainage, lowered the mortality from a previous 60% to 12% in 29 comatose children with Reye's disease.

All the series reported have not been equally successful, but clearly the control of RICP does substantially reduce the mortality.

The residual mortality and morbidity is probably determined by the neuronal injury which is the pathological basis of the brain swelling. The degree of primary brain damage is thus very important and the rise of ICP is probably largely, but not linearly, related to the severity of the neuronal damage.

Nevertheless, active treatment of the RICP may reduce the mortality significantly and is clearly of value.

Anoxic Brain Damage

In anoxic brain damage it appears likely that a rise of ICP is truly an index of neuronal damage and that its level is directly related to the degree of neuronal damage.

Bruce reported on two comparable series of nearly drowned children, each consisting of 17 patients. In one series all the patients who were in coma were intubated and ventilated, with the monitoring and control of ICP to below 20 mm Hg, if possible. Steroids, mannitol, furosemide, and pentobarbital were administered as was felt necessary. In the second series the patients received ventilatory and metabolic support, but ICP received no attention. In both series, eight of the 17 patients died. In the series in which an attempt was made to control ICP, only two patients survived in good condition or with moderate impairment. In the series in which no attention was paid to ICP, six patients survived in good condition or with moderate impairment.

Bruce's conclusion were that the RICP that occurs after the anoxia of near drowning, is the result of neuronal swelling and is an indication of irrevocable neuronal damage. There does not appear to be an indication for the monitoring and control of ICP in near drowning, nor in other states of anoxic damage such as that occurring with cardiac arrest.

Head Injuries

While there is a clear correlation between the level of ICP and the outcome of a head injury, a high ICP following head injury does not necessarily indicate severe, extensive brain damage. Conversely severe extensive brain damage usually, but not invariably, produces a considerably elevated ICP. The difficulty is to know whether the intracranial hypertension after head injury is a manifestation of brain swelling secondary to an irreversible lesion, or whether it is playing an important part in causing the problem. It can be concluded that brain damage does often produce intracranial hypertension proportional to the degree of damage, but there surely is little doubt that a space-occupying lesion, such as an intracerebral haematoma, may itself lead to brain shifts and hernias which cause brain damage. These space-occupying lesions usually produce an RICP of a magnitude reasonably in accordance with their size and the amount of shift they produce, so that the measured ICP is an approximate index of their size and progress. If the ICP is high or rising, the presence of a mass lesion must therefore be excluded by computerized tomographic (CT) scanning or angiography before other causes are postulated and the RICP treated by means other than surgical. The magnitude of brain shift is of course always of more importance than the level of ICP. In the unconscious patient it is difficult to detect the development of such a haematoma which may make its appearance unexpectedly after a few days if ICP monitoring is not carried out. A haematoma usually heralds its presence by a rise of ICP long before clinical deterioration becomes evident, and its early detection may improve the very poor survival figures of delayed traumatic intracerebral haematomas.

Although there is not a great deal of firm statistical evidence to indicate that the monitoring of ICP, and even the treatment of RICP, in the adult patient improves the outcome, the same cannot be said of the child.

The proclivity of the immature brain to generalized swelling following even relatively minor trauma has been recognized for many decades, and the commonest pathological changes in children who die from head injury have been found to be diffuse brain swelling and venous congestion. In the series of Bruce et al diffuse swelling occurred in 41% of children with a Glasgow Coma Scale (GCS) score of 8 or less, and in 15% with a GCS score of greater than 8. The swelling was shown to be produced by an increase of the intracerebral blood volume and occurred after all degrees of head injury. When associated with a lucid interval minimal brain injury is likely and recovery with proper treatment should be complete. If loss of consciousness occurs with impact, invariable, but not necessarily great, brain damage occurs. As many as 50% of children who die following head trauma, are conscious on admission. With active treatment in the face of deterioration, Bruce et al have reduced the incidence of "talk and die" patients in the paediatric age group to zero, or virtually so.

A few other convincing examples of improved survival rates without an apparent increase in morbidity may be quoted.

Saul and Ducker reduced the mortality figure in a group of patients with GCS score of 7 or less from 46 to 28%, simply by treating elevations of ICP above 15 mm Hg instead of doing so at a level of 20-25 mm Hg.

Bowers and Marxhall, in a co-operative study from San Diego County, showed that the monitoring and control of ICP, at below 15 mm Hg if possible, reduced the mortality of patients with a GCS score of 3 to 5 from 62% to 39%. Patients with a GCS score of above 5 did not benefit from ICP monitoring.

These clear reductions in mortality are not universally obtained, but those who monitor ICP and treat rises vigorously, almost invariably produce better results than those who do not. It is admittedly not, however, very fruitful to compare the results of different series, and particularly of series from different institutions.

In agreement with Galbraith and Teasdale we have found ICP measurement useful in monitoring the progress of patients with traumatic subdural or intracerebral haematomas. They found that operations always became necessary if the initial pressure was over 30 mm Hg, they became necessary in half the patients if it was between 20 and 30 mm, and was rarely necessary if it was below 20 mm. We have found this a useful guide, but it must remain a guide. We would not rely on measured ICP to guide us in our decision to operate on temporal lobe contusions, just as we would not wait for a significant degree of midline shift to occur in such patients. If the size of the temporal lobe contusion is worrying, possibly with encroachment on the supra-sellar cistern by the uncus, operation is generally indicated. Carotid angiography will almost invariably show marked elevation of the middle cerebral artery in these patients. ICP monitoring is an investigation and it must be interpreted with good judgement.

That good results can be obtained in the management of head injuries without ICP monitoring has been shown by Stuart et al. Their results are comparable to those from a series in which ICP was carefully monitored and controlled. They used assisted ventilation to provide optimal oxygenation, rather than as a means of reducing ICP. Mannitol was used mainly prior to craniotomy for a mass lesion in rapidly deteriorating patients, and thereafter if pupillary dilatation occurred in patients without an intracranial haematoma. Dexamethasone was used in just over half the patients without apparent effect.

Apart from the evacuation of haematomas, their treatment was good supportive care with which they were more successful than the authors reporting most similar series. Nevertheless, I believe that this is acceptable treatment and that it is, in fact, preferable to aggressive attempts at control of ICP outside the confines of a neurosurgical intensive care unit. I have little doubt that inadequately controlled aggressive treatment is worse than no treatment.

The case for monitoring and controlling ICP in selected head injured patients in the setting of an intensive care unit is open to discussion. I do believe, however, that not to do so represents less than optimal care.

Patients Selection for ICP Monitoring Following Head Injury

It is obviously not necessary to monitor all patients following head injury. In general patients must be in coma and not showing signs of early recovery of consciousness. Coma is defined in conformance with the recommendation of the Head Injuries Committee of the World Federation of Neurosurgical Societies. This definition, which is in wide international use, requires that the patients do not open their eyes in response to any stimulus, speak no recognizable words, and do not obey commands.

Should all patients in coma then be subjected to ICP monitoring? One monitors ICP only if one suspects that it is likely to be raised or to become raised. The guidelines established by Narayan et al are useful in predicting which patients are likely to develop RICP. Patients of all ages are represented.

They found that when the CT scan was abnormal, showing either high-density or low-density lesions, RICP occurred in 57% of patients. In the presence of surgically significant haematomas the ICP was raised in a predictably larger percentage. To the CT scan abnormalities predictive of high ICP we would add the appearance of compressed or absent basal cisterns, which is also of poor prognostic import.

When the CT scan was normal, they found that only 13% of patients developed ICP above 20 mm Hg. Those patients who did develop RICP could be reliably predicted on the basis of certain clinical features:

- Hypotension, defined as a systolic blood pressure below 90 mm Hg. Only 6% with a systolic blood pressure greater than 90 mm Hg developed RICP compared to 45% with a systolic blood pressure below 90 mm Hg.

- Motor posturing. In the absence of abnormal flexor and extensor responses only 5% developed RICP compared to 17% when unilateral posturing was present and 44% when bilateral posturing was present.

- Age. With normal scans 4% of patients under 20 developed RICP, 17% between 20 and 40, and 30% over 40.

These figures were calculated by excluding abnormal CT scans and not by excluding other adverse features, which appeared to interact in the production of RICP.

