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Neuromuscular Blockade in a Patient with Active Dermatomyositis

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Dermatomyositis is a disease of unknown etiology characterized by proximal muscle weakness secondary to muscle destruction from a nonsuppurative inflammatory process. To date, few reports have described in detail the effects of muscle relaxants on neuromuscular blockade in patients with this disease entity. We present a case involving the use of muscle relaxants in a patient with active dermatomyositis undergoing cholecystectomy.

CASE REPORT

A 33-yr-old white woman with a 5-year history of dermatomyositis presented with symptoms of acute cholecystitis and was subsequently scheduled for cholecystectomy. Her medical history was remarkable for insulin-dependent diabetes mellitus and hyperlipidemia, both secondary to steroid therapy used to control acute exacerbations of dermatomyositis. Medications included cyclosporine, 40 mg prednisone daily for 3 weeks prior to surgery (5–65 mg daily for several years), and a divided daily dose of NPH insulin. Preoperative examination of the patient demonstrated generalized erythematosus that was more pronounced on her face, chest, and arms. Proximal muscle weakness was also evident because of her inability to rise from a squatting position and abduct her arms. Laboratory studies revealed an increased creatine kinase level of 382 μ/L (normal, 32–289 μ/L) and sedimentation rate of 53 mm/h (normal, 0–20 mm/h). Pulmonary function testing showed an $FEV_{1.0}$ of 3.30 L, FVC of 3.68 L, and $FEV_{1.0}$/FVC of 89%. Chest x-ray and electrocardiogram results were normal.

The patient was brought to the operating room where routine monitors including a five-lead electrocardiogram, finger pulse oximeter, and an automated blood pressure cuff were applied. A Puritan-Bennett NMT221 neurologic stimulator/monitor (Helsinki, Finland) *via* two electrode pads placed adjacent to the ulnar nerve proximal to the wrist delivered supramaximal, square-wave impulses of 0.1-ms duration with a 0.5-s interval between stimuli in a train-of-four (TOF) sequence (2 Hz). Train-of-four stimuli were repeated at intervals of 20 s. In addition, two electrode pads were placed over the thenar muscles to obtain electromyographic responses, which were displayed on a built-in thermal recorder. The amplitude of the first response in each train (T_1) and the ratio of the amplitude of the fourth response to the first (TOF ratio) were measured.

Baseline electromyographic recordings were obtained and anesthesia was induced with 0.035 mg sufentanil, 250 mg propofol, and oxygen. The patient's lungs were easily ventilated by mask and a cumulative dose of 140 mg succinylcholine was administered in increments of 20 mg, 40 mg, and 80 mg (fig. 1, *top*). Partial recovery of twitch height was allowed between incremental doses. The first dose of succinylcholine administered reduced twitch height to 28% of control in 75 s, the second dose reduced twitch height to 10% of control in 30 s, and the third dose completely abolished the twitch response almost immediately after injection. At this time, satisfactory muscle relaxation was obtained, the trachea was intubated, and the lungs were mechanically ventilated. The time from injection of the first incremental dose to complete recovery of twitch height after administration of subsequent incremental doses of succinylcholine was approximately 20 min.

Anesthesia was maintained with nitrous oxide, oxygen, and an intravenous infusion of sufentanil. The patient was then given 20 mg atracurium (ED_{95} dose, 0.25 mg/kg) and repeated TOF stimuli were monitored (fig. 1, *bottom*). Atracurium produced almost complete ablation of TOF in just over 2 min from the time of injection. The time from injection to 25% recovery of T_1 of the TOF ratio following complete recovery from atracurium was 33 min.

For the remainder of the case (total duration, 3 h), the patient was given an additional 35 mg atracurium intravenously in incremental doses of 5–10 mg (total dose, 55 mg) for muscle relaxation. No muscle relaxants were administered to the patient during the last 45 min of the procedure. At the completion of surgery, the TOF ratio was 70%. Residual neuromuscular blockade was reversed with 30 mg edrophonium and 1.2 mg atropine. No clinical weakness was observed at this time, and her trachea was extubated. The patient's postoperative course was uneventful, and she was discharged on the fourth postoperative day. A creatine kinase level obtained at that time was 367 μ/L.

