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Always Check the Muscle Twitch: Residual Neuromuscular Block After Removal of a Gastric Balloon

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The Case

A 38-year-old female patient with a height of 65 inches (165 cm) and weight of 153 kg (body mass index, 56.2 kg/m^2) required removal of a gastric balloon under general anesthesia. She required a relatively large dose of rocuronium for endotracheal intubation, and she was given intravenous sugammadex (200 mg) at the end of the procedure to reverse the neuromuscular block. A quantitative neuromuscular block monitor was not used but reliance was placed on clinical signs.

Upon awakening from general anesthesia, the patient was extubated and taken to the post-anesthesia care unit (PACU). However, shortly after arrival in the PACU, she couldn't move or open her eyes and became jittery with low oxygen saturation. Quantitative blockade monitoring revealed a "train of four" (TOF) ratio less than 70%, so she was given another 200 mg of intravenous sugammadex with return of normal motor function.

The Commentary

By Christian Bohringer, MBBS, and Sharon Ashley, MD

Clinical Background

The incidence of obesity is increasing in many countries as part of a global obesity pandemic.[1,2] Class 3 obesity, with body mass index (BMI) of 40 or higher, poses special risks to patients undergoing anesthesia. Both mask ventilation and endotracheal intubation may be difficult. Patients with class 3 obesity are also at significant risk of postoperative apnea and respiratory arrest.[3,4]

Persistent neuromuscular blockade in the recovery room from inadequate reversal of neuromuscular blocking agents (NMBAs) like rocuronium is an ever-present danger whenever they are used as part of the anesthetic.[5] Persistent paralysis is particularly dangerous for severely obese patients because they must perform increased work of breathing at baseline to compensate for increased upper airway resistance. The American Society of Anesthesiology (ASA) has recently recommended using a nerve stimulator for assessing adequate recovery from neuromuscular blockade. Quantitative monitors that can measure the TOF ratio more accurately than is possible with visual and tactile evaluation of thumb movement have recently become available and the ASA advocates their use.[6,7]

The Case

This 38-year-old woman has class 3 or severe obesity, which is defined by a BMI greater than 40 kg/m^2. This patient's BMI of 56.2 kg/m^2 is associated with substantially higher perioperative risk when compared to patients with normal BMI.[8]

The patient in this case presumably received an intragastric balloon as a temporary and minimally invasive therapy for weight loss. The balloon is filled with water and acts as a space occupying device that reduces stomach capacity, resulting in decreased hunger and food intake.[9] A recent trial demonstrated a 14% total body weight loss over a 4-month study period with this type of device.[10]

A large dose of rocuronium was given to this patient at the beginning of this short procedure to remove the gastric balloon. This large dose ensured adequate paralysis to allow for uncomplicated endotracheal intubation. Insufficient depth of neuromuscular blockade during intubation may lead to gagging and closing of the vocal cords, resulting in failed intubation and/or failed ventilation.

In this case, recovery from the neuromuscular blockade was unfortunately not monitored with a nerve stimulator, as is now recommended by the ASA.[7] The anesthesiologist relied instead on assessing neuromuscular recovery clinically by looking at the patient's chest and body movements. The patient received only 200 mg sugammadex to reverse the large dose of rocuronium given at the beginning of this short procedure. The standard reversal dose is 2 mg/kg, so this 153 kg patient should have received 306 mg of sugammadex.

This patient was too weak to take deep breaths in the recovery room. Hypoxemia presumably occurred because the persistent neuromuscular block induced atelectasis at the base of her lungs. She had difficulty opening her eyes, which is a typical feature of persistent neuromuscular blockade because the extraocular muscles are more sensitive to the effects of neuromuscular blocking agents (NMBAs) than most other muscles in the body. When neuromuscular function was formally assessed with a quantitative nerve stimulator, the TOF ratio was only 0.7. This means that there was a fade of the contraction of the opponens pollicis muscle with each subsequent stimulus over a series of four electrical impulses applied to the ulnar nerve. An additional dose of sugammadex fully reversed her residual weakness and eliminated the need for ventilatory assistance or reintubation.

Approaches to Improving Patient Safety

Educate patients about the dangers of super obesity

The prevalence of obesity is increasing worldwide; anesthesia care providers must understand the adverse cardiopulmonary consequences of class 3 obesity. They have the opportunity to educate patients during the perioperative period about the benefits of weight loss before any future procedures. Anesthesiologists are well-positioned to help identify sleep apnea because they essentially perform a drug-induced sleep study whenever they administer anxiolytic medications before an operation. If patients develop airway obstruction before losing consciousness, they should be advised to seek evaluation by a sleep apnea specialist who can prescribe effective treatment for this condition.[4]

Avoid or carefully titrate the dose of NMBAs

NMBAs can improve operating conditions for the surgeon by completely abolishing any patient movement under anesthesia. These drugs are necessary for many operations; however, their use always carries the potential risk of persistent neuromuscular blockade in the recovery room. Therefore, if the procedure can be performed without NMBAs, these drugs should be avoided.

