AANA Journal Course 2

Update for nurse anesthetists

6 CE Credits*

An overview of multiple sclerosis and implications for anesthesia

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Multiple sclerosis is the most common demyelinating, chronic disease of the central nervous system diagnosed in and affecting young adults. This disease can significantly alter the life of affected people, and there is no known cure. It is important to understand and review this disease process because anesthesia providers are likely to encounter patients with multiple sclerosis in their practice. The purpose of this AANA Journal course is to present an overview of multiple sclerosis. The pathophysiologic features, symptoms, manifestations, diagnosis, and pharmacologic treatment are discussed, with a focus on the implications of multiple sclerosis for general and regional anesthesia.

Key words: **Anesthesia, general anesthesia, multiple sclerosis, regional anesthesia.**

Objectives

At the completion of this course, the reader should be able to:

1. Explain the pathophysiologic features and different types of multiple sclerosis.
2. List symptoms and clinical manifestations of multiple sclerosis using a systems approach.
3. Describe the various pharmacologic treatments available to treat multiple sclerosis.
4. Discuss principles of anesthetic management and implications for patients with multiple sclerosis.
5. Discuss issues regarding the use of general vs regional anesthesia in patients with multiple sclerosis.

Introduction

Multiple sclerosis (MS) is a term that means many scars and is the name given to a disease process defined as a demyelinating, autoimmune disease that attacks the white matter of the central nervous system (CNS). Patients affected are primarily between the ages of 20 and 40 years. Causative factors of MS are unknown but may include genetic factors and environmental, viral, and infectious exposures. Women are affected more than men by a rate of 2:1.[1,2] Multiple sclerosis affects whites more than African Americans, Hispanics, Asians, and other ethnic groups. In one study of 747 patients with MS, 86% were white and 13% were African American.[3] This study found that African Americans experienced earlier system involvement, increased severity of the disease process, and higher rates of disability than did whites.[3]

Geographically, MS primarily affects people who live in countries 40° north or south of the equator with a lower incidence in the warm climates closer to the equator. Worldwide, 1 million people are affected by MS. In the United States, at least 350,000 people have MS, with an estimated 10,000 new cases diagnosed every year.[4] Once diagnosed, MS can affect people throughout the life span, and there is no known cure. Therefore, nurse anesthetists may encounter patients requiring surgery and anesthesia who have MS. This article discusses the pathophysiologic features, symptoms and manifestations, diagnosis, pharmacologic treatment, and anesthetic implications, including general and regional anesthesia.

Pathophysiologic features

Multiple sclerosis is characterized by inflammation, demyelination, and axonal damage in the brain and spinal cord, with a loss of myelin that covers the axons followed by the formation of scar tissue or plaques.[2] Multiple sclerosis begins when immune-mediated inflammation activates T cells, along with other

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immune mediators, to cross the blood-brain barrier into the CNS and attack the oligodendrocytes. These cells produce myelin, the insulation surrounding the axons, which protects the axons and nerve fibers and enables nerve impulse transmission in the CNS. When oligodendrocytes are attacked, demyelination results and myelin is replaced by scar tissue, forming plaques throughout the CNS. With this damage to the myelin sheath, the ability to transmit and conduct nerve impulses along the spinal cord to the brain is disrupted. Nerve impulse transmissions become slowed, producing diminished function of the CNS, including muscle weakness, loss of coordination and balance, fatigue, cognitive impairments, and other symptoms characteristic of MS.

During periods of remission, the myelin can regenerate and symptoms can resolve, but myelin cannot be repaired completely. With progression of the disease, the myelin is destroyed, nerve impulse transmission becomes further slowed or absent, and symptoms worsen.

