Recent advances in neuroanaesthesia

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In order to provide optimum intracranial operating conditions for neurosurgery, anaesthetists must have a thorough understanding of brain physiology and how this is affected by pathology and anaesthetic drugs and techniques. This article discusses the current understanding of cerebral vascular physiology and how novel neuroanaesthetic drugs and techniques affect it.

Neuroanaesthesia, perhaps more than any other field of anaesthesia, has represented an area where the expertise and competence of the anaesthetists can influence patient outcome. Developments in neurosurgery, neuro-intensive care and neuroradiology have only been possible because of the concomitant advances in neuroanaesthesia. These advances have been underpinned by experimental research and the availability of novel drugs and monitoring modalities. While many of the advances in neuroanaesthesia are based on high quality clinical research with outcome data, some are based on personal experience and data obtained from better understanding of the clinical pathophysiology.

In order to provide optimum intracranial operating conditions for neurosurgery, anaesthetists must have a thorough understanding of brain physiology and how this is affected by pathology and anaesthetic drugs and techniques. This means ensuring that the intracranial pressure (ICP) does not rise and that, when the skull is open, brain bulk is not increased. The neurosurgeon must be able to retract the brain easily; too high a pressure on the retractors may result in neuronal damage. This article discusses the current understanding of cerebral vascular physiology and how novel drugs and techniques in neuroanaesthesia affect it.

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CEREBRAL PHYSIOLOGY

Intracranial pressure and cerebral blood volume

An increase in any of the intracranial contents, including the brain, CSF and blood, can lead to raised ICP. Most of the intracranial blood volume (about 200 ml) is contained in the capacitance venous vessels of the cerebral circulation. A reduction in this volume by physiological or pharmacological interventions may be used to compensate for rises in brain or CSF volume. However, this compensatory mechanism can be easily exhausted, and at the point of decompensation (arrow in *Figure 1*), small changes in volume will lead to dramatic variations in ICP.

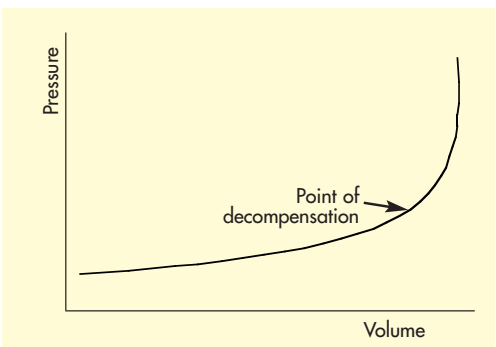
Cerebral perfusion pressure

Cerebral blood flow (CBF) is determined by the cerebral perfusion pressure (CPP); the difference between the mean arterial pressure and ICP. In patients with critical intracranial compliance, extremes of CPP will lead to an increase in the ICP. Low CPP will lead to cerebral vasodilatation, an increase in venous blood volume and ICP. At very high CPP, when the upper limit of cerebral autoregulation is exceeded, increases in ICP are the result of increases in CBF and cerebral blood volume (CBV).

Factors determining cerebral blood flow

Flow metabolism coupling: Brain blood flow is closely coupled to its metabolic activity. This coupling may be mediated by either metabolic (potassium and adenosine) or neurogenic pathways — acetylcholine (Edvinsson et al, 1993), nitric oxide (Kontos, 1993) and dopamine. Intravenous anaesthetics, such as the barbiturates, produce cerebral vasoconstriction indirectly by reducing cerebral metabolism. Maximal metabolic

Figure 1. Pressure–volume intracranial compliance curve.



reduction coincides with the onset of electrical silence in the electroencephalogram (EEG), beyond which no further reduction in cerebral metabolic rate or CBF occurs with further administration of the agent (Matta et al, 1995a).

The effect of inhalational anaesthetics on the cerebral circulation can be explained by the dual action hypothesis (Matta et al, 1995b). Briefly, this states that inhalational agents have a direct cerebral vasodilatory action independent of cerebral metabolism and an indirect effect consequential of the normal flow–metabolism coupling. The net effect of an inhalational agent on CBF depends on the balance between its direct vasodilatory action and its vasoconstrictive cerebral properties secondary to its cerebral metabolic depressant effects.

