Adequate cerebral blood flow to each part of the brain is essential for its normal functioning as cerebral tissues are very intolerant of hypoxia. The cessation of cerebral blood flow/circulation for a few minutes can produce permanent damage to the brain. In addition, the CBF determines the cerebral blood volume (CBV) which itself is the net result of cerebral arterial supply and venous drainage. The CBV being a major (60-65%) and variable component of intracranial contents, plays an important role in determining its total volume. The cranium being a closed hard case (except in young children with open fontanelles or suture lines) with limited compliance, the abnormal changes in CBV and intracranial contents manifest in the form of a proportionate change in intracranial pressure (ICP). An idealized pressure/volume (ICP/CBV) relationship can be described to represent the intracranial compliance curve (Fig.1). Initially (Phase A), when the intracranial volume increases, the intracerebral pressure does not increase suggesting the presence of compensatory mechanisms. However, when the elastance of the system is limited (Phase B), a further increase in volume translates into a rise in pressure. Finally, when the compensatory mechanisms have been exhausted, any slight change in volume leads to an exponential rise in pressure (Phase C)\(^1\). However, if the cranium is open, an increase in CBV will cause a brain bulge (tight brain) and a decrease will result in brain relaxation.

Physiological Considerations

Cerebral Blood Supply: Although only 2% of body weight, the brain accounts for 20% of resting oxygen consumption at a rate of 3-3.5ml/100g brain/min which is even more in infants and young children (5ml/100g brain/min)\(^2\). Due to its lack of substrate storage and high metabolic rate, the brain is very sensitive to oxygen deprivation. For its optimal oxygen supply, the brain receives almost 15% of total cardiac output i.e. nearly 700-750 ml/min or 55 ml/min/100 gm brain tissue.

The principal pathways are contained in the circle of Willis. Potential collateral pathways exist between the external and internal carotid arteries via the ophthalmic
and meningeal vessels. Also important are the leptomeningeal collaterals. These collateral arterial pathways are the corner stone of CBF compensation during ischemia. However, in the event of occlusion of one of the major cortical vessels, infarction is almost certain because of inadequate anastomotic connections, particularly in the deeper brain structures.

**Physical Principle**: Blood flow is dependent on blood pressure and vascular resistance i.e. flow = pressure/resistance.

In case of CBF, the pressure denotes the Cerebral perfusion pressure (CPP) and the resistance is Cerebral vascular resistance (CVR) Thus CBF = CPP/CVR

As CPP = Mean arterial pressure (MABP) - Cerebral venous pressure.

CBF = MABP - Cerebral venous pressure / CVR

As cerebral venous pressure is only slightly more (nearly equal) to ICP, we assume for clinical purposes that CBF = MABP-ICP / CVR

Thus we may conceptualize as pressure and resistance being independent variables and flow as the dependent variable. For example drugs exert effects on CBF by changing CPP and CVR directly by vasodilatation and indirectly by metabolic suppression.

**CBF Regulation**: A precise regulatory system has evolved in the brain whereby the cerebral blood flow and metabolism are coupled. Thus the local nutrient supply matches to metabolic demand in the functionally diverse central nervous system e.g. the CBF in the white matter is 20 ml/100 gm/min whereas in gray matter it may be up to 80-100ml/100gm/min and in younger children the average flow may be as high as 100ml/100g brain/min². Clinical levels of anaesthesia do not uncouple flow and metabolism, although they may reset their relationship. The flow-metabolism coupling is critical during times of stress or extreme conditions such as hypotension or hypoxia. These processes involve following regulatory mechanisms to maintain flow at required levels.

1. **Haemodynamic Auto-regulation**: It refers to cerebral haemo-dynamic response in the form of an active arteriolar vasomotion to maintain a constant CBF to wide range of changes in CPP (lower limit 50-70 mmHg and upper 130-150 mmHg), independent of flow to metabolism coupling. Beyond these limits, the CBF changes proportionately to the MABP. It must be remembered that auto-regulation in children occurs at lower absolute values than adults, as the MABP may not reach 60mmHg (torr) until months after birth and it reaches to approximately 75 mmHg at one year of age.