Steering clear of NMBAs also significantly reduces the incidence of intraoperative anaphylaxis, since these drugs are among the most frequent causes of this clinical syndrome.[11] When NMBAs are indispensable, the dose should be titrated carefully to the clinical need. For laryngeal and mandibular surgery, dense neuromuscular blockade has been shown to improve operating conditions for the surgeon.[12,13] If deep paralysis is requested for this type of procedure, additional sugammadex must be given at the end so that the patient will not be weak in the recovery room.

Assess the depth of neuromuscular blockade with a nerve stimulator

Different patients metabolize rocuronium at different rates. Elimination is significantly increased in patients taking anticonvulsant medications and significantly reduced in patients with impaired renal and hepatic function.[14,15] It is therefore very important to assess the depth of paralysis with a nerve stimulator whenever NMBAs are used as part of the anesthetic regimen. It is better to evaluate the contraction of the thumb than the facial muscles because clinical trials have shown that facial muscle monitoring underestimates the depth of the neuromuscular block.[7]

Evaluation with a nerve stimulator is especially important before extubation. A patient with persistent partial paralysis can usually breathe very well while the endotracheal tube is splinting the larynx open because the diaphragm is very resistant to NMBAs, relative to the laryngeal muscles. A patient with persistent neuromuscular block therefore often develops upper airway obstruction after the endotracheal tube has been removed because the laryngeal muscles are still too weak to keep the airway patent.

Use quantitative neuromuscular monitoring whenever available

Quantitative assessment of the degree of paralysis by either electromyography or acceleromyography can measure the TOF ratio accurately. This is not possible by simply looking at the thumb or feeling its movements.[7] A fading TOF ratio can be easily missed if a quantitative assessment is not performed.

Reduce the dose of rocuronium in patients with renal and hepatic impairment

The amount of sugammadex needed at the end of the procedure depends on the total amount as well as the timing of the rocuronium doses given, based on the duration of the procedure. Older patients and patients with impaired renal and hepatic function are at particular risk for persistent neuromuscular blockade in the (PACU).[16] Anesthesia care providers may not always appreciate how significantly the impairment of renal and hepatic function can affect elimination of NMBAs. Cis-atracurium does not rely on renal and hepatic function for elimination, but it has the significant disadvantage that deep levels of neuromuscular block cannot be reversed because it does not bind to sugammadex.[17]

Use sugammadex instead of neostigmine for reversal of rocuronium

Sugammadex has significant advantages over neostigmine for reversing rocuronium.[18] Even very deep neuromuscular blockade can be completely reversed by increasing the dose of sugammadex. By contrast, neostigmine has a ceiling effect; when the dose is increased above 50 mcg/kg, a cholinergic crisis may ensue at the neuromuscular junction that potentiates rather than reverses the neuromuscular block. Neostigmine also produces cholinergic side effects like salivation, nausea, vomiting, bowel cramps, bronchospasm, and bradycardia. An anticholinergic agent like glycopyrrolate or atropine therefore should be administered together with the neostigmine dose to lessen these side effects. Recovery of neuromuscular function is also significantly faster with sugammadex versus neostigmine.[19] Ten randomized trials reported lower rates of residual paralysis with sugammadex than with neostigmine. The ASA taskforce on neuromuscular blockade therefore recommended it over neostigmine in their latest report.[7]

Another advantage of sugammadex is that it can be used to rapidly ameliorate anaphylactic shock caused by rocuronium. Sugammadex can remove rocuronium very quickly from the blood stream by binding to it and forming an inactive complex that does not stimulate the immune system.[11] Many case reports describe very significant attenuation of the allergic response after rapid high dose (16 mg/kg) reversal with sugammadex.[20,21]

The dose of sugammadex should be based on total body weight

The dose of sugammadex used to reverse the neuromuscular block should be based on actual body weight rather than ideal body weight.[22] A standard 2 mg/kg dose would have been 306 mg for this 153 kg patient. The patient in this case therefore received an insufficient dose of the reversal drug, which might have been identified through a clinical decision support tool in the electronic health record.

Recognize the signs of persistent neuromuscular block

The clinical signs of persistent neuromuscular block are subtle, and anesthesia care providers without extensive experience can easily miss them. These signs include tachypnea, low tidal volume, and lid lag when the patient attempts to open their eyes. The absence of sustained antigravity power produces the classical “fish out of water” syndrome, which is characterized by flailing about with the upper limbs. Because the frontalis muscle is very resistant to the effect of NMBAs, patients still manage to frown even when many of their other muscles remain deeply paralyzed. On the other hand, extraocular muscles are very sensitive to NMBAs, leading the eyes to diverge in different directions causing diplopia.[5] Diplopia can contribute to nausea and vomiting in the PACU.