When cerebral metabolism is already depressed, the agent increases CBF by vasodilation of cerebral vessels. However, should the agent be administered to patients who are in a 'light plane of anaesthesia', its cerebral metabolic depressant effect leads to a decrease in CBF. Therefore, at low concentrations, inhalational agents tend to reduce the CBF in keeping with flow metabolism coupling; at higher concentrations, their vasodilatation properties predominate, leading to increases in CBV and ICP. This direct vasodilatation may also explain the steal of blood from ischaemic areas. The vasodilatory properties of inhalational agents may be partly counteracted by hypocapnic hyperventilation. Inhaled anaesthetic agents are best avoided when ICP is elevated, or if the surgical field is persistently tight.

Cerebral vascular reactivity to carbon dioxide: CBF is directly proportional to arterial carbon dioxide tension ($PaCO_2$). On average, each kPa change in $PaCO_2$ produces a change of about 15 ml/100 g/min in CBF. Prostaglandins (Stringer et al, 1993) and nitric oxide (Maktabi, 1993) may mediate the vasodilatation produced by carbon dioxide (CO_2). Moderate hypocapnia ($PaCO_2$ <3.5 kPa) has long been used to reduce CBV in intracranial hypertension, but this practice is now questioned, because of the risk of inducing cerebral ischaemia. The use of hypocapnia is based on the assumption that reduction in $PaCO_2$ is accompanied by similar reductions in CBV, ICP and hence better cerebral perfusion (Kosteljanetz, 1986). Although this can be readily shown in animal models, this relationship is less robust in the injured human brain. Hypocapnia may reduce CBF without any significant reductions in CBV, leading to brain hypoperfusion. It has been shown that 'traditionally acceptable' levels of hypocapnia in head injured patients can result in dangerously low regional CBF levels (Stringer et al, 1993; Chesnut, 1997). Paraesthesia and EEG abnormalities have been reported in volunteers with $PaCO_2$ of 2.5 kPa and were reversed by the administration of hyperbaric oxygen. In addition to its direct effect on CBF, hyperventilation shifts the oxygen–haemoglobin dissociation curve to the left, thus reducing the amount of oxygen released from haemoglobin to brain tissue.

Cerebral pressure autoregulation: In the normotensive human, autoregulation is operative over a range of mean CPP of 50–160 mmHg (*Figure 2*). Within this range, cerebral vascular resistance will vary directly with CPP to keep CBF constant. In the event of an abrupt change in CPP, blood flow will initially change correspondingly for a brief period (1–5 seconds) before the autoregulatory mechanism returns flow to control levels. As is the case with CO_2 reactivity, some pathological states and certain pharmacological interventions can modify or abolish autoregulation. For example, chronic hypertension shifts the autoregulatory curve to the right, leading to a higher autoregulatory range than observed in normal healthy patients.

While it is reasonable to assume that autoregulation is effective between 50 and 160 mmHg in the majority of patients, this is by no means universal (Bentsen et al, 1975). Upper and lower limits are affected by pathology, drugs and techniques used. For example, pharmacologically-induced hypotension preserves CBF better than haemorrhagic hypotension. Similarly, neuronal function is better preserved at similar levels of hypotension produced by halothane, nitroprusside or isoflurane compared with trimetaphan (Michenfelder and Theye, 1977). It has been demonstrated that symptoms of cerebral ischaemia appear when the mean arterial pressure falls below 60% of an individual's lower autoregulatory threshold (Strandgaard, 1976).

Oxygenation: Transcranial Doppler ultrasonography studies have demonstrated that increases in CBF occur once the PaO_2 falls below 8.5 kPa (~ 89–90% SaO_2) (Gupta et al, 1997a).

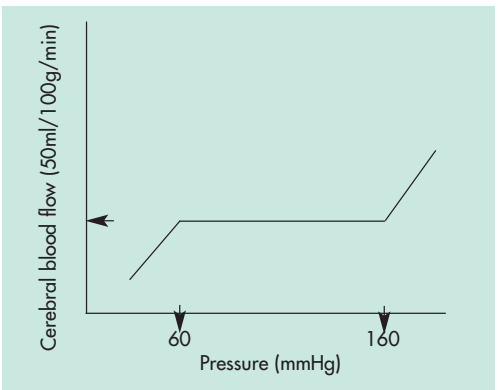


Figure 2. Cerebral pressure autoregulation curve. Cerebral blood flow remains relatively constant in the autoregulatory range of mean arterial pressure of 60–160 mmHg.

Unlike the vascular response to CO_2, the vasodilator responses to hypoxaemia appear to show little adaptation with time (Krasney et al, 1990), but may be substantially modulated by $PaCO_2$ levels (Krasney et al, 1984, 1985). Although hyperoxia may cause cerebral vasoconstriction, this appears to be clinically insignificant (Matta et al, 1994).

