THE MANAGEMENT OF THE UNCONSCIOUS HEAD-INJURY PATIENT

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[PART I]

It is unfortunately true that a patient with a severe head injury, and in deep coma, presents a distressing clinical picture to the doctor. This feeling of hopelessness, helplessness and disaster is caused by the frequent fatal outcome. To him, as well as to the lay mind, a profound brain injury spells doom to the patient, and death is shrugged off as unavoidable. Unfortunately this is too frequently the outcome. The brain-complex, however, from its known and unknown characteristics, is endowed with a remarkable resilience and potentiality for recovery in sublethal injuries so that, when the full acumen of modern medical science is energetically applied, a satisfactory therapeutic response may result.

The doctor is perhaps negatively conditioned during his training in the wards and in the medico-legal laboratories, where the high percentage of fatal head injuries accentuates their mortal nature. During his internship he may also lose touch with the actual management of these patients, because they are often referred to special units. This is unfortunate for it is essential that every doctor becomes conversant especially with the early management, which is often crucial to the survival of the patient. The total care and management requires specialized experience, which is often not available to those who are suddenly presented with such a problem. It is my purpose to indicate the details of the modern approach and the therapeutic means, which, if applied as surgical intervention, can save many of the increasing numbers of casualties, suffering from head injury and unconsciousness.

Cook, in a recent editorial on cranio-cerebral injuries,* states: 'Daily experience indicates how, over periods of months or even years, remarkable recovery can ensue in patients with so-called severe brain injury, if they are carried through the acute phases of the injury and subsequently intercurrent infection or pulmonary dysfunction does not develop.

'Most certainly increased interest and attention to the various systemic pulmonary and metabolic alterations in patients with severe cranio-cerebral injury will repay the surgeon and the patient with a hope of survival far greater than that which could be anticipated in the past.'

The unconsciousness caused by a head injury can conveniently be divided into three categories:

1. Concussion.
2. Short-term coma.
3. Prolonged coma.

CONCUSSION

In this event the duration of unconsciousness is short—from a fraction of a second to not longer than half an hour. Concussison is not necessarily associated with a benign lesion, and deaths have occurred in which autopsy showed no lesions detectable microscopically. This, however, is rare and, in clinical jargon, concussion denotes a temporary loss of consciousness following the application of force to the skull and its contents. The mechanism leading to this physiological change has not yet been fully elucidated[5, 6, 24]—currently the theory attributing it to inhibition of the reticular activating formation in the brain-stem, which is credited with activating or alerting the cerebral cortex along defined pathways, is favoured. In concussion *per se* the sudden application of force to the skull causes an interference or inhibition or physiological interruption in the conducting mechanism by stress or torsional strain at the mid-brain level, and hence a failure of the alerting mechanism. The patient thus reverts to the so-called sleep state.

Nature of Interrupting Mechanism

It is important to bear in mind the nature of the interrupting mechanism. In pure concussion it is, in effect, a conduction or apraxia phenomenon and usually results in complete recovery. Should the stress or torsional mechanism, however, be so severe that it subjects the tissues (nerve tracts and vessels) to trauma beyond the limits of elasticity or plasticity, actual break in continuity will occur, with all the secondary manifestations of haemorrhage, oedema, swelling, venous stasis and tissue and parenchymal damage. In this event the outlook takes on a different and grimmer outlook. No longer can we expect the rapid recovery of function, characteristic of the concussive state. Physiological recovery now depends on pathological resolution which, in turn, conditions anatomical reconstitution. Brain can only recover from submicroscopical or conductive lesions or from the effects of the compression of oedema or pathological fluid collections (here we are referring to traumatic states only). The value of surgery in such instances is so limited that it plays a negligible part. Adjuvant measures to hasten and encourage such resolution are indeed available, but again, of limited use. Natural resolution, aided therapeutically, is the most that can be expected. The time factor for this process varies naturally and may take days, weeks, months or even years.

Thus, while the pathological effects are still active and natural resolution awaited, the patient may remain in a state of continued, impaired consciousness. It is incumbent on us to maintain life until the limits of resolution have been attained.

