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Anesthetic Considerations for the Use of Acceleromyography for Neuromuscular Blockade

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ANESTHETIC CONSIDERATIONS FOR THE USE OF ACCELEROMYOGRAPHY FOR NEUROMUSCULAR BLOCKADE

by

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Abstract

Title: Anesthetic Considerations for the Use of Acceleromyography for Neuromuscular Blockade

Background: Acceleromyography is a quantitative, objective neuromuscular monitor utilized to monitor neuromuscular blockade during the perioperative period. Failure to adequately monitor neuromuscular blockade can result in residual paralysis. Although the prevalence of monitoring neuromuscular blockade remains low, monitoring neuromuscular blockade after a patient has received a neuromuscular blocking agent is a standard of care

Purpose: The purpose of this independent project is to review the current literature and practice recommendations regarding clinical management using acceleromyography neuromuscular monitoring in the perioperative setting.

Process: A comprehensive review of the literature was performed utilizing CINAHL and PubMed databases from the University of North Dakota Harley French Library. The findings were analyzed to determine significance for this project.

Results: Motor neuron and muscle cell communication takes place at the neuromuscular junction. The post-synaptic junction at the neuromuscular junction is the site where both depolarizing and non-depolarizing neuromuscular blocking agents act. Anticholinesterase medications antagonize the effects of non-depolarizing neuromuscular blocking agents by competitively binding to the post-synaptic receptor at the neuromuscular junction. The acceleromyography neuromuscular monitor is an objective, quantitative monitor utilized to assess blockade in patients who received neuromuscular blocking medications during surgery. Calibrating the acceleromyography monitor is recommended in scientific research however, it is still undecided if it is warranted in the clinical setting. Monitoring at the adductor pollices muscle

is the most accurate monitoring site. The oribularis oculi muscle site is not recommended when using an acceleromyography monitor. The flexor hallucis brevis muscle may be considered as an alternative site. The primary advantage of using an acceleromyography monitor over qualitative neuromuscular monitors is its ability to provide a train of four ratio. A train of four ratio of greater than or equal to 0.9 decreases incidence of residual paralysis.

Implications: Incidence of residual paralysis may be reduced with the implementation of acceleromyography neuromuscular monitoring.

Keywords: acceleromyography, train of four monitoring, quantitative train of four monitoring, residual paralysis, residual neuromuscular blockade

Anesthetic Considerations for the Use of Acceleromyography for Neuromuscular Blockade

Acceleromyography neuromuscular monitoring was introduced in the late 1980s (Claudius & Viby-Mogensen, 2008). It is a quantitative monitor utilized to assess the level of neuromuscular blockade in patients who received neuromuscular blocking medications during surgery. Monitoring the depth of neuromuscular blockade can also be evaluated with clinical tests such as head lift and grip strength. However, these tests have been found to be subjective, unreliable and require patient cooperation (Claudius & Viby-Mogensen, 2008).

Prevalence of monitoring using qualitative, subjective nerve stimulators is less than 40% (Brull & Kopman, 2017). Prevalence of monitoring using quantitative, objective neuromuscular monitoring is even lower at less than 17% (Brull & Kopman, 2017). Accurately monitoring neuromuscular blockade can affect the patient's outcome postoperatively. A case report of a young, healthy woman undergoing a parietal craniotomy utilizing a TOF-WATCH™ acceleromyography monitor will be discussed. A review of the literature including pathophysiology, pharmacology, train of four monitoring, acceleromyography monitor calibration, acceleromyography monitoring sites, and residual paralysis will be presented.

Purpose

The purpose of this independent project is to provide anesthesia practitioners with evidence-based research regarding the use of acceleromyography neuromuscular monitoring. Assessing neuromuscular blockade when a neuromuscular blocking agent is utilized is a standard of care for anesthesia practitioners. Therefore, having the ability to know how many receptors continue to be occupied by the neuromuscular medication is essential for the provider to be able to safely extubate the patient.

Case Report

A 32-year-old, 150 cm, 55 kg, female presented for a left parietal craniotomy for the treatment of a meningioma brain tumor. Her medical history included meningioma, latent tuberculosis of the lungs, and chronic left sided headaches. Surgical history included a cerebral angiogram the day prior to the proposed surgery. She had no history of anesthetic complications. Current medication included Isoniazid.

The patient was considered an American Society of Anesthesiologists physical status level three. Airway evaluation revealed a Mallampati score of 2 with a thyromental distance of 3 fingerbreadths. Bilateral breath sounds were clear to auscultation. She had a regular rate and rhythm, no murmurs, gallops or rubs. Neurologically, she presented with chronic left sided headaches. Preoperative vital signs were: blood pressure 127/92 mmHg, heart rate 103/min, respirations 20/min, room air oxygen saturation (Sp02) 96%, and temperature 97.6° Fahrenheit.

The patient was given midazolam 1 milligram (mg) intravenously (IV) upon arrival to the operating room (OR). She was assisted onto the operating room table per OR staff. Standard non-invasive monitors were applied simultaneously which included: finger pulse oximetry, five-lead electrocardiogram (EKG), and a blood pressure cuff.

