Anesthetic Implications for the Patient With Osteogenesis Imperfecta

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Osteogenesis imperfecta is an inherited disorder of the connective tissue stemming from gross abnormalities in collagen formation and structure. Affected patients fall into 4 classifications each displaying the similar properties of easily fractured bones, hypermobile joints, blue or gray sclera, skeletal deformities, and fragile skin. More severe forms of the disease may manifest platelet dysfunction, cardiac anomalies, hypermetabolic syndromes, respiratory compromise, and/or basilar invagination. Treatment of osteogenesis imperfecta is mainly supportive, consisting of prompt surgical treatment of fractures to prevent deformity and maintaining mobility to lessen the chance of pulmonary or cardiovascular complications. These treatment modalities make anesthesia of paramount importance.

Current literature exposes many potential anesthetic complications associated with osteogenesis imperfecta. The research shows that implications range from simply positioning the patient on the operating room table to management of rare occurrences such as malignant hyperthermia and basilar invagination. Commonly encountered complications include a difficult airway, intraoperative bleeding due to platelet dysfunction, respiratory compromise due to skeletal deformity, and congenital cardiac anomalies. Proper preparation and preoperative assessment is important, as is the choice of anesthetic technique. Correct identification of risk factors and optimization of health before surgery should lead to an uneventful anesthetic course.

Keywords: Anesthesia, collagen, osteogenesis imperfecta.

Osteogenesis imperfecta is an inherited disorder of connective tissue usually transmitted as an autosomal dominant trait. The pathophysiology of the disease revolves around defects in collagen formation, leading to common characteristics that are present in each of the 4 currently identified types. Treatment modalities for osteogenesis imperfecta primarily serve to limit progression of cardiopulmonary complications and any decreases in mobility as well as to promptly perform surgical intervention of fractures. Recognition of potential complications of the disorder is vitally important for the anesthesia provider as well as for all staff involved in the continuum of care of patients with osteogenesis imperfecta.

This review of the literature will focus on positioning, airway management, spinal deformities, pulmonary mechanics, cardiac anomalies, bleeding diathesis, hyperthermia, and the rare occurrence of basilar invagination. Proper preoperative preparation, strict intraoperative monitoring, and supportive postoperative care will dramatically lessen the chance of adverse outcomes in these patients.

History and Review of Literature

The first recorded case of osteogenesis imperfecta was in a partially mummified body discovered in ancient Egypt, but it was not until 1835 that the term was coined and the disease was truly understood.[1] Lobstein was the first to correctly identify the pathophysiology of the disease and first termed the disorder Lobstein's disease.[1] The name was later changed to osteogenesis imperfecta, which means "imperfect bone formation." It is now known that osteogenesis imperfecta is one of the most common skeletal dysplasias, affecting approximately 6 to 7 per 100,000 people, and occurring in approximately 1 in 20,000 births.[2] It appears to affect males and females equally and has no partiality for a particular race.[2] Osteogenesis imperfecta is classified as either *congenita* meaning that fractures are present at or before birth, or *tarda* based on the presence of fractures after birth.[3]

The common characteristic of all cases of osteogenesis imperfecta is a gene mutation that leads to either defective collagen formation or a reduction in collagen formation.[2] The formation of bone consists almost entirely of collagen, with type I being the most prevalent.[4] Type I collagen is also the most abundant protein in the skin and other tissues providing strength and structure to the body. With defects in its structure, connective tissues are weakened, causing bones to be brittle and to fracture easily. About 90% of all cases of osteogenesis imperfecta are due to mutations in the genes responsible for making the proteins that are used to assemble type I collagen.[2] The structure of collagen is complex but mainly consists of 2 α-1 protein chains and a different α-2 chain wound together in a semirigid rod.[4] Molecular defects in these chains will help to confirm diagnosis and differentiate between types of osteogenesis imperfecta.

The characteristic features of different types of osteogenesis imperfecta overlap, but specific changes in bone and genetic factors along with signs and symptoms are used to classify them. Before classifying specific types of osteogenesis imperfecta, it is first necessary to recognize many of the common characteristics associated with the disease. These include but are not limited to blue or gray sclera, thin skin, opalescent teeth (dentinogenesis imperfecta), presenile hearing loss, hyperextensible joints, cardiac valve anomalies, and thoracic and spinal deformities.[4] Currently, there have been 4 major types of osteogenesis imperfecta identified.

