Cerebral blood flow during induced hypotension

Induced hypotension has been used in anaesthetic practice since the 1940s [1]. It has been argued that it should not be used to make a difficult operation easy but is justified to make the impossible possible. It would seem reasonable to take this argument a stage further by stating that induced hypotension is justified if it reduces the overall morbidity of an operation. The fact that moderate hypotension has been used for many years with minimal morbidity [2, 3] lends further support to the continued use of this technique.

The relative safety of the technique in healthy individuals is well documented [2, 3] and the anaesthetic technique does not appear to affect outcome significantly after hypotensive anaesthesia. However, there are several contraindications to the technique, including respiratory, and in particular, cardiovascular disease. It is now generally accepted that, except during surgery for cerebral arteriovenous malformations, cerebral perfusion pressure should not be allowed to decrease during cerebrovascular surgery. The best method of cerebral protection is maintenance of an adequate mean arterial pressure and cerebral perfusion pressure. This is particularly important when cerebral vasospasm occurs after a subarachnoid haemorrhage or the cerebral arteries are occluded by atheroma, thrombus, or both. In these situations mean arterial pressure needs to be well maintained to optimize collateral circulation [4].

It is likely that induced hypotension will continue to be used during many surgical procedures. This will usually be at the request of the surgeon, and the anaesthetist must establish that it is necessary before complying. If it is accepted that there is a role for induced hypotension in modern anaesthetic and surgical practice, it is important that the effects of different techniques of hypotensive anaesthesia on cerebral blood flow to essential organs are elucidated. Cerebral blood flow is maintained at a lower mean arterial pressure when hypotension is induced with vasodilators than when it is induced by haemorrhage, because the latter causes increased sympathetic activity and constriction of the major vessels supplying the brain. Cerebral vasodilators effectively shift the limits of autoregulation of cerebral blood flow to the left [5].

In this issue, Tsutsumi and colleagues [6] report the effects of profound hypotension, to a mean arterial pressure of 30 mm Hg for 30 min, using haemorrhage, trimetaphan or nitroprusside on local cerebral blood flow (CBF) in rats. A mean arterial pressure of 30 mm Hg was chosen because maintenance of local CBF and survival are compromised below this level [7]. Haemorrhagic hypotension and trimetaphan-induced hypotension reduced local CBF in most regions of the brain but most notably in the neocortex and telencephalon, whereas hypotension induced with nitroprusside caused a significantly smaller reduction in CBF. EEG activity, which indicates continuing neuronal function, was maintained for longer in the nitroprusside group. These results are supported by the earlier work of McDowall and colleagues [8, 9] who showed that induced hypotension with nitroprusside reduced cerebral cortical blood flow, cerebral electrical activity and brain-surface oxygen tension to a lesser extent than similar levels of hypotension with trimetaphan or haemorrhage. The present work is useful because it shows that flow to subcortical regions was maintained better during hypotension with nitroprusside than with trimetaphan or haemorrhage. However, it should be noted that hypotension to a mean arterial pressure of 40 mm Hg, even when induced with nitroprusside, is associated with cerebral metabolic abnormalities [10].

Halothane anaesthesia was used despite the known detrimental effect of high concentrations on brain energy metabolism [11, 12]. The same work could be performed with other anaesthetics, such as isoflurane and propofol, which depress cerebral metabolic rate, to establish if local cerebral blood flow is maintained better with these agents. Published data indicate that isoflurane has properties which could be useful during hypotensive anaesthesia. In humans, trimetaphan-induced hypotension to a mean arterial pressure of 40 mm Hg caused EEG amplitude depression during 0.5 % halothane anaesthesia whereas there was no EEG depression with 1 % isoflurane [13]. Despite a similar depression in cerebral metabolic rate, isoflurane-induced hypotension was associated with higher cerebral blood flow than that during hypotension induced with a peripheral vasodilator during anaesthesia with propofol and alfentanil [14], and also blood flow was surplus to metabolic requirements when hypotension was induced with isoflurane [15]. Information on the cerebral effects of the newer anaesthetic agents is limited. Hypotensive anaesthesia with desflurane reduces CBF by 36 % and reduces cerebral metabolic rate by a similar amount [16], and 4 MAC of sevoflurane has no adverse effects on brain energy metabolism [12]. Therefore, it would appear to be safe to use these agents during hypotensive anaesthesia.