The patient's vital signs before induction were: 147/93 mmHg, heart rate 103/min, respirations 21/min, 99% SpO2, 25 mmHg end-tidal carbon dioxide (ETCO2). Inhalation and intravenous induction ensued with oxygen at 10L per minute and sevoflurane end-tidal concentrations of 1.25%. Fentanyl 100 micrograms (mcg) IV, lidocaine 40 mg IV, and propofol 110 mg IV were administered for induction. The ability to ventilate was confirmed with chest rise, ETCO2 and mask fogging. Subsequently, rocuronium 30 mg IV was administered. A 7.0 millimeter cuffed endotracheal tube (ETT) was placed by direct laryngoscopy utilizing a MAC

three blade with a grade one Cormack and Lehane view. After intubation, bilateral breath sounds were auscultated, ETCO2 monitoring was present, and the ETT was secured at 21 centimeters (cm) at the lip. The patient was placed on volume control mode with a respiratory rate of 12/min, and a tidal volume of 460 mL. Sevoflurane was turned off, and desflurane was initiated with end-tidal concentrations of 5% throughout the procedure. Inspired oxygen was titrated to 60% and air 40% was administered to keep flows at 1 liter (L) per minute. A nasopharyngeal probe was inserted for temperature monitoring with temperatures of 36.7° C throughout the procedure. After induction, two 18-gauge IV's were placed in addition to the preexisting 20-gauge IV. A dexmedetomidine infusion was initiated at 0.4mcg/kg/hr intravenously for multimodal amnesia. A remifentanil infusion was initiated at 0.15 mcg/kg/min intravenously for multimodal analgesia. After several attempts by multiple anesthesia practitioners, a 20-gauge arterial line was successfully placed in the left radial artery to monitor precise blood pressures. The patient's blood pressure decreased to 61/33 mmHg with a mean arterial pressure (MAP) of 44 mmHg. A phenylephrine 200 mcg bolus was given IV. A phenylephrine drip was started intravenously at 5 mL/hr to maintain systolic blood pressures greater than 100 mmHg to ensure adequate perfusion to her brain. For nausea prophylaxis, ondansetron 4 mg IV and dexamethasone 10 mg IV were administered. Acceleromyography, to monitor neuromuscular blockade, was applied without calibration with a standard current of 50 mA to the right thumb and electrodes on the ulnar nerve to elicit contraction of the adductor pollices muscle. The train of four (TOF), which was used to assess neuromuscular blockade showed one out of four (1/4) twitches, thirty-one minutes after the last dose of rocuronium. Two grams (g) of cefazolin was administered IV for a preoperative antibiotic.

The patient was positioned, per OR staff, supine with her arms tucked at her sides ensuring the ability of the right thumb to freely move. The OR table was turned 180 degrees away from the anesthesia provider. A lower Bair Hugger was placed on the patient with a temperature setting of 43° C. Levetiracetam 1000 mg was given IV for seizure prophylaxis. The dexmedetomidine infusion was decreased to 0.3 mcg/kg/hr due to the medication's hypotensive side effects and the patient's corresponding hypotension. The patient had 1/4 twitches on the acceleromyography monitor forty-five minutes after the last dose of rocuronium. The phenylephrine drip was discontinued, twenty-nine minutes after initiation, as the blood pressures were 123/80 mmHg with a MAP of 100 mmHg. Fifty-one minutes after the last dose of rocuronium, a TOF was checked revealing 2/4 twitches. Rocuronium 10 mg IV was administered to maintain a TOF less than 2/4 twitches. Mannitol 30 mg IV was administered over twenty-five minutes to decrease intracerebral volume. TOF was again checked thirty-two minutes after the last dose of rocuronium, revealing 2/4 twitches. Rocuronium 10 mg IV was administered. Ofirmev 1 g IV was administered for multimodal analgesia. Two and a half hours after the case began, an arterial blood gas and labs were drawn revealing: Ph 7.38, PCO2 33 mmHg, PO2 157 mmHg, O2 99%, base excess -6, HCO3 19 mmol/L, HBG 11.6 g/dL, HCT 34 g/dL, glucose 162 mg/dL, NA 138 mmol/L, K 4.3 mmol/L, Ionized Ca 1.03 mmol/L. Calcium gluconate 1 g IV was ordered and administered to increase the patient's intravascular calcium as her Ionized Calcium was 1.03 mmol/L. Albumin 5% (250mL) IV was administered for fluid replacement. TOF was again checked thirty-eight minutes after the last dose of rocuronium, revealing 2/4 twitches. Rocuronium 10 mg IV was administered. A TOF was again checked thirty-eight minutes after the last dose of rocuronium, revealing 2/4 twitches. An additional dose of rocuronium 10 mg IV was administered to maintain paralysis. At the same time, albumin 5% IV was given for volume

replacement. Twenty-eight minutes after the last dose of rocuronium, a TOF was checked revealing 1/4 twitches. The surgeon began closing the wound; paralysis was no longer warranted, and the rocuronium boluses were discontinued. A TOF was checked 40 minutes after the last dose of rocuronium, revealing 4/4 twitches. Ondansetron 4 mg IV and cefazolin 2 g IV were administered as it had been four hours since they were administered. Four and a half hours into the case a second set of arterial blood gases and labs were drawn revealing: Ph 7.38, PCO2 33 mmHg, PO2 167 mmHg, O2 99%, base excess -6, HBG 9.9 g/dL, HCT 29 g/dL, glucose 175 mg/dL, Na 139 mmol/L, K 4.4 mmol/L, Ionized Ca 1.33 mmol/L. The dexmedetomidine infusion was discontinued. The phenylephrine infusion was restarted at 5 mL/hr IV for seven minutes due to systolic blood pressures being less than 100 mmHg. The remifentanil infusion was decreased to 0.05 mcg/kg/min. Glycopyrolate 0.2 mg IV and neostigmine 2.5 mg IV were administered to antagonize any remaining neuromuscular blockade. Lidocaine 50 mg IV was administered to decrease coughing. The patient met extubation criteria as evidenced by regular spontaneous respirations of 8/min, tidal volume consistently greater than 300 mL and bilateral equal hand grip strength.

During the five-hour thirty minute surgical case, the patient received a total of 3,000 mL of normal saline, 475 mL lactated ringers and 500 mL 5% albumin. Estimated blood loss was 850 mL and urine output was 2,635 mL. The patient was discharged home on postoperative day thirteen with home care.

