Review Article

Regional anesthesia and muscle-wasting diseases in pediatrics: A focused educational review

ABSTRACT

The muscular dystrophies or muscle-wasting diseases include a diverse group of genetic disorders, which result in progressive degeneration of skeletal muscles, progressive muscle weakness, and comorbid multi-system involvement. Duchenne muscle dystrophy is the most common type of muscular dystrophy with a reported incidence of 1 in every 3500–6000 male live births in the United States. Given the progressive nature of these disorders, skeletal muscle weakness frequently progresses to loss of the ability to ambulate and perform functions of daily life. In addition to affecting the skeletal musculature, many muscular dystrophies have effects on both cardiac and smooth muscles. As respiratory muscles are one of the most frequently affected muscles in patients with muscular dystrophies, progressive respiratory insufficiency may occur with dependance on non-invasive forms of respiratory support. Given the progressive multi-system involvement associated with the muscular dystrophies, perioperative care and the use of general anesthetic agents and opioids may result in postoperative respiratory failure. In an effort to avoid the deleterious effects of anesthetic agents and opioids on hemodynamic and respiratory functions, regional anesthesia may be used as an adjunct to or instead of general anesthesia. This manuscript provides a literature review and educational summary regarding the use of regional anesthetic techniques in pediatric-aged patients with muscular dystrophies.

Key words: Duchenne muscular dystrophy, muscular dystrophy, pediatric anesthesia, regional anesthesia

Introduction

In 1830, Sir Charles Bell, a Scottish surgeon, physiologist, neurologist, and anatomist first wrote about a disease affecting primarily boys and causing progressive muscle weakness. In 1836, Italian researchers documented the case of two brothers who experienced generalized weakness, muscle damage, and replacement of damaged muscle tissue with fat and connective tissues. However, during that period, these symptoms were initially attributed to tuberculosis. During the 1850s, additional reports appeared in the literature, describing boys, who progressively weakened, lost their ability to walk, and experienced an early demise. In 1868, the French neurologist, Guillaume Duchenne provided a comprehensive account of 13 boys affected by the most common and severe form of muscular dystrophy, which now bears his name, Duchenne muscular

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Amr Elhamrawy[1], Islam Elmitwalli[1], Candice Burrier[1,2], Giorgio Veneziano[1,2], Joseph D. Tobias[1,2]

[1]Department of Anesthesiology and Pain Medicine, Nationwide Children's Hospital, Columbus, Ohio, [2]Department of Anesthesiology and Pain Medicine, The Ohio State University College of Medicine, Columbus, Ohio, United States

Address for correspondence: Dr. Joseph D. Tobias, Department of Anesthesiology and Pain Medicine, Nationwide Children's Hospital, 700 Children's Drive, Columbus, Ohio - 43205, United States.
E-mail: Joseph.Tobias@Nationwidechildrens.org

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dystrophy (DMD). Subsequently during the 1950s, German physician, Peter Emil Becker, was the first to document a milder variant of a related muscular dystrophy. He placed his own name on this condition, which is now known as Becker muscular dystrophy (BMD). Both DMD and BMD result from mutations in the same gene, exhibit similar symptoms with the progressive development of skeletal muscle weakness, generally starting in early childhood.[1,2] These two disorders represent two of the more common disorders, which are generally classified as muscular dystrophies.

Muscular dystrophies (MDs) include a diverse group of inherited genetic disorders, which result in progressive degeneration of skeletal muscles, resulting in progressive muscle weakness. Related effects in cardiac and smooth muscles result in associated comorbid multi-system involvement. There is a wide spectrum of muscle involvement, end-organ involvement, and disease progression in muscular dystrophies including Duchenne/Becker muscle dystrophy, myotonic muscle dystrophy (MMD), congenital muscle dystrophy (CMD), limb-girdle muscle dystrophy (LGMD), distal muscular dystrophy, oculopharyngeal muscular dystrophy (OPMD), and facioscapulohumeral muscular dystrophy (FSHD). The clinical signs and symptoms, age of onset, muscle groups affect, and involvement of other end-organs (cardiac) vary depending on the type of muscular dystrophy.