SHORT-TERM COMA

This is a state in which unconsciousness is prolonged beyond a few seconds or minutes, i.e. from $\frac{1}{2}$ to ± 12 hours. Here, the intrinsic cerebral damage can be recognized microscopically or macroscopically and results in a more serious disturbance of cerebral function—not only of the 'centre' of consciousness, whether in the brain-stem or cerebral cortex, but also of vital and other processes. The patient will come out of coma when the normal, natural conductive mechanisms reassert themselves. He passes from deep coma, successively through all the phases of impaired consciousness, with awareness of environmental stimuli, both internal and external—possibly interposed with periods of restlessness and irritability—finally to resolution, lucidity and full or partial recovery.

It is now that the effects of our therapeutic measures can be properly judged.

Observation

In this phase of short-term coma the patient must particularly be observed as he passes from a primary condition of semi-coma to coma or awaits spontaneous recovery of conscious and vital functions. This 12-hour period can indeed be the most trying part of the medical attendants' supervision of the head-injured patient.

In this 12-hour period possibly three mechanisms are in force to account for the continued unconsciousness—not only the initial short phase of concussion, with nerve cell and tract dysfunction, but also actual organic damage to these structures.

*Surg. Gynec. Obstet., March 1964.

In addition there may be the development of a space-occupying, compressional lesion superimposing itself gradually as the mechanism now producing the unconsciousness. This latter aspect is of paramount importance to the surgeon at this stage, since it is obvious that the prognosis may be seriously jeopardized if this compressional lesion is not removed surgically.

Surgery Indicated

Many state that, excluding the extradural haemorrhages, surgery in the first 12 hours gives such poor results that it is not worth while. It is true that the effects of the primary brain damage (concussion, contusion, laceration) are of over-riding importance, but there is absolutely no doubt that the prompt and efficient removal of intracranial collections of blood may favourably tilt the factors towards recovery. It is not my purpose to detail this aspect of the management of the unconscious head-injury patient. It will be assumed that all adequate investigations and surgical treatments have been effected—but that the patient still remains in deep coma. However, from time to time the acutely-expanding, intracranial lesion requiring urgent surgical evacuation is encountered.

A patient arriving in semi-coma, but rapidly passing into deep coma, an unconscious patient with equal and reacting pupils in whom one pupil is rapidly enlarging, or one in whom the pulse rate slows to 50 per minute or less, is a candidate for immediate surgery, or preparation for surgery and not for watchful waiting.

PROLONGED COMA

Here the intrinsic damage—disruption of coordinating tracts by oedema and/or haemorrhage—is such that the natural processes of recovery do not result in the return of cerebration. Such primary injury results in the so-called 'vegetative' states and forms of coma, from which recovery is uncommon. In the early phase it is difficult to distinguish between conduction and organic interruption so that 'time only will tell'.

However, the patient must not be allowed to die while awaiting the slow and often prolonged process of recovery.

This is the crux of the therapeutic principle of the management of this type of injury—a certain number of patients in prolonged coma may and do recover function in whole or in part indicating that surgical knowledge and equipment must be applied to make this a possibility. The most hopeless case may, surprisingly, halt its deterioration and recover. In head injuries the dictum 'while there is life there is hope' has a profound application—although the surgeon must be prepared for more disappointments than successes.

MANAGEMENT

Emergency measures for the immediate maintenance of life obviously take precedence to all others.[2, 7, 13, 14, 17, 19] After these are effected, a base-line neurological examination is carried out (Summary I). It must be fully realized at this stage—which may be within the first half hour of injury—that the duration of the coma is entirely conjectural. Different patients, presenting identically, may show signs of recovery while being initially examined or only after hours, days, weeks or months later, or never. The same intensive approach must therefore be adopted.

SUMMARY I. MANAGEMENT OF A COMATOSE PATIENT

Attention should be paid to the following:

Aeration and oxygenation
Shock
Record of level of consciousness—'vital signs'
The irritable violent patient
Hypothermia
Dehydrating agents
Feeding and metabolic care
Tracheostomy
The tracheo-bronchial toilet
The inspired air
Physiotherapy
Care of the bladder
Care of the bowels
The use of drugs

It must be determined whether: (1) The patient suffers purely from a head injury, or (2) Whether there are associated serious injuries, which may require immediate supervision or operation (e.g. active haemorrhage, ruptured abdominal viscus, gross compound fractures of the limbs). The recognition and care of these injuries are not under discussion in this article and have recently been adequately stressed and described.[26-28] We are concerned entirely with the cranio-cerebral aspect of head injuries.

