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Case Report

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Perioperative Care of a Child With Molybdenum Cofactor Deficiency

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Abstract

Molybdenum is a trace mineral that is a key component of several enzyme systems. In the human body, molybdenum is complexed with a pterin-based molybdenum cofactor (MOCO), to form the active center of molybdenum-based enzymes. MOCO is synthesized in a four-step process involving six proteins, iron, ATP, and copper. Defects in any of the individual genes involved in this biosynthesis can result in molybdenum cofactor deficiency (MoCD). MoCD is an autosomal recessive disorder with an estimated incidence of 1 in 100,000 - 200,000 live births. Although most patients appear normal at birth, intractable seizures typically develop within hours to days of life, along with feeding difficulties, and subsequent microcephaly, brain atrophy, and severe developmental delay. Mortality is high, with a reported median survival of 2.4 to 3 years. Given the associated end-organ involvement, anesthetic management may be required during radiologic imaging or surgery procedures. We present a 6-year-old child with MoCD type A, who required anesthetic care for a magnetic expansion control (MAGEC) rod insertion with posterior spinal instrumentation under general anesthesia. End-organ involvement of MoCD is presented, previous reports of anesthetic care reviewed, and options for perioperative management discussed.

Keywords: Molybdenum cofactor deficiency; Pediatric anesthesiology; Posterior spinal fusion; Molybdenum

Introduction

Molybdenum is a chemical element with an atomic number of 42. It is present as an essential trace mineral in the human body, being a key component of several enzyme systems [1]. Molybdenum is catalytically inactive in biological systems unless it is complexed with a pterin-based molybdenum cofactor (MOCO), forming a key component of the active centers of molybdenum-based enzyme systems [2]. MOCO is an essential organometallic cofactor, that is synthesized in a four-step process involving six proteins, iron, ATP, and copper. Defects in any of the genes involved in this biosynthetic pathway can lead to impaired activity of MOCO-dependent enzymes, resulting in the accumulation of toxic metabolites.

At least 50 molybdenum-containing enzymes have been identified in bacteria, plants, and animals. Molybdenum-bearing enzymes are the most common bacterial catalysts for breaking the chemical bond in atmospheric nitrogen, a key step in the nitrogen fixation process, a chemical process used by microorganisms to convert atmospheric nitrogen into ammonia. The enzymes also catalyze redox reactions, which are responsible for the degradation of various toxic substances and metabolites in the body. A deficiency in MOCO biosynthesis can lead to an inherited metabolic disorder, molybdenum cofactor deficiency (MoCD).

MoCD is a rare autosomal recessive syndrome with an estimated incidence of 1 in 100,000 - 200,000 live births with approximately 200 cases reported worldwide [3]. It was first described in 1978 by Duran et al in a 10-day-old newborn who presented with feeding difficulties, severe neurological impairment, lens dislocation, and dysmorphic head features [4]. Laboratory and chromatographic investigations revealed a combined deficiency of xanthine oxidase and sulfite oxidase. Both enzymes required molybdenum as an essential cofactor, linking the condition to impaired MOCO biosynthesis [5, 6].

Given the associated end-organ involvement, anesthetic management may be required during radiological imaging or surgical interventions in patients with MoCD. We present a 6-year-old child with MoCD type A, who required anesthetic care for magnetic expansion control (MAGEC) rod insertion with posterior spinal instrumentation to treat progressive neuromuscular scoliosis. End-organ involvement of MoCD is presented, previous reports of anesthetic care reviewed, and options for perioperative management discussed.

Case Report

Review of this case and presentation in this format followed the guidelines of the Institutional Review Board of Nationwide Children's Hospital. This review was conducted

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in compliance with the ethical standards of the responsible institution on human subjects as well as with the Helsinki Declaration.

