Essentials of neuromonitoring

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The essentiality of neuromonitoring lies in the fact that the outcome of the surgery depends on the basic pathophysiological disease process and effect of anaesthesia and surgical stress on the disease process and on the cerebral perfusion pressure, regionally and globally. Anaesthesia for neurosurgery and spine surgery requires the standard ASA monitoring for physiologic parameters. These parameters may not give the overall impression of wellbeing of the patient. Therefore, specific neuromonitoring may play a role to understand the integrity of the CNS that may specifically detect CNS function, perfusion or metabolism.

The ideal neuromonitoring technique preferred would be:

1. Simple technique
2. Bedside monitor, applicable both in ot and icu settings
3. Non-invasiveness
4. Easy to interpret
5. Should truly reflect the degree of injury to the nervous system
6. Able to monitor therapeutic interventions and
7. Able to provide online feedback

But there is no single technique which reflects all the ideal properties. Neuromonitoring techniques currently used for varied indications can be categorized as:

1. Routine monitoring
2. Electroencephalography And Cerebral Function Monitoring
3. Electromyography and cranial nerve monitoring
4. Evoked potentials
5. Monitoring Cerebral Perfusion
6. **Monitoring cerebral oxygenation**
7. Evaluation of Cerebral metabolic parameters – Micordialysis
8. Multimodality monitoring

ROUTINE PHYSIOLOGIC MONITORING

Bradycardia rather than tachycardia is an ominous signal in many neurosurgical conditions.[1] This occurs in conjunction with Cushings reflex, most commonly seen in conditions with increased ICP.

The causes of bradycardia include

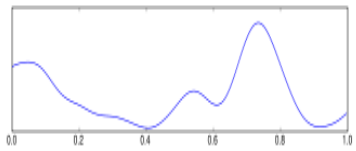
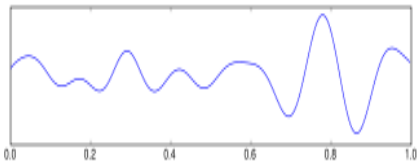
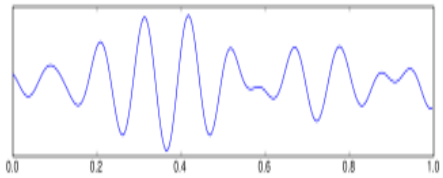
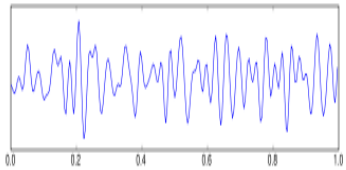
- space occupying lesion involving or compressing the brain parenchyma (subdural haematoma, tumours, hydrocephalus),
- neurosurgical procedures (neuroendoscopy, placement of extradural drains),

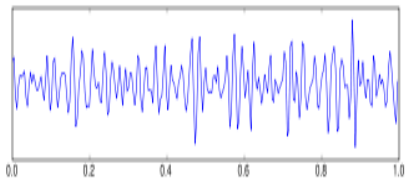
- epileptic and non-epileptic seizures,
- trigemino-cardiac reflex,
- cerebellar lesions,
- spinal lesions (neurogenic shock, autonomic dysreflexia) and
- many other rare causes (Ventricular catheter obstruction in cases of hydrocephalus, colloid cysts related acute neurogenic cardiac dysfunction, Ondine's curse syndrome, etc.).

Brain stem compression or stretching is a serious consequence. It usually presents either as bradycardia or tachycardia, associated with increased blood pressure. These patients may finally rest in asystole. On the other hand, tachyarrhythmias are common in patients with subarachnoid haemorrhage. Another important parameter is $EtCO_2$ monitoring which is an early and useful physiologic tool in the diagnosis of venous air embolism.

ELECTROENCEPHALOGRAPHY AND CEREBRAL FUNCTION MONITORING

The electrical activity of the brain comes from axon currents. This produces voltage differences between different places on the brain surface or scalp. The electroencephalogram is a collection of voltage-time tracings obtained from 10 or 20 electrodes at specific locations on the scalp. Frequency analysis of brain electrical signals reveals that on the scalp, one detects signals in the range of 0.5 to 30 Hz.

Frequency range (in Hz)		Description of the waves	Characteristics
0.5 to 4		Delta waves, δ	Slow wave sleep
4 to 7		Theta waves, θ	Drowsiness
8 to 13		Alpha waves, α	Quiet wakefulness
13 to 30		Beta waves, β	Intense mental activity

> 30		Gamma waves, γ	Sudden sensory stimuli

ELECTROCORTICOGRAM Intraoperatively electrodes can be placed on the surface of the cortex to obtain an electrocorticogram. These recordings help identify the origin of the epileptic foci and their relationship to cortical areas that direct speech and motor activity.

