BRONCHOSPASM INDUCED BY SUXAMETHONIUM
A Case Report

A. M. KATZ AND PATRICIA G. MULLIGAN

SUMMARY

The case is presented of a 53-year-old woman with an allergic diathesis who developed bronchospasm believed to be due to the administration of suxamethonium. The differential diagnosis is discussed.

Bronchospasm attributable to suxamethonium is unusual, although several case reports have been published (Smith, 1957; Kepes and Haimovici, 1959; Fellini, Bernstein and Zauder, 1963; Jerums, Whittingham and Wilson, 1967; Eustace, 1967; Bele-Binda and Valeri, 1971). We have recently observed a case of bronchospasm following suxamethonium administration.

CASE REPORT

A 53-year-old female was scheduled for elective cholecystectomy. During the preoperative interview, the patient stated that she was allergic to sulphur medications, which produced a generalized rash, and that she suffered from "hay fever" for which she took Ornade spansules (a preparation containing chlorpheniramine, phenylpropanolamine, and isopropanamide). She denied any history of asthma and did not smoke. Physical examination revealed an obese woman, a 1.5 cm nodule in the left thyroid gland, but no other physical abnormalities. Laboratory tests showed Hb 14.7 g/100 ml, WBC 6000/ cu.mm with normal differential count, normal urinalysis, chest X-ray and electrocardiogram.

On the morning of surgery she was premedicated one hour preoperatively with morphine 8 mg and hyoscine 0.4 mg i.m. The preanaesthetic blood pressure was 120/80 mm Hg and pulse 96 beats/min. A solution of 5 per cent dextrose in Ringer-lactate solution was administered intravenously. Anaesthesia was induced using thiopentone 250 mg and halothane 1 per cent in a mixture of nitrous oxide 3 l/min and oxygen 3 l/min by mask. Gallamine 10 mg was given intravenously. Three minutes later suxamethonium 80 mg was given i.v. in a bolus injection and the expected degree of relaxation ensued. Intubation was easily accomplished with a No. 36 Magill endotracheal tube the cuff of which was inflated with 6 ml of air. Approximately one min after the suxamethonium injection, wheezing, difficulty in ventilation requiring a greater than usual peak inspiratory pressure and prolonged expiratory phase were noted. The blood pressure was 120/80 mm Hg and pulse was 80 beats/min. A diagnosis of bronchospasm secondary to a light plane of anaesthesia was suspected and the anaesthetic level was deepened using 2 per cent halothane in nitrous oxide and oxygen together with hyperventilation. Over a period of 8 to 10 min the systolic blood pressure dropped to 75 mm Hg and the pulse rate dropped to 68 beats/min with no lessening in the degree of bronchospasm. Atropine 0.4 mg was given intravenously and the heart rate rose to 96 beats/min but the pulse became temporarily impalpable. The difficulty in ventilation continued and halothane was discontinued. The position of the endotracheal tube was checked. Suction was applied down the endotracheal tube but no secretions were obtained. The breath sounds were noted to be of equal quality in both lung fields. Both inspiratory and expiratory wheezing were heard. The cuff of the endotracheal tube was easily deflated but this produced no change in breath sounds or ability to ventilate and the cuff was then reinflated. The systolic blood pressure rose after 5 min on 50 per cent nitrous oxide and 50 per cent oxygen to 120 mm Hg and the pulse remained at 96 beats/min. Suxamethonium (two 1 g ampoules of suxamethonium powder dissolved in 1 l. Ringer-lactate solution) was administered by intravenous infusion. Halothane 0.5 per cent was added to the inspired gas mixture. An isoproterenol aerosol was inserted into the breathing circuit. The bronchospasm lessened after two spray doses administered during inhalation and ventilation became easier, but within 5 min returned to the original severity. At this time the suxamethonium infusion was discontinued and a third dose of isoproterenol administered by aerosol. Gallamine 60 mg was given intravenously to provide surgical relaxation. The difficulty in ventilation and wheezing disappeared within three min. Systolic blood pressure was 120 mm Hg and pulse 96 beats/min. After 15 min of normal ventilation a challenge dose of suxamethonium (10 mg in 0.5 ml of the original solution used for intubation) was given intravenously. Wheezing and difficulty in ventilation began again within one min and continued for 12 min, regressing gradually without treatment. There was a concurrent fall in blood pressure to 100/70 mm Hg and it then returned to 120/80 mm Hg.