Literature Search

A review of the evidence was pursued with an online search using CINAHL, PubMed, and the American Association of Nurse Anesthetists (AANA) website. CINAHL and PubMed databases were accessed utilizing the University of North Dakota Harley French Library. All

journal articles were carefully analyzed and selected to provide evidence regarding the use of acceleromyography.

CINAHL was utilized for its association with nursing health research (Mateo & Foreman, 2014). Four searches were performed in the CINAHL database. The first search utilized the search words "train of four monitoring" with the all text option. This resulted in 7,633 articles. The all text option was changed to the abstract option to narrow the results. This resulted in forty-six articles, of which five were saved.

A second search within the CINAHL database was performed with the search words "qualitative train of four monitoring" with the all text option selected, which yielded 2,815 articles. Again, the all text option was changed to the abstract option to narrow the results. This resulted in five articles, two of which were duplicates, no articles were saved.

A third search within the CINAHL database was performed with the search word "acceleromyography" with the all text option. This resulted in thirty-nine articles of which two new articles were saved with three articles previously found in the first search.

A fourth search within the CINAHL database was performed with the search words "residual neuromuscular blockade" with the all text option selected. This resulted in 238 articles. The all text option was changed to the abstract option to narrow the results. This resulted in thirty-two articles of which one new article was saved.

PubMed was utilized for its large amount of research in the biomedical arena (Mateo & Foreman, 2014). Three searches were performed. The first search utilized the search words "train of four monitoring" in all fields. This resulted in 2,979 articles. To narrow the search, the all fields option was changed to title/abstract. This yielded thirty-eight articles of which one new article was saved, and one article was a duplicate from prior searches.

A second search in the PubMed database was performed with the search words "qualitative train of four monitoring" in all fields. This resulted in twenty-nine articles. Five new articles were saved, and two were duplicates from prior searches.

A third search in PubMed database was performed with the search word "acceleromyography" in all fields. This resulted in two-hundred and forty articles. In an attempt to narrow the search, the all fields option was changed to title/abstract. This yielded the same, two-hundred and forty articles. Eight new articles were saved, and seven articles were duplicates. In one of the eight saved articles, the reference list provided an additional article to use by searching the PubMed database.

The AANA website was accessed with the search terms "standards of care". This resulted in one new article.

After searches within the CINAHL and PubMed databases had been completed, a total of twenty-four articles were saved and reviewed. A review of the literature will be discussed in detail in the following section.

Review of Literature

Pathophysiology

The neuromuscular junction is the site where the motor neuron and muscle cell communication occurs. At the neuromuscular junction, the neuron and muscle fibers are separated by the synaptic cleft. When the nerve's action potential becomes depolarized, an influx of calcium ions travels through the voltage-gated calcium channel into the nerve cytoplasm. This allows storage vesicles to fuse together with the terminal plasma to release acetylcholine (ACh). The ACh molecules move across the synaptic cleft and bind to nicotinic cholinergic receptors on the muscle membrane's motor end-plate. (Butterworth, Mackey, & Wasnick, 2013)

The makeup of the ACh receptors changes in different tissues and at different times in development. Each ACh receptor consists of five protein subunits: two α, one β, δ, and ε or γ. The γ subunit is substituted instead of the ε when the receptor is fetal or immature. Only two identical α subunits can bind to ACh molecule. If both ACh binding sites are occupied, the ion channel will open. Conversely, if only one ACh binding site is occupied, the ion channel will not open. (Butterworth et al., 2013)

The end-plate potential is generated by sodium and calcium flowing through the open ACh receptor channel and potassium flowing out. When enough ACh receptor sites are occupied, the end-plate potential will depolarize the perijunctional muscle membrane. Voltage-gated sodium channels within the perijunctional muscle membrane open when a threshold voltage is developed across them. The opening of the sodium channels allows for the release of calcium from the sarcoplasmic reticulum. The intracellular calcium is what provides muscle contraction. (Butterworth et al., 2013)

ACh is hydrolyzed into acetate and choline by the enzyme acetylcholinesterase. This enzyme is located on the motor end-plate adjacent to the ACh receptors. After unbinding ACh, the receptors' ion channels close, allowing the end-plate to repolarize. Calcium moves back into the sarcoplasmic reticulum causing muscle relaxation. (Butterworth et al., 2013)

Pharmacology

Neuromuscular blocking drugs are frequently used in the perioperative setting. There are two different classes of neuromuscular blocking drugs that can be used; depolarizing or non-depolarizing. The type of neuromuscular blocker used is based on numerous factors such as perception of ease of intubation with an endotracheal tube, laboratory values specifically potassium, length and type of surgical procedure, as well as practitioner preference.

Depolarizing and non-depolarizing neuromuscular blocking agents (NDMA) provide paralysis by acting on the post-synaptic junction at the neuromuscular junction. Depolarizing neuromuscular blockers are composed of two ACh molecules whereas NDMA's are composed of one ACh molecule.

Anticholinesterase medications are used to antagonize the effects of NDMA's. They work by competitively binding to the postsynaptic receptor at the neuromuscular junction (Brull & Kopman, 2017; Kopman & Eikermann, 2009). Anticholinesterases slow the breakdown of ACh thereby increasing the concentration of acetylcholine at the post-synaptic nicotinic receptor on the muscle, thus re-establishing normal function (Brull & Kopman, 2017; Kopman & Eikermann, 2009; Nagelhout, 2013). Once the enzyme is inactivated, additional anticholinesterase will not increase the availability of ACh.

Depolarizing neuromuscular blockers are not able to be antagonized with an anticholinesterase medication because the depolarizing neuromuscular blocker binds to both ACh sites thereby not allowing a site of action for the anticholinesterase medication. Depolarizing neuromuscular blockers are reversed by the body's plasma cholinesterase.

