AANA Journal Course

Anesthesia and Perioperative Considerations for Patients With Myasthenia Gravis

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Patients with neuromuscular diseases such as myasthenia gravis can present as complicated anesthetic cases. This article reviews anesthetic considerations for optimal perioperative care of patients with myasthenia gravis. The pathophysiology of myasthenia gravis, cholinergic and myasthenic crises, and perioperative management are discussed; this includes the pharmacology of acetylcholinesterase inhibitors vs sugammadex, extubation criteria, pain management, and risk factors for postoperative myasthenic crisis. Anesthesia recommendations include reversal of nondepolarizing neuromuscular blockade agents with sugammadex, obtaining sufficient spontaneous breathing with absolutely no residual curarization before extubation, limited use of opioids and sedatives, avoidance of routine admission to the intensive care unit, and consideration of peripheral nerve blocks for adjunct pain control.

Keywords: Anesthesia, myasthenia crisis, myasthenia gravis, postoperative management, risk factors.

Objectives
At the completion of this course, the reader should be able to:
1. Summarize the pathophysiology underlying myasthenia gravis.
2. Explain factors that contribute to myasthenic and cholinergic crises.
3. Describe the implications that immunosuppressants and anticholinesterase inhibitors have on neuromuscular blockade.
4. Compare the advantages of using sugammadex vs anticholinesterase inhibitors in patients with myasthenia gravis.
5. Identify the risk factors associated with postoperative myasthenic crisis.
6. Describe the specific considerations involving postoperative management.

Introduction
Patients with neuromuscular diseases can present as complicated anesthetic cases, often requiring specific and tailored perioperative considerations. One such disease is myasthenia gravis (MG), an autoimmune disease that specifically targets and reduces availability of nicotinic acetylcholine receptors (AChRs) at the postsynaptic neuromuscular junction.1 This decrease of nicotinic AChR results in varying degrees of muscle paralysis, because the neuromuscular junctions cannot sufficiently transmit an appropriate number of signals between the nerve and muscle fibers. The potentials that are generated at the muscle fibers are too weak to open sodium channels that would otherwise allow for muscle fiber depolarization to occur. Muscle weakness—along with the potential for myasthenic crisis—can occur and lead to poor outcomes.2

Increases in the incidence and prevalence of MG have been noted, with a pooled incidence rate of 5.3 per million person-years and an estimated pooled prevalence rate of 77.7 per million persons.3 This increase may be a result of better diagnosis. Whereas MG mainly used to be of greater prevalence in young women, it is now also found in the elderly population, in which it tends to be underdiagnosed.4 Treatment of MG has improved over the years, which in theory leads to an increased number of patients with MG presenting for surgery.

The aim of this review was to search the literature and highlight best practices that can be used in anesthesia to help decrease weakness after neuromuscular blockade and the precipitation of a myasthenic or cholinergic crisis in patients with MG. As part of the review, a summary of the pathophysiology of MG as well as identification
and treatment of myasthenic crisis is provided, including the implications that immunosuppressants and anticholinesterase inhibitors (AChEIs) have on neuromuscular blockade and the advantages of using sugammadex vs AChEIs in patients with MG. Providers are responsible for treating MG-related comorbidities and should consider multifactorial case management for reducing anesthetic complications perioperatively.

**Methods**

This review of the literature began with a search for articles pertinent to this topic. Multiple literary search engines were used to search for articles including Academic Search Complete, CINAHL (Cumulative Index to Nursing & Allied Health Literature) Complete, MEDLINE Complete, EBSCOhost, JBI EBP Database, PubMed, Ovid, and The Cochrane Library. Article search criteria were limited to articles in English, published in the last 15 years, and with available full text. One article published before 2004 was necessary to include for the literature review because it contained foundational knowledge pertinent to our topic. Searches were conducted using keywords, phrases, and specific subject headings including myasthenia gravis, acetylcholinesterase inhibitors, recurrarization, residual neuromuscular block, rocuronium, succinylcholine, sugammadex, extubation, maintenance, induction, risk factors, anesthesia, and pathophysiology. The evidence was evaluated using the method described by Melnyk and Fineout-Overholt.

**Pathogenesis**

The extent of MG disease correlates directly with the extent of the paralysis. Hallmark characteristics of MG include generalized muscle weakness that improves with rest and an inability to sustain repetition-based activities. If severe enough, the patient may die due to paralysis of the respiratory muscles. Paralysis symptoms that are more characteristic of the disease and are of lesser severity include bulbar-type symptoms (eg, ptosis and diplopia occurs in >50% of patients) and paralysis of the oropharyngeal muscles, proximal limbs, and shoulder girdle.

