An infusion of polygeline was commenced. Five minutes later, the arterial pressure decreased to 40 mm Hg systolic and erythema appeared over the body. Despite treatment with antazoline, calcium chloride, phenylephrine and an infusion of plasma protein, the arterial pressure remained at a low value for 30 min and the operation was cancelled. The patient made an uneventful recovery and 7 days later the same solution of polygeline gave no reaction in one of us.)

The second patient was a 53-year-old man known to be allergic to phenytoin, carbamazepine and some tar oils. Bronchomediastinoscopy was performed for the investigation of a suspected tumour of the left lung. The arterial pressure before surgery was 150/100 mm Hg. After induction of anaesthesia with enibomal, suxamethonium was given i.v. to facilitate endotracheal intubation. Anaesthesia with tetracaine, halothane, and nitrous oxide in oxygen, comprised: enibomal, suxamethonium, halothane, and nitrous oxide in oxygen. There was a gradual decrease in arterial pressure to 90/70 mm Hg and a polygeline infusion was started. Within 10 min, the arterial pressure increased to 120/100 mm Hg, but a sudden decrease to 40 mm Hg systolic occurred thereafter. Simultaneously severe bronchospasm developed with the appearance of widespread urticaria. The operation was cancelled. Following oxygenation and the administration of aminophylline, phenylephrine and calcium chloride, the patient recovered completely within half-an-hour. Five days later the patient had an uneventful bronchoscopy performed under a anaesthetic technique which included enibomal, suxamethonium, halothane, and nitrous oxide in oxygen. The third patient was a 23-year-old man (without a known allergy) scheduled for a gastrectomy. The arterial pressure before surgery was 120/60 mm Hg. The drugs administered during the anaesthetic comprised: enibomal, suxamethonium, halothane, nitrous oxide in oxygen and gallamine. After the patient had been placed in the lateral position, the arterial pressure decreased to 80/60 mm Hg and an infusion of polygeline was started. Five minutes later the arterial pressure decreased to 50 mm Hg systolic and a generalized urticaria appeared. Despite treatment with phenylephrine, antazoline, calcium chloride, adrenaline and albumin, hypotension persisted for 25 min and the operation was cancelled. There was an uneventful recovery and 7 days later the patient had an uncomplicated anaesthetic with enibomal, suxamethonium, cyclopropane and gallamine. In five patients, who received enibomal, suxamethonium, halothane and nitrous oxide in oxygen during anaesthesia, polygeline was administered for minor degrees of hypotension. An urticarial rash developing 15–20 min after the start of the infusion was observed in all five patients. Arterial pressure did not decrease significantly and there were no other allergic manifestations. No urticarial eruptions occurred.

All eight patients developed skin reactions of varying degrees. Four of the patients developed marked hypotension and one had a severe attack of bronchospasm. In five out of six an intradermal skin test with polygeline diluted 1:10 showed a positive reaction. (An intradermal test with the same solution of polygeline gave no reaction in one of us.) None of the patients received a blood transfusion.

Following the infusion of polygeline in man, there have been other reports of urticaria (Bark, 1964; Eberlein and Dobberstein, 1962) and one instance of anaphylactic shock with urticaria, bronchospasm and hypotension (Bortoluzzi et al., 1967). In dogs acute hypotension and the liberation of histamine have been demonstrated (Messmer et al., 1969).

Our observations provide very strong circumstantial evidence that polygeline may induce a hypersensitivity reaction in patients.

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REFERENCES


PREOPERATIVE ASSESSMENT FOR ANAESTHESIA

Sir,—We would like to congratulate you on your leading article on “Preoperative assessment for anaesthesia” (Editorial, 1974), which advocates, among other things, the provision of a “pre-admission unit” in the outpatient departments of our hospitals. We would, however, like to remind your readers that at the Southend Hospital (Lee, 1949) and, indeed, at other hospitals mentioned in the same number of your journal (Kyei-Mensah and Thornton, 1974; Green and Howat, 1952; Loder and Richardson, 1955; Burn, 1972; de Baas, 1972), such departments have been actively occupied in making patients safer for surgery, and for the past 25 yr in our own hospital continue to be kept busy.

