FOETAL ACID-BASE BALANCE AT CAESAREAN SECTION

Sir.—In the Correspondence section of the Journal (1972, 44, 233) Dr Marx mentioned that foetal acid-base values obtained from umbilical artery blood at Caesarean sections did not correlate with the condition of the newborn infant at birth. I should like to provide evidence that acid-base values of umbilical artery blood are not reliable at Caesarean sections under general anaesthesia.

We measured foetal pH, base excess (BE) and Pco₂, from serial scalp capillary blood samples during 26 Caesarean sections under thiopentone/nitrous oxide/oxygen anaesthesia. The first scalp sample was obtained in all cases immediately before the induction with thiopentone. The last sample was obtained during the last 2 minutes before the delivery of the foetus in 24 cases. The umbilical cord was double clamped as soon as possible in all 26 cases. Induction-delivery (ID-) time ranged from 4½ to 14 minutes (mean 9 min 7 sec). The means (±SD) of foetal scalp capillary blood immediately before the induction of anaesthesia were for pH 7.318 (±0.033), for BE -6.70 (±2.41) m.equiv/l. and for Pco₂ 37.17 (±6.13) mm Hg. Thirteen of the 26 newborn infants were given an Apgar score of 9 or 10 at the age of one minute, 6 a score of 7 or 8, and 7 a score of 6 or less. The means of the foetal acid-base values immediately before the induction of the anaesthesia did not differ significantly between the three groups of newborn infants. A more detailed description of methodology and the patient material has been published previously (Teramo 1968, Teramo et al., 1971).

There was no correlation between the pH of the umbilical artery blood and the condition of the newborn infant or the ID-time (fig. 1). However, there was a good correlation between the pH change of foetal scalp capillary blood during Caesarean section and the condition of the newborn infant (fig. 2). The pH difference between the scalp capillary blood obtained immediately before the induction and umbilical artery blood did not correlate with the condition of the newborn infant (fig. 3).

![Fig. 1. pH values of umbilical artery blood and induction-delivery time in 26 Caesarean section cases under thiopentone/nitrous oxide/oxygen anaesthesia and Apgar scores of infants at the age of one minute. Foetal pH was measured from capillary blood samples immediately before the induction of anaesthesia and within a 2-minute interval before the delivery of the foetus. The mean pH change in the asphyxiated group differed highly significantly from the corresponding mean change of the group with Apgar score 9 or 10 (t=4.85, P<0.001).](https://academic.oup.com/bja/article-abstract/44/9/988/242054)

![Fig. 2. Change in foetal pH during Caesarean section under thiopentone/nitrous oxide/oxygen anaesthesia and Apgar scores of infants at the age of one minute. Foetal pH was measured from capillary blood samples immediately before the induction of anaesthesia and within a 2-minute interval before the delivery of the foetus. The mean pH change in the asphyxiated group differed highly significantly from the corresponding mean change of the group with Apgar score 9 or 10.](https://academic.oup.com/bja/article-abstract/44/9/988/242054)

![Fig. 3. Change in foetal pH during Caesarean section under thiopentone/nitrous oxide/oxygen anaesthesia and Apgar scores of infants at the age of one minute. Foetal pH was measured from capillary blood samples immediately before the induction of anaesthesia and from umbilical artery blood at birth. The mean pH change in the asphyxiated group did not differ significantly from the group with Apgar scores 9 or 10.](https://academic.oup.com/bja/article-abstract/44/9/988/242054)
 Obviously, scalp capillary blood gives a better assessment of the condition of the foetus and the newborn infant than umbilical artery blood at Caesarean sections with this type of anaesthesia.

The aetiology of the foetal pH decrease and the asphyxia of the newborn infants in the present series may well have been the supine position of the mothers during the operation as Dr Marx pointed out. However, other causes, e.g., thiopentone, nitrous oxide and maternal hypovolaemia cannot be excluded.

