METOCLOPRAMIDE AND GASTRIC EMPTYING

Sirs,—In view of increasing interest in the effect of metoclopramide on the gastric emptying rate, may I briefly describe the results of a short trial carried out in the clinical situation as an addition to the information so far available on a drug action which could be of importance to anaesthesia.

Under double-blind conditions, 39 patients appearing consecutively for emergency surgery were given intravenously, 1-2 hours before operation, the contents of randomly numbered ampoules containing normal saline or metoclopramide 5 mg/ml. The dosage of metoclopramide given was 1.5 mg/10 kg. No other medication was given and any patients with evidence of intestinal obstruction were excluded from the trial. Following the induction of anaesthesia, a wide-bore stomach tube was passed and the gastric contents aspirated as thoroughly as possible. The quantities were measured. It was felt that the inaccuracies occurring at this stage would affect both groups equally.

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<th>TABLE I</th>
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<td>ml</td>
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<td>0–10</td>
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<td>11–20</td>
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At the completion of the trial, the key to the numbered ampoules showed that 20 patients had received metoclopramide and 19 normal saline. The data distribution is shown in table I. The data demonstrated a wide variation in control levels, reflecting a wide variation in initial stomach contents. There was no statistically significant drug effect at this dosage level, although there was a suggestion of a drug effect at large initial volumes.

I thank Prof. W. Hugin of the Anaesthetic Department of Basel City Hospital for his permission to undertake this study.

RODNEY F. ARMSTRONG
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ANAESTHESIA AND SICKLE-CELL HAEMOGLOBIN

Sirs,—Whilst I write to congratulate Dr T. Hilary Howells and his colleagues on their valuable contribution to this subject (Brit. J. Anaesth., 1972, 44, 975), they should permit me to make comments on their paper, especially on their recommendation for exchange transfusion for sickle-cell traits undergoing major thoracic surgery.

In Korle Bu Teaching Hospital, Accra, Ghana, hundreds of patients with sickle-cell trait have been successfully anaesthetized for all forms of major surgery (excluding open heart surgery using the bypass). Exchange transfusion or any other existing drug prophylaxis has not been employed prior to anaesthesia. However, apart from other measures, all patients, whether known sicklers or not, receive induction of 100% oxygen before induction. This was omitted in the patient of Dr Hilary Howells and his colleagues although they recommended this practice later in the paper.

Another precipitating factor to be considered in this particular patient is cardiovascular collapse from propanidid. There have been reports of such cardiovascular reaction (sudden severe hypotension, and red blotchy skin) from propanidid (Evans, 1971; Spreadbury and Marrett, 1971). In this consideration the complication might have occurred whether the patient was a sickler or not.

In Accra, many patients with HbSS, HbSC and HbS Thalassaemia and HbCC have had uneventful general anaesthesia and surgery without exchange blood transfusion. Among these was a 10-year-old HbSS anaemic and toxic girl who at the time of anaesthesia was in crisis. Her general condition was so grave that apart from sickling crisis due to splenic abscess, typhoid fever was also suspected as a probable diagnosis. Her haemoglobin was 4.7 g/100 ml and her temperature was swinging. She recovered after a successful splenectomy. The spleen had multiple infarcts and abscesses. Our anaesthetic management of sicklers has been discussed elsewhere (Searle and Oduro, 1972).

Dr Hilary Howells and his colleagues did realize that “hypoxia and a transient polycythaemia presumably caused by dehydration” might have precipitated the infarctive crisis. I am happy they did not give urea intravenously. Blood viscosity would have been increased considerably to add its own dangers.

I believe that fatalities in patients with sickle-cell trait tend to occur in short and minor surgery where the usual “quick whiff” is given or anaesthesia is by an intravenous induction agent alone.

Until an ideal drug prophylaxis prior to anaesthesia is introduced, anaesthesia for these patients must be simple and cautious.

K. A. ODURO,
Saskatoon

REFERENCES

Sirs,—We were delighted to receive the comments of Dr Oduro, who, like Dr Searle, is expert in the anaesthetic management of patients with sickle-cell syndromes. The subject in our case report did not receive preoxygenation and falls precisely into that category of patient described by Dr Oduro as at risk.

Our case report was a cautionary tale and not an example of good management. While it is reasonable to consider that a propanidid sensitivity reaction might have brought about our patient's collapse, the absence of urticaria and the localization of splenic pain which preceded the incident suggests that a splenic infarct was the most likely diagnosis.

T. HILARY HOWELLS
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CIRCULATORY EFFECTS OF CONTROLLED ARTERIAL HYPOTENSION WITH TRIMETAPHAN DURING NITROUS OXIDE/HALOTHANE ANAESTHESIA

Sirs,—Dr Scott and his colleagues (Brit. J. Anaesth., 1972, 44, 523) may anticipate subsequent normal essential organ function in their 10 patients aged 26 to 67 years and hypotended to a systolic pressure of 60 mm Hg (5 cases showing a fall in cardiac output, the maximum being 27%), but without suitable function studies their statement “without risk” is still supported merely by presumption and the same applies to their unpublished cases of “25 years of experience”, if inadequately investigated.