

Technique	Variable Measured	Major Indications	Disadvantages
Whole-brain monitoring			
Serial neurologic examination (15)	Qualitative assessment of brain function	All neurocritically ill patients	Constricted by pharmacologic interventions; operator dependent; qualitative
Intracranial pressure devices (16, 17)	Intracranial pressure; cerebral perfusion pressure	Coma and abnormal head CT; cerebral edema; midline shift; acute hydrocephalus	Invasive; ICH (<3%); infection (<14%); malfunction
Internal jugular bulb catheters (18, 19)	Oxygen saturation of venous return from the brain; cerebral oxygen extraction can be derived	Severe traumatic brain injury; diffuse cerebral edema	Line sepsis; venous thrombosis; carotid puncture; does not measure regional oxygenation
Continuous electroencephalography (20)	Brain electrical activity with close relationship to cerebral metabolic rate	Status epilepticus; altered level of consciousness to detect nonconvulsive status epilepticus; pentobarbital coma	Qualitative evaluation; frequent artifacts; interference with head imaging studies
Regional/focal brain monitoring			
Transcranial Doppler ultrasound (21, 22)	Cerebral blood flow velocity	Monitoring of cerebral vasospasm; vessel recanalization after thrombolytic therapy; confirmation of brain death	Operator dependent; may be limited by cranial anatomy
Near-infrared spectroscopy (23, 24)	Cerebral tissue oxymetry	Severe traumatic brain injury	Sensitivity to extraneous light; motion artifact; signal drift
Xenon-133 clearance (25, 26)	Regional cerebral blood flow	Severe traumatic brain injury; subarachnoid hemorrhage	Measures superficial blood flow; unreliable with abnormal blood-brain partition coefficient
Laser-Doppler flowmetry (27)	Qualitative regional cerebral blood flow	Under study for severe traumatic brain injury and massive cerebral edema	Probe malfunction requiring replacement
Thermal diffusion flowmetry (28)	Regional cerebral cortical blood flow	Under study for severe traumatic brain injury and massive cerebral edema	Infection (low risk); signal distortion; small region monitored
Microdialysis catheters (29, 30)	Various substances in the extracellular space	Severe traumatic brain injury; subarachnoid hemorrhage	Infection (low risk); may not detect all ischemic regions
Brain tissue probes (31, 32)	Regional brain P_{O_2} , P_{CO_2} , pH, and temperature	Severe traumatic brain injury; subarachnoid hemorrhage; diffuse cerebral edema	ICH and infection (low risk)

ICH, intracerebral hemorrhage; CT, computed tomography.

acute ischaemic stroke:

- (i) rt-PA:
 - rt-PA within 3 hrs of symptom onset can result in clinical and statistical significant improvement
 - at least 30% of treated patients will be completely independent 3 months after treatment.
- (ii) intraarterial thrombolysis:
 - may increase the window treatment to 6 hrs
 - may be used as rescue therapy after intravenous thrombolysis
- (iii) ultrasound-enhanced systemic thrombolysis
 - uses transcranial doppler to enhance thrombolysis

intracerebral haemorrhage

- One of the major discoveries has been the realisation that intraparenchymal hematomas grow in about 38% of patients within 3hrs of onset . Such growth may result in increased mortality.
- (i) recombinant factor VIIa
 - The most promising intervention to limit hematoma growth with resulting improved mortality and functional outcome thus far has been the administration of recombinant activated factor VIIa.
- (ii) Surgical evacuation
 - Surgical evacuation of intracerebral hematomas within 24 hrs has not shown benefit when compared with initial conservative treatment
 - The one group of patients who may benefit from surgery with improved functional outcome is that with cerebellar hematomas.
- (iii) Blood pressure management:
 - On the one hand, there is the risk of increasing the size of the hematoma if blood pressure remains elevated, and on the other, there may be the theoretical risk of causing cerebral ischemia if blood pressure is reduced.
 - A prospective study is under way to evaluate the optimal blood pressure control level in these patients.

subarachnoid haemorrhage:

- (i) cerebral vasospasm treatments:
 - Cerebral vasospasm is a frequent cause of cerebral ischemia after subarachnoid hemorrhage.
 - Calcium antagonists, particularly nimodipine and possibly magnesium, reduce the risk of poor outcome and cerebral ischemia.
 - volume expansion has been commonly used under the assumption that hypovolemia is related to cerebral ischemia. However, convincing evidence of its benefit is lacking.
- (ii) hypothermia:
 - The use of intraoperative (i.e., during aneurysm clipping) hypothermia does not improve clinical outcome
- (iii) coiling vs clipping:
 - randomized trial revealed that in patients with ruptured cerebral aneurysms, for which both endovascular coiling or surgical clipping are treatment options, the outcome in terms of disability at 1 yr is better for patients undergoing endovascular coiling
- (iv) hypoxic ischaemic insult:
 - Evidence from randomized controlled trials has demonstrated that institution of mild-to-moderate hypothermia results in improved survival and functional outcome of these patients.
- (v) traumatic brain injury:
 - patients admitted to specialized trauma centers are more likely to experience a reduced hospital length of stay and mortality, with improved functional outcome.
 - aggressive management of hypotension in the prehospital setting is important. A recent randomized clinical trial of aggressive fluid management in the prehospital setting revealed decreased mortality, most likely related to avoidance of hypotension.
 - induced hypothermia has also been applied to patients with severe traumatic brain injury. Studies have revealed that induced hypothermia may confer benefit particularly to those patients with elevated intracranial pressure. However, the routine use of this treatment remains controversial.
- (vi) treatment of fever:
 - Elevated core body and brain temperature is associated with worsening neurologic injury and functional outcome of critically ill neurologic patients, regardless of the type of injury. Such association is very important because fever is a frequent occurrence in neurologic patients while in the ICU.
 - What remains to be answered is whether effective fever reduction results in significantly improved functional outcome and mortality rates in these patients.

monitoring

neurocritical care
[created by Paul Young 02/10/07]

therapy

general

- The birth of neurocritical care stemmed from the appreciation that an already affected brain (primary injury) is greatly influenced by systemic alterations that may adversely affect its function (secondary injury).

outcome measures

Indicator	Type of Scale/Outcome	Advantages	Disadvantages
Mortality (4-7)	General	Easy to obtain	Lack of indication of quality of life of survivors; greatly influenced by discharge practices
Length of stay (4-7)	General	Easy to obtain; surrogate for resource utilization	Crude measure of resource utilization; greatly influenced by discharge practices
APACHE Score (8)	Severity of illness	Widely used in critical care; risk adjustment	Neurologic information is limited to GCS
GCS (9)	Disease specific; measures level of consciousness	Easy to obtain; good interobserver and intraobserver reliability	Crude measure of neurologic injury
COS (10)	Disability scale	Easy to obtain; good interobserver and intraobserver reliability	Crude measure of disability
Barthel index (11)	Functional scale; measures ability to perform activities of daily living	Easy to obtain; good interobserver and intraobserver reliability	Lack of measurement of quality of life
Rankin Scale Score (12)	Disability scale	Easy to obtain; good interobserver and intraobserver reliability	Lack of measurement of quality of life
SF-36 (13)	Multidimensional health status	Attempts to measure quality of life	Floor effect in the physical function and role domains in neurologic patients
NHSS (14)	Disease specific; measures stroke severity	Easy to obtain; good interobserver and intraobserver reliability	It applies to stroke patients only

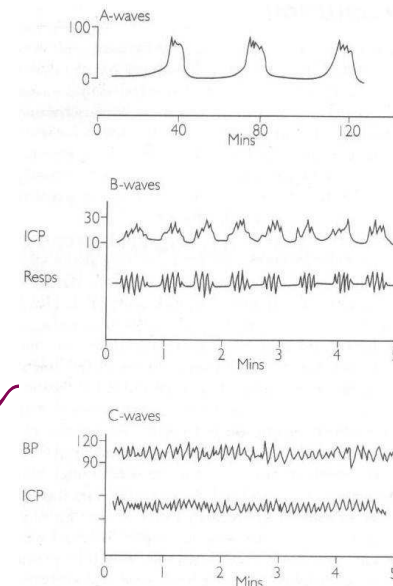


fig. 43.3 ICP waveforms. A-waves are plateau waves of 0–100mmHg, sustained for 5–15 minutes. Associated with raised ICP and compromised CBF. B-waves are small changes in pressure every 0.5–2 minutes, often associated with breathing patterns and possibly due to local variations in the arterial pressure of oxygen and carbon dioxide. C-waves are low amplitude oscillations with a frequency of about 5 per minute. Associated with variation in vasomotor tone.