In approximately 80% to 85% of patients with MG, there are specific antinicotinic AChR antibodies that are detected and pathologically significant for the disease. In most of these seropositive patients, the thymus is affected. The thymus gland is believed to contribute to the production of the autoantibodies responsible for MG, and thymectomy is often indicated for those with this form. Other treatment options for MG are dependent on the disease stage based on the Osserman classification and can include nonspecific immunosuppressive therapy such as corticosteroids, methotrexate, immunoglobulins, cyclosporine, plasmapheresis, and, of course, AChEIs. These treatments or the lack thereof have implications for the anesthesia plan of care and are discussed later in this article.

- **Seronegative Myasthenia Gravis.** Up to 20% of patients with MG are actually seronegative for the antinicotinic AChR antibodies and are instead seropositive for the muscle-specific tyrosine kinase (MuSK) antibodies. Patients with these genotypes typically exhibit bulbar symptoms but can also experience generalized weakness. Of importance, cholinesterase inhibitors have no effect on the disease process and may actually worsen the disease itself.

- **Myasthenic and Cholinergic Crisis.** Regardless of the type of pathognomonic process underlying MG, a patient with this disease is at risk of development of either a myasthenic crisis or a cholinergic crisis. Myasthenic crisis is an exacerbation of the disease, in which the patient begins to exhibit increased muscle weakness and respiratory distress. It is generally attributed to multifactorial precipitants, including respiratory tract infections, stress, and surgery. The severity of this particular crisis and predictors for death include older age, slow recognition of the crisis, and need for endotracheal intubation. This type of crisis is treated with additional doses of cholinesterase inhibitor, and if no improvement, immunoglobulins, plasmapheresis, and endotracheal intubation.

Cholinergic crisis is when the patient is overdosed with cholinesterase inhibitors, and symptoms can range in severity from sweating, abdominal cramping, excessive salivation, urinary urgency, and bradycardia to muscle fasciculations and/or muscle weakness. When this type of crisis occurs, treatment is warranted, primarily with cessation of cholinesterase inhibitors and atropine, but it can require endotracheal intubation until resolved.

Even for the skilled provider, it may be difficult to distinguish between the 2 types of crises. The situation becomes more complicated because if not quickly and correctly recognized, the first-line treatment of myasthenic crisis can exacerbate a cholinergic crisis. To distinguish between myasthenic crisis and cholinergic crisis, it may be helpful to give a single dose of edrophonium, because symptoms will improve with the myasthenic crisis or remain unchanged, but potentially worsen with a cholinergic crisis.

**Preoperative Considerations**

In reviewing the care plan with the patient, the provider must underline that physical stress such as that associated with surgery can exacerbate the disease process, but that the disease process generally returns to baseline. If the surgery is elective, a time when the patient is in a stable phase should be chosen for optimal surgery and anesthesia outcomes. As a rule, most immunosuppressants will not interact with anesthesia. However, if the patient is receiving azathioprine, the medication will prolong the effect of succinylcholine and inhibit the effects of the nondepolarizing neuromuscular blocking agents (NMBAs). If the patient is experiencing an
acute exacerbation or having a myasthenic crisis but requires emergent surgery, every effort should be made to optimize the patient’s condition first. Optimization includes plasmapheresis, immunosuppressive therapy such as exogenous corticosteroids, and discussing postoperative ventilation therapy with the patient. There is an advantage to discontinuing the patient’s pyridostigmine before surgery, as it will allow for a quicker onset of the nondepolarizing NMBA as well as a smaller dose. This practice should be used with caution as the results of one study indicated that patients with MG who did not take their morning dose of pyridostigmine on the day of surgery were at higher risk of development of respiratory distress. Avoidance of respiratory depression should also be a priority for the anesthesia provider, and premedication with sedatives or opioids should not be routine practice, as it is extremely difficult to predict which patients with MG will undergo postoperative myasthenic crisis (PMC).

Considerations for Anesthesia Management

- **Anesthesia Technique.** Whenever possible, peripheral nerve blocks should be used for the MG patient population. If a regional blockade is performed on an upper extremity, be advised that the patient with MG is more vulnerable to respiratory failure. Two such blocks of concern would be either supraclavicular or interscalene, because blocking the phrenic nerve would cause direct paralysis of the diaphragm and compromise respiratory function. For the purpose of this article, the focus will be on general anesthesia management using surgical paralysis, and the sole use of volatile anesthetics, as regional anesthesia is beyond the scope of this review.