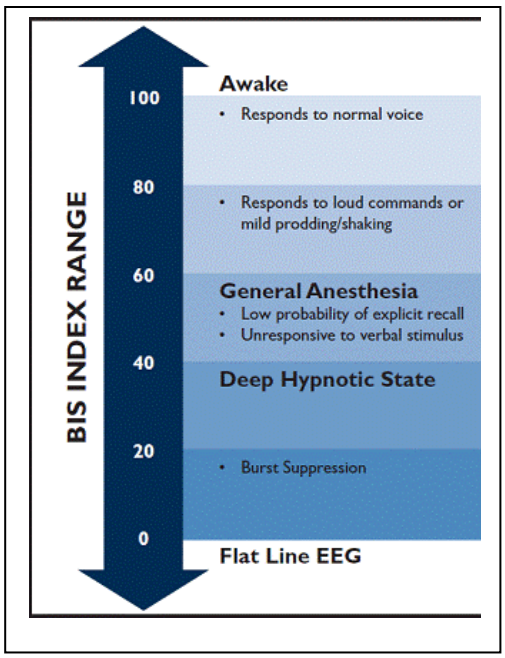
Modern digital signal processing and a reduced scalp montage have been used to derive indices that correlate with "depth of anesthesia." Depth of Anaesthesia monitors use three electrodes that are usually placed in the frontal region.[2, 3] Two electrodes provide a differential amplifier with the voltage difference between their locations, whereas the third electrode provides an obligatory reference signal. Brain monitors digitally record voltage tracings for fixed times known as epochs, typically seconds. Microprocessors in the monitors are used to process the tracings at the end of each epoch and display the analyzed data on a time scale of seconds. Figure displays the different states of anaesthesia with the values and tracings of brain monitors.

INDICATIONS FOR EEG MONITORING

During anaesthesia	1.Carotid endarterectomy 2. Cardioplmonary bypass procedures 3. Cerebrovascular surgery a. aneurysm surgery involving temporary clipping b. vascular bypass procedures 4. When burst suppression is desired for cerebral protection
In the ICU	1.Barbiturate coma for patients with traumatic brain injury 2.when subclinical seizures are suspected 3.determination of brain death (isoelectric flat EEG run twice 24 hrs apart)

BISPECTRAL ANALYSIS

This is based on Fourier analysis, a computational approach that permits the calculation of a mathematically complex index of wave synchrony known as interfrequency phase coherence. Two waves of the same frequency are in phase with each other when their maxima and minima occur simultaneously. When such repeated phase relationships persist, BIS analysis produces an interfrequency phase coherence of 100%.If a brain is having consistent signal repetitions, be it from pain, thinking, or other sensations, one must be concerned that the anesthetic effects are not dominant and the brain in question may not be adequately anaesthetized. The above concepts outline the basis for BIS monitoring.[4]



ENTROPY ANALYSIS

This is based on frequency and amplitude information in the EEG.[5, 6]

After Fourier analysis, computations determine the plot of EEG power versus frequency, which is then divided into N bins, with each bin given a probability value pi that is used for calculating spectral and response entropy:

Spectral entropy – where analyzed frequencies are in the range of 0.8 to to 32 Hz, thereby emphasizing pure brain electrical signals.

Response entropy – where analyzed frequencies are in the range of 0.8 to 42 Hz, with the frequency region being above 32 Hz, including EMG components from scalp and frontal muscle activity.

For both BIS and RE, a number between 1 and 100 is calculated whereas 91 is the maximum value for SE.

NARCOTREND

The Narcotrend algorithm is based on pattern recognition of the raw electroencephalogram (EEG) and classifies the EEG traces into different stages from A (awake) to F (increasing burst suppression down to electrical silence) and is divided into 14 substages (A, B0-2, C0-2, D0-2, E0,1, F0,1). The newest Narcotrend software version includes a dimensionless Narcotrend index from 100 (awake) to 0 (electrical silence). The Narcotrend monitor provides a vast amount of information: the actual Narcotrend stage and index, the trend (‘cerebrogram’), the raw EEG signal and a power spectrum and several derived EEG parameters.

THE PATIENT STATE INDEX (PSI)

The PSI algorithm relies on 4-channel EEG providing information on power, frequency and phase from anterior–posterior relationships of the brain as well as coherence between bilateral brain regions. The referential and bipolar combinations of the four channels used in PSI allow enhanced capabilities to detect subtle regional differences that cannot be captured in a single channel system.[7]

ELECTROMYOGRAPHY AND CRANIAL NERVE MONITORING

Spontaneous Electromyography. The technique of Electromyography allows continuous assessment of the integrity of cranial and peripheral motor nerves. Impingement on a nerve root by an instrument will cause immediate motor activity that is spontaneously detectable, thus guiding the surgeon to modify the technique.[8] Surgery in the posterior cranial fossa and adjacent to the brainstem places the surgeon in close proximity to the cranial nerves. Generally, only the integrity of nerves with motor components can be detected, either through spontaneous EMG or through EMG evoked by local electrical stimulation. These include cranial nerves V, VII, IX, XI and XII.

This is commonly used to monitor facial nerve while resecting tumours like acoustic neuroma, also can be used to monitor other cranial nerve integrity.[9] It is frequently used to detect injury to the spinal cord and spinal nerve roots during cervical and lumbar spine surgery where the brachial plexus and lumbosacral plexus are encountered. It is not affected by anaesthetics, but muscle relaxants should be avoided.