DMD is the most common type of muscular dystrophy, affecting 1 in every 3500–6000 male live births in the United States. DMD is an X-linked recessive, resulting from mutations in the dystrophin gene.[1] In muscle cells, the dystrophin complex localizes at the membrane, connecting the intracellular cytoskeleton to the cell membrane and the extracellular matrix.[2] The dystrophin complex functions as a membrane stabilizer during muscle contraction to prevent contraction-induced damage to the cellular structure. Dysfunction of the dystrophin complex leads to progressive muscle damage, degeneration, and cell death [Figure 1]. DMD is typically diagnosed when a child fails to meet gross motor milestones, frequently with an onset as early as 2 years of age. Progressive involvement of the skeletal muscle manifests as difficulty managing stairs or assuming a standing position. By adolescence, patients lose their ability to ambulate independently, becoming wheelchair-bound. In addition to its impact on the function of skeletal muscle, dysfunction of the dystrophin complex leads to progressive effects on cardiac muscle. This result in myocardial dysfunction and eventual development of cardiomyopathy characterized by a progressive decline in ejection fraction, which generally becomes clinically evident during the teenage years or early adult life.[3,4] Given the associated respiratory and cardiac involvement as well as the cellular dysfunction of muscle cells, these patients have an exaggerated risk of morbidity and mortality during perioperative care.

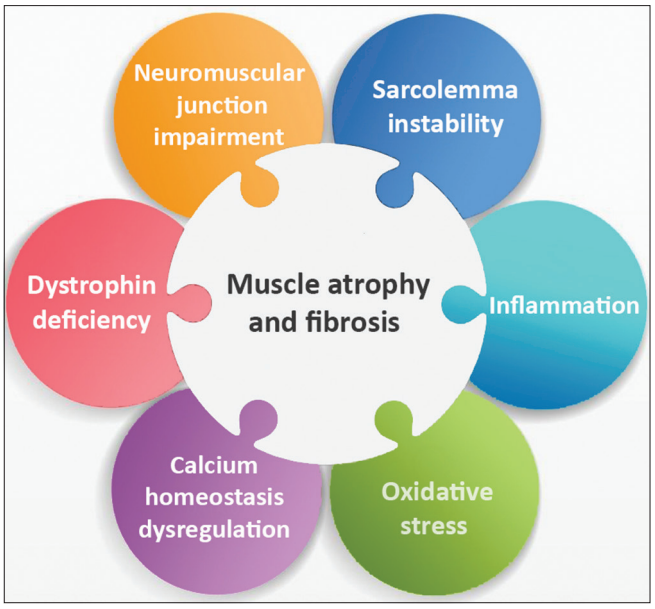

Figure 1: Representation of the varied pathophysiology resulting in muscle dysfunction, damage, and atrophy in patients with muscular dystrophy

With the ongoing multi-system involvement, surgical intervention may be required to correct sequelae of the primary disease progression including progressive orthopedic deformities or treatment of conditions unrelated to the primary disease process. In an effort to avoid the deleterious effects of anesthetic agents and opioids on hemodynamic and respiratory functions as well as adverse effects related to end-organ involvement, regional anesthesia may be used instead of general anesthesia. This manuscript provides a literature review and educational summary regarding the use of regional anesthetic techniques in pediatric patients with muscular dystrophies as the primary anesthesia technique to avoid general anesthesia.

Perioperative Complications and Muscular Dystrophy

Because of related structural protein mutations affecting the skeletal muscle cells, patients with MD present several challenges during anesthetic care. The risks related to anesthesia in patients with various muscular dystrophies can be divided into those related to the primary cellular defect involving the muscle itself as well as the risks related to progressive end-organ involvement. Depending on the specific type of MD, risks related to the primary cellular muscle defect include the potential for an exaggerated hyperkalemic response to depolarizing neuromuscular

blocking agents (succinylcholine), heightened sensitivity to non-depolarizing neuromuscular blocking agents, a predisposition for malignant hyperthermia in a subset of these patients (central core disease, King–Denborough syndrome), a predisposition to depolarization of the sarcolemma resulting in rhabdomyolysis and hyperkalemia with prolonged administration of halogenated volatile anesthetic agents, and exacerbation of mitochondrial dysfunction with prolonged administration of propofol.[5-8] End-organ involvement may include involvement of the skeletal, smooth, and cardiac muscles. Primary perioperative concerns include upper airway involvement with obstructive sleep apnea and a higher incidence of difficulties with airway management and endotracheal intubation related to prolonged involvement of skeletal muscles of the face and jaw.[9] Respiratory insufficiency is common with many patients, requiring non-invasive ventilation including bilevel positive airway pressure (BiPAP), especially nocturnally. Upper airway and pharyngeal involvement may not only result in OSA and airway effects, but also swallowing dysfunction, a higher incidence of gastroesophageal reflux, and the potential for aspiration. Respiratory skeletal muscular involvement may result in poor cough effort, perioperative atelectasis, and ventilation–perfusion inequalities, and the chronic predisposition to recurrent pneumonia. Cardiac involvement is common, especially in DMD, manifesting as dilated cardiomyopathy during the second and third decades of life. When postoperative mechanical ventilation is needed, difficulties weaning from mechanical ventilation may be encountered. All factors may impact perioperative outcomes, increasing the incidence of morbidity and mortality following all types of surgical intervention. Given these concerns, investigators have reported the use of regional anesthetic techniques (neuraxial and peripheral nerve blockade) as a means of avoiding the potential perioperative risks involved with the administration of general anesthesia to patients with MD.