Injuries which do not threaten life should not immediately be treated by surgery in the unconscious patient. (e.g. Colles', Pott's fractures, foreign bodies, etc.). A few days' delay will not materially affect the result.

Modern anaesthesia, well-conducted and controlled, not only may do *no* harm to the patient but may well improve the patient's milieu while under the anaesthetic. However, the ideal state does not always exist and, generally speaking, there is an added metabolic 'insult' which may very well be harmful, apart from the histotoxic effects of the drugs *per se*.

The post-anaesthetic phase likewise may be fraught with sufficient added risk and complications which may move the pendulum away from the patient's recovery. Apart from the actual anaesthesia the various manipulations required may also engender physiological and metabolic disturbances, the summation of which may impede the natural recovery, which is the object of the total care of the head-injury patient.

Aeration and Oxygenation

A clear airway, important as it is, is not sufficient. The free way for the air having been provided and maintained, it is incumbent to provide maximal utilization of the air presented to the lungs. In effect, this means that all fluid and accumulations must be prevented from entering the broncho-alveolar tree. Forcible aspiration of saliva, mucus, blood, vomitus, alcohol, cerebrospinal fluid and, unfortunately not infrequently, of injudiciously administered fluids by bystanders or 'first-aiders' can not only nullify the provision of an 'adequate' airway, but rapidly contribute to the death of the patient.

Dentures are immediately removed from the patient's mouth and the patient is placed in the coma position: The patient lies on his side—whichever is most convenient for supervision—in the semi-prone position, the lower leg (i.e. resting on the bed) with the knee extended and the upper leg flexed in order to prevent the unconscious patient from rolling over completely. The chin is directed towards the bed (or stretcher) while the head is supported by a small pillow or blanket. The foot of the bed is elevated 9 - 12 inches. This position allows the tongue to fall forward (especially if there is concomitant fracture of the mandible) and the relatively free escape of blood etc. from the nose and oropharynx out of the corner of the mouth.

A Davis' mouth gag is immediately inserted into the mouth and in the absence of suction, the mouth is continuously mopped and swabbed until relatively dry. If suction is available, it is used to keep the oropharynx as free of fluid as possible. It may be necessary, in suspected inhalation, to elevate the foot of the bed up to 2 feet in order to allow adequate gravitation of fluid to the exterior. This position, in the acute head injury, must not be maintained beyond the minimum period necessary, since the venous engorgement produced may be deleterious and, theoretically, promote further intracranial bleeding. A suitable rubber or metal airway is introduced into the mouth and suction carried out around and through it.

Laryngeal intubation should be resorted to if this procedure is inadequate, i.e. if the patient still labours with respiratory difficulty owing to obstruction further down. This, in a patient bleeding from the nasopharynx and vomiting through the oropharynx, in acute respiratory distress, can be a trying and arduous procedure but should be rapidly accomplished since the patient can drown in his own fluids.

Tracheostomy must immediately be resorted to if intubation encounters unexpected difficulties, or a suitable tube is not immediately available. In the immediate phase of commencement of treatment the 'maintenance of an open airway' is of paramount importance. Carbon dioxide accumulation increases intracranial tension and cerebral oedema and, most important, completely nullifies the beneficial effects of hypothermia (see below). Brain tissue, especially the traumatized areas around the lesion, is extremely sensitive to lack of oxygen, and prolonged anoxia can readily convert a reversible to an irreversible state. By the judicious use of suction, airways, intubation, tracheostomy and oxygen administration, this can to some extent, be averted.

The alcoholic patient with a head injury presents a serious problem. On no account should a diagnosis of drunkenness be made in a head injury, despite 'strong' evidence of odour, vomitus, etc., until the presence of intracranial injury has been ruled out, i.e. treat such cases as serious injuries until proved otherwise. Gastric lavage, when carefully carried out, can be of considerable and immediate benefit.