The patient was a 6-year-old, 23.1 kg male, born at term via vaginal delivery. Prenatal history was notable for a 20-week ultrasound showing a choroid plexus cyst that resolved on subsequent evaluation. Although the initial newborn examination was normal, at 8 - 12 h of life, the patient stopped breastfeeding, and at 16 h of life, he began having intractable seizures, leading to admission to the neonatal intensive care unit (NICU). Magnetic resonance imaging (MRI) was consistent with severe hypoxic-ischemic changes. Seizure workup including family history, genetic analysis including karyotype and microarray, newborn screening, and metabolic studies was negative. Treatment for the seizures was started with intravenous (IV) phenobarbital. However, subsequent video electroencephalogram revealed ongoing subclinical seizures which improved with the addition of levetiracetam. Due to persistent episodes of apnea and cyanosis, the patient required tracheal intubation for 9 days during his NICU admission. His clinical status progressively deteriorated with severe neurodevelopmental impairment, spastic quadriplegia, microcephaly, intractable seizures, neuromuscular scoliosis, gastrostomy tube dependence, and recurrent aspiration pneumonia. At 2 years of age, he was referred to our institution for evaluation of static encephalopathy and microcephaly. At that time, family history was notable for a cousin with an autosomal recessive neurodevelopmental disorder, as reported by the mother. Considering the family history, along with maternal concerns about the risk of recurrence in subsequent pregnancies and the possibility of overlooking disease-specific therapies, further genetic testing was performed. This included a single nucleotide polymorphism microarray followed by reflex whole exome sequencing, which confirmed the diagnosis of MoCD type A. Additional biochemical testing revealed undetectable blood uric acid, elevated urinary S-sulfocysteine, increased levels of xanthine and hypoxanthine, and low urinary uric acid, thereby confirming the diagnosis of MoCD type A. Disease-specific therapy with fos-denopterin (substrate replacement therapy) was started with the aim of slowing clinical deterioration. Dosage was gradually increased to the maximum of 0.9 mg/kg/day as mother reported some improvements in the patient's oxygenation levels. However, despite therapy, his clinical course was marked by multiple hospital admissions and emergency visits, due to recurrent respiratory and central line-associated infections. Given his worsening neuromuscular scoliosis, the patient was scheduled at 6 years of age for MAGEC rod insertion, extending from T4/T5 to the pelvis. His past surgical history was relevant for a low-profile gastrostomy device placed to provide long-term feeding support, circumcision, and a Broviac catheter placement in the right internal jugular vein for fos-denopterin infusions. At the time of the procedure, the patient's medication regimen included clobazam to prevent seizures, baclofen to treat spasticity, and oral glycopyrrolate and sublingual atropine to control oral secretions. The patient's weight was 23.1 kg and his perioperative vital signs showed a temperature of 37.2 °C (98.9 °F), pulse 112 beats/min, blood pressure 102/82 mm Hg, and oxygen saturation of 98% on room air. On physical examination, the Mallampati class was unable to be assessed; however, his thyromental distance was more than 3 fingerbreadths. Cardiac and respiratory examinations were otherwise unremarkable. Preoperative laboratory evaluation included a complete blood count and a comprehensive metabolic panel, which were within normal limits. The patient was held *nil per os* (NPO) for 8 h and transported to the operating room. Routine American Society of Anesthesiologists monitors were applied, and anesthesia was induced by the inhalation of incremental concentrations of sevoflurane in nitrous oxide and oxygen. A peripheral IV cannula was placed followed by the administration of propofol (4 mg/kg) to deepen the level of anesthesia. The patient's trachea was intubated orally with a 4.5 mm cuffed endotracheal tube without difficulty. A second peripheral IV cannula was placed and a left radial arterial line inserted under ultrasound guidance. The patient was positioned prone on the Jackson table. Normothermia was maintained by control of the room temperature and a forced air warming blanket. To facilitate intraoperative neurophysiological monitoring (somatosensory and motor evoked potentials), our standard protocol was used [7]. This included no additional neuromuscular blocking agents (NMBAs) and maintenance anesthesia with remifentanil (0.05 - 0.25 µg/kg/min), methadone (0.15 mg/kg), lidocaine (1 mg/kg/h), desflurane (0.5 MAC), and intermittent doses of midazolam to maintain the bispectral index at 50 - 60. Strategies to limit the need for allogeneic blood products included controlled hypotension, intraoperative cell salvage, and tranexamic acid (loading dose 50 mg/kg and maintenance infusion at 5 mg/kg/h). Prophylaxis to prevent surgical site infection included cefazolin (50 mg/kg every 3 h) and one dose of gentamicin (5 mg/kg). The intraoperative course was uneventful. Two transient episodes of hypotension were treated with ephedrine. Low ionized calcium was treated with a single dose of calcium gluconate. Toward the end of the procedure, the remifentanil and lidocaine infusions were stopped, and a bolus dose of hydromorphone (0.02 mg/kg) was administered. A total volume of 820 mL of Normosol-R was administered during the 5 h and 35 min of anesthetic care. Estimated blood loss was 100 mL. At the completion of the procedure, the patient was turned supine and his trachea extubated when awake. He was transported to the post-anesthesia care unit (PACU) and thereafter admitted to the pediatric intensive care unit (PICU) for postoperative care and monitoring. His pain control regimen included IV morphine via nurse-controlled analgesia, scheduled IV acetaminophen, scheduled IV ketorolac, and IV diazepam as needed. On postoperative day 1, the patient developed a fever, which was attributed to his post-surgical status rather than an infection, as confirmed by a negative blood culture. Attempts to discontinue supplemental oxygen resulted in mild hypoxemia. These episodes were attributed to poor airway clearance, atelectasis, and limited respiratory reserve. A chest radiograph revealed low lung volumes with perihilar and lower lobe atelectasis. Aggressive chest physiotherapy was instituted to treat atelectasis and improve airway clearance. Eventually, the patient was weaned off supplemental oxygen. He was transferred to the inpatient ward on postoperative day 2 and discharged home on day 6.

