Monitoring the brain

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Monitoring the brain during anaesthesia has not been routine because: (1) in patients free of neurological disorders, maintenance of adequate haemodynamic and respiratory status implies integrity of CNS function; (2) efforts to monitor the brain during anaesthesia are hampered by the lack of suitable, affordable and reliable equipment; (3) incomplete understanding of neurophysiology and cerebral vascular haemodynamics leaves the anaesthetist helpless even if significant changes are detected.

Cerebral haemodynamics

Intracranial pressure: Monitoring intracranial pressure allows one to assess cerebral perfusion as well as institute treatment.[1] The ventricular catheter allows monitoring of ICP, assessment of intracranial compliance and treatment of elevated ICP by drainage of CSF. Alternatives include the subarachnoid screw and the extra-dural transducer.

Cerebral blood flow (CBF): Global cerebral blood flow (normally $50\,ml \cdot 100\,gm^{-1} \cdot min^{-1}$ between a cerebral perfusion pressure of 50 to 150 mmHg) is measured either by using inert gas uptake (N_2O) or washout (^{133}Xe or ^{85}Kr). Use of a multidetector scintillation counter allows determination of regional cerebral blood flow.

Stump pressure: Pressure in the distal carotid artery ("stump pressure") above 50 mmHg is generally thought to provide adequate collateral circulation during clamping of the carotid. This, however, is influenced by anaesthetic technique as well as the arterial PCO_2.

Cerebral metabolism

Cerebral metabolic rate for oxygen ($CMRO_2$) and glucose (CMR_g): When CBF values are available, measurement of jugular venous bulb oxygenation ($PjvO_2$) and glucose concentration allows calculation of global cerebral oxygen consumption and glucose use. In addition, a $PjvO_2$ above 35 mmHg usually indicates adequate oxygen delivery.

Cerebral function

Electro physiological activity: Intraoperative electroencephalogram monitoring has not been popular because of technical complexity, difficulty with interpretation of a voluminous amount of data, and the variable influences of anaesthetic agents on the EEG. The conventional EEG now enjoys only limited use during carotid endarterectomy.[2] Because of these limitations, devices such as the compressed spectral array (CSA) and the cerebral function monitor (CFM)[3] have been designed to process the data into a more interpretable form. The CSA takes a four-second epoch of a bipolar EEG signal and separates the components into their respective power spectrum, converting the EEG data from a time to a frequency domain. A three dimensional graph is obtained and a long period of recording can be compressed and displayed on a single piece of paper. The CFM represents the other extreme of processing raw data. Frequencies between 2 to 15 Hz are extracted from a single channel EEG, rectified, averaged and displayed as a continuous line, the height of which is dependent both on the voltage as well as the frequency of the electrical activity. Thus, it is a crude measure of electrical activity without discrimination of the contribution of different frequencies. Attempts have been made to use this to determine the depth of anaesthesia and the tolerance limit of hypotension. Its simplicity, however, is equally matched by its non-specific interpretation and lack of sensitivity.

Monitoring of sensory evoked responses (SER) represents the most promising monitor of electrical activity during anaesthesia.[4] While EEG monitors spontaneous activity of non-specific nature the SER is functionally pathway specific. Modalities employed include somatosensory, visual and auditory. Cortical-evoked responses are influenced by anaesthetic agents in a dose-related manner whereas brain stem-evoked responses are relatively resistant to anaesthetic influences. Somatosensory-evoked responses have been used successfully during spinal column surgery to monitor neurological functions. Visual-evoked responses have been used to assess integrity of the optic pathway during surgery in proximity to the optic chiasma. Brain stem auditory-evoked responses have been used to monitor

and preserve the integrity of the eighth nerve during posterior fossa surgery, as well as brain stem function during operations on aneurysms of the vertebral basilar artery system.

References

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Monitoring the lung

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With induction of anaesthesia and its attendant alterations in gas exchange, the anaesthetist monitors lung function using instruments that must not only measure but warn. Measurement and warning are not necessarily synonymous and the latter function is often assumed by the anaesthetist. Thus, all mechanical and electronic methods are supplemental to physical examination, i.e., *inspection, palpation, percussion* and *auscultation*. The use of a precordial or oesophageal stethoscope is a mandatory intraoperative monitor; the risks are nil, the benefits enormous. But, just as the presence of electrical cardiac activity does not guarantee systemic perfusion, neither does the presence of breath sounds guarantee adequate alveolar gas exchange. Thus ancillary techniques and instrumentation have been developed to monitor both carbon dioxide and oxygen exchange.

Carbon dioxide

Inspection of the reservoir bag permits an estimate of minute volume (VE) in spontaneously breathing subjects. However, anaesthetic circuits or faulty valves which permit rebreathing, can mislead the clinician. The fundamental equation of factors that determine alveolar CO_2 concentration has been described.[1] Since anaesthesia can produce respiratory depression and rebreathing of CO_2 can be highly variable, the use of qualitative monitors of spontaneous ventilation, such as respiratory rate, tidal volume, movement of reservoir bag, chest-abdominal movement, the stethoscope and transthoracic electrical impedance measurements, are unreliable substitutes for measurement of alveolar or arterial PCO_2. End-tidal CO_2 can be monitored using an infra-red capnograph or mass spectrometer. However the end-tidal CO_2 to arterial PCO_2 gradient increases in patients with pulmonary disease, making this monitor unreliable in the high-risk group. Transcutaneous CO_2 monitoring ($TcPCO_2$) has limitations, but may be useful in specific cases.

Oxygen

Minor alterations of $PaCO_2$ are usually benign; however, this is not true for PaO_2. Potential causes of hypoxaemia are:

1. decreased FiO_2
2. decreased alveolar ventilation (VA)
3. increased V/Q mismatching
4. any right to left shunt
5. decreased PvO_2 in – anaemic states
   – low cardiac output states
   – increased VO_2 conditions.

FiO_2 should always be monitored with a properly calibrated oxygen analyser in the inspiratory limb of the breathing circuit. Future design improvements[3] will improve FiO_2 monitoring and reduce the incidence of hypoxic gas delivery due to human error or machine mulfunction.

The most frequent cause of decreased VA induced hypoxaemia is a disconnection during mechanical ventilation.[4] Anaesthetic ventilators should only be used in conjunction with low-