ICP elevation could be accurately predicted 92% of the time in patients with normal CT scans with these three indicators. Analysis showed that when a patient demonstrated two or more of these adverse features on admission, the incidence of RICP was 60%, compared to 4% when only one or none of these features was present. These findings suggest that ICP monitoring need not be routinely performed in patients with normal CT scans unless they present two or more of the adverse features noted above.

However, we regard abnormal motor posturing as sufficient reason to monitor ICP, as any deterioration from this state results in loss of motor activity and a patient who is usually beyond salvage. The decerebrate or decorticate state is usually due to diffuse primary brain damage, but clearly there is no way in which the supervention of RICP, whether it be due to haematoma or brain swelling, can be clinically suspected in such a patient. Thus, although we accept that the incidence of RICP is low if there are not other adverse factors, the price that is paid by such a patient who develops and intracranial haematoma is unacceptably high if an early warning system is not installed. It is impractical to scan patients frequently enough to always detect such haematomas in time, although we do routinely scan all patients who are not improving the second or third day to exclude the possibility of delayed intracranial haematomas.

Miller monitors ICP pressure in all head injured patients who are being artificially ventilated. This includes patients who were in coma prior to the evacuation of an intracranial haematoma, patients who score 5 or less on the GCS score, and patients in whom the CT scan shows loss of the perimesencephalic cisterns, midline shift of more than 5 mm, or dilatation of the contralateral ventricle. He also advised ICP monitoring in head injured patients who are being artificially ventilated for respiratory reasons such as pulmonary contusion and post-aspiration atelectasis.

The patients studied by Narayan et al were largely an adult group. What of the child with his propensity for diffuse brain swelling, a feature not shared by adults? Bruce gives the following guidelines:

- Focal lesion on brain scan.

- GCS score of 5 or below. A Glasgow Coma Scale score of 5 almost always invariably means a patient with flexor posturing of the arms, absent verbal responses, and absence of eye opening to any stimulus.

- Shock. He advises early insertion of an ICP monitor if the patient requires vigorous resuscitation. With a low blood pressure the intracranial vessels dilate and when large volumes of fluid are rapidly infused the brain may swell acutely due to perfusion of these dilated vessels.

The indications are thus much the same as those of Narayan et al except that one adverse factor alone is regarded as sufficient indication. RICP in children occurs more frequently than in adults and Shapiro and Marmarou found that 86% of monitored children with a GCS of 8 or less developed an ICP above 20 mm Hg.

Methods of Monitoring Intracranial Pressure

Extracranial

Apart from a tonometer which measures the tension of the infant's fontanelle there are no extracranial devices in clinical use. I have no experience of these devices and there is scant evidence in support of their accuracy.

Extradural

Extradural monitoring is the least invasive of the techniques in common use, and is virtually free of serious complications as the dura mater is not breached. Intracranial infection and haemorrhage are virtually unknown and the device can remain in place for as long as is considered necessary.

The measuring surface must be coplanar with the dura, and if the dura is not deformed by the sensor, dural tension will not influence measurement of intradural pressure. To satisfy these requirements the technique of placement is important, a poor technique inevitably leading to unreliable results. In our unit we have for the past seven years employed epidural monitoring, using mainly the Hellige device because of its cost-effectiveness, and also the Ladd fibre-optic monitor, particularly for children in whom the skull is too thin to accept a bolt which must be seated in it. The Hellige device screws into a burrhole and accepts a Statham transducer in such a fashion, if correctly inserted, that its membrane lies in complanar contact with the dura mater without deforming it. The sensor of the Ladd fibre-optic monitor is slipped in between the dura mater and the skull through a burrhole after stripping the dura mater adequately from the skull to prevent the device from becoming wedged between the two. Freeing the dura mater from the skull to provide an adequate area of mobile membrane is critically important with both devices and failure to do so is a major reason for unreliable readings.

We have recently embarked on a study to compare epidural pressures with ventricular pressures, and our findings in the first 11 patients are very similar to those of Dorsch and Symon who found that the epidural pressure is usually 5 to 10 mm Hg higher than the intraventricular pressure, but that the difference may increase up to 20 mm Hg with high intracranial pressure. It is not clear at present how much, if any, of the pressure difference is real and due to tissue pressure gradients, and how much is due to erroneous readings, possibly inherent in the epidural technique. We have not so far found the rapid early upward drift, recently reported by Power and Crockard, which would invalidate epidural recordings

if substantiated. Our satisfaction, and that of Dorsch and Symon, with epidural monitoring is shared by Gobiet et al and by Sündbarg and Nomes who found pressure differences between the intraventricular and epidural spaces of similar magnitude to those reported by Dorsch and Symon. They felt that this pressure gradient was real and not artificial.

Subdural

A technique which employs a monitoring device in the subdural space eliminates any possible pressure-distorting effect of the dura mater. Many of the devices which are used to measure the pressure in the subdural space, or even in the cortical subarachnoid space, are open lumen screws such as the Richmond screw, or catheters which also depend on the free transmission of pressures along fluid-filled pathways. The theoretical objection that the orifices of these tubes are very likely to become obstructed, particularly when the brain surface becomes apposed to the dura under pressure, has been substantiated in studies such as those of Mendelow and colleagues. They showed that the Richmond type screw with its terminal hole is only a little more likely to give a false low reading than the Leeds type screw with its lateral holes, especially when the pressure rises above 20 mm Hg. The same objection cannot be made to subdurally placed monitoring devices which are not open to the subdural space.

Infection nevertheless remains a problem and this will be discussed when we consider intraventricular monitoring.

Intraparenchymal

Intraparenchymal pressure monitoring is not in clinical use and is mainly an experimental tool to investigate intracranial pressure gradients.

Intraventricular

This is the standard against which all other methods are measured. It is virtually the only method in which the accuracy of the reading can be vouched for if the catheter is demonstrably patent. But this may not necessarily be adequate reason to reject subdural and epidural readings which do not conform to ventricular pressures, as this presupposes the erroneous assumption that the brain behaves as a fluid and that pressure gradients are not possible.

Apart from usually reliable readings, for they can also mislead, ventricular catheters offer the opportunity of venting CSF and thus lowering ICP.

There are, however, major disadvantages with any method which requires that the dura mater be breached. In the Richmond series the infection rate with ventricular catheters was 6.3% and with subarachnoid screws 5.3%. They removed the devices as soon as possible after three days, at a time we have often found them to be as useful as in the previous few days. Eighty-five per cent of patients who developed infection had been monitored for five days or longer. No infections occurred in the first two days. This still implies that 15% of their infections occurred on the third and fourth days.

In this series intracerebral haemorrhage occurred in 1.4%.

Thus when the dura is breached there is a risk of major complications, even with only a few days of monitoring, of about 7%. This is a not inconsiderable figure and may justify opposition to ICP monitoring if this were the only method available.

The Treatment of Raised Intracranial Pressure

In the absence of a surgically removable space-occupying lesion, ICP can be lowered only by reducing the volume of intracranial blood, or of the CSF, or of the water content of the brain. All measures that are taken to reduce ICP are aimed at one of these compartments and which compartment will be evident from the discussion.

Once the decision has been made to do so, the ICP monitor should be inserted as soon as possible. We have discussed the reasons for doing so when active resuscitation is necessary for head injured patients in shock, particularly in the case of children. It is also of considerable prognostic help at this early stage after head injury. If the GCS score is 5 or less and the initial ICP is greater than 40 mm Hg the outcome is almost invariably fatal unless this early pressure rise can be rapidly controlled. In patients with fixed dilated pupils and flaccidity in whom the ICP and mean arterial pressure approach each other, cerebral angiography will show non-filling of cerebral vessels and resuscitation can be abandoned. This saves considerable anguish in relatives and nursing staff.

If a decision has been taken to monitor ICP, the patient is intubated. This is usually most easily done after administering Pavulon (pancuronium bromide) 0.2 mg/kg and sodium pentothal 3-5 mg/kg.

The level of ICP at which treatment is instituted to lower it varies from unit to unit, but it is usually at a point between 15 and 25 mm Hg. We have noted that Saul and Ducker significantly reduced the mortality in their patients with head injury by commencing to treat RICP at a level of 15 mm Hg rather than 20 to 25 mm. It has also been our policy for some years to commence treatment at a pressure of 15 mm Hg. The normal ICP in adults and in older children is in the region of 10 mm Hg and does not exceed 15 mm. In children the pressure is between 3 and 7.4 mm and in infants between 1.5 and 5.9 mm Hg.