Train of Four Monitoring

Dosing of NDMA's and antagonizing their effects can be decided by evaluating the patient condition, surgical procedure type and length, time from the last dose of NDMA, as well as using neuromuscular monitors. The American Association of Nurse Anesthetists (2013) states, "when neuromuscular blocking agents are administered, monitor neuromuscular response to assess depth of blockade and degree of recovery" (p. 1). Although it is a standard of care to monitor neuromuscular blockade and recovery, less than 40% of patients are monitored with a qualitative monitor and less than 17% with a quantitative monitor (Brull & Kopman, 2017).

The gold standard of neuromuscular monitors is mechanomyography, a quantitative, objective monitor (Brull & Kopman, 2017; Heir & Hetland, 1999). Mechanomyography is not used in clinical practice due to the complexity of its setup (Brull & Kopman, 2017). There are several quantitative and qualitative neuromuscular monitors available in clinical practice.

A qualitative, subjective neuromuscular monitor such as the peripheral nerve stimulator (PNS) is used most often in clinical practice (Bhananker et al., 2015; Brull & Kopman, 2017). The PNS is classified as a qualitative measure since it relies on the provider's subjectivity to visually and/or tacitly assess the patient's response to an electrical stimulus. The PNS elicits an electrical stimulus to a peripheral nerve corresponding with a muscle contraction. The response seen from the contraction of the muscle is referred to as a twitch (Welliver, Murphy, Kopman, & Brull, 2015). Many PNS' can assess a single twitch, TOF count, double burst stimulation, and post-tetanic count. The TOF count is the most commonly used function of a qualitative monitor (Bhananker et al., 2015; Brull & Kopman, 2017). A TOF of 1 to 2 twitches provides an adequate amount of muscle relaxation for most procedures (Nagelhout, 2013). Fade is determined by subjectively evaluating the force of the muscle contraction of the 4th twitch compared to that of the 1st twitch. The absence of fade occurs when the force of contraction at the 4th and 1st twitch is equal. The PNS' major limitation beyond its subjectivity, is that it does not provide a ratio of receptor blockade once the TOF count has reached 4/4.

Mechanomyography, electromyography, kinemyography, phonomyography, and acceleromyography are objective and quantifiable monitors available to assess neuromuscular blockade (Claudis & Viby-Mogenson, 2008). These quantifiable monitors detect a TOF count and a TOF ratio. The ratio can be calculated once the TOF count reaches 4/4 and provides the difference of amplitude between the fourth and first twitch on a neuromuscular monitor (Thilen

& Bhananker, 2016). Of the quantitative, objective neuromuscular monitors available, the acceleromyography monitor is the most widely used (Claudius & Viby-Mogenson, 2008; Fuchs-Buder, Nemes, & Schmartz, 2016). A cohort study by Bhananker et al. (2015), found anesthesia providers overestimate the TOF count, especially during counts of 1, 2, and 3, utilizing a qualitative peripheral nerve stimulator in comparison to the quantitative acceleromyography monitor, TOF-WATCH SX™.

Acceleromyography can be used to measure the patient's amount of blockade through quantifying the number of twitches via the acceleration of muscle tissue in response to nerve stimulation (Brull & Kopman, 2017). Acceleromyography is based on Newton's second law of motion where force equals mass times acceleration (Claudius & Viby-Mogensen, 2008; Welliver et al., 2015; Brull & Kopman, 2017). Brull and Kopman (2017) state:

> A piezoelectric transducer is attached to a muscle, and when the innervating nerve is stimulated, the muscle movement is sensed by the transducer; a voltage is generated in the piezoelectric crystal, and this electrical signal is analyzed by the acceleromyography monitor. (p. 186)

For example, if the piezoelectric crystal is attached to the thumb (constant mass), the acceleration from the contracted adductor pollices muscle is proportionate to the force needed to move the thumb.

Gätke et al. (2002) completed a prospective, randomized and double-blind study evaluating the effects of monitoring with an acceleromyography monitor, TOF-Guard Bextel®, and intermediate acting NMDA, rocuronium. The use of the acceleromyography monitor did not allow for more accurate titration of rocuronium during the intraoperative period. The lack of titration ability found by Gätke et al. (2002) may have been due to the length of surgery with a

mean of 66.5 minutes and range of 14 to 409 minutes. Since the duration of action of rocuronium is thirty to sixty minutes, the surgeries observed by Gätke et al. (2002) may not have required additional paralysis.

Acceleromyography Calibration

Calibration of the acceleromyography monitor is recommended after administering induction agents and prior to administering neuromuscular blocking agents (Schreiber, 2014). The purpose of calibration is to adjust a supramaximal intensity current that is specific to each patient before receiving a NMDA (Schreiber, Mucha, & Fuchs-Buder, 2011; Colegrave, Billard, Motamed, & Bourgain, 2016). Calibration is recommended in scientific research. However, it is still undecided if it is warranted in the clinical setting. Calibrating the acceleromyography does not provide 100% reliability and may overestimate recovery of neuromuscular blockade by up to 15% (Schreiber, 2014).

Schreiber et al. (2011) found at a TOF greater than or equal to 1.0; the non-calibrated TOF-WATCH SX™ acceleromyography monitor is as reliable as a calibrated TOF-WATCH SX™ acceleromyography monitor to exclude residual neuromuscular blockade. Eliminating calibrating prior to the administration of NMDA's decreases a step during the busy phase of induction. The TOF-Scan™ acceleromyography monitor bypasses the need for calibration compared to its counterpart TOF-WATCH SX™ (Colegrave et al., 2016). Colegrave et al. (2016) found no significant difference during the onset of neuromuscular blockade but significant differences during recovery of moderate and deep neuromuscular blockade between the TOF-Scan™ and the TOF-WATCH SX™. Regardless of the differences during recovery of the blockade, the TOF-Scan™ is "an acceptable clinical alternative to detect residual paralysis before recovery in routine practice" (Colegrave et al., 2016, p.227).