The disease is characterized by unpredictable remissions occurring during several years. There are 6 types of MS: relapsing-remitting (RRMS), secondary-progressive, primary-progressive, benign, progressive-relapsing, and malignant or fulminant (Table 1). The most common of these types is RRMS, affecting 80% of people with a diagnosis of MS. The RRMS type is characterized by exacerbations or attacks lasting 1 to 3 months, followed by remissions lasting as long as a year.[4]

The second phase of RRMS is secondary-progressive MS, which can affect 90% of people with RRMS within 25 years of diagnosis. In secondary-progressive MS, symptoms worsen and may occur with or without relapses. The other 20% of people with MS have primary-progressive MS, characterized as the steady accumulation of neurological problems without relapses and remissions.[4]

The other types of MS are less common. Benign MS is characterized by few attacks and little or no changes after 20 years, progressive-relapsing MS is characterized by a progressive course of disease with relapses, and malignant or fulminant MS is characterized by a rapid and progressive disease course.[4]

Causative factors for MS are unknown. Genetic factors and environmental, viral, and infectious exposures may contribute to the development of MS. The risk of developing MS is, on average, 0.1% or 1 in 1,000 people. The risk is increased to 3% to 4% if a first-degree relative has MS. In cases of identical twins in which one twin has MS, the risk is increased to 31% for the other twin compared with 5% in nonidentical twins.[4]

Cigarette smoking is associated with the development of MS, especially among women. An analysis of 2 large, prospective cohort studies, the Nurses Health Study and Nurses Health Study II, showed a 60% greater incidence of MS in current smokers compared with nonsmokers and a higher incidence of MS associated with an increased pack-year history of smoking.[5] Prospective studies can infer causality by demonstrating that risk factors such as smoking occurred before the diagnosis of MS and can be correlated with the diagnosis of MS.

The link between smoking and MS is unclear, but smoking has been associated with an increased risk for developing certain types of autoimmune diseases, including rheumatoid arthritis, systemic lupus erythematosus, Graves disease, and Crohn disease. Nicotine, one component of cigarette smoke, has been shown to have direct effects on the blood-brain barrier in rats by increasing microvascular blood flow in the brain and raising the influx of permeable solutes across this bar-

Table 1. The six types of multiple sclerosis (MS)

Type	Characteristics
RRMS	The most common type affecting 80% of people given a diagnosis of MS
	Exacerbations or attacks last 1 to 3 months followed by recovery and remission of symptoms
Secondary-progressive MS	The second phase of RRMS
	Worsening of symptoms that may occur with or without relapses
Primary-progressive MS	Affects the other 20% of people given a diagnosis of MS
	Steady, progressive accumulation of neurological problems without relapses and remissions
Benign MS	Few exacerbations or attacks with little to no change in 20 y
Progressive-remitting MS	Progressive course of disease with relapses
Malignant or fulminant MS	Rapid and progressive disease course

RRMS indicates relapsing-remitting MS.

rier. Such leakage across the blood-brain barrier may lead to the development of MS. Cyanide, another component of cigarette smoke, is known to cause demyelination in the CNS. Furthermore, smoking increases the incidence of developing respiratory infections, which also may increase the risk of developing MS.[5]

Another theory of the possible cause of MS involves slow-acting viruses. After exposure to viruses such as measles, herpes, human T-cell lymphoma, or Epstein-Barr, MS may develop in genetically susceptible people. Exacerbations of MS also can be triggered by viruses and infections, with urinary tract infections shown to be the most common illness to cause an exacerbation of MS.[4] It is important to remember that causes of MS are speculative. No exact trigger or cause of MS is known, and research is ongoing.

Symptoms and manifestations

Multiple sclerosis has multifocal involvement; symptoms and clinical manifestations of the disease reflect the various sites or parts of the CNS affected by demyelination (Table 2). Common symptoms include motor disorders; cerebellar signs; spasticity; fatigue; sensory disorders; pain; visual complaints; bladder, bowel, and sexual dysfunction; paroxysmal symptoms; mood disorders; cognitive impairment; and psychosocial symptoms.[6] Motor disorders can lead to weakness or paresis of the lower extremities that may progress to paralysis manifesting as gait dysfunction from cerebellar demyelination, muscle weakness, visual disturbances, and decreased proprioception. Cerebellar manifestations include ataxia, nystagmus, dysarthria, slurred speech, intention tremors, and vertigo. Spasticity is most common in the calf, thigh, back, and groin muscles. Fatigue can increase the severity and intensity of other symptoms, including motor weakness.[6]

