Demyelinating Disorders
Scott E. Rudkin

As in men and more frequently afflicts Caucasian patients, but all races can be affected. Onset of disease usually occurs in people between 20 and 50 years of age, with a peak occurring in those 30 years of age. The prevalence of MS varies widely with location; the highest prevalence is found at higher latitudes. This geographic variation suggests that MS may in part be caused by the action of some environmental factor.

GBS has an incidence of 3 per 100,000 individuals, which makes it the most common cause of flaccid paralysis. It has a slight male preponderance. The age at onset is bimodal, with the elderly and young adults most commonly being afflicted. There does not appear to be a geographic effect.

PATHOPHYSIOLOGY
The exact cause of demyelinating conditions is unknown, but an autoimmune or immune-mediated etiology is most likely. A molecular mimicry model postulates that an autoimmune attack on myelin is precipitated by an infectious organism that contains a protein similar to a myelin protein. The infection elicits an immune response by lymphocytes that recognize the cross-reactive protein; the activated lymphocytes then damage the myelin. The majority of the demyelinating disorders cause demyelination of the myelin sheath with relative sparing of the axon.

MS, transverse myelitis, and optic neuritis all affect the central nervous system. GBS, which primarily afflicts the peripheral nervous system, is now believed to cause both demyelination of the myelin sheath and axonal loss. Inflammatory lesions in peripheral nerve fibers cause focal demyelination with resultant slowing of conduction. Cranial nerves can also be affected.

PRESENTING SIGNS AND SYMPTOMS
In general, all the demyelinating disorders are characterized by an abrupt episode of loss of function. Depending on the area of the brain or nervous system affected, the patient may have sensory, motor, or autonomic symptoms.

MULTIPLE SCLEROSIS
In MS, the initial attack occurs abruptly (minutes to hours) from a single lesion. These attacks last between 6 and 8 weeks. Recovery between bouts of demyelination can be incomplete
or complete, depending on the amount of remyelination. Any part of the central nervous system can be affected. In decreasing order of frequency, the patient may exhibit optic neuritis, paresthesias in a limb, diplopia, trigeminal neuralgia, urinary retention, vertigo, or transverse myelitis. Depending on the spinal cord level, transverse myelitis can also cause loss of bladder or bowel function.

Ocular findings are the most common initial symptom. Optic neuritis is manifested as subacute monocular vision loss, although it can affect both eyes, and pain exacerbated with eye movement. It is the initial symptom in 25% and ultimately affects 50% of patients. The course usually progresses over a period of 2 weeks and may be include headache, retroorbital or periorbital pain, and alterations in color vision and visual fields. Slit-lamp examination may demonstrate cell and flare in the anterior chamber. The optic disk is frequently swollen on initial evaluation. In addition to optic neuritis, the patient may have an afferent pupillary defect (Marcus Gunn pupil, or decreased pupillary constriction on direct light confrontation but a normal consensual response) or intranuclear ophthalmoplegia, which is characterized by dysconjugate gaze with limited adduction of one eye and nystagmus in the abducting eye on lateral gaze as a result of a lesion involving the medial longitudinal fasciculus.

Sensory symptoms in patients with MS usually include numbness, tingling, pins and needles sensation, and tightness and coldness of the limbs and trunk. Radicular pain and itching may also occur. Symptoms result from involvement of the spinthalamic, posterior column, and dorsal nerve roots. The loss of vibration sense is often most prominent. Ataxia is uncommon at the onset of MS, but it occurs to some degree in most patients. Exacerbation of sensory symptoms can occur frequently and in different patterns with a patchy distribution. Patients may note either paresthesias or loss of sensation.

Sensitivity to heat is a characteristic complaint. Exercise, fever, a hot bath, or other activities that raise body temperature may result in the appearance of new symptoms or the recurrence of old symptoms. These events occur as a result of a temperature-induced conduction block across partially demyelinated fibers. Symptoms resolve when body temperature returns to normal.

In addition to loss of sensation, patients may also report “positive” symptoms. In addition to causing a slowing of conduction, demyelination may result in ectopic impulses with resultant abnormal signal transmission and abnormal mechanical sensitivity. These aberrant signals can produce the Lhermitte sign—an electric-like tingling or vibrating sensation in the torso or extremities with neck flexion. The patient may also report flashes of light (phosphenes) and paroxysmal symptom, including trigeminal neuralgia, ataxia, and dysarthria or painful tetanic posturing of the limbs triggered by touch or movement.

Motor weakness may occur in any pattern, including paraparesis, hemiparesis, and monoparesis; the lower extremities are usually affected more than the upper ones. Upper motor neuron dysfunction accompanied by spasticity and increased reflexes may also be present. Transverse myelitis with ascending weakness and numbness below the level of the lesion can occur as an initial symptom.

