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ESSENTIALS Normal intracranial pressure is between 5 and 15 mm Hg in supine subjects. Intracranial hypertension (ICP >20 mm Hg) is common in many central nervous system diseases and in fatal cases is often the immediate cause of death. Aetiology and pathogenesis—increases in intracranial volume and hence—given the rigid skull—intracranial pressure may be the consequence of (1) brain oedema, (2) increased cerebral blood volume, (3) hydrocephalus, and (4) space-occupying lesions. Brain perfusion depends on the cerebral perfusion pressure, which is mean arterial pressure minus intracranial pressure. The normal brain autoregulates cerebral blood flow down to a lower limit of cerebral perfusion pressure of about 50 mm Hg in healthy subjects, and perhaps 60–70 mm Hg in disease. Cerebral perfusion pressure reduction to below these values results in cerebral ischaemia. Clinical features—the cardinal symptom of intracranial hypertension is headache, which may be accompanied by vomiting, visual disturbance, and alterations in mental function or conscious state. Papilloedema is the classical sign, but may be absent. Severe elevation of intracranial pressure can result in bradycardia and hypertension (Cushing's response), abnormalities of breathing (Cheyne-Stokes respiration, central neurogenic hyperventilation, 'ataxia of breathing'), and various forms of cerebral herniation. Investigation—computed tomography or magnetic resonance imaging is the investigation of choice; if lumbar puncture is considered (e.g. for diagnosis of meningitis), imaging must be done first and lumbar puncture avoided if the basal cisterns are effaced by cerebral oedema. Management—this involves (1) ensuring normoxia and normocapnia ($P_{aO_2} > 11$ kPa, P_{aCO_2} 4.5–5 kPa), with tracheal intubation and ventilatory support where required; (2) treating precipitating factors such as seizures, fever, and electrolyte abnormalities; (3) treating raised intracranial pressure with mannitol, dexamethasone (for tumours), hyperventilation (if pupillary dilatation/clinical picture merits); and (4) monitoring intracranial pressure if appropriate (e.g. trauma). **Introduction** The normal intracranial pressure (ICP), measured at the level of Monro's

foramen, is between 5 and 15 mm Hg in supine subjects. Intracranial hypertension (ICP >20 mm Hg) is a common accompaniment of many central nervous system (CNS) diseases. In many of these situations intracranial hypertension is the most important cause of symptoms and determinant of outcome, and in fatal cases is often the immediate cause of death. Epidemiology Intracranial hypertension is a pathophysiological mechanism common to many diseases. Acute intracranial pressure elevation is commonly encountered in traumatic brain injury, haemorrhagic and large ischaemic strokes, and intracranial infection. Subacute and chronic intracranial hypertension are seen in intracranial tumours. Less commonly, intracranial hypertension may be observed without any underlying cause—the syndrome of idiopathic intracranial hypertension. Pathophysiology The cranial cavity contains brain (80%), blood (10%), and cerebrospinal fluid (CSF; 10%). These incompressible contents are bounded by a rigid skull with a fixed capacity. Consequently, an increase in volume of any of these contents, or the presence of any space-occupying pathology, results in an increase in intracranial pressure unless one of the other constituents can be displaced or its volume decreased (Fig. 17.7.1). This principle is referred to as the Monro-Kellie doctrine. Increases in intracranial volume may be the consequence of:

- Brain oedema, which may have different pathogenic mechanisms:
 - Cytotoxic oedema occurs as a result of cell swelling, most commonly due to ischaemic energy depletion and increases in intracellular Na^+ and water.

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- Vasogenic oedema results from an increased permeability of the blood-brain barrier with an expansion of the extracellular fluid compartment.
- Interstitial oedema occurs in the context of hydrocephalus, where increased intraventricular cerebrospinal fluid pressures result in permeation of cerebrospinal fluid into adjacent brain, typically in the frontal periventricular regions.
- Vascular engorgement that results from increased cerebral blood volume. This may be due to the vasodilatation that accompanies normal or abnormal (e.g. epileptiform) neuronal activity. In other situations vasodilatation may be due to loss of vasoregulation, either due to disease (vasoparalysis), or due to the effect of potent physiological (carbon dioxide) or pharmacological (nitrates and other nitric oxide donors) cerebral vasodilators.
- Hydrocephalus, which may be noncommunicating (where an obstruction prevents the ventricular system communicating with the subarachnoid space), or communicating (where there is a defect in cerebrospinal fluid reabsorption).
- Space-occupying lesions (SOL), which may be either chronic (e.g. intracranial tumours) or acute (e.g. intracranial haematomas associated with trauma).