Acceleromyography Monitoring Sites

The effect of NMDA's are muscle dependent. Stimulating the ulnar nerve at the adductor pollices muscle on the arm is the most common mode of intraoperative monitoring in the United States (Thilen et al., 2012). Other options, if the hand and arm are inaccessible, are to stimulate the facial nerve or the tibial nerve (Heier & Hetland, 1999; Gätke et al., 2002; Larsen et al., 2002; Thilen et al., 2012).

A study performed by Capron, Fortier, Racine, & Donati (2006) examined the application of electrodes over the ulnar nerve at the wrist versus electrodes on both sides of the hand over the adductor pollices muscle with acceleromyography in comparison to the gold standard mechanomyography at the adductor pollicis muscle. Capron et al. (2006) found the best correlation between acceleromyography and mechanomyography is to have the electrodes placed on the hand rather than the wrist.

Typically, the orbicularis oculi muscle site is used as an alternative for qualitative monitoring. This monitoring location is especially useful if arms are tucked due to the nature of the surgical procedure. The orbicularis oculi muscle has been shown to correlate more closely with diaphragm paralysis than the adductor pollices muscle (Gätke et al., 2002). Therefore, monitoring at the orbicularis oculi muscle may be more prudent during the onset of neuromuscular blockade. The adductor pollices is more sensitive to NDMA's than the diaphragm, therefore, monitoring at the adductor pollices during recovery of neuromuscular blockade may prove to be optimal as the diaphragm has already recovered (Aflfille, Merritt, Chamberline, & Eikermann, 2009). Anticholinesterase medications guidelines to antagonize NDMA's are based on assessing the degree of neuromuscular blockade only at the adductor pollices muscle (Thilen et al., 2012).

A study performed by Gätke et al. (2002) identified correct placement of the electrodes for monitoring with acceleromyography is lateral to the eye or along the zygomatic arch. The acceleration transducer is placed in the middle of the eyebrow to elicit the greatest response of the orbicularis muscle. However, the corrugator supercilii may also be elicited (Gätke et al., 2002).

Larsen et al. (2002) found the maximum first response of TOF may be nearly 0% at the orbicularis oculi. Meanwhile the maximum first response of TOF at the adductor pollices is between 0% and 100% during the onset of neuromuscular blockade. Recovery of neuromuscular blockade also varied with a TOF ratio of 1.0 at the orbicularis oculi muscle and 0.20-0.40 at the adductor pollices (Larsen et al., 2002). Monitoring one nerve and muscle in the face has been shown to be challenging due to the vast amount of nerves and muscles located there (Gätke et al., 2002; Larsen et al., 2002; Thilen et al., 2012). Monitoring the orbicularis oculi muscle with acceleromyography requires increased stimulation to elicit the orbicularis oculi response, therefore, increasing the likelihood of eliciting responses from other facial muscles (Gätke et al., 2002; Larsen et al., 2002). Patients are also at increased risk for residual paralysis monitoring at the eye muscles than at the adductor pollices (Thilen et al., 2012).

Monitoring at the tibial nerve at the ankle joint to elicit the flexor hallucis brevis muscle may be considered as an alternative site with the use of acceleromyography (Heier & Hetland, 1999). A study performed by Heier & Hetland (1999) examined the effectiveness of monitoring acceleromyography at the tibial nerve on the ankle joint versus the gold standard of monitoring with mechanomyoraphy at the ulnar nerve. Monitoring at the tibial nerve at the ankle joint with the acceleromyography is consistent with mechanomyography at the ulnar nerve during the period of 0/4 TOF (Heir & Hetland, 1999). The adductor pollices continues to be the

recommended site of action to monitor with acceleromyography during induction, duration of action, and recovery of neuromuscular blockade (Heier & Hetland, 1999).

Residual Paralysis

Incomplete recovery of neuromuscular blockade (residual paralysis) after anesthesia is a serious event. Several studies have attributed residual paralysis with adverse events in the postoperative period including inspiratory obstruction, hypoxemia-related increase in ventilation, postoperative critical respiratory events, intraoperative awareness, and unpleasant symptoms of muscle weakness (Fuchs-Buder, Nemes, & Schmartz, 2016; Kopman & Eikermann, 2009; Sauer et al., 2011). Residual paralysis may be in part due to dosing and timing of NDMA's and anticholinesterase medications, type of monitor used, and/or clinical assessment.

Specific anticholinesterase medication dosing recommendations are based on quantitative or qualitative neuromuscular monitors (Bhananker et al., 2015). Therefore, this may lead to inaccuracy of recommended dosage depending on which anticholinesterase medication was administered and what type of monitor was used. One anticholinesterase medication, neostigmine, has dosage guidelines based upon a qualitative TOF monitor (Bhananker et al., 2015). Kopman and Eikermann (2009) outline guidelines for neostigmine administration with a quantitative neuromuscular monitor such as an accelermyograph are: with a TOF count with no response the provider should delay administering anticholinesterase until a TOF count of 2 has been obtained; with a TOF ratio < 0.4 or TOF count of 2-3 the provider can give neostigmine dose 0.02 – 0.05 mg/kg; with a TOF ratio 0.4 – 0.9 a neostigmine dose of 0.015 – 0.025 mg/kg; with a TOF ratio > 0.9 the provider does not need to use anticholinesterase to antagonize the NDMA previously given.

