BRONCHOSPASM DUE TO SUXAMETHONIUM

Report of a Case

BY

ANDREW A. FELLINI, RALPH L. BERNSTEIN AND HOWARD L. ZAUDER

Department of Anesthesiology, Bronx Municipal Hospital Center, Albert Einstein College of Medicine, New York, U.S.A.

SUMMARY

Severe bronchospasm developed in an 11-year-old female during intra-abdominal surgery. Suxamethonium was incriminated as the aetiological agent. This was confirmed by repeated administration of the relaxant and by intradermal skin-testing.

All muscle relaxants in clinical use exhibit histamine-releasing properties. “The mere possession of such an action is of little significance, for it is displayed by a very large range of bases, if sufficiently high concentrations are administered. The crucial point lies in the comparison of doses required to release histamine with that required to produce neuromuscular block” (Paton, 1959). It is generally agreed that, of all the muscle relaxants, d-tubocurarine is the most potent in releasing histamine in doses within the range of clinical practice (Comroe and Dripps, 1946; Landmesser, 1947; Paton, 1959). Suxamethonium, on the other hand, has but 1/100 the histamine-releasing activity of d-tubocurarine (Bourne, Collier and Somers, 1952). Histamine release may manifest itself as bronchospasm. The bronchospastic properties of curare, secondary to histamine release in the anaesthetized patient, are well known. Bronchospasm following the usual clinically administered dose of suxamethonium is virtually unknown. When spasm of the bronchi and bronchioles occurs it is usually the result of poor technique (Davis, 1956). The presence of an oropharyngeal airway or of an endotracheal tube in a patient who is too lightly anaesthetized, an excess of secretions, and a sudden increase in the concentration of irritant anaesthetics, have all been incriminated as the offending agents. Although the administration of massive doses of suxamethonium is capable, at least theoretically, of producing bronchospasm, this has not been reported in man (Paton, 1959). Similarly, normal doses are thought to be capable of inducing bronchospasm as a result of histamine release in the sensitized or hyper-reactive patient. Laurie Smith (1957) reported that in four patients weal and flare formation had been demonstrated following 10-mg doses of suxamethonium administered intradermally. Bronchospasm occurred in one of these patients during anaesthesia. The aetiology was thought to be histamine release secondary to suxamethonium; however, the patient was a known asthmatic, presenting with partial bronchospasm. Anaesthesia was induced with thiopentone, an endotracheal tube was in situ. The inhalation anaesthetic agent used was not mentioned by the author. The following case illustrates, it is believed, bronchospasm appearing in a patient under anaesthesia, secondary to histamine release due to administration of suxamethonium.

CASE REPORT

E.M., an 11-year-old white female, was admitted to hospital with a diagnosis of acute appendicitis. Height 5 ft. 5 in. (165 cm), weight 150 lb. (68 kg). There was no history of prior illness and she gave no history of food allergy, hay fever, or asthma. Other than for the local condition, physical examination was negative. Her physical status was classified as E-1.* Hematocrit 39 per cent; urine analysis was negative.

Premedication, consisting of pentobarbitone 75 mg and atropine 0.4 mg, was administered intramuscularly half an hour prior to anaesthesia. Anaesthesia was induced with 50 per cent cyclopropane and 50 per cent oxygen in a closed circle carbon dioxide absorption system. Induction was smooth and anaesthesia was continued with intermittent cyclopropane. Suxamethonium 0.1 per cent was administered by intravenous drip for muscular relaxation after the

* According to the classification of physical status adopted by the American Society of Anesthesiologists, this denotes a normal healthy patient undergoing an emergency operation.
peritoneum was opened. Approximately 15 minutes had elapsed between the induction of anaesthesia and the first administration of muscle relaxant.

Coincident with the administration of the muscle relaxant, the patient's lungs became difficult to ventilate, she began to manifest tachycardia (100-140/min), a rise in blood pressure (120/75-140/80 mm Hg), and slight cyanosis. Bilateral apical wheezes, with decreased aeration of both lungs, were noted on auscultation. Approximately 10 minutes after the appearance of the respiratory difficulty, cyclopropane was discontinued. Anaesthesia was continued with ether and oxygen in a closed system. Anaesthesia was continued in this manner for another 20 minutes, the patient continuing to have partial bronchospasm. Laryngoscopy was performed but no secretions were noted, nor were any anatomical abnormalities seen. At this point the suxamethonium drip was discontinued and the anaesthesia changed to nitrous oxide, oxygen with ether given in a semiclosed system. As the child began to breathe spontaneously, the wheezes became minimal; respiratory exchange was judged adequate. At this time the suxamethonium drip was again turned on to obtain further muscle relaxation; once again the patient became extremely difficult to ventilate, wheezes appeared in both lung fields, and the blood pressure and pulse rate started to rise. The suxamethonium drip was again turned off; the wheezes once again disappeared with the onset of normal spontaneous respiration. Considering the possibility of a contaminated or mis-labelled bottle the intravenous fluid bottle containing the 0.1 per cent suxamethonium was exchanged for a fresh bottle. Once more, with the administration of this fresh solution bronchospasm occurred. At this time the peritoneum was closed, suxamethonium was discontinued; with the appearance of normal spontaneous respiration the patient again ventilated adequately with no sign of bronchospasm.