Temporal patterns of intracranial pressure change Initial increases in intracranial volume are buffered by displacement or reduction in volume of other contents. Thus, cerebral oedema may result in compression of the ventricles, with translocation of cerebrospinal fluid to the spinal subarachnoid space, and compression of cerebral vasculature. Over longer time periods, normal brain may be compressed and cerebrospinal fluid production diminished. The relationship between intracranial volume (ICV) and intracranial pressure is commonly depicted as a hyperbolic curve, with an initial flat part during which compensatory mechanisms are effective, a knee that represents their progressive exhaustion, and a steep phase when even small increases in intracranial volume produce large increases in intracranial pressure. However, the extent and efficiency with which these mechanisms buffer increases in volume depend on the speed of progression of disease. Given these considerations, it is more appropriate to depict the evolution of pathophysiology as a family of curves, with variable rates of progression (Fig. 17.7.2). It is important to make three further points in this context:

- First, a precipitating factor may suddenly

increase the speed of progression of a relatively slow pathophysiological process, and be the proximate cause of symptomatic decompensation. • Secondly, acute changes in cerebrovascular physiology are an important cause of such deterioration. Both hypoxia and hypercarbia can cause cerebral vasodilatation and elevate intra- cranial pressure. While severe hypertension may result in cere- bral oedema, it is far more common to find that relatively minor reductions in mean arterial pressure compromise cerebral per- fusion and trigger reflex vasodilatation and secondary increases in intracranial pressure. Such haemodynamic instability may be the underlying cause of phasic increases in intracranial pressure (Fig. 17.7.3).

(a) AV FM SSAS ICP 100 Arterial Venous CSF CP Brain 0 Sol Pressure transducer (b) AV SSAS ICP 100 Arterial Venous CSF CP Brain 0 Pressure transducer

Fig. 17.7.1 Schematic diagram showing intracranial contents in the normal brain (a) and with elevated intracranial pressure (b). Note that cerebrospinal fluid (CSF) is produced by the choroid plexus (CP), circulates freely, passing through the foramen magnum (FM) into the spinal subarachnoid space (SSAS), before absorption by arachnoid villi (AV) in the cerebral venous sinuses. Increases in intracranial pressure may be due to brain oedema, vascular engorgement, space-occupying lesions (SOL), or impaired CSF circulation or absorption. Compensatory mechanisms include translocation of CSF to the SSAS, and compression of cerebral vascular beds. The intracranial pressure trace shows a higher mean value, and the inability of the noncompliant brain to cope with increased blood during each systole results in an increased pulsatility of the intracranial pressure waveform.

40 ICP (mmHg) 30 20 10 0 Rate of rise of ICP X Intracranial volume Slow Fast

Fig. 17.7.2 Intracranial volume/pressure curves. Increases in intracranial volume (ICV) are initially buffered by compensatory mechanisms, but eventually result in intracranial pressure (ICP) elevation. The ability to buffer ICV increases depends on the speed at which pathology develops. Gradually progressive increases in ICV (such as those produced by a slow growing tumour) may be well compensated, until a precipitating factor (e.g. the development of hydrocephalus, denoted by X in the diagram) shifts the relationship to a steeper curve.