A TOF ratio of less than 0.9 using the ulnar nerve to assess the adductor pollices muscle utilizing a quantitative neuromuscular monitor allows for increased incidence of residual neuromuscular blockade (Brull & Kopman, 2017; Fuchs-Buder et al., 2016; Murphy et al., 2013; Thilen et al., 2012). A TOF ratio greater than 0.4 does not allow providers to accurately assess for the presence of fade (Welliver et al., 2015). Thus, if the patient does not have fade, then the patient may still be at risk for residual blockade. A TOF count of 4/4 does not eliminate residual neuromuscular blockade; in fact, up to 75% of neuromuscular receptors may still be blocked. There is evidence of residual paralysis in up to 64% of cases when utilizing qualitative monitoring methods (Thilen et al., 2012).

Quantitative train of four monitoring is the best way to detect residual neuromuscular blockade (Gätke et al., 2002). Furthermore, "the acceleromyographic TOF ratio, even in the uncalibrated mode, remains the most accurate test to reliably detect residual paralysis" (Capron et al., 2006, p. 1582).

Discussion

The patient in this case review was undergoing a parietal craniotomy. Per surgeon request, she was pharmacology paralyzed for the duration of the surgical procedure. The patient was paralyzed with a TOF of 2/4 twitches or less. She underwent general anesthesia the day before without a history of difficult intubation or anesthetic complications.

After induction and prior to neuromuscular blockade, attempts were made to apply an acceleromyography monitor to the right thumb with electrodes on the ulnar nerve. Due to challenges with the acceleromyography such as the ability to turn on or calibrate, placing the acceleromyography monitor was aborted until after intubation. Calibration is not a necessary step in clinical practice according to the literature.

The patient was intubated with an intermediate acting NMDA, rocuronium. The acceleromyography monitor was switched for another monitor and again applied to the right thumb with electrodes on the ulnar nerve without calibration. The piezoelectric transducer was taped to the patient's thumb, and the thumb was allowed to move freely. This is congruent with the evidence that if the patient has a thumb that is allowed to move freely, this is the best place to monitor neuromuscular blockade.

After the initial intubating dose of rocuronium 30 mg IV, it took the patient fifty-one minutes to reach a TOF of 2/4 twitches. Subsequently, frequent TOF monitoring was performed throughout the surgical procedure. Rocuronium was re-dosed at 10 mg increments when the patient's train of four reached 2/4 twitches. On average, it took the patient 36 minutes to reach a 2/4 TOF after each 10 mg IV bolus of rocuronium. With the use of acceleromyography in this patient, it was predictable that she would require an addition bolus of paralytic around 36 minutes from the prior dose.

During emergence, the patient received glycopyrrolate 0.2 mg IV and neostigmine 2.5 mg IV. The patient had a TOF count of 4/4 over an hour prior to receiving an anticholinesterase medication, therefore a TOF ratio was not performed. The acceleromyography monitor is superior over peripheral nerve stimulators as it provides the practitioner a TOF ratio. The TOF ratio can help guide the practitioner as to appropriate anticholinesterase dosages. Twelve minutes after glycopyrrolate and neostigmine were administered, the patient received lidocaine 50 mg to attenuate the coughing reflex and minimize any increases in intracranial pressures. The patient was subsequently extubated without signs of residual paralysis.

Conclusion

Acceleromyography has been shown in the literature to be a useful, qualitative monitor to help aid the anesthesia provider in guiding neuromuscular blockade at induction, maintenance, and recovery. However, there is a gap in the literature regarding how frequently to monitor neuromuscular blockade to provide a moderate block with the use of an acceleromyography monitor.

Further research is needed regarding the necessity of calibrating the acceleromyography in clinical practice. An alternative acceleromyography monitor that does not require calibration is the TOF-Scan™.

Appropriate choice and dosage of NDMA's may be guided by using the acceleromyography monitor. NDMA's and anticholinesterase inhibitors guidelines should include monitoring site when discussing antagonizing the effects of NDMA's. Monitoring the facial nerve at the eye muscles may be an acceptable alternative for qualitative monitoring, however, research has found limitations with the use of acceleromyography for monitoring the facial nerve (Gätke et al., 2002; Larsen et al., 2002; Thilen et al., 2012). This is in part due to not being able to differentiate specific eye muscles that are elicited by the pizo electric stimulus. The preferred site of neuromuscular blockade monitoring is at the adductor pollices muscle.

The acceleromyography monitor is one tool that can be used to aid the anesthesia provider in making an appropriate judgment for tracheal extubation to reduce the incidence of residual paralysis. The acceleromyography monitor provides the practitioner with a TOF ratio in addition to a TOF count that qualitative monitors provide. A TOF ratio of 0.9 or greater has been shown to decrease residual neuromuscular blockade. Therefore, tracheal extubation should not occur until a TOF ratio of 0.9 has been achieved.

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Appendix A

Anesthetic Considerations for the use of Accelerometry for Neuromuscular Blockade

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Introduction

- Acceleromyography neuromuscular monitoring was introduced in the late 1980s.
- Acceleromyography is a quantitative neuromuscular monitor utilized to assess the level of neuromuscular blockade in patients who received neuromuscular blocking drugs during surgery.

