CONVULSIONS FOLLOWING SURGERY

Sir,—A fit African female, 35 years of age, weighing 51 kg, having no history of previous fits, was admitted for hysterectomy for menorrhagia due to large multiple fibroids. She received pethidine 50 mg and atropine 0.6 mg intramuscularly 1 hour before operation.

In the operating theatre an intravenous infusion was started and an upper arm cuff applied. The systolic arterial pressure, measured by aneroid manometer, was 110 mm Hg. With the patient in the lateral position, a 16-gauge spinal needle was introduced aseptically into the epidural space at L2-3 interspace through the lateral approach, using loss of resistance with air technique. When no blood or cerebrospinal fluid appeared, 18 ml of 0.5 per cent bupivacaine with 1:200,000 adrenaline was injected at 8.40 a.m.

Seven minutes after the epidural injection the arterial pressure was 120 mm Hg systolic and, having ascertainment that movement of the toes was still present, sleep was induced with methohexital 1 per cent and maintained with 30 per cent oxygen with nitrous oxide and minimal trichloroethylene; the systolic pressure now was 90 mm Hg. Ten minutes after the epidural injection the table was tilted at 30° Trendelenburg and operation started. At 9.10 a.m. the arterial pressure fell from 85 to 40 mm Hg following rapid blood loss, this was treated by methotamine 10 mg intravenously and rapid infusion of 500 ml plasma expander (Haemocel) followed by 500 ml blood. Within 5 minutes the arterial pressure returned to 70 and settled at 80 mm Hg. At no time was the patient pulseless and apparently normal spontaneous respiration continued (the inspired oxygen concentration was increased to 50 per cent).

The hysterectomy was completed at 9.50 a.m. and the patient was allowed to wake up; she answered questions, was able to move her toes but not her legs. Her systolic pressure was 90 mm Hg and she was detained in the recovery ward on oxygen and the foot of the bed was kept elevated. At 10.40 a.m. the patient had a convulsion, followed, after a short lucid interval, by another convulsion starting with twitching of the face and becoming generalized. These were treated by giving 100 per cent oxygen via a face mask, thiopentone 100 mg and frusemide 40 mg intravenously. Because the tidal volume appeared to be small, tracheal intubation was performed with the aid of suxamethonium 50 mg and IPPV instituted using a Cyclator ventilator giving 50 per cent oxygen and nitrous oxide. Two further convulsions recurred both treated with thiopentone 100 mg.

At 3.30 p.m. adequate spontaneous respiration was present and IPPV was discontinued and the trachea extubated. The patient was conscious but confused, but no paresis was noted. She was returned to the ward and given 30 per cent oxygen via an Edinburgh mask. At 7 p.m. the patient had two convulsions treated with thiopentone 100 mg intravenously. A further convulsion occurred at 7.30 p.m.; diazepam 10 mg was given intravenously. The systolic arterial pressure had remained above 90 mm Hg throughout the post-operative period and good urinary output had been maintained. No further convulsions occurred and the patient’s confused mental state improved in the next 24 hours, although phenobarbitone 30 mg was given prophylactically for the next three days. She made a slow recovery, complicated by urinary infection, and was discharged on the 12th postoperative day. There was no obvious neurological impairment, but intelligence and psychological assessment were not performed. When she was seen four weeks after discharge she appeared fully recovered from her experience.

The occurrence of convulsions after epidural anaesthesia may be attributed to the toxic effects of the absorbed drug, or to cerebral hypoxia due to hyperventilation or hypotension. In this case the convulsions occurred 2 hours after the epidural injection, thus excluding the possibility of occurrence of intravascular or subarachnoid injection of the drug. In this case the adoption of the Trendelenburg position 10 minutes after the epidural injection may have promoted cephalad displacement of the drug, causing some hyperventilation and hypotension which became marked with rapid blood loss. Thus, these convulsions of late onset could have been due possibly to the occurrence of either cerebral hypoxia causing transient central depression or of hepatic hypoxia causing very slow breakdown of bupivacaine, or may have been coincidental.

F. F. CASALE
Lusaka

EMO VAPORIZER: EXPIRATORY VALVES

Sir,—We would like to reassure Drs D. E. Das Gupta and D. B. Deval (correspondence, Brit. J. Anaesth. (1970), 42, 808) that the faults they report in the expiratory valves supplied with the EMO vaporizer have already been corrected.

In fact the valve to which they refer must have been manufactured three or four years ago and is in no sense "new". This design was an attempt to produce a valve whose operation was more obvious to the less skilled EMO user but it was quickly realized that it was not satisfactory and only about 100 were produced.

We have already replaced the majority of these valves with our current, more orthodox, model. We would like to use your columns to extend to other EMO users who may still have expiratory valves of the type described by Dr Das Gupta, the offer of free replacement.

B. R. SUGG
for Longworth Scientific Instrument Co. Ltd

HYPERTONIC SYNDROME ASSOCIATED WITH SUXAMETHONIUM ADMINISTRATION

Sir,—A more likely cause of the profound circulatory depression noted in the case report of Dr Daviea (Brit. J. Anaesth. (1970), 42, 656) would be hyperkalaemia subsequent to suxamethonium administration noted in patients with various neurological and muscular diseases.

L. E. COOPERMAN
Philadelphia

REFERENCE