

ICP monitoring

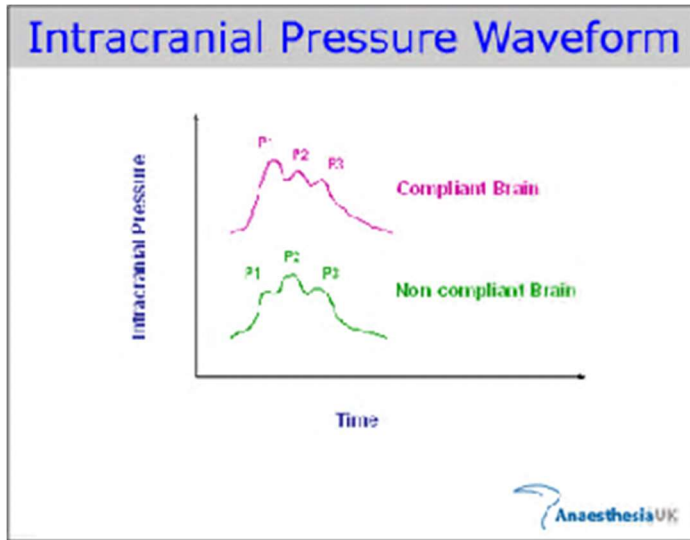
P1 systolic contraction, systole

P2 cerebral compliance, correlates with AV closure

P3 or part of P2 – CHECK -- dichrotic notch

P3 tidal wave, corrs w/ anterograde flow during diastole

Cerebral bld flow incs in resp to hypoxia and causes profound elev in ICP.



$P2 > P1$ as \downarrow intracranial compliance

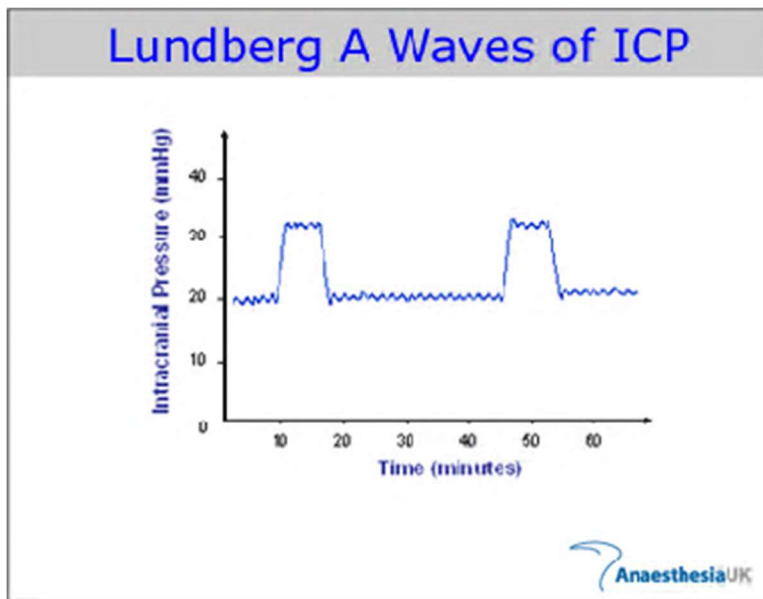
Lundberg waves

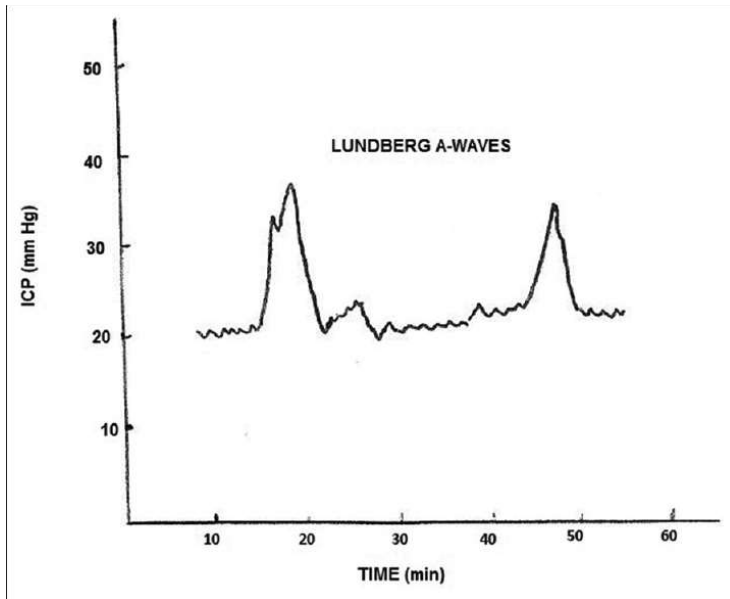
look at chg in ICP over prolonged time

A-waves steep incs in ICP for 5-10 min at a time, always pathological, usually represent early brain hern