The relative safety of hypotension with nitro-
prusside was confirmed by work in healthy patients anaesthetized with enflurane when there was no change in CBF or cerebral metabolic rate on reducing mean arterial pressure to 50 mm Hg [17]. Combinations of sodium nitroprusside and trimetaphan have been used to combine the best features of both agents, and a 1:5 mixture appears to provide hypotension with little change in heart rate [18]. It would be useful to investigate the effect of such a combination on CBF. The main function of the circulation is the delivery of oxygen and nutrients to the tissues, so knowledge of the effects of hypotensive agents on the microcirculation of the brain is desirable. Red blood cell flow in the cerebral microcirculation of rats is greater during isoflurane than halothane anaesthesia [19], and the deformability of red blood cells is improved by prostaglandin E\(_1\) [20].

There are many situations where induced hypotension during surgery can be shown to be beneficial. Hypotensive anaesthesia produced by extradural analgesia has been shown to improve penetration of cement into bone, reducing blood loss during total hip replacement [21]. For many years hypotensive anaesthesia has been advocated for middle ear surgery [3] and is associated with minimal morbidity. In ASA I and II patients, induced hypotension to a mean arterial pressure of 50-55 mm Hg, during propofol or enflurane anaesthesia supplemented by labetalol and nitroglycerin, had no effect on psychomotor function [22]. Similarly, Toivonen, Kuikka and Kaukinen [23] detected no significant effects on mental function when labetalol and isoflurane were used to induce hypotension for middle ear surgery.

The use of induced hypotension during neurosurgical operations is diminishing. In spinal surgery, bleeding is mostly venous in origin and so the operative technique is the most important determinant of blood loss and induced hypotension is unnecessary [24]. However, it is useful in reducing morbidity associated with haemorrhage and blood transfusion during excision of vascular tumours and arteriovenous malformations (AVM). Cerebral vasospasm does not occur when an AVM is the cause of a subarachnoid haemorrhage so hypotension may be used safely during operation. In the early postoperative period, autoregulation and carbon dioxide reactivity are disturbed in the brain underlying the AVM and hypertension may cause cerebral oedema leading to neurological deterioration. It may be necessary therefore to use hypotensive agents to maintain mean arterial pressure at the lower end of the normal range. When rupture of an aneurysm on a cerebral artery is the cause of subarachnoid haemorrhage, cerebral vasospasm is a common complication and induced hypotension may further impair cerebral oxygen supply by compromising the collateral circulation. In experimental work, arterial pressure has been shown clearly to be an important determinant of collateral flow during focal cerebral ischaemia [4] and, in the author's department, continuous monitoring of oxygen saturation in the jugular bulb in patients undergoing clipping of cerebral aneurysms has shown that about 50% have a critical mean arterial pressure below which there is hypoperfusion [unpublished observations]. However, in a series of 112 patients undergoing surgery for cerebral artery aneurysms, induced hypotension did not alter outcome or the incidence of re-rupture compared with normotension [25]. In another study, patients in whom hypotension was used during rupture of the aneurysm had a worse outcome than those in whom tamponade or temporary clipping was used to control haemorrhage [26]. As the primary objective of induced hypotension in these patients is to reduce the incidence of intraoperative rupture, these studies show that induced hypotension is usually not appropriate during surgical occlusion of cerebral aneurysms. If there is a neurological deficit after the aneurysm has been clipped, hypervolaemic, hypertensive haemodilution can be used. This improves oxygen supply to ischaemic areas of the brain and improves the chances of survival of critically ischaemic neurones.

In conclusion, induced hypotension is a safe technique in healthy patients and depression of cerebral metabolic rate during general anaesthesia reduces the chance of cerebral complications. During moderate hypotension it is advisable to maintain normocapnia, but at mean arterial pressures below 50 mm Hg the level of Pa\(_{\text{CO}}\_2\) is not important because carbon dioxide reactivity of the cerebral circulation is lost [7]. The safety of the technique is increased by the use of direct acting vasodilators such as nitroprusside, glyceryl trinitrate or isoflurane which shift the range of cerebral autoregulation to the left. In previously normotensive patients who do not have cerebrovascular disease, a mean arterial pressure of between 40 and 50 mm Hg, measured at the level of the head, is well tolerated for several hours. However, the decision to use induced hypotension depends on clinical judgement and entails weighing the risks of the technique against the benefits it could provide. Extra care should be taken in the elderly who are often hypertensive and have cerebrovascular disease which cause susceptibility to ischaemic brain damage during hypotension.

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References
Editorial