**Carbon dioxide** is a powerful modulator of cerebrovascular resistance, and perhaps clinically most accessible one. Any change in arteriolar PaCO₂ within the range of 20 to 80 torr has a direct effect on cerebral vessels except in neonates, in whom the response to PaCO₂ below 30 mmHg may be blunted. The CBF changes 1-2ml/100g/min for every 1mmHg change in PaCO₂ within physiological ranges. On the other hand, PaO₂ does not affect CBF and only a reduction of PaO₂ to <50 torr has a significant effect, by stimulating arteriolar dilation. The hyperoxia decreases CBF, producing a modest 10-15% decrease at 1 atmosphere. Hyperbaric oxygenation may further decrease CBF. The relationship of CBF to CPP, arterial blood carbon dioxide and oxygen is shown in the following diagram (Figure 2).

2. **Metabolic Mediators and Chemoregulation**: As mentioned above the products of metabolism control CBF by acting as local vasodilators. An overwhelming number of metabolic mediators like, H⁺, K⁺, adenosine and phospholipid metabolites are known. Recently a few endothelium-derived factors have been described. Out of these, the nitric oxide (NO) has been subjected to intense scrutiny. Nitric oxide is supposed to be a vascular tone mediator functioning as an endothelium-derived relaxing factor (EDRF) and it also acts as a neurotransmitter.

3. **Neural Control**: Though the precise function of autonomic nerves innervating the perivascular area remains obscure, current belief holds that these are not necessary for but may modify the regulatory responses. Nonetheless this view may change as NO is increasingly being recognised as a neurotransmitter.
As a neurotransmitter, the nitric oxide undoubtedly will have a therapeutic role in cerebral ischemic states\(^9\).

4. Circulatory Peptides: The circulatory vasoactive substances such as angiotensin II may also have influence on CBF.

Thus the interaction between salient CBF control mechanisms - mainly the chemo regulation operating through changes in PaCO\(_2\), PaO\(_2\), extracellular pH and metabolic products as mentioned above, and a responsive auto-regulation, may be summarized schematically as shown in Figure 3.

![Figure 3: The CBE and its control mechanisms (Taken from KW Lindsay and Ian Bownes, In Neurology and Neurosurgery Illustrated)](image)

As is evident in the figure above, a decrease in CPP will cause vasodilation of cerebral vessels avoiding cerebral ischemia, whereas, an increase will cause vasoconstriction avoiding hyperemia\(^10\).

Cerebral Steal and Inverse Steal: The cerebral steal refers to stealing of blood from one area of the brain to another. The stealing is possible only if there is a pressure gradient between the circulatory beds of two areas. In clinical practice the steal refers to no increase in CBF of an ischemic area as compared to the normal area when hypercapnia is instituted (causing cerebral vasodilation) or other vasodilators, e.g. volatile anaesthetics, sodium nitropruside and nitroglycerine etc are administered. As the blood vessels of the ischemic area are maximally dilated with an exhausted "cerebro-vascular reserve", they do not respond by any further vasodilation receiving the same or even lesser blood flow, whereas, the normal adjacent brain regions get vasodilated and receive an increased flow.

Conversely to above, a vasoconstriction caused by hypocapnia or a suitable anaesthetic agent such as thiopentone will cause a reduced blood flow to the normal responsive regions of the brain resulting into redistribution of blood to ischemic regions. This is called, "Robin Hood Phenomenon" or the "inverse- steal". Thus the inverse steal redistributes more CBF to ischemic areas\(^11\).

Measurement of CBF

Out of the many methods available for CBF measurement most are difficult to use in clinical settings and those, which can be used with relative ease, do not give a numerical value of global or regional CBF. The choice of a method will depend upon its availability, expertise, cost and ability to perform repeated measures in a given situation.

1. Kety-Schmidt's Arteriovenous Difference Method: Most of the techniques in use today, are either derived from the work of Kety and Schmidt or its some variation. This method uses an inert, radioactive (Xenon\(^{133}\) or Krypton\(^{85}\)) and freely diffusible tracer, measuring its concentration in the arterial and venous (Jugular bulb) blood at different time intervals, to find out arteriovenous desaturation or rarely saturation curves. If CMRO\(_2\) remains constant, the relative changes in arteriovenous difference in oxygen content (Ca-Cv) must reflect global CBF.

Thus CBF=a (Ca-Cv) where a is a proportionality constant.

This method has the disadvantage of being cumbersome and invasive and may over estimated the CBF in low perfusion states.

2. Positron Emission Tomography (PET): It allows for precise imaging of CBV, CBF, cerebral glucose and oxygen utilization (CMRO\(_2\)), pH and neurotransmitter events etc. Its disadvantages are cost and complexity.