CLINICAL NEUROANAESTHESIA

In order to provide the best possible operating conditions, the patient must be optimized. This necessitates a thorough evaluation of the patient, concentrating on current problems, associated risk factors and current medication.

A routine general anaesthetic assessment will include the patient's past medical history, medications, allergies and previous general anaesthetics. The extent of the neurological and myocardial injury are of particular interest in patients who have suffered a subarachnoid haemorrhage (SAH), as many of those patients suffer myocardial injury as a result of the intracranial bleed, which is worse in poor grade patients. In addition, blood results, electrocardiography and echocardiography, when indicated, chest X-ray, computed tomography scans, angiography and transcranial Doppler findings should be available. Although it is not routine practice in the UK to obtain transcranial Doppler measurements before aneurysmal surgery, this is the authors' practice. Proper communication between the anaesthetist and the operating surgeon is likely to lead to the best perioperative care.

The use of sedative premedications is controversial as pre- and postoperative neurological assessment may be difficult. Premedicants may also cause respiratory depression, leading to hypercarbia, hypertension and increased CBF, CBV and ICP. Intravenous midazolam can be administered in the anaesthetic room. Midazolam reduces the cerebral metabolic rate, and hence CBF and CBV without significantly affecting cerebral CO_2 reactivity or autoregulation. However, the agent should be titrated carefully as hypotension must be avoided.

Monitoring

Routine monitoring will include electrocardiography, pulse oximetry, end-tidal capnography, urinary output and temperature. Direct blood pressure measurement allows accurate, beat-to-beat observation of blood pressure and is also useful for intraoperative blood gas and haemoglobin measurements. Central venous pressure is measured in all patients presenting for major neurological surgery to guide fluid management as these patients often receive repeated doses of diuretics and/or mannitol. A pulmonary artery catheter may be required in elderly patients and those with cardiac disease. The authors routinely use a jugular bulb catheter to measure jugular venous oxygen saturation and lactate, and to monitor alterations in ventilation and blood pressure.

Induction of anaesthesia

The aims are to titrate the depth of anaesthesia and the blood pressure to match surgical need, control ICP, minimize cerebral metabolic demands, prevent cerebral ischaemia, ensure good operating conditions and allow rapid awakening. With the exception of ketamine, any intravenous induction agent can be used. Thiopentone, etomidate and propofol are the main agents used for induction of anaesthesia, with muscle relaxation usually achieved with vecuronium, atracurium or pancuronium. When rapid control of the airway is required, suxamethonium can be used, but it may result in a transient increase in ICP (Cottrell et al, 1983). This potential increase in ICP and its possible effect on aneurysmal rupture is balanced against the risk of aspiration, hypoxaemia and hypercapnia.

If a rapid sequence is not required, the patient's lungs are denitrogenated and anaesthesia is induced with the intravenous agent of choice in combination with a short-acting opioid (e.g. fentanyl 1 μg/kg). The agent is titrated to blood pressure and heart rate. There is no place for high dose opioid anaesthesia as this will result in catastrophic hypotension, cerebral vasodilatation, reduced CPP and cerebral ischaemia. Once the patient is judged ready for tracheal intubation (by the use of peripheral nerve stimulator), further aliquots of induction agent, fentanyl, labetalol, esmolol or lidocaine can be used to attenuate the response to laryngoscopy and intubation. The lungs are then ventilated to mild hypocapnia ($PaCO_2$ ~ 4–4.5 kPa). The endotracheal tube is then secured, wide bore intravenous access established and the eyes protected. Local anaesthesia or further doses of induction agent or opioid can be used to attenuate the response to head pin insertion (Colley and Dunn, 1979). Patients are then transferred into theatre and positioned 15–30° head up to aid venous drainage. Once in position, all pressure areas are cared for and thromboembolic prophylaxis (flowtron boots) is instituted.

Maintenance

There is currently no evidence from prospective randomized trials to suggest that a particular anaesthetic technique is superior in patients undergoing neurological surgery. However, the 'best' anaesthetic technique produces a 'slack'

brain so that retraction pressure is low while ensuring maximal cerebral protection by keeping cerebral metabolic requirements to a minimum. Those agents that maintain cerebral vasoreactivity to CO_2 and autoregulation may reduce fluctuations in CBF, ICP and CPP when blood pressure changes with varying surgical stimuli.