Intracranial hypertension is managed in the following way:

The patient is nursed with the head elevated 15 to 30° and unrotated, unless the maintenance of blood pressure gives cause for concern. Durward et al have shown that ICP falls significantly with elevation of the head to 15°, and a little further with elevation to 30°, without significant change in the cerebral perfusion pressure (CPP). WIth elevation of the head to 60°, however, they found that ICP often rose to a level higher than when the patient is in a horizontal position. This probably occurs as a result of a rise of central venous pressure following upward pressure of the abdominal contents on the diaphragm. Cerebral vasodilation which occurs in response to a fall in intracranial arterial pressure also probably plays a role. In this position, with a rise in ICP and a fall in intracranial arterial pressure, CPP may fall

significantly. Occasionally, at 60° elevation, ICP fell further with maintenance of CPP so that this degree of elevation may be cautiously explored if the need arises.

Rosner and Coley found that CPP was maximal when the patients were nursed flat in bed, but perfusion pressure is not, of course, the only consideration. It is common experience that rotation of the head may cause marked elevation of ICP, probably because of kinking of the vertebral venous plexus, with cerebral engorgement.

The patient is hyperventilated with a volume-cycled ventilator to maintain a $PaCO_2$ of 25 to 30 mm Hg. The tidal volume is adjusted to about 12 to 15 mL/kg, after which the ventilatory rate is adjusted to effect the desired $PaCO_2$.

The patient must be adequately relaxed and sedated so that movements, and in particular decerebrate posturing, do not raise central venous pressure (CVP) through increased venous return. If he "fights" the respirator, his intrathoracic pressure and CVP will also rise and cause a rise of intracranial venous pressure.

As an emergency measure for rapid reduction of ICP, vigorous ventilation using an Ambu bag or similar device can be employed. While rapidly reducing ICP, vigorous hand ventilation may cause a significant fall in blood pressure with a resultant fall in CPP and possible cerebral ischaemia. Positive end expiratory pressure (PEEP) variably affects cerebral perfusion pressure, but we have found it wise to allow the merits of the pulmonary pathology dictate its employment and, within limits, its magnitude up to 10 cm H_2O.

The $PaCO_2$ may be lowered to 20 mm Hg, if necessary, and experience has shown that children respond to changes in $PaCO_2$ well below the 20 mm Hg level suggested for adults. It has been suggested that jugular venous PO_2 be measured to guard against cerebral ischaemia at these low levels of $PaCO_2$, and that hyperventilation be reduced if the jugular venous PO_2 drops below 20 mm Hg. It has, however, never been shown that structural damage to the brain can be caused by the cerebral vasoconstriction that is brought about by hyperventilation.

Cerebral blood flow changes by about 1.8 mL/100 gm brain/1 mm Hg change in $PaCO_2$, and cerebral blood volume by about 0.04 mL/100 gm brain/1 mm Hg change in $PaCO_2$ in the normal person. These changes are probably much greater when we are dealing with a hyperaemic brain.

If ICP rises above an acceptable level, and we have taken this to be 15 mm Hg, further active steps have to be taken. If ICP is being measured using an intraventricular catheter it is expedient to remove CSF by allowing it to escape spontaneously against a pressure of about 150 mm H_2O. This prevents ventricular collapse. We do not usually use a ventricular catheter so that our next step is the administration of mannitol.

It is usually administered as a rapid intravenous infusion of 0.25 gm/kg and, depending on the level of the ICP and its response, administration is repeated at this dosage or at 0.5 mg/kg at about four to six-hourly intervals, or as indicated. Serum osmolality levels monitor the administration and are kept below 310 or at most 325 mOsm/L to prevent renal tubular damage. It is important to prevent dehydration by carefully maintaining fluid balance.

There is still controversy concerning the mechanisms of action of mannitol. The oldest and simplest view is that the rise in serum osmolality that it causes withdraws water from the brain across the blood-brain barrier. Its effect on areas with impairment of the blood-brain barrier is less certain and it has been proposed that it may pass through the defective barrier and increase oedema by attracting water into the tissues. It has also been suggested that mannitol can withdraw water from the ventricles into the bloodstream and so reduce CSF volume. Mannitol causes an increase in cardiac output and consistently increases cerebral blood flow in patients whose perfusion pressure is below 60 mm Hg. It is also well recognized that mannitol may improve cerebral blood flow in ischaemia without greatly altering perfusion pressure. This is probably due to a combination of factors such as the improved blood rheology due to haemodilution as well as the increased cardiac output. Mannitol may have an even greater effect on cerebral compliance than an absolute ICP and thus protect the patient against ICP rises following intracranial volume increases.

In children mannitol must be used a little cautiously as the cause of RICP is so often cerebral hyperaemia with increased intracerebral blood volume. Increasing the blood flow to the brain may be counterproductive in these circumstances, although it has been suggested that when autoregulation is intact, mannitol may carebral vasoconstriction, with a reduction in cerebral blood volume.

If the addition of mannitol fails to control ICP, one may abandon further attempts at reducing intracranial hypertension with the dubious consolation that there does not appear to be incontrovertible statistical evidence that, with the exception of children with brain swelling, the reduction of RICP is important in the management of head injured patients.

The next line of defence is admittedly of more doubtful effectiveness than the previous, and consists of the employment of anaesthetic agents such as althesin (now withdrawn from the South African market) and barbiturates. We have no experience with gammahydroxybutyrate and etomidate, nor of ethacrynic acid. We have, however, employed barbiturates extensively to reduce RICP which has resisted other attempts to do so, and are impressed by their effectiveness in achieving this. We, however, have no statistical evidence that the patient's chances of survival are enhanced, although Saul and Ducker, Marshall et al and many others have adduced evidence that this is so. Ward et al found that prophylactic barbiturate coma did not lead to better ICP control in a series of patients, nor did it enhance survival. They cautiously concluded, however, that although prophylactic barbiturate coma appeared to be ineffective, baribiturates may still be necessary to control high ICP when all other methods fail. Miller who is sceptical of the value of baribiturates, nevertheless still uses them when the other measures fail. He states that he now uses them in a different wat by administering thiopental as a single intravenous infusion "hoping for a prolonged effect from an average dose in adults of 500 mg". We would nevertheless agree with Miller when he states that no verdict can yet be brought in and that there is need for properly controlled trials before a final decision can be made concerning their role in reducing mortality and morbidity following head injury.

What is the rationale for using barbiturates in patients with RICP? Barbiturates in therapeutic doses may be expected to reduce cerebral metabolism by up to 55% and cerebral blood flow by 48%, at which point cerebral vascular resistance is increased by 80%. Such a vasoconstrictor effect is the only tenable explanation for the rapidity of action of barbiturates

in lowering ICP. Because cerebral vascular dilatation is such a common cause of RICP in children following head injury, it is not surprising that barbiturates are especially effective in lowering intracranial hypertension under these circumstances. It would be out of context to discuss the other possible beneficial effects of barbiturates on the brain here.

To justify the often rather emotional objections to the use of barbiturates there must obviously be undesirable side effects. It is so that the patients become anaesthetized for prolonged periods and require vigilant care. They are, however, in coma to start with and by this stage are undergoing artificial ventilation and ICP monitoring, for barbiturates would not be used in other circumstances, nor would their use be recommended outside the confines of a neurosurgical intensive care unit. In these circumstances the administration of barbiturates do not in out experience add to the burdens of patient care.

Arterial hypotension is, however, a major undesirable side-effect. Barostatic reflexes are impaired, but convincing evidence of a direct effect on the myocardium has not been documented. The main cause of the hypotension appears to be a relaxation of tone in venous capacitance vessels with a diminution of circulating blood volume. It is thus of paramount importance to ensure that the patients are not hypovolaemic, as they often are after prolonged mannitol administration. This lesson has been learnt in the prevention and treatment of cerebral vasospasm following subarachnoid haemorrhage. Central venous pressure is maintained at an adequate level and if there are any uncertainties concerning cardiac output, a Swan-Ganz catheter should be inserted. An adequate cerebral perfusion pressure must be maintained, and it is almost invariably necessary to administer pressor agents such as dopamine or dobutamine.

I am not aware of acceptable documented evidence showing a clearly higher infection rate in patients undergoing treatment with barbiturates. There is, however, a dose-dependent interference with phagocytosis of leucocytes exposed to thiopental in therapeutic doses, as well as a depression of lymphocyte activation. Of the drugs examined, namely phenobarbital, pentobarbital, and thiopental, only the latter exhibited this effect in concentrations likely to be attained during the treatment of patients. Dangerous levels of hypothermia must be guarded against.