Section 17 Critical care medicine 3894 • Finally, since patients with significant intracranial hypertension operate on the steep part of the ICV/ICP curve, even small de- creases in intracranial volume (e.g. a 5 ml decrease in cerebral blood volume produced by mild hyperventilation) can have grati- fyingly large effects on intracranial pressure. Why treat intracranial hypertension? Brain perfusion depends on the cerebral perfusion pressure (CPP), the difference between mean arterial pressure (MAP) and intracra- nial pressure. While the normal brain autoregulates cerebral blood flow across a large range of CPP values, the lower limit of such autoregulation is about 50 mm Hg in healthy subjects, and may be sig- nificantly higher (60–70 mm Hg) in disease. CPP reduction below the lower limit of autoregulation results in cerebral ischaemia, and even minor reductions in CPP may trigger reflex vasodilatation and increase intracranial pressure in a noncompliant intracranial cavity. Such cere- bral ischaemia is important in its own right. For instance, intracranial hypertension may be the direct cause of neurocognitive deficits in sur- vivors of traumatic brain injury. It is very likely that both the severity and duration of ICP elevation contribute to the ‘dose’ of intracranial hypertension that is responsible for its adverse effect on outcomes. An expanding focal mass can generate pressure gradients within the intracranial cavity, and the resulting displacement of brain against rigid structures, and protrusion (herniation) of brain through narrow openings between intracranial compartments can press on vital structures and result in death (Fig. 17.7.4). Prolonged intracranial hypertension may result in permanent damage to critical structures. Thus, benign intracranial hyperten- sion rarely results in herniation syndromes, but if left untreated, fre- quently results in optic atrophy.

Clinical features Symptoms The symptoms that

accompany intracranial pressure elevation can be nonspecific and insensitive. The cardinal feature of intracranial hypertension is headache, which may be described as severe ('worst ever') and explosive in onset in the setting of intracranial haemorrhage. The headache of intracranial tumour is often progressive, worst on awakening (possibly due to intracranial pressure elevations associated with the supine position and PaCO₂ elevation in sleep), and is exacerbated by coughing and straining. However, it may be indistinguishable from common tension headache, and dangerous intracranial hypertension may occur without headache. The headache is often accompanied by vomiting, which is classically described as projectile and not preceded by nausea. Visual disturbances are often reported, which may be attributable to optic or oculomotor nerve compression (with accompanying visual failure or diplopia, respectively). Alterations in mental function or consciousness may be observed, ranging from impaired concentration, through increased irritability, impaired cognition and memory, and altered personality, to increased somnolence and deep coma. Signs While papilloedema is the classical sign associated with intracranial pressure elevation, it is not seen with acute intracranial hypertension, and may be absent even with large intracranial masses. Pressure on cranial nerves may result in weakness of ocular movement. The abducens nerve is often involved in such a process due to its long intracranial course, and the resultant diplopia provides the CPP ICP CBV 140 [A] 120 MAP (mmHg) 100 60 45 40 5 min 25 5 ICP (mmHg) 80 [B] Cerebral vasodilatation CPP (mmHg) CBV Cerebral vasoconstriction CPP ICP Fig. 17.7.3 Intracranial pressure (ICP) traces show phasic variations which may last several minutes (Lundberg A waves; (A)) or may be more transient (Lundberg B waves; (B)). ICP elevations are often initiated by reductions in mean arterial pressure (MAP), which trigger compensatory vasodilatation and increase cerebral blood volume (CBV) and ICP. This vicious cycle may be terminated by spontaneous hypertension associated with a Cushing's response (arrow in MAP and ICP traces), or by therapeutic elevation of MAP, which triggers compensatory cerebral vasoconstriction and reductions in ICP. Note that a period of stable MAP greater than 100 mm Hg is associated with a low, stable ICP. Modified from Rosner MJ (1993). Pathophysiology and management of increased intracranial pressure. In: Andrews BT (ed) Neurosurgical intensive care, p. 75. McGraw-Hill, New York. 1 2 3 4 Fig. 17.7.4 Cerebral herniation may be (1) subfalcine (beneath the falx cerebri), (2) transtentorial (through the tentorial hiatus with compression of the midbrain and posterior cerebral artery), (3) tonsillar (where the cerebellar tonsils herniate through the foramen magnum and compress the lower brainstem upper cervical cord), or (4) transcalvarial (through a traumatic or surgical defect in the roof of the cranial cavity). Modified from Fishman RA (1975). Brain edema. *New Engl J Med*, 293, 706-11.

