MASSETER SPASM FOLLOWING INTRAVENOUS SUXAMETHONIUM

A Case Report

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SUMMARY
An abnormal response to suxamethonium is reported. Masseter spasm followed intravenous administration of the drug, and was associated with an elevated serum creatine phosphokinase at rest. The implications of the reaction are discussed.

CASE REPORT
A woman of 25 was admitted to Princess Mary's Royal Air Force Hospital, Halton, for tubal ligation. Preoperative history and examination revealed no abnormality. Chest X-ray was normal, and the haemoglobin level was 14.3 g/100 mL. Premedication was administered 1 hour prior to induction of anaesthesia and consisted of papaveretum 15 mg and hyoscine 0.3 mg.

Anaesthesia was induced with methohexitone 70 mg and was followed by suxamethonium 70 mg to facilitate endotracheal intubation. Moderate muscle fasciculations occurred after the injection. The lungs were inflated without difficulty with 100% oxygen. Subsequent laryngoscopy was impossible because the teeth were tightly clenched due to contractions of the masseter; oral intubation was clearly not feasible. Lung inflation was continued for 2 minutes with 2% halothane vaporized in 50% nitrous oxide and oxygen. Atropine 0.4 mg was administered intravenously and followed by suxamethonium 50 mg taken from a different batch. Mild muscle fasciculation followed the injection but the masseter muscles remained in spasm and it was not possible to open the mouth. The arms were passively flexed and there was no abnormal tone. The operation was abandoned, and the patient recovered consciousness 15 minutes after induction. Her oral temperature was 37.8°C at this time; it returned to normal on return to the ward and remained so.

Subsequent investigation.
Blood was withdrawn at the termination of the anaesthetic, the serum potassium value was 4 m-equiv/l. and creatine phosphokinase 163 mu/ml (CPK was measured using the UV system. CPK supplied by Boehringer, Mannheim; normal values up to 50 mu/ml). On the following day the patient complained of severe muscle pain in the loins, abdomen and legs. Urine was found on ward testing to be hazy and contained blood. The CPK was determined after overnight rest and under fasting conditions, and was found to be 63 mu/ml. In view of the abnormal response to suxamethonium, coupled with a high resting CPK, the operation was performed under epidural anaesthesia and intravenous diazepam.

The patient was subsequently subjected to electromyographic examination of the biceps and quadriceps muscles. The results were entirely normal.

DISCUSSION
It is well known that patients suffering from myotonia congenita, myotonia dystrophica or other rare muscle diseases can react to suxamethonium with an increase in muscle tone instead of relaxation (Paterson, 1962; Thiel, 1967; Lody, 1968). In these cases, muscle spasticity develops most often in the jaw muscles and this is not followed by the usual relaxation but persists.

A case in which suxamethonium 100 mg failed to produce muscular relaxation was described by Jensen and associates (1968). In this case a total of 250 mg of suxamethonium was administered to facilitate endotracheal intubation. The anaesthetic was followed by myoglobinuria and elevated levels of creatine kinase. Other cases of myoglobinuria following anaesthesia including suxamethonium have been described (Bennike and Jarnum, 1964; Airaksinen and Tammisto, 1966). In the case reported the urine was hazy and macroscopically contained blood on the day after administration of suxamethonium. Unfortunately a sample of urine sent for detection of myoglobin was lost. It is possible that had
further investigation is warranted. CPK estimation and electromyography should be repeated in the future with the addition of motor point muscle biopsy. The patient's relatives should have CPK estimations before general anaesthesia. In cases where a normal dose of suxamethonium fails to produce muscular relaxation, further doses should be administered with caution.

REFERENCES