Type I is characterized by bony fractures during childhood that can result from mild trauma.[2] It is the mildest and most common form of the disease.[1] Affected individuals have a nearly normal stature, little bone deformity, and characteristically blue sclera. Frequent fractures begin to occur with ambulation during childhood but decrease dramatically after puberty[4]; however, osteoporosis is present throughout life.[5] Hearing loss may manifest during the second or third decade with all types of osteogenesis imperfecta.[5] This first type of osteogenesis imperfecta has clearly been defined as autosomal dominant, resulting in decreased type I collagen formation and normal type III collagen synthesis.[4]

Type II is the most severe mutation, with bony changes becoming evident at 15 weeks' gestation. Death usually occurs in utero or within 24 hours of birth due to respiratory failure.[5] Upon autopsy, affected individuals have bones that are easily crushed between the thumb and forefinger.[4] Most of these patients will not survive to undergo surgical procedures, so this type will not be discussed further.

In severity, type III ranks second.[4] Fractures during childhood are common and persist throughout life, resulting in progressive growth failure and bone deformity. Some patients may be able to ambulate but most require assistive devices later in life.[5] Frequent fracture results in bowing of long bones, dwarfism, and severe kyphoscoliosis that may put the patient at risk for respiratory and cardiovascular complications. Its inheritance is autosomal recessive and autosomal dominant, resulting in an absence of α-2 chains in type I collagen.[4] This type of osteogenesis imperfecta may pose the greatest risk to a patient undergoing surgical procedures that require either general or regional anesthesia.

Type IV has primarily autosomal dominant inheritance and lacks progressive deformities and deterioration.[4] Other distinguishing characteristics are the normal or grayish sclera and mild to moderate short stature.[5] There is moderate to severe fragility of bones due to a reduced type I collagen production and small mutations in its α-2 chains. In severity, this type falls between type I and type III.[4]

The current treatment of osteogenesis imperfecta is aimed at preventing gross deformity and maintaining mobility, with prompt surgical treatment of fractures, and at reducing scoliosis to lessen the likelihood of pulmonary or cardiovascular complications.[5] These treatment modalities make intraoperative anesthesia of paramount importance and necessitate an in-depth discussion of the currently identified anesthetic implications of osteogenesis imperfecta. These include the constant management of multiple anatomic and physiologic abnormalities associated with the disease.[6]

Positioning
Positioning of the patient during the start of a procedure is an integral part of any surgery and is the responsibility of the anesthesia provider as well as the surgeon and other operating room staff. Careful positioning of a patient with osteogenesis imperfecta is of utmost importance to avoid fractures.[6] Simply moving the patient from the stretcher to the operating room table can result in a fracture. The table should be padded with careful consideration to pressure points. Joint laxity can lead to dislocation, implying that care should be taken to avoid overextension during positioning for surgical exposure. If available, a molding mattress is preferred, as these patients may be unable to lay flat and supine.[7] An arterial line should be considered in patients with severe osteogenesis imperfecta, as pressure from an automated blood pressure cuff can cause fractures. During insertion of an intravenous catheter, light pressure should be applied above the site of insertion, or a loosely applied tourniquet may be used. Skin may be extremely thin and easily torn with even small amounts of adhesive tape. After positioning and monitor placement is complete, attention is turned to induction of anesthesia and airway management.

Airway Management
A difficult airway must always be assumed in patients with osteogenesis imperfecta; however, proper preparation for laryngoscopy and intubation can lead to an uneventful anesthetic course during induction.[7] Overextension of the cervical spine can lead to odonto-axial dislocation or fracture and must be avoided.[8] Upward translocation of the cervical spine, known as basilar invagination, can distort the airway anatomy and will be discussed later. If the provider has experience in fiberoptic intubation, this would appear to be the preferred method of securing the airway.[7] Other methods for maintaining cervical spine immobility during intubation include the use of an intubating laryngeal mask airway or, in emergency cases, the use of a stylet. A short neck, a protruding mandible, and the presence of a pigeon chest will make visualization of the glottis difficult and should be anticipated.

