toxicity related to the central nervous and cardiovascular symptoms.[6] Clinical trials have documented side effects when low-dose bupivacaine is administered with epidural morphine.[7] Of the references cited by Drs. Maier and Wulf to support their statement, one is not yet published and the other reports the occurrence of high epidural blocks as well as one intrathecal migration of an epidural catheter.[8] While no serious morbidity resulted from these events, you cannot use this as evidence that epidural local anaesthetics are safer than epidural opioids.

Despite the above concerns, we believe the combination of low-dose epidural bupivacaine and fentanyl or morphine does improve the efficacy of epidural analgesia and is safe on general postoperative wards provided the nursing staff are appropriately trained, monitoring protocols established, and physician assistance is available 24 hours a day. It is also our impression that since our survey was done, more centres in Canada are using epidural opioids alone or in combination with low-dose bupivacaine to improve the management of postoperative pain.

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REFERENCES

1 *Maier C. Wawersik J, Wulf H.* Ergebnisse einer Fragebogenerhebung zur Praxis und Organisation der postoperativen Periduralanalgesie an 461 Fachabteilungen für Anästhesiologie. [Results of a questionnaire survey of the practice and organization of postoperative peridural analgesia 461 anesthesial departments]. Reg Anaesth 1991; 14: 61–9.
2 *Ready LB, Oden R, Chadwick HS, et al.* Development of an anesthesiology-based postoperative pain management service. Anesthesiology 1988; 68: 100–6.
3 *Ready LB, Edwards WT.* Postoperative care following intrathecal or epidural opioids. II. Anesthesiology 1990; 72: 213.
4 *Ready LB, Loper KA, Nessly BS, Wild L.* Postoperative epidural morphine is safe on surgical wards. Anesthesiology 1991; 75: 452–6.
5 *Baxter AD.* Editorial: Respiratory depression with patient-controlled analgesia. Can J Anaesth 1994; 41: 87–90.
6 *Grichnik K, Ginsberg B.* Epidural analgesia for patients recovering from surgery. *In*: Sinatra RS, Hord AH, Ginsberg B, Preble LM (Eds.). Acute Pain: Mechanisms and Management. Chicago: Mosby Year Book, 1992.
7 *Jayr C, Thomas H, Rey A, et al.* Postoperative pulmonary complications; epidural analgesia using bupivacaine and opioids versus parenteral opioids. Anesthesiology 1993; 78: 666–76.
8 *Schug SA, Torrie JJ.* Safety assessment of postoperative pain management by an acute pain service. Pain 1993; 55: 387–91.

Plasma cholinesterase activity in infants

To the Editor:

We have read the Case Report by Pasquariello and Schwartz,[1] describing plasma ChE deficiency in a two-day-old neonate. Indeed, this is the youngest reported patient to exhibit apnoea after succinylcholine. Laboratory testing confirmed plasma cholinesterase deficiency as the mechanism for prolonged neuromuscular relaxation. We would like to bring to the attention of readers that some neonates can have decreased plasma cholinesterase activity within the first two weeks of life. This activity usually reaches normal levels within one month of age.[2] With respect to the infant described in the Case Report, analysis of plasma ChE activity should be made at an older age before a definitive diagnosis of cholinesterase deficiency is made. This is especially important since both parents and an older sibling had normal cholinesterase activity. Genetic analysis of the neonate's DNA would confirm the presence of a silent gene.

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REFERENCES

1 *Pasquariello CA, Schwartz RE.* Plasma cholinesterase deficiency in a neonate. Can J Anaesth 1993; 40: 529–31.
2 *Strauss AA, Modanlon HD.* Transient plasma cholinesterase deficiency in preterm infants. Dev Pharmacol Ther 1986; 9: 82–7.

