The measurement of intracranial pressure (ICP) as an aid in the assessment of certain neurosurgical patients is now widely accepted, following the initial descriptions of the technique (Guillaume and Janny, 1951; Lundberg, 1960).

An understanding of the dynamics of ICP changes is important for many anaesthetists, not only those who are working in specialized neurosurgical units, but also those who are involved in the care of the unconscious patient with a head injury, either in the Accident and Emergency Department or in the wards and operating theatres. For a more complete treatment of the effects of anaesthesia on ICP, the reader is referred to the review of McDowall (1975).

THE INTRACRANIAL STATE

The rigid, and virtually closed, container which is the adult skull contains four constituents:

(i) brain substance (including interstitial fluid),
(ii) cerebrospinal fluid (c.s.f.),
(iii) arterial blood,
(iv) venous blood.

As is well known, an increase in the volume of any one constituent occurs at the expense of another; for example, an increase in the apparent volume of brain substance, such as is seen with cerebral oedema, is balanced, in the acute phase, by the loss of c.s.f. from the skull to the spinal space, and also by the extrusion of blood from the thin-walled cerebral veins.

The initial expansion, then, of an abnormal intracranial lesion is accompanied by only a moderate increase in ICP, because an equal volume of c.s.f. and venous blood, or both, is lost from the skull. Eventually, if the expansion of the lesion continues, this compensation becomes exhausted and, at this point, a large increase in ICP is produced. The relationship between the abnormal volume expansion within the skull and ICP is graphically represented by the volume/pressure curve of the intracranial contents (fig. 1). The initial part of this curve is relatively flat, as a large increase in volume of one of the intracranial components is associated with only a small increase in ICP. As the abnormal volume expansion continues, however, large increases in ICP are produced for a small volume increase. The steep part of the curve represents the situation in patients with advanced intracranial disease who are in a critical condition. Clearly, then, it is important to ensure that an increase in the volume of one of the intracranial components, caused by disease or injury (for example, oedema, haematoma or tumour), is not further compounded by an iatrogenic increase in intracranial volume (for example, cerebral venous obstruction, administration of volatile anaesthetic agents, hypoxia, hypercarbia or airway obstruction). The effect of any of these iatrogenic causes is to increase the abnormal amount of intracranial space occupied by disease, so that the patient is “moved to the right” towards the steep part of the volume/pressure curve, and the primary lesion is rendered more serious than it would otherwise be.

THE MEASUREMENT OF INTRACRANIAL PRESSURE

The two sites commonly used for ICP measurements are the ventricular system and the extradural space.
Ventricular system

The measurement of ICP is traditionally made from one or other lateral ventricle by the insertion of a catheter into the ventricle, either through a burr hole or through a twist drill hole in the skull. Commonly, the catheter is connected to a pressure transducer which is usually mounted on the patient's head, and the trace so obtained is displayed on a chart recorder.

The advantages of the ventricular method of ICP measurement are considerable. The measurements are made from a fluid-filled space and it is therefore possible to achieve high-quality recordings showing variations in ICP with both pulse and respiration. C.s.f. can be removed either for the relief of high pressure states or for acid-base measurements.

There are some disadvantages to the ventricular measurement of ICP, however (Turner et al., 1975), and these include difficulty in catheterizing the ventricles especially when the brain is distorted by tumour or haematoma, or when the ventricles have been compressed. The fact that the dura has been breached opens the brain to the possibility of infection, especially as fluid pressure measurements may be required for some time. Some difficulty may be experienced in maintaining an adequate pressure trace since this may become damped as a result of blockage of the catheter or of impaction of the catheter tip against the ventricular wall, which is presumably caused by brain shift.

The extradural space

Measurement of ICP from the extradural space is often technically easier to institute as it requires only the fixation of an appropriate device in a burr hole. The same good-quality recordings can be obtained from the extradural route as from the ventricular route, but, to achieve this, care must be taken to prevent any leakage of fluid from the site of the extradural pressure-measuring device. The fact that the dura is intact makes the possibility of serious infection much less likely.

The major disadvantages of the extradural route for ICP measurement centre around the relationship of the pressure-sensitive element of the transducer to the dura, and the effect of the dura on the pressure measured (Dorsch and Symon, 1975). The transducer should be fixed so that it is in contact with the dura but not indenting it. The transducer should also be parallel, or coplanar, with the dura, otherwise the dura will be stretched and the pressure recorded will then be affected by the compliance of the dura (Schettini et al., 1972). The implantation of a transducer in a burr hole may result in inaccurate results if the zero-point or the calibration of the transducer varies. Many workers have attempted to overcome this difficulty by the development of more reliable transducers with low zero-drift and also instruments in which the zero-point may be checked in use.

Another solution to this problem is to use a transducer mounted externally on the skin surface, connected to a hollow screw fixed in the skull (Coroneos et al., 1972), and mounted so that the inner surface of the device is flush with the inner surface of the skull. If the dura is opened, such a device may also be used most satisfactorily for subdural pressure measurements (Vries, Becker and Young, 1973). Calibration of the externally mounted transducer is as easily accomplished as for the ventricular pressure transducers.

