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Case Report

Prolonged effect of succinylcholine following reversal of residual rocuronium bromide block: A case report

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ABSTRACT

Succinylcholine is administered in small doses to relieve post-extubation laryngospasm. We report a 3-month-old infant weighing 5.1 kg who developed severe laryngospasm after tracheal extubation following reversal of rocuronium bromide neuromuscular block. Succinylcholine 2.5 mg promptly relieved the spasm but apnea lasted for nearly an hour in a patient with normal dibucaine number. We postulate that this prolonged apnea was related to the timing of succinylcholine administration in relation to the degree of recovery from non-depolarising neuromuscular block.

1. Introduction

The incidence of laryngospasm is higher during the perianesthetic period in infants younger than 1 year in comparison with older children.[1] When initial steps of treating laryngospasm such as positive pressure mask ventilation have failed to relax the vocal cords, succinylcholine administration can be effective.[2,3] It relieves the spasm quickly and patient resumes spontaneous respiration within 4-6 min when administered in doses of <0.5 mg/kg. Recently, we noted an unexpected response of prolonged apnea following succinylcholine in a patient with laryngeal spasm after nearly full recovery from non-depolarising block.

2. Case Report

A 3-month-old male infant weighing 5.1 kg was scheduled for elective repair of cleft lip. The infant was not known to have any associated syndromes and was evaluated as American Society of Anesthesiologists (ASA) physical status I. His laboratory findings were all within acceptable limits. However, history was positive for upper respiratory tract infection approximately 2-weeks prior to surgery, but he was currently asymptomatic.

The infant did not receive any premedication and had fasted for 5 hours. Anesthesia was induced with sevoflurane in 100% oxygen via facemask. After intravenous (IV) cannulation, fentanyl 7.5 mg (1.5 μ/kg) and rocuronium 4 mg (0.8 mg/kg) were administered. Tracheal intubation was uneventful. Anesthesia was maintained with sevoflurane 2-3% in a 50%-50% mixture of nitrous oxide and oxygen, with pressure controlled, volume guaranteed mode of ventilation. Paracetamol suppository (125 mg) was administered per rectum and dexamethasone 1 mg was given IV. Surgery lasted approximately one hour. No further muscle relaxant or fentanyl was administered. After conclusion of surgery, sevoflurane and nitrous oxide were discontinued and the infant started making respiratory efforts. A mixture of neostigmine 0.2 mg (40 μg/kg) and atropine 0.1 mg (20 μg/kg) was administered. Within 5-6 minutes, patient started having adequate respiratory efforts, and was moving all extremities. Assessment of neuromuscular function was made subjectively, based on clinical signs (tidal volume and peripheral muscle function adequacy) without train-of-four (TOF) monitoring. Almost immediately after tracheal

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extubation, the infant's end-tidal carbon dioxide ($EtCO_2$) waveform was lost, despite attempts at spontaneous ventilation as evidenced by suprasternal retractions and abdominal muscle contractions. A diagnosis of laryngeal spasm was made. Despite jaw thrust maneuver and positive pressure ventilation with 100% O_2 via facemask, obstruction was not relieved and SpO_2 decreased from 96% to 56%, and the infant's heart rate decreased from 110 beats-per-minute (BPM) to 82 BPM. Since small doses of propofol may be ineffective in terminating laryngospasm in up to 25% of children,[4] the attending anesthesiologist administered succinylcholine 2.5 mg (0.5 mg/kg) IV. Almost immediately, the laryngeal spasm was relieved and the infant was easily mask-ventilated resulting in improvement of SpO_2 to 98% and heart rate to 112/BPM.

Recovery from succinylcholine depolarizing block usually occurs within 4 to 8 min; in this infant, however, the neuromuscular block lasted for nearly 50 min, with no response to TOF monitoring at orbicularis oculi muscle. Over the next 25 min, the infant gradually started breathing spontaneously and respiration was fully restored within 30 min. TOF responses of equal intensity was noted prior to transferring the patient to recovery room and thereafter to high dependency unit with no further untoward incident. A month later, patient was investigated for pseudocholinesterase deficiency, and dibucaine number was reported as 82. At one and three months follow-up visits, the infant continued to be healthy and was developing normally.

