INTENSIVE CARE IN THE NEUROSURGICAL UNIT*

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ANY ASSAULT UPON the brain, whether traumatic or surgical, may result in unconsciousness. This may cause secondary respiratory problems, but brainstem dysfunction may directly produce hyperventilation, periodic breathing or hypoventilation. Add to this the problems of metabolic balance, hyperpyrexia, status epilepticus, and the need for frequent assessment of neurological signs, sometimes prolonged over long periods of weeks or even months, and the demands which neurosurgical patients make on medical and nursing staff are clearly seen.

Intensive Care has, of course, been practised by nurses in the wards for many years, but it is only in recent years that specialized units have been established for this purpose. This paper explores the special problems of neurosurgical patients in the light of contemporary thinking in intensive care.

Unconsciousness associated with intracranial surgery is often uncomplicated by respiratory, cardiovascular or renal malfunction, features which so often befog the issue in general intensive care work; neurosurgical patients are therefore ideally suited for instruction of nurses, undergraduates and postgraduates in the basic management of the unconscious patient.

TYPE OF PATIENT

The most common indication for instituting intensive care in the neurosurgical patient is an altered state of consciousness, but it is advisable to include most patients who cannot be fed orally, all those who have had a craniotomy however minor, for the immediate postoperative period, and of course, any other patient in whom there is a real threat of sudden loss of consciousness.

The diagnosis and frequency of the various types of patient admitted to the ICU at the Institute of Neurological Sciences, Glasgow (at Killearn), over a period of a year are illustrated in Table I. (Those admitted only for post-general anaesthetic recovery, are excluded.) It will be noted that the need for intensive care was not limited to the postoperative period, as many patients were admitted preoperatively; nor did all the patients undergo neurosurgery, as quite frequently patients were transferred from the neurological wards of the Institute for intensive care. In addition, many unconscious patients were admitted as emergencies, presumptively for surgery, which proved to be based on mis-diagnoses in the referring hospital, and it was not considered safe for these patients to be moved again; treatment (e.g. of meningitis or encephalitis) was therefore often continued at the Institute. Neoplasms (27 per cent) were the most frequent cause of admission, the next most

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TABLE I
NEUROSURGICAL INTENSIVE CARE—ONE YEAR

Diagnosis	Number admitted	Per cent
Intracranial neoplasm	179	27.2
Head injury	167	25.4
Ruptured aneurysm	146	22.2
Other CVA's	54	8.2
Cerebral abscess	14	2.1
A-V malformation	13	2.0
Encephalitis	13	2.0
Status epilepticus	11	1.7
Spinal cord compression	11	1.7
Unknown coma	7	1.1
Meningitis	6	0.9
Miscellaneous	26	0.9
Total	657	

frequent being head injuries (25 per cent) – a very important group indeed, as these patients often make good recoveries after proper care, without the need for operative surgery.

DURATION OF STAY

Average stay was 8 days, but stay could vary between 24 hours and, in one case, 11 months. This emphasizes the need for skill in the management of prolonged unconsciousness in this group of patients.

The number of deaths in the year was 121 out of a total of 657 patients admitted, i.e. a mortality rate of 18.4 per cent, which seems to compare tolerably well with other reported experience, but admission criteria may vary so widely that such comparisons are probably not valid.

MANAGEMENT

In the management of these patients there are always two main requirements: firstly, maintenance of satisfactory respiration and, secondly, maintenance of fluid balance and nutrition. Many other problems arise, but these two are constant and require an efficient routine for their successful management.

PREOPERATIVE PROBLEMS

As these patients are often admitted in a confused or unconscious state, we are often ignorant of their previous "normal" state, or the history of their illness. The diagnosis is often in doubt and the patient is frequently admitted in an unstable, fluctuating state of consciousness which may deteriorate due to a sudden rise in intracranial pressure, with resulting apnoea. It is therefore most important to ensure minute-to-minute observation of level of consciousness, adequacy of airway, respiration and blood pressure.

This is well illustrated in the management of head injury. This may prove fatal if mismanaged, when often quite simple measures will ensure the patient's survival. Such patients are often admitted deeply unconscious, areflexic and with partial

airway obstruction causing hypoxia, hypercarbia and raised central venous pressure; this may be aggravated by inhalation of secretions and blood, which may precipitate or accentuate increase in intracranial pressure. Progression to medullary coning may follow further respiratory depression, resulting in a dangerous vicious circle involving further increase in intracranial pressure.