EVOKED POTENTIALS

This technique of monitoring involves elicitation of a response following application of a specific stimulus. Sensory evoked potentials may be elicited after various types of sensory input: somatosensory (SSEP), visual (VEP), or auditory (brainstem auditory [BAEP]). Motor evoked potentials (MEPs) detects signal in the spinal cord, peripheral nerves and muscles following the result of specific stimulation of the motor cortex. Any neurologic injury typically leads to prolonged patency and decreased amplitude. The common definition of significant change is a 50% drop in amplitude or a 10% increase in latency, or both. Evoked potentials are adversely affected by anesthetics in the following order: VEP> SSEP/MEP> BAEP. Inaccurate interpretation may be seen in conditions where physiologic alterations are expected such as hypotension, anaemia, hypoxia, and hypothermia. The anaesthetic agents and its effects on somatosensory and motor evoked potentials is tabulated below:

Relative effects of anaesthetic agents on somatosensory and motor evoked potentials

Agent	Cortical SSEPs		MEPs
	Latency	Amplitude	Amplitude
Volatile agents*	↑↑↑	↓↓↓	↓↓↓
Nitrous oxide	↑	↓↓	↓
Barbiturates*	↑↑	↓↓↓↓	↓↓

Propofol*	↑↑	↓↓	↓↓
Benzodiazepines	↑	↓	↓↓
Narcotics/opioids	+/-	+/-	+/-
Ketamine	↑	↑	+/-
Etomidate	↑	↑↑	↑
Muscle relaxants*	0	0	↓↓↓

*- the degree of response is highly dose dependent.

↑- mild increase, ↑↑- moderate increase, ↑↑↑- significant increase, ↓- mild decrease, ↓↓- moderate decrease, ↓↓↓- significant decrease

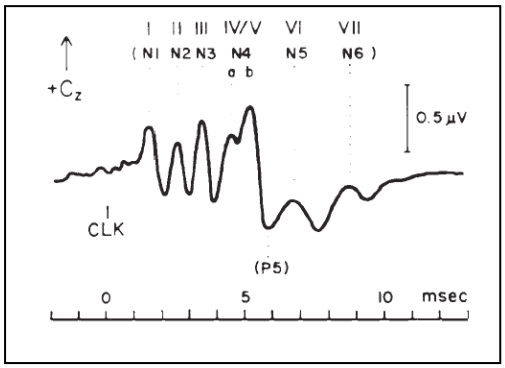
Visual evoked potentials: monitors the visual pathway from the eye through the optic nerve and chiasm to the visual cortex. By using special goggles or contact lenses, a series of bright LED lights are focused on to the eyes to stimulate the retina while potentials are recorded by the scalp electrodes.

Examples for VEP monitoring	
Pathway involved	Indication for surgery
Optic nerve and chiasm	Pituitary surgery
Visual cortex	Tumours in the occipital cortex Surgery involving the posterior circulation
Eye (ischemia)	Prone spine surgery

BRAINSTEM AUDITORY EVOKED POTENTIALS (BAEPs)

BAEPs infer the integrity of the auditory pathways, starting from the ear (tympanic membrane, ossicles) and including the nervous system structures such as hair cells, spiral ganglion, cranial nerve VIII, cochlear nuclei, superior olivary complex, lateral meniscus, inferior colliculi and medial geniculate thalamic nuclei.[10]

Technique: The stimulus is typically a series of standardized clicks produced from a transducer placed in the external auditory canal. The response is recorded from the electrodes placed over the scalp. Multiple signals (~2000) are averaged to yield a series of six or seven positive waves, named I to VI or VII, where the latency of each peak has significance with respect to the integrity of various parts of the auditory pathway.



INTERPRETATION

Middle ear or cochlear deficits	No waves recorded
Eighth nerve injury	All waves are affected except wave I
Cerebellar retraction	Prolongation of inter peak latency between waves I and V
Permanent damage to auditory tract	Disappearance of later waves
Compression of cranial nerves – V and VII Microvascular decompression Damage to auditory nerve Resection of acoustic neuromas	

Somatosensory evoked potentials (SSEPs)

SSEPs monitor the integrity of the sensory pathways, including peripheral nerves, the spinal cord, the brainstem, subcortical structures and the sensory cortex. A repetititve electrical stimulus is applied to a peripheral nerve and responses are measured over the cerebral cortex with scalp electrodes.[11]

Ischemia secondary to spinal distraction and disruption of perforating radicular vessels- Soine surgery	Spinal cord
Direct trauma Pedicle screw placement or other instrumentation Resection of pathologic lesion (proximate to the sensory tracts)	Spinal cord
Adequacy of perfusion to the cortex Intracranial surgery – aneurysm Extracranial surgery – carotid endarterectomy	Sensory Cortex (ACA – posterior tibial nerve stimulation; MCA- median nerve stimulation)

Motor evoked potentials (MEPs)

MEPs involve stimulation of the motor cortex to activate the motor pathways and elicit a movement response. With MEP, latency of the signal is unreliable, rather 50% decrease in amplitude is clinically significant.[12] Its use is still being evaluated for optimal benefit in detecting and preventing motor injury.