Sensory disorders manifest as numbness and paresthesias in the face and extremities and impaired vibration, temperature, and depth perception. Pain can be chronic or acute, with acute pain manifesting as retro-orbital pain associated with optic neuritis, burning, itching, and aching associated with paroxysmal limb pain, trigeminal neuralgia, headache, and pain from spasticity.[6]

Visual complaints include manifestations such as the

Table 2. Symptoms and manifestations of multiple sclerosis by system

System	Symptoms and manifestations
Eye, ear, nose, throat	Visual disturbances, including temporary or unilateral loss of vision, diplopia, blurred vision, scotoma, red-green distortion, and Marcus Gunn pupil
	Optic neuritis manifesting as retro-orbital pain
Neurological	Cerebellar signs, including ataxia, nystagmus, dysarthria, slurred speech, intention tremors, and vertigo
	Sensory disorders, including numbness and paresthesias in the face and extremities
	Impaired vibration, temperature, and depth perception
	Paroxysmal symptoms, including localized or generalized seizures, trigeminal neuralgia, and painful spasms of the hands and feet
	Headaches
	Mood disorders manifesting as emotional lability, euphoria, or depression
	Cognitive impairment manifesting as difficulty concentrating, planning, or maintaining attention and deficits in judgment and problem solving
Renal	Bladder dysfunction manifesting as increased urinary urgency and frequency, nocturia, overflow incontinence, hesitancy, and a feeling the bladder has not emptied after voiding
Gastrointestinal	Bowel dysfunction manifesting as diarrhea, incontinence, and constipation
Musculoskeletal	Paroxysmal limb pain manifesting as burning, itching, and aching
	Spasticity, most common in the calf, thigh, back, and groin muscles, resulting in pain
	Motor disorders manifesting as gait dysfunction and muscle weakness of the lower extremities that may progress to paralysis
	Decreased proprioception
Other	Fatigue that increases the severity and intensity of other symptoms
	Sexual dysfunction manifesting in women as difficulty achieving orgasm and in men as erectile dysfunction and difficulty ejaculating
	Psychosocial symptoms manifesting as hopelessness, loss of control, fear, and uncertainty, which can lead to further deterioration of physical function

temporary unilateral loss of vision, diplopia, blurred vision, red-green distortion, scotoma or blind spots in the eyes, and Marcus Gunn pupil or pupillary dilation in response to light.[6]

Bladder dysfunction can include manifestations such as increased urinary urgency, frequency, nocturia, overflow incontinence, hesitancy, and the feeling the bladder has not emptied after voiding. There also is an increased risk of developing urinary tract infections related to MS. Bowel dysfunction manifests as diarrhea, incontinence, and constipation. Sexual dysfunction in women can include difficulty achieving orgasm and in men, erectile dysfunction and difficulty with ejaculation.[6]

Paroxysmal symptoms manifest as local or generalized seizures, trigeminal neuralgia, and painful spasms of the hands and feet. Mood disorders manifest as emotional lability, euphoria, and depression. Manifestations of cognitive impairment include difficulty concentrating, planning, and maintaining attention and deficits in judgment and problem-solving. Psychosocial symptoms can manifest as hopelessness, loss of control, fear, and uncertainty, all of which can lead to further deterioration of physical function.[6]

The symptoms and manifestations may develop during a period of a few days, stabilize for a few weeks, and then show improvement with exacerbations triggered by such things as infection, exhaustion or fatigue, heat, depression, and stress. Remyelination in the CNS is unlikely; therefore, remissions are attributed to correction of transient chemical and physiologic disturbances interfering with nerve conduction in the absence of complete demyelination.