(Claudius & Viby-Mogensen, 2008)

Introduction Continued

- Clinical tests used to evaluate depth of neuromuscular blockade:
  - Head lift
  - Grip strength
    - These tests have been found to be subjective, unreliable and require patient cooperation
- Prevalence:
  - Using qualitative, subjective nerve stimulators is < 40%
  - Using quantitative, objective nerve monitoring is < 17%

Case Information

- Left parietal craniotomy
- 32-year-old female
- 150cm
- 55kg
- No known drug allergies
- ASA physical status: level 3

Pre-operative Evaluation

- Past Medical History:
  - Meningioma
  - Chronic left sided headaches
  - Latent lung tuberculosis
- Surgical History:
  - Cerebral angiogram performed the day prior to the proposed surgery
- Anesthetic History:
  - Uncomplicated

Pre-operative Evaluation Continued

- Pre-op VS:
  - Blood pressure: 120/92 mmHg
  - Heart rate: 103/min
  - Respiratory rate: 20/min
  - Room air oxygen saturation (SpO_2): 96%
  - Temperature: 97.6° Fahrenheit
- Mallampati score: 2
- Thyromental distance: 3 fingerbreadths
- Basic metabolic panel and complete blood count laboratory results: WNL

Anesthetic Course

- Pre-induction:
  - Midazolam 1mg IV
- Inhalational and intravenous induction:
  - Oxygen at 10L per minute and sevoflurane end-tidal concentrations of 1.25%, fentanyl 100 mcg IV, lidocaine 40 mg IV, propofol 110 mg IV, rocuronium 30 mg IV
- Airway & Ventilation:
  - 7.0 mm cuffed ETT, volume control, respiratory rate of 12 breaths/min, tidal volume 460mL
- Sevoflurane turned off and desflurane initiated with end-tidal concentrations of 5% throughout the procedure
- Other medications administered:
  - Dexmedatomidine IV gtt: 0.4mcg/kg/hr, remifentanil IV gtt: 0.15mcg/kg/min, cefazolin 2 g IV, ondansetron 4mg, levetiracetam 1000 mg IV, mannitol 30 mg IV, Ofirmev 1 g IV
- 20-gauge arterial line placed in the left radial artery
- Acceleromyography neuromuscular monitor applied without calibration to the right thumb and electrodes on the right ulnar nerve

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Intraoperative Issues

- Hypotension:
  - Treated with:
    - Phenylephrine bolus x 1: 200mcg bolus IV
    - Phenylephrine infusion: 5mL/hr
    - Dexmedatomidine infusion: decreased to 0.3mcg/kg/min
- Moderate neuromuscular blockade (train of four < 2/4):
  - Rocuronium 10mg boluses were administered x 4 (after initial intubating dose)
- Hypocalcemia (ionized calcium 1.03 mmOl/L):
  - Treated with:
    - Calcium gluconate 1 g IV
  - Repeat lab: ionized calcium 1.33 mmOl/L

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Post-operative Course

- Emergence:
  - Remifentanil IV gtt decreased: 0.05mcg/kg/min, glycopyrrolate 0.2 mg IV, neostigmine 2.5 mg IV, lidocaine 50 mg
- No signs of residual paralysis
- Total procedure time: five hours and thirty minutes
  - Estimated blood loss: 850mL
  - Urine output: 2,635mL
  - Volume replacement:
    - Normal saline: 3,000mL
    - Lactated ringers: 475mL
    - 5% albumin: 500mL
- Discharged home on postoperative day thirteen with home care

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Pathophysiology

- The neuromuscular junction is the site where the motor neuron and muscle cell communication occurs.
- When the nerve's action potential becomes depolarized, an influx of calcium ions travels through the voltage-gated calcium channels into the nerve cytoplasm.
- The ACh molecules move across the synaptic cleft and bind to nicotinic cholinergic receptors and the muscle membrane's motor end-plate.

([Butterworth, Mackey, & Wasnick, 2018])

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Pathophysiology Continued

- Only two identical α subunits can bind to a ACh molecule.
- When enough ACh receptor sites are occupied, the end-plate potential will depolarize the perijunctional muscle membrane.
  - Sodium channels open which allows for the release of calcium from the sarcoplasmic reticulum.
- ACh is hydrolyzed into acetate and choline by the enzyme acetylcholinesterase located on the motor end-plate adjacent to the ACh receptors.
- After unbinding ACh, the receptors' ion channels close, allowing the end-plate to repolarize and move calcium back into the sarcoplasmic reticulum.

([Butterworth et al., 2018])

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Pharmacology

- There are two different classes of neuromuscular blocking drugs that can be used in the perioperative setting:
  - Depolarizing or non-depolarizing (NDMA)
    - Both agents act on the post-synaptic junction at the neuromuscular junction.
- Depolarizing neuromuscular blockers are composed of two ACh molecules.
- NDMA's are composed of one ACh molecule.

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Pharmacology Continued

- Anticholinesterase medications:
  - Used to antagonize the effects of NDMA's
  - Work by competitively binding to the post-synaptic receptor at the neuromuscular junction
  - Slow the breakdown of ACh
    - Increasing the concentration of ACh at the post-synaptic nicotinic receptor on the muscle, thereby re-establishing normal function
- Depolarizing neuromuscular blockers are not able to be antagonized with an anticholinesterase medication.
- Depolarizing neuromuscular blockers are reversed by the body's plasma cholinesterase.

(Brull & Kopman, 2017; Kopman & Eikermann, 2009; Hagelhoul, 2018)

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Train of Four Monitoring

- The AANA (2013) states, "when neuromuscular blocking agents are administered, monitor neuromuscular response to assess depth of blockade and degree of recovery" (p.1).
- Gold standard of neuromuscular monitors:
  - Mechanomyography, a quantitative, objective monitor
- Of the quantitative, objective neuromuscular monitors available, the acceleromyography monitor is most widely used.
- A cohort study by Bhananker et al. (2015) found anesthesia providers overestimate the TOF count, especially during counts 1, 2, and 3 utilizing a qualitative, peripheral nerve stimulator in comparison to the quantitative, acceleromyography monitor, TOF-WATCH SX™.

(Brull & Kopman, 2017; Claudius & Viby-Mogensen, 2009; Fuchs-Buder, Hennes, & Schmartz, 2018; Hall & Helland, 1999)

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Train of Four Monitoring Continued

- Acceleromyography
  - Can be used to measure the patient's amount of blockade through quantifying the number of twitches via the acceleration of muscle tissue in response to nerve stimulation.
  - Based on Newton's second law of motion: force = mass x acceleration
  - "A piezoelectric transducer is attached to a muscle, and when the innervating nerve is stimulated, the muscle movement is sensed by the transducer; a voltage is generated in the piezoelectric crystal, and this electrical signal is analyzed by the acceleromyography monitor" (Brull & Kopman, 2017, p. 186).