A delicate mandible and the presence of dentinogenesis imperfecta can lead to jaw fracture and easily chipped or dislodged teeth. If teeth are missing or a cleft palate is present, the laryngoscope blade may slip out of place and make intubation difficult.[7] Often, the tongue is large in

proportion to the mouth, making visualization difficult. The optimal patient position can be achieved by carefully placing a blanket beneath the upper back to help restore anatomical relationships. Direct laryngoscopy should be performed with minimal disturbance to the mucosa, as bleeding and bruising occur easily.[9] After the airway has been secured, attention is turned to spinal deformities and their effect on the pulmonary mechanics of a patient with osteogenesis imperfecta.

Spinal Deformity

Kyphoscoliosis and thoracic cage deformity is prevalent in the osteogenesis imperfecta population.[10] The incidence of scoliosis in patients with osteogenesis imperfecta under age 5 years is approximately 26% and increases to 82% in older children. Respiratory complications secondary to chest deformity and scoliosis lead to limitations in thoracic function and make it the principal cause of death in most patients with osteogenesis imperfecta.[3]

Thoracic spine curvature smaller than 35 degrees will impair respiratory function during exercise, whereas curves of more than 50 degrees will result in a decreased vital capacity at rest. Morbidity and mortality increase dramatically when thoracic curves surpass 80 degrees. Deformities in the thoracic spine and abnormal positioning of the ribs limit thoracic movements and lung expansion. These deficiencies are worsened by rib fractures and respiratory muscle weakness.[3]

Pulmonary Mechanics and Anesthesia Considerations

Spinal deformities will predispose the patient with osteogenesis imperfecta to pulmonary disease, with increasing scoliosis strongly correlating to restrictive pulmonary disease.[3] Severe thoracic deformity will cause a reduced vital capacity, decreased chest wall compliance, and hypoxemia due to ventilation/perfusion mismatch.[11] A diagnosis of severe dysfunction is made when vital capacity falls below 15 mL/kg (normal, greater than 70 mL/kg).[12] Forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) are decreased secondary to small lung volumes. An FEV_1-FVC ratio of greater than 80%, present in many patients with osteogenesis imperfecta, supports the diagnosis of restrictive lung disease.[3]

Anesthesia in patients with respiratory disease will present a challenge to the anesthesia provider. Airway instrumentation may precipitate laryngeal or bronchial spasm, and the presence of an endotracheal tube allows inspired gases to bypass the filtering, humidifying, and warming functions of the upper airway. Without adequate humidification and warming of the anesthetic gases, mucosal drying will occur and pathogens may gain entry into the lung parenchyma. If available, a heat and moisture exchanger may help to prevent these manifestations. A reduction in functional residual capacity will pre-

dispose these patients to rapid hypoxemia following apnea with induction of anesthesia.[12] Positive pressure ventilation will worsen the already present ventilation-perfusion mismatch in patients with osteogenesis imperfecta and may necessitate an increase in the inspired oxygen content and/or positive end-expiratory pressure.[13] Decreased compliance will lead to higher than normal peak inspiratory pressures and can lead to barotraumas. Tidal volumes should be reduced to 4 to 8 mL/kg, with a compensatory increase in respiratory rate to minimize increases in end-tidal carbon dioxide. Airway pressures should generally be kept below 30 cm of water (H_2O) in patients with restrictive lung disease.[12] Volatile anesthetics depress the respiratory drive (enflurane, desflurane, isoflurane, sevoflurane, halothane), diminishing the body's natural response to hypoxia and hypercapnia.[13]

Regional techniques will help to avoid the pulmonary complications associated with general anesthesia, but performing regional blocks may be difficult or impossible because of orthopedic and anatomical anomalies in patients with osteogenesis imperfecta.[6] Platelet dysfunction is a concern for the anesthesia provider performing spinal blocks and epidurals. The platelet level should be checked and platelet transfusions administered if necessary. Coagulopathies are of concern also, so tests such as prothrombin time, partial thromboplastin time, and thrombin time should be performed to evaluate primary hemostasis.[12] If regional anesthesia is not an option, many decisions regarding general anesthetic technique will help to facilitate smooth induction and maintenance of anesthesia. Premedication with an inhaled β-2 agonist such as albuterol is prudent, but use of opiates and benzodiazepines can cause respiratory depression and should be used with caution.[13]

Propofol, thiopental, and etomidate are acceptable intravenous induction agents; however, ketamine is preferable in patients with osteogenesis imperfecta. Ketamine has the ability to preserve respiratory drive and will maintain airway reflexes and oral musculature, eliminating the need for an oral airway. Nondepolarizing muscle relaxants are preferable, as fasciculations associated with succinylcholine may produce fractures and muscle damage. In emergency cases when succinylcholine may be necessary, pretreatment with a nondepolarizing muscle relaxant will attenuate fasciculations. Muscle relaxation may be maintained with a nondepolarizing agent, although atracurium should be avoided because of its propensity to cause histamine release, leading to exacerbations of restrictive airway disease. Intraoperative analgesia should consist of cautious opioid administration. Morphine may cause histamine release, and nonsteroidal anti-inflammatory drugs may exacerbate asthma, so these agents should be avoided.