3. Single Photon Emission Computed Tomography (SPECT): SPECT is the image produced by gamma scintillation counting that is reconstructed in three dimensions by a rotating camera. Recently, dedicated brain scanners specifically optimized for the intracranial cavity have become available. Though SPECT offers slightly less resolution than PET, yet it gives far more specific anatomical information and is cheaper than PET. Thus SPECT imaging is likely to become increasingly commonplace in the management of cerebro-vascular disease.

4. Magnetic Resonance Imaging (MRI): MRI angiography supplants the standard contrast x-ray techniques. By using paramagnetic tracers excitable in a magnetic field, one may directly examine cerebral perfusion. Moreover, with development of freely diffusible drugs, it will be possible to determine wash-in and wash-out as is currently done in radio-isotopic methods. MRI
by providing both the CBF and structural informations could become the “gold-standard” method in future.

5. Doppler Techniques: Transcranial Doppler (TCD), Laser Doppler and other ultrasonic devices are in wide use for clinical imaging. The methodology is similar for all applications, using a probe placed over low density bone regions of skull. The ultrasonic beam is focussed on the desired vessel and its Doppler shift is recorded which is proportional to the blood flow velocity of the vessel in focus. Thus the Doppler measures the flow velocity of the cerebral vessels individually and not of all together i.e. not the global CBF or CBV. The other problem of Doppler relate to variability of exact angle of insonation and difficulty in finding a vessel.

The greatest advantages of TCD are that it is relatively inexpensive, noninvasive, and non-radioactive and it furnishes a continuous information about the cerebral circulation.

Thus we find that different methods examine different aspects of the same or related phenomenon and different techniques may be required to completely elucidate a process.

Effect of Anaesthetic Agents on CBF

Abnormal changes in CBF (excess or low) adversely affect the outcome of neurosurgical patients. Most of the anaesthetics and adjuvants cause atleast some alteration in CBF. But the exact mechanisms involved for alteration in CBF with different drugs appear to be different for different classes of agents. Following is a brief mention of changes in CBF in a clinical point of view, caused by individual drug class/drug.

Inhalational Agents

Nitrous Oxide: The literature is controversial regarding the effects of nitrous oxide (N₂O) on brain primarily because of species differences. In humans, if used alone (in oxygen), the N₂O is a potent cerebral vasodilator and may also increase the ICP but without any appreciable change in CMRO₂. When combined with barbiturates, narcotics, volatile agents or hypocarbia, the effects on CBF and ICP are attenuated or abolished. Still, it would seem prudent, in the event of a “tight brain” intra-operatively, that discontinuation of N₂O should be considered among possible beneficial interventions.

Volatile Anaesthetics: All the volatile anaesthetics cause dose related increase in CBF and a decrease in CMRO₂. Because these agents cause both an increase in CBF and a decrease in brain metabolism, it is unlikely that cerebral hyperemia is metabolically mediated.

Although in vitro data suggest that halothane is more potent vasodilator than isoflurane, the direct vasodilator potential in vivo appears similar between the two. As isoflurane causes greater reduction of CMRO₂ than halothane, some investigators have reported a greater cerebral vasodilation by halothane as compared to isoflurane. Considering the net result, isoflurane is preferred over halothane in patients with increased ICP, as it does not increase the CBF at doses less than 1-1.5 MAC, whereas, the halothane consistently increases CBF. The enfurane has fallen out of favour as it increases the ICP significantly in patients with space occupying lesions and has epileptogenic properties. Both desflurane and sevoflurane have a special role in neuroanaesthesia as they ensure a rapid onset of and emergence from anaesthesia. They cause a dose related rise in both CBF and ICP and a fall in CMRO₂, at least up to 2 MAC. These changes closely resemble those of isoflurane.

The cerebral auto-regulation is preserved at 1 MAC concentration of almost all the volatile anaesthetics. Also there is no alteration in cerebral vascular response to hypercapnia over time with all, except sevoflurane, where it is slightly blunted.

Intravenous Agents

Barbiturates: The barbiturates decrease both CBF and CMRO₂ in humans. Treatment with barbiturates does not prevent CBF auto-regulation, however, the response to hypoxia and hypercapnia is attenuated because of metabolic depression. In addition to their effect on metabolism, barbiturates may have direct effects on vascular tone causing cerebral vasoconstriction hence a decrease in CBF and ICP. The mechanism for vasoconstriction appears to be due to influx of calcium into vascular smooth muscles. Incremental doses of pentothal may decrease the CBF and CMRO₂ by 55-60 percent.

