


Sir,—Thank you for giving us an opportunity to comment on the letter received from Drs Vesey and Cole.

We were delighted to see that their work explains the slow and progressive development of the toxic signs of nitroprusside overdosage. Of course we agree that it is the rate of infusion and not the total dose which is important, and indeed quoted our results in terms of dose per hour. We seem to be in agreement also that hydroxocobalamin might act clinically as an effective cyanide antagonist.

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ON THE CIRCULATORY AND TOXIC EFFECTS OF SODIUM NITROPRUSSIDE

Sir,—Apparently, some confusion surrounds the relative importance of arterial pressure and blood flow in the successful use of deliberate hypotensive techniques. Induced hypotension is utilized for one of two aims: (1) facilitation of vessel surgery (such as intracranial aneurysms and coarctation of the aorta), and (2) reduction of blood loss (such as radical neck operations). Hypotension may facilitate vessel surgery without a reduction in cardiac output being necessary. Under these circumstances it is the decrease in vessel tension and not the decrease in blood flow that is desirable. Hypotensive drugs which provide minute-to-minute control of arterial pressure, such as nitroprusside or trimetaphan, administered in an intravenous infusion, would then be most useful. However, the success of induced hypotension in producing a relatively bloodless operative field depends on reduction of the cardiac output (or the blood flow at the operative site). This has been shown clearly by Dieder, Clagett and Theye (1965).

It has been demonstrated that the increase in cardiac output with nitroprusside is related to the increase in heart rate (Wildsmith et al., 1973; Styles, Coleman and Leary, 1973). One may seriously question the success and safety of such a technique in reducing the amount of bleeding. Tachycardia during induced hypotension may result in increased oozing as a result of repetitive spike filling of the vessels (Larson, 1964), and may ultimately lead to failure of the technique (Larson, 1964; Hellewell and Potts, 1966; Salem and Ivankovic, 1970). If sodium nitroprusside is used, this may result in the administration of a large and toxic dose of the drug. This is likely to occur in children who are known to be resistant to the induction of hypotension (Anderson, 1955; Salem et al., 1974). Increasing the rate of infusion is not the answer to the problem, but with beta-adrenergic blockade the total dose of nitroprusside may be kept to a minimum, thus avoiding catastrophes similar to the case reported by Merrifield and Blundell (1974).

It must be emphasized that acidosis is not a feature of induced hypotension when other drugs are used, provided that adequate oxygenation is maintained and the value of arterial FCO₂ is within normal limits. On the other hand, the acidosis seen with large doses of nitroprusside results from depressed oxygen uptake as a result of its conversion to thiocyanate and subsequent oxidation to cyanide (McDowall et al., 1974). This was observed by Dr Paul Boyan, 20 years ago, but has now been published.

For these reasons, nitroprusside seems to be a poor choice in long operations in which reduction of bleeding is desirable. In contrast, pentolinium appears to offer advantages. The use of nitroprusside should be restricted to procedures where minute-to-minute control of pressure is desirable for a short period.

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REFERENCES


ANAPHYLACTIC REACTIONS INDUCED BY INFUSION OF POLYGELINE (HAEMACCEL)

Sir,—We read with interest the report of Lund (1973) concerning a case of anaphylactic reaction to polygeline (Haemaccel). It was our practice to infuse polygeline if the arterial pressure decreased during and after induction of anaesthesia, but following anaphylactic reactions to polygeline in eight patients, this practice has stopped.

The first patient was a 40-year-old healthy man who was scheduled for lumbar laminectomy. There was no history of allergy. The arterial pressure was 120/70 mm Hg before operation. Following induction of anaesthesia by the i.v. injection of a barbiturate (enflurane—a short acting barbiturate differing from thiopentone only by substitution of N for the S atom), tracheal intubation was performed with the aid of suxamethonium i.v. and anaesthesia was maintained with nitrous oxide in oxygen, halothane and gallamine. The patient was turned to the prone position. Shortly afterwards the arterial pressure decreased to 100/80 mm Hg.
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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 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 was maintained with nitrous oxide in oxygen and methoxyflurane. 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 an 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 urtiartrial 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 ALTHESIN 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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