Frequently the only treatment these patients require is establishment of a proper airway. Scalp lacerations will require debridement after appropriate local infiltration analgesia. Blood loss can be considerable from the head wound itself, but in addition serious blood loss may occur unnoticed elsewhere, at the site of other injuries of which the unconscious patient cannot complain. Sometimes the attendants are so anxious to get on with treating the head injuries, that other injuries (e.g. ruptured spleen) may be missed, with fatal outcome.

Another hazard is the ever-present danger from a full stomach. At the risk of seeming trite, the suggested drill in these cases should be:

- a. Establish airway.
- b. Pass stomach tube.
- c. Transfuse if necessary.
- d. Carry out complete physical examination.
- e. Specific measures.

It should always be borne in mind that immediate remedial measures may be instituted should signs of rapidly increasing intracranial pressure intervene, e.g. infusions of hypertonic solutions and the opening of the skull for evacuation of clot or tapping of the ventricles.

POSTOPERATIVE AND LONG TERM MANAGEMENT

Postoperative and long term management amounts simply to management of the unconscious patient, for which all anaesthetists should be well trained, but certain complications may occur, in some cases due to specific neurological lesions, or as a result of a prolonged period of increasing debility prior to admission and sometimes simply because of long duration of unconsciousness.

Respiratory care

The dangers of cerebral acidosis have been well emphasized by Alexander and Lassen¹ and to this end the importance of ensuring adequacy of cerebral oxygenation and CO₂ elimination need hardly be repeated, but some details should be emphasized:

(a) *Airway control.* It is often possible to manage patients at the lightest level of unconsciousness (i.e. those who respond vigorously to stimulation of upper airway by suction, or insertion of oropharyngeal airway by coughing, swallowing, biting and phonating) simply by posture in semi-prone position and moving the patient from one side to the other; but careful observation is essential, particularly in the immediate postoperative state, or immediately after admission when the level of unconsciousness is either unknown or fluctuating. Intermittent pharyngeal suction not only removes secretions, but also confirms that the level of consciousness is not deteriorating. The stomach should be kept empty by suction through a naso-gastric tube until the patient's level of consciousness has stabilized.

More deeply unconscious patients may require an oropharyngeal airway to maintain the airway even when semi-prone. The fact that the patient will tolerate an oropharyngeal airway or even pharyngeal suction, with little or no coughing, indicates that inhalation of secretions will almost certainly occur. Unless rapid improvement is anticipated, endotracheal intubation or tracheostomy is indicated. Until the airway is protected by a cuffed tube, the stomach should, of course, be kept empty by suction. If the patient will not tolerate an endotracheal tube, or if more than a few days' unconsciousness at this level is anticipated, a tracheostomy should be established. Endotracheal intubation is most frequently useful in patients with head injuries, as many of these can make a rapid recovery.

Profoundly unconscious patients (i.e. areflexic, showing no evidence of coughing or swallowing, and with stertorous respiration tending to obstruct) require endotracheal intubation initially and usually tracheostomy, except for the occasional patient who makes an unusually rapid recovery.

Tracheostomy may also be indicated when certain cranial nerve palsies have affected normal coughing and swallowing mechanisms (e.g. acoustic neuroma, posterior fossa and brainstem lesions). It is not intended to discuss tracheostomy management here, but it should be emphasized that this procedure should not be undertaken lightly, as there are many possible complications. The tracheobronchial tree nearly always becomes infected, and the problems resulting from cross-infection can be very serious unless strict isolation is possible.

(b) *Assisted or Controlled Ventilation.* Assisted or controlled ventilation may be indicated by obvious clinical signs of ventilatory inadequacy or by a deterioration in arterial blood gas values, which should be measured routinely in all but the patient who is obviously breathing adequately.

Controlled ventilation (IPPV) is most commonly required when apnoea occurs due to rising intracranial pressure, and it may only be required briefly until therapeutic measures have time to have their effect. If the condition is not amenable to and promptly treated by such measures, apnoea is almost certainly going to be permanent, and justification for prolonging IPPV under these conditions must be carefully considered.

Other less frequent causes of apnoea or respiratory insufficiency which may require assisted or controlled ventilation are:

1. Multiple injuries with crushed chest.
2. Posterior fossa lesions or surgical damage to the respiratory centre in the brainstem.
3. Following cervical cordotomy for intractable pain (especially if bilateral) – very rarely.
4. Rewarming recirculation, if induced hypothermia has been used during the operation. This is an occasional, but real possibility.