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REFERENCES


CORRESPONDENCE

Sir,—There have been several reports recently of anaphylactic and hypotensive reactions to propanidid (Thornton, 1971; Turner, Keep and Bartholomaeus, 1972; Lorenz et al., 1972). I would like to describe another case of apparent anaphylaxis.

The patient was an 8-year-old boy who had sustained a severe Colles fracture, which was initially reduced using methohexitone, nitrous oxide-oxygen-halothane anaesthesia. The anaesthetic was uneventful. This reduction was unsatisfactory and the fracture was remanipulated the following day using propanidid 250 mg, nitrous oxide-oxygen-halothane, this too was an uneventful anaesthetic. Following day using propanidid 250 mg, nitrous oxide-oxygen-halothane, this too was an uneventful anaesthetic.

Fourteen days later remanipulation was again necessary and anaesthesia was induced with propanidid with the following result:

1405 Propanidid 250 mg intravenously over 45 seconds into a vein in the left antecubital fossa. Shortly after the period of hyperventilation, during which time the patient was breathing nitrous oxide-oxygen and 0.5% halothane, the patient became very agitated and started to thrash his limbs around and to cough, dislodging the intravenous needle.

1407 A generalised reddish flush appeared over the upper part of the body, maximal on the upper chest. Coughing continued and there was copious production of thin saliva. The pulse had fallen to 30 beats/min and was regular but poor in volume.

1408 100% oxygen was administered and the patient tipped head down. The patient maintained his own airway adequately and the nasopharynx was repeatedly aspirated. The erythema decreased but a petechial rash appeared on the upper chest. There was now some oedema and erythema at the injection site.

1409 Gross pallor of the face developed. However, there was sparing of the lips and ears which remained pink and appeared well perfused.

1410 There was considerable sweating, mainly confined to the face. The pulse was barely palpable. However, there was evidence of good peripheral perfusion. Piriton 10 mg and Hydrocortisone 200 mg was given intramuscularly, the patient being so collapsed that a vein could not be found. The patient continued to breathe adequately and there was no facial or laryngeal oedema and no bronchospasm.

1411 The pulse rate rose to 100 beats/min regular, with a fair volume, and the facial pallor disappeared. Coughing continued but the volume of saliva was much reduced.

1412 The patient regained consciousness but was very drowsy, responding only to simple commands.

1415 The petechial rash remained and the boy was much more alert. He did not complain of any abdominal pain but had been doubly incontinent.

The boy remained unwell for a further two days with violent diarrhoea. However, he was sufficiently recovered after four days for a further anaesthetic to be administered. This time induction was with nitrous oxide, oxygen and halothane and there were no untoward effects.

On further questioning the mother of this boy, it became clear that there was a family history of allergy; the mother was allergic to elastoplast, and a brother aged 10 had a history of eczema. The patient was said to have developed an allergic type rash while on holiday in France at the age of 4. This was on exposed areas of the body and was said at the time to be due to local dust. The boy had had no contact with oil of cloves at any time.

This case would appear to have followed the general pattern of some of those reactions already reported in that hypersensitivity developed a fortnight after an initial uneventful exposure to propanidid. It would have been of interest to have re-anaesthetized this child with propanidid, after antihistamine cover, after the manner described by Lorenz et al. (1972) on a volunteer anaesthetist patient. However, in the circumstances, this was considered unethical.

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REFERENCES


A CIRCUIT TO REDUCE THE INHALATION OF GASES BY ANAESTHETISTS

Sir,—I read with interest the letter from Dr C. D. G. Evans-Prosser in the April issue (Brit. J. Anaesth., 44, 412) as it details apparatus similar to that which I have evaluated in a recent article.

It will be of interest to those concerned with the removal of exhaust gases to note that such apparatus is now available commercially from Monitor Medical Products Ltd., Robin Hill, Castle Hill, Prestbury, Cheshire SK10 4AS.

The components consist of a curved manifold corrugated tubing and terminal safety T-piece and will fit the standard BOC expiratory valve or the M.I.E. expiratory valve with an available modification, the whole apparatus weighing less than 5 ounces.

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REFERENCE