In some patients, the disease is characterized by infrequent and mild symptoms, whereas severe disability from the variety of symptoms and clinical manifestations, including failed vision, ataxia, spastic skeletal muscle weakness, and urinary incontinence, can persist during periods of remissions in other patients. The onset of MS after age 35 years has been associated with slower disease progression.[2] With time and progression of MS, remissions become less complete and the disease becomes incapacitating, characterized by muscle weakness, with 50% of affected people requiring help walking within 15 years of diagnosis.[1]

Diagnosis

Multiple sclerosis cannot be diagnosed based solely on the presence of clinical symptoms and manifestations because numerous possibilities exist to explain these symptoms. A diagnosis of MS requires that all other possible causes of symptoms have been eliminated.[7] Therefore, only a presumptive diagnosis of MS can be made based on the presence of these clinical symptoms and manifestations. Confirmation of the presence of MS requires diagnostic testing such as magnetic resonance imaging (MRI), computed tomography scanning, visual evoked potentials, somatosensory evoked potentials, or lumbar puncture. The cerebrospinal fluid from a lumbar puncture or spinal tap can be analyzed for the presence of elevated immunoglobulin G and the presence of a specific immunoglobulin G that appears as oligoclonal bands in the cerebrospinal fluid.[7] Visual and somatosensory evoked potential testing look for the prolonged latency of evoked potentials resulting from demyelination and slowed nerve conduction.

The presence of signal changes from plaques in the white matter can be shown on cranial MRI, which is used to confirm the presence and diagnosis of MS because of its sensitivity for showing the demyelinative plaques associated with MS.[2] The most common diagnostic test is an MRI of the brain, which has been shown to be abnormal in 90% of people with a definite diagnosis of MS.[7]

Pharmacologic treatment

As previously mentioned, there is no known cure for multiple sclerosis. Various pharmacologic treatments are available to treat MS (Table 3), including corticosteroids, interferon beta-1a (Avonex and Rebif), interferon beta-1b (Betaseron), glatiramer acetate (Copaxone), and mitoxantrone (Novantrone).[4]

Corticosteroids are considered a principal treatment for relapses of MS because of their anti-inflammatory effects that restore the blood-brain barrier, decrease edema, and improve axonal nerve conduction. The use of intravenous corticosteroids to shorten the duration of attacks of MS is well reported in the literature. For example, one case study described a patient with MS in whom acute respiratory failure developed as a result of an upper respiratory tract infection; mechanical ventilation was required.[8] The patient needed mechanical ventilation for 2 weeks and, after receiving an intravenous course of methylprednisolone (1 g/d for 3 days), was weaned successfully from the ventilator. The authors concluded that corticosteroids may not be used in the majority of cases due to concerns over immunosuppression and the risk of opportunistic infections.[8] Side effects of long-term use of corticosteroids include hypertension, diabetes, osteoporosis, cataracts, and ulcers, which may outweigh the possible benefits of long-term corticosteroid use.[9]

Immunotherapy is used to treat MS and decreases the occurrences of episodes of MS by reducing inflammation and inhibiting the autoimmune response. Interferon beta-1a (Avonex), interferon beta-1b (Betaseron), and glatiramer acetate (Copaxone) are called the *ABCs* of immunotherapy and may reduce the frequency of relapses of MS by as much as 30%.[1]

Table 3. Pharmacologic treatment of multiple sclerosis (MS)