Thiopental, pentobarbital, and phenobarbital have all been used to lower RICP. We have found thipental undesirable because of its high sodium content, its irritating effect on veins, and its long half-life of 38-86 hours with prolonged administration. Phenobarbital also has a long half-life of 60 to 120 hours but has the advantage of having the least effect on the systemic circulation. However, it appears to have the smallest effect on ICP. The most frequently used agent is pentobarbital which has a half-life of about 26 hours. We have used if for a number of years.

A loading dose of 3-5 mg/kg is given slowly intravenously and is followed by maintenance at as low a dose as is appropriate to control ICP, usually in the range of 1-3 mg/kg/hour. Additional boluses may be necessary to control pressure peaks, especially during manoeuvres such as tracheal toilet. A pentobarbital serum level of above 50 mg/L probably does not enhance its effectiveness a great deal, and hypotension increasingly becomes a problem as the level rises.

Barbiturate therapy is stopped when the ICP has been below 15 mm Hg for three days or if intracranial hypertension has become unmanageable despite its administration. When the ICP approaches the mean arterial pressure, brain death becomes certain. Absence of cerebral circulation on 4 vessel arteriography is, however, the only certain way of demonstrating that a patient on barbiturate therapy has sustained brain death.

We have not discussed the use of steroids in the management of RICP. Their considerable and uncontested efficacy in the management of the cerebral oedema caused by brain tumours and brain surgery was first documented by Galicich and French. It was eminently reasonable to extrapolate from this that dexamethasone and other corticosteroids should be effective in the control of oedema after trauma to the brain. Steroids were accordingly used worldwide for many years for this purpose. Certain studies such as those of Gobiet et al and Faupel et al seemed to indicate efficacy in a very high dose range, but subsequent well-controlled studies have failed to substantiate this. Steroids do not appear to reduce the incidence or magnitude of intracranial hypertension, nor to influence the outcome of head injury.

Saul and Ducker were somewhat cautious in their conclusions and suggested, on the basis of a tendency that did not reach statistical significance, that there might be a group of patients in whom steroids may prove beneficial.

We still occasionally uyse dexamethasone, often with apparently good result in lowering RICP, in patients with a focal contusion and CT scan evidence of local oedema round it. But the evidence as it stands at present seems convincing enough not to use steroids as a routine treatment following head injury, nor routinely in the control of RICP after such injury.

When all has been said concerning the control of intracranial hypertension following head injury, it must be clearly stated that this probably plays a relatively small part in improving the outcome of the patients as a group. Much more important is the preservation of the well-being of the patient as a whole, with particular attention to meticulous respiratory care, and the maintenance of the fluid and electrolyte balance and nutritional status.

Chapter 4.4: Spinal Cord Compression

J C de Villiers

Introduction

Compression of the contents of the spinal canal can result from a wide variety of lesions encountered in different specialities so that an individual practitioner may not see many examples unless his practice is pre-occupied with diseases of the spine and spinal cord. It is therefore difficult to form an estimate of the incidence of spinal cord compression in any population, as local conditions and the prevalence of particular pathological entities may determine its occurrence in a community, i.e. tuberculosis in the southern African population.

Spinal cord compression will inevitably lead to paraplegia which is the single term into which a patient's complex disability is contracted. For the patient it means loss of

mobility, dependence on others for all his needs, loss of skin sensation with risk of unnoticed damage to skin and subsequent septic complications, incontinence of urine and faeces as well as sexual incompetence. All these occur in patients who usually have perfect intellectual function and it is a condition which should be avoided at all costs. The eventual cost to a community to care for one such patient is difficult to determine.

Definitions

Gradual or sudden intrusion into the spinal canal by any expanding lesion will lead to compression of spinal cord, nerve roots and their blood supply. The resultant weakness due to spinal cord dysfunction may be either slight (paraparesis) or total (paraplegia). Different types of paraplegia may be encountered even when the patient is first seen due to delay in presentation.

Upper Motor Neurone Flaccid Paraplegia (Spinal Shock)

This occurs when there is complete loss of function (sensory, motor and autonomic) below a lesion of the spinal cord of rapid or abrupt onset. It can occur even after long antecedent progressive disease such as tuberculosis of the spine or slow-growing metastatic neoplasm, when eventual vertebral collapse may cause sudden functional transsection of the cord.

The limb musculature below the lesion is atonic and all sensation is lost; there is absence of sweating and a paralytic ileus may be present. Bladder distension occurs painlessly and overflow incontinence may ensue. Absence of tone is gradually replaced by paraplegia in flexion if the lesion is complete.

Upper Motor Neurone Spastic Paraplegia in Flexion

This may follow upper motor neurone flaccid paraplegia or a progressive compressing lesion, such as a benign spinal cord tumour, may lead to it. There is no voluntary motor function below the level of the lesion and impaired or total loss of sensation. The tone in the limbs is increased, tendon reflexes are exaggerated and plantar responses are extensor. Painful flexor spasms which form a prominent part of the clinical picture are precipitated by even light cutaneous stimuli and may be accompanied by voluntary evacuation of bladder and bowel (the mass reflex).

Upper Motor Neurone Spastic Paraplegia in Extension

There is weakness or total paralysis of the lower limb accompanied by elevation of tone mainly affecting the extensor muscles. This is usually associated with incomplete lesions of the cord (such as benign compression). The reflexes are exaggerated, plantar responses are extensor, sensation may be partially impaired or totally lost and bladder control similarly ranges from almost normal function to complete loss of voluntary control.

The Brown-Sequard syndrome may occur, particularly in benign lesions, compressing the spinal cord antero-laterally and thus affecting mainly the one half of the cord resulting in

a clinical picture of a spastic paralysis of the leg on the ipsi-lateral side, with signs of spino-thalamic sensory loss on the opposite side.

Lower Motor Neurone Flaccid Paraplegia

This results from acute or chronic lesions of the *cauda equina*. Flaccid weakness and wasting of muscles occur in the final stages and tendon reflexes at the knee or ankle are absent while the plantar responses may not be obtainable. Sensory loss is in the saddle area and involves sacral and lumbar dermatomes so that rectal, vaginal and urethral sensation may be absent.

Conus medullaris lesions display a mixture of upper and lower motor neurone paralysis with absence of tendon reflexes but extensor plantar responses associated with saddle anaesthesia and retention of urine.

Lesions of the spinal cord above the fourth cervical segment will lead to respiratory failure.

Surgical Anatomy and Physiology

The dural sac which encloses the spinal cord and its emerging and entering nerve roots, extends from the foramen magnum to the level of the body of S2. Within this dural sheath, the spinal cord extends from the cervico-medullary junction to the *conus medullaris* at the L1/L2 level and the *cauda equina* continues from that level to S2. Lesions below the conus will cause lower motor neurone symptoms and signs and those above that level, upper motor neurone symptoms and signs.

Fat, which surrounds the dura within the spinal canal, is continuous through the intervertebral foraminae with retromediastinal and retroperitoneal fat. Blood vessels enter along the nerve root, some of them being particularly significant in supplying the spinal cord. The intervertebral foraminae are also portals of access to the posterior mediastinal and retroperitoneal spaces so that pathological processes in these spaces may enter the spinal canal and cause compression via this route.

The spinal canal is a rigid, unyielding space (like the cranial cavity) and any space-demanding lesion in it will only be able to expand at the expenses of the CSF, vascular or neural elements giving way in order to accomodate the growing mass.

Progressive compression causes a transverse spinal lesion which will affect the root(s) at that level so that local radicular loss of function (sensory and motor) will occur while cord function (sensory, motor and autonomic) will be affected to a variable extent below that level.

Pathology

The spinal cord may be compressed by any mass lesion occurring within the spinal canal or encroaching upon it from without. A time-honoured and practical classification takes the dura, with its high resistance to transgression by tumour and infection, as the dividing line between the two main types of compression: extradural and intradural.

Those masses which occur intradurally can also be divided into those which occur primarily in the neural tissue and those which lie outside it but still within the sheath of the dura:

- extradural - intramedullary

- intradural - extramedullary.

An exhaustive encyclopaedic list of all the causes of spinal cord compression may leave a newcomer to the field in confusion and would serve little purpose. Consideration of the more common causes and paying particular attention to the earliest clinical neurological features of spinal cord compression can be carried out as soon as possible, will be to the patient's greatest benefit.