An intradermal skin test was made using 0.1 ml of the original solution of 1 per cent suxamethonium and 0.1 ml of 0.9 per cent NaCl as a control. An erythematous weal 1 cm in diameter appeared at the suxamethonium injection site but no reaction developed at the control site.

The remainder of the surgical procedure continued without incident. The patient required no more than the original 60 mg dose of gallamine for muscle relaxation. A total of atropine 1.2 mg and neostigmine 3.0 mg intravenously was required to reverse the neuromuscular block.

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The views expressed herein are those of the authors and do not necessarily reflect the views of the United States Air Force or the Department of Defense.
Table I. Suxamethonium induced reactions.

<table>
<thead>
<tr>
<th>Author</th>
<th>Sex</th>
<th>Age</th>
<th>Allergy</th>
<th>Asthma sux.</th>
<th>Exp to Bronchos</th>
<th>BP</th>
<th>Cyan-Card mani-</th>
<th>Skin Sur-</th>
<th>Skin Survived</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smith (1957)</td>
<td>M</td>
<td>72</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>?</td>
<td>?</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Kepes (1959)</td>
<td>M</td>
<td>61</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Fellini (1963)</td>
<td>F</td>
<td>11</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Jerums (1967)</td>
<td>F</td>
<td>26</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Eustace (1967)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 1</td>
<td>M</td>
<td>57</td>
<td>possible</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Case 2</td>
<td>F</td>
<td>54</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Bele-Binda (1971)</td>
<td>F</td>
<td>18</td>
<td>-</td>
<td>?</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>?</td>
</tr>
<tr>
<td>Katz (1972)</td>
<td>F</td>
<td>53</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
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DISCUSSION

The diagnosis of sensitivity to suxamethonium was reached by ruling out other more common causes of bronchospasm, and confirmed by the administration of a challenge dose of suxamethonium. The possibility that a light level of anaesthesia was responsible is unlikely because there was no response to the increase in the concentration of halothane to the point of obvious cardiovascular depression. A true asthmatic attack was also unlikely considering the absence of a history of asthma and the second attack of bronchospasm following the challenge dose of suxamethonium. Mechanical obstruction as a cause was considered and excluded.

It is conceivable that the patient was sensitive to the Ringer-lactate solution that served as a carrier for the suxamethonium. However, the onset of bronchospasm shortly after the initial bolus dose, and its subsequent demonstration after the second challenge dose, incriminates the suxamethonium itself rather than the carrier solution. It is possible that the patient was sensitive to the preservative or solution carrying the suxamethonium in the original vial. However the manufacturer states that powdered suxamethonium contains no preservative. The patient's reaction to the suxamethonium in the continuous infusion would then necessitate the presumption of a reaction to the preservative in both the Ringer-lactate solution and the original vial of suxamethonium. Sensitivity to suxamethonium is the most likely explanation.

It is unlikely that thiopentone was the agent responsible for the sequence of events. The prompt disappearance of bronchospasm after discontinuing the suxamethonium infusion and its redevelopment shortly after the test dose of suxamethonium are more indicative of a reaction to suxamethonium than to thiopentone.

The hypotension noted in this patient was most probably accounted for by the deeper level of anaesthesia caused by hyperventilation with halothane and was not anaphylactic in nature. The prompt recovery, lack of associated tachycardia, and the fact that a challenge dose of suxamethonium elicited only slight and possibly fortuitous change in vital signs, support this thesis.

Although there was a positive skin test response to intradermal injection of suxamethonium, this reaction is nonspecific and probably acts more as an irritant than an antigen. The diagnosis must rest primarily on the clinical response of the patient to the administered drugs.

