ratories, our technique may be considered when more conventional ones fail.

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Succinylcholine Resistance in a Patient with Juvenile Hyaline Fibromatosis

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SUCCINYLCHOLINE resistance is a rare phenomenon that may occur in patients with increased plasma cholinesterase[1-3] or in myasthenic patients.[4-6] The present report describes resistance to succinylcholine in a patient with juvenile hyaline fibromatosis (JHF),[7,8] who had normal cholinesterase values and normal neurologic and neuromuscular functions.

Case Report

A 47-yr-old man, 84 kg, who suffered since childhood from multiple soft-tissue masses, up to 5 cm in diameter and scattered all over the body presented. Histopathology after excisional biopsy revealed darkly stained fibroblasts scattered in hyalinized tissue. The case was diagnosed as JHF.

The patient was operated on in our institution at least 10 times during the past 40 yr for excision of the JHF nodules. Originally, general anesthesia was inhalational N_2O:O_2 supplemented either by ether or by halothane. When succinylcholine was introduced into our practice, anesthesia was induced with thiopental followed by succinylcholine. However, succinylcholine administration was always followed by inadequate jaw relaxation, despite the administration of repeated doses. The patient was labeled as resistant to succinylcholine.

In the present hospital admission, the patient was scheduled for laparoscopic cholecystectomy. Review of his old chart disclosed several anesthesia notes referring to resistance of the patient to succinylcholine. Preoperative analysis of the plasma cholinesterase activity, using benzoylcholine as a substrate, showed normal values. The plasma cholinesterase was 3.84 U/ml (normal values, 4.65-12.2 U/ml), dibucaine number was 87.3%, and fluoride number was 74.6%. Neurologic examination, nerve conduction studies, and electromyography did not detect any abnormalities.

The patient was premedicated with promethazine, 25 mg, meperidine, 75 mg, and atropine, 0.6 mg. Before induction of anesthesia and throughout surgery, neuromuscular transmission was monitored by Datex® (Helsinki, Finland) electromyography. The ulnar nerve was stimulated supramaximally (train-of-four) at the wrist every 20 s while displaying the resulting integrated electromyographic response of the adductor pollicis muscle. The patient was monitored continuously

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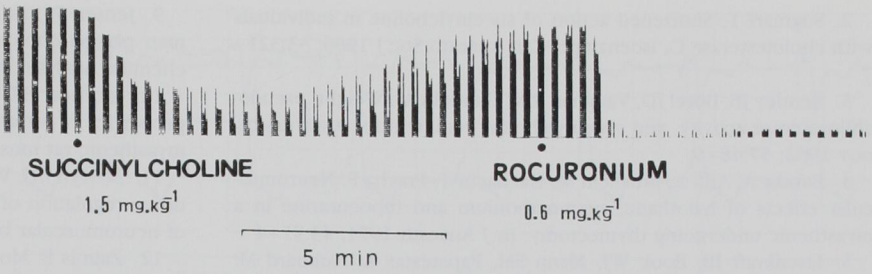

Fig. 1. Electromyographic response of the adductor pollicis muscle to train-of-four stimulation of the ulnar nerve. Succinylcholine, 1.5 mg/kg, resulted in about 50% neuromuscular block. After recovery, rocuronium, 0.6 mg/kg, resulted in rapid and complete neuromuscular block.

during anesthesia by electrocardiography (ECG), pulse oximetry, end-tidal capnography, esophageal temperature probe, and by noninvasive blood pressure monitor. After induction of anesthesia with thiopental, 5 mg/kg, succinylcholine, 1.5 mg/kg, was administered. Succinylcholine administration was not followed by muscle fasciculations and produced only a partial and transient neuromuscular block at the adductor pollicis (fig. 1). The mouth could not be opened, and laryngoscopy was impossible. However, the patient's lungs could be ventilated by a face mask. Rocuronium, 0.6 mg/kg, was administered intravenously and was rapidly followed by complete jaw relaxation and by complete neuromuscular block of the adductor pollicis muscle (fig. 1). Laryngoscopy and tracheal intubation could be easily achieved, and anesthesia was maintained with N_2O:O_2 supplemented by fentanyl. Recovery of T_4:T_1 ratio to 10% was observed after 40 min. Throughout this period, ECG was normal, $SPO_2 > 97\%$, end-tidal PCO_2 30–35 mmHg, and esophageal temperature was 36.5°C. Also, blood gases and electrolytes were within the normal values.