DISCUSSION

Dermatomyositis is one of several diseases classified as an idiopathic inflammatory myositis histopathologically represented by perivascular nonsuppurative inflammatory infiltrates leading to muscle fiber degeneration.[1] Clinically, it is characterized by proximal muscle weakness and tenderness.[2] There is no evidence that the disease affects the neuromuscular junction, however. The disease is fairly uncommon, with only 5–10 new cases per million persons per year in the United States.

In this report, we present a patient with active dermatomyositis in whom the neuromuscular responses to succinylcholine and atracurium were evaluated and found to be within the normal range. Complete recovery from succinylcholine was slightly prolonged because of the dosing technique employed in this study. Extrapolation of the recovery response after the first dose revealed that complete recovery would have occurred in approximately 7 min.

Recovery time from atracurium-induced neuromuscular blockade was similar to the mean of 39.1 min determined in another study of the clinical effects of atra-

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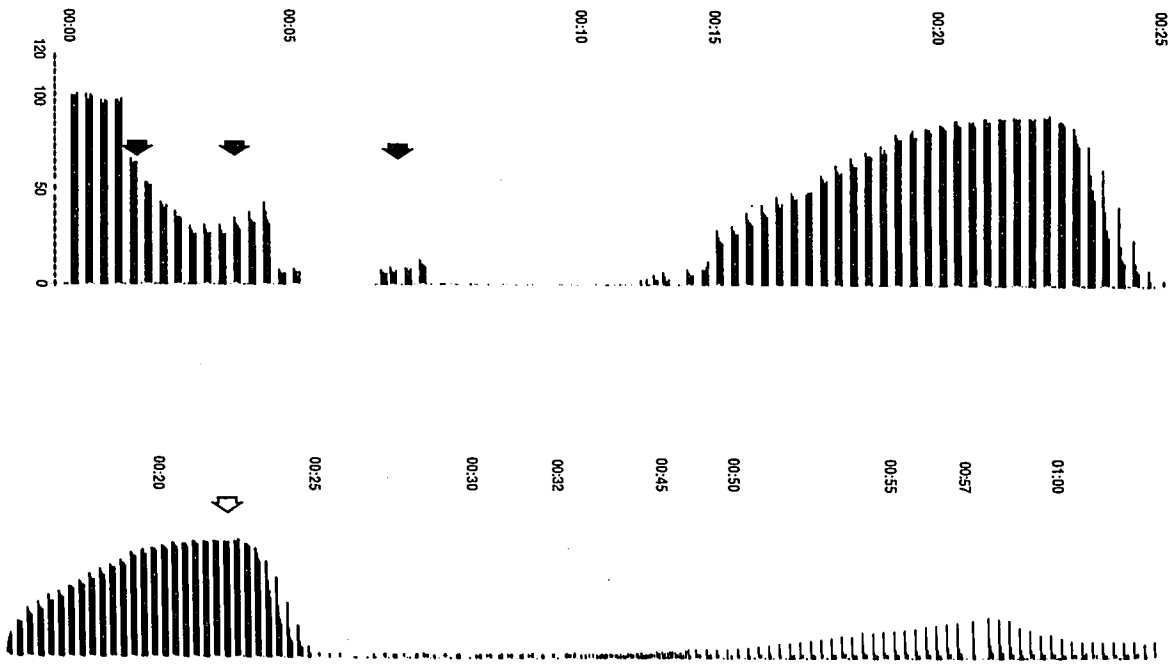

FIG. 1. Recording of train-of-four stimuli at 20-s intervals over approximately a 1-h period. *Top:* Control measurements made at extreme left of strip (twitch height 100%), followed by 25% decrement in twitch height after induction of anesthesia. Filled arrows represent administration of incremental doses of 20, 40, and 80 mg succinylcholine. *Bottom:* Continuation of top panel with recovery from succinylcholine at 20 min followed by administration of 20 mg atracurium (open arrow).

curium 0.4 mg/kg during nitrous oxide, oxygen, and fentanyl anesthesia.[3] The response time determined in this case also would fall within the upper limits of the normal range (16–46 min) for 95% recovery from atracurium following succinylcholine-induced neuromuscular blockade reported in a previous investigation using a similar anesthetic technique and smaller dose of atracurium.[4]