A combination of a propofol infusion and an opioid is increasingly used to maintain anaesthesia during neurosurgery. Propofol allows rapid adjustment of anaesthetic depth with more rapid recovery than either thiopentone or isoflurane (Ravussin and de Tribolet, 1993). Propofol has no intrinsic vasodilatory effect and therefore does not result in increases in CBF, CBV or ICP. Furthermore, propofol has been shown not to affect cerebral autoregulation or CO_2 reactivity even at doses high enough to produce EEG isoelectricity (Matta et al, 1995a). It also reduces the cerebral metabolic rate, with cortical structures being depressed to a greater extent than subcortical structures, and may be neuroprotective. Although the reduction in CBF is secondary to flow metabolism coupling and a reduction in metabolism, this coupling may not be perfect and incidences where CBF reduced to a greater extent than metabolism have been reported (Jansen et al, 1999).

Inhalational anaesthetic agents have a dual effect on CBF: a reduction consequential to the decrease in cerebral metabolism and an increase secondary to their direct cerebral vasodilatory effect. The 'net' effect of an inhalational agent on CBF therefore depends on the level of cerebral metabolism at the time the agent is introduced (Matta et al, 1995b). When cerebral metabolism is low, as in patients with SAH grades 3 or 4, the net effect may be vasodilatory, with increases in CBF and ICP accompanying the introduction of the agent. However, in patients with good grade SAH 1 or 2 in whom cerebral metabolism is high, inhalational agents primarily reduce CBF secondary to the reduction in cerebral metabolism, therefore, inhalational agents can be safely used in these patients. When there is uncertainty about the level of cerebral metabolism, or when signs of significant cerebral oedema are present, total intravenous anaesthesia is the preferred option.

With the exception of sevoflurane, inhalational agents impair autoregulation in a dose-dependent manner (Strebel et al, 1995a). Therefore, isoflurane in concentrations less than 1.0% can be used to supplement intravenous anaesthesia. The epileptic activity of enflurane prevents its use in neurosurgery. Desflurane increases ICP and this may be related to its sympathoadrenal effects (Ebert and Muzi, 1993).

Sevoflurane has been shown not to alter cerebral autoregulation in concentrations up to 1.5 MAC (the minimal alveolar concentration — the inhaled anaesthetic concentration at which 50% of patients do not move in response to a defined surgical stimulis) (Katsuyasu et al, 1993; Cho et al, 1996; Gupta et al, 1997b). However, at 1.5 MAC sevoflurane, brain oxygen consumption was reduced by 25% and therefore a degree of luxury perfusion (above metabolic requirements) may occur (Heath et al, 1997). Sevoflurane has a relatively low blood partition coefficient and is non-irritant to the respiratory tract. Because this permits smooth rapid induction and a quick clear-headed recovery from anaesthesia, sevoflurane has largely replaced halothane as the agent of choice for inhalational induction in children. Given its other favourable cerebral haemodynamic properties, sevoflurane might be a useful and safe agent for neurosurgical anaesthesia.

Nitrous oxide has a number of advantages: it has a rapid onset and offset, is easy to use and relatively inexpensive. However, its routine use in neuroanaesthesia is discouraged at the authors' institution as Matta and Lam (1995) found that nitrous oxide increases ICP and CBF by stimulating cerebral metabolism. Although nitrous oxide increased CBF velocity when used in combination with isoflurane (Strebel et al, 1995b), this effect can be attenuated by hyperventilation (Hormann et al, 1995) and propofol (Eng et al, 1992).

Opioids generally have negligible effects on CBF and metabolism. However, the newer synthetic opioids, fentanyl, sufentanil and alfentanil, can increase ICP in patients with tumours and head trauma (Albanese et al, 1993). This increase, originally assumed to be secondary to an increase in CBF, is more likely to be the result of changes in $PaCO_2$ and systemic hypotension (Mayer et al, 1990; Trindle et al, 1993; Werner et al, 1995). Irrespective of the actual mechanism causing the increase in ICP, these observations highlight the importance of administering these agents judiciously and carefully to avoid systemic hypotension. Fentanyl, with its medium duration of action and its negligible cerebral vascular effects, is the agent of choice in many neurosurgical intensive care units. Remifentanil, a new opioid with a rapid onset and short half-life, is being investigated for neurosurgery. Remifentanil appears to compare favourably with fentanyl in patients undergoing elective supratentorial surgery (Guy et al, 1997). The authors have shown that remifentanil, when combined with 0.5 MAC sevoflurane, does not alter cerebral autoregulation in individuals undergoing non-intracranial neurosurgical procedures (Godsiff et al, 1998).