From a practical point of view, the commonest cause of spinal cord compression encountered in adults will be compression by metastatic deposits in the vertebrae with or without vertebral collapse. After that, tuberculosis is the commonest cause, particularly in childhood, in southern Africa.

Causes of Spinal Cord Compression

Extradural

Tumouor

- Secondary deposit in bone

- Primary bone tumour

- Retroperitoneal tumour extending into the vertebral canal

Infection

- Extradural pyogenic abscess

- Spinal tuberculosis

Haemorrhage

- "Spontaneous", anticoagulants

- Arterio-venous malformation

- Trauma

Traumatic Compression

Degenerative Spinal Disease

Central Intervertebral Disc Prolapse

Intradural

Intramedullary

- Primary neural tumours

- Cysts

- Lipoma

Extramedullary

- Neurofibroma/Neurilemmoma

- Meningioma

Clinical Diagnosis

The best hope of effecting a cure or alleviating the symptoms of a patient with spinal cord compression lies in early diagnosis and surgical decompression. The patient's earliest complaints are therefore all-important.

Sensory Dysfunction

Pain

Since pain in the back is a common symptom and in the lay mind usually attributed to strain, fatigue, "fibrositis", "rheumatism" and other plausible explanations, it is often neglected. Pain occurring at a localized site in the spine, particularly if it radiates around the trunk into the anterior chest wall or abdomen, and if associated with local tenderness, is highly suggestive of a potentially compressive lesion. (At times such pain radiating to the front, or even localized to the front of the trunk, is attributed to cardiac, pleuritic or upper gastrointestinal causes.) Pain of recent onset which is progressive is more significant than intermittent chronic pain.

Three types of pain are described: Vertebral, radicular, and tract pain.

Vertebral Pain

This is well localized and due to involvement of the bone by a metastatic or infective process. It tends to be deepseated and boring in character, is constant, often worse at night

and not relieved by change in posture. In rare instances pain or malignancy may be aggravated or initiated by minimal alcohol intake.

It is important to note that bone pain may precede radiographic and other objective manifestations of metastatic disease, so that it may be mistakenly diagnosed as degenerative disease of arthritis. A dictum that will save many patients much discomfort and even avert disaster is:

Thoracic back pain in a patient over 40 is never insignificant.

Radicular Pain

The significance of radicular pain is that it localizes the lesions to a specific dermatome and hence also to a segment of the spinal cord. It is lancinating and aggravated by manoeuvres that raise intra-thoracic and abdominal pressure and may wake the patient from sleep. It indicates a localized partial compression of a nerve root with irritation when the intrathecal pressure is transiently elevated. Pain is felt in a root distribution and afterwards as a tight band round the trunk.

The diagnostic error most likely to be encountered is pain occurring in the lumbar region with radiation into the groin or leg. This is almost invariably attributed to degenerative or disc disease, forgetting the possibility of intraspinal or retroperitoneal involvement of the lumbar or sacral plexuses by neoplasia.

Tract Pain

This pain, which is diffuse, aching and continuous in nature and is often associated with increased sensitivity and dysaesthesia of the skin to touch, is probably due to spinothalamic tract involvement.

The following important associations with pain should be recognized:

- a painful, stiff back in a child indicates a spinal tumour until disproved

- a painful, stiff back with severe local tenderness and fever is highly suggestive of a spinal extradural abscess

Sensory Symptoms and Signs

These may include numbness and tingling in the legs or feet or be described as if walking on cotton wool. Sensations of heat or cold in the legs are particularly common in mid-dorsal compression. Paraesthesia may be associated with little or no sensory loss, or conversely, the patient may be totally unaware of his sensory loss and present with peripheral skin excoriation and ulceration. Numbness or loss of urethral, vaginal or rectal sensation may be the complaint in *cauda equina* lesions.

The finding of a sensory level where one or all of the sensory modalities may be altered, significantly localizes the spinal segment involved. The importance of careful testing

for sensory levels, especially examination of the sacral segments (saddle area), in every patient with a suspected spinal lesion cannot be stressed enough.

Motor Dysfunction

Weakness is usually the earliest symptoms and may manifest itself in many ways: stiffness, clumsiness or catching the toes when climbing stairs. Sudden dramatic loss of power may occur after a minor injury, spinal manipulation or merely getting out of bed when collapse of a diseased vertebra occurs.

The inability to manipulate small objects (i.e. fastening buttons) may be the first indication of weakness of the small hand muscles but commonly the patient will complain of numbness of "wooliness" of the fingertips rather than of weakness.

Examination to confirm power loss must involve all the main muscle groups, and includes evaluation of tone and reflex testing. Slow onset spinal cord lesions will produce distal, spastic weakness (upper motor neurone) while flaccidity and wasting (lower motor neurone) will occur at the level of the lesion, due to local root involvement.

Cauda equina compression occurs below the level of L1 vertebral body, and motor paralysis (lower motor neurone type) corresponds to the root or roots involved.

Visceral Manifestations

Sphincter dysfunction of the bladder manifestations itself initially as urgency, progressing to painless retention. Overflow incontinence or reflex bladder emptying may be mistakenly interpreted as intact bladder function by the patient or doctor, unless further clues are sought in history and examination, loss of rectal sphincter function may be associated with incontinence or constipation.

Examination should include abdominal palpation and percussion to ascertain distension of the bladder together with rectal sphincter assessment, bulbocavernous and anocutaneous reflex testing. A spinal cord lesion (upper motor neurone) will be associated with a small reflex emptying bladder and positive bulbocavernous and anocutaneous reflexes. (Acute compression results in "spinal shock" with features similar to a lower motor neurone lesion.) *Cauda equina* or *conus medullaris* involvement is associated with a large bladder which is percussible abdominally and leads to overflow incontinence (lower motor neurone lesion) with absence of bulbocavernous and anocutaneous reflexes.

Sphincter changes may occur early and be the only manifestation of compression of the *cauda equina* or *conus medullaris.*

Autonomic Manifestations

Impotence is the dominant symptom. Hypotension is found in cervical and upper thoracic lesions while hypertension may occur because of auto-isolated spinal segments being stimulated by bowel or bladder distension. Sweating abnormalities and Horner's syndrome may be present and indicate levels involved.

Vertebral Column

Deformity such as scoliosis or loss of lordosis indicates muscle spasm irritation due to focal lesion. Limitations of movement also indicates muscle spasm or mechanical dysfunction due to a bony lesion or disc prolapse. A kyphus usually suggests tuberculosis.

A paraspinal mass is indicative of a tumour or infective lesion. A midline dimple, sinus, tuft of hair or skin angioma may indicate an underlying lesion such as an epidermoid with or without infection.

Local tenderness may identify the level of a bony lesion.

Special Investigations

A full blood count, determination of the erythrocyte sedimentation rate, blood chemical estimations and serological tests for syphilis are standard investigations for all neurological complaints. Alterations in serum protein or presence of Bence-Jones proteinuria may assist in diagnosis of myleoma.

Plain Radiography

It is advisable to take radiographs of the complete spine including the craniocervical junction. Antero-posterior and lateral projections are usually sufficient except in cases where a neurofibroma involving an intervertebral foramen is suspected, in which case oblique views characteristically show expansion of such a foramen. Although radiographs of the spine are often diagnostic, they may show no abnormality in the initial stage of the disease when pain is the only feature.

Abnormalities commonly seen in metastatic disease include vertebral collapše, erosion of pedicles and spinous processes without early disc-space involvement. Infection causes vertebral collapse with early disc-space narrowing and in tuberculosis and tumour a paravertebral mass may be present. Erosions produced by benign tumours or slow-growing entities cause well-defined, clearly outlined defects and scalloping of the vertebral bodies with maintenance of the disc-space integrity.

Chest radigraphs are advisable in all cases, since a primary neoplasm, other secondary deposits or tuberculosis may be demonstrated.

Radio-Isotope Studies

Radio-isotope bone scans demonstrate lesions at an early stage, often before plain radiographic changes occur. These are relatively non-invasive and easily available investigations. It is often less informative than radiological studies in myeloma.

Lumbar Puncture

Lumbar puncture without associated myelography as a diagnostic procedure is not indicated when a compressive spinal lesion is suspected. Indeed, the withdrawal of

cerebrospinal fluid removes the cushioning effects of the fluid between the spinal cord and the compressing agent and this may change a partial dysfunction into total paralysis.

