*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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# *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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## *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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