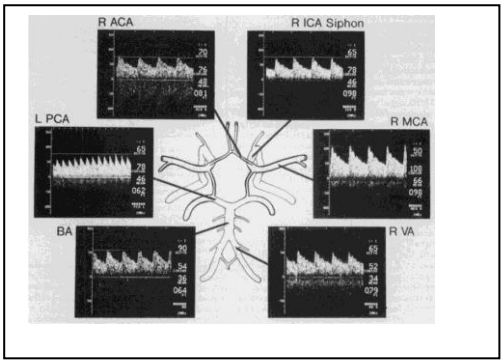
Examples for MEP monitoring	
Pathway involved	Indication for surgery
Descending motor pathway (spine)	Spine surgery
Motor cortex	Tumours adjacent to the motor cortex

MONITORING OF CEREBRAL PERFUSION

TRANSCRANIAL DOPPLER ULTRASONOGRAPHY

It is a real time noninvasive monitor that provides indirect information about cerebral blood flow. It evaluates the relative changes in flow through the large basal arteries of the brain (i.e., the circle of Willis).[13] It calculates the red cell flow velocity (FV) from the shift in frequency spectra of the Doppler signal and can be used both intraoperatively and in the icu. Changes in FV correlate closely with changes in CBF, provided that the angle of insonation (the angle between the axis of the vessel and the ultrasound beam) and the diameter of the vessel insonated remain constant.

Vessels insonated	Approach
Anterior, middle and cerebral arteries	Transtemporal approach
Carotid siphon	Trans orbital approach
Basilar and vertebral vessels	Suboccipital approach



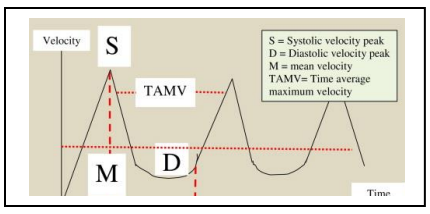
MCA is most commonly insonated because it is simple to detect and allows easy probe fixation, thereby providing a constant insonation angle. It can be used both in adult and pediatric patients.

Factors to ensure that changes in flow velocity reflect cerebral blood flow

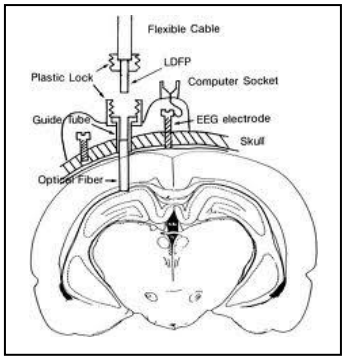
1. Minimal changes in arterial carbon dioxide tension and blood pressure
2. Able to insonate conductance vessels
3. The probe should be fixed in position
4. Steady state anaesthesia to be maintained

FVmean (weighted mean velocity) – most physiologic and best correlates with the actual CBF.

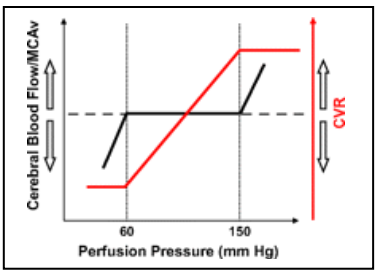
Waveform pulsatility – This is determined by the arterial blood pressure waveform, the viscoelastic properties of the cerebral vascular bed, and blood rheology. With normal



properties of the above parameters, the pulsatility of the waveform indicates distal cerebrovascular resistance. This is quantified as Pulsatility Index: PI or Gossling index = (FVsystemic – Fvdiastolic)/FVmean. Normal PI ranges from 0.6 to 1.1. Cerebral autoregulation - the static rate of autoregulation or the index of autoregulation (IOR) is the ratio of percent change in estimated cerebral vascular resistance (CVRe) to percent change in mean blood pressure. IOR = % change in CVRe/%change in MAP Value of 1 – perfect autoregulation; zero denotes complete disruption of autoregulation. Dynamic autoregulation (dRoR) – rate of restoration of FV (%/sec) with respect
to the drop in perfusion pressure.
Normal dRoR is 20%/sec (i.e., dynamic autoregulation is complete within approximately 5 seconds).
Clinical applications:
TCD allows assessment of cerebral vascular reserve by examining CO2 reactivity, detection of emboli, monitoring of cerebral perfusion during cross clamping of the carotid artery, and testing of autoregulation.
>40% of the preclamping value – absent ischemia
16 to 40% - mild ischemia
<15% - severe.



High FV indicates vasospasm or hyperemia. Vasospasm is present if the ratio between FVmca and FVica (Lindegaard ratio) exceeds 3.0 or FVmca > 120 cm/sec.[14]
Other applications include severe head injury and stroke. Continuous measurement of FVmca, the autoregulatory "threshold" or "break point" [cerebral perfusion pressure (CPP) at which autoregulation fails] can be easily detected. This provides a target CPP value for treatment. It may also be used to assess changes in CBF in patients with secondary causes of raised ICP.
LASER DOPPLER FLOWMETRY
This technique allows continuous real time measurement of local microcirculatory cortical blood flow.[15] A 0.5 to 1mm diameter fiberoptic laser probe is placed in contact with or within brain tissue and conducts reflected light back to a photodetector within the flowmeter sensor. The signal is processed to give the blood flow.
Disadvantages include invasiveness, not a quantitative measure, measures CBF in a small brain volume, and is prone to artifacts.
THERMAL DIFFUSION FLOWMETRY
The thermal conductivity of cerebral cortical tissue varies proportionally with CBF. The monitor consists of two small thermistors, one of which is heated. Insertion of a thermal diffusion probe on the surface of the brain allows CBF to be calculated from the temperature difference between the plates.[16]
Advantages include potentiality for bedside monitoring of



cerebral blood flow at tissue level. Disadvantages include its invasiveness.