REPLY

We thank Drs. Vassallo and Goudsouzian for their comments. We have been attempting to contact the family for a follow-up plasma cholinesterase level (PChE). Strauss[1] reported a group of premature infants, 16% of whom had abnormally low PChE. The lowest value he reported in that group was 4 U· ml^{-1} (normal >7 U· ml^{-1}) or approximately 57% of normal. This included the values of a pair of twins with persistently low PChE, thought to be a genetic abnormality. Zsigmond and Downs[2] found the mean PChE activity of infants and newborns to be approximately 50% of adults. These results double and approach adult values, by several weeks of age. Because the PChE level of our patient[3] was 0.2 U· ml^{-1} (5.8% of normal), we felt that this showed practically no PChE activity and an increase of 10–20-fold to reach "normal" values would be highly unlikely given the above data. If we are able to obtain a follow-up plasma cholinesterase level on our patient, we will report our findings.

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REFERENCES
1 *Strauss AA, Modanlou.* Transient plasma cholinesterase deficiency in preterm infants. Dev Pharmacol Ther 1986; 9: 82–7.
2 *Zsigmond EK, Downs JR.* Plasma cholinesterase activity in newborns and infants. Can Anaesth Soc J 1971; 18: 278–85.
3 *Pasquariello CA, Schwartz RE.* Plasma cholinesterase deficiency in a neonate. Can J Anaesth 1993; 40: 529–31.

Drug labels on anaesthesia carts

To the Editor:
In the January edition of the Journal, Drs. Boldt and Renwick[1] stated that they had identified a safety hazard associated with 1 millilitre glass vials produced by Sabex Inc. The problem which they have identified occurs not only with Sabex.

We agree that medication error is an important cause of patient morbidity, and that to eliminate medication error there can be no substitute for reading a label. It is, however, the responsibility of hospital pharmacies and departments of anaesthesia to take special precautions to assure that medications with similar labeling are not placed in anaesthetic carts. Institutions and governments now frequently choose to purchase pharmaceuticals from the manufacturer who can supply product at the best price. Anaesthetists and pharmacists must take a proactive role in assuring that ampoules of similar colour and labeling are not placed side by side. In our hospital, we have a cooperative arrangement between the Department of Anaesthesia and the Pharmacy, assuring that changes in product are not introduced to the operating room without prior consultation. We have agreed that we will not place ampoules of similar size and colour on our anaesthetic carts where possible; to that end, we have occasionally requested a change in supplier simply to modify the size and colour of an ampoule. We have also occasionally requested a change in product concentration (e.g., atropine 0.4 $mg \cdot ml^{-1}$ instead of atropine 0.6 $mg \cdot ml^{-1}$) specifically to obtain a different colour of ampoule. Where we previously had ampoules of atropine, adrenaline, and heparin all of the same size and colour, they are now very different from one another.

In discussing the problem of labeling of ampoules with manufacturers, we have found them sympathetic to our objective of reducing medication error, but reluctant to change their labeling to meet the needs of individual institutions. Our experience with Sabex Inc., however, has been positive and we concur with Mrs. Levesque's response.

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REFERENCE
1 *Renwick JE, Boldt C.* Safety hazard – Sabex drug labels (Letter). Can J Anaesth 41: 1: 75–6.

Anaesthesia for cardiac transplant patients

To the Editor:
We read with interest the article of Cheng and Ong[1] concerning anaesthesia for non-cardiac surgery in heart-transplanted patients. We would like to address the issue of the reported lack of effect of anticholinesterases on heart rate. The authors observed that "Paralysis was easily reversed with neostigmine with or without atropine. No significant effect on heart rate was recorded," and later claim that " ... heart rate shows no response to ... anticholinesterases (neostigmine, edrophonium, pyridostigmine, physostigmine) ... " While this view seems to be generally accepted[2–4] we have demonstrated in cats that neostigmine can still evoke a marked, dose-dependent bradycardia when autonomic efferent activity to the heart is interrupted. The mechanism by which this occurs appears to involve direct activation by neostigmine of excitatory cholinergic receptors on cardiac ganglion cells, which results in release of acetylcholine from their terminals and subsequent activation of inhibitory cardiac receptors.[5] We have also demonstrated that clinically relevant doses of neostigmine produce an atropine-sensitive bradycardia in both recently and remotely transplanted patients.[6] We wish to draw attention to our observations that neostigmine can produce a clinically significant bradycardia in the heart transplant patient and we suggest that muscarinic antagonists be administered routinely with reversal agents to block cardiac and other muscarinic side effects of the anticholinesterases.

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