In the clinical setting, if it is desirable to pursue near maximal brain metabolic suppression with thiopental for brain protection, an end point of complete burst suppression on the EEG introduces an increased risk of significant haemodynamic depression with little further cerebral metabolic suppression as compared to a safer burst suppression EEG pattern with 3-6 residual bursts per min. However, it may be argued that even a burst suppression pattern may not be safe, as this too requires a relatively higher dose of pentothal. Warner et al. in their study using one third of burst suppression dose found an adequate protective benefit expressed as reduction of intact size.

Propofol: The cerebral vascular and metabolic effects of propofol resemble nearly the effects of...
barbiturates. The propofol decreases CBF and CMRO₂ and may also decrease the ICP particularly when combined with hyperventilation. The decrease in CBF with propofol does not appear due to any direct vascular effects (unlike pentothal) but due to a reduction in CMRO₂. It causes a dose dependent but not necessarily an identical reduction in MABP and ICP.

Though quite a few studies have reported that the fall in ICP may be lesser than MABP, resulting into a more marked fall of CPP (and CBF) yet others contradicted this fear and found an identical fall in CBV (i.e. CPP) and ICP after continuous propofol anaesthesia. However, due precaution is needed to avoid unwanted hypotension.

**Etomidate**: The etomidate resembles thiopental in many respects in terms of effects on CBF and CMRO₂. Compared to thiopental, there is only minimal haemodynamic suppression even in high doses. Despite these advantages, the etomidate is rarely used in neuroanaesthesia and intensive care due to high incidence of myoclonus and adrenal cortical suppression.

**Benzodiazepines**: As their site of action is receptor specific and saturable, all of these drugs would be similar in terms of CBF and CMRO₂ effects. They all cause a reduction in CBF due to a decrease in both CVR and CMRO₂. The reduction in CBF and CMRO₂ appears to be less than that observed with other intravenous anaesthetics. Forster et al., found a 30–34% reduction in CBF after administering 0.15mg/kg of midazolam to awake healthy human volunteers. This fall in CBF is not associated with any cerebral ischemia, as there is no impairment of brain glucose, lactate or pyruvate.

**Flumazenil**: Flumazenil itself is virtually without any effect on the central nervous system but if given after benzodiazepines, such as midazolam, it may reverse their cerebral vascular and metabolic effects, with the possibility of an overshoot above pre-midazolam levels in CBF and ICP. Flumazenil may be avoided or used with caution to reverse benzodiazepine sedation in patients with impaired intracranial compliance.

**Ketamine**: It causes a significant increase in CBF and ICP but has lesser effect on CMRO₂. Given the fact that there are many available alternatives to ketamine, it seems reasonable to avoid its use in patients at risk of increased ICP. However, ketamine may emerge as an useful drug in cerebral ischemia due to its NMDA antagonist property. Shapira et al., demonstrated its cerebral protective effect in experimental head injury models.

**Narcotics**: The opioids have been traditionally used in neuro-anaesthesia and neurointensive care, because, they provide potent analgesia associated with a stable haemodynamics with low sympathetic tone and a relaxed brain.

There are conflicting reports on the effect of narcotics on CBF and CMRO₂. In general they have a minimal to modest depressive effects on CBF and CMRO₂. But it appears that the effect is dictated by the control circumstances. Thus if a cerebral vasodilator was used for general anaesthesia (GA), narcotics induce a decrease in CBF. If however, a vasoconstricting anaesthetic or no anaesthetic was used, narcotics either had no effect or increased the CBF. In clinical doses the conventional narcotics usually do not induce major changes in cerebral haemodynamics, but there are occasional reports of fentanyl, alfentanil and sufentanil-induced increases in ICP in head trauma or brain tumour patients. Because narcotics do not alter CO₂ reactivity, their use in combination with hypocapnia accounts for a favourable effect upon brain bulk and ICP.

The collective available data suggest that fentanyl may be the ideal narcotic to give to patients with intracranial mass lesions. Marx et al., compared the effects of equipotent doses of fentanyl, alfentanil and sufentanil on ICP and CPP under general anaesthesia (GA) for brain tumor surgery. The fentanyl had no significant effect on ICP but it decreased the CPP by 28%. The sufentanil caused an 87% increase in ICP and a 24% decrease in CPP and the effects of alfentanil were intermediate between those of fentanyl and sufentanil. In another study the cerebral blood velocity was found to increase by 20% after giving either fentanyl or sufentanil.