RELATIONSHIP OF INTRAVENTRICULAR AND EXTRADURAL PRESSURES

In brief, the intracranial pressure as observed by the extradural route appears to be 1–2 mm Hg higher than the ventricular pressure at normal intracranial pressures (that is normal ICP = 5–13 mm Hg), but at high intracranial pressures the extradural pressure considerably over-reads the ventricular pressure (Coroneos et al., 1972; Gobiet et al., 1972; Jørgensen and Riishede, 1972; Sundbärg and Nornes, 1972). In more than 60 patients studied in Leeds, no patient has had an abnormally high intraventricular pressure in the presence of normal extradural pressure (Gibson et al., 1975; Turner et al., 1975).

CAUSES OF INCREASED INTRACRANIAL PRESSURE

Disease
- (a) cerebral tumour
- (b) intracranial haematoma
- (c) cerebral oedema
- (d) benign intracranial hypertension

Iatrogenic
- (a) hypoxia
- (b) hypercarbia
- (c) halogenated anaesthetics
- (d) coughing and straining
- (e) physiotherapy
- (f) head-down tilt
- (g) badly adjusted pressure ventilation
- (h) compression of jugular vein

(CBF = cerebral blood flow; CBV = cerebral blood volume.)
INDICATIONS FOR ICP MEASUREMENT

Patients with a severe closed head injury

In the acute stage of the management of head injury, ICP measurement alerts the clinician at an early stage to the occurrence of brain compression, caused either by oedema or by haematoma formation. It is not possible to differentiate between these two alternatives solely on the basis of ICP measurement, but if the possibility of intracranial bleeding exists, the patient requires either urgent angiography or burr hole exploration. An EMI scan, if available, is an alternative. For example, we have seen two patients in whom a posterior fossa collection of blood produced a high ICP by obstructing c.s.f. drainage. In each case the presence of the clot was suspected initially from the high ICP and confirmed by ventriculography and craniotomy (Gibson et al., 1975). If the clinician is certain that cerebral compression is caused by oedema and not by bleeding, then medical treatment of cerebral oedema with dehydrating agents, diuretics and perhaps steroids is indicated. In carrying out this treatment under the rapidly changing conditions which exist in the acute stage, continuous monitoring of ICP is of considerable assistance.

Neurosurgical patients who require artificial ventilation

Many neurosurgical patients benefit from a period of artificial ventilation. One obvious example is the patient with head injuries and associated chest injuries, especially if he is hypoxic. Such a patient, when seen initially, may appear to have extremely severe head injuries, but only after the establishment of good oxygenation and pulmonary ventilation can an estimate of the true extent of his intracranial pathology be made.

The establishment of artificial ventilation often involves the use of muscle relaxants or of sedative drugs to reduce the amount of straining against the ventilator which restless patients often exhibit. Such straining causes poor pulmonary ventilation, and a high thoracic venous pressure which is transmitted to the cerebral veins, resulting in an increase in ICP. The use of either muscle relaxants or sedatives will result in the loss of one of the most useful methods of monitoring—the assessment of responsiveness, including level of consciousness. If opiates are used for sedation, then pupillary signs are obscured. In these circumstances ICP is a most valuable guide to the patient's hour-by-hour condition and the combination of artificial ventilation and ICP measurements may allow the use of potent analgesics in patients who require them. It may also allow the effective suppression of convulsive activity by the use of sedative and hypnotic drugs.

Postoperative neurosurgical patients

In the few days immediately following neurosurgery many derangements of ICP may take place. Nakagawa, Yada and Tsuru (1975) have reported a peak of ICP within 12 h of the end of neurosurgery, and another at 26–48 h. Cerebral oedema particularly may cause periods of high ICP after operation, especially in patients who have had extensive brain retraction or profound degrees of induced hypotension. Following such brain "insults" normal autoregulation is impaired or lost, so that any increase in arterial pressure is accompanied by an increased CBF and so by an increased ICP (Keaney, Pickerodt et al., 1973; Keaney, McDowall et al., 1973). It is known from animal experimental work (Haggendal and Johansson, 1971/72) that increases in arterial pressure in the absence of autoregulation may result in cerebral oedema formation, so that it is important to be able to detect by ICP monitoring such arterial pressure-dependent changes and to initiate treatment to avoid periods of systemic hypertension in the early period after surgery.

Recognition of pressure waves

Lundberg (1960) first described these patterns of abnormal ICP. "A" waves are large increases of ICP above base-line, commonly to 60–80 mm Hg and of about 20 min duration. Such waves are particularly sinister, as they denote a patient who is near the limit of compensation for intracranial volume changes. An example is included in figure 2. "B" waves are large decreases of ICP below base-line, commonly to 10–20 mm Hg and of about 10 min duration. Such waves are particularly ominous, as they denote a patient who is near the limit of compensation for intracranial volume changes. An example is included in figure 2.