XENON 133 WASHOUT TECHNIEQUE

Regional decay in radioactivity after intracarotid or intra-aortic injection of ^{133}Xe is measured by scintillation counters positioned over the head. The slope of the wash-out curve is proportional to regional CBF. The curve is biexponential, the fast and slow components possibly representing blood flow in gray and white matter. This method estimates predominantly cortical blood flow.

TOMOGRAPHIC TECHNIQUES

Dynamic computed tomography (CT) - quantifies the washout of inhaled xenon or intravenous radioiodinated contrast agents to measure regional CBF.

Positron emission tomography (PET) - provides images of flow throughout the brain and enables assessment of regional variations and the CBF response to increased stimulation/metabolism

SPECT (SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY) – produces qualitative images of blood flow across all areas of the brain by using γ-emitting technetium 99. It also provides information about cerebral blood volume, oxygen extraction, and the cerebral metabolic rate of oxygen (CMRO2).

FUNCTIONAL MRI – uses MRI to map changes in brain hemodynamics in response to brain neural activity. An intravenous paramagnetic contrast agent or decreases in regional deoxyhemoglobin levels can produce tomographic images of regional CBF.

OTHER TECHNIQUES FOR ESTIMATING CEREBRAL BLOOD FLOW

Intraoperative angiography

Ultrasonic Perivascular Flow Probe

Contrast enhanced Ultrasonography

Laser Speckle Imaging

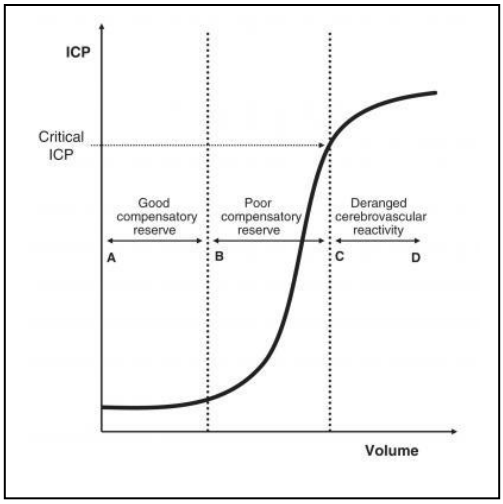
INTRACRANIAL PRESSURE MONITORING

ICP is one of the essential modality in cranial based monitoring systems. It can be measured both invasively and noninvasively. It indirectly helps in the estimation of cerebral perfusion pressure (CPP). It also provides information regarding cerebral blood flow, pressure reactivity, and compliance of the cerebrospinal system.[17, 18]

Methods of measurement:

INVASIVE TECHNIQUES

1. Microtransducers – ventricular, subdural or intraparenchymal
   Intraparenchymal- Codman or camino system
   Disadvantages: Cannot be recalibrated after insertion. Zero drift seen after long term monitoring. Measures local CSF pressure
2. Intraventricular drains –connected to external pressure transducer; gold standard
   Advantages: periodic external calibration possible, csf drainage
   Disadvantages: risk of infection is high

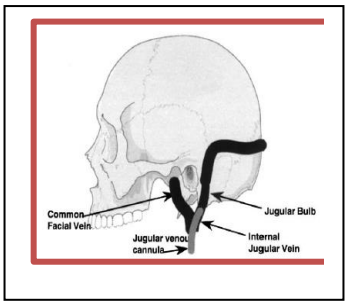


3. Others include epidural probes and lumbar CSF drains- rarely used

NON-INVASIVE TECHNIQUES – includes mainly

1. Transcranial Doppler
2. Tympanic membrane displacement
3. Ultrasound
4. Pupillometry
5. Optic Nerve sheath diameter
6. Ophthalmodynamometer
7. MRI technology

Pressure Reactivity index (PRx) reflects the autoregulatory reserve of cerebral blood vessels.[19] When the cerebral vessels are pressure reactive ABP and ICP show negative correlation, i.e., with increase in ABP, there will be cerebral vasoconstriction, and decrease in ICP, termed a negative pressure reactivity index. Hence, just the opposite, a positive correlation coefficient indicates failed cerebrovascular pressure reactivity. This value may fluctuate but on average this may be helpful to correlate with cerebrovascular dynamics. Optimal CPP- can be extrapolated from the "U"-shaped curve obtained from PRx plotted against CPP, especially in adult patients with head trauma.[20] The outcome depends upon the distance the current CPP has to overcome to reach the optimal CPP. This indicates that both too low and too high a CPP are quite detrimental. This is a very useful guide in refining the therapy as per CPP guided management of head injury patients. Also analysis of the PRx and the ICP pulse waveform gives additional information regarding the adequacy of CPP.