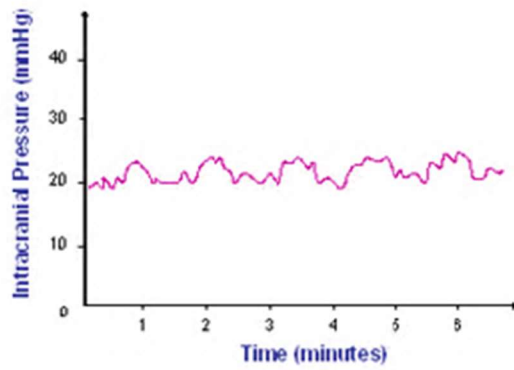
B-waves slow oscillations q 1-2 min rep pot vasospasm

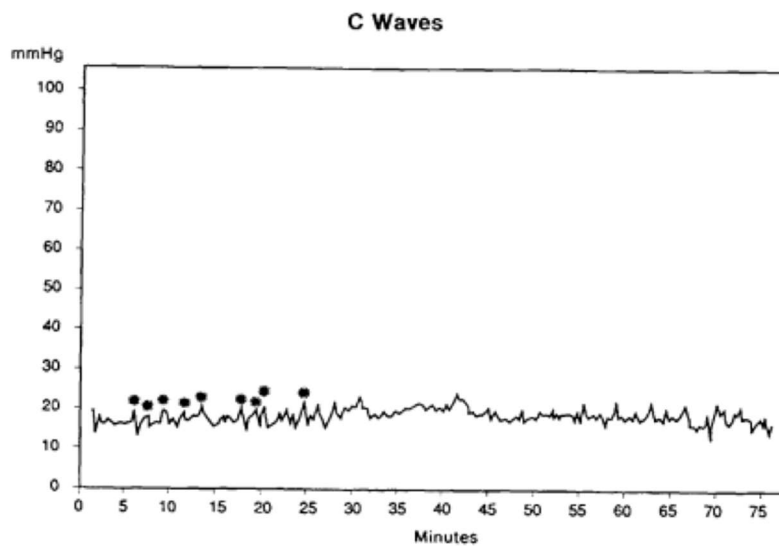
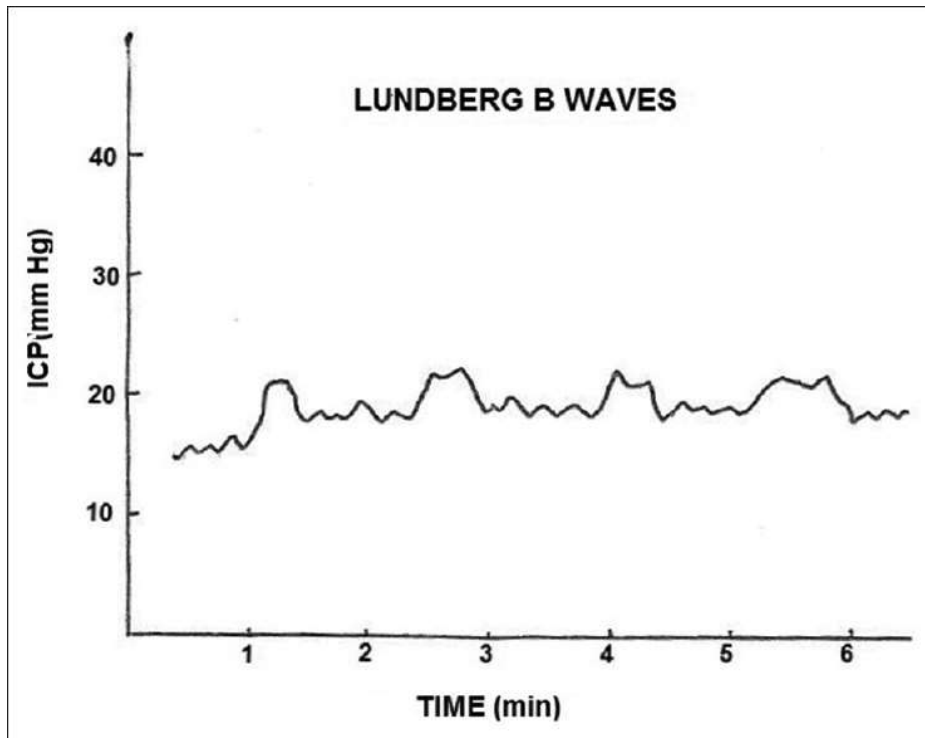
C-waves physiologically nl fast oscills, 4-8x/min