Classification and drug(s)	Comments
Corticosteroids	Principal treatment for relapses used to shorten duration of attacks of MS
Methylprednisolone (Solu-Medrol)	Anti-inflammatory effects restore the blood-brain barrier, decrease edema, and improve axonal nerve conduction
	Concerns involve immunosuppression and risk of opportunistic infections
	Side effects of long-term use include hypertension, diabetes, osteoporosis, cataracts, and ulcers
	Side effects may outweigh possible benefits of long-term use
Immunotherapy	Reduces inflammation and inhibits the immune response; may reduce frequency of relapses by as much as 30%
Interferon beta-1a (Avonex and Rebif)	Avonex: immune-system modulator with antiviral properties; administered IM; side effects include flulike symptoms and headaches
	Rebif: an immune-system modulator with antiretroviral properties; administered SQ; side effects include flulike symptoms, reactions around the injection site, and abnormal blood cell count and liver function test results
Interferon beta-1b (Betaseron)	Interferon beta-1b: immune-system modulator with antiviral properties; administered SQ; side effects include flulike symptoms, reactions around the injection site, and abnormal blood cell count and liver function test results
Glatiramer acetate (Copaxone)	Glatiramer: immune-system modulator blocking the destruction of myelin; administered SQ; side effects include reactions around the injection site; can also cause a systemic reaction 5-15 min after injection manifesting as anxiety, flushing, chest tightness, palpitations, and shortness of breath; symptoms last a few minutes and do not require treatment
Immunosuppressants	Use considered when there is no response to treatment with immunotherapy drugs
Azathioprine (Imuran)	Purine analogue depressing cell-mediated and humoral immunity; decreases rate of relapses but does not affect progression of MS
Antineoplastics	
Mitoxantrone (Novantrone)	An immune-system modulator and suppressor; administered as an IV infusion every 3 mo; side effects include nausea, thinning hair, loss of menstrual periods, bladder infections, mouth sores, and bluish discoloration of urine and sclera
Methotrexate (Amethopterin)	Risk of cardiotoxic effects requiring routine cardiac testing, WBC counts, and liver function tests; may not be used longer than 2-3 years due to cardiotoxic effects
Cyclophosphamide (Cytoxan)	Folic acid antagonist and antineoplastic with anti-inflammatory effects inhibiting cell-mediated and humoral immunity; attempts to slow progression of MS
Other	
Carbamazepine (Tegretol)	Used to treat paroxysmal symptoms
Tricyclic antidepressants	Used to treat depression
Bethanechol	Used to treat urinary retention
Baclofen, benzodiazepines, and dantrolene	Used to treat muscle spasticity
Anticholinergics	Used to treat urinary incontinence

IM indicates intramuscularly; SQ, subcutaneously; IV, intravenous; and WBC, white blood cell.

Interferon beta-1a is an immune-system modulator. The commercial preparation Avonex has antiviral properties, is administered by intramuscular injection, and is associated with side effects such as flulike symptoms and headaches. Rebif, another interferon beta-1a preparation, has antiretroviral properties. It is administered subcutaneously; side effects may include flulike symptoms, reactions around the injection site, and abnormalities in blood cell counts and liver function test results.

Interferon beta-1b also is an immune-system modulator with antiviral properties. It is administered by subcutaneous injection and is associated with side effects such as flulike symptoms, reactions around the injection site, and abnormalities in blood cell counts and liver function test results. The side effects for the interferon preparations usually are temporary and can be prevented or managed.

Glatiramer acetate is an immune-system modulator that blocks the destruction of myelin. It is administered by subcutaneous injection and is associated with reactions around the injection site. Glatiramer also can cause a systemic reaction 5 to 15 minutes after injection in 10% of patients that manifests as anxiety, flushing, chest tightness, dizziness, palpitations, and shortness of breath. These symptoms last for a few minutes, do not require treatment, and have no long-term effects.[4]

Mitoxantrone is an antineoplastic agent that acts as an immune-system modulator and suppressor. Mitoxantrone is administered as an intravenous infusion every 3 months and usually is well tolerated. Side effects include nausea, thinning hair, loss of menstrual periods, bladder infections, mouth sores, and bluish discoloration of the urine and the sclera. Because of the risk of cardiotoxic effects, mitoxantrone often can be used for only 2 to 3 years. Routine cardiac testing and monitoring white blood cell counts and liver function test results are required.[4]

Other pharmacologic therapy includes immunosuppressant and antineoplastic drugs. Azathioprine (Imuran) is a purine analogue that depresses cell-mediated and humoral immunity. The use of azathioprine is considered when there is no response to treatment with the interferons or glatiramer. It is used to decrease the rate of relapses of MS but does not affect the progression of MS. Methotrexate is a folic acid antagonist and antineoplastic drug with anti-inflammatory effects that inhibits cell-mediated and humoral immunity.[2] Cyclophosphamide (Cytoxan) is another antineoplastic agent used to attempt to slow the progression of MS.[1]