Discussion

Juvenile hyaline fibromatosis is a rare syndrome characterized by onset in early life and the presence of numerous dermal and subcutaneous nodules with unique histologic features. The patient may also have hypertrophic gingivae, osteolytic bone lesions, and stunted growth, with normal mental development.[7,8] Since childhood, our patient had JHF nodules scattered all over his body. During his last anesthetic and during his previous anesthetics, succinylcholine did not produce adequate muscle relaxation, despite adequate dose administration. The fact that this man had a history of inadequate muscle relaxation after succinylcholine, had normal plasma cholinesterase levels, had normal neurologic and neuromuscular functions, and had only 50% twitch depression of the adductor pollicis muscle suggests the diagnosis of the unusual phenomenon of succinylcholine resistance.

Resistance to succinylcholine may have a pharmacokinetic basis. The drug is rapidly hydrolyzed by the plasma cholinesterase.[1] Thus, an increase in the cholinesterase activity,[1–3] whether inherited (C_5 isoenzyme variant) or acquired, may decrease the response to succinylcholine. In obese patients, there is an increase of plasma cholinesterase that is matched by an increase in extracellular fluid, so that succinylcholine requirements may increase.[3] Our patient had a normal plasma cholinesterase activity and normal dibucaine and fluoride activity. However, a recent report by Jensen *et al.* has pointed out that many of the alleles of the gene for plasma cholinesterase cannot be detected by the usual imbibition studies of catalytic activity and require techniques of molecular biology to identify.[9] Resistance to succinylcholine also may be a result of pharmacodynamic causes as observed in experimental myasthenia[10] and in clinical myasthenia gravis.[4–6] In myasthenia, there is a decrease in the functional acetylcholine endplate receptors, with a consequent decrease in the response to the chemical transmitter acetylcholine an to other depolarizing agents such as succinylcholine.[6,11] The ED_{50} and ED_{95} in myasthenic patients are 2.0 and 2.6 times normal, respectively.[5] However, our patient did not show clinically or by nerve conduction studies and electromyography any neurologic or neuromuscular disorder that may "down-regulate"[11] the endplate receptors.

Resistance to succinylcholine has also been attributed to species variations.[12,13] In a previous report, we have shown in the isolated phrenic nerve–diaphragm of different species, that the rat preparation is extremely resistant to succinylcholine and sensitive to d-tubocurarine compared with the cat.[13] The resistance of our patient to succinylcholine and his relative sensitively to rocuronium may simulate the response in the rat species. However, previous reports of JHF have shown a normal response to muscle relaxants.[14,15]

In conclusion, the present report describes the occurrence of unusual succinylcholine resistance in a patient with JHF. The patient had normal plasma cholinesterase activity and normal neurologic and neuromuscular functions.

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Rate-adaptive Cardiac Pacing: Implications of Environmental Noise during Craniotomy

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IT has recently been estimated that approximately 1 million Americans have permanent implantable cardiac pacemakers. Of these, approximately 50-70% use rate-adaptive technology.[1,2] We report a case in which a patient undergoing a suboccipital craniotomy experienced vibration-mediated malfunction of a rate-adaptive cardiac pacemaker.

This Case Report is accompanied by a commentary from the manufacturer. See Correspondence, page 1261.

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Case Report

A 63-kg, 70-yr-old man with glossopharyngeal neuralgia presented for left suboccipital craniotomy with section of the left glossopharyngeal nerve and superior fibers of the vagus nerve. His medical history was remarkable for a third-degree atrioventricular (AV) heart block, which was treated with a permanent implantable dual-chamber rate-adaptive (DDDR) cardiac pacemaker (Trilogy™, Pacesetter, Sylmar, CA, model 2350). Eight months before surgery, the pacemaker was functioning normally with lower and upper rate limits of 60 and 120 beat/min, respectively. He denied having other cardiac diseases, cardiac symptoms with exercise, or problems related to his pacemaker. Preoperative medications included carbamazepine and baclofen (drugs being used for symptomatic therapy of his glossopharyngeal neuralgia) and atenolol. Pertinent preoperative laboratory values were within normal limits. Preoperative electrocardiogram (ECG) documented AV sequential or dual-chamber pacing at a rate of 77 beat/min. A chest radiograph demonstrated appropriate placement of pacemaker leads in the right atrium and ventricle, and the pulse generator was located in the left prepectoral region. A cardiology specialist was consulted and recommended placing the electrocautery grounding pad on the right thigh during surgery.

General anesthesia was induced with intravenous thiopental, fentanyl, and vecuronium. The trachea was intubated, and anesthesia was maintained with isoflurane, nitrous oxide, fentanyl, and vecuronium. A right radial arterial catheter and 18-gauge lumbar malleable needle (for cerebrospinal fluid drainage) were placed. Anesthesia proceeded uneventfully, the patient remained in the supine position throughout surgery, and an electrocautery grounding pad was placed