the face and body. This was accompanied by deep cyanosis of the lips and nail beds. Pulse and blood pressure were unrecordable, although spontaneous ventilation appeared adequate. The cyanosis did not improve with 100 per cent oxygen and the pupils dilated. Since ventilation remained satisfactory, the patient was not intubated. An intravenous infusion with Ringer-lactate solution was commenced through the needle which had been used for the propanidid injection. Intravenous hydrocortisone hemisuccinate 100 mg produced no improvement. An electrocardiograph showed a supraventricular tachycardia. The heart rate was 180 beats/min. Unconsciousness and profound hypotension continued for about 30 minutes. Recovery of consciousness was associated with marked restlessness. At 30 minutes, systolic blood pressure was 70 mm Hg and heart rate 160 beats/min. During the next 2 hours, blood pressure rose to 110/70 mm Hg and heart rate fell to 80 beats/min. Over the total period of resuscitation (i.e. 24 hours), the patient received 540 ml of each of the following: Ringer-lactate solution, Rheomacrodex 10 per cent and plasma. Subsequent recovery was uneventful and the patient was discharged 2 days later, after the operation had been performed with local anaesthesia.

Later investigations, including chest X-ray, serum electrolytes, haemoglobin and p.c.v., skin sensitivity by intradermal injection of propanidid, ASO titre and virus studies, have all yielded normal results.

This patient does not appear to show a true drug idiosyncrasy since a previous administration was uneventful. It is possible that this was a hypersensitivity reaction causing massive histamine release, although there was no clinical evidence of bronchospasm, no urticaria or angio-oedema, also skin testing was negative. Finally, we cannot rule out direct cardiovascular depression causing severe hypotension with compensatory tachycardia.

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MEASUREMENT OF DEPTH OF ANAESTHESIA
Sir,—Prof. Robson’s article (Brit. J. Anaesth. (1969), 41, 785) is very important and timely. It tends to stimulate interest in the recent developments relating to this fascinating subject. The article was mainly concerned with depth in the range required by modern anaesthesia, that is, the lightest anaesthesia and the means of measuring it in clinical practice. I presume, therefore, that for this reason two objective methods to investigate anaesthetic depth were not mentioned in Prof. Robson’s article. I refer to (a) the minimum alveolar concentration (m.a.c.) measurements recently described by Prof. Robson to prevent gross movement in response to a painful stimulation and (b), the recording of Hoffman (H) reflex, a spinal monosynaptic reflex, from the calf muscles.

The ratio of alveolar concentration of an anaesthetic to m.a.c. reflects anaesthetic depth. Anaesthetic depth in regard to multiples of m.a.c. has been described for various anaesthetic agents: halothane, nitrous oxide, cyclopropane, diethyl ether and methoxyflurane (Merkel and Eger, 1963; Eger, Saidman and Brandstater, 1965).

De Jong, Hershey and Wagman (1967) found a strong association between progressive depression of the H reflex amplitude and four clinical levels of muscular relaxation. The decrease in the amplitude of the H-reflex was then compared with inspired anaesthetic concentration and with m.a.c. units (De Jong, Hershey and Wagman, 1967; De Jong et al., 1968). Thus the measurement of both m.a.c. and the amplitude of the H-reflex provide a quantitative index for depth of anaesthesia. At present small significant changes in the depth of anaesthesia, as pointed out by Prof. Robson, are difficult to detect. Future research may make possible the demonstration of those changes by the use of either one or a combination of these new methods. A comparative m.a.c. value for different anaesthetic agents could be determined for every clinical plane of anaesthesia, even the one associated with amnesia, the individual’s appreciation of the passage of time (Robson et al., 1960) or with cortical evoked potentials (Robson, 1967).

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REFERENCES


USE OF FARMAN ENTRAINER AND EMO VAPORIZER WITH IPPV
Sir,—It may be that some anaesthetists are using the Farmen Entrainer (Farmen, 1965) together with the EMO vaporizer for paediatric anaesthesia in a way not originally described by Dr Farmen.

He advocates and gives valid reasons for a non-return valve being placed between the vaporizer and the Ayre T-piece with Jackson Rees attachment, when the apparatus is being used for IPPV.

Briefly, this is to prevent reverse flow through the vaporizer which would have the dual effect of decreasing the fresh gas flow and allowing the gases more than one passage through the vaporizing chamber, when the gas pressure in the T-piece as the result of squeezing the bag exceeds that which is delivered by the entrainer (10–15 cm H2O maximal).

The Oxford inflating bellows can be conveniently used for this purpose. The manufacturers’ usage