Brain relaxation: The aim is to produce a 'slack' brain so that retraction pressure can be kept to a minimum. Several methods are employed to reduce brain bulk, CSF volume and CBV. These include a 15–30° head-up position, mild hypocapnia (~ 4–4.5 kPa), mannitol and frusemide.

Mannitol is an osmotic diuretic used to reduce cerebral tissue water (Pollay et al, 1983; Archer, 1996). It is usually administered (0.5–1.0 g/kg) as a 20% solution. Mannitol probably acts on all three intracranial compartments via different mechanisms. It may reduce brain bulk by osmotic dehydration, CBV by improving rheology of red blood cells, thus decreasing blood viscosity (Muizelaar et al, 1983), and CSF production (Sahar and Tsipstein, 1978; Donato et al, 1994). Mannitol is also a free radical scavenger. Mannitol's high osmolarity causes an immediate but transient increase in intravascular volume, CBF, CBV and ICP. This is followed by a reduction in ICP and CBV, which is at a maximum at 45–60 minutes. Therefore, care must be taken when administering mannitol to patients with poor cardiac function as they may develop congestive cardiac failure and pulmonary oedema. Frusemide can be used in conjunction with mannitol, or it can be used alone in those patients with poor myocardial function who may be sent into cardiac failure with mannitol (Pollay et al, 1983). Hypertonic saline has been advocated as an alternative to mannitol, although its action is transient and its overall effects remain untested.

The potential ischaemic effects of marked hyperventilation must be balanced against the benefits of reducing CBV. When used properly, hyperventilation is a quick and effective tool for reducing CBV, provided a measure of cerebral oxygenation is employed. Although it cannot detect regional ischaemia, jugular bulb oxygen saturation (SJO_2) will reflect the balance between cerebral oxygen supply and demand. It is probably unwise to induce hypocapnia if the SJO_2 is <50%.

CBV can also be reduced pharmacologically by reducing cerebral metabolism and hence CBF. This is achieved by bolus intravenous administration of thiopentone (3–5 mg/kg), propofol (1–2 mg/kg) or lignocaine (1.5 mg/kg). If brain condition improves after the bolus, a continuous infusion is started.

Cerebral protection: Various cerebroprotective methods have been used including hypothermia, additional doses of intravenous or inhalational anaesthetics, deliberate hypertension and drugs such as mannitol, phenytoin and calcium channel-blockers (Warner, 1994). These methods may be used routinely or their use may be guided by changes to the EEG or evoked potentials. Although the exact mechanism for neuroprotective action of anaesthetic agents is not fully understood, barbiturates and propofol may produce their effect by reducing the cerebral metabolic rate. However, doses of intravenous anaesthetic high enough to suppress EEG activity cause marked cardiovascular depression (Todd et al, 1987). The cerebral protective effects of barbiturates may be secondary to their ability to reduce calcium influx, inhibit free radical formation, potentiate γ-aminobutyric acid (GABA)-ergic activity, reduce cerebral oedema and inhibit glucose transfer across the blood–brain barrier.

In contrast to pharmacological agents which only reduce the active component of cerebral metabolism, moderate (32–35 °C) hypothermia reduces both the active and basal components thereby increasing the period of ischaemia tolerated (Steen et al, 1983; Doppenberg and Bullock, 1997). There is no clinical evidence for its use which is based on animal models. However, trials are currently being performed. Cerebral metabolism is approximately 15% of normal at 20 °C. It is now commonly accepted that hypothermia may be neuroprotective because it affects factors other than cerebral metabolism, e.g. cytokines, free radicals and glutamate (Lei et al, 1997; Marion et al, 1997; Bart et al, 1998).

Problems associated with hypothermia include reliability of temperature measurement, the optimal temperature needed to offer the best benefit–risk ratio, and the best method of rewarming the patient safely (Cheney et al, 1994) so that normothermic temperatures are reached before emergence. Other problems include delayed awakening, postoperative shivering, coagulation disorders and aggravation of myocardial disease.

Hyperglycaemia (leading to lactic acidosis) has been shown to correlate with poor outcome after neurological injury (Lam et al, 1991). Therefore, as part of cerebral protection, the use of glucose-containing solutions is discouraged unless hypoglycaemia is suspected. Hyperglycaemia should be actively treated and blood glucose levels controlled with an infusion of insulin.