- **General Anesthesia.** If general anesthesia must be used, induction should be performed using short-acting intravenous agents, with the understanding that any respiratory depressant effects may be augmented. Maintenance of anesthesia is achieved with volatile anesthetics with or without nitrous oxide. The use of volatile anesthetics may decrease or even eliminate the need for muscle relaxants.

- **Muscle Relaxants.** If the patient with MG requires surgical paralysis, a nondepolarizing neuromuscular blockade is generally selected to sustain appropriate relaxation throughout the surgery. The dose of the nondepolarizing NMBA should be reduced by one-half to two-thirds. A depolarizing NMBA such as succinylcholine has an unpredictable response in those with MG, as this population has poorly functioning postsynaptic AChRs and will likely not receive an adequate blockade or conversely, if the patient is receiving treatment with cholinesterase inhibitors, he or she will have a prolonged blockade. When a nondepolarizing NMBA is used, these responses are not an issue, but sensitivity to nondepolarizing NMABs is increased because of the reduced number of receptors in this patient population. The dose of nondepolarizing NMABs should thus be reduced.

- **Neuromuscular Blockade Monitoring.** The conventional method for monitoring recovery from neuromuscular blockade is with a peripheral nerve stimulator, and the most common method for monitoring neuromuscular blockade recovery is the train-of-four (TOF). After delivery of 4 electrical stimuli—each 0.5 seconds apart—the fourth twitch is either qualitatively or quantitatively compared with the first twitch. Quantitative measurements, usually accomplished with acceleromyography, are a much better method of measurement compared with the subjective qualitative observation.

A residual block is said to be present when the TOF ratio is less than 0.9. This definition is based on upper airway and laryngeal muscle recovery. Patients with MG are at greater risk of residual blockade secondary to their preexisting pathophysiology of neuromuscular transmission deficits.

When using a peripheral nerve stimulator to monitor recovery from NMABs, the provider should be aware that twitch response at the orbicularis oculi muscle may overestimate the depth of neuromuscular blockade, but monitoring at this site may also help to avoid unrecognized persistent neuromuscular blockade in patients with MG.

- **Acetylcholinesterase Inhibitors.** Historically, the

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**Table 1. Osserman Classification of Myasthenia Gravis With Modifications From the Committee of the Myasthenia Gravis Foundation of America**

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<td>1</td>
<td>Specific to ocular weakness. All other muscles function normally.</td>
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<td>2</td>
<td>Mild generalized muscle weakness. May have ocular muscle weakness of any severity.</td>
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<td>2a</td>
<td>Predominant limb and/or axial involvement.</td>
</tr>
<tr>
<td>2b</td>
<td>Predominant oropharyngeal and/or respiratory involvement.</td>
</tr>
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<td>3</td>
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<tr>
<td>4</td>
<td>Severe generalized muscle weakness. May have ocular muscle weakness of any severity.</td>
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<tr>
<td>5</td>
<td>Requiring intubation except during perioperative management.</td>
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reversal of surgical paralysis in patients with MG was accomplished through the administration of an AChEI, such as neostigmine or edrophonium. Acetylcholinesterase inhibitors work by inhibiting acetylcholinesterase, thereby increasing the availability of acetylcholine in the junctional cleft at the receptor site. The increased acetylcholine at the junctional cleft provides competitive action with the neuromuscular blockade for the receptor site. Although this competitive action created by AChEIs at the receptor site is desirable in a healthy patient, it presents concerns in the MG patient population.

The administration of AChEIs in patients with MG presents concerns for 2 reasons: (1) undermedicating with an AChEI could result in a myasthenia crisis, in which severe muscle weakness and respiratory paralysis ensues, and (2) overmedicating could result in a cholinergic crisis, in which excessive acetylcholine causes a depolarizing-like block and profound muscular weakness in addition to the muscarinic side effects. What is disconcerting is that the prolonged blockade may not be as clinically obvious as a myasthenic or cholinergic crisis and may simply go unnoticed. Decreased muscle strength may manifest in hypoventilation, hypercapnia, aspiration, hypoxia, and possible postoperative pneumonia. There is a clinical ceiling effect of AChEIs or, in other words, maximal inhibition. This occurs if the anesthetic provider continues to administer an AChEI when all the receptors have been antagonized. This is important to recognize for 2 main reasons: (1) It can cause paradoxical muscle weakness and (2) when neuromuscular blockade is more profound, the ability of the AChEIs to reverse the blockade is limited. In essence, when the ceiling effect is reached, muscle weakness may actually be precipitated.