During the postoperative period, any residual effects

of anesthesia or neuromuscular blockade will depress upper airway muscular tone, and airway obstruction may occur. An already weakened chest wall will make the patient with osteogenesis imperfecta unable to compensate for this obstruction, and delayed extubation should be anticipated. Pretreatment of airway edema with dexamethasone may be considered.[7] If extubation is allowed, oxygen should be administered until the patient is fully awake and recovered from anesthesia. Effective analgesia will reduce the incidence of postoperative respiratory complications. Most respiratory problems encountered during recovery are due to shallow breathing, poor lung expansion, atelectasis, and subsequent infection.[13] An antiemetic should be administered to prevent aspiration and/or fracture of brittle facial bones due to vomiting during the postoperative period.[7]

Cardiac Anomalies

Type I collagen makes up approximately 85% of cardiac muscle and provides stiffness to the ventricular wall. Decreased collagen diameter and amount leads to decreased ventricular stiffness and major alterations in the structure and mechanics of the myocardium. Myocardial fiber stiffness is affected by many properties of collagen, mostly collagen diameter, which is reduced up to 42% in patients with osteogenesis imperfecta. This would imply that there are important differences in the left ventricular stress-strain relationship. Studies have shown that collagen cross-linkage is increased to compensate for the decreased myocardial fiber stiffness in people with osteogenesis imperfecta. Some studies have shown that the osteogenesis imperfecta–affected heart also contains a compensatory increase in type III collagen.[14]

The abnormal myocardial collagen amount and structure in osteogenesis imperfecta hearts has led to reports of aortic dissection, left ventricular rupture, and aortic or mitral valve incompetence.[14] Atrial or ventricular septal defects or a patent ductus arteriosus have been reported and must be dealt with accordingly. It is prudent to perform echocardiography to rule out cardiac abnormalities in high-risk patients with osteogenesis imperfecta before performing surgical intervention.[7] The most commonly reported cardiovascular abnormality in osteogenesis imperfecta patients is mitral valve prolapse; however, this anomaly is also one of the most common abnormal traits in humans. This observation makes it difficult to determine whether or not the irregularity is simply the result of an autosomal dominant trait. The most frequently reported cardiac abnormality specific to osteogenesis imperfecta is that of aortic root dilatation.[15]

Many of these abnormalities may prove insignificant and nonprogressive, requiring no surgical intervention. If surgical repair is required, patients with osteogenesis imperfecta carry a much higher risk of complications related to intrinsic tissue weakness, delayed or impaired wound healing, musculoskeletal weakness and deformity, platelet dysfunction, and capillary fragility.[16]

Hemorrhagic Diathesis

Bleeding and bruising tendencies in these patients are well documented. If the patient is undergoing anesthesia for the first time or if there is a history of hemorrhagic diathesis, platelet function tests should be performed before surgery.[7] However, it should be noted that intraoperative or postoperative hemorrhage could still occur despite normal bleeding times and coagulation studies.[10] Evidence has shown increased capillary fragility, decreased platelet retention, decreased factor VIII production, and decreased collagen-induced platelet aggregation.[17] Decreased release of platelet factor in these patients can also lead to hemorrhagic diathesis.[11] The underlying collagen abnormality can result in delicate tissues and small blood vessels that are unable to adequately constrict. All can lead to excessive intraoperative bleeding and slow oozing during the postoperative period.[17]

Clotting abnormalities in osteogenesis imperfecta usually exist at the stage of platelet plug formation. After a blood vessel is damaged, the vessel should constrict and a platelet plug should begin to form. Exposed collagen normally attracts platelets, which in turn cause the release of adenosine diphosphate, leading to further platelet aggregation. A defective platelet response will prevent effective clotting and can lead to excessive bleeding. In rare cases, disseminated intravascular coagulopathy can result. The possibility of this potentially fatal complication should be seriously considered. Treatment may consist of the administration of platelets, fresh frozen plasma, cryoprecipitate, and/or packed red blood cells.[17]