17.7 Management of raised intracranial pressure 3895 classical example of a false localizing sign. Lesions that irritate the posterior fossa dura can produce neck stiffness. Progressive increases in intracranial pressure result in bradycardia and hypertension, which constitute the Cushing's response and signify stimulation of brainstem autonomic nuclei. Worsening brain stem compression and/or ischaemia result progressively in Cheyne-Stokes respiration, central neurogenic hyperventilation, and irregular respiratory patterns ('ataxia of breathing'). Both neurogenic pulmonary oedema and the adult respiratory distress syndrome have been associated with intracranial hypertension; sudden massive increases in ICP, such as occur with a high grade subarachnoid haemorrhage, cause a 'catecholamine storm' with severe acute systemic arterial hypertension that causes both pulmonary vascular and myocardial damage. Severe elevation of intracranial pressure may result in herniation of the temporal lobe through the tentorial notch (Fig. 17.7.4). This produces clinical features due to pressure on the ipsilateral oculomotor nerve

(ipsilateral pupillary dilatation), pyramidal tract (contralateral weakness), and brainstem (Cushing's response and abnormal respiratory patterns followed by circulatory collapse and respiratory arrest). The posterior cerebral artery is frequently compressed by the herniating temporal lobe, and successful resuscitation from threatened or early transtentorial herniation may leave a patient with an ipsilateral occipital infarction.

Clinical investigation

Imaging

The most informative standard imaging in patients with intracranial hypertension is computed tomography (CT), which may reveal subarachnoid or intracerebral blood, contusions, or a tumour. In addition, cerebral oedema may be manifest by loss of sulci, compression of the third and lateral ventricles, and effacement of the perimesencephalic and suprasellar cisterns. Unilateral lesions may result in midline shift (which can occur without pupillary asymmetry), compression of the ipsilateral lateral ventricle, and in some cases dilatation of the contralateral ventricle due to obstruction of Monro's foramen. It is important to recognize that overt ventricular dilatation may be absent when hydrocephalus coexists with cerebral oedema. Indeed, the presence of normal sized ventricles in the context of intracranial hypertension (demonstrated by intracranial pressure monitoring) should suggest the possibility of coexisting hydrocephalus and trigger the consideration of cerebrospinal fluid drainage as a means of therapy. Magnetic resonance imaging may provide better definition of underlying pathology, particularly in the posterior fossa, and its multiplanar capability may provide a better appreciation of the extent of space-occupying lesions. Modern imaging methods can also detect patients who may have relatively normal intracranial pressure, but are at high risk of severe intracranial hypertension. For example, patients with a middle cerebral artery (MCA) territory infarction are at high risk of severe brain swelling if more than 50% of the MCA territory is hypodense.

Lumbar puncture

A lumbar puncture offers the opportunity to directly measure cerebrospinal fluid pressure, and can be the defining investigation in meningitis, subarachnoid haemorrhage, or benign intracranial hypertension. However, in the context of clinical features that suggest intracranial hypertension, a lumbar puncture must be preceded by CT, and avoided if the basal cisterns are effaced by cerebral oedema. Removal of cerebrospinal fluid from the lumbar subarachnoid space under these circumstances can markedly increase the pressure differential between the infratentorial and supratentorial compartments, or the intracranial and spinal compartments, and precipitate transtentorial or cerebellar herniation, respectively. At the extreme it can be fatal.

Monitoring intracranial pressure

The clinical evaluation of intracranial hypertension is difficult due to its nonspecific clinical picture and phasic variations. Management may therefore be greatly facilitated by direct monitoring of intracranial pressure using intraparenchymal or ventricular monitoring devices. Such monitoring is most commonly used in severe intracranial hypertension and in sedated or deeply unconscious patients, in whom changes in clinical signs do not provide an alternative means of assessing progress and response to therapy. Although authoritative guidelines recommend such an approach, the one randomized clinical trial that evaluated intracranial pressure monitoring in traumatic brain injury found no outcome benefit when compared to a protocol based on clinical evaluation and serial imaging. This trial was conducted in South America, as intracranial pressure monitoring in patients with moderate to severe traumatic brain injury is considered a standard of care in many high-income countries.