MONITORING OF CEREBRAL OXYGENATION

Jugular Venous Oximetry (Sjvo2)

It is the most widely used monitor for estimating global cerebral oxygenation and metabolism in neuroanaesthesia and intensive care.[21] Measurement can be intermittent or continuous. For determination of cerebral metabolism, it utilizes the equation and relationship between $AVDO_2$, CBF, and $CMRO_2$; $CMRO_2 = AVDO_2 /CBF$. Keeping one of the factors constant, changes of other two factors is interdependent, depending upon their relationship. If CBF is coupled with $CMRO_2$, then $AVDO_2$ remains constant as CBF changes. However, if CBF and $CMRO_2$ are uncoupled, then changes in CBF while $CMRO_2$ remains constant are reflected as changes in $AVDO_2$. Except during ischemia or other pathological events characterized by an extremely low $CMRO_2$, CBF has been shown to correlate well with $AVDO_2$. In severely head-injured patients with an $AVDO_2$ of less than 2.9 ml/dl, an average CBF value of 53±18 ml/100 g/min has been demonstrated; in those with an $AVDO_2$ between 2.9 ml/dl and 6.8 ml/dl, an average CBF value of 42±12 ml/100 g/min was shown; and in those with an $AVDO_2$ greater than 6.8 ml/dl an average CBF value of 23 ± 7 ml/100 g/min was found.

Monitoring of oxygenation:

Insertion of fiberoptic catheters enables continuous monitoring of $Sjvo_2$, with normal values ranging from 55% to 85%. The saturation of jugular venous blood demonstrates whether CBF is

sufficient to meet the cerebral metabolic rate for oxygen ($CMRO_2o$) of the brain. This is particularly useful in monitoring interventions such as hyperventilation therapy. Major limitation is lack of sensitivity to regional changes.

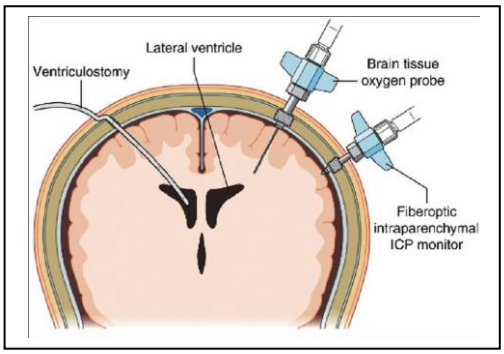
Measurement of Brain Tissue Oxygenation

Brain tissue oxygenation is the partial pressure of oxygen in the extracellular fluid of the brain and reflects the availability of oxygen for oxidative energy metabolism thus represents the balance between oxygen delivery and consumption.[22, 23] Potential benefits include:

1. Optimization of cerebral oxygen delivery
2. Early detection and possible attenuation of secondary cerebral insults
3. Monitoring of focal areas of injured brain
4. Monitoring of uninjured brain and thus allowing assumption about global cerebral oxygenation
5. Greater assessment of therapeutic interventions such as manipulation of CPP
6. Elucidation of underlying pathophysiology after brain injury

It helps in continuous monitoring of cerebral oxygenation but main disadvantage is that it is an invasive procedure and its sensors are fragile.

Most commonly used:



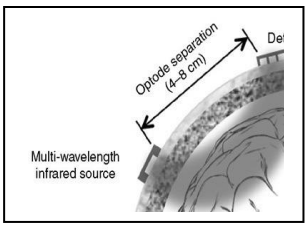
1. Neurotrend sensor (Codman, Johnson and Johnson, Raynham, MA)- measures pbo_2 , $pbco_2$, pH and temperature, now abandoned.
2. Licox (GMS, Kiel-Mielkendorf, Germany) – measures pbo_2 and temperature.

This is usually placed in conjunction with a fiberoptic ICP monitor through the burr hole. Exact localization of the sensor tips on computed tomography after insertion is essential for accurate interpretation and use. Measurement of $Pbto_2$ has been significantly correlated with cerebral venous blood Po_2, $Sjvo_2$, regional cerebral blood flow, positron emission tomography (PET) - derived end-capillary Po_2, and microdialysis glucose and lactate. $Pbto_2$ sensors are accurate with minimal complications and are more reproducible and robust than $Sjvo_2$. CPP and $Paco_2$ levels can be optimized with $Pbto_2$ sensors. $Pbto_2$ complements ICP information in that it provides insight into oxygen delivery.

NORMAL VALUES – 37 to 45 mmHg; a level of 10 mmHg is taken as hypoxic threshold.

CLINICAL APPLICATIONS

Trends in cerebral oxygenation assist in early detection and treatment of secondary insults, as well in assessing responses to therapeutic interventions such as hyperventilation, especially in patients with ischemia following TBI. Measurement of Pbo_2 allows assessment of the effect and reversibility of temporary aneurysm clipping, as well as correct positioning of the permanent clip. It can also be applied to resection of arteriovenous malformations and brain tumours and determination of pharmacodynamics of anaesthetic agents.