She was allowed to continue in this manner for another 15 minutes, when a single 20-mg dose of suxamethonium was administered intravenously. Again, the patient manifested the same response, becoming "tight", wheezes appeared bilaterally; the blood pressure and pulse rate at this time were unchanged. The respiratory difficulty again cleared when the child was allowed to respire spontaneously. Anaesthesia was continued with nitrous oxide, oxygen and ether until the end of the operation when the patient was given oxygen for 5 minutes; following this a single dose of 20 mg of suxamethonium was given intravenously. The patient at this time was virtually awake. Again manual ventilation became difficult, wheezes appeared in both lung fields. Within 5 minutes, she began to breathe spontaneously, the wheezes disappeared and the ventilatory exchange was normal. A total dose of 200 mg suxamethonium was administered. In the immediate postoperative period,

![FIG. 1](https://academic.oup.com/bja/article-abstract/35/10/657/241925)

Response to intradermal suxamethonium and saline solution.
her lungs were clear, blood pressure and pulse were at pre-operative levels. She was returned to the ward in good condition.

On the third postoperative day an intradermal skin test was performed with 2 mg of suxamethonium. The test solution was prepared with pure lyophilized powder, thereby avoiding non-specific reactions to preservatives. A typical weal and flare with pseudopod formation resulted (fig. 1). A control injection of 0.9 per cent sodium chloride failed to elicit this response. Repeat skin test the next day half an hour after the intramuscular administration of diphenhydramine 75 mg, showed marked attenuation of the weal and flare formation; pseudopods were absent.

DISCUSSION
Although several cases of bronchospasm have been reported as having been due to suxamethonium, there were so many variables present that it is almost impossible to incriminate the relaxant as the aetiological agent. Light anaesthesia, the presence of secretions, airways, and endotracheal tubes, and the use of parasympathomimetic drugs were usually concomitant with bronchospasm. More often than not suxamethonium is given as an aid in the differential diagnosis of bronchospasm and chest wall spasm (Gold and Helrich, 1963).

In the case presented, when the bronchospasm first manifested itself, all parasympathomimetic drugs were eliminated. An oropharyngeal airway was placed only when the patient was adequately anaesthetized; no secretions were present nor was any abnormal pathological condition noted during laryngoscopy. Once the diagnosis of bronchospasm was made, ether was administered in increasing concentrations, to no avail. Only when the suxamethonium was discontinued did the bronchospasm recede, to return when suxamethonium was readministered.

The mechanism by which histamine is released from the tissues is not well understood (Paton, 1957). Why this idiosyncrasy is demonstrable only in the occasional patient remains unexplained. Kepes and Haimovici (1959) observed hypotension and the skin manifestations of histamine release following suxamethonium. In their case intracutaneous tests to the drug were negative. However, intravenous administration identified the relaxant as the offending agent. It should be emphasized that these reactions are not true allergies. In the true sense of the word, “allergy” implies previous sensitization. There was no indication of previous administration of suxamethonium either in the case reported above or in the case report of Kepes and Haimovici. One should, therefore, consider these responses to be an idiosyncrasy or a hypersensitivity to suxamethonium.

REFERENCES


Smith, N. Laurie (1957). Histamine release by suxamethonium. Anaesthesia, 12, 293.

UN CAS DE SPASME BRONCHIQUE Dû AU SUXAMETHONIUM

SUMMARY
Chez une petite fille âgée de 11 ans survint au cours d'une intervention intra-abdominale un spasme bronchique grave. Le Suxaméthonium, aussi tôt souçonné comme cause, était réellement l'agent étiologique. La remission après administration répétée d'une substance de détente appropriée et la réaction au test intradermique en fournissaient ensuite la confirmation.

BRONCHOSPASMUS DURCH SUXAMETHONIUM

ZUSAMMENFASSUNG