CSF analysis (biochemistry and cells) are rarely of diagnostic value in compressive lesions. Queckenstedt's test is too unreliable to be of assistance in diagnosis.

Myelography

This is still the diagnostic procedure of choice and should be carried out as a prelude to surgery after consultation with a neurosurgeon. It should be a planned investigation so that CSF can be obtained for whatever analysis may be thought necessary and also for surgery to follow if indicated.

Computed Tomographic (CT) Scan of Spine

CT scanning of the spine has limited application as a screening procedure at present but can clarify the nature of the lesion when the level is known. It is particularly helpful in further defining bony lesions and tumours with both extra- and intraspinal components. The diameter of the bony canal and laterally situated lesions are well demonstrated. It is most useful in the diagnosis of intramedullary cystic lesions and lipomas.

Treatment

Treatment should be regarded as an extremely urgent matter. A delay may mean the difference between permanent paralysis and good functional recovery. Time should not be spent unnecessarily in diagnostic pursuit when urgent decompression - which will also provide histological certainty - is required.

The modes of surgical treatment are tailored to the individual lesion and should be carried out in centres equipped to deal with this adequately. In general, posteriorly situated lesions should be dealt with by laminectomy, while compression of the cord from anteriorly usually demands an anterior approach if the site of the lesion and the patient's condition permit it. In some situations bone grafting or other forms of stabilization (metal struts, acrylic) may be required after tumour removal or decompression.

Should surgery not be indicated in malignant disease, needle biopsy of a paraspinal mass or affected bone may be necessary to establish a diagnosis. In some malignant tumours radiotherapy with or without chemotherapy may be indicated without surgical decompression. This is also indicated in multi-level metastatic compression.

Following the acute surgical management, the patient is rehabilitated so as to recover maximum functional capacity as soon as possible. Rehabilitation of the spinal paralysed patients is an integral part of management and should commence from the time the patient is first seen: prevention of deformity and of pressure sores as well as the patient's psychological status should receive careful consideration.

Prognosis depends on the nature of the causative agent, i.e. whether benign or malignant. In the latter instance, the degree of malignancy and response to therapy will

determine the patient's life expectancy. Neurological outlook is determined by the degree of functional loss, duration of paralysis and to some extent rapidity of evolution. Long-standing total paralysis caused by a benign lesion will not benefit from surgery and life expectancy will depend on the management of paraplegia.

Chapter 4.5: Intracranial Sepsis

R Lipschitz, P Miller

Surgical intracranial sepsis is a collection of pus in extradural, subdural or intracerebral location. These discrete or localized collections exert mass effects intracranially and are amenable to surgical drainage. Meningitis and ventriculitis (i.e. spreading diffuse inflammation of the meninges or ependymal lining) are excluded from the discussion, but it must be appreciated that it occasionally co-exists with intracranial pus collections. Osteomyelitis of the skull is also excluded from the discussion since it is, strictly speaking, not intracranial in nature, although it may lead to intracranial sepsis. Cerebritis will be discussed later.

The incidence of intracranial surgical sepsis varies widely, but it is by no means a rare condition. The neurosurgical unit at Baragwanath Hospital, drawing the black populations of the Witwatersrand, treats approximately 80 cases of primary intracranial sepsis per year. Iatrogenic sepsis, i.e. following a previous intracranial operation for aneurysm or tumour, is not included. The unit at Johannesburg Hospital, drawing the white population, treats only five to eight cases of intracranial sepsis per year. It is thus evident that socio-economic factors play a role in the aetiology of intracranial sepsis.

There is always a primary lesion which is responsible for intracranial sepsis. This should be found and eradicated as part of the treatment.

Ethnic Groups

Amongst the black population the most common cause is ENT sepsis like frontoethmoiditis and/or mastoiditis. This sepsis may be acute or chronic, and often may not have been treated at all, or inadequately treated. When neurological signs and symptoms are found in association with ENT sepsis, one should immediately think of, and exclude, intracranial infection.

Age

Cerebral sepsis occurs most often in young persons between 15 and 25. ENT sepsis may cause meningitis, extradural empyemas, subdural empyemas, cerebral cerebritis and cerebral abscesses which may be single or multiple. Cerebral abscesses and subdural empyemas are the most frequent. The infection may spread locally, by eroding through and infecting the bony plate between the inflamed cavity and the intracranial components. Thus, there may be an associated area of localized osteomyelitis and the infection may spread externally, to form large subcutaneous pockets of pus. The infection also spreads by venous "seeding" - since there is free communcation between the veins outside the skull and the intracranial veins. Thus fronto-ethmoiditis may be associated with cavernous sinus infection

(which must be differentiated clinically and radiologically from orbital cellulitis). Mastoiditis may cause thrombosis of the transverse (lateral) sinus. Subdural empyema is frequently associated with a thrombosis of the superficial cortical veins which are in contact with the pus collection. These venous problems often exacerbate the condition.

Abscess Formation

Fronto-ethmoiditis most often gives rise to abscess formation in the frontal lobe, although rarely temporal and occipital collections may occur. Sinusitis may give rise to a spreading subdural empyema which, because of the nature of the subdural space, frequently extends over the convexity of the involved hemisphere. This empyema may be loculated, is usually unilateral, and is often associated with an interhemispheric collection of pus. Occasionally the empyema is purely in an interhemispheric location. The extradural empyemas which occur are always frontal in location, small or sliver-like, and may contain gas or air.

Mastoiditis usually results in abscesses lying either in the ipsilateral temporal lobe or cerebellar hemisphere, although collections of pus lying over the tentorium are not uncommon.

Treatment

The treatment of the IC sepsis include eradication of the ENT sepsis, or the intracranial conditions will not subside.

The second most common cause of intracranial sepsis is an infected compound skull fracture. This leads to single or multiple abscesses (which are usually localized to the area under the septic skull wound) or to an ipsilateral subdural empyema. At the time of presentation the skin wound overlying the fracture site is usually grossly septic, and often there is a "boggy" tender swelling discharging pus. However, only a minority of penetrating skull wounds become septic and some injuries, especially penetrating wounds from pitchforks and slivers of wood, may lead to anaerobic infection, with gas formation in the areas of IC sepsis. It is noteworthy that fractures of the skull base, with CSF leakage, rarely lead to abscess or empyema formation, but rather to acute or chronic meningitis.

An underlying IC haematoma or an intracerebral clot may provide a nidus for infection, especially if it is in communication with a compound fracture.

It is important to differentiate on history and clinical examination between the primary neurological effect resulting from a head injury and the subsequent further deterioration which may occur when IC sepsis ensues.

Post-Operative and Co-Existing Sepsis

Neurosurgical deterioration occurring some days after an operation for a traumatic intracranial haematoma should alert to recurrence of intracranial clot and/or intracranial sepsis. In any case of IC sepsis occurring after trauma, the presence of a retained foreign body, i.e. wood, sliver of brick, sand, or a retained knife blade fragment should be excluded.

The third cause of IC sepsis is from co-existing chest sepsis. The patients often have coexisting TB (active or healed) underlying the bacterial chest infection, and those cases are often malnourished or alcoholics, causing a general lowering of the body's immune systems. The method of spread to the brain is haematogenous (arterial) seeding, and the cases classically have multiple abscesses scattered throughout the brain, i.e. there is no anatomical localization within a particular lobe or area of the brain. Abscesses may also arise as a result of metastatic lung carcinoma with secondary sepsis and a biopsy of the abscess wall may reveal co-existent carcinoma.

Although extremely rare, IC sepsis has occurred after infections as varied and widespread as gangrene of one foot, hepatic abscesses, insect bites on the neck, osteomyelitis of the mandible, and infected carious teeth; in fact from any sepsis elsewhere in the patient.

IC abscess may occur in association with congenital heart disease associated with acute or subacute endocarditis. These cases usually have right to left shunts, i.e. ASD or VSD with an Eisenmenger complex. Immune suppression may result in IC infection, i.e. diabetes, renal transplant patients and patients with lymphoma, etc.

In up to 20% of cases, no cause will be found to account for the intracranial sepsis.

