AN UNUSUAL CAUSE OF APNOEA ON THE TABLE

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SYNOPSIS

A case of apnoea due to absorption of Streptomycin from the orbital cavity is reported and discussed.

INTRODUCTION

Experimental studies in the recent past (1, 2, 3) on neuromuscular blockade produced by antibiotics has prompted this case report.

CASE REPORT

A six year old boy weighing 20 kgs was to have an evisceration of the right eye. Atropine 0.5 mg was given I.M, 20 minutes before induction. Induction was carried out using halothane, and suxamethonium 35 mg was given I.V for intubation. Anaesthesia was maintained using 1.5% halothane, oxygen and nitrous oxide. After the eviscération which took 20 minutes, about 100 mg of Streptomycin was introduced into the orbital cavity. It was noticed that the child becamed apnoeic within 5 minutes after the addition of Streptomycin and controlled respiration was started using Magill's open circuit. Fortunately respiration recommenced 15 minutes later and remained normal thereafter. There was therefore no necessity to put him on a ventilator. In the absence of a nerve stimulator a clinical diagnosis of apnoea due to streptomycin was made.

DISCUSSION

Prolonged apnoea related to neuromuscular blocking agents can occur with both depolarising relaxants and the competitive blocking agents. In this instance. the apnoea was related to the addition of Streptomycin. Aminoglycosides like streptomycin, neomycin and other antibiotics are known to potentiate the competetive neuromuscular blocking agents and care should be exercised when streptomycin or neomycin is introduced intraperitoneally during abdominal surgery. In this case however the small amount of streptomycin introduced into the orbital cavity has produced generalised neuromuscular block and apnoea in a patient breathing spontaneously. This is rather unusual. The patient was however breathing spontaneously on halothane which is known to have relaxant properties and it is probable that the two effects had been additive resulting in generalised neuromuscular blockade.

This facts are quite in agreement with the experimental evidence obtained by Singh, Marshall and Harvey (1) on anaesthetised cats. They noticed that the time of maximum twitch depression was 4 minutes when amikacin was given I.V. They also found that the time of recovery from 75% depression to 25% depression was 10 minutes. When tubocurarine 0.3-0.5 mg/kg was given and the animals allowed to recover until 50% twitch depression remained, the dose of amikacin required for adequate neuromuscular block was 16 time less.

In 1982 Singh et al (3) found that the concentration of streptomycin required to produce paralysis in the nerve muscle preparation of rats was 0.6 mmol/L and this of course is many times the therapeutic dose. In our patient it may be concluded that sufficient streptomycin had been absorbed to produce an obvious degree of neuromuscular blockade in conjunction with halothane.

The mechanism of action of most of the antibiotics which produce neuromuscular blockade have recently been elucidated (3). The aminoglycosides streptomycin, amikacin and neomycin act like magnesium prejunctionally at nerve endings. They compete with Calcium and so prevent to release of acetylcholine. This action can be overcome by increasing the concentration of Ca^{++}. Other antibiotics which act in a similar mannar are Polymyxin B and Oxytetracycline.

The aminoglycosides also have an action on the post-synaptic membrane of nerve endings. They reduce the ionic conduction by blockade of the receptor activated ion channels and thereby prevent depolarisation. Other antibiotics which have an effect here are Lincomycin, Clindamycin and Polymyxin B. The site of action of tetracycline is not very clear.

The prejunctional effect could be distinguished from the postjunctional effect by their effects on miniature and plate potential (m.e.p.p). The former reduces the m.e.p.p while the latter abolishes the m.e.p.p. Further, the prejunctional effect can be reversed by increasing the Calcium concentration. Aminopyridines are potentially useful in the reversal of both prejunctional and postjunctional block (1). while neostigmine is effective in the postjunctional block. In a clinical situation however it is wiser to rely on ventilation till the effect of the drug wears off.

Prigden in 1956 (4) has reported 4 cases of apnoea due to intraperitoneal instillation of neomycin. The mechanism of the respiratory arrest was probably not well known then; as this was shortly after the introduction of muscle relaxants into clinical anaesthesia. This was also a time when intestinal antisepsis was in practise (5). Of the 4 cases reported two were infants who ended tragically. In these two cases five tenth gram of neomycin in a dilute solution was instilled into the peritoneal cavity during anaesthesia and surgery. It must be remembered however that absorption can occur later in the ward and lead to apnoea. Also the competitive muscle relaxants like tubocurarine. gallamine and volatile anaesthetic agents like ether. halothane and methoxyflurane can be potentiated by antibiotics, and great care should be exercised when using them in conjunction.

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