Discussion

This case description is unique as it provides extensive details on the general anesthetic care of a pediatric patient with MoCD during a major surgical procedure. This is of clinical interest considering the paucity of literature on anesthetic management in children with this rare genetic condition. The name, molybdenum, is derived from the Greek word "molybdos," which means "lead-like." Molybdenum is a naturally occurring element that is obtained from various dietary sources. It was identified as an essential element for plants before identification of its critical role as a cofactor for four human enzymes: sulfite oxidase (SOX), xanthine oxidase (XO), aldehyde oxidase, and mitochondrial amidoxime-reducing component (mARC) [2, 3]. MOCO, which is the active moiety, is composed of the molybdenum oxide ion and molybdopterin, which is synthesized *de novo*.

MOCO is synthesized through a three-step pathway involving the molybdenum cofactor synthesis genes (MOCS): MOCS1, MOCS2, and MOCS3, with the final step catalyzed by the multi-domain protein gephyrin, encoded by the GPHN gene. Depending on the specific biosynthetic step affected, MoCD can be classified into three types (A, B, and C). MoCD types A and B account for the majority of cases seen, while type C is extremely rare with a more rapid and severe clinical presentation. This is in part because the product "*gephyrin*" of the mutated gene (GPHN) has a dual function in both MOCO synthesis and as a neuroreceptor clustering protein, leading to more severe neurological symptoms [8-10]. Mutation of any of these genes leads to the pleiotropic loss of all molybdenum-dependent cellular processes and the subsequent accumulation of toxic metabolites including sulfite, taurine, s-sulfocysteine, and thiosulfate.

As noted in our case, patients may initially appear healthy at birth and then within hours to days of life develop myoclonic encephalopathy along with feeding difficulties and irritability, which quickly progresses to intractable seizures, encephalopathy, and severe neurological impairment. Although head circumference may be normal at birth, microcephaly with brain atrophy develops within the neonatal period. This leads to severe developmental delay with affected patients unable to feed, sit or speak. Lens dislocation during infancy and nephrolithiasis have also been reported. Mortality is high, associated with recurrent lower respiratory tract infections and seizures, with a reported median survival of 2.4 - 3 years. Replacement therapy with cyclic pyranopterin monophosphate (cPMP), a missing component for MOCO synthesis in MoCD, may be effective for patients with MoCD type A, offering improved neurodevelopmental prognosis when started early [11]. Fosdenopterin, a synthetic form of endogenous cPMP, is the only approved drug treatment for MoCD. It has demonstrated substantial effectiveness in treated patients across two prospective clinical trials, and a retrospective observational study. The study results when compared to a genotype-matched observational cohort of untreated patients, showed evidence of survival benefit [12, 13]. The success of this substrate replacement therapy depended not only on the postnatal age at which therapy is started, but also on the presence of irreversible cerebral lesions before treatment initiation. As a result, a favorable outcome is linked to a timely intervention [3, 11, 13]. Notably, our patient was diagnosed at the age of 3 years, and fos-denopterin was initiated 6 months later; therefore, this therapy would not be expected to result in the reversal of symptoms. Instead, the main goal was to halt or slow disease progression and prevent further clinical deterioration.