Recovery: In patients with initial good neurological state, a rapid return of consciousness is aimed for to allow early neurological assessment. Provided no problematic events occurred intraoperatively, these patients are extubated. Ideally, all postoperative neurosurgical patients extubated at the end of the procedure should be monitored in a high dependency area. However, in reality, many of the patients are transferred to neurologic wards where the staff are skilled in observing subtle changes in neurology. Patients with poor preoperative conscious level and ven-

tilatory state are usually transferred to the neurocritical care unit for a 24–48-hour period of elective postoperative ventilation.

When surgery is complete, the anaesthetic agents are discontinued and 100% oxygen is given. Residual neuromuscular blockade is reversed, the airway suctioned and the patient extubated on regaining consciousness. Boluses of short-acting opioids, propofol or lignocaine can be used to facilitate extubation and control the blood pressure. Uncontrolled hypertension in the immediate postoperative phase can precipitate intracerebral haemorrhage. In general, the blood pressure is kept to within 20% of normal. If the patient remains hypertensive (systolic pressure greater than 200 mmHg) in recovery despite adequate pain relief, esmolol, labetalol or nifedipine are used.

CONCLUSIONS

The maintenance of adequate cerebral perfusion, the avoidance of hypotension and the prevention and treatment of raised ICP are essential for good recovery in patients undergoing neurosurgery. Therefore, clinicians caring for these patients must have a thorough understanding of cerebral physiology and the factors that affect cerebral haemodynamics. HM

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KEY POINTS

- Cerebral blood flow is determined by the cerebral perfusion pressure, which is the difference between mean arterial blood pressure and intracranial pressure.
- Cerebral autoregulation is a sensitive mechanism which maintains brain blood flow constant when cerebral perfusion pressure changes. This mechanism is abolished in a dose-dependent manner by halothane, isoflurane, desflurane but not sevoflurane.
- During neurosurgery, the aims are to titrate the depth of anaesthesia and the blood pressure to match surgical need, ensure good operating conditions and allow rapid awakening.
- Although moderate hypothermia (32–35 °C) has been shown to be neuroprotective in animal models of cerebral ischamia, its efficacy as a first-line treatment in brain injury humans has not been demonstrated so far.
- Optimizing cerebral perfusion pressure, avoidance of hypotension and the prevention and treatment of raised intracranial pressure are essential for good recovery in patients undergoing neurosurgery.

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In the Public's View...

The same the world over

At last, we are able to do tonsillectomies again. Faced with an unknown risk of instruments contaminated forever with indestructible prions, the decision was made that all instruments used for the operation must be disposable.

Travel broadens the mind. Travel also makes me realize how narrow minds are or, at least, how narrow are the rails on which minds run. Travelling over the last few months for work or pleasure to the USA and Italy, and meeting people from Australia and Canada, has made me realize that we're all in this together. The *Adirondack Explorer* is not the world's best known newspaper. It's not even the best known newspaper in the USA or even in New York state, which is where the Adirondacks are, but its lead story in May was of an orthopaedic surgeon forced to resign after operating on the wrong knee. The *Adirondack Daily Enterprise* had another medical headline, 'Hospital must tell patients about mistakes'. This idea has gone so far that the American Hospital Association is advising that patients in their hospitals be told even about mistakes that don't cause any harm. This advice is the eventual fallout of an Institute of Medicine report published in 1999, which estimated that up to 100 000 patients die each year in American hospitals because of medical errors.

And in Italy? Italian anaesthetists at a conference in May were talking of the requirement for patients to be more directly involved in their treatment. Australian and Canadian anaesthetists spoke of the same pressures.

It's all too easy, when the government berates us and compares the NHS unfavourably with other countries, to feel hounded and isolated. We should not feel like that. It's the same the whole world over — except in those parts of the world where things are even worse. The phrase 'paradigm shift' is one of those clichés that people apply to changes to make them sound more important than they really are. Few people actually know what a paradigm is, and even fewer have read what the originator of its shifting actually wrote, so I shall refrain from using the phrase now. But there are big changes occurring in the relations between different groups in society, and the idea of authority is mutating. It is not just that we no longer doff our caps to the socially more advantaged; even the authority that used to come from experience and knowledge is questioned.

I take comfort that there has been little anti-doctor invective in the UK for a little while now. Politicians may have realised that it is best not to destroy all authority. The Department of Health has published a glossy document about ensuring quality and pursuing excellence which, although it does little more than regurgitate well-worn platitudes, recognizes the malign effect of the media (although not the politicians' role in encouraging this) and is co-signed by a number of influential doctors.

As I write, the publication of the Bristol paediatric heart surgery enquiry is due. If the government fails this test, I retract all I said, and suggest we retreat further into paranoia. HM

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