Shock

Having attended to the prime requirements of aeration and oxygenation, attention is directed consecutively or concomitantly to the possible presence of the shock syndrome. If it is present look for the causes of it—head injury, *per se*, unless in the decompensated phase, is not characterized by it. A frequent cause of this syndrome is from blood loss from scalp wounds, which can be considerable. Witness the excessive blood loss that may accompany uncontrolled excision of scalp wounds. Should shock be present, irrespective of the cause, it is absolutely mandatory to resort to immediate blood, plasma, dextran or emergency fluid replacement, since hypovolaemic shock, by increasing the anoxia of the brain, may cause rapid deterioration of the cerebral state.

The severely shocked patient must be suspected to have other injuries in addition to those sustained intracranially. While treating shock these associated shock-producing lesions must be immediately attended to as well, e.g. immobilization of fractured limbs, traction of fractured femora, treatment of flail chest and its associated paradoxical respiration (here immediate tracheostomy and the application of intermittent positive pressure can indeed be life-saving and supersedes all other forms of treatment of this condition, if available). A ruptured viscus (liver, spleen, bowel, aorta) may well be the cause of shock which fails to respond to intensive routine care.

Preparation of the patient. In the comatose patient in whom such a lesion is suspected the procedure recommended is as follows: The head of the patient is shaved, the abdomen prepared, a cerebral angiogram[3, 32] carried out and the patient taken to theatre where, once the blood pressure has been brought up to the 'surgical' level of 100 mm. Hg systolic pressure, the patient is explored. Concomitantly, should the angiogram be positive, intracranial exploration is carried out as well. It will be clear that, because of the urgency of the condition, multiple, synchronous procedures are carried out by separate teams. It is only in this way that the patient with severe, multiple injuries requiring emergency surgery in their own right, can possibly be saved.

Radiology. A word of warning must be sounded about unnecessary radiological examinations. The prevalent practice of subjecting the unconscious or violently restless patient to a full, skull-routine must be condemned. The emergency X-rays required (bed-side unit)[5] are a right and left lateral (to exclude a fracture-line crossing a middle meningeal vessel marking) and a lateral X-ray of the neck, preferably in flexion, to exclude a concomitant cervical spine injury. Extracranial examinations must, of course, be carried out where indicated.

RECORD OF LEVEL OF CONSCIOUSNESS AND 'VITAL SIGNS'

This is charted at $\frac{1}{2}$ - 1-hourly periods, and in rapidly deteriorating patients even at 15-minute intervals. The progress is indicated on a chart depicted in Fig. 1, and used in the Professsional Unit of the Johannesburg General Hospital. The following classification of the levels of disturbance of consciousness is found to be the most practical:[25]

1 Coma—A complete lack of response to painful stimulation of any kind, e.g. pinching of the skin, supra-orbital pressure, pressure below and behind the angle of the jaw, pressure on the testicles.

2 Semi-coma—Grimacing movements and attempts to push the hand away in response to painful, or even normal stimuli. Swallowing movements are present, the patient wets the bed and the corneal reflex is present.

3 Severe confusion—The patient makes an attempt to obey commands, e.g. 'open your eyes, put out your tongue, take my hand'.

4 Moderate confusion—The patient answers simple questions, e.g. 'What is your work?,' etc. The patient is out of touch with his surroundings.

5 Mild confusion—The patient is capable of carrying on a conversation, but is unable to concentrate for long.

6 Fully conscious.

Charting the level of consciousness: This charting is more important than the simultaneous recording of the changes, if any, (on separate charts) in the so-called 'vital