It is theorized that this paradoxical muscle weakness may be caused by desensitization of the AChRs, which in turn creates a depolarization block leading to failure of the signal transmission. What is surprising is this effect can be induced in the clinically recommended dosing of neostigmine at 0.04 to 0.07 mg/kg; doses above 0.06 mg/kg carry a threefold increase affecting postoperative atelectasis, longer PACU recovery times and hospital stays, thus increasing overall risk of postoperative respiratory morbidity. Furthermore, there is an increased risk of pulmonary edema and reintubation when unwarranted doses of neostigmine are given (ie, not guided by TOF monitoring). As for the limited ability of the AChEI to reverse a neuromuscular blockade, this can occur when the concentration of the nondepolarizing muscle relaxant is elevated at the neuromuscular junction (determined by a TOF ≤0.2). Spontaneous recovery (represented by a TOF >1) must be present before the use of an AChEI can help achieve recovery to a TOF greater than 0.9. This reversal is a timely process and is generally unachievable within 10 minutes of neostigmine delivery from any degree of paralysis during a volatile-based anesthetic.

- Sugammadex. Sugammadex, approved by the US Food and Drug Administration in December 2015, is a reversal agent for steroidal NMBAs and works by encapsulation via tight water-soluble complexes with the amino steroids (eg, rocuronium, vecuronium, pancuronium) rather than increasing acetylcholine at the neuromuscular junction. Neostigmine competitively antagonizes NMBAs and will reverse paralysis only if there is at least a 1 of 4 on TOF. Sugammadex completely avoids interference with the neuromuscular junction, and cholinergic transmission maintains homeostasis. There is no impact surrounding myasthenic or cholinergic crisis, and twitch is not required for complete and rapid reversal.

Benefits of Sugammadex for Patients With Myasthenia Gravis
The use of sugammadex in patients with varying stages of MG has been described in several publications throughout the last decade. For this review, articles with more than 100 cases involving patients with MG undergoing surgical procedures ranging from thoracic operation, thymectomy, cholecystectomy, cataract removal, endoscopy, abdominal hysterectomy, and herniated disk repair were evaluated. Results demonstrated that sugammadex provides rapid and complete reversal of NMBA while eliminating residual curarization.

Vymazal et al describe a series of 117 cases of MG in which patients underwent various surgeries and had both a rapid reversal of a rocuronium-induced blockade and extubation following sugammadex administration. Patients in various stages of MG received 0.6 mg/kg of rocuronium on induction of anesthesia. To maintain relaxation, a TOF ratio less than 0.6 was maintained with rocuronium, 0.15 mg/kg. For reversal, patients then received sugammadex, either 4 mg/kg if TOF was 1 or less, or 2 mg/kg if TOF was 2 or more. The authors noted that the time to achieving a TOF ratio of 0.9 or greater took a mean of 117 seconds (SD, 10-12 seconds). Extubation after sugammadex administration took 276 seconds (SD, 24-29 seconds). In all cases, no oxygen desaturation (SpO₂ <95%) was noted 24 hours postoperatively. No hypercapnia ( PCO₂ elevation >10% from baseline) or reintubations were noted within 48 hours postoperatively. No postoperative pneumonias occurred within 120 hours. Overall, no signs of postoperative residual curarization or respiratory depression were noted.

In another study, Sungur et al demonstrated full and rapid reversal of a rocuronium-induced blockade with sugammadex in 10 cases of video-assisted thorascopic extended thymectomy, all patients with MG in varying stages. Induction was achieved with rocuronium, 0.3 mg/kg, and supplemental dosing of rocuronium given at one-
fourth the initial dose if there was a TOF value greater than 25% or if diaphragmatic movement was observed. Before sugammadex administration, TOF values ranged from 0% to 50%. All patients received sugammadex, 2 mg/kg, for reversal, and the total mean rocuronium dose was 48 mg (SD, 16 mg). The mean time for a TOF ratio greater than 0.9 after sugammadex administration was 111 seconds (minimum, 35 seconds; maximum, 240 seconds), and all patients were successfully extubated in the operating room. The overall mean operative time was 62 minutes (SD, 16 minutes). All patients were monitored in the postanesthesia care unit (PACU) for 4 to 6 hours and then transferred to the ward. Not a single patient required reintubation due to respiratory distress or myasthenic crisis, again demonstrating that sugammadex allows for a rapid and complete recovery from neuromuscular blockade in patients with MG.22,25

