ume teaching unit, on a regular basis, in order to maintain their expertise and to learn new techniques.

The second concern relates to the management of potential complications when an anaesthetist may not be physically present. This concern also relates to higher volume units where the anaesthetist may be "tied up" in an operating room with a surgical case. In fact the guidelines actually contain the answer in that they state that "an appropriate protocol for the management of these epidurals is in place."[1]

I would suggest that the setting up of these protocols is the most important aspect of this document. It is essential that the nurses caring for these parturients be educated in the recognition of complications and their initial management. This education, in the low-volume units, must not be a one-time event. It should be repeated and reemphasized at regular intervals. Additionally, other physicians who might be physically present in the hospital should be educated and trained in the management of these complications. This would include obstetricians, family and emergency physicians.

The other recommendation is that all hospitals practicing obstetrics should have a regular program of CPR training with emphasis on the parturient.[3] The importance of uterine displacement in critical situations may not be recognized unless specifically included in the protocols. The normal response to finding a hypotensive, apnoeic patient is to place him/her supine in the Trendelenburg position. This could prove fatal in the parturient.

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REFERENCES

1 Appendix IV Epidural analgesia during childbirth. *In*: Guidelines to the Practice of Anaesthesia as Recommended by the Canadian Anaesthetists' Society. Revised Edition.
2 *Palahniuk RJ*. Obstetrical epidural anaesthesia in remote hospitals (Editorial). Can J Anaesth 1988: 35: 488–50.
3 *Rees GAD, Willis BA*. Resuscitation in late pregnancy. Anaesthesia 1988; 43: 347–9.

Anaesthesia and myasthenia gravis

To the Editor:

I was disappointed that the comprehensive review article by Baraka[1] made little or no mention of the benefits of combined regional/general anaesthesia in these patients. Although Dr. Baraka pointed out that the use of regional or local anaesthesia "seems warranted in these patients," he did not expand on this concept when discussing the management of retrosternal thymectomy.

A thoracic or high lumbar epidural anaesthetic in combination with balanced general anaesthesia provides excellent analgesia both intraoperatively and during the period following the thymectomy.[2] This decreases the stress response to the sternotomy, decreases opioid requirements and perhaps even the dose of neuromuscular blocking agents. This in turn allows these patients to meet extubation criteria earlier and in some individuals prevents the need for postoperative ventilation.

The current trend for anaesthetists to emphasize aggressive pain management,[3] not only for aesthetic reasons, has seemed to miss a beat in this article. I hope this letter may perhaps remind readers of the obvious benefits of a combined regional/general anaesthetic technique, using an indwelling epidural catheter for retrosternal thymectomy.

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REFERENCES

1 *Baraka A*. Anaesthesia and myasthenia gravis. Can J Anaesth 1992; 39: 476–86.
2 *Kirsch JR, Diringer MN, Borel CO, et al.* Preoperative lumbar epidural morphine improves postoperative analgesia and ventilatory function after transsternal thymectomy in patients with myasthenia gravis. Crit Care Med 1991; 19: 1474–9.
3 Commission on the Provision of Surgical Services. Report of the Working Party on Pain after Surgery. The Royal College of Surgeons of England and College of Anaesthetists. September 1990.

REPLY

Thank you for referring to me the letter of Dr. Gambling. I agree with him that thoracic or simply high lumbar epidural analgesia using local anaesthetic and/or narcotic, in combination with a balanced general anaesthesia, provides excellent analgesia both intraoperatively and postoperatively. The benefits of this combination are not limited to myasthenic patients undergoing thymectomy[1] but also apply to other patients undergoing any thoracic[2] or major abdominal and vascular surgery.[3] However, the concept is still controversial.[4] Many of our patients do not accept the idea of having a needle or a catheter in their "back" as a modality for pain relief. Also, because of the possible delayed respiratory depression, all patients having epidural morphine must be observed in an ICU setup for at least 12–24 hr postoperatively.

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REFERENCES
1 *Kirsch JR, Diringer MN, Borel CO, et al.* Preoperative lumbar epidural morphine improves postoperative analgesia and ventilatory function after transsternal thymectomy in patients with myasthenia gravis. Crit Care Med 1991; 19: 1474–9.
2 *Shulman M, Sandler AN, Bradley JW, et al.* Post thoracotomy pain and pulmonary function following epidural and systemic morphine. Anesthesiology 1984; 61: 569–75.
3 *Yeager M, Glass D, Neff R, et al.* Epidural anesthesia and analgesia in high-risk surgical patients. Anesthesiology 1987; 66: 729–36.
4 *Shenaq S.* Con: epidural anesthesia is not a valuable adjunct to general anesthesia for abdominal vascular surgery. Cardiothoravasc Anesth 1989; 3: 509–12.

