Personal practice

Intracranial pressure monitoring

R A MINNS

Department of Child Life and Health, University of Edinburgh

Increasing awareness of the part played by raised intracranial pressure in brain damage together with clinical signs which are often unreliable and the static nature of ultrasound or computed tomography for what is essentially a dynamic problem have meant that there is now widespread acceptance of intracranial pressure monitoring in children. The reasons for monitoring are to detect raised pressure, to quantitate this, and to determine the effectiveness of treatment.

Techniques and indications

This review article mentions only a few of the many devices and techniques presently available for intracranial pressure monitoring. For routine clinical use the keynote should be simplicity—in the type of equipment used, method of calibration, routine for checking time and temperature drift, sterilisation, and in a standard zero reference value. Basic equipment, which can be used in most circumstances and is inexpensive compared with modern hospital equipment, consists of a small Luer-locking solid state transducer (for example, Gaeltec or Statham), a pre-amplifier, and a physiological pen recorder. More sophisticated display, storage, and data processing with microprocessors or computers may, however, be desired. There are three principal opportunities for measuring intracranial pressure.

(1) At the time of lumbar puncture. Traditional practice has been to measure cerebrospinal fluid pressure at lumbar puncture by open ended manometry but this method is fraught with inaccuracies in routine practice. The mere fact that cerebrospinal fluid is displaced from a closed system to fill a column is fundamentally error producing; ‘eyeballing’ a fluctuating meniscus and the need for several pairs of hands have negated much of its usefulness. Measurement can now be accomplished simply without displacement of cerebrospinal fluid with the patient in the lateral recumbent position for lumbar puncture and with no undue abdominal compression, flexion or extension of the neck. Where there is free communication between the spinal and cranial compartments the measured pressure reflects the intracranial pressure. A paper recording of a few minutes duration is made before cerebrospinal fluid collection, with the transducer attached to the lumbar puncture needle by a three way connection. The presence of a cardiorespiratory artefact verifies the space and because there is no displacement of cerebrospinal fluid this method is theoretically safer. The mean pressure is independent of the internal bore of the lumbar puncture needle, although oscillations will be damped with fine bore needles. This simple adaptation improves appreciably the routine practice of estimating cerebrospinal fluid pressure at the time of lumbar puncture and equipment for this should be available in most treatment rooms and accident departments. Measurement can be performed after brief instruction on how to calibrate and sterilise the equipment; most children find it no more uncomfortable than the lumbar puncture procedure has always been; and it can be carried out in any paediatric age group.

(2) When the ventricles are enlarged. The most accurate intracranial pressure recordings are obtained from the ventricles, either directly from ventricular cannulation (burr or twist drill hole) or via a ventriculostomy reservoir connected to an external transducer. This is the method of choice in the older child where the ventricles are easily located. As little cerebrospinal fluid as possible should be split and all joints must be watertight. It allows easy recalibration, a means of controlling pressure (by drainage against a pressure of 10 mmHg) and has the additional advantage of enabling pressure volume responses (PVI in mmHg/ml) to be carried out, thus identifying patients at risk of pressure decompensation.1 For the older child with active hydrocephalus, management is dramatically altered by placing a...
separately sited reservoir (usually in the right frontal horn) at the same time as definitive cerebrospinal fluid shunt insertion, thus providing both access and drainage. When there is subsequent block or infection of the shunt simple tapping of the reservoir and direct intracranial pressure recording will often delineate the problem. Equivocal pressure results may necessitate a provocation record through several rapid eye movement sleep phases when intracranial pressure increases as a result of enhanced cerebral blood flow.

Ventricular pressure monitoring is also helpful in children with intermittently active hydrocephalus and long standing pressure symptoms and those with space occupation from tumour, clot, cyst, or abscess. The use of this method in tuberculous meningitis, ventriculitis, and other meningitides has also allowed better control of intracranial pressure while the infection is being treated.

A telemetric pressure sensor for hydrocephalus and ventricular shunt systems combines a telemetric device attached to either the proximal limb of a cerebrospinal fluid shunt or a reservoir. This allows in vivo confirmation of zero point and pressure calibrations by means of pressure balanced telemetry. This may prove of value in the outpatient assessment of hydrocephalic states in the future and is currently under evaluation in Edinburgh.

In neonates with ventriculomegaly short opportunistic records may be obtained at the time of percutaneous ventricular puncture but prolonged recordings require that a finer, non-compliant catheter be left in situ. If the ventricles are not easily located this way no more than two attempts should be allowed before alternative techniques are sought. Insertion of a miniaturised ventriculostomy reservoir of the type recently designed (Steers J; personal communication) may be a useful alternative to repeated puncture or hardware of adult dimensions.