Platelet counts of less than $50,000 \times 10^9$/L are associated with increased blood loss during surgery. Patients with preoperative platelet counts of less than 10,000 to $20,000 \times 10^9$/L should receive platelet transfusions before surgery because of an increased risk for spontaneous hemorrhage. A goal of $100,000 \times 10^9$/L is optimal, with each unit of platelets raising the count by 10,000 to $20,000 \times 10^9$/L. Patients with osteogenesis imperfecta who have decreased factor VIII production and deficiencies of other clotting factors will benefit from the preoperative administration of fresh frozen plasma. Fresh frozen plasma contains all plasma proteins as well as all clotting factors, each of which will be increased by approximately 2% to 3% per unit administered.[12] If specific factor concentrates are unavailable, factor VIII deficiencies may also be corrected with the administration of cryoprecipitate. This blood product is prepared from plasma and contains fibrinogen, von Willebrand factor, factor VIII, factor XIII, and fibronectin. The usual adult dose for correcting factor VIII deficiencies is 6 to 12 bags, each containing approximately 150 U of the factor.[18] Careful monitoring, early recognition, and ag-

gressive treatment will help to avoid fatality in the patient with osteogenesis imperfecta who presents with inadequacies in coagulation.

Hyperthermia

Perhaps the most worrisome anesthetic implication of osteogenesis imperfecta is the possibility for malignant hyperthermia. Malignant hyperthermia is an inherited condition presenting during general anesthesia as a progressive increase in body temperature, associated with hypercapnia, metabolic acidosis, and usually muscle rigidity.[19] Many cases of hyperthermia in osteogenesis imperfecta are not of the malignant type but the result of a hypermetabolic state of which the pathogenesis is unknown.[20] It has been suggested that hyperthermia in patients with osteogenesis imperfecta is the result of either abnormal central nervous center temperature regulation or abnormal cellular energy metabolism.[11] Children with osteogenesis imperfecta have been reported to have a multitude of metabolic defects, including a higher-than-normal body temperature, heart rate, and respiratory rate. They may also have an increase in basal creatine kinase and pyrophosphate concentrations, both of which have been proposed as predictors for malignant hyperthermia.[19] At least 50% of patients with osteogenesis imperfecta also have an elevated serum thyroxine level, leading to elevated oxygen consumption and heat production relative to their body weight. Excessive thyroid hormone will uncouple oxidative phosphorylation, leading to energy required for adenosine triphosphate to be lost as heat. This energy waste may serve as an explanation for the cause of hyperthermia.[6]

Patients with osteogenesis imperfecta, specifically type III, often complain of heat intolerance, and during anesthesia, these patients have been described as developing signs and symptoms similar to those of malignant hyperthermia. The triggering agents for malignant hyperthermia have been found to consist of both potent inhalational agents and the depolarizing muscle relaxant succinycholine.[20] Anticholinergics have also been shown to produce hyperthermia and should be avoided if a history of malignant hyperthermia is suspected.[9] Malignant hyperthermia–triggering agents cause an increase in calcium release in the muscles of susceptible individuals. This high concentration of calcium causes contracture of skeletal muscles and an increase in the metabolism of glucose, leading to both respiratory and metabolic acidosis as well as an increase in temperature.[20]

Important preoperative assessments should include obtaining a careful personal and family history and laboratory measurements of serum creatine kinase and pyrophosphate concentrations. Avoidance of common malignant hyperthermia–triggering agents should be considered, and as always, temperature monitoring and provision of necessary drugs and cooling devices should be immediately available.[19] In patients with osteogenesis imperfecta, the presence of hyperthermia under anesthesia can usually be controlled with cooling, supplemental oxygen, sodium bicarbonate, cardiovascular stimulants, and intravenous dantrolene.[6]

Basilar Invagination

Perhaps the rarest complication associated with osteogenesis imperfecta is the potential for basilar invagination. The disorder is commonly described as the upward displacement of basilar and condylar portions of the occipital bone, causing an infolding of the foramen magnum and leading to a translocation of the upper cervical spine into the brainstem. Craniocervical malformation is usually suspected before neurologic studies owing to a combination of direct compression of the brainstem and vascular disruption. Symptoms can be progressive, and the disorder may prove fatal if left undiagnosed. Structures involved include the upper cervical cord, medulla, pons, midbrain, cerebellum, and the vertebrobasilar system.[21]