**Fig. 2.** An "A" wave, as defined by Lundberg. This tracing was obtained from a head-injured patient and shows a typical "A" wave, with ICP increasing by more than 50 mm Hg. The time axis is in 10-min intervals. (Reproduced from McDowall (1976), by kind permission of the editors and publishers.)
waves occur about once a minute. They are less immediately sinister than "A" waves but, nevertheless, they denote definite intracranial pathology. "C" waves occur usually at rates up to 6/min. Action should be taken if any of the above derangements of pressure patterns are seen, to avoid the development of further intracranial compression.

For delineation of prognosis

Vapalahti and Troupp (1971) have pointed to the poor outcome in most patients with an ICP above 60 mm Hg for any length of time. The appearance of waves of increased ICP implies a poor prognosis, especially if high amplitude waves occur frequently (Cold, Enevoldsen and Malmros, 1975). Extreme caution must be exercised, however, in using ICP measurement for assessing prognosis in individual patients (Miller, Garabi and Pickard, 1973).

ASSESSMENT OF THE VOLUME/PRESSURE RELATIONSHIP IN CLINICAL PRACTICE

As discussed above, the measurement of ICP will provide valuable clinical information in certain patients, but because of the volume compensation available to the brain, ICP changes little in the early stages of a space-occupying lesion, especially if it is growing slowly. In order to assess the degree of intracranial compression present, therefore, it is sometimes valuable to test the response of ICP to the sudden addition of volume to the intracranial space; this is termed a test of intracranial compliance. At present there are two ways of doing this.

Pressure/volume testing

Some workers have measured the pressure increase that occurs after an injection of a standard volume (say, 1 ml) of saline into the lateral ventricle, while ICP measurement is in progress (Miller, Garabi and Pickard, 1973). The greater the pressure increase that occurs, the greater the brain compression present, and the less the compensation still available. Miller, Leech and Pickard (1975) quote an increase in pressure of more than 2 mm Hg following injection of 1 ml saline into the ventricles as being significant, and Leech and Miller (1975) have shown improvements in the volume/pressure response following treatment with mannitol or betamethasone.

Analysis of ICP variation

The idea of observing the response of ICP to a standard stress can be extended by observing the natural variation in ICP. Even with no intracranial disease, each heart beat adds a volume of blood to the cerebral blood volume and a pressure increase is seen.

In the patient with an intracranial space-occupying lesion, the intracranial pulse pressure widens as the lesion increases in volume. In such a patient other causes of a temporary increase in ICP, such as a cough, will produce a greater and more sustained pressure increase than in the normal patient.

The amount of variation around a mean intracranial pressure then may be indicative of the degree of intracranial compression that exists. The greater the variation, the more nearly exhausted are the compensating mechanisms for intracranial volume increase (Szewczykowski et al., 1975).

Thus, more intensive analysis of ICP variations has been attempted by many workers (Janny et al., 1972; Kullberg, 1972). We have used a computer-based system for ICP measurement and analysis which samples ICP at a pre-set rate (usually once every 4 s). At regular time intervals (usually 30 min), the computer calculates the mean and standard deviation of the last block of results, then prints a frequency histogram and calculates the degree of skewness. The standard deviation, of course, increases as the variation of ICP increases and may be taken as an index of the degree of decompensation existing in the skull. Figures 3A and B show successive frequency histograms from a patient who had suffered a head injury. Figure 3A shows an acceptable distribution of pressure, with a normal mean ICP and standard deviation. Figure 3B shows the next period of observation in which the mean ICP is still within the normal range, and most observations fall below 15 mm Hg. The standard deviation of this block, however, is much greater and reflects the increased variation of ICP which preceded an increase in ICP to 25–30 mm Hg.

TREATMENT OF INCREASED ICP

Prevention

The avoidance of increases in ICP during the care of neurosurgical patients, whether in the ward or operating theatre, depends on careful attention to all details of good management, which are described elsewhere in this issue.

Hyperosmolar therapy

Mannitol, laevulose and glycerol are the agents usually recommended for the reduction of ICP. Their
action is moderately quick in onset. They are not always effective, hence the importance of ICP measurements to ensure an adequate dose. Electrolyte disturbances are not uncommon following their use.

**Steroids**
High doses of dexamethasone and betamethasone are most effective in the relief of pressure states from tumour, especially metastatic tumour or cerebral abscess. They appear to be less useful for the oedema which complicates head injury. Patients who receive steroid therapy should also receive oral antacids to guard against gastric ulceration. Steroid action starts after some hours and therefore is best preceded by the administration of hyperosmolar agents for a more rapid effect.

**C.s.f. removal**
C.s.f. removal should not be undertaken lightly, as it is possible to promote brain shift if aspiration is performed too rapidly. Aspiration of c.s.f. from compressed ventricles is not always easy.

**Hyperventilation**
Hyperventilation should perhaps be used in the treatment of high-pressure states more often than at present, and Shapiro, Wyte and Loeser (1975) have recorded the treatment of intractable high ICP with i.v. barbiturates.
REFERENCES


