Pseudocholinesterase abnormalities are a genetic cause of aberrant metabolism of the depolarizing muscle relaxant succinylcholine. This article examines a case where succinylcholine was chosen to facilitate intubation due to its ultra short duration and the request of the surgeon to monitor motor evoked potentials. Following succinylcholine administration the neurophysiologist was unable to obtain motor evoked potentials. This case study highlights the intraoperative and postoperative management of an elderly patient with an unknown pseudocholinesterase deficiency.

Keywords: Anesthesia, dibucaine number, motor evoked potentials, pseudocholinesterase deficiency.
The neurophysiologist attempted to establish the patient’s baseline MEPs. At 0901 the neurophysiologist made the surgeon, CRNA, and student registered nurse anesthetist aware that motor evoked potentials were unable to be elicited. After confirming with the anesthesia team that succinylcholine had been administered the neurophysiologist began to troubleshoot the monitoring equipment.

The student registered nurse anesthetist checked a train of four (TOF) response using the nerve stimulator to evaluate the patient’s recovery from neuromuscular blockade. The lack of response led to a presumptive diagnosis of pseudocholinesterase deficiency. The battery and electrodes of the nerve stimulator were checked and confirmed to be in working order. The surgeon and anesthesiologist were made aware of the lack of response. The surgeon stated the case could be safely continued without the motor evoked potentials and requested that MEPs periodically be followed to detect the return of muscle responsiveness.

Early in the case the patient experienced hypotension and a phenylephrine infusion was initiated and titrated to maintain a mean arterial pressure of 70 or greater. Otherwise, the case remained uneventful. Approximately 3 hours into the case, at 1202, the neurophysiologist notified the team that the MEPs had returned to the patients’ presurgery baseline. The student registered nurse anesthetist rechecked the TOF response and noted 4 strong equal twitches. MEPs were then monitored continuously throughout the remainder of the surgery.

The case was completed at 13:04. Five hours and 10 minutes had elapsed since the succinylcholine had been administered. After discussion among the anesthesia team, propofol and remifentanil were discontinued in an attempt to maintain a mean arterial pressure of 70 or greater.

Discussion
Prolongation of succinylcholine can be caused by either a decreased quantity or quality of pseudocholinesterase. Diminished quantities may be seen in the presence of malignancies, pregnancy, liver disease, collagen vascular disease, malnutrition, and hypothyroidism. In this case, the dibucaine inhibition test was drawn and sent for analysis to determine if the patient had an atypical variant of PChE. The normal result of the dibucaine inhibition test is 80. This means that 80% of the PChE activity was inhibited by the local anesthetic dibucaine. These individuals are labeled homozygous normal and would be briefly paralyzed by succinylcholine. Those with a dibucaine number of 20 would be homozgyous atypical and can be expected to have a marked response to succinylcholine with paralysis typically exceeding 1 hour. A dibucaine inhibition test result of 60 would be defined as heterozygous and generally only produce a slight prolongation of succinylcholine. Postoperatively, it was noted that the patient’s dibucaine inhibition test result was 27. This indicates an atypical PChE variant with the genetic label of homozygote atypical. The incidence of a patient homozygous for pseudocholinesterase mutations is one in 2,500 patients.

Studies have shown that patients with pseudocholinesterase deficiency have a normal response to remifentanil, leading the author to believe it is unlikely that the remifentanil contributed to the prolonged recovery of the patient. Remifentanil is a synthetic opioid with swift onset and short duration. It allows predictable titration of anesthesia with rapid recovery of consciousness and respiration independent of the duration of infusion. The context-sensitive half-life is 3-4 minutes. Remifentanil is metabolized by nonspecific esterases in tissues and blood and is not mediated by pseudocholinesterase.

An indication that this patient may have had a pseudocholinesterase deficiency came from the preoperative interview when the patient stated that he was unable to move during a prior anesthetic. Past surgical records for the aortic aneurysm repair did include the administration of succinylcholine followed by rocuronium. TOF response had not been charted between the two, however 6 hours later a four-twitch response to TOF was documented prior to reversal. The patient was taken to PACU intubated and the reason indicated on the anesthesia discharge summary was pulmonary edema. The lobectomy had occurred after the AAA repair and succinylcholine had not been administered. This explains why the patient had indicated he could not recall having an anesthetic complication with his most recent surgery.

The author acknowledges that a baseline TOF should have been elicited prior to the administration of succinylcholine. An early discovery could have alerted the surgeon to the inability to elicit intraoperative MEPs prior to skin incision. Multiple case reports describe the importance of monitoring neuromuscular blockade when administering succinylcholine.
When faced with a patient experiencing a prolonged duration of paralysis, in addition to safety, a primary goal is comfort of the patient. The patient was found to be awake and able to blink eyes in response to questioning. The patient was reassured, sedated, and ventilated until it was decided by the anesthesiologist that the patient satisfactorily met the extubation criteria. The patient was educated regarding his atypical enzyme and given a letter regarding his pseudocholinesterase deficiency for future anesthesia experiences.

Conclusion
In summary, this was a unique case of unanticipated prolonged paralysis observed in an elderly patient during a surgery where motor evoked potentials were being monitored. The inability to elicit a TOF response led the student registered nurse anesthetist to an early presumptive diagnosis of pseudocholinesterase deficiency. In this patient population education regarding the cause of the paralysis is important to decrease anxiety and avoid the future use of depolarizing muscle relaxants by other healthcare providers.

REFERENCES

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