In addition, IPPV may be used therapeutically in patients with raised intracranial pressure (e.g. due to cerebral oedema associated with head injury), to assist in lowering blood flow and therefore intracranial pressure by inducing cerebral hypocarbia.^{2,3}

Patients with central nervous system damage who hyperventilate present a tricky problem. It has been shown that many of these patients have a low csf pH

which stimulates respiration and lowers Paco_2 .⁴ In addition, although Pao_2 may be normal, this is frequently very low also, probably due to pulmonary v/q imbalance. Whether Pao_2 is normal or low, these hyperventilating patients may become exhausted and suddenly die. For this reason it is advisable to institute controlled ventilation in all these patients, adding oxygen if necessary, to reduce the work of breathing and to lower Paco_2 further in an attempt to correct intracerebral acidosis.¹ Care should be taken, however, not to reduce Paco_2 below 20 Torr when anoxic changes can occur due to severe cerebral vasoconstriction.⁵ If a systemic metabolic acidosis is present, this should obviously be corrected. In addition to extra oxygen, these patients require an increased water and calorie intake.

Another instance where airway and respiration require special care is in the management of the patient in status epilepticus. In these patients convulsions are nearly always controlled satisfactorily with a thiopentone⁶ or diazepam⁷ intravenous drip, without necessarily rendering the patient unconscious. Our clinical impression is that thiopentone is the more efficacious in the resistant case. Cases not controlled by thiopentone may occasionally require to be curarized and ventilated until the cause of the convulsions has been removed or an adequate level of long-acting anti-convulsant drug builds up in the tissues. Often all that is required is to break the vicious circle involving hypoxia – convulsion – hypoxia.

CARDIOVASCULAR PROBLEMS

Postoperative hypotension is a frequently occurring cardiovascular problem. A combination of low systemic blood pressure and high intracranial pressure can result in a low cerebral arterial perfusion pressure with possible occurrence of cellular hypoxia.

There are many possible causes for arterial hypotension in the neurosurgical patient, and the most relevant are listed in Table II.

TABLE II
HYPOTENSION AND NEUROSURGERY

Inadequate transfusion
Induced hypotension
Induced hypothermia
Cerebral decompression
Hypothalamic lesions
Pituitary failure
Vasomotor centre lesion
Cervical cordotomy
Dehydration
Electrolyte imbalance
Curling's ulcer

Hypovolaemia is avoided by accurate measurement and replacement of operative blood loss, and by central venous pressure manometry. Hypotension due to dehydration and electrolyte imbalance should respond readily to routine measures. Operative drug-induced hypotension may rarely cause post-operative hypotension when the longer acting ganglioplegics have been used, and should be managed by posture and the use of a peripherally-acting vasopressor if necessary. Incidentally,

as these patients will have fixed dilated pupils, the intensive care staff should be advised when ganglioplegic drugs such as trimetaphan have been used. Hypotension following hypothermia and rewarming may be due to flooding of the circulation with acid metabolites from the periphery during rewarming, resulting in myocardial depression or peripheral vasomotor failure, or both.⁸ This syndrome is best avoided (1) by encouraging systemic vasodilatation during operation, (2) by not unduly prolonging hypothermia below 30°C, and (3) by slow rewarming.

Pathological processes, or surgical interference with the vasomotor centre in the brainstem, or of the descending vasomotor efferent fibres during bilateral cervical cordotomy may cause hypotension due to loss of peripheral vascular tone. Conversely, systemic hypertension associated with medullary coning and possibly due to partial ischaemia of the vasomotor centre may cause compensatory vasomotor hyperactivity, and if sudden cerebral decompression occurs, there may be a precipitous fall in systemic blood pressure. These hypotensive episodes should be adequately dealt with by posture, and judicious use of vasomotor drugs.

Adrenocortical failure may occur, not only following pituitary ablation, but may result post-operatively because of inadequate "cover" in patients previously or currently under treatment with steroids, which are frequently used in the treatment of patients with threatened or established elevation of intracranial pressure. Hypotension may occur very suddenly following pituitary ablation; possibly hypothalamic vasomotor function is deranged during hypophysectomy. Whatever the precise mechanism, "cover" with large intravenous doses of hydrocortisone should be ensured and posture, peripherally-acting vasopressors, and plasma volume expanders may be used. In this context, the possibility of acute gastro-intestinal ulceration (Cushing's Ulcer, Anterior Hypothalamic Syndrome⁹) with perforation or intestinal haemorrhage must not be overlooked as a cause of unexplained shock in these patients.