Several investigators have reported a prolonged response to neuromuscular blocking agents in patients with active myositis. Churchill-Davidson and Richardson were concerned about the use of muscle relaxants in patients with dermatomyositis because of an association with carcinoma of the lung and subjective findings of improvement in peripheral weakness following the administration of anticholinesterase drugs.[5] Flusche *et al.*, while monitoring neuromuscular function in a similar manner as we did, observed significantly delayed recovery from a single dose of vecuronium in an elderly patient with multiple medical problems including polymyositis.[6]

The results of this case differ from those of Flusche *et al.* for several reasons. They studied an elderly patient with polymyositis and multiple chronic medical problems including long-standing rheumatoid arthritis, peripheral vascular disease, cerebrovascular disease, hypertension, renal insufficiency, and urinary tract infections for which she was receiving aminoglycoside antibiotics. In addition, a single large dose of vecuronium and a potent volatile agent were used in her anesthetic management. Several of these factors independent of polymyositis may have prolonged vecuronium-induced neuromuscular blockade in that patient.

Prior to anesthesia, our patient was receiving concomitant medications including prednisone, cyclosporine, and insulin that could have impacted neuromuscular function, such that the findings of this report were a result of several contradictory effects. Patients with adrenal cortical insufficiency appear to have a defect in neuromuscular transmission that is reversed by the administration of corticosteroids.[7] In addition, cortisol or adrenocorticotrophic hormone can improve neuromuscular function in patients with myasthenia gravis. However, acute administration of corticosteroids to normal patients intraoperatively had no effect on nondepolarizing neuromuscular blockade.[8] Chronic steroid therapy may produce proximal muscle weakness and histologic evidence of myopathy that is distinct from its effects on the neuromuscular junction.[9] Since our patient was receiving chronic corticosteroid therapy, it is possible that a defect in neuromuscular function due to dermatomyositis could have been obscured.

Patients receiving cyclosporine appear to be sensitive to the effects of vecuronium-induced neuromuscular blockade.[10] A direct effect of cyclosporine on the neuromuscular junction has not been demonstrated to our knowledge. However, chronic therapy causes a deterioration in renal function that can increase the duration of nondepolarizing neuromuscular blockade.

Similarly, insulin therapy does not directly affect neuromuscular function clinically, but chronic complications of diabetes mellitus such as renal insufficiency could result in prolonged effects from nondepolarizing muscle relaxants. The patient in this case had normal renal function. In addition, she received atracurium, which is less dependent upon the renal route of elimination than other nondepolarizing neuromuscular blocking agents.

Theoretically, one concern involving the administration of succinylcholine in a patient with a muscle disorder is the excessive release of potassium secondary to depolarization of abnormal cells. Although this problem has not been documented in patients with dermatomyositis, we attempted to measure any changes in serum potassium following the administration of succinylcholine in our patient. Unfortunately, the blood sample was lost. However, no electrocardiogram changes or clinical signs of hyperkalemia were evident during the care of this patient.

In summary, the results of this case study demonstrate a normal onset, peak effect, and recovery from the neuromuscular blocking actions of succinylcholine and atracurium in a patient with active dermatomyositis. However, interactions between concomitant medications and muscle relaxants used intraoperatively may have influenced the results of this report through effects on neuromuscular transmission.

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The Effect of Positive End-expiratory Pressure on Right-to-left Shunting at the Atrial Level as Documented by Transesophageal Echocardiography

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The combination of a patent foramen ovale and positive end-expiratory pressure (PEEP) has been shown to be associated with an increased incidence of paradoxical embolism, significant shunting at the atrial level, and refractory hypoxemia.[1] We report a case of intraoperative development of right ventricular failure secondary to right ventricular infarction during coronary artery bypass graft surgery. After the development of severe refractory hypoxemia, bedside transesophageal echocardiography (TEE) was performed and confirmed the presence of a right-to-left shunt at the atrial level. This case report demonstrates the deleterious effect of increasing PEEP on the shunt across a patent foramen ovale as documented by TEE.

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Case Report

A 74-yr-old man with a history of angina pectoris of recent onset was scheduled for coronary artery bypass graft surgery. He had a long-standing history of hypertension and non–insulin-dependent diabetes mellitus. His blood pressure was 148/86 mmHg with a regular heart