REFERENCES


BRONCHOSPASME SUSCITE PAR SUXAMETHONIUM: DESCRIPTION D'UN CAS

SUMMARY

Les auteurs décrivent le cas d'une femme de 53 ans avec diathèse allergique, qui développait un bronchospasme, supposé dû à l'administration de suxamethonium, et discutent du diagnostic différentiel.
BOOK REVIEW

Lidocaine in the Treatment of Ventricular Arrhythmias.

The book reports the Proceedings of a Symposium held in Edinburgh in September 1970. Though its title suggests a consideration of ventricular arrhythmias, it is, in fact, devoted to the treatment of those arrhythmias associated with myocardial infarction. Indeed, all but two of the contributors are cardiologists, pharmacologists or electrophysiologists. Nevertheless, there is much that is of interest and of use to the anaesthetist.

The book is conveniently divided into four sections corresponding with the four separate sessions of the Symposium.

Section I is devoted to a consideration of the genesis of cardiac arrhythmias. The paper by Vaughan-Williams on the basic electrophysiology of arrhythmias is commendably clear and concise when one considers the volume, complexity, and often contradictory nature of the results of investigations in this field. In view of this, his classification of types of anti-arrhythmic agents into three broad groups and his conclusions are attractively and acceptably simple. Oliver's account of metabolic factors in the genesis of arrhythmias is similarly clear and concise. Both papers end with lists of up-to-date references for the reader who wishes to pursue the subjects in more detail. The third and fourth papers in this section are too hypothetical, too technical, and too confusing to be of any use to other than a researcher in this field. Moreover, the frequent use by Bigger of a multiplicity of home-made abbreviations I found distinctly irritating and far from conducive to a rapid grasp of the subject matter.

Section II is devoted to a series of papers describing the prophylactic and therapeutic uses of lignocaine in myocardial infarction and to a paper by Katz and Katz on its use in anaesthesia. The first paper in this section is a practical presentation of the problems of diagnosis, treatment and prevention of the arrhythmias of myocardial infarction. It makes the first of several important references to the possibility of altered responses to anti-arrhythmic drugs produced by certain other drugs and by anaesthetic agents. Several subsequent papers report the results of clinical trials of lignocaine. The wide variations in the results of therapy reported in these papers are not, in fact, contradictory, but merely witness to the fact that the results of clinical trials cannot be compared unless the population samples are identical. The use only of Pantridge of the term "dysrhythmias" is an inconsistency the editors might well have eliminated in the written text, particularly since even he used the term "arrhythmias" in his title.

The views presented by Katz and Katz are more or less a repeat of those expressed elsewhere in the anaesthetic literature and should already be familiar to most anaesthetists. The dispute which exists in anaesthetic circles about the relevance of anaesthetic arrhythmias is resurrected in the discussion following this paper and, as usual, is not resolved. However there is support for the view that lignocaine is the drug of choice if anaesthetic arrhythmias are not to be ignored.

The third section, which deals with the pharmacology and toxicology of lignocaine, is, on the whole, well written and useful, if somewhat repetitive. It rightly emphasizes the desirability of frequent blood level estimations, the danger of asystole following intravenous lignocaine in patients with bradycardia or atrioventricular block and, in a moment of true inspiration, the fact that lignocaine has its failures and that its presence in the blood does not guarantee immortality.

Section IV compares the haemodynamic effects and efficiency of lignocaine with those of other anti-arrhythmic agents. One is left with the impression—and rightly so—that both lignocaine and practolol are equally efficient and have comparably minor effects on haemodynamics in therapeutic doses.

The book is neatly produced with few spelling mistakes, contains many useful references, and has an adequate index. Since first edition and reprint both appear within a year, it obviously has a wide market despite its rather high price of £3.

For individual anaesthetists it contains too little of relevance to justify its price tag and is, to a degree, already out-of-date. However, as a volume for an anaesthetic departmental library it could provide a useful reference background for a logical approach to the treatment of ventricular arrhythmias.

W. Ryder