Regional Anesthesia and MDs

A systematic search of PubMed®, EMBASE, and Web of Science was conducted using the following terms: muscular dystrophy, dystrophinopathy, Duchenne, pediatric, regional anesthesia, peripheral nerve blockade, and neuraxial anesthesia. The abstracts from the publications were reviewed and those pertaining to the use of regional anesthetic techniques, including neuraxial anesthesia and peripheral nerve blockade, were included for further review. Additionally, the reference list of these publications was reviewed to ensure that all applicable manuscripts had been identified.

Our review revealed only 16 previous reports of 33 pediatric patients, outlining the use of regional anesthesia instead of general anesthesia in pediatric patients with muscular diseases [Tables 1 and 2].[9-24] This included 10 reports of neuraxial (caudal/epidural/spinal anesthesia) including 26 pediatric patients as well as 6 reports of peripheral nerve blockade including 7 pediatric patients. Several patients had associated cardiac involvement with dilated cardiomyopathy and depressed ejection fraction. Neuraxial techniques included 12 caudal epidural, 2 lumbar epidural, and 12 spinal anesthetic techniques. Peripheral blockade included upper extremity blocks (one supraclavicular and one axillary block), lower extremity blocks (one femoral–obturator and one bilateral popliteal-sciatic block), and truncal blocks (PEC and intercostal blockade in two patients and unilateral ESP block in one patient). The patients ranged in age from 5 weeks to 18 years. A wide range of surgical procedures was reported including hernia repair, appendectomy, muscle biopsy, amputation, Achilles tenotomy/extension, fasciotomy, hip muscle release, automatic implantable cardiac defibrillato (AICD) placement, orchidopexy and video-assisted thoracoscopic surgery (VATS). All patients who had a peripheral nerve blockade had DMD; however, the neuraxial group was more varied (three patients with DMD, one with Emery–Dreifuss, and the remaining with congenital muscular dystrophy). The majority of patients received procedural sedation during performance of the regional block and during the surgical procedure with a variety of agents including ketamine, dexmedetomidine, midazolam, and/or propofol.

The most commonly used local anesthetic (LA) agent in caudal/epidural/spinal blockade was bupivacaine (0.25–0.5%) ± epinephrine. The most common LA dose for spinal anesthesia was 0.3 mg/kg. One case report outlined the use of 3% chloroprocaine for epidural anesthesia. For peripheral nerve blockade, 0.5% ropivacaine ($n = 3$), 0.5% bupivacaine ($n = 3$), and 1% lidocaine ($n = 1$) were used with volumes ranging from 15 to 30 mL. Regional anesthesia was performed either as the primary anesthetic technique (neuraxial/peripheral nerve blockade) or in combination with sedation with a native airway and maintenance of spontaneous ventilation. A single anecdotal case reported the induction of anesthesia with the inhalation of halothane in nitrous oxide and oxygen to facilitate the placement of an intravenous cannula and performance of a single-shot caudal block. Following the placement of the regional anesthetic technique, halothane and nitrous oxide were discontinued, allowing the caudal block to provide the primary surgical anesthetic without the administration of other agents. In these anecdotal reports, no intraoperative or postoperative concerns were noted. All

Table 1: Neuraxial blockade instead of general anesthesia in pediatric patients with muscular dystrophy