Casarotti et al22 outlined 2 case reports in which high-dose rocuronium for rapid sequence induction and reversal with sugammadex was used in 2 myasthenic patients. The first case involved a 2-hour emergent laparotomy, during which a 48-year-old man received reversal with sugammadex, 4 mg/kg of actual body weight, on 1 posttetanic twitch. A TOF ratio greater than 0.9 was restored within 3 minutes. The patient was monitored for an additional 30 minutes to ensure there was no residual curarization; at the end of this timeframe, sedation was withdrawn and the patient was extubated. Postoperative pain management used morphine and ketorolac, and the patient was transferred to the ward within hours.22

The second case reported by Casarotti et al22 involved an emergent surgery for endoscopy in a 71-year-old woman who had massive hematemesis. The duration of surgery was approximately 1 hour, and at the end of the procedure, the patient had a 1 of 4 TOF. Reversal was again achieved with sugammadex, 4 mg/kg of actual body weight, and a TOF ratio greater than 1 was observed within 2 minutes. The patient was monitored for an additional 40 minutes for recurarization and was extubated and transferred to the ward within 2 hours. In both cases, sugammadex proved to be a fast and reliable reversal agent for recovery of neuromuscular function when high-dose rocuronium was used for rapid sequence induction.

Postoperative Considerations and Management

Due to longer life expectancy and medical advancements, there is a growing population of elderly patients with MG.27 It is important to recognize that patients with MG are undergoing an increasing number of surgeries that may be nonrelated to MG (eg, thymectomy).28 These patients will require a more specific and disease-oriented care plan.

In a retrospective cohort study by Chang et al,28 a total of 2,290 patients undergoing major surgery with a preoperative diagnosis of MG were analyzed over a 6-year timeframe and compared with more than 22,000 randomly selected surgical patients without MG. Procedures were matched by propensity scoring, and overall, surgical patients with MG had higher risk of postoperative complications (ie, pneumonia, septicemia, and intensive care unit [ICU] admission), longer hospitalizations, and higher medical expenditures. The postoperative risks were higher in patients with MG receiving emergency care or undergoing hospitalization or thymectomy.28

Regarding development of PMC, a study by Yu et al29 concluded that patients with MG with a history of preoperative myasthenic crisis, preoperative Myasthenia Gravis Foundation of America (MGFA) class, presence of bulbar symptoms, longer surgical times, greater amount of blood loss, and the presence of thymoma were statistically significant risk factors for PMC.29 Thymoma occurs in approximately 10% to 15% of patients with MG, most commonly found with the early-onset or generalized form of the disease. The relevance of thymoma is that it is associated with hematologic autoimmune disorders, myocardiitis, heart conduction irregularities, and some cancers; thus, thymoma is a complicating factor for negative postoperative outcomes.28 This further corroborates the need for optimization of the perioperative management of patients with MG.

Postoperative considerations for patients with MG include specific extubation criteria, avoidance of ICU admission, and analgesia. Proposed criteria for extubation in a patient with MG include the patient having a normal level of consciousness, a tidal volume of at least 5 mL x body weight (in kilograms), and a respiratory rate less than 30/min.1 The most crucial aspect before extubation is to ensure no residual curarization, making it imperative to use either TOF monitoring in the unconscious patient or a head lift longer than 5 seconds in the conscious patient.1

Most patients with MG undergoing elective surgery can be discharged without an ICU stay or consultation with a neurologist.1 Routine ICU admission with mechanical ventilation should be avoided if at all possible and is not recommended because of the increased risk of respiratory failure and airway-associated complications.1 Prolonged intubation may result in atelectasis,23,30 delay in ambulation, and prolonged ICU length of stay.30 These factors can contribute to an increased risk of emboli and ventilator-associated pneumonia.28 If the patient begins to exhibit signs of deterioration or warrants ICU admission, a neurologist should be consulted as soon as possible.1

Opioid analgesics produce respiratory depressant effects that could be dangerous in the postoperative period, especially if superimposed on muscle weakness, but it is equally important to avoid excessive surgical stress (ie, pain), which could precipitate a myasthenic crisis. Therefore, optimal pain management is crucial. If
avoidance of opioids is a viable option, then whenever possible, regional anesthesia can be used. If opioids are needed, the provider should consider smaller doses of rapid, short-acting medications. Nonsteroidal anti-inflammatory drugs can also reduce some of the need for opioids but may not completely eliminate the necessity. When gastrointestinal tract function is not decreased with use of opioids, patients with MG can resume their usual oral medication of cholinesterase inhibitors sooner. See Table 2 for a list of anesthesia and perioperative recommendations for patients with MG.