Neurosurgical patients with persistent hypotension, often accompanied by spontaneous, irreversible hypothermia, have probably sustained irreversible hypothalamic damage, and the prognosis is very poor indeed.

The existence of an association between acute cerebral damage and a specific form of myocardial damage described as a focal myocytolysis with a variety of arrhythmias and other ECG changes has been shown by Connor,¹⁰ and this may present a diagnostic and therapeutic problem. Connor suggests that presence of such myocardial lesions may explain sudden death in patients with severe CNS damage and apparently intact cardiorespiratory systems. He also suggests that as massive vagal overactivity combined with release of catecholamines may be the cause of the syndrome, treatment with atropine and possibly propranolol may be indicated.

FLUID AND ELECTROLYTE BALANCE

With adequate gastric tube feeding, this should not be a problem. If an intravenous regimen is required for a short time, there should be no difficulty when therapy commences with the patient in a stable state. If the fluid and electrolyte status are not stable at the beginning of therapy, establishing satisfactory balance may be difficult. Neurosurgical patients are frequently admitted as emergencies,

undiagnosed, with no history regarding fluid intake or loss, often apparently dehydrated, oliguric or anuric. They may have in fact undergone, over a number of consecutive days, a series of investigations for which oral feeding is withheld and it may be difficult and time-consuming to establish whether anuria is due to dehydration or renal damage. Estimation of urine/plasma osmolality and urine/plasma urea ratios may be helpful.¹¹ Treatment of increased intracranial pressure with osmotic dehydrating agents may also cause considerable changes in tissue and plasma electrolyte levels.

In the post-operative phase, bizarre fluctuations of electrolyte levels occur in many of these patients. Rightly or wrongly these changes are usually attributed to hypothalamic dysfunction. At one end of the scale we may find the patient water logged and with dilutional hyponatraemia. With or without a concomitant rise in intracranial pressure, such a patient may develop symptoms simulating postoperative intracranial haemorrhage or infarction, i.e. confusion, semi-coma etc. This picture may be attributable to a manifestation of the post-traumatic response to injury, to a hypothalamic lesion or hypophysectomy, or due simply to excessive intravenous administration of fluids. The routine use of intravenous 5 per cent dextrose postoperatively should be discouraged and mild restriction of water administration during the first three days after surgery is probably a good policy.

At the other end of the scale, and less common, is the "hypertonicity" syndrome, which may also be the result of a hypothalamic lesion (i.e. diabetes insipidus) or due to administration of salt-retaining adrenal hormones, or simply due to fluid deprivation. Over a 6-month period at Killearn, 27 (4 per cent) patients exhibited abnormalities of fluid and electrolyte balance, of sufficient extent to be reflected in a fluctuation or serum electrolyte levels outside the accepted range of normality. Of these, the vast majority (22) were combined hyponatraemia and hypochloraemia.

With careful charting of clinical state, weight, fluid intake and output, and measurements of urine specific gravity and electrolyte levels, urine/plasma osmolality and urea ratios, haematocrit and blood electrolyte levels, fluctuations in fluid and electrolyte balance can usually be restored to a stable state in these patients.

CONTROL OF CSF PRESSURE

Control of cerebrospinal fluid pressure is primarily a surgical problem, but much can be done in the intensive care unit to prevent unnecessary rise in intracranial pressure, by rational positioning of the patient, avoidance of unnecessary coughing and straining, prevention of hypercarbia or hypoxia by close surveillance of respiration, and cautious administration of intravenous fluids, to avoid overhydration.

Currently, 20 per cent mannitol intravenously is widely accepted as the hypertonic dehydrating agent of choice for short term management of acute increase in intracranial pressure. Dexamethazone is also used in many patients for longer term management of patients with increased intracranial pressure.¹²

Increasing use of continuous ventricular fluid pressure monitoring in recent years has facilitated the early detection and management of rising intracranial pressure – particularly in patients with head injuries, where monitoring may be usefully continued for several days.³