Given the end-organ involvement that invariably occurs with MoCD, anesthetic care may be required for various imaging or surgical procedures. As with the anesthetic care of all patients, effective perioperative care of these patients begins with a thorough preoperative examination, the identification of end-organ involvement, and identification of potential perioperative concerns. To date, there is only one previous report of anesthetic care in a patient with MoCD, thereby limiting the information available in the literature [14]. Alkan et al reported perioperative care of a 4-year-old boy with MoCD diagnosed at 5.5 months of age who presented for dental rehabilitation. Comorbid conditions included lens subluxation, bilateral vision loss, mental-motor impairment, and cerebral atrophy. General anesthesia with spontaneous ventilation and a native airway was initiated by the administration of IV ketamine (0.5 mg/kg) and midazolam (0.5 mg/kg) followed by maintenance anesthesia with 2% sevoflurane in nitrous oxide and oxygen via a nasal mask. The needed depth of anesthesia was maintained throughout the procedure along with spontaneous respiration, using additional doses of IV ketamine. A local anesthetic agent was injected where necessary for tooth extraction and filling. The procedure lasted 50 min and no perioperative adverse effects were reported. The authors identified three major perioperative concerns of these patients including the associated seizure disorder, risk of regurgitation leading to pulmonary aspiration/infection, and the potential for adverse effects with the administration of NMBAs due to hypotonia and spastic quadriparesis. They reasoned that the choice of anesthetic should be one that does not trigger seizures while preserving respiratory drive and protective airway reflexes.

In patients with inherited or genetic disorders, one of the primary concerns related to anesthetic care is the potential for difficulties with airway management and endotracheal intubation [15]. Our preoperative evaluation did not identify anatomic findings suggesting that airway management would be problematic such as micrognathia or limited mouth opening. Additionally, the limited information regarding patients with MoCD requiring anesthetic care does not suggest this as a consequence of the disorder. We noted no concerns with bag-valve-mask ventilation or endotracheal intubation. In patients with cognitive impairment, airway management may be further compromised by the risk of aspiration and difficulties with secretion management as noted in our patient. These concerns may warrant the use of rapid sequence intubation (RSI). Given the confirmed NPO status of our patient and limited clinical concerns of significant aspiration, inhalation induction was chosen [16].

The presence of seizures is a universal comorbid feature of patients with MoCD. There may be a risk of developing breakthrough seizures during the perioperative period. Preoperatively these patients should be evaluated for increased frequency of seizures at baseline, history of a recent seizure close

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to the day of the procedure, presence of seizure triggers (stress, sleep deprivation), and specifically any disruption in anti-seizure medications or recent changes in drug regimen perioperatively which can lead to subtherapeutic medication levels [17]. Patients should continue their routine anti-seizure medications including on the morning of the surgery [18]. These should be administered despite concerns of the patient's NPO status [19]. When enteral administration is not feasible, alternative routes of delivery including IV administration may be feasible for several anticonvulsant agents [20]. Consultation with the neurology or pharmacology service is suggested when questions arise concerning dosing conversion from enteral to IV administration. The choice of anesthetic agents in patients with seizure disorders remains somewhat controversial [21]. Although it has been postulated that specific agents may activate the electroencephalogram (EEG) and hence augment seizure activity, in general, the inhalational and IV anesthetic agents are anticonvulsants [22, 23].

Central nervous system involvement in patients with MoCD results in hypotonia which may impact choice and dosing of NMBAs [24, 25]. Although there is little to no evidence-based medicine on which to base the clinical decision making process, theoretical concerns may suggest the need to avoid succinylcholine, given the potential for rhabdomyolysis and hyperkalemia [26]. Although non-depolarizing NMBAs may be used, their response and duration may be exaggerated in patients with hypotonia and neuromuscular involvement. For our patient, endotracheal intubation was performed under deep inhalational anesthesia with sevoflurane, supplemented with propofol to avoid these issues and allow for effective neurophysiological monitoring (motor-evoked potentials). When NMBAs are administered, sugammadex may provide an additional margin of safety for reversal of neuromuscular blockade, especially in patients with hypotonia or neuromuscular disorders [27]. Train-of-four monitoring is recommended when using NMBAs in patients with rare neurometabolic disorders as a means to titrate dosing and document effective reversal of neuromuscular blockade.

Perioperative care with an increased risk of adverse respiratory effects may be impacted by poor airway control, hypotonia, and respiratory muscle insufficiency in patients with central nervous system (CNS) disabilities. These factors resulted in poor airway clearance, atelectasis, and postoperative hypoxemia in our patient. These findings demonstrate the importance of postoperative respiratory monitoring and aggressive chest physiotherapy to improve airway clearance. As the residual effects of anesthetic agents may impact upper airway control and postoperative respiratory function, short-acting agents whose effects dissipate rapidly should be considered. In our patient, it is likely that the combined effects of the comorbid involvement of hypotonia, poor airway control, decreased cough effort, mucus plugging, and sensitivity to the respiratory depressant effects of opioids resulted in hypoventilation and atelectasis. These preoperative concerns led to our choice of postoperative care in the PICU setting. Non-invasive respiratory support (bilevel positive airway pressure (BiPAP)) may prevent the need for reintubation due to respiratory insufficiency/failure [28].