**Discussion**

The anesthesia care for patients with MG primarily centers around NMBs and residual curarization, with good reason. There are other important aspects of anesthesia care that need to also be considered. Although it is advantageous to the anesthesia provider to perform a thorough preoperative assessment of every patient, it is especially important to create a patient-oriented care plan that addresses the specific needs of a patient with MG. Consideration of risk factors that can lead to PMC should be screened for, including a history of myasthenic crisis, preoperative MGFA class, bulbar symptoms, and presence of thymoma.

Preoperative

- Educate patient and family that physical stress associated with surgery can temporarily exacerbate disease process.2
- If surgery is elective, it should be performed during a stable phase of the disease process.2,7
- If surgery is emergent and patient’s condition is unstable, every effort should be made to optimize patient’s condition first.7
- Discontinuation of pyridostigmine treatment before surgery is debatable; it allows for quicker onset and smaller dosing of nondepolarizing NMBAs but also increases risk of respiratory distress.8
- In general, most immunosuppressants do not interact with anesthesia.
- Azathioprine therapy should be discontinued, as it prolongs effect of succinylcholine and inhibiting effects of nondepolarizing NMBAs.2
- Avoid premedication with sedatives or opioids as routine practice because of CNS depression effects.2

Intraoperative

- Peripheral nerve blocks benefit patient population, requiring smaller doses of local anesthesia.2
- Caution is advised with any regional blockade that might impede diaphragm function, eg, supraclavicular or interscalene blocks, because blocking phrenic nerve paralyzes diaphragm and impedes respiratory function.9,10
- If patient requires surgical paralysis, choose a nondepolarizing NMBA for a more predictable and adequate blockade.4
- Use sugammadex for rapid and complete reversal of NMBA.11,18-22
- Sugammadex can completely reverse NMBA with high-dose rocuronium used in RSI.18
- Avoid AChEIs for reversal of NMBA because of ceiling effect,12 paradoxical muscle weakness,13 and potentiation of a myasthenic or cholinergic crisis.1
- If neostigmine is used, doses 0.04-0.07 mg/kg can induce paradoxical muscle weakness; doses >0.06 mg/kg carry threefold increase for postoperative complications.15

Postoperative

- Patients with MG have higher risk of complications, particularly with emergent care, hospitalization, and thymectomy.28
- Patients at higher risk of PMC include those with history of myasthenic crisis, preoperative MGFA class, presence of bulbar symptoms, long surgical times, greater amount of blood loss, and presence of thymoma.29
- Specific considerations beyond standard extubation criteria: ensure normal level of consciousness, tidal volume of ≥5 mL/kg, respiratory rate <30/min.2
- Most importantly, ensure sufficient spontaneous breathing and absolutely no residual curarization.2
- Avoid routine ICU admission with mechanical ventilation.2
- If ICU admission is warranted or patient exhibits signs of deterioration, consider immediate consult with neurologist.2
- Physical stress should be minimal, emphasizing optimal pain management. Consider regional anesthesia, rapid and short-acting opioids if indicated, and NSAIDs.2

**Table 2. Anesthesia and Perioperative Recommendations for Patients With Myasthenia Gravis**

Abbreviations: AChEIs, acetylcholinesterase inhibitor; CNS, central nervous system; ICU, intensive care unit; MG, myasthenia gravis; MGFA, Myasthenia Gravis Foundation of America; NMBA, neuromuscular blocking agent; NSAID, nonsteroidal anti-inflammatory drug; PMC, postoperative myasthenic crisis; RSI, rapid sequence intubation.

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From a postoperative perspective, avoidance of ICU admission and delayed extubation should be primary foci. Analgesia to prevent exacerbation of physical stress and, in turn, worsening of the disease process, is also of importance. Because every attempt should be made to reduce opioid consumption, the use of regional anesthesia, nonsteroidal anti-inflammatory drugs, or shorter-acting, more rapid-acting opioids can all aid in recovery.
REFERENCES


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DISCLOSURES
The authors have declared no financial relationships with any commercial entity related to the content of this article. The authors did not discuss off-label use within the article.