SUXAMETHONIUM-INDUCED CARDIAC ARREST AS AN INITIAL MANIFESTATION OF DUCHENNE MUSCULAR DYSTROPHY

Sir,—Duchenne muscular dystrophy (DMD) has been reported previously in association with cardiac arrest after the administration of suxamethonium (Genever, 1971). We report the occurrence of this complication in a 3-month-old child with unsuspected DMD during bilateral herniorrhaphy. After induction of anaesthesia with halothane and nitrous oxide in oxygen, atropine 0.125 mg i.v. was given, followed by suxamethonium 8 mg. Immediately after intubation of the trachea, his face turned pale, widened QRS complexes appeared in the ECG, pupils were dilated and his carotid pulse could not be felt. The patient was successfully resuscitated, and the operation was abandoned. The rectal temperature was 37 °C, urine colour of several samples was normal. Serum potassium concentration was not determined. On the following day, serum potassium concentration was 5.5 mmol litre⁻¹, both ECG and 2D-echocardiogram were normal. Seven days after the event, creatine phosphokinase (CPK) was 706 u. litre⁻¹ (normal < 55). The EMG was consistent with a myopathic process. Duchenne's muscular dystrophy was suspected. The operation was performed 6 months later. Anaesthesia was successful, and consisted of ketamine 100 mg plus hyoscine 0.1 mg i.m. for induction, with i.v. supplementary doses of ketamine up to 25 mg for maintenance. The child breathed 100% oxygen spontaneously. Quadriceps muscle biopsy confirmed DMD.

There are few reports of suxamethonium-induced cardiac arrest in unsuspected DMD in the literature (Genever, 1971; Linter et al., 1982; Henderson, 1984), and this complication is an uncommon initial manifestation of Duchenne disease (Seary, Ziter and Thompson, 1978). Although hyperkalaemia has been thought to be the possible cause of the arrest (Genever, 1971; Linter et al., 1982), this has only been confirmed on one occasion (Henderson, 1984). Cardiac involvement occurs in nearly all patients with DMD, and it has been suggested that an underlying cardiomyopathy could be a contributing factor (Genever, 1971; Henderson, 1984). Several features make our patient noteworthy. This is the youngest patient reported; rigidity, masteter spasms, and myoglobinuria were all absent. Although hyperkalaemia could have been the mechanism behind the arrest (Henning and Busch, 1982), unfortunately, we were unable to measure the serum potassium concentration immediately. The absence of any cardiac abnormalities is of interest.

In conclusion, in every unexpected cardiac arrest after the administration of suxamethonium in a child, serum potassium concentration should be determined, and patients should be evaluated for evidence of muscular dystrophy.

REFERENCES


NORMAL PREGNANCY FOLLOWING NITROUS OXIDE EXPOSURE IN THE FIRST TRIMESTER

Sir,—Nitrous oxide is widely used as an analgesic agent and as an adjunct to anaesthesia. Its use for over 100 years is testimony to its safety, but evidence now suggests that it is less innocuous than previously thought. The inactivation of vitamin B₁₂ by nitrous oxide, with the resulting haematological consequences, is well documented (Nunn and Chanarin, 1985). Less is known, however, about the possible teratogenic effects of nitrous oxide.

Female rats exposed to nitrous oxide in early pregnancy show an increased incidence of resorptions and both soft tissue and skeletal abnormalities in their offspring (Fink, Shepard and Blandau, 1967; Lane et al., 1979). Spermatogenesis is reduced in male rats after long-term exposure to nitrous oxide (Kripke et al., 1976). The evidence for a teratogenic effect in human embryos lies in a number of epidemiological surveys. Operating theatre nurses, female anaesthetists, and dental practitioners and dental assistants all have a higher incidence of spontaneous abortion than control groups (Cohen, Bellville and Brown, 1971; Cohen et al., 1980). The incidence of congenital abnormalities in the children of women occupationally exposed to nitrous oxide is also increased (Corbett et al., 1974; Cohen et al., 1980). Other anaesthetic agents have been investigated and found not to be teratogenic (Halsey et al., 1981).

Information regarding the possible risk to pregnant patients exposed to nitrous oxide is limited since anaesthesia and surgery are seldom undertaken during early pregnancy. An increased incidence of birth defects following surgery in the first trimester was demonstrated in one study, but no details were given to incriminate nitrous oxide as the causative factor (Shnider and Webster, 1965). It has also been suggested that nitrous oxide is contraindicated in the first two trimesters of pregnancy (Nunn and Chanarin, 1985).