Joint and soft tissue contractures or orthopedic deformities may impact intraoperative positioning, especially in the prone position on the Jackson table. Padding of bony prominences is imperative to prevent skin breakdown during prolonged procedures or immobilization. Growth failure, nutritional issues, and CNS disabilities may predispose to hypothermia. Intraoperative care includes continuous temperature monitoring and the use of overhead heating lights, forced air warming devices, and increased room temperature.

In summary, we present the anesthetic management of a 6-year-old child with MoCD. To date, this is only the second report of anesthetic care in such patients. Anesthetic concerns included the presence of a comorbid seizure disorder, potential for postoperative respiratory failure related to hypotonia, poor airway control, and pulmonary aspiration, as well as the impact of hypotonia on choice of NMBA. Given the severe associated CNS involvement, short-acting anesthetic agents (desflurane, remifentanil) may be preferred to facilitate emergence from anesthesia. A thorough perioperative evaluation and adopting an individualized anesthetic plan can allow for safe anesthetic care in patients with rare neurometabolic disorders.

Learning points

Anesthetic care in patients with MoCD can be challenging as no formal guidelines concerning management have been established. However, employing an individualized anesthetic plan based on the patient's disease severity, comorbidities, and surgical procedure may be helpful. This should involve a multidisciplinary approach including but not limited to neurologists, geneticists, surgeons, and clinical pharmacology services. Anesthetic concerns to anticipate in these patients include breakthrough seizures, anesthetic-agent related adverse effects, risk of postoperative respiratory failure, and the need for mechanical ventilation and/or prolonged hospitalization. Perioperative continuation of routine anti-seizure medications, avoidance of general anesthetic agents that could precipitate or lower seizure thresholds, adequate intraoperative airway management, and commencement of early postoperative respiratory management is suggested. Short-acting inhalational and IV anesthetic agents can be effective and were safely tolerated by our patient. Given the associated hypotonia, we would recommend avoidance of succinylcholine, use of short-acting NMBAs, and effective documentation of reversal prior to tracheal extubation. Postoperative monitoring of respiratory and hemodynamic function may be required based on associated comorbid conditions.

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Financial Disclosure

None to declare.

Conflict of Interest

None to declare.

Informed Consent

Informed consent was obtained for hospital/anesthetic care and the use of de-identified information for publication.

Author Contributions

EEV: preparation of initial, subsequent, and final drafts; AS: direct patient care, literature review, review of drafts and final document; ICO: literature review, review of drafts and final document; JDT: concept, writing, and review of all drafts.

Data Availability

Any inquiries regarding supporting data availability of this study should be directed to the corresponding author.