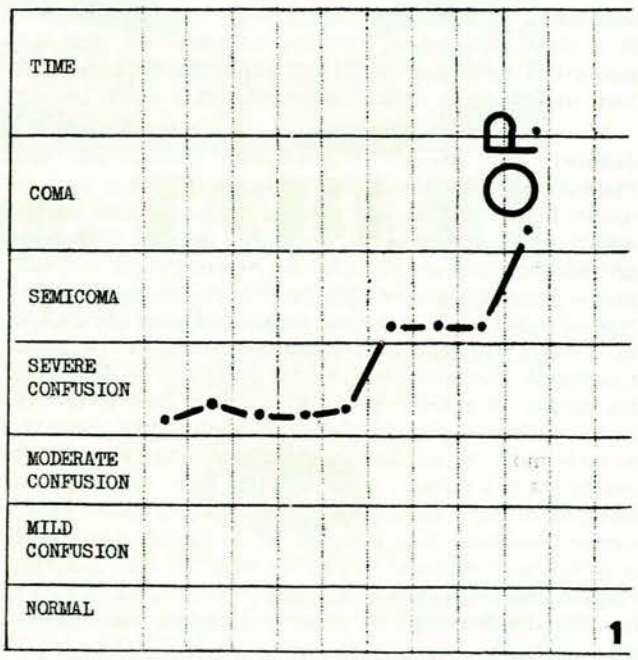

Fig. 1. Record of level of consciousness.

signs', i.e. pulse, blood pressure, temperature and respiration (Fig. 1). It is the most important part of what our American colleagues aptly describe as 'personalized unremitting care'. Classically, in compensated cerebral com-

pression the pulse-rate slows to 60 per minute or even less, the temperature rises to 101 - 103°F, the respiration slows down to the twelves and the systolic blood pressure rises to 140 - 160 mm. Hg resulting in an increased pulse-pressure of 50 - 70 mm. Hg—all part of a homeostatic mechanism to maintain the oxygenation of the vital centres (cardiac, vasomotor, respiratory). *It is my opinion, from the observation of hundreds of cases of serious head injuries, that these 'vital signs' are of no significant value in* 95% *of cases.* If you are watching the patient you must watch the state of consciousness or else your watchful inactivity will lead the patient to the mortuary instead of the operating table.

The experienced observer (whether surgeon or nurse) will readily detect signs of deterioration of the conscious state —even the comatose patient will manifest changes in increasing flaccidity of all the limbs, increase in stertor of breathing and absence of even a flickering response to painful stimulation. The nurse is instructed to elicit the signs every hour, i.e. to try to 'wake' the patient since there may be considerable overlap of the unconscious phase of primary cerebral injury by the superimposed coma of cerebral compression. The true lucid interval is, unfortunately, with the violence associated with the usual impact, all too frequently absent.

THE IRRITABLE, VIOLENT PATIENT

Although not a phase of disturbed consciousness irritability is closely associated with all levels except that of deep coma. Its management can tax those in charge of the case to their extreme limits. As a rule, violence associated with severe confusion denotes a favourable prognosis, but a number of patients with head injuries will die after being in a state of 'cerebral irritation' continuously, and lapse into coma only shortly before death. Restlessness, therefore, may at times indicate increasing intracranial pressure.

Restraining violence by violence must be strongly condemned. Beds should be fitted with padded side rests. Padded manacles should be applied only as a very last resort. Full-length padded arm splints are applied and the splints securely tied to the frame of the bed in order to prevent threshing around and the patient pulling out nasogastric, intravenous and catheter tubes. The 'boxing-glove' type of bandage, in which the fingers and wrist are enclosed by a crepe bandage over cotton-wool, allows the patient freedom of movement without his being able to do himself, his tubing, or others, harm. Although it fails in the violently restless, it should always be applied first. Side-rests to beds must on no account be left off after attending or examining the patient. Several deaths have occurred from falls out of bed almost immediately the attendant's back is turned. Intramuscular injection of 5 - 10 ml. paraldehyde is a well-tried measure, which recently fell into disfavour. Phenobarbitone, similarly administered in doses of 3 - 5 G, has the disadvantage of possibly clouding consciousness excessively.

The neuroplegic drugs are being used increasingly. The most frequently employed and popularized by Lazorthes of France[12] and the Oxford school,[14, 17] is a combination of chlorpromazine ('largactil'), promezathine ('phenergan') and pethilorfan. The latter is given to relieve painful stimuli which may be playing a part in the abnormal restlessness. The patient may be helped to settle on doses of 25 - 50 mg. of each, given intramuscularly. These medications can be repeated 4 - 6 hourly *p.r.n.* Despite their slight masking effects the experienced surgeon will readily recognize a deteriorating level of consciousness. *Morphia must never be given,* particularly in the elderly, even for severe associated injuries.