A comparison of equipotent doses of alfentanil, fentanyl and remifentanil, given to patients undergoing tumor surgery showed more similarities in effects than differences, although patients given remifentanil did awaken slightly faster. It may be remembered that the rapid offset of remifentanil may be accompanied by some "overshoot" in both MABP and ICP.

**Naloxone**: Although intermittent reports of positive effects of naloxone on ischemic stroke continue to appear in literature, the overall effects of naloxone on CBF are variable and clinically insignificant.

**Local Anaesthetics**: The local anaesthetics (LA) rapidly cross the blood brain barrier and because of their membrane stabilizing effects, exert and effect on cerebral function, CBF and CMRO₂. In subtoxic doses, LA causes a modest and transient decrease in CMRO₂ and CBF.
(coupled with an increase in CVR). After administering 5 mg/kg of lidocaine over 30 minutes in conscious human volunteers Lam et al. observed CBF and CMR reduction of 24 and 20% respectively. When pushed to the point of seizures, both the CBF and CMRO$_2$ increased over control values.

**Effect of Ancillary Drugs and Methods**

**Muscle Relaxants**: Although muscle relaxants do not cross the blood-brain barrier, secondary effect are possible mainly due to histamine release, systemic haemodynamic effects and altered afferent inputs from muscle spindles. On the other hand, muscle relaxation may reduce ICP by preventing coughing and staining, which results in a lowering of central venous pressure.

The succinylcholine can cause impressive increases in CBF and ICP, which is secondary to increased muscle spindle activity. However, it was found to be safe in most neurosurgical cases when used following a smooth induction, more so when a prompt airway control was indicated via intracranial considerations. In an other and more recent study on patients with brain injury, no clinically significant increase in CBF or ICP was observed after succinylcholine.

**d-Tubocurarine** (dTC) is known to release histamine and cause a modest rise in CBF and CSF pressures after a bolus dose. Simultaneously it decreases the MABP and coupled with an increase in ICP, it is expected to cause a modest reduction in CPP. These changes are more significant in haemodynamically unstable patients.

In this regard, atracurium causes less concern, as it is associated with less degree of histamine release. Of great interest is one of its metabolites, laudanosine. The laudanosine readily crosses the blood brain barrier and may cause seizures in large doses. In clinically relevant doses, its cerebral effects are insignificant as under halothane anaesthesia of 1 MAC, atracurium in a cumulative dose of 3.5 mg/kg did not cause EEG arousal response or any change in CBF, CMRO$_2$ or ICP. However, the use of large doses of atracurium, as may be possible in prolonged procedures or in neurointensive care, is controversial and should be avoided, even though a documented case report indicated that after 71 days of infusion, the accumulation of laudanosine was minimal.

Pancuronium, though causes a rise in MABP and pulse rate yet any effect on canine CBF, CMRO$_2$ or ICP could not be demonstrated on giving 0.1-0.2 mg/kg of pancuronium. However, in patients with intracranial pathology and defective auto-regulation, pancuronium induces increases in CBF and ICP.

The vecuronium appears to be without any cerebral effects whatsoever. Predictably, it has no effect on ICP and is most commonly used relaxant in neurosurgical patients. Currently, vecuronium is attracting much interests in neuroanaesthesia mostly as an alternative to suxamethonium for providing fairly rapid intubating conditions. Its haemodynamic effect is lesser than pancuronium, atracurium and mivacurium and comparable to vecuronium, making it more popular. However, the increased cost of using newer relaxants during long neurosurgical procedures must be kept in mind.

**Drugs for Induced Hypotension**: The sodium nitroprusside (SNP) is normally characterized as a central vasodilator but during therapeutic hypotension in men it causes a consistent increase in ICP and either no change or a decrease in CBF. However, if SNP is given without inducing hypotension (partial aortic occlusion), the canine studies showed an increase in both CBF and ICP. The nitroglycerine induced hypotension also has almost similar effects. b-Blockers such as, esmolol, labetalol or metoprolol are safer but unpredictable in their effect. However they are good for treating intraoperative hypertension.