Persistent or recurrent neuromuscular blockade should always be reversed emergently as soon as it is discovered to provide patient comfort and avoid reintubation. When patients feel weak and struggle to breathe in the PACU, they often worry that they had a stroke while they were under anesthesia. Verbal reassurance that the weakness will only be transient is necessary to alleviate the patient's fears. An awake patient with persistent neuromuscular block should not be subjected to electric shocks from the nerve stimulator because this can be very painful and the patient cannot take evasive action because they are still paralyzed. The only hint for the clinician that the patient is aware but unable to move may be a sudden rise in heart rate and blood pressure with the tetanic stimulus. In a complex clinical scenario where the diagnosis is uncertain, it is prudent to administer some propofol prior to applying the nerve stimulator so that the patient does not remember the electric shocks. This is especially important when the patient has dilated pupils because this clinical sign may indicate that the patient is awake and aware underneath the neuromuscular blockade.

The nerve stimulator should not be relied on as the only source of information to determine if it is safe to extubate. The patient may be weak from electrolyte abnormalities like hypokalemia or hypermagnesemia. Residual effects of volatile anesthetic agents may also contribute to muscle weakness. Weakness from these causes may be present despite an adequate TOF ratio. The discomfort from nerve stimulation limits the ability to repeatedly reevaluate awake patients. Anesthesia care providers therefore need to be able to clinically recognize residual or recurring weakness in the PACU by assessing the patient's breathing pattern and body movements.

Continue to monitor the patient for clinical signs of persistent neuromuscular block

The patient should be continuously evaluated for persistence or recurrence of neuromuscular weakness in the recovery room or PACU. Recurrence of neuromuscular block has been reported after both neostigmine and sugammadex.[23-25] The patient should not be discharged from the PACU if there is any concern for persistent neuromuscular weakness.

When anesthesia staff fail to identify the NMBA as the cause of residual neuromuscular weakness, they may request a neurology consult. Given that neurologists never see residual neuromuscular blockade in their outpatient practices, they may misdiagnose a transient intranuclear ophthalmoplegia.[25] Anesthesia care providers should be comfortable with the diagnosis of persistent neuromuscular block in the PACU, but a neurology consult is indicated when the patient's clinical course suggests an exaggerated response to NMBAs that is not readily explained by electrolyte disturbance. Prolonged paralysis in this setting is frequently due to subclinical myasthenia gravis, especially after eyelid surgery for ptosis if the ophthalmologist failed to recognize myasthenia as the cause of the ptosis. Administering NMBAs is a pharmacological stress test for the neuromuscular junction; myasthenic patients frequently have a history of unexplained reintubation after surgery before their definitive diagnosis.

Be prepared to administer additional doses of sugammadex

Residual or recurring paralysis should be reversed with further doses of sugammadex to maximize patient comfort and eliminate nausea-inducing diplopia, even if it is not severe enough to require reintubation.

Summary

This patient suffered an avoidable complication but was able to avoid reintubation or hypoxemic cardiac arrest as a result of rapid intervention by the anesthesia care team. Assessing the depth of neuromuscular blockade with a nerve stimulator would have clearly demonstrated the need for a larger dose of sugammadex prior to extubation. It is the responsibility of the anesthesiologist to ensure that the neuromuscular blockade has been fully reversed before extubation and transport of the patient to the recovery room. Monitoring the depth of neuromuscular blockade with a nerve stimulator is essential when using NMBAs as part of the anesthetic combination.

Take-Home Points

- Patients with class 3 obesity are at increased risk of postoperative respiratory arrest.
- Persistent neuromuscular blockade in the recovery room is a known risk, especially in patients with impaired elimination of NMBAs, or after multiple or large NMBA doses.
- The depth of neuromuscular block should be assessed quantitatively with a nerve stimulator before extubation.
- Quantitative monitors can now measure the TOF ratio precisely and are recommended by the American Society of Anesthesiologists.
- The dose of sugammadex should be based on total rather than ideal body weight.
- When large doses of rocuronium are used, the dose of sugammadex also should be increased.
- The patient should be carefully evaluated for the clinical signs of persistent neuromuscular blockade prior to extubation as well as during the entire recovery period.
- Look for and correct electrolyte disturbances that may exacerbate muscle weakness.
- When there is an exaggerated response to NMBAs, the patient may have undiagnosed myasthenia gravis.
- Clinicians should have a low threshold for administering additional doses of sugammadex in the operating or the PACU if the patient still appears weak.

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