Capsules

Every abscess or empyema stimulates an attempt by the body's defence systems to localize and wall off the infection. Thus the pus collection may have a poorly or well developed capsule at different stages of its evolution. The more chronic and longstanding the process, the thicker and tougher the capsule and the more completely separated and delineated from the normal brain the abscess will be. The more chronic the process, the more likely the abscess or empyema will become loculated into different cavities. The earliest stage of abscess formation consists of cerebritis, i.e. an area of inflammation, oedema and necrosis at the site where pus will eventually form a collection if the process is not halted by natural or medical means. The area of cerebritis breaks down and eventually a frank abscess collection occurs. Although the area of cerebritis does exert mass effect, it is not amenable to surgical treatment, as no pus has yet formed, and at this stage antibiotics must be used in an attempt to halt the infection.

Meningitis

An inflammatory mass presenting concomitantly with a florid meningitis is unlikely at that stage to show true abscess formation ready for surgical drainage, as the meningitis implies that the walling off and localizing process has not yet occurred. A true abscess will most probably not be present for at least three to five days after the meningitis has abated. This accounts for the frequent clinical picture of obvious localizing neurological signs becoming more prominent as the meningitis begins to resolve.

However, an abscess can "de-localize", i.e. it bursts, or ruptures, most often into the ventricle through the thin lining layer of white matter between the abscess and ventricular wall (this area provides at least support against the pressure which builds up in the abscess cavity). This leads to ventriculitis, which is often a terminal event, the patient manifesting an

acute deterioration as the infected material floods the ventricles and spreads to the CSF spaces. Ventriculitis occurs as a spontaneous phenomenon in large untreated abscesses, but may rarely occur at the time of surgery, where the abscess is inadvertently opened into the ventricle. The iatrogenic variety of ventriculitis is usually less hazardous than the spontaneous event, probably because a large volume of pus is drained externally by the surgeon at the same time as the ventricle is breached, and the resulting septic load is less and as the abscess often has been walled off for some time, the patient has built up a resistance to the organisms in the pus.

As with many other CNS lesions, the blood-brain barrier around the area of an abscess breaks down, leading to a spreading vasogenic cerebral oedema which may contribute greatly to the mass effect of an abscess. This accounts for the dramatic recoveries which may transiently occur when steroids (which stabilize the BBB and allow the oedema to reabsorb) are used. The use of steroids in cases with IC sepsis is controversial, however, and will be discussed later.

Effects on Brain Tissueš

Abscesses do not destroy brain tissue to any large extent, but tend rather to displace the tissue around it, so the prognosis for recovery may be reasonable if timely treatment is undertaken. The area around the abscess or empyemas may be transiently or permanently "unstable" so that epilepsy results. A concomitant meningitis or ventriculitis may affect the brain's ability to reabsorb CSF, so that hydrocephalus results.

The bacteriology may vary according to the etiology. The data in table 4.5.1. is gleaned from a Baragwanath series of 300 cases (unpublished).

The gram-positive organisms are predominantly staph. aureus and staph. epidermidis infections. The gram-negative group has a high incidence of *Proteus mirabilis* organisms, with *E. coli* and *Enterobacter* species coming a close second.

Cases present with signs of toxicity and infection, combined with neurological symptoms and signs. The more chronic the presentation, the less the signs of toxicity. The neurological picture may be of general dysfunction, with symptoms and signs of raised intracranial pressure, and may in addition manifest localizing signs relating to the area of the lesion. Neck stiffness is frequently present and severe, and may be caused by inflammation (meningitis) or raised intracranial pressure.

The clinical spectrum is wide, ranging from headache only in early cases, to confusion, coma and a state of decerebracy or absent brainstem function in the very late cases.

Thirty to 40% of the cases have epilepsy at some stage during the course of their illness, either on presentation, or during the course of their illness, either on presentation, or during the convalescent period after drainage. Some develop late epilepsy weeks or months after full recovery.

As stated previously, the clinical signs relating to the primary process which caused the intracranial sepsis may be overt or florid at the time of presentation.

The presence of chest sepsis (pneumonia) does not necessarily mean that the chest is the primary cause of the neurological condition, as many cases in deep coma after developing IC sepsis will develop chest infection as a result of aspiration and hypostatic pneumonia.

On presentation patients may need general resuscitation before any investigations are carried out. Those in deep coma may need to be intubated to ensure airway patency. They are often dehydrated as many have not had an adequate fluid intake during the period of confusion and drowsiness preceding the onset of coma. Epilepsy may need to be controlled during or before investigations are carried out.

If surgical IC sepsis is suspected, and surgical drainage is planned within a few hours of admission, antibiotics are withheld until pus is obtained for culture. Some cases will already have received a course of antibiotics for the primary infective lesion which has presented some days or weeks prior to the intracranial problem. In these cases pus obtained at operation is often sterile.

Investigations

Investigations must include chest X-rays to assess the chest as a site of primary sepsis and as a routine preoperative investigation, and a skull X-ray to assess the ENT sites or skull fractures as a cause of sepsis. Rarely a huge abscess with anaerobic infection may show the presence of gas in the abscess cavity. A patient with IC sepsis following recent trauma may show the presence of air in various intracranial compartments on skull X-ray.

For practical purposes, a pre- and postcontrast CT scan is the investigation which confirms the diagnosis. Angiography is no longer used, except in hospitals where CT scanning is not available. MRI may replace the CT scan in the future, but at present not enough is known of the advantages of MRI in IC sepsis.

CT scan has resulted in diagnosing IC sepsis at a much earlier stage when the lesion(s) are smaller. Because it is less invasive than angiography and ventriculography it can be used repeatedly, on a daily basis if necessary, to monitor the course of cerebritis or a small abscess. Since the advent of CT scanning the diagnosis of multiple abscesses and empyemas has increased. Previous methods and investigations tended to show the larger lesions, but were relatively insensitive in detecting the presence of associated smaller lesions. CT scanning is invaluable in showing the morphology of the lesion like loculation of the abscesses/empyemas, the presence of daughter abscesses which will require separate drainage, etc. CT scanning is useful in showing the state of the ventricles. Dilated ventricles will establish that hydrocephalus is present. When the walls of the ventricles "light up" with contrast, ventriculitis is present. CT scanning will also assess the status of the sinuses and mastoid air cells, and will establish if infarction (i.e. from venous thrombosis) and cerebral oedema are present. CT scanning should be done with and without contrast, as the abscess wall and the empyema membrane are both composed of inflammatory vascular granulation tissue, and will therefore enhance on contrast scan.

Cerebritis, on the other hand, shows an irregular homogenous hyper- or hypodense lesion on scan, which may enhance irregularly with contrast, but the typical "light-like" abscess capsule is absent.

Empyema cavities may appear small and sliver-like on scan but, when compared to small subdural haematomas in the same locations, are often associated with a greater amount of shift, swelling and cerebral oedema.

The clinical picture must never be neglected, because the typical ring-like appearance of an abscess on CT scan may resemble:

- a resolving IC haematoma

- a cystic brain tumour

- parasitic cysts

Furthermore, the CT scan will not be able to distinguish between bacterial, tubercular or fungal abscesses. The CT scan may not distinguish between empyemas and chronic subdural collections. It is the clinical picture of neurological symptoms and signs, pyrexia, toxicity and a primary source of sepsis, that establishes the diagnosis. If the presence of intracranial surgical pus (a mass lesion) is suspected, the patient should not have a lumbar puncture (LP) as the danger of cerebral coning after CSF pressure has been lowered is very real, and often leads to a rapid deterioration, usually within 12-36 hours. If an LP is inadvertently performed, it may show:

- a frank meningitis picture with turbid CSF

- a "neighbourhood syndrome" - i.e. a raised protein together with some inflammatory cells (normally 20-40 lymphocytes)

- a raised CSF pressure - this is valid only if it is measured with the patient truly at rest, i.e. relaxed, not straining, not restless and lying in a straightened-out position after being uncurled from the normal LP position

- normal appearing CSF.

Management

Most cases of IC sepsis are taken to theatre and drained as an emergency because the potential for further deterioration and death is high. Some cases with small, purely extradural collections, and who are in a good neurological state, can be operated on electively. The primary cause of sepsis must be dealt with at the same time. In a case of ENT sepsis the ENT surgeons must be consulted with a view to draining the septic ENT cavities at the same time. Similarly a patient with IC sepsis following a compound skull fracture should have a debridement/sequestrectomy under the same anaesthetic.

