alothane 0.5 to 1 per cent. She was allowed to breathe spontaneously. The recovery period was identical to that of Case 2, except that she failed to respond to verbal command on admission to the Recovery Room. She also responded well to diazepam.

There was no hypoxia or hypercarbia during any of the anaesthetic procedures.

Prolonged sleep and rigidity with apnoea and cyanosis should now be added to the list of known reactions to Alfathesin.

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SERUM CHOLINESTERASE ACTIVITY FOLLOWING ANAESTHESIA

SIR,

I was interested to read the article on serum cholinesterase activity following enflurane anaesthesia by Kaniaris, Fassoulaki and Liarmakopoulou in the November 1978 issue of the Canadian Anaesthetists' Society Journal. In a smaller, less well controlled study, I have reached the same conclusion, demonstrating an initial small drop in cholinesterase activity one hour after starting enflurane anaesthesia, with return to normal 24 hours later. In addition to ten patients having enflurane, I also looked at five patients having halothane and five patients having spinal anaesthetics.

The 20 patients, ranging in age between 24 and 83 years were premedicated with diazepam 10–20 mg orally and were undergoing simple urological, orthopaedic and general surgical procedures in which the average estimated blood loss was less than 50 ml. Those patients having general anaesthesia were given thiopentone 250–500 mg and anaesthesia was maintained by

spontaneous ventilation, through a circle carbon dioxide absorber circuit, with 66 per cent nitrous oxide with oxygen and either 1 to 3 per cent of enflurane or 1 to 2 per cent halothane. Those patients having spinal anaesthetics were given a subarachnoid injection of tetracaine 12–15 mg in 10 per cent dextrose. All patients had an intravenous infusion of lactated Ringer's solution of which, in the first two hours, the enflurane patients received 400 ml, the halothane patients 500 ml and the spinal patients 900 ml. The average length of anaesthetic was 56 minutes with enflurane and 76 minutes with halothane, while in the spinal anaesthetic group the average operating time was 67 minutes. Blood samples were taken preoperatively and at one hour, two hours, four hours and 24 hours after the start of anaesthesia. Serum was obtained by centrifugation of clotted blood and it was then frozen until an estimation of cholinesterase level could be made, which was done using acetyl thiocholine as a substrate.

The results are summarised in the Table.

Thus it appears that halothane and spinal anaesthesia should be considered with enflurane and methoxyflurane¹ as capable of causing a small acute drop in serum cholinesterase activity, which returns to normal within 24 hours.

An older paper by McIntyre and Campbell² was primarily concerned with the effects of neostigmine on serum cholinesterase activity during a thiopentone, succinyl choline, nitrous oxide, oxygen, halothane and curare anaesthetic, which was not shown by patients who had fentanyl and droperidol rather than halothane. In Palahnuik's methoxyflurane study, control patients who had thiopentone, succinyl choline, nitrous oxide and oxygen, and curare, showed levels of cholinesterase activity to be slightly depressed initially, but raised above normal at two hours and 24 hours.

Another study³ suggests that an initial drop in cholinesterase activity is common following

SERUM CHOLINESTERASE ACTIVITY

	Preop	1 hour	2 hours	4 hours	24 hours
Enflurane (10 patients)	3452	3130	3274	3224	3442
Halothane (5 patients)	2627	2483	2863	2517	2946
Spinals (5 patients)	3431	3287	3210	3173	4363

surgery and anaesthesia. Perhaps the effects of surgery and anaesthesia on liver perfusion are more important than the effects of anaesthetic drugs on enzyme activity.

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