(Brull & Kopman, 2017; Claudius & Viby-Mogensen, 2009; Weiller et al, 2018)

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Acceleromyography Monitor Calibration

- The purpose of calibration is to adjust a supramaximal intensity current that is specific to each patient prior to receiving a NDMA.
- Calibration is recommended in scientific research however, it is still undecided if it is warranted in the clinical setting:
  - Schreiber (2014) recommends calibration prior to administering neuromuscular blocking agents.
    - Does not provide 100% reliability and may overestimate recovery of neuromuscular blockade by up to 15%.
  - Capron et al. (2006) states "even in the uncalibrated mode, (acceleromyography) remains the most accurate test to reliably detect residual paralysis" (p. 1582).
  - Colegrave et al. (2016) concluded at a TOF ≥ to 1.0, the non-calibrated TOF-WATCH SX™ is as reliable as a calibrated TOF-WATCH SX™ .
  - Schreiber et al. (2011) concluded that the TOF-Scan™ bypasses the need for calibration compared to its counterpart TOF-WATCH SX™.
- Eliminating calibrating prior to the administration of NMDA's decreases a step during the busy phase of induction.

(Colegrave, Ellard, Molamad, & Bourgain, 2016; Schreiber, 2014; Schreiber, Musha, & Fuchs-Buder, 2011).

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Acceleromyography Monitoring Sites

- Adductor Pollices
  - The most common mode of intraoperative monitoring in the United States.
    - Capron et al. (2006) found the best correlation between acceleromyography and mechanomyography is to have the electrodes placed on the hand rather than the wrist.
- Oribularis Oculi
  - Requires increased stimulation to elicit the orbicularis oculi response therefore increasing likelihood of eliciting responses from other facial muscles.
  - Increased risk for residual paralysis monitoring at the eye muscles than at the adductor pollices.
- Flexor Hallucis Brevis
  - May be considered as alternative site with the use of acceleromyography.

(Abbasi et al., 2002; Hebl & Helland, 1999; Larsen et al., 2001; Tiller et al., 2012)

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Residual Paralysis

- Several studies have attributed residual paralysis with adverse events:
  - Inspiratory obstruction, hypoxemia-related increase in ventilation, postoperative critical respiratory events, intraoperative awareness, and unpleasant symptoms of muscle weakness
- A TOF ratio < 0.9 using the ulnar nerve to assess the adductor pollices muscle utilizing a quantitative neuromuscular monitor allows for increased incidence of residual neuromuscular blockade.
- A TOF ratio > 0.4 does not allow providers to accurately assess for the presence of fade.
- Residual paralysis has occurred in up to 64% of cases when utilizing qualitative monitoring methods.

(Brull & Kopman, 2017; Fuchs-Buder, Hennes, & Schmartz, 2018; Kopman & Eikermann, 2009; Murphy et al., 2013; Saxen, Stahn, Schaaf, Noldge-Schomburg, Meuske, 2011; Tiller et al., 2012; Weiller et al., 2018)

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Residual Paralysis Continued

- Specific anticholinesterase medication dosing recommendations are based on quantitative or qualitative neuromuscular monitors.
  - Kopman & Eikermann (2009) outline guidelines for neostigmine administration with a quantitative neuromuscular monitor such as an accelermyograph:
    - TOF count with no response the provider should delay administering anticholinesterase until a TOF count of 2 has been obtained
    - TOF ratio < 0.4 or TOF count of 2-3 the provider can give neostigmine dose 0.02 – 0.05 mg/kg
    - TOF ratio 0.4 – 0.9 a neostigmine dose of 0.015 – 0.025mg/kg
    - TOF ratio > 0.9 the provider does not need to use anticholinesterase to antagonize the NDMA previously given

(Bhananker et al., 2015)

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Recommendations

- Further research is needed regarding the necessity of calibrating the acceleromyography in clinical practice.
- Further research is needed regarding how frequently to monitor neuromuscular blockade to provide a moderate block with the use of acceleromyography monitoring.
- Further research is needed regarding monitoring sites other than the adductor pollices muscle in the event the adductor pollices muscle monitoring can not be performed.
- A TOF count and ratio should be obtained prior to antagonizing the effects of NDMA's.
  - Tracheal extubation should not occur until a TOF ratio of 0.9 has been achieved.

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Conclusion

- This patient underwent a parietal craniotomy and remained paralyzed intraoperatively at a TOF of 2/4 or less with the aid of an acceleromyography monitor.
- The piezoelectric transducer was taped to the patient's thumb and the thumb was allowed to move freely.
  - This is congruent with the evidence that if the patient has a thumb that is allowed to move freely, this is the best place to monitor neuromuscular blockade.
- With the use of acceleromyography, it was predictable the patient would require additional boluses of paralytic around 36 minutes from the prior dose.
- The patient had a TOF count of 4/4 over an hour prior to receiving an anticholinesterase medication, therefore a TOF ratio was not performed.
  - The acceleromyography monitor is superior over the peripheral nerve stimulator as it provides the practitioner with a TOF ratio. The TOF ratio can help guide the practitioner as to appropriate anticholinesterase dosages.
- The acceleromyography monitor is one tool that can be used to aid the anesthesia provider in making appropriate judgement for tracheal extubation to reduce the incidence of residual paralysis.
  - A TOF ratio of 0.9 or greater has been shown to decrease residual neuromuscular blockade.

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Thank You
Are There Any Questions?

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