**Hypothermia**: Deliberate hypothermia is often used in many neurosurgical operations, as a measure for brain protection. Deliberate or passive reduction in the brain temperature causes a parallel reduction in CMRO$_2$ and CBF. The decrease in CBF has some heterogeneity, so that CBF changes are most apparent in the cerebral and cerebellar cortex, less in thalamus and minimal in hypothalamus and brainstem. Mild hypothermia also reduces ICP and preliminary clinical trials are promising in terms of neurological outcome. A hypothermia of 2°C to 3°C increases the tolerance of neurons to hypoxia by 25-30 percent. On the other hand, severe hypothermia is less beneficial or even may be detrimental to the outcome. It may lead to metabolic and a relative brain tissue acidosis which can abolish the CBF auto-regulation. The rewarming will cause opposite effects in CMRO$_2$, CBF and ICP but there may be more marked rise in ICP.

**Nitric Oxide**: The NO causes generalized including cerebral vasodilatation, thus raising the CBF. The vasodilatation caused by inhalational anaesthetics is also least in partial due to production of NO. The inhibition of NO synthase results in an increase in MABP and systemic vascular resistance but it prevents cerebral hyperemia caused by N$_2$O, halothane and isoflurane. NO is also implicated with neurotoxicity. It may react with other radical species and form lethal radical forms.

**Free Radicals and Scavengers**: There is substantial but indirect evidence that free radicals (oxygen or others)
play an important role in tissue damage and neurotoxicity including cerebral ischemia. These radicals may alter primary regulatory mechanisms of CBF, especially during disease and head injury. Though quite a few antioxidants are in clinical use and still many others are undergoing trials, none so far has demonstrated a clear neuroprotection. More recently a class known as 21-aminosteroids has been tested in animals and is under going multicentric clinical trial. The preliminary studies have found these as a potent free radical scavenger and provider of neuroprotection in cerebral ischemia.

Cholinergic Agonists: It has been proved that the natural sleep is maintained by acetylcholine. Likewise, the rapid eye movement (REM) sleep can be induced chemically by injecting cholinergic agonists - carbechol, neostigmine and pilocarpine. The GA mimics REM sleep in many ways, hence cholinergic agonists are expected to help in maintaining unconsciousness. Oxotremorine, a cholinergic agonist is known to cause cerebral hyperemia, which is accentuated during isoflurane but not pentobarbital anaesthesia. Thus the agonists mentioned above are expected to increase the CBF and accordingly the anticholinergics to decrease it.

Calcium and Calcium Entry Blockers: The direct evidence of the effect of calcium on CBF is lacking. Studies on inhalational anaesthetics have shown that they cause a depression in both calcium and potassium currents and channel blockade. This leads to membrane depolarization, vascular relaxation and an increase in CBF. The calcium entry blockers like nimodipine are understood to protect cerebral ischemia, improve regional CBF, decrease infarct size and improve the neurological status. Inspite of favourable results in small trials, not all investigations in stroke victims have confirmed the benefits of nimodipine.

Reversal/Prevention of Ischemia & Enhancing CBF

The cerebral ischemia may be prevented or reversed by avoiding hypovolemia and maintaining adequate intravascular volume, thereby an adequate CBF. Various factors which are helpful in raising the CBF, may be shown schematically as following (Figure 3). In the absence of randomized, prospective and controlled trials of volume expansion in the prevention and treatment of cerebral ischemic due to vasospasm, the clinician must proceed with the available information. As anaesthesiologist, we are often responsible for patients in whom hypertensive hypervolemia has already been instituted and we need to maintain it or else initiate and maintain the volume expansion.

Maintenance of a high perfusion pressure in combination with optimal viscosity and oxygen delivery may reduce cell death in threatened vascular territory as evident by an improvement in cerebral perfusion, evoked responses and neurologic outcome. By augmenting systemic perfusion pressure, one can mitigate the pressure drop across a stenotic vessel. However, induced hypertension is controversial as it is not without hazards such as worsening cerebral oedema, haemorrhage and risk of cardiac ischemia.

As mentioned above, the cerebral perfusion to an ischemic area may be enhanced by inducing ‘inverse-steal’ through methods such as hypocapnia and administration of thiopentone. The resultant cerebral vasoconstriction, causes a preferential higher flow towards, ‘non-responsive’ ischemic area from the ‘responsive’ (constricted) healthy brain areas.

Whether steal or inverse-steal have any bearing on anaesthetic management is open to debate. Inspite of this, the anaesthesiologist may be called upon to try some pharmacological manipulation to enhance the regional flow to an ischemic area. The clinical role of barbiturates remains controversial, as outcome studies are lacking. The choice is limited to only other two or three agents such as propofol, midazolam and etomidate. Even these drugs have not been convincingly demonstrated to be cerebro-protective.

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References


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