Autonomic symptoms are a frequent finding. Patients have difficulty with bladder function, including frequency and urgency, and may experience urge incontinence from bladder spasticity or hesitancy, retention, and overflow incontinence from poor signal conduction. Constipation is the most common bowel complaint. This autonomic dysfunction is frequently very embarrassing and distressful.

Normal disease progression is variable: MS may remain indolent or occur in a progressive manner, with steady accumulation of neurologic deficits in the absence of clearly defined exacerbations. Typically, acute exacerbations are followed by partial or complete resolution. New neurologic deficits develop over the course of several hours or days, remain stable for a few days to a few weeks, and then gradually improve.

With repeated exacerbations, permanent neurologic deficits tend to develop. Patients usually have symptom-free intervals of months or years between attacks. Patients who initially have relapsing-remitting disease (two or more episodes lasting more than 24 hours separated by more than 1 month) and who then enter a progressive phase are said to have secondary progressive disease (initial exacerbations and remissions followed by slow progression over at least a period of 6 months), whereas those whose symptoms are progressive from the onset are said to have primary progressive disease (slow or stepwise progression over a period of at least 6 months). About 15% of patients have primary progressive disease; of those who initially have relapsing-remitting disease, 30% to 50% will experience progressive symptoms during the first 10 years.

**OPTIC NEURITIS**

Optic neuritis may occur by itself or be the initial symptom of MS. The relationship of optic neuritis to MS is controversial. Some regard optic neuritis as a distinct entity, but others consider it part of the clinical continuum of MS. More than half of all patients with MS have optic neuritis at some time during the disease. Patients with completely normal results on magnetic resonance imaging (MRI) and comprehensive cerebrospinal fluid (CSF) evaluation seldom progress to MS. Optic neuritis is usually manifested initially as unilateral vision loss and retrobulbar pain with eye movement.

**TRANSVERSE MYELITIS**

Like optic neuritis, transverse myelitis may occur in isolation or be part of the symptomatology of MS. It is usually manifested as paraparesis, which is initially flaccid and then spastic; as loss of sensation at a sensory level on the trunk; and as bowel and bladder dysfunction. Back pain precedes the neurologic symptoms, and the sensory symptoms may begin distally and ascend. The thoracic cord is most often affected.

**GUILLAIN-BARRÉ SYNDROME**

Patients with GBS have weakness, paresthesias, and decreased or absent deep tendon reflexes. The distribution typically includes distal involvement with symmetric paresthesias (pins and needles). It spreads proximally, with weakness occurring a few days later; weakness also progresses to involve the upper extremities. Weakness is most prominent in the lower extremities and tends to involve the proximal muscles. It is usually first manifested as difficulty rising from a chair. The disease progresses from a few days to 3 to 4 weeks (progressive phase), followed by a plateau phase (days to weeks) and then by a recovery phase lasting from weeks to months. Weakness is varied but can be profound and involve the face and
respiratory muscles. A loss or decrease in deep tendon reflexes is frequently the initial finding, and these reflexes should be tested if GBS is suspected. Variants include the acute axonal form, which has a poor prognosis, and the Miller-Fisher syndrome, which is characterized by ataxia, ophthalmoplegia, and areflexia.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The differential diagnosis of demyelinating diseases includes conditions that cause progressive weakness. In due course the diagnosis of a demyelinating disorder becomes clear because few disorders relapse and remit over time. The role of the EP is to exclude other diseases that need immediate treatment.

Clinical factors that suggest a diagnosis other than MS (Table 98.1) include normal findings on neurologic examination, aphasia, predominance of pain, abrupt hemiparesis, quick (seconds or minutes) resolution of symptoms, and age younger than 10 or older than 50 years.

The diagnosis is usually made from the clinical signs and symptoms; MRI and other laboratory tests play a supporting role. Diagnosis requires evidence of dissemination of lesions in time and space and careful exclusion of other causes. The patient should have had more than one episode of neurologic dysfunction and evidence of white matter lesions in more than one part of the central nervous system or a change in a previous lesion. Although it is possible for a neurologist to diagnoses MS during an initial attack, provided that two clinical lesions are present and with corroborating laboratory testing, it is prudent for the EP to use a more conservative approach that requires two distinct attacks.

The EP should consider the diagnosis in a young adult with a history of two or more clinically distinct episodes of central nervous system dysfunction or the presence of highly suggestive symptoms (optic neuritis or intranuclear ophthalmoplegia). In 2005, the McDonald criteria revised the 2001 guidelines of the International Panel on MS to incorporate specific MRI findings and reaffirm the need for separation of clinical events and lesions in space and time. Diagnostic confidence is based on whether the criteria are fully met (diagnosis of MS), partially met (possible MS), or not met (not MS). Table 98.2 lists characteristic factors differentiating demyelinating disorders.