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REFERENCES


THE USE OF ALTHEIN FOR SEDATION

Sir,—In this hospital, Althesin has been used for sedation during surgery performed under spinal or extradural analgesia. Sedation was commenced with Althesin 0.5–1.0 ml and maintained with 10–15 ml/hr of a solution of Althesin 10 ml, diluted to 100 ml with isotonic saline. The patient was kept in a state of light sleep and maintained his own airway. If more sedation was required the rate of infusion was increased, and if the patient’s airway became obstructed a Guedel airway was inserted and was tolerated well.

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This procedure has been used for 25 patients who were premedicated with pethidine 50 mg, phenergan 25 mg, and atropine 0.6 mg all given i.m. and who were undergoing surgery lasting 1–1.5 hr. In the anaesthetic room, an i.v. infusion of Ringer’s lactate was commenced and a spinal or extradural block was performed. This produced a reduction in arterial mean pressure of 5–30 mm Hg (mean 15 mm Hg) and an increase in heart rate of 5–15 b.p.m. (mean 10/min). Sedation with Althesin was then started and further changes in arterial pressure occurred in the range +5 to —10 mm Hg (mean —5 mm Hg). There was no significant change in heart rate. The respiratory rate decreased by an average of 2 b.p.m. (range +3 to —5). In two patients undergoing total cystectomy the central venous pressure was measured, and no significant changes were observed during the infusion. At the end of surgery, the Althesin infusion was stopped and recovery, as judged by an awake and clear-headed patient, occurred within 6–12 min.

In the past, we have used diazepam for sedation during local anaesthesia but this drug has an unpredictable and often prolonged effect (Brown and Dundee, 1968), there is restlessness, especially in the older patient (Du Cailar and Gestin, 1966), occasional profound respiratory depression may occur (Buskop, Price and Molnar, 1967) and there may be thrombophlebitis at the site of injection.

Althesin is a short acting drug with little tendency to produce cumulative effects or tolerance (Ramsey et al., 1974). A very light level of sedation can be maintained with stability of the cardiovascular and respiratory systems, and recovery from anaesthesia occurs quickly (Ramsey et al., 1974). We suggest that this method of sedation is both safe and easy to administer and has advantages over other drugs (Du Cailar, 1972).

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REFERENCES


A MODIFICATION TO THE SERVOMEX OA 272 OXYGEN ANALYSER

Sir,—We have modified the Servomex OA 272 oxygen analyser to provide a 50% oxygen range on the instrument. The modification is particularly useful for the measurement of the oxygen concentration in anaesthetic mixtures.

The 333-ohm resistor R32 in the potentiometer chain, shown in figure 1, is replaced by three, 111-ohm resistors connected in series and a tap is taken off the chain, at the point shown in figure 1, to provide a 50% range. A single pole toggle switch, mounted on the front panel of the instrument, allows selection of either the 50% or 100% range when the range selection switch is in the 100% position. This obviates the need to alter the wiring of the range selection switch.

Fig. 1. Electrical circuit diagram of analyser before and after modification.

The potentiometer chain resistors are located on a small panel mounted on the meter terminals and the added resistors can be accommodated on this panel. It is essential that precision resistors (tolerance ±0.1%) are used in order that the relationship between ranges is correct. The electrical output of an instrument modified as described above was measured with a digital voltmeter, using room air in the analyser. The output was 8.423 mV on the 50% range and 4.211 mV on the 50% range, showing good agreement between the ranges. The precision resistors required for the modification were kindly supplied by Taylor Servomex Limited, Crowborough, Sussex.

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OCCLUSION OF THE INFERIOR VENA CAVA

Sir,—We wish to report a problem, similar to that described by Malatisinsky and Kadlic (1974), of inferior vena cava occlusion causing severe arterial hypotension.

A 54-year-old healthy male suddenly developed severe arterial hypotension (arterial systolic pressure 40 mm Hg) during the operation of cholecystectomy for cholelithiasis. At this stage the surgeon was attempting to delineate the junction of the cystic duct with the hepatic duct. After excluding anaesthetic causes of hypotension, the surgeon was asked to remove the pack from Morrison’s pouch, and also the retractors which were applied to the stomach. The arterial pressure then increased to 100 mm Hg systolic. It seemed that the packs and retractor were compressing both