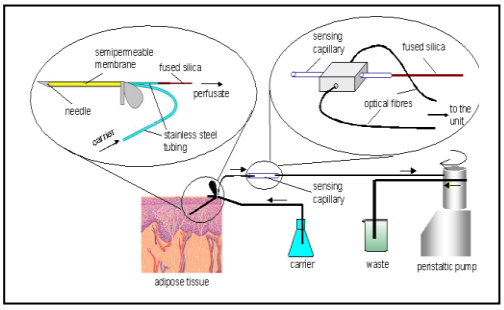
NEAR-INFRARED SPECTROSCOPY

NIRS is a transcutaneous noninvasive monitor of regional cerebral oxygenation.[24] Monitoring is based on the differential absorption of near-infrared light by oxyhaemoglobin, deoxyhaemoglobin, and cytochrome oxidase. It is well established in neonats and provides brain hemoglobin oxygen saturation, cerebral blood volume, and cerebrovascular responses to therapeutic interventions. Although it does not measure CBF specifically, it provides an indication of the balance between flow and metabolism. In adults, it is predominately used to monitor patients with TBI and SAH, undergoing carotid endarterectomy and other neurovascular procedures. Falling saturation indicates a decline in cerebral perfusion. [25]

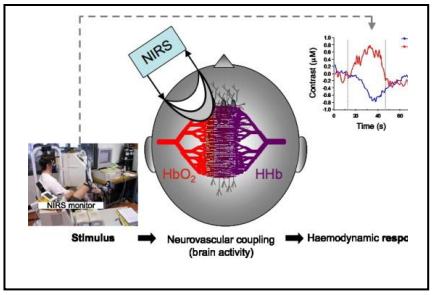
Tissue Spectroscope (TiSpec)Optical monitoring of various tissue physiological and biochemical parameters in real-time represents a significant new approach and a tool for better clinical diagnosis. It enables the monitoring of microcirculatory blood flow (O_2 supply), mitochondrial NADH redox state (O_2 balance), and tissue reflectance, which correlates to blood volume.[26]

MICRODIALYSIS

Cerebral Microdialysis allows in vivo bedside analysis of brain tissue biochemical substances, i.e., markers of energy metabolism – glucose, lactate and pyruvate in the extracellular fluid.[27, 28] The system consists of a perfusion pump, catheter implanted in the tissue, microvials for collection of the dialysate and an analyzer. The catheter can be located either in areas of injured or uninjured areas of the brain and measures the extracellular markers around that area. The L/P ratio is a sensitive marker of impaired aerobic metabolism. Other additional markers include glutamate and glycerol which get



elevated during adverse conditions like ischaemia and hypoxia. Trends of these substances as a part of the multimodality monitoring may provide clinical information regarding the dynamic state of the patient's condition.[29] Its clinical applications include TBI, SAH, stroke, epilepsy, tumours, infections and hepatic encephalopathy. Microdialysis may help evaluate safety of intraoperative manipulations and the duration of hypoperfusion (e.g., during temporary clipping or anastomosis).



Multimodality Monitoring

The concept of multimodal monitoring involves continuous monitoring of more than one parameter using two or more of the techniques described above.[30] This helps to overcome the limitations of each individual method of monitoring thereby enabling more accurate analysis of changes in the measured parameters.

Conclusion

Neuromonitoring is a field with upcoming research and ability and should be an essential part of modern anaesthesia and critical care. Trading with the facts and information provided by multimodality monitoring can be very useful clinically and awe-inspiring. Resourceful use of such information requires techniques to assimilate varied sets of information, and methods to access the online monitoring information remotely and at any time, day or night. Up to date knowledge in brain monitoring in neurointensive care is incorporating these technologies into patient care using telemedicine methods.

References

1. Agrawal A, Timothy J, Cincu R, Agarwal T, Waghmare LB. Bradycardia in neurosurgery. Clinical neurology and neurosurgery 2008; 110**:** 321-327.

2. Bruhn J, Myles PS, Sneyd R, Struys MM. Depth of anaesthesia monitoring: what's available, what's validated and what's next? British journal of anaesthesia 2006; 97**:** 85-94.

3. Practice advisory for intraoperative awareness and brain function monitoring: a report by the american society of anesthesiologists task force on intraoperative awareness. Anesthesiology 2006; 104**:** 847-864.

4. Johansen JW. Update on bispectral index monitoring. Best practice & research. Clinical anaesthesiology 2006; 20**:** 81-99.

5. Viertio-Oja H, Maja V, Sarkela M, Talja P, Tenkanen N, Tolvanen-Laakso H, et al. Description of the Entropy algorithm as applied in the Datex-Ohmeda S/5 Entropy Module. Acta anaesthesiologica Scandinavica 2004; 48**:** 154-161.

6. Vakkuri A, Yli-Hankala A, Talja P, Mustola S, Tolvanen-Laakso H, Sampson T, et al. Time-frequency balanced spectral entropy as a measure of anesthetic drug effect in central nervous system during sevoflurane, propofol, and thiopental anesthesia. Acta anaesthesiologica Scandinavica 2004; 48**:** 145-153.

7. Prichep LS, Gugino LD, John ER, Chabot RJ, Howard B, Merkin H, et al. The Patient State Index as an indicator of the level of hypnosis under general anaesthesia. British journal of anaesthesia 2004; 92**:** 393-399.

