INTRACRANIAL PRESSURE Intracranial pressure and ce

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The brain depends on continuous perfusion for oxygen and glucose delivery , and hence survival. Normal cerebral blood flow (CBF) is about 55 /uni00A0 mL/min for every 100 /uni00A0 g of brain tissue. Ischaemia results when this rate drops below 20 /uni00A0 mL/min, and even lower levels will result in infarction unless promptly corrected. † The learning objectives of this chapter are aligned with the Intercollegiate Surgical Curriculum Programme (ISCP) ST3 Neurosurgery Knowledge Requirements in Cranial Trauma and comprise: • the resuscitation, assessment, investigation and continuing care of head-injured patients • the prevention and detection of secondary intracranial and systemic insults. Alexander Monr o (secundus) , 1733–1817, Scottish anatomist, physician and medical educator. George Kellie , 1770–1829, Scottish surgeon and pupil of Alexander Monro (secundus). Flow depends on cerebral perfusion pressure (CPP), which is the di ff erence between the mean arterial pressure (MAP) and the intracranial pressure (ICP): CPP (75–105 /uni00A0 mmHg) = MAP (90–110 /uni00A0 mmHg) – ICP (5–15 /uni00A0 mmHg) Typical normal values are given in parentheses. In fact, in - the normal brain, variations in vascular tone maintain a con - stant CBF across a range of MAP between 50 and 150 /uni00A0 mmHg (or higher in the setting of chronic hypertension) and a cor - responding range of CPP – the process of cerebral auto- regulation . Autoregulation can be impaired in the context of trauma, so that MAP and ICP must be actively regulated in these patients to maintain proper perfusion. The Monro–Kellie doctrine and herniation syndromes Alexander Monro observed in 1783 that the cranium is a ‘rigid box’ containing a ‘nearly incompressible brain’. Any expansion in the contents, especially haematoma and brain swelling, may be initially accommodated by exclusion of fluid components, venous blood and cerebrospinal fluid (CSF). Further expansion is associated with an exponential rise in ICP (Figure 28.1). Uncontrolled increases in ICP result in cerebral herniation (Figure 28.2). Typically , herniation of the uncus of the tem - poral lobe over the tentorium results in pupil abnormalities (see Pupils), usually occurring first on the side of any expand - ing haematoma. Cerebellar tonsillar herniation through the foramen magnum compresses medullary vasomotor and

The resuscitation, assessment, investigation and • continuing care of head-injured patients The prevention and detection of secondary intracranial • and systemic insults

(b) respiratory centres, classically producing Cushing’s triad hypertension, bradycardia and irregular respiration. The patient is then said to be ‘ coning ’, and brainstem death will result without immediate intervention. Summary box 28.1 Intracranial pressure /uni25CF /uni25CF Harvey Williams Cushing , 1869–1939, Professor of Surgery , Harvard University Medical School, Boston, MA, USA, considered the founding father of modern neurosurgery .

Normal Venous Arterial Brain CSF blood blood Skull Mass lesion – compensation phase Ve nous Arterial Brain CSF Mass blood blood Skull Mass lesion – brain herniation Arterial Brain CSF Mass blood blood Venous Skull 90 80 Brain 70 herniation 60 50 Point of 40 Compensation decompensation 30 20 Intracranial pressure (mmHg) 10 0 10 20 30 40 50 60 70 80 90 100 110 120 Mass lesion size (arbitrary units) Figure 28.1 The Monro–Kellie doctrine accounts for the ability of the intracranial compartment to

accommodate expanding mass lesions, primarily by excluding venous blood and cerebrospinal /f_l uid (CSF), and the rapid rise in pressure associated with exhaustion of this compensation. A continuous supply of oxygenated blood is essential for brain survival Raised ICP can compromise cerebral perfusion, resulting in a cycle of secondary brain injury and swelling 1 2 4 3 5 Figure 28.2 Brain herniation. (1) Subfalcine herniation – the cingulate gyrus is herniating under the falx cerebri. (2) Midline shift is evident. (3)

/uni00A0 Uncal herniation – the temporal lobe is herniating over the ten

torium cerebelli, where it can compress the third nerve. (4) Central herniation and (5) tonsillar herniation result in brainstem compromise, manifesting as Cushing’s triad.

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