Perioperative stroke

To the Editor:
While reading DHW Wong's extensive and informative two-part review "Perioperative Stroke,"[1,2] I came across a statement in Part I which troubled me, not because of what was said but by what was implied: "Halothane produces cerebral vasodilation with an increase in cerebral blood flow and increased intracranial pressure. On the other hand, isoflurane reduces cerebral oxygen consumption, preserves the normal energy state, and has minimal effect on intracranial pressure.[228-233]"

In fact, things are not so clear as this. Dr. Michenfelder (Dr. Wong's reference 231), concluded that reasons for the apparent benefit from isoflurane in carotid endarterectomy were not as obvious as this. While most authors agree that halothane increases cerebral blood flow more than isoflurane, agreement on their effects on intracranial pressure is not as universal. For example, Kaieda *et al.*, [3] in a rabbit model with a cryogenic cortical injury, were unable to demonstrate any difference in intracranial pressure at 1 MAC of isoflurane or halothane, or an equal dose of pentobarbitol. Mutch *et al.*[4] in a similar model, found no difference in intracranial pressure until mean arterial pressure had been increased 60% above baseline with phenylephrine.

Particularly given the difference in distribution of cerebral blood flow effected by the two agents,[5] I believe that Todd and Drummond are correct when they conclude that "the simple fact that isoflurane produced consistent dose-related increases in intracranial pressure ... indicates that it should be used with caution in situations where intracranial compliance is compromised..."[6]

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REFERENCES
1 *Wong DHW.* Perioperative stroke. Part I: General surgery, carotid artery disease, and carotid endarterectomy. Can J Anaesth 1991; 38: 347–73.
2 *Wong DHW.* Perioperative stroke. Part II: Cardiac surgery and cardiogenic embolic stroke. Can J Anaesth 1991; 38: 471–88.
3 *Kaieda R, Todd MM, Weeks JB, Warner DS.* A comparison of the effects of halothane, isoflurane and pentobarbitol anesthesia on intracranial pressure and cerebral edema formation following brain injury in rabbits. Anesthesiology 1989; 71: 571–9.
4 *Mutch WAC, Patel PM, Ruta TS.* A comparison of the cerebral pressure-flow relationship for halothane and isoflurane at haemodynamically equivalent end-tidal concentrations in the rabbit. Can J Anaesth 1990; 37: 223–30.
5 *Hansen TD, Warner DS, Todd MM, et al.* Distribution of cerebral blood flow during halothane versus isoflurane anesthesia in rats. Anesthesiology 1988; 69: 332–7.
6 *Todd MM, Drummond JC.* A comparison of the cerebral, vascular and metabolic effects of halothane and isoflurane in the cat. Anesthesiology 1984; 60: 276–82.

REPLY

Dr. Gilmour was quite correct in pointing out that the observed effects of halothane and isoflurane on intracranial pressure (ICP) are not uniform. In neurosurgical patients, for example, isoflurane anaesthesia has been shown to produce no significant effect on ICP.[1] In animal studies, when compared with equipotent concentrations of halothane, isoflurane produces lower[2,3] or similar ICP levels.[4-7] The observed differences may be due to differences in species studied, experimental design, instrumentation, supplementary anaesthetic drugs, and concomitant drug therapy. Furthermore differences in MAP and $PaCO_2$, presence or absence and type of ischaemic injury may have overriding effects. ICP changes are important as they directly influence cerebral perfusion pressure (CPP), which equals the difference between mean arterial pressure (MAP) and ICP.

Todd and Drummond[5] showed that in cats, halothane produced greater increases in CBF and less decreases in $CMRO_2$ than isoflurane. In spite of the differences in CBF, the ICP changes produced by the two agents were similar. Since ICP changes should follow the changes in cerebral blood volume (CBV), there appears to be a dissociation between the effects of anaesthetics on CBF and CBV. One explanation is that the study animals with no intracranial pathology may have high cerebral compliance.[6,8] Another explanation is that halothane affects the inflow and outflow resistance equally, whereas isoflurane has less effect on the arterioles and even less effect on the outflow resistance, thus allowing accommodation of a larger CBV and resulting in higher ICP at lower CBF.[4,5] It is also possible, since the xenon clearance method used measures, predominantly, cortical CBF, that the observed differences in CBF were not representative of whole-brain differences.[9]

It has been suggested that the control of CPP may not be crucial during light isoflurane anaesthesia, as the uncoupling of local flow and metabolism by isoflurane is dose-dependent.[10-12] Isoflurane produces less cerebral vasodilation compared to enflurane and halothane, and might therefore cause a relatively