The symptoms and manifestations of MS can be treated with pharmacologic therapy including carbamazepine (Tegretol) for paroxysmal symptoms; tricyclic antidepressants to treat depression; baclofen, benzodiazepines, or dantrolene to treat muscle spasticity; bethanechol to treat urinary retention; and anticholinergic drugs to treat urinary incontinence.[1,10]

Surgery, anesthesia, and MS

Controversy exists about the effects of surgery and anesthesia on MS. The National Multiple Sclerosis Society (NMSS) states that the stress of surgery will not exacerbate symptoms of MS. Instead, it is the complications of surgery such as infection and fever that can exacerbate the symptoms.[11]

The impact of surgery and anesthesia on MS is related to the severity of the disease process. The risk of elective surgery in the presence of MS is the same as that in the general population for young and otherwise healthy adults, and in these patients, a diagnosis of MS is not an absolute contraindication for surgery. However, considerations need to be made for patients who are severely disabled or who have respiratory problems as a result of MS. Patients with muscle weakness or who are confined to bed may have difficulty recovering from surgery and may require physical therapy to recover. In patients with respiratory problems, the nature of the surgery, including the potential need for prolonged respiratory support and mechanical ventilation, should be considered.[11] Patients should be informed in advance of these possibilities so they can decide whether to proceed with surgery.

MS is most prevalent in women of childbearing age.[12] Therefore, nurse anesthetists may encounter patients with MS who require surgery and anesthesia related to pregnancy and childbirth. A prospective study of 254 women with MS during 269 pregnancies found that the frequency of relapses decreased during pregnancy, especially during the third trimester, and relapses increased during the first 3 months of the postpartum period compared with the year preceding pregnancy. One possible explanation for these findings is that MS is a T cell–mediated autoimmune disease, as discussed, and a normal pregnancy is associated with a shift toward humoral immunity (B-cell) and away from cellular-mediated (T cell) immunity.[13]

Surgery and anesthesia also may be required for orthopedic, urologic, and neurological complications.[10] Thus, it is necessary to discuss management of anesthesia as it relates to patients with MS.

Anesthetic management and implications

Anesthetic management of and implications for patients with MS should consider the possibility of exacerbating symptoms of MS through surgical complications such as infection and fever. An important consideration is monitoring for increases in body temperature. An increase in body temperature of as little as 1°C can result in exacerbation of MS symptoms.

Increases in temperature block the conduction of demyelinated nerves, lead to the deterioration of nerve tissue at the sites of demyelination, and are more likely than the drugs used for anesthesia to result in postoperative exacerbation of symptoms of MS.[2]

In a study focusing on cooling in patients with MS, participants reported less fatigue and improvements in the quality of life as a result of reductions in core body temperature achieved by wearing a specially designed cooling suit.[14] The core body temperature of study participants decreased from 0.1°C to 1.0°C. Even though the sample included only 8 patients with various forms and degrees of MS, all participants reported positive outcomes, with feelings of comfort and less fatigue reported.[14] Fatigue was measured quantitatively using the Fatigue Impact Scale and severity graded according to a visual analogue scale. Qualitative data also were collected through interviews using open-ended questions that asked study participants to describe in-depth how they experienced fatigue. Participants also were asked about their experiences and conditions before and after using the cooling suit and about activities, advantages, and disadvantages during and after use of the cooling suit.[14]

Before surgery, patients with MS should receive a thorough preoperative evaluation, including assessment of the neurological and respiratory systems. A respiratory system evaluation is especially important because respiratory insufficiency frequently is a major cause of death in the presence of a neuromuscular disorder such as MS.[10] All findings should be documented because they may be useful in the detection of postoperative exacerbations of MS.