NUTRITION

Nutrition is too often neglected in the unconscious patient. The postoperative neurosurgical patient who may be unconscious or unable to feed normally for some considerable time, is particularly vulnerable. Infusion of intravenous crystalloids can, of course, be only a temporary expedient to maintain fluid and electrolyte equilibrium until proper nutrition can be instituted. In the absence of an adequate source of calories and amino-acids, the patient will rapidly pass into negative nitrogen balance. For this reason, in the absence of contraindications, naso-gastric tube feeding is established as soon as possible. Contraindications to tube feeding are: (1) Unprotected airway – in these patients the tracheobronchial tree must be protected against inhalation either by an active cough reflex, or by a cuffed endotracheal or tracheostomy tube; (2) A fluctuating level of consciousness, or threat of fluctuation in the level of consciousness, as for example in the immediate postoperative period, or in the acute head injury; (3) Absence of gastric absorption – this usually occurs anyway during the first 24–48 hours postoperatively, and in the presence of any acute illness, fever, infection, shock, etc. The possibility of enteritis must not be forgotten as a cause of nonabsorption, also.

INTRAVENOUS NUTRITION

Tube feeding is frequently not possible due to one or more of the above contraindications. If this state of affairs persists for more than 48 hours, intravenous nutrition must be established. The main problem here is concentration of sufficient calories and protein-building substances into solution in the patient's daily allowance of fluid – as it is only too easy to overload these patients with water. One of the proprietary preparations of protein hydrolysate or synthesized amino-acids may be used, which usually also contain a useful quantity of carbohydrate in the form of glucose or fructose. In addition the proprietary fat-emulsion preparations are very useful and compact source of calories, and may be used with the amino-acids preparations. It is nearly always necessary to add some potassium to the solutions and parenteral vitamins must be administered as well. The hyperventilating or pyrexial patient will, of course, require extra calories and water, as indicated earlier.

TEMPERATURE CONTROL

Temperature controlling centres are located in the hypothalamus, and pathological or surgical lesions may cause either elevation or depression of body temperature.

It is generally agreed that the hyperpyrexial patient should, if possible, be actively cooled to a temperature as near normal as possible. This will include many patients with head injuries. Some authorities believe that patients with head injuries benefit from induced hypothermia, and the use of this technique seems to be gaining favour. This remains controversial, and presently the most common practice seems to be prevention of pyrexia without lowering body temperature below normal. Whether to actively rewarm the patient who has been deliberately cooled for surgery, also remains controversial. Most authorities seem to practice prompt and active rewarming after surgery, with careful avoidance of shivering, on rationale

that shivering will produce increased oxygen uptake of muscle and brain. That oxygen uptake of muscle should be increased is obvious, but it is a little more difficult to understand why oxygen uptake of brain should be increased (if this does indeed occur). Increased oxygen uptake in shivering muscles will cause increased desaturation of mixed venous blood, which may not be accompanied by an appropriate increase in cardiac output¹³ and the now well documented post-anaesthetic arterial desaturation will be exaggerated and possibly accompanied by a metabolic acidosis. Whether this mechanism actually occurs or not, any risk of hypoxaemia or acidosis in these patients is, of course, to be deplored. The relatively small number of postoperative blood gas observations we have obtained in shivering patients do not bear out this gloomy picture, but this should obviously be the subject of a carefully controlled investigation.

There is a good argument for prolonging hypothermia into the post-operative period, possibly for days, to minimize damage resulting from cerebral oedema or infarction. This can be technically tedious in practice and very demanding upon nursing faculties, but is by no means impossible and may be very desirable. In the meantime, whilst these problems remain controversial, it is generally our practice to actively rewarm patients immediately after induced hypothermia.

Occasionally a patient admitted after a severe head injury remains in a persistent hypothermic state, or fails to rewarm after induced hypothermia. This is a sign of serious hypothalamic damage, and is of grave prognostic significance in any patient with intracranial pathology, especially when combined with persistent hypotension. Over a 6-month period, 29 out of a total of 333 patients (8.7 per cent) exhibited a picture of persistent hypothermia ($<35^{\circ}\text{C}$) accompanied by hypotension (<100 mm Hg systolic) and 21 of these were also apnoeic. All except one of these patients subsequently died. The one survivor was not apnoeic at any time.