As soon as pus is obtained for culture, antibiotic treatment is commenced aggressively. In the first instance, while awaiting laboratory reports, frontline drugs are used, i.e. penicillin G and chloramphenicol. These are given in "meningitic" dosages, i.e. pen G is given at a dose of 2 million units two-hourly IVI. Other frontline drugs are ceftriaxone or cefatazyne and chloromycetin. Antibiotics are modified and changed when the culture results become

available. In general, antibiotics are used for six to twelve weeks after drainage. They should be continued until the patient has no further signs of toxicity and infections, and until the follow-up CT scan demonstrate that the septic lesion has been eradicated.

Antibiotics used must be those capable of crossing the blood brain barrier.

It is known that abscesses, especially small ones of 0.5-2 cm, may resolve on antibiotics without surgical drainage. Many units treat the small abscesses conservatively. In a case with multiple abscesses they may drain the larger lesions which contribute the most "mass effect", and elect to leave the smaller ones and follow the progress of the patient with CT. In a case with multiple small abscesses, it is recommended that at least one of the small abscesses be drained in order to acquire pus for culture, so that the correct antibiotic treatment can be carried out. It can be very difficult to drain one of these small abscesses "freehand", especially if it is deep, and repeated attempts to find it may do more harm than good. Today more of these abscesses are being aspirated safely by stereotactic methods.

The problem with conservative management of small abscesses is that it is difficult to predict which will heal on antibiotics, and which will enlarge and ultimately require drainage. Even if the small abscesses do heal without operation, there is uncertainty whether this method increases or decreases the morbidity and mortality.

On the other hand, even small empyemas are drained surgically. There are three reasons for this:

- Small empyemas are generally easier to locate surgically, since they are on the surface of the brain, except for deep interhemispheric collections, which may be difficult to locate and require extensive brain retraction.

- The CT scan can be misleading. "Small" empyemas contain more pus than one would estimate on the CT scan appearance.

- Even small empyemas can cause thrombosis of cortical veins.

Abscesses may be drained by one of many methods. The methods all have advantages and depending on the patient's condition, the site, size and number of lesions, the amount of brain swelling and retraction of the brain that may be needed. There is some controversy over the best method to be used in terms of the morbidity, mortality, and complication rate.

Different methods in use are:

- Aspiration through a burrhole followed by washout with saline with or without a particular antibiotic solution. No drain is left in situ and repeated follow-up scans and aspiration are done as necessary. Aspiration may be done freehand or stereotactically.

- Aspiration through a burrhole with drainage. The drain is usually a no 6 red rubber catheter, which is allowed to drain freely into the head bandages. The drain is unblocked daily with saline, and is removed when all discharge from it ceases, usually at ten days when a

track will have formed. There is controversy about which of these two methods give better results.

- Craniectomy or craniotomy to
    - excise the abscess capsule partially, entering the cavity, and sucking out and washing out the pus under vision, and leaving a "window" for drainage
    - excise the abscess and its capsule in toto.

This is usually done for the more chronic, thick-walled abscesses, where the capsule may be extremely tough, and does not crenate down when the pus is evacuated.

Craniectomy

Posterior fossa abscesses are usually drained through a craniectomy, to decompress vital structures (the brainstem) in addition to removing the abscess, as medullary coning and death is always a possibility with sepsis in the small "tight" posterior fossa. Compound skull fractures usually have a craniectomy en route to the abscess, as the infected bone must be removed as part of the treatment of the primary cause of sepsis. Craniotomies are more acceptable cosmetically, but the replaced bone flap carries the risk of becoming infected.

Empyema

Empyemas are drained as follows:

- Burrhole aspirations and washouts with drainage via at least one large catheter

- Craniectomy and drainage

- Craniectomy

Cases should have a repeat CT scan at five to ten days, depending on the clinical picture. If a scan has reached a "plateau" and is not improving, or the patient has a bulging burrhole, or continuing pyrexia and toxicity, he should be rescanned immediately. Twenty per cent of cases require repeat drainage at least once of abscesses which are crenating but persisting, or new collections of pus which have formed since the previous drainage procedure. Recurrence or formation of new abscesses is usually obvious clinically, as the clinical picture either fails to improve, or deteriorates. Recurrence should also prompt one to assess whether the primary cause of the IC sepsis has been adequately treated.

Steroids

The use of steroids is controversial. Steroids act as cell membrane and blood brain barrier stabilizers, retard oedema formation and can result in rapid clinical recovery. Steroids are also immune suppressants and can therefore worsen the infection process and retard its localization and capsule formation. Steroid use also has other complications such as peptic ulceration with an increased incidence of gastro-intestinal tract bleeding, thus many neurosurgical units do not use steroids in the management of these cases.

Prognosis

The most important factor influencing outcome is the patient's neurosurgical-mental state preoperatively. The mortality ranges from 10% in those who are alert or only confused on admission, to over 80% in those who are in deep coma with decerebrate posturing before drainage.

However, many cases who survive recover from their neurological deficits. Thus cases with initial hemiplegia or hemiparesis, speech defects, cerebellar ataxia, etc, may almost fully recover a few weeks after drainage. Nevertheless, some deficits only recover after months, so ancillary measures such physiotherapy, occupational therapy and speech therapy must be persisted with.

Once clinical and CT examinations indicate total eradication of the lesion after two to three months, it is very rare for late recurrence to occur, unless the primary lesion which led to the sepsis recurs.

Cases of epilepsy are managed with Epanutin (phenytoin) or other anti-epileptic medication. A minority of cases may need ventrico-peritoneal shunts to treat ongoing post-sepsis hydrocephalus. Permanent shunting must never be done while foci of sepsis exist in the parenchyma or CSF as the shunt itself is likely to become acutely or chronically infected. If raised CSF pressure presents a problem at this stage, recourse muct be had to direct ventricular drainage.

TB Abscesses

This has become a very rare occurence. TB affecting the CNS most commonly presents as vertebral TB which extends extradurally from a focus in the vertebral body to endanger the spinal cord. Intracranial infection usually manifests as TB meningitis, and mass lesions such as tuberculomas have become rare since the introduction of modern anti-tuberculosis treatment.

If the tuberculoma, a mass of gaseous and granulation tissue which presents as a typical isodense lesion on CT scan, is excluded, and one considers only those cases with tuberculosis inspisated pus forming a mass lesion, then the condition is even rarer.

As mentioned previously, cases with active or inactive pulmonary TB may develop ordinary bacterial infections intracranially, as a consequence of the secondary chest sepsis superimposed on pulmonary TB.

If a tuberculoma or TB abscess is diagnosed, it is important to treat with anti-TB drugs which cross the blood brain barrier.

If a tuberculoma is proved histologically at operation, the question of whether to attempt an often hazardous removal, or have recourse to anti-TB medication instead, arises. This issue is still controversial.

Fungal Abscesses

They are more common than TB lesions, although still rare. For this reason, all pus specimens submitted for culture should be accompanied by a request that testing for TB and fungi be carried out.

Fungal abscesses occur in an "odd" setting, which differ from the circumstances of bacterial abscesses:

- A small number have no discernible cause.

- A small number have diabetes and may have mucormycosis infection in the frontal and ethmoidal sinuses, with or without cavernous sinus infection. The cerebral abscesses in this setting, however, may not be fungal, but may be ordinary bacterial infections.

- Some cases arise from trauma. The history of trauma is of longer duration than in ordinary bacterial abscesses. Frequently, the infection is intracranial and that of the scalp and bone, if present is chronic and indolent.

- Many cases are immuno-suppressed. There is an association of Nocardia and other fungal abscesses with patients undergoing immunosuppression, i.e. for renal transplants.

Some patients are undergoing chemotherapy treatment for malignancy. More fungal abscesses may be seen as AIDS becomes widespread.

- Other fungal infections, usually actinomycosis, occurs in conjunction with pyogenic brain abscesses, especially secondary to long sepsis or teeth caries.

At operation the abscess capsule may be extremely thick-walled. There may be a collection of small loculated abscess cavities. The pus found may be thin and watery. On microscopy, fungal elements may be found in the capsule itself. It is for this reason that it is recommended that reliance should not be placed on aspiration alone, but that if possible a biopsy of the capsule be done.

The prognosis is often poor and is worse than for the ordinary bacterial abscesses. The infection may be refractory or very difficult to eradicate.

Amphotericin B is used to treat most fungal abscesses other than actinomycosis. A full course must be given despite the fact that the drug administration is fraught with problems in terms of patient discomfort and complications.