8. Holland NR. Intraoperative electromyography. Journal of clinical neurophysiology : official publication of the American Electroencephalographic Society 2002; 19**:** 444-453.

9. Harper CM. Intraoperative cranial nerve monitoring. Muscle & nerve 2004; 29**:** 339-351.

10. Legatt AD. Mechanisms of intraoperative brainstem auditory evoked potential changes. Journal of clinical neurophysiology : official publication of the American Electroencephalographic Society 2002; 19**:** 396-408.

11. Banoub M, Tetzlaff JE, Schubert A. Pharmacologic and physiologic influences affecting sensory evoked potentials: implications for perioperative monitoring. Anesthesiology 2003; 99**:** 716-737.

12. Lotto ML, Banoub M, Schubert A. Effects of anesthetic agents and physiologic changes on intraoperative motor evoked potentials. Journal of neurosurgical anesthesiology 2004; 16**:** 32-42.

13. Singh V, McCartney JP, Hemphill JC, 3rd. Transcranial Doppler ultrasonography in the neurologic intensive care unit. Neurology India 2001; 49 Suppl 1**:** S81-89.

14. Aaslid R. Transcranial Doppler assessment of cerebral vasospasm. European journal of ultrasound : official journal of the European Federation of Societies for Ultrasound in Medicine and Biology 2002; 16**:** 3-10.

15. Nakase H, Kaido T, Okuno S, Hoshida T, Sakaki T. Novel intraoperative cerebral blood flow monitoring by laser-Doppler scanner. Neurologia medico-chirurgica 2002; 42**:** 1-4.

16. Carter LP. Thermal diffusion flowmetry. Neurosurgery clinics of North America 1996; 7**:** 749-754.

17. Czosnyka M, Pickard JD. Monitoring and interpretation of intracranial pressure. Journal of neurology, neurosurgery, and psychiatry 2004; 75**:** 813-821.

18. Balestreri M, Czosnyka M, Steiner LA, Schmidt E, Smielewski P, Matta B, et al. Intracranial hypertension: what additional information can be derived from ICP waveform after head injury? Acta neurochirurgica 2004; 146**:** 131-141.

19. Steiner LA, Czosnyka M, Piechnik SK, Smielewski P, Chatfield D, Menon DK, et al. Continuous monitoring of cerebrovascular pressure reactivity allows determination of optimal cerebral perfusion pressure in patients with traumatic brain injury. Critical care medicine 2002; 30**:** 733-738.

20. Hlatky R, Valadka AB, Robertson CS. Intracranial hypertension and cerebral ischemia after severe traumatic brain injury. Neurosurgical focus 2003; 14**:** e2.

21. Robertson CS, Gopinath SP, Goodman JC, Contant CF, Valadka AB, Narayan RK. SjvO2 monitoring in head-injured patients. Journal of neurotrauma 1995; 12**:** 891-896.

22. Stiefel MF, Spiotta A, Gracias VH, Garuffe AM, Guillamondegui O, Maloney-Wilensky E, et al. Reduced mortality rate in patients with severe traumatic brain injury treated with brain tissue oxygen monitoring. Journal of neurosurgery 2005; 103**:** 805-811.

23. Nortje J, Gupta AK. The role of tissue oxygen monitoring in patients with acute brain injury. British journal of anaesthesia 2006; 97**:** 95-106.

24. Al-Rawi PG, Kirkpatrick PJ. Tissue oxygen index: thresholds for cerebral ischemia using near-infrared spectroscopy. Stroke; a journal of cerebral circulation 2006; 37**:** 2720-2725.

25. Calderon-Arnulphi M, Alaraj A, Amin-Hanjani S, Mantulin WW, Polzonetti CM, Gratton E, et al. Detection of cerebral ischemia in neurovascular surgery using quantitative frequency-domain near-infrared spectroscopy. Journal of neurosurgery 2007; 106**:** 283-290.

26. Mayevsky A, Manor T, Pevzner E, Deutsch A, Etziony R, Dekel N, et al. Tissue spectroscope: a novel in vivo approach to real time monitoring of tissue vitality. Journal of biomedical optics 2004; 9**:** 1028-1045.

27. Bellander BM, Cantais E, Enblad P, Hutchinson P, Nordstrom CH, Robertson C, et al. Consensus meeting on microdialysis in neurointensive care. Intensive care medicine 2004; 30**:** 2166-2169.

28. Engstrom M, Polito A, Reinstrup P, Romner B, Ryding E, Ungerstedt U, et al. Intracerebral microdialysis in severe brain trauma: the importance of catheter location. Journal of neurosurgery 2005; 102**:** 460-469.

29. Vespa P, Bergsneider M, Hattori N, Wu HM, Huang SC, Martin NA, et al. Metabolic crisis without brain ischemia is common after traumatic brain injury: a combined microdialysis and positron emission tomography study. Journal of cerebral blood flow and metabolism : official journal of the International Society of Cerebral Blood Flow and Metabolism 2005; 25**:** 763-774.

30. Timmons SD. An update on traumatic brain injuries. Journal of neurosurgical sciences 2012; 56**:** 191-202.