At present, we assume that the elevated activity and biochemical abnormalities of muscle creatine-phosphokinase we found in the family of a patient who recovered from and lost two children from malignant hyperpyrexia, may be some of the aetiologic factors. Therefore we again recommend the routine use of CPK-test prior to inhalational anaesthesia.

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REFERENCES


SIR,—May I offer some comments on the article by Drury and Gilbertson? (Brit. J. Anaesth. (1970), 42, 1021).

(1) A likely cause of the hyperpyrexia is the combination of pethidine and atropine in conjunction with anaesthesia. Pethidine potentiates atropine and probably atropine potentiates pethidine. Pethidine is not needed for premedication, and probably can be dispensed with safely.

(2) It is difficult to see why a patient's temperature should be allowed to reach 109°F before someone in the operating room notices that something is wrong. Temperature measurement is one of the simplest techniques in medicine. One can use an ordinary thermometer, or a thermistor. If the anaesthetist were to measure axillary temperatures every 5 or 10 minutes, many more cases of hyperthermia would be recognized in time to save the patient's life.

Malignant hyperthermia is probably more frequent than the reports suggest, and routine temperature monitoring in surgery ought to be considered.

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CONVULSIONS FOLLOWING SURGERY

SIR,—In reference to the interesting case reported by Casale (Brit. J. Anaesth. (1970) 42, 1024), I want to recall a similar case of recurring convulsions in a 20-year-old primigravida after the injection of 40 ml of 2 per cent carbocaine without adrenaline from South Africa (Van Dongen and Glietenberg, 1961). Another similar case was discussed by Moore (1966) at the III European Congress of Anesthesiology. As far as I know, these are the only reported cases of recurring convulsions after the use of amide-type of local anaesthetic. Dr Moore termed this type of reaction "cumulative type". It is unlikely, however, that even in the presence of severe hepatic damage, the delayed metabolism of bupivacaine alone could account for a persistent high brain level of this agent, resulting in recurrent convulsive episodes 6 hours after the injection of the local anaesthetic. Therefore, one must suspect a possible genetic abnormality in drug metabolism in these individuals. Blood level determinations would have been useful to clarify this question.

To clarify further the patho-etiologic of convulsions in this case, it would be of great interest to know whether this 35-year-old African female has an abnormal Hb-S, since a sickle-cell crisis might have resulted in the described sequelae. An increase in blood viscosity over 14 times the normal at a shear-rate of 11 seconds⁻¹ during a hypotensive episode in combination with hypoxia and blood loss which all occurred during anaesthetic management, might have led to intravascular sickling (Motulsky and Stamatoyannopoulos, 1968). This in turn might have resulted in pulmonary and cerebral embolization, and congestive failure, which was reported in the case discussed. Since many of the features of sickling crisis were present in this case and since this patient may require surgery again in the future, it will be imperative to recall her and study her for haemoglobinopathies.

SIR,—In reference to the interesting case reported by Casale (Brit. J. Anaesth. (1970), 42, 1024), I want to recall a similar case of toxicity to excess Carbocaine with probable reactivity of rheumatoid disease. S. Afr. med. J., 73.


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HAEMODYNAMIC EFFECTS OF OXPRENOLOL AND PRACTOLOL DURING HALOTHANE ANAESTHESIA

SIR,—Drs A. J. Strong et al. in their letter (Brit. J. Anaesth. (1971), 43, 580) think that my dose of practolol 15 mg i.v. is high by their standards. The reason for our somewhat different findings on the effect of practolol on cardiac output is that my patients were under adrenergic stress when the drug was given and their patients were not. The considerable decreases in cardiac output below the resting levels in their patients after the administration of practolol 15 mg i.v. is not surprising as the patients were not catecholamine depleted. Catecholamine depletion with drugs such as reserpine is essential before a conclusion can be drawn concerning the "direct" myocardial effect of beta adrenergic blocking drugs.

It was interesting to note that their dye dilution technique showed a 25 per cent reduction in cardiac output after the administration of the beta blockers to non-stressed patients. This compares with a reduction of 27 per cent as assessed by venous occlusion plethysmography in vasodilated patients with adrenergically hyperactive hearts in sinus rhythm (Johnstone, 1969). In the latter conditions the fall in cardiac output after beta adrenergic blockade, cardioselective or otherwise, did not exceed the increase caused by the adrenergic stimulation of the heart. This may illustrate the competitive action of the beta blockers when used in the circumstances for which they were intended.

Beta blockers do not invariably lower the cardiac stroke volume and output. When used to control supraventricular or ventricular tachycardias of adrenergic origin they increase the stroke volume by providing a longer diastolic filling time. They increase the stroke volume and output when used to treat a rapid multifocal ventricular tachycardia of similar origin.