At the brain surface. In older children with conditions of brain swelling in coma and decerebrate states such as Reye’s syndrome, anoxic ischaemic encephalopathy, and head injury a fluid filled metal screw is mounted in a twist drill hole and placed in the subarachnoid space. This avoids brain puncture, is secure on the skull, and is independent of shifts or small ventricular size. Several types of screw have been used such as the single lumen screw and the Leeds screw but my preference is for the Newell modification of the Leeds screw which is stainless steel, simple in design, without movable parts, easily autoclaved, and has a luer connection for attachment to the transducer. It is imperative that a good bolt-space connection is maintained and the sloping thread of this screw fits neatly into the cranial bones after a threader has been used on the inner aspect of the drill hole. Again this equipment is inexpensive and the apparatus may be calibrated externally.

The use of screws in the epidural space, which avoids opening the dura and theoretically lessens the risk of seizures, haematoma, or infection has not proved reliable because of signal damping. Subdural placement also records disparate pressures (compared with ventricular pressure) at values greater than 20 mmHg. Intracranial catheter tip pressure transducers for epidural use and implanted epidural transducers probably only approximate cerebrospinal fluid pressure and are inferior to the screw in my experience.

There are additional problems in infancy and in the newborn—swollen brain syndromes consequent on anoxic ischaemic injury, central nervous system infections etc. Neonatal methods which have been included measurements from most of the surface spaces such as the subarachnoid or subdural where a wide bore medicut is introduced via the anterior fontanelle using a conventional subdural puncture procedure and leaving a teflon catheter in situ. (These are attractive because they are easy to insert). It is important, however, to ensure that movement of the catheter does not cause air liquid interfaces thus falsifying the recorded intracranial pressure. These methods require further validation by comparison with ventricular pressures. A recently designed, plastic miniaturised screw is currently on trial for this group of children. Its smaller size and light weight make it more desirable for the thin cranial bones of infancy.

Many forms of fontonometry have been tried over the past 10 years such as the Ladd device, stethoscope ‘pick ups’, tambours, modified Schiotz tonometer, aplanation transducers, pressure activated fibreoptic sensors, oscillographic technique, sagittal sinus pressure or bloodflow, and impedance, and while varying degrees of success have been achieved fundamental problems still exist with coplanimetry, the external pressure applied, tension in the fontanelle etc, making these methods suitable for research but of limited value in routine clinical practice.

Recordings

The zero reference value is usually taken as the foramen of Munro (mid-cranium) with the patient supine. The hydraulic system of blood brain and cerebrospinal fluid is responsible for the normal intracranial pressure. For older children and adults intracranial pressure values less than 15 mmHg are considered normal, values in excess of 20 mmHg are...
unequivocally high, and values greater than 40 mmHg severely so. There is a gradual increase in the normal intracranial pressure from birth through childhood with values of approximately 2 mmHg in the newborn, up to 5 mmHg mercury by the end of infancy, and between 6 and 13 mmHg for the child up to 7 years of age.

Critical intracranial pressure values cannot be deduced from pressure recordings alone. The cerebral perfusion pressure (mean systemic arterial pressure minus mean intracranial pressure) is the important parameter and the critical value of cranial perfusion pressure is 40 mmHg, below which cerebral blood flow falls precipitously in adults and older children with intact autoregulation. Infants with their additional intracranial buffering should tolerate lower cranial perfusion pressure but children with loss of autoregulation will require a higher value. Cranial perfusion pressure may be prognostic in birth asphyxia.

The intracranial pressure record can be varied by means of the chart speed and amplifier gain to identify the ‘shape of the trace’ which is decided by arterial input, intracranial contents, and venous outflow. The configuration of the pulse wave represents a complex sum of various components, although the two predominant frequencies are of respiratory and cardiac origin. There are three fairly consistent components of the arterial pulse wave, a ‘percussion wave’ which originates in the choroid plexus and the large intracranial conductive vessels, the ‘tidal wave’ which ends in a dichrotic notch and may reflect variations in cerebral bulk compliance, and the ‘dichrotic wave’ after which the pressure wave tapers to its diastolic position. The mean intracranial pressure is usually taken at the midpoint of these fluctuations. With increasing intracranial pressure the pulse pressure artefact increases in width and the respiratory component assumes less importance.

Transitory effects on the pressure tracing from events such as coughing, crying, passage of nasogastric tube, and nappy changes need to be differentiated from abnormal wave forms. Many abnormal wave forms based on amplitude and frequency have now been described, the most important of which are the plateau waves (A waves) resulting from acute non-compliance. These are seen frequently in children with swollen brain or hydrocephalics with acute non-communication in whom pressure may exceed 50 mmHg and may be raised for 20 minutes. Although there is usually no clinical warning of acute non-compliance, these episodes may end in cardiorespiratory arrest. They do not occur in the newborn or early infancy. Other abnormal wave forms described include sinus and ramp B waves, C waves, ‘hills’ or pre-plateaus, S (negative) waves, and scallop waves.

Finally, there is a slight risk of complication depending on the method used for invasive techniques. This risk is estimated as between one and five per cent and complications may take the form of intracranial haemorrhage or infection. The latter is related to the length of time cerebrospinal fluid is open to the atmosphere and also to the use of prolonged steroid treatment.

References