Author	Age/gender	Surgical procedure	Anesthesia type	Comments
Alexander *et al*.[10]	2-year-old, 8.9 kg. girl with CMD	Bilateral repair of talipes equinovarus.	Caudal anesthesia (halothane/nitrous oxide)	Caudal block with 8 mL of 0.5% bupivacaine. Halothane was discontinued. Procedure time was 150 min. No intraoperative or postoperative concerns. Discharged on POD 5.
Bray *et al*.[11]	5-week-old, 2.1 kg. boy with CDM	Bilateral inguinal hernia repair.	Caudal block	Caudal block with 2.1 mL of 0.25% bupivacaine with 1:400,000 epinephrine. No premedication was used. No intraoperative or postoperative concerns.
Tobias[12]	11-year-old, 32 kg boy with MD	Tendon transfers in the lower extremities.	Epidural anesthesia (L3-4) under sedation with ketamine	Initial bolus dose of 15 mL of 3% chloroprocaine followed by a continuous infusion of 15 mL/hr. A sensory level of T_{12} was obtained. The procedure lasted for 3 h. The epidural catheter was left in place for 72 h.
Zanette *et al*.[13]	17 infants (11 males, 6 females), weight 6.38±5.5 kg	Idiopathic clubfoot correction with muscle biopsy for suspected muscle dystrophy.	Caudal (n=8)/ spinal anesthesia (n=9)	Sedation included ketamine and diazepam. Caudal was selected for bilateral procedures and spinal for unilateral. Caudal block performed using bupivacaine 0.25% with epinephrine 1:200,000. Spinal anesthesia with bupivacaine 0.5% based on body weight.
O' Higashi *et al*.[14]	9-year-old boy with CMD. Weight: not listed	Bilateral hip muscle release.	Caudal-epidural block	Sedation with diazepam and pentazocine. No intraoperative or postoperative concerns.
Shiraishi *et al*.[15]	2-year-old boy with CMD. Weight: not listed	Extension of the Achilles tendon.	Caudal block	Sedation with ketamine. Caudal block with lidocaine 1% (10 mL). No intraoperative or postoperative concerns.
Shende *et al*.[16]	5-year-old boy with DMD. Weight: not listed	Orthopedic surgery with temporary pacemaker insertion.	Epidural anesthesia (L3-4)	Epidural anaesthesia was administered with bupivacaine 0.5% (10 mL) with epinephrine 1:200,000. Sensory block to T8 level was achieved. The operation lasted for 60 min. Discharged home on POD 2.
Caliskan *et al*.[17]	3-year-old, 15 kg with DMD	Orchidopexy.	Spinal anesthesia (L5-S1)	Sedation with midazolam 0.05 mg/kg and propofol 20 mg. Spinal anesthesia with 1.6 mL of 0.5% hyperbaric bupivacaine. Sensory blockade to T6-7. Surgical time 25 minutes. Uneventful intraoperative and postoperative course.
Apiliogullari *et al*.[18]	10-year-old, 23 kg. boy with DMD	Plantar fasciotomy and achilloplasty	Unilateral spinal anesthesia (L4-5)	Spinal anesthesia with 0.3 mg/kg of 0.5% hyperbaric bupivacaine. Sensory block to T6-7 level. The patient was kept in left lateral decubitus and reverse Trendelenburg position. The procedure lasted for 25 minutes with no intraoperative or postoperative adverse events
Özmete *et al*.[19]	2-year-old, 9 kg. boy with DMD	Bilateral inguinal herniorrhaphy.	Spinal anesthesia (L5-S1)	Spinal anesthesia with 0.3 mg/kg 0.5% hyperbaric bupivacaine was injected in subarachnoid space. Sedation was with intravenous midazolam (1 mg) and ketamine (10 mg). Sensory coverage was confirmed to T10. The first need for analgesics was 4.5 h after the operation (oral paracetamol suspension). Discharged home on POD 1

MD: myotonic dystrophy; DMD: Duchenne muscular dystrophy; POD: postoperative day; POH: postoperative hour; TIVA: total intravenous anesthesia; PACU: postanesthesia care unit; VATS: video-assisted thoracoscopy surgery; CMD: congenital muscular dystrophy; MMD: myotonic muscle dystrophy; CDM: congenital dystrophia myotonica; EDMD: Emery–Dreifuss muscular dystrophy

patients experienced an uncomplicated postoperative course, and they were discharged home without delay.

Discussion

Given the primary defect and associated end-organ involvement, the conduct of general anesthesia in patients with any of the various MDs can be challenging, with the literature demonstrating an increased incidence of perioperative morbidity and even mortality. Given these concerns, various authors have presented anecdotal experience with the use of regional anesthesia instead of general anesthesia in patients ≤18 years of age. Our review identified 16 previous reports including 33 pediatric patients with various MDs in whom regional anesthesia was used instead of general anesthesia. A neuraxial technique was used in 26 patients, whereas a peripheral nerve block was placed in 7 other patients. The neuraxial technique included caudal epidural, lumbar epidural, and spinal anesthesia, whereas peripheral nerve blockade included upper extremity blockade, lower extremity blockade, as well as truncal blockade (PEC, intercostal, and ESP block).