HYPOTHERMIA[12, 15, 17, 29]

The temperature on admission is usually normal or subnormal but may rise within a few hours to 103°F or even higher. This pyrexial reaction must be avoided and combated, since hyperpyrexia harms traumatized cerebral tissue by increasing the cellular metabolism excessively. It is being recognized increasingly that hypothermia should be induced early, as soon as it is realized that the craniocerebral injury is serious and likely to lead to prolonged coma. (Summary II). By this time a few hours have probably elapsed, the patient is being reasonably assessed and observed and the whole weight of therapeutic acumen can be applied. There need be no fear in applying hypothermia; even to the otherwise shocked patient. There is no danger of ventricular fibrillation at the level recommended.

SUMMARY II. BENEFITS OF HYPOTHERMIA

1. **Decrease O2 consumption by cerebral tissues by 50%.**
2. **Slows down cerebral metabolism.**
3. **Reduces CSF pressure.**
4. **Lessens hyperpnoea.**
5. **Diminishes excessive bronchial secretions.**
6. **Diminishes reactivity of the reticular formation.**
7. **Allows survival at lower metabolic level in areas anoxic from oedema or vascular insufficiency but potentially reversible.**

Hypothermia decreases oxygen consumption by the cerebral tissues by 50% (gray matter utilizes 8 times more oxygen than the white), slows down cerebral metabolism, reduces cerebrospinal-fluid pressure, lessens hyperpnoea, diminishes excessive bronchial secretions, diminishes the reactivity of the reticular formation and allows survival and function at a lower metabolic level to continue in areas already anoxic from oedema or vascular insufficiency but potentially reversible. During this inhibitory phase of cerebral metabolism, time is gained for the natural processes of resolution to become effective.

Brain-stem Lesions

Brain-stem lesions—to which hypothermia is mainly directed—comprise intrinsic damage and the effects of compression. As a consequence two clinical phases arise.[12]

1 Vagal—the patient is pale, hypertensive, bradycardiac, hypothermic and has slow respiration. A high level of acetylcholine in the cerebrospinal fluid may be related to this phase which is relatively short-lived (if present at all).

2 Sympathetic—there is hypermetabolism, and a febrile state results with an increase in pulse rate and respiration. There may be associated visceral dysfunctions, pulmonary oedema, haemorrhage in the upper gastro-intestinal tract—all evidences of irritative phenomena of the vegetative centres. According to Lazorthese there is also an escape of K from and entry of Na into the cells.

All these vegetative-centre phenomena can be considerably reduced and inhibited by hypothermia. It is therefore advisable, even imperative, to initiate this therapy before

the second phase arises, and it is considered that hypothermia may fail if applied in the later phase of relative irreversibility.

Technique of Hypothermia

The ideal is to aim for a rectal temperature of 95°F (axillary or any other method of measurement is undesirable). This is kept as constant as possible, any variation rather favouring the lower than the higher level.

First, apply the 'exposure method'. The patient is completely uncovered (properly screened off and minimal coverage supplied for the sake of modesty) and one, two or more electric fans directed to the body surface. Chlorpromazine, 50 mg., promezathine, 50 mg., and pethilorfan, 50 mg. (the hypothermic 'cocktail'), are given every 6 hours. These 'neuroplegic' drugs, besides inhibiting the reticular formation, are anti-oedematous, have a central sedative effect (particularly valuable in the restless patient), and cause moderate vasodilatation and, hence, aid heat loss necessary for hypothermia and, most important, prevent or greatly reduce the shivering response to the excessive cooling (Summary III). This is extremely important since uncontrolled shivering could easily nullify the effect of hypothermia by increasing the metabolism.