3. Discussion

In our case, laryngeal spasm could have been precipitated by any one of the known triggers of reflex laryngeal response that existed in the patient such as secretions, blood, removal of pharyngeal pack and oropharyngeal suctioning via catheter in an airway that has recently recovered from upper respiratory tract infection.[5]

So what could have produced this abnormally prolonged spell of apnea after a small dose of succinylcholine in this infant? Search of literature provided an interesting and perhaps lesser-known fact about this response. Rouse and Bevan in 1979[6] reported a variable response to succinylcholine when it was administered during recovery from non-depolarising muscle relaxant; the response to succinylcholine appeared to depend upon the degree of recovery from non-depolarising block.[6] It was postulated that when succinylcholine is administered in the early phase of recovery from a non-depolarising blockade, many neuromuscular receptors are still occupied by the nondepolarizing blocking agent. In this situation, succinylcholine, by its depolarising action, partially antagonizes the neuromuscular blockade and produces some degree of temporary reversal. In contrast, when succinylcholine is given when more than 50% recovery has taken place (or later than 5 minutes after neostigmine administration, when recovery is more advanced), the non-depolarizing agent has been displaced from the postsynaptic receptors sufficiently; thus, the butyrylcholinesterase responsible for degradation of succinylcholine has been effectively inhibited by this time, resulting in a prolonged depolarizing block that may last up to 60 minutes.[6,7]

In our patient, the non-depolarising block had recovered sufficiently, as evidenced by the presence of adequate respiratory and voluntary muscle movements at the time of succinylcholine administration. It is therefore reasonable to postulate that the prolonged (50 min) duration of succinylcholine block at this level of non-depolarizing recovery was the result of butyrylcholinesterase inhibition, as reported previously.[6]

This case report should remind clinicians of the potential for prolonged duration of succinylcholine when it is used to treat post-extubation laryngospasm, particularly when it is administered at more than 50% recovery of non-depolarizing block. Similarly, the dose (not just the timing) of succinylcholine may impact its effects on recovery from a non-depolarizing block: a small dose of succinylcholine (0.1 mg/kg) at 50% non-depolarizing recovery will elicit a temporary twitch height increase (i.e., recovery), while a larger dose (0.3 mg/kg) may result in a decrease in twitch height (i.e., enhanced block).[6]

In a known case of patient recovering from upper respiratory tract infection, one should consider pre-emptive strategy to reduce the risk of post extubation laryngospasm. Lignocaine both topical and IV may be considered but keeping in mind the potential complications of topical application.[8] A meta-analysis of 1,416 pediatric patients found that both topical and intravenous lidocaine was effective in preventing laryngospasm following general anesthesia.[9] The salutary effects of lidocaine, however, must be weighed against the potential for complications associated with its use: for instance, the clinician must consider the effect of topically applied lidocaine on the sensitivity of upper airway reflexes and ability to swallow, the duration that airway protective reflexes are obtunded, and the safe maximum dose of lidocaine that should be used in the pediatric population.[10,11] Unlike lidocaine, propofol helps in relieving laryngospasm by deepening the plane of anaesthesia but may be ineffective in 25% of cases, and laryngospasm may recur upon re-emergence.

Finally, a brief but very important comment must be made about the use of succinylcholine in the pediatric population. Because of instances of life-threatening malignant hyperthermia and reports of rare, but often fatal, hyperkalemic cardiac arrests in young boys with undiagnosed muscular dystrophy, the U.S. Food and Drug Administration (FDA) issued a warning in 1994, "the use of succinylcholine in pediatric patients should be reserved for emergency intubation or instances where immediate securing of the airway is necessary."[12]

This case report would be of great value when deciding on the use of succinylcholine to relieve laryngeal spasm after reversal of non-depolarizing block.

The learning points from this case report are:

1. Laryngospasm in pediatric patient is a life threatening complication that needs urgent management
2. The first line of pharmacological treatment of post-extubation laryngospasm might be propofol rather than succinylcholine.
3. In case succinylcholine is to be used, especially later during the post-non-depolarizing reversal period, one should keep in mind the possibility of prolonged apnea.
4. The use of succinylcholine in the pediatric population should be reserved for emergency settings in which rapid airway management is paramount.

4. Source of Funding

None.

5. Conflict of Interest

None.

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