Despite the efficacy of regional anesthesia, specific challenges exist with such techniques. The performance of these blocks and subsequent care of these patients require pediatric anesthesiologists who are trained and experienced in performing regional anesthesia, especially in patients with significant comorbid conditions including MDs. The increased use of ultrasound has facilitated such procedures, especially peripheral and truncal blockade. Given the age and cognitive states of patients, some degree of sedation may be required during performance of the regional anesthetic technique. Given the depth of sedation required, adverse effects may occur related to the impact of sedative and analgesic agents on respiratory and hemodynamic functions. When these techniques are continued into the postoperative period to

Table 2: Peripheral nerve blockade instead of general anesthesia in pediatric-aged patients with muscular dystrophy

Author	Age/gender	Surgical procedure	Anesthesia type	Comments
Büget MI *et al*.[20]	17-year-old male adolescent with DMD. Dilated cardiomyopathy with EF 23%.	Amputation of left arm (large rhabdomyosarcoma).	Single shot supraclavicular nerve blockade.	Local anesthetic dose: 30 mL of 0.5% bupivacaine. Fever to 39°C on POD 3, which resolved with cold compresses and paracetamol. No acidosis, hyperkalemia, or hypercapnia. Discharged home on POD 3.
So M *et al*.[21]	14-year-old, 35 kg. boy with DMD.	Left gracilis muscle biopsy for suspected DMD.	Femoral-obturator nerve blockade.	Local anesthetic dose: 15 mL of 1.5% lidocaine. No adverse events were noted in the perioperative period.
Froyshteter AB *et al*.[22]	16-year-old, 144 kg. adolescent male and 25-year-old, 72 kg. male with DMD. Both with dilated cardiomyopathy.	AICD placement for recurrent episodes of ventricular tachycardia.	Unilateral PEC, intercostal blocks, and local anesthetic infiltration with sedation.	Sedation included dexmedetomidine infusion (0.3–0.7 μg/kg/min), midazolam, and ketamine. Local anesthetic dose: ropivacaine 0.5% with epinephrine 1:200,000 (25 mL for the 1st patient and 20 mL for the 2nd one). Both patients tolerated the procedure well and were discharged home the following day.
Tladi R *et al*.[23]	12-year-old male with DMD. Weight: not listed.	Bilateral Achilles tenotomy for clubbed feet.	Bilateral popliteal-sciatic nerve blockade under sedation.	Sedation with midazolam 1 mg and ketamine 15 mg. Local anesthetic dose: 20 mL of 0.5% bupivacaine. The procedure lasted for 40 minutes. He was discharged home the following morning
Şahin AT *et al*.[24]	18-year-old male with DMD. Weight: not listed.	VATS exploration.	Unilateral ESPB under sedation.	Sedation with midazolam and dexmedetomidine. Local anesthetic dose: 20 mL of 0.5% bupivacaine. Decortication surgery was performed in 3 hours. Hemodynamic parameters were stable, and he had no pain or any complications.
Yadav *et al*.[25]	16-year-old adolescent with DMD. Weight: not listed. Dilated cardiomyopathy (EF 15-20%).	Fasciotomy for compartment syndrome.	Axillary brachial plexus block.	Local anesthetic dose: 15 mL 0.5% ropivacaine. Intraoperative and postoperative course were uneventful. The patient was discharged from the hospital on POD 10.

AICD: automatic implantable cardiac defibrillator; EF: ejection fraction; PEC: pectoralis; TAP: transversus abdominis plane; PACU: post anesthesia care unit; ESPB: erector-spina plane block; CHF: congestive heart failure; VATS: Video-assisted thoracoscopy surgery; POD: post-operative day

provide ongoing analgesia, monitoring of these patients is required with specific dosing protocols, including the choice of the LA agent and the doses used. Even with such care, adverse effects may occur related to the placement of the catheter or its subsequent use, including local anesthetic systemic toxicity. Whenever feasible, peripheral nerve blockade may be preferable to epidural/spinal anesthesia to avoid or limit sympathetic blockade, especially in patients with comorbid cardiac involvement. Additional attention is required when patients with comorbid cardiac involvement are receiving acute or chronic anticoagulation therapy.

The absence of prospective trials limits the evidence-based medicine available that can be used to develop guidelines for the optimal local anesthetic agent to use, its concentration, the rate of infusion, and the choice of adjunctive agents. Additionally, although the anecdotal evidence supports the efficacy and safety of these techniques, there is no evidence-based medicine to definitely show their superiority over general anesthesia.

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Conflicts of interest
There are no conflicts of interest.

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