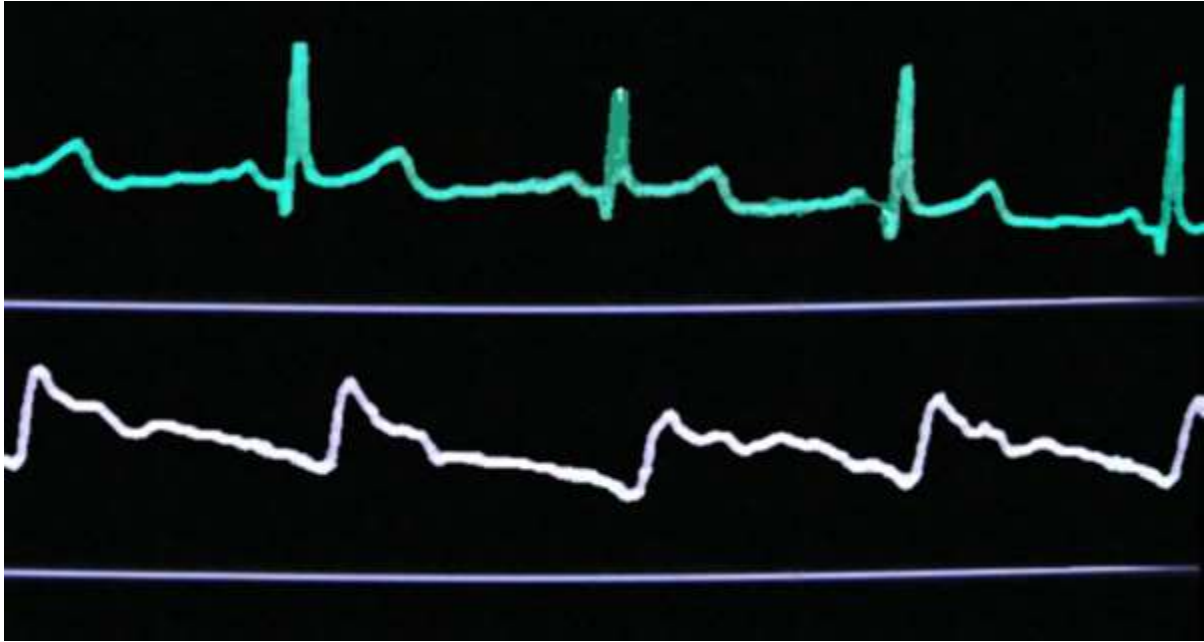


Lundberg B Waves of ICP

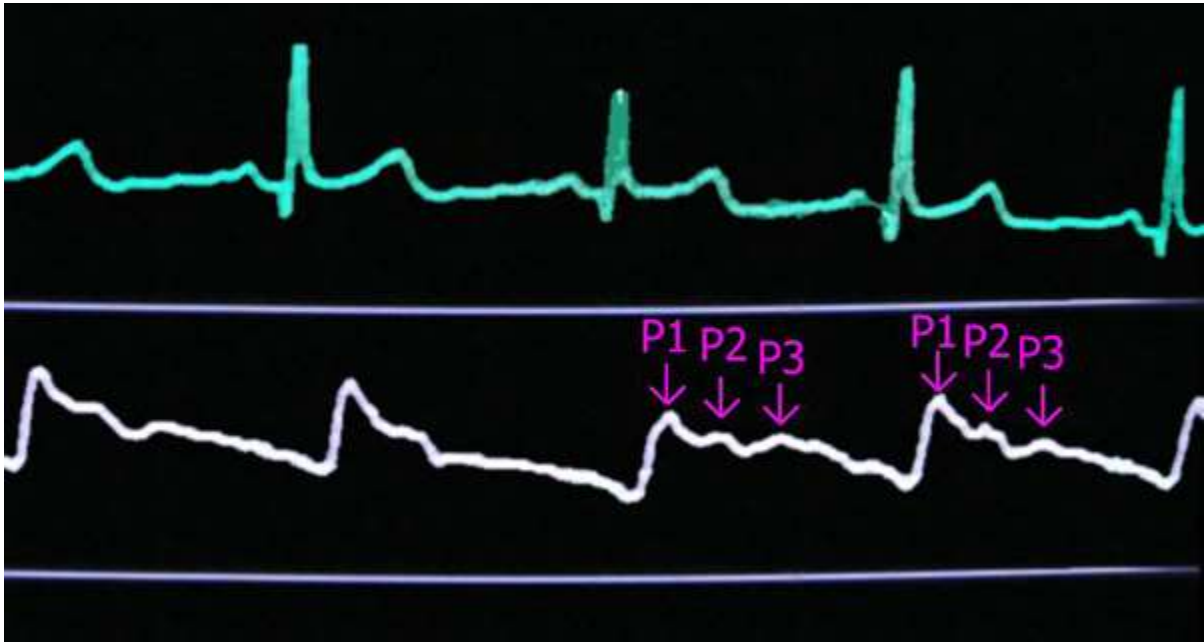




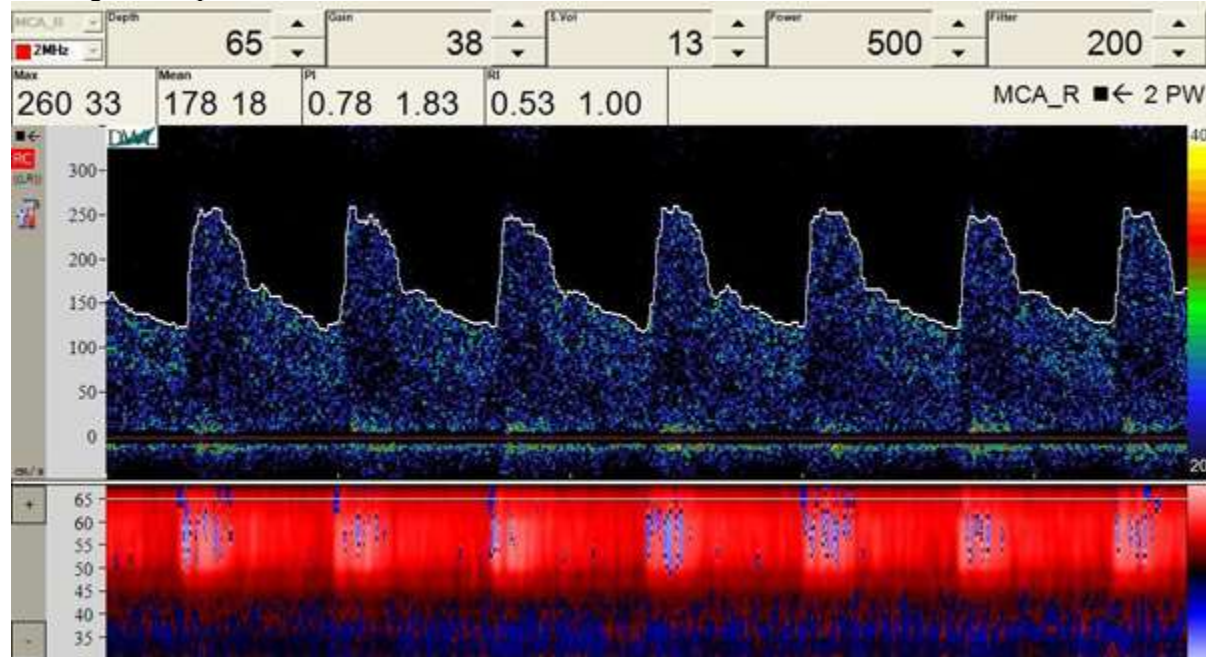
The white waveform shown is of the ICP waveform of a patient with a severe traumatic brain injury. The pressure at this time was 14mmHg. The aim of therapy was <20mmHg. Give 2 reasons why you think the reading is likely to be an accurate reflection of intracranial pressure.



A normal non-damped waveform is seen with a clearly visible P1, P2 and P3. If intracranial compliance was poor a prominent P2 would be expected.



A 36 year old patient is day 5 of his admission to the ICU following a witnessed collapse. The CT brain showed a Fischer grade 3 subarachnoid hemorrhage. His level of consciousness deteriorated from withdrawing from a central noxious stimulus, to extension to the same stimulus. His pupils remain equal and reactive to light. His EVD does not appear to have increased in its output rate. The following investigation is performed.



- What is this investigation?
- What does it demonstrate?
- List 3 management options.