References

1. Oskarsson A, Kippler M. Molybdenum - a scoping review for Nordic Nutrition Recommendations 2023. Food Nutr Res. 2023;67:10326. doi pubmed
2. Mendel RR. The molybdenum cofactor. J Biol Chem. 2013;288(19):13165-13172. doi pubmed
3. Atwal PS, Scaglia F. Molybdenum cofactor deficiency. Mol Genet Metab. 2016;117(1):1-4. doi pubmed
4. Duran M, Beemer FA, van de Heiden C, Korteland J, de Bree PK, Brink M, Wadman SK, et al. Combined deficiency of xanthine oxidase and sulphite oxidase: a defect of molybdenum metabolism or transport? J Inherit Metab Dis. 1978;1(4):175-178. doi pubmed
5. Johnson JL, Rajagopalan KV. Purification and properties of sulfite oxidase from human liver. J Clin Invest. 1976;58(3):543-550. doi pubmed
6. Cohen HJ, Johnson JL, Rajagopalan KV. Molecular basis of the biological function of molybdenum. Developmental patterns of sulfite oxidase and xanthine oxidase in the rat. Arch Biochem Biophys. 1974;164(2):440-446. doi pubmed
7. Martin DP, Bhalla T, Thung A, Rice J, Beebe A, Samora W, Klamar J, et al. A preliminary study of volatile agents or total intravenous anesthesia for neurophysiological monitoring during posterior spinal fusion in adolescents with idiopathic scoliosis. Spine (Phila Pa 1976). 2014;39(22):E1318-1324. doi pubmed
8. Johannes L, Fu CY, Schwarz G. Molybdenum cofactor deficiency in humans. Molecules. 2022;27(20):6896. doi pubmed
9. Reiss J, Lenz U, Aquaviva-Bourdain C, Joriot-Chekaf S, Mention-Mulliez K, Holder-Espinasse M. A GPHN point mutation leading to molybdenum cofactor deficiency. Clin Genet. 2011;80(6):598-599. doi pubmed
10. Feng G, Tintrup H, Kirsch J, Nichol MC, Kuhse J, Betz H, Sanes JR. Dual requirement for gephyrin in glycine receptor clustering and molybdoenzyme activity. Science. 1998;282(5392):1321-1324. doi pubmed
11. Schwahn BC, Van Spronsen FJ, Belaidi AA, Bowhay S, Christodoulou J, Derks TG, Hennermann JB, et al. Efficacy and safety of cyclic pyranopterin monophosphate substitution in severe molybdenum cofactor deficiency type A: a prospective cohort study. Lancet. 2015;386(10007):1955-1963. doi pubmed
12. Kang C. Fosdenopterin: first approval. Drugs. 2021;81(8):953-956. doi pubmed
13. Farrell S, Karp J, Hager R, Wang Y, Adeniyi O, Wang J, Li L, et al. Regulatory news: Nulibry (fosdenopterin) approved to reduce the risk of mortality in patients with molybdenum cofactor deficiency type A: FDA approval summary. J Inherit Metab Dis. 2021;44(5):1085-1087. doi pubmed
14. Alkan M, Kip G, Sahin S, Atabek D. Choice of anesthesia in molybdenum cofactor deficiency: A case report. J Res Med Sci. 2014;19(11):1103-1105. pubmed
15. Butler MG, Hayes BG, Hathaway MM, Begleiter ML. Specific genetic diseases at risk for sedation/anesthesia complications. Anesth Analg. 2000;91(4):837-855. doi pubmed
16. Tobias JD. Rapid sequence intubation: what does it mean? Does it really matter? Saudi J Anaesth. 2014;8(2):153-154. doi pubmed
17. Niesen AD, Jacob AK, Aho LE, Botten EJ, Nase KE, Nelson JM, Kopp SL. Perioperative seizures in patients with a history of a seizure disorder. Anesth Analg. 2010;111(3):729-735. doi pubmed
18. Perks A, Cheema S, Mohanraj R. Anaesthesia and epilepsy. Br J Anaesth. 2012;108(4):562-571. doi pubmed
19. Jones CT, Raman VT, DeVries S, Cole JW, Kelleher KJ, Tobias JD. Optimizing anticonvulsant administration for children before anesthesia: a quality improvement project. Pediatr Neurol. 2014;51(5):632-640. doi pubmed
20. Dhallu MS, Baiomi A, Biyyam M, Chilimuri S. Perioperative management of neurological conditions. Health Serv Insights. 2017;10:1178632917711942. doi pubmed
21. Zuleta-Alarcon A, Castellon-Larios K, Moran KR, Soghomonyan S, Kurnutala LN, Bergese SD. Anesthesia-related perioperative seizures: pathophysiology, predisposing factors and practical recommendations. Austin J Anesth Analg. 2014;2(4):1026.
22. Wong BJ, Agarwal R, Chen MI. Anesthesia for the pediatric patient with epilepsy and minimally invasive surgery for epilepsy. Curr Anesthesiol. 2021;11:233-242.
23. Bloor M, Nandi R, Thomas M. Antiepileptic drugs and anesthesia. Paediatr Anaesth. 2017;27(3):248-250. doi pubmed
24. Azar I. The response of patients with neuromuscular disorders to muscle relaxants: a review. Anesthesiology. 1984;61(2):173-187. doi pubmed
25. Ragoonanan V, Russell W. Anaesthesia for children with neuromuscular disease. Contin Educ Anaesth Crit Care

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Pain. 2010;10:143-147.
26. Martyn JA, Richtsfeld M. Succinylcholine-induced hyperkalemia in acquired pathologic states: etiologic factors and molecular mechanisms. Anesthesiology. 2006;104(1):158-169. doi pubmed
27. Tobias JD. Current evidence for the use of sugammadex in children. Paediatr Anaesth. 2017;27(2):118-125. doi pubmed
28. Jaber S, Michelet P, Chanques G. Role of non-invasive ventilation (NIV) in the perioperative period. Best Pract Res Clin Anaesthesiol. 2010;24(2):253-265. doi pubmed

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