MONITORING

As in any intensive care unit, efficient and comprehensive monitoring of vital functions are essential, and two schools of thought prevail – The “all-automated, centralized, minimum-nursing-personnel” school, and the “two nurses per patient, 24 hours” school. This is not the place to take sides in such a controversy, save to point out that pre- and postoperative neurosurgical patients may very suddenly and quietly become apnoeic due to insidious rise of intracranial pressure, and as yet no 100 per cent reliable respiration monitor has been devised; nor has there yet been invented an instrument that will signal the sudden appearance of dilated, fixed pupils. The writer would personally prefer to be monitored by a reasonably intelligent nurse, full time. This of course makes huge demands on nursing manpower. It is essential to classify patients into groups of varying degrees of risk, and to distribute available nursing staff and monitoring facilities to best advantage, always bearing in mind the limitations of the mechanical system.

A thoracic impedance plethysmograph¹⁴ may be helpful, not only in warning nursing staff of diminishing respiratory amplitude, but also in providing a spiograph showing respiratory patterns. The instrument is foolproof as regards power failure or detachment of chest leads – the latter being very important in the restless neurological patient. In its present form, the instrument does not always give

warning of respiratory inadequacy due to early airway obstruction and is, therefore, of most use in the intubated patient. The manufacturers consider that the instrument can quite easily be modified to warn of respiratory inadequacy due to obstruction, and it is hoped that this will become available.

Continuous monitoring of ventricular fluid pressure (VFP) by means of a catheter placed in the subdural or intraventricular space through a burr-hole has been developed in recent years, particularly by Lundberg³ who describes various waveforms associated with raised intracranial pressure. Solid state transducers placed on the dura or under the skin over a burr-hole are also being investigated. VFP monitoring can give early warning of the development of cerebral compression and one can observe the effectiveness or otherwise of such measures as controlled hyperventilation and osmotic dehydrating agents in lowering intracranial pressure. Another useful observation may be changes in pressure caused by use of drugs such as the narcotics.

In many patients continuous ECG monitoring will be desirable, especially in view of the risk of myocardial lesions described earlier. In some patients, such as those with head injury and unknown blood loss, and following prolonged surgery involving a steady blood loss where measurement is difficult because of frequent irrigation, central venous manometry is most helpful.

SEDATION

Many of these patients are confused, restless, and difficult to control. The temptation to administer potent depressing drugs to facilitate nursing must be resisted. These drugs not only mask signs of hypoxia and the onset of intracranial trouble, but may actually cause sufficient respiratory depression to precipitate a progressive rise in intracranial pressure in patients who are marginally compensating for a rise in pressure due to a space-occupying lesion.

It is wise policy to restrict sedation to aspirin and codeine phosphate in judicious dosage; restless patients should be restrained by use of padded cot sides, with a well secured overhanging net if necessary. In some cases it may be necessary to administer more potent analgesics as when using hypothermia in treatment of head injury; in which case the drugs should be given only in small doses intravenously and when ventricular fluid pressure is being monitored. It may also be necessary to administer these drugs in combined head and chest injuries when the patient is being ventilated. Incidentally, whereas in the conscious patient adaptation to the ventilator occurs, in these head injuries it is continually necessary to be giving more drugs, and the problem of tolerance may arise.

PHYSIOTHERAPY

Physiotherapy is of tremendous importance, not only in preventing or treating pulmonary complications in the patient who may require intubation or IPPV for months, but also in preventing the onset of flexion contractures of the limbs and bedsores in patients with acute neurological deficits.

BLADDER CONTROL

It is obviously essential that bladder infection be avoided if possible in these

patients. Not only can this lead to destructive renal disease, but the antibiotic-resistant bacteria flourishing in the renal tract seem to have a remarkable knack for finding their way up to the respiratory tract. In addition it is important in the prevention of bedsores that beds be kept dry.

A system using condom and closed drainage in males, and catheterization with closed drainage into totally disposable sterile closed plastic bags in females, should be routine. In spite of this, it remains very difficult to keep bladder infection rate down, particularly in females and when, in the latter case, urinary tract infection does become established, as well as using the appropriate antibiotic, a closed bladder irrigation system using a urinary antiseptic in the irrigating solution, may be helpful.

CROSS-INFECTION

In any intensive care unit cross-infection is difficult to control. In neurosurgical practice where coma is sometimes prolonged, the hazard is particularly great, and is an ever present problem.

Antibiotics should not be used routinely to provide "cover", but should only be administered where a specific indication exists. Indiscriminate use of antibiotics undoubtedly causes widespread and rapid appearance of resistant strains in any intensive care unit.

Emphasis should be upon spacing of beds, liberal use of disposable equipment, and scrupulous sterilization of pulmonary ventilators and humidifiers. Segregation of infected cases and barrier nursing should be employed as much as possible.