Strategies for therapy

Management focuses on four areas, which are described next.

Monitoring progression of disease and response to therapy

Monitoring will depend on the clinical context. Repeated clinical examination with regular charting of the Glasgow Coma Scale may suffice in many cases. Patients with benign intracranial hypertension may require regular visual field assessment, while those with traumatic brain injury, intracranial haemorrhage, or severe cerebral oedema may benefit from direct intracranial pressure monitoring. The value of intracranial

pressure monitoring may be substantially enhanced by the use of other monitoring modalities such as brain tissue oximetry. Maintenance of stable physiology and removal of precipitating factors Hyponatraemia and low plasma osmolality will tend to worsen cerebral oedema by favouring water entry into the brain, and should be vigorously corrected. Maintenance of cerebral perfusion pressure with fluid resuscitation and vasoactive agents will prevent cerebral ischaemia. Comatose patients should have arterial blood gas analysis, and intubation and ventilatory support provided if airway protection is required or gas exchange is impaired. While hyperventilation has been widely used to control intracranial pressure in the past, there is increasing concern regarding the induction of critical cerebral ischaemia by hypocapnic vasoconstriction.

Section 17 Critical care medicine 3896 Current recommendations suggest that near normal P_{aCO_2} levels (4.5–5 kPa) should be maintained, with moderate hyperventilation (P_{aCO_2} 4.0–4.5 kPa) guided by brain tissue or jugular bulb oximetry; and more profound reductions in P_{aCO_2} reserved for control of acute episodes of severe intracranial hypertension, evidenced by monitoring or clinical signs (such as pupillary dilatation). Intensive glucose control targeting a blood glucose concentration of 4.5–6.0 mmol/litre significantly increases the risk of inducing moderate and severe hypoglycaemia, but the effects on clinical outcomes are currently unclear. Most current recommendations are to treat blood glucose when it is greater than 10 mmol/litre, with a target range of 6.0–10.0 mmol/litre. Attention should also be paid to treating epilepsy and significant fever, both of which can precipitate rises in intracranial pressure, and to discontinuing or mitigating the cardiorespiratory effects of drugs such as opioids, which may be responsible for physiological derangements that precipitate intracranial pressure elevation. Treatment of the underlying condition Early neurosurgical evaluation and operative therapy may be life-saving if a patient has an acute intracranial haematoma, a large tumour, or established hydrocephalus. Specific antimicrobial therapy may be required for meningitis, encephalitis, or brain abscess. Systemic hypertension commonly accompanies intracranial hypertension, and should generally not be treated because it may be needed to preserve cerebral perfusion. If therapy is needed for extreme hypertension or for hypertensive encephalopathy, then it is best to avoid nitric oxide donors such as nitrates or sodium nitroprusside, which can cause cerebral vasodilatation and further increase intracranial pressure. Table 17.7.1 Treatment of intracranial hypertension CPP augmentation (by increasing MAP) Maintenance of CPP >60–70 mm Hg prevents ischaemia, and further increases (90–100 mm Hg) may reduce ICP by autoregulatory cerebral vasoconstriction. Efficacy demonstrated in traumatic brain injury. Corticosteroids Reduce vasogenic oedema by restoring BBB integrity. Particularly effective in peri-tumoural oedema and benign intracranial hypertension. No outcome benefit in trauma. Prophylactic use may reduce incidence of hydrocephalus and other sequelae in tuberculous and acute bacterial meningitis. Diuretics Furosemide used to potentiate mannitol. Acetazolamide and thiazide diuretics used in benign intracranial hypertension. Osmotic agents Mannitol is effective in emergencies and can be used repeatedly if effective and plasma osmolality ≤ 325 mOsm/litre. Hypertonic NaCl (3–30%) may reduce ICP either as first line agent or when mannitol is ineffective and tends to cause less problems with major fluid shifts. Hyperosmotic agents may be less effective when there is widespread disruption of the blood-brain barrier. Reduction of cerebral blood volume Sedation and treatment of seizures can produce reductions in CBF and CBV that are coupled to reduction of neuronal metabolism. Hyperventilation has been commonly used to reduce CBV by inducing cerebral vasoconstriction, but can produce critical reductions in CBF. Needs to be used with care and with monitoring of cerebral oxygenation (usually with brain tissue or jugular bulb oximetry). Hypothermia Mild to moderate hypothermia (32–36°C)

