CORRESPONDENCE

cryotherapy is considered to be of little value in the treatment of elapid bites (Minton, 1974). In Australia many hundreds of snake-bite victims are treated successfully with antivenom every year. Artificial ventilation is necessary only if antivenom has been delayed unduly. Deaths from snake bite in Australia in recent years have all resulted from failure to administer antivenom, or from an inadequate dose of antivenom or the use of the wrong antivenom (Sutherland, 1977). Pleas have been made in editorials for the better management of snake bite (Editorial, 1975; Editorial, 1978).

In their paper, Naphade and Shetti do not disclose the species of snake, give details of the source, specificity, quantity or potency of the antivenom, or state when antivenom was given and how much later the neostigmine was administered. This lack of information implies that the writers doubt the efficacy of antivenom, yet they list in detail a number of dangerous venom components it is known to neutralize. Since antivenom was given there is no evidence to show that this was not the cause of the child's improvement.

They present no evidence that antivenom is responsible for the neuromuscular block, rather than the neurotoxins. We believe that neostigmine might be of slight help in the minority of snake-bite cases after adequate treatment with antivenom. Neostigmine will not improve victims of the bite of snakes of which the venom has potent neurotoxins which act presynaptically. A number of the most dangerous snake venoms have been found to be predominantly presynaptic in action, and these include the Asian krait (Bungaris multicinctus), the Australian tiger snake (Notechis scutatus) and the taipan (Oxyuranus scutellatus). There is no place for the use of neostigmine in the management of snake bite in Australia. We believe it has not been demonstrated to be of use in the treatment of snake bite in other countries.

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REFERENCES


DENTAL ANAESTHESIA

Sir,—Having read Dr Scurr's letter (1978) relating to improvements to training schemes for dental anaesthesia and sedation in the United Kingdom, I thought your readers may be interested to know of some recent developments in Australia.

In November 1975, the Health Commission of New South Wales appointed a committee to investigate the use of relative analgesia, i.e. sedation and general anaesthesia in dental practice. Its report (Wright et al., 1978) contains many recommendations including the establishment of a Department of Clinical Pharmacology and Anaesthesia within the Faculty of Dentistry at Sydney University. The Committee has accepted that the usage of relative analgesia, i.e. sedation and general anaesthesia is part of the present-day practice of dentistry, and it has recommended that dentists who wish to include general anaesthesia in their clinical practice be given the opportunity to complete a suitable course of instruction which would include residency at a major teaching hospital.

The Australian Society for the Advancement of Anaesthesia and Sedation in Dentistry (A.S.A.A.S.D.) will commence an entirely new teaching programme in 1979 which is designed to equip general dental practitioners with the necessary skills to use safe, simple techniques. The programme consists of six units of instruction and each candidate will be required to:

(a) complete each unit in its correct order;
(b) work for one year with a recognized practitioner;
(c) be evaluated by a review committee at the completion of the programme.

This programme has been approved by the Australian Dental Association, the Faculty of Anaesthetists of the Royal Australasian College of Surgeons and the Oral Surgeons Society of N.S.W. Full details of this new programme were published recently (Sheridan et al., 1978) and further information can be obtained by writing to me.

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REFERENCES


HYPERSENSITIVITY TO ATROPINE

Sir,—With reference to the letter of Dundee and Mirakhur (1978), it is worth pointing out that glycopyrronium is not yet available in Greece.

Some months ago, a 4-yr-old baby boy of 18 kg body weight had to undergo an operation for congenital cataract. Allergy to atropine—mentioned by his parents—was proved by testing with atropine 1 : 1 000 000. The anaesthetic management comprised premedication with droperidol.
1.6 mg and fentanyl 0.03 mg 45 min before surgery, and anaesthesia was induced with ketamine 50 mg. Endotracheal intubation was facilitated with suxamethonium 20 mg i.v. Anaesthesia was maintained with nitrous oxide in oxygen 2 : 1 and ventilation was controlled with pancuronium 1.5 mg. The neuromuscular blockade was antagonized by hyoscine 0.2 mg and neostigmine 1 mg i.v.

Arterial pressure was maintained at 110/80 mm Hg and the heart rate at 120 beat min⁻¹ until the injection of hyoscine which increased the rate to 160 beat min⁻¹. This effect lasted for about 5 min until the neostigmine produced a gradual reduction of heart beat to 68 beat min⁻¹, and 30 min later the heart rate remained stable at 100 beat min⁻¹.

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REFERENCE

ASPIRATION SYNDROMES IN PREGNANCY
Sir,—In the past decade it has become accepted practice to give oral alkali to patients in labour as prophylaxis against the acid aspiration syndrome.

We report two patients who received appropriate alkali therapy, in which the aspiration of alkaline stomach contents into the lungs during anaesthesia for Caesarean section resulted in differing clinical courses.

The first patient, a 25-year-old primigravida, underwent an emergency Caesarean section under general anaesthesia after prolonged labour. Routine precautions at induction did not prevent the regurgitation and inhalation of stomach content, which was seen as a clear yellow fluid. Tracheal intubation was performed rapidly, and a suction catheter was inserted via the tube. A healthy child was delivered by routine lower segment Caesarean section. After antagonism of residual neuromuscular blockade and removal of the tracheal tube, there was ventilatory inadequacy and peripheral circulatory collapse. The patient was transferred to the intensive therapy unit, for treatment of the acid aspiration syndrome. Investigations included chest radiography (fig. 1) and analysis of the gastric aspirate. The patient died 8 h after the induction of anaesthesia.

The second patient, a 32-year-old primigravida, underwent an emergency Caesarean section under general anaesthesia because of obstructed labour. Before induction a nasogastric tube was passed and the stomach aspirated. Following induction, tracheal intubation was carried out without incident. During the operation, the cuff of the tracheal tube burst, laryngoscopy was performed immediately, and the tracheal tube changed without incident. Ten minutes later the cuff of the second tube burst, and at laryngoscopy a pool of whitish, minty-smelling fluid was observed in the pharynx; this was removed by suction, and a third tracheal tube inserted. The operation was completed uneventfully, and the patient recovered fully from anaesthesia, but 30 min later she developed tachypnoea, cyanosis and tachycardia. Chest radiography (fig. 2) was performed, and the patient was transferred to the intensive therapy unit for treatment of the acid aspiration syndrome. The patient recovered completely.