POSTOPERATIVE COMA

Prolonged postoperative coma is bound to occur in a certain percentage of neurosurgical patients, the commonest causes probably being recurring haemorrhage producing subdural haematoma, cerebral infarction possibly due to vascular spasm, and cerebral oedema causing midbrain distortion.

Of the less common causes which may be overlooked, hyponatraemia, adrenocortical failure, and myxoedema should be mentioned.

An occasional patient fails to return to full consciousness following cerebral angiography and air encephalography. The reasons for this are not always clear. In many cases coma will be due to increase in intracranial pressure. In the case of air encephalography, it is possible that an excessive amount of cold gas or air has been injected into the subarachnoid space, with subsequent warming and expansion of the air producing a rise in intracranial pressure resulting in medullary "coning." Use of nitrous oxide as a general anaesthetic for angiography or air encephalography may exaggerate this.¹⁵ If general anaesthesia using a volatile agent or involving any degree of respiratory depression has been employed for cerebral angiography, increased cerebral blood flow may account for rising intracranial pressure and "coning" when a space occupying lesion is present.¹⁶ In any event, it is important that patients should always be carefully observed following these radiological investigations, so that should prompt recovery from anaesthesia fail to occur, or deepening of conscious level or other sign of rising intracranial pressure be observed, steps to decompress the brain may be promptly instituted.

MANAGEMENT OF SPECIFIC NEUROLOGICAL LESIONS

Aspects in the care of many specific neurosurgical conditions have already been mentioned, but there remain some conditions which occasionally give rise to difficulty and which should be mentioned specifically.

1. During or following posterior fossa exploration, damage to the medullary respiratory centre may cause persistent postoperative apnoea, or apnoea may suddenly occur postoperatively due to infarction, haemorrhage or oedema in the operative site.

2. Following cervical cord exploration or cervical cordotomy for intractable pain, there may be postoperative respiratory inadequacy if some of the phrenic nerve roots have been sectioned, especially if bilateral tractotomy has been performed. It has already been mentioned that simultaneous section of descending sympathetic efferent fibres in this area may cause hypotension.

3. Excision of an acoustic neuroma may involve dissection in the region of cranial nerves 7, 9, 10, or 11 with frequent occurrence of facial nerve palsy and less frequently, but more seriously, pharyngeal or laryngeal palsies with anaesthesia, which result in postoperative difficulty in swallowing or coughing, and may necessitate establishment of a permanent tracheostomy in some cases.

ETHICAL PROBLEMS

Unavoidably, a large neurosurgical unit will accumulate a small but persistent number of patients who, if not permanently "unconscious" are completely incapacitated by severe cerebral damage, and arranging long-term care for these patients can put a serious strain on social workers' and almoners' facilities.¹⁷ The need for establishment of special units where these patients can be cared for is urgent. There is a tendency for these patients to be discharged from the intensive care unit to small peripheral or cottage-type hospitals for after-care, and after a disconcertingly short interval, it is heard that the patient has died, having been kept alive and possibly improving for months in the neurosurgical unit.

An even more acute problem is that of the patient who has sustained very serious cerebral damage, who is apparently decerebrate, whose cardiovascular system is intact and whose respiration is maintained by a mechanical ventilator. Defining cerebral "death" and making the decision to discontinue artificial respiration is not easy, and in addition to careful clinical assessment, appraisal should include discussion between the neurosurgeon or neurologist in charge of the patient, the anaesthetist caring for the patient's vital functions, and the relatives. It is quite commonly believed that if a patient is still unconscious after 4 weeks, the chances of recovery are almost nil. In fact, at Killearn, over a period of one year, of 31 patients who had been unconscious for more than 4 weeks in this unit, 16 eventually recovered consciousness, at least sufficiently to be fed orally.

NURSING STAFF

This type of work makes great demands on nursing staff, both in the number of nurses required per patient, and in the standard of nursing, for which special training is required. The state of the patient must be carefully discussed with the nursing

staff, and explicit instructions given with regard to management, but leaving room for intelligent discretion on the part of the nursing staff in the immediate care of the patient.

Nursing staff should be provided with good equipment and be exhorted to observe and record assiduously. They must be encouraged and congratulated on every successful step achieved towards the patient's recovery. It is only with the help of such enthusiastic and persistent attention to details of observation and treatment, that many of these very ill patients may be returned to a useful life.

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