is neuroprotective in experimental models, but clinically unproven. The neuroprotective benefit of hypothermia following cardiac arrest has been challenged by more recent data that show no benefit when compared to avoidance of hyperthermia. Hypothermia is effective at controlling refractory intracranial hypertension by multiple mechanisms, including metabolic suppression and anti-inflammatory effects, but outcome benefits have not been demonstrated. Indeed, when used early as an ICP lowering intervention in TBI, it may result in worse outcomes. CSF drainage Ventriculostomy provides emergency drainage of CSF in trauma, acute hydrocephalus (subarachnoid haemorrhage, tumours). Ventriculoperitoneal, ventriculoatrial, and lumboperitoneal shunts provide chronic CSF diversion in idiopathic or secondary hydrocephalus. Endoscopic third ventriculostomy provides communication between ventricular and cisternal CSF in noncommunicating hydrocephalus. May remove the need for shunts and the associated risk of shunt malfunction and sepsis. Surgical decompression In TBI, early decompressive craniectomy worsens outcome in the absence of severe intracranial hypertension; used for the treatment of refractory intracranial hypertension it reduces mortality, but increases the number of survivors with severe disability or in a vegetative state. Decompressive craniectomy improves survival, and probably functional outcome, in 'malignant' MCA stroke with severe cerebral oedema. In traumatic brain injury, early decompression results in worse functional outcomes. When used in refractory intracranial hypertension, it can increase survival and the proportion of survivors at least independent at home, but with some increase in severely disabled survival. Optic nerve decompression may prevent visual deterioration in benign intracranial hypertension. BBB, blood-brain barrier; CBF, cerebral blood flow; CBV, cerebral blood volume; CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; ICP, intracranial pressure; MAP, mean arterial pressure; MCA, middle cerebral artery; TBI, traumatic brain injury

Fig. 17.7.5 Management of the unconscious patient with intracranial hypertension. CPP, cerebral perfusion pressure; MAP, mean arterial pressure.

17.7 Management of raised intracranial pressure 3897 Specific treatment of intracranial hypertension Several therapies can be used to reduce intracranial pressure, and their application will depend on the cause and severity of intracranial pressure elevation. Commonly used interventions and their indications are outlined in Table 17.7.1, but it must be pointed out that few of these have been assessed by good quality outcome studies. Treatment pathways for the emergency management of an unconscious patient with suspected intracranial hypertension are outlined in Fig. 17.7.5. **FURTHER READING** Andrews PJ, et al. (2015). Eurotherm3235 Trial Collaborators: hypothermia for intracranial hypertension after traumatic brain injury. *N Engl J Med*, 374, 1385. Brain Trauma Foundation (2016). Guidelines for the Management of Severe Traumatic Brain Injury, 4th edition. https://braintrauma.org/uploads/03/12/Guidelines_for_Management_of_Severe_TBI_4th_Edition.pdf Chesnut RM, et al. (2012). A trial of intracranial-pressure monitoring in traumatic brain injury. *N Engl J Med*, 367, 2471–81. Cooper DJ, et al. (2011). Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med*, 364, 1493–502. Hofmeijer J, et al. (2009). Surgical decompression for space-occupying cerebral infarction (the Hemicraniectomy After Middle Cerebral Artery infarction with Life-threatening Edema Trial [HAMLET]): a multicentre, open, randomised trial. *Lancet Neurol*, 8, 326–33. Hutchinson PJ, et al. (2016). Trial of decompressive craniectomy for traumatic intracranial hypertension. *N Engl J Med* 2016, 375, 1119. Menon DK, Ercole A. (2017). Critical care management of traumatic brain injury. *Handb Clin Neurol*, 140, 239. Posner JB, et al. (eds) (2007). Plum and Posner's diagnosis of stupor and coma, 4th edition. Oxford University Press, Oxford. Reilly PL (2005). Management of intracranial pressure

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