SUMMARY III. NEUROPLEGIC DRUGS

1. **Inhibit the reticular formation.**
2. **Are anti-oedematous.**
3. **Have a central sedative action.**
4. **Cause vasodilatation—aid heat loss.**
5. **Inhibit the shivering response to cooling.**

Should these methods prove inadequate to reduce or maintain the temperature at the required level, then more intensive procedures should be applied. A thin, well-soaked sheet is draped over the front and sides of the body, and large bowls containing ice are placed in front of the fans, thereby cooling the air blown over the patient. Should these measures still remain inadequate, then fragmented commercial ice (or ice-cubes) is packed around the body. The patient lies on a large rubber mackintosh and the head of the bed moderately elevated in order to facilitate the drainage of water from melted ice into suitably placed receptacles. In the rare cases of persistent elevation of temperature, iced enemata and/or the lavage of the stomach with ice water through a naso-gastric tube may be necessary. The essential object is to lower the temperature to hypometabolic levels and this should be achieved within 2 - 3 hours.

Once the desired level (94 - 95°F) is attained, further untoward lowering must be avoided and the intensity of cooling modified to meet changing conditions (of environment) and requirements. The drug schedule may also require intensification (up to 100 mg. of each) or diminution (in the absence of a shivering reaction—which is rare but a serious prognostic sign).

Special hypothermia baths, beds and blankets are available but are rarely required since the above measures, in our experience, have always brought about the desired response.

Once started the hypothermic level is maintained until signs of improvement become manifest, as shown by a lightening of the unconscious state: increasing movements or return of movements, swallowing reaction, coughing, etc.

It has been stated that hypothermia should be applied for a total period of 4 - 5 days,[17] but it is our practice to maintain it for weeks if necessary. At intervals it is good practice to withhold the neuroplegic drugs in order to remove their slightly masking effect. Should signs of returning consciousness become evident then the dosage is gradually diminished and the temperature allowed to stabilize itself. It is assumed arbitrarily that after 2 weeks oedema and haemorrhages of the midbrain will have been largely absorbed and cellular activity of the vital areas regained so that after this period, in the still fully comatose patient, hypothermia can gradually be dispensed with. The temperature-regulating mechanism, however, has been disturbed considerably, both pathologically and iatrogenically and, not infrequently, there may be a considerable overswing with marked hyperpyrexia (in one case to 108°F—with eventual survival). In such cases hypothermia must be immediately re-instituted.

There is, therefore, no hard and fast rule regarding the duration of the hypothermia—the surgeon must be guided by the clinical state of the patient and by the pathological history.

DEHYDRATING AGENTS

Dehydration, as such, plays no part in routine therapy. The recently introduced urevert solution (with strong dehydrating properties) has certain limited indications and should only be administered under skilled supervision. It is essentially a measure for an acute emergency. It is given intravenously as a 30% solution of urea,[9] prepared by adding 210 ml. of sterile 10% invert sugar to 90 G lyophilized urea. The recommended dose is 1 - 1·5 G/kg. of body weight, injected at a rate of about 60 drops per minute. No other solution is given simultaneously. It finds its most useful application before exploratory craniotomy. According to Javid,[9] it is desirable to administer ⅔ of the dose before opening the dura, though, in life-saving procedures the whole dose can be given. A marked dehydrating action on the brain tissue occurs with 'shrinking' of the brain. Intracranial manoeuvres, such as removal of subdural haematomata, are facilitated since there is more room for irrigation and suction. A moderate rise in blood pressure (20 - 26 mm. Hg) occurs.

In a separate 'drip' the normal requirements of fluid are administered as indicated by the patient's condition (electrolytes, urinary excretion, etc.).

Apart from its pre-operative indication urevert also finds use in cases where, after angiographic and exploratory measures, there is evidence of absence of a space-occupying lesion in the presence of a tense non-pulsatile brain (revealed at exploration), and in those cases where lumbar puncture had been performed and a markedly raised cerebrospinal fluid pressure found. The dose can be repeated in no more than 3 units per 24 hours, and, in our opinion, for no longer than 48 hours. It should only be used as an emergency measure. Owing to the intense diuresis which results, an indwelling Foley's catheter must be inserted into the bladder. *A blood-urea examination must be made before its administration.* Owing to the large volume of fluid eliminated via the kidneys, the use of urevert presupposes a good knowledge of electrolyte control.

An added note of warning—there are some who maintain that, because of the shrinkage induced in the brain-substance and diminution of volume, increased, or reactivation of, intracranial bleeding may ensue. The surgeon must be prepared to re-open the cranium in order to evacuate a re-accumulated haematoma.

(To be continued)