Neurologic symptoms vary greatly due to the close proximity of the neuroanatomical structures and the disruption of both blood flow and cerebrospinal fluid. Symptoms include but are not limited to occipital headaches precipitated by cough and laughter, trigeminal neuralgia, vertigo precipitated by movement of the head and neck, weakness in the upper and lower extremities, sleep apnea, seizures, and nystagmus. Symptoms may be acute due to trauma but likely will develop over time; headache is the most common presenting complaint. Cranial nerves are often affected, with trigeminal neuralgia occurring more frequently, presumably because of its large size and extension of the nerve into the pontomedullary region. The facial nerve is less commonly involved and is probably the result of posterior circulation abnormalities.[21]

In patients with osteogenesis imperfecta, invagination occurs as the weight of the cranium progressively deforms the soft skull base; this allows the soft bone to slide over the cervical spine and leads to extension into the foramen magnum. It occurs in approximately 25% of patients with osteogenesis imperfecta and should always be considered in adults with osteogenesis imperfecta who report new onset of localizing neurologic signs or symptoms.[21] These patients are at risk for brainstem compression leading to severe neurologic changes, respiratory distress, and even sudden death.[10]

Anesthetizing a patient with brainstem compression is taxing, especially if respiratory compromise is already advanced and neurologic pathways are disrupted. Translocation of the cervical spine will distort the airway, shorten the chin to chest distance, and limit neck movement, making an already difficult intubation even more challenging.[10] Overextension of the neck can lead to vertebral

Concern	Management
Bony fractures	Careful positioning needed Avoid NIBP cuff, tourniquets, and depolarizing fasciculations
Airway management	Airway is often distorted; avoid overextension of cervical spine Teeth are easily chipped or dislodged; consider less invasive manipulation techniques (LMA, FOB)
Pulmonary mechanics	Restrictive disease and V/Q mismatch lead to hypoxemia; may require high Fio_2 or PEEP
Cardiac anomalies	Increased ventricular compliance; high incidence of aortic dissection, left ventricular rupture, and valve incompetence Check electrocardiogram and echocardiogram
Bleeding diathesis	Tendencies to bleed and bruise; impaired platelet plug formation Administer platelets and fresh frozen plasma as needed
Malignant hyperthermia	Risk increased in osteogenesis imperfecta; creatine kinase and pyrophosphate levels are predictors of malignant hyperthermia Use standard precautions against malignant hyperthermia
Basilar invagination	Patients are at risk for brainstem compression; avoid vertebral artery compression and overextension of the neck Compromised cerebrospinal fluid drainage can lead to increased intracranial pressure; consider LMA or FOB

Table. Perioperative Issues With Osteogenesis Imperfecta

NIBP indicates noninvasive blood pressure; LMA, laryngeal mask airway; FOB, fiberoptic intubation; V/Q, ventilation/perfusion; Fio_2, fraction of inspired oxygen; PEEP, positive end-expiratory pressure.

artery compression, resulting in cerebral ischemia.[22] Fiberoptic intubation or the use of an intubating laryngeal mask airway should again be considered.[10] Cerebrospinal fluid drainage may be compromised, leading to a diagnosis of hydrocephalus and increased intracranial pressure. In cases such as this, the use of the preferred induction agent for patients with osteogenesis imperfecta—ketamine—would be contraindicated.[21]

Summary

It is prudent to conclude that no matter how rare the anesthetic complication in osteogenesis imperfecta, these patients must be considered as potentially possessing any or all of these anesthetic obstacles (Table). Anesthetic management begins with an in-depth preoperative assessment of airway anatomy, coagulation studies, heart imaging studies, and pulmonary function testing. Testing for malignant hyperthermia and basilar invagination should be performed if indicated based on an in-depth history and physical findings. Cardiopulmonary status should be optimized before surgery and proper materials for aggressive management of any complications should be immediately available during the intraoperative course.

The anesthesia provider and the surgeon as well as any recovery room staff should continue to monitor the patient closely during the postoperative period. Proper planning should prevent poor performance and maximize the patient's opportunity for a favorable outcome.

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