Following surgery, a postoperative neurological examination also should be performed, with new or exacerbated symptoms documented and appropriate follow-up care provided. Furthermore, if the patient was treated with corticosteroids preoperatively, intravenous supplementation is indicated.[15]

General vs regional anesthesia

General and regional anesthesia have been used in patients with MS. General anesthesia most often is chosen for use with patients who have MS.[2] There are no unique interactions between MS and the drugs used for general anesthesia, and no evidence supports recommendations for specific inhaled or intravenous anesthetic drugs.[2] However, there are possibilities of exaggerated potassium release following succinylcholine administration and prolonged responses and resistance to the effects of nondepolarizing muscle relaxants in patients with MS.[2] Patients with MS who are severely debilitated with neurological deficits and muscle atrophy are at risk for hyperkalemia following succinylcholine administration, whereas, it may be used safely in patients who are in remission or have mild symptoms.[2,15]

Prolonged responses to the paralytic effects of nondepolarizing muscle relaxants are consistent with the existing degree of skeletal muscle weakness and decreases in skeletal muscle mass. Resistance to the effects of nondepolarizing muscle relaxants also is possible and has been reported, possibly related to the proliferation of extrajunctional cholinergic receptors in upper motor neuron lesions.[2] Therefore, care must be individualized, with a plan of care made based on the extent of the disease process.

Disagreement exists regarding the use of regional anesthesia. The NMSS asserts that there is no reason for patients with MS to avoid the use of local anesthetics. In a study reported by the NMSS, of 98 patients with MS who received a total of more than 1,000 doses of local anesthetics, only 4 cases of exacerbations of MS were found following the administration of local anesthetics.[16]

With the young age and female predominance for MS, concerns about the use of regional anesthesia for labor and delivery arise. Again, MS is most prevalent in women of childbearing age compared with any other group.[12] In a study reported by the NMSS comparing pregnant women with MS who received or did not receive an epidural for labor and delivery, there were no differences in the number of relapses of MS following anesthesia between the 2 groups.[16]

A case study described the successful use of epidural anesthesia for a cesarean section in a patient with von Hippel-Lindau disease and MS.[17] The patient did not experience any neurological complications or exacerbations of MS following the use of epidural anesthesia. The authors of the case study concluded that the choice of anesthetic technique must be made on an individualized basis, including an evaluation of the extent of the disease process and consideration as to the surgical procedure, circumstances surrounding the surgery, and desires of the patient.[17]

It has been reported that the use of spinal anesthesia has resulted in exacerbations of MS, whereas epidural anesthesia and peripheral nerve blocks have not been implicated in postoperative exacerbations of MS.[2] The explanation for this is unknown, but the demyelination that occurs with MS and the resulting lack of a protective nerve sheath around the spinal cord may increase the risk of neurotoxic effects from local anesthetics.

Epidural anesthesia results in a lower concentration of local anesthetic in the white matter compared with spinal anesthesia, thus decreasing this risk.[2,15] This advantage of epidural anesthesia may decrease with repeated and continuous epidural administrations that may be required for the management of

labor and delivery in a pregnant patient with MS.[15] Overall, epidural anesthesia seems to be well tolerated in patients with MS, but spinal anesthesia is not always recommended due to concerns by some neurologists of a greater risk of postoperative complications.[16] Despite the differences of opinion about the safety and use of regional anesthesia in patients with MS, it is important to inform patients about the possibility of exacerbations of symptoms resulting from regional anesthesia and obtain informed consent before administration of regional anesthesia.

Summary

Perhaps the most important point to remember about MS is that the disease process involves a variety of symptoms and different levels of severity. Multiple sclerosis is unpredictable, as are the effects of anesthesia in patients with MS. An overview of MS has been provided for anesthesia providers for a better understanding of the disease process, including an explanation of the pathophysiologic features, symptoms and manifestations, diagnosis, pharmacologic treatments, risks and potential effects of surgery, and the use of general vs regional anesthesia. Patients should be informed and understand that exacerbations of MS are possible following surgery and anesthesia.

Patients should be informed and receive a thorough explanation of the benefits, risks, and potential complications of general and regional anesthesia based on the extent of the disease process. Preoperative and postoperative neurological and respiratory assessment findings should be documented to provide a basis of comparison for the effects of surgery and anesthesia on MS. It is important to review pharmacologic treatments used by patients with MS and provide supplementation as needed. Anesthesia providers also should continuously monitor body temperature and avoid hyperthermia in patients with MS. Individualized care of patients with MS is required to provide safe anesthetic management and to promote optimal recovery from surgery and anesthesia with minimal effects and complications in the postoperative period.

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