ADVERSE REACTIONS TO INTRAVENOUS ANAESTHETIC AGENTS

Sir,—Recent investigations have suggested that certain transient changes may occur in the blood of patients who suffer an adverse reaction following the administration of an intravenous anaesthetic agent. Alterations in the concentrations of circulating components of the complement system and white cell numbers have been apparent from serial sampling of blood from patients with an anaphylactic type of response following administration of thiopentone, methohexitone or Althesin. Insufficient data have been collected to allow a full understanding of the significance of these findings, but we believe that they will assist towards an understanding of the mechanism underlying the reactions.

We invite all anaesthetists who see an anaphylactic type of response in a patient following shortly after the induction of anaesthesia to take blood samples, adhering as closely as possible to the protocol described below, and to contact Dr J. Watkins, Protein Reference Unit, Department of Immunology, Hallamshire Hospital Medical School, Sheffield, S10 2RX (0742-26484 ext. 232 or 229). We advise that skin testing of the patient should be delayed until examination of the blood samples has been completed.

Five millilitre of venous blood should be collected into a heparinized tube as soon as possible after the reaction begins. In view of the rapid deterioration of blood samples, it is suggested that the plasma is separated and stored at −25 °C if difficulty is experienced in contacting Dr Watkins. It would also help the investigation if a sample of blood (taken in an EDTA tube) immediately at the time of reaction, and 10 min later, is sent to the local Haematology Laboratory for total and differential white cell count. Further samples of 5 ml heparinized blood should be taken at 3 hr, 6 hr and 24 hr after the reaction. If possible, another 5 ml should be taken not sooner than 5 days after the event.

Full records of the incident, including batch numbers of the drugs given at induction, should be kept and the first series of samples dispatched as soon as possible. Receipt of the blood samples will be acknowledged and accompanied by a standard form for collecting full details associated with the reaction. Results of the blood assays will be communicated as soon as available when advice will be offered on the best means of investigating the patient further (for example, skin tests) to identify or confirm the agent responsible for the reaction.

J. WATKINS
J. A. THORNTON
Sheffield
R. S. J. CLARKE
Belfast

ENTONOX AND HALOTHANE IN DENTAL SURGERY

Sir,—I have read with interest the article on the “Use of Entonox plus carbon dioxide in dental surgery” by Davies, Burns and Bracken (1975), but I was surprised that they chose to use the Goldman Halothane Vaporizer. I used this vaporizer with a premixed gas unit, specially made for me by the British Oxygen Company Limited in 1966 (Rollason, 1967). I found the concentration of halothane from this vaporizer as monitored by a Hook and Tucker meter, during outpatient dental anaesthesia, decreased rapidly from an initial 2% to 0.5% although the vaporizer remained fully “on”. Accordingly I replaced it by a Cyprane A.E. temperature and flow-compensated vaporizer and I found this much more satisfactory (Rollason and Dundas, 1969).

NORMAN ROLLASON
Aberdeen

REFERENCES


Sir,—Dr Rollason has commented on the performance of his Hook and Tucker meter, rather than on the behaviour of his patients. We found that with 7% carbon dioxide added to Entonox, the Goldman Vaporizer delivered more than sufficient halothane to keep our patients adequately anaesthetized. The vaporizer was seldom “on” for more than 20 sec. A safe vaporizer is one which is small in volume and inefficient. Efficient vaporizers can harbour a concentration of 28% halothane and can fail, thus increasing considerably the risk to the patient.

T. H. S. BURNS
London

ENTONOX IN DENTAL SURGERY

Sir,—In the article by Dr Davies and his colleagues (1975) on the use of Entonox in dental surgery, two statements are made which deserve comment. These are: “Intravenous drug administration requires that one person injects the drug while another supervises the patient’s airway”, and “Advantages of inhalation anaesthesia for outpatients . . . the anaesthetist can control the airway as he gives the anaesthetic”.

May I refer the authors and any interested readers to two publications which demonstrate the use of an indwelling intravenous needle and extension tubing which brings the anaesthetic syringe to the anaesthetist’s hand whilst he sits in the traditional position for maintaining a patent airway and controlling any supplementary inhalation, at the patient’s side. This position may be used whether the patient is dangerously upright in the old-fashioned “barber’s chair”, or supine on a modern adjustable couch (Green and Coplans, 1973). The second publication by the Society for the Advancement of Dental Anaesthesia (1973) demonstrates a position by the patient’s side whereby the
anaesthetist can monitor the pulse, observe respirations and support or elevate the mandible by reaching across the patient's chest from the front when access to the head is blocked by the seated dentist and his assistant.

DONALD BLATCHLEY
London

REFERENCES


ACID PULMONARY ASPIRATION
Sir,—Dr Gordon Taylor's report (1975) on “Acid pulmonary aspiration” occurring following the aspiration of gastric contents with a pH of 3.5 is extremely disquieting to those of us who use prophylactic antacids and have, hitherto, considered a pH of 2.5 as critical. Careful scrutiny of the time sequence, however, casts doubt on whether or not aspiration really occurred at the stated pH. The patient’s first dose of antacid was at 2 a.m. and the second dose, 5.5 hr later at 7.30 a.m. Our work shows that an interval of more than 4 hr after ingestion of oral antacid may be followed by acid rebound (Roberts and Shirley, 1974). Hence it is possible that at 7.30 a.m., the pH was lower than it would have been had the first dose been omitted, particularly in view of the large volume involved. Aspiration occurred at 7.50 a.m., 20 min after the second oral dose of antacid. At 8.00 a.m., more senior anaesthetic assistance arrived and the gastric sample was stated to be taken at the time of endotracheal intubation. We must assume that some time passed during which the senior anaesthetist performed endotracheal intubation and brought the situation under control. As such, 8.05 a.m. is probably the earliest time at which the sample could have been taken. Our own work shows that up to 30 min may be required for adequate mixing and buffering of gastric contents by antacids, and if the pH was 3.5 at 35 min after ingestion of antacid, it was not necessarily 3.5 at 20 min after ingestion.

The use of a mixture of aluminium and magnesium hydroxide in 200 cases resulted in the increase of the pH to more than 5 in 67% of elective Caesarean sections and 64% of vaginal deliveries. However, if the antacid was given more than 4 hr before delivery, only 27% and 30% of patients in the latter two groups respectively had a pH higher than 5, while 64% and 44% of patients in the same two groups had a pH of below 2.5.

I believe Dr Taylor's present contribution in no way diminishes the value of his classical work in introducing the use of prophylactic antacids (Taylor and Pryse-Davies, 1966), but points out again that prophylactic antacids are only one of a number of preventive measures which must be adhered to rigorously in order to prevent the occurrence of the acid pulmonary aspiration syndrome.

R. BRYAN ROBERTS
New York