Differential diagnoses for optic neuritis include anterior ischemic optic neuropathy, which is usually painless and found in patients older than 50 years; hereditary diseases such as Leber hereditary optic neuropathy; and toxic or nutritional optic neuropathies.

Transverse myelitis may superficially resemble GBS, but its asymmetric involvement, definite sensory level, lack of upper extremity involvement, urinary incontinence symptoms, and CSF pleocytosis make the diagnosis of GBS less likely. The differential diagnosis includes other causes of acute myelopathy such as compression of the cord by an extradural structural lesion, spinal cord neoplasms, ischemia, and systemic lupus erythematosus.

Heavy metal poisoning can also mimic GBS, but it is usually preceded by a gastrointestinal phase with vomiting and diarrhea. As discussed previously, transverse myelitis can superficially resemble this syndrome.

**Table 98.1** Differential Diagnosis of Multiple Sclerosis

<table>
<thead>
<tr>
<th>DISEASE/SYSTEM</th>
<th>IMPORTANT FACTORS</th>
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<tbody>
<tr>
<td>Seizures, syncope, or dementia</td>
<td>Present diffusely or globally; MS is usually focal; consider Todd paralysis in seizure patients</td>
</tr>
<tr>
<td>SLE</td>
<td>Neurologic findings normally occur in patient with a known diagnosis of SLE</td>
</tr>
<tr>
<td>Sarcoid</td>
<td>CNS and pulmonary involvement normally occurs in patients with known disease</td>
</tr>
<tr>
<td>Lyme disease</td>
<td>Can mimic MS; look for tick exposure, travel history, and Lyme disease titers</td>
</tr>
<tr>
<td>CNS infection</td>
<td>Intracranial abscess, meningitis/encephalitis, or epidural abscess can produce focal findings</td>
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<tr>
<td>Bleeding (CNS)</td>
<td>Subdural, subarachnoid, intraparenchymal, or epidural hemorrhage can produce focal findings</td>
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<tr>
<td>Neoplasm</td>
<td>Usually progressive course with a more insidious onset</td>
</tr>
<tr>
<td>Vascular</td>
<td>Patients with thrombosis, embolism, or vasculitic conditions do not usually have resolution of symptoms</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Vitamin B12 deficiency, hypoglycemia, hyperglycemia (hyperosmolar)</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Migraine headache, postictal state, Bell palsy</td>
</tr>
<tr>
<td>Psychiatric</td>
<td>Diagnosis of exclusion; includes conversion reaction</td>
</tr>
</tbody>
</table>

**Table 98.2** Characteristic Factors Differentiating Demyelinating Disorders

- **Central nervous system (CNS) infection**: May involve CNS infection, meningitis, or subarachnoid hemorrhage.
- **Sarcoidosis**: Characterized by systemic involvement and extrapulmonary manifestations.
- **Systemic lupus erythematosus (SLE)**: Can mimic MS, but specific clinical and laboratory features help distinguish between the two.
- **Bleeding (CNS)**: Includes conditions such as subdural hematoma, subarachnoid hemorrhage, and intraparenchymal hemorrhage.
- **Neoplasm**: Often manifests with rapid progression and can be associated with symptoms such as headache, vomiting, and cognitive changes.
- **Vascular**: Includes conditions such as stroke, transient ischemic attack, and aneurysmal subarachnoid hemorrhage.
- **Metabolic**: Includes conditions such as diabetes mellitus, hyperthyroidism, and hypothyroidism.
- **Neurologic**: Includes conditions such as Guillain-Barré syndrome (GBS), chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), and motor neuron disease.
- **Psychiatric**: Includes conditions such as depression, anxiety, and posttraumatic stress disorder.

**DIAGNOSTIC TESTING**

Findings on routine laboratory tests are usually normal in patients with myelinating disorders, including MS, transverse myelitis, and GBS. Because the respiratory muscles are frequently affected in GBS, measurement of forced vital capacity (FVC) is essential to determine disposition.

Most cases of optic neuritis are diagnosed clinically. In questionable cases of optic neuritis, serum testing (erythrocyte sedimentation rate, angiotensin-converting enzyme, rapid plasma reagin, thyroid function testing, and antinuclear antibody studies) can be ordered to exclude other causes of optic neuritis.

**CEREBROSPINAL FLUID ANALYSIS**

With MS, CSF analysis (with electrophoresis) frequently demonstrates albuminocytologic dissociation with increased protein (usually less than 100 mg/dL) and a normal cell count.
However, 10% of patients have a normal CSF protein level. Mild mononuclear cell pleocytosis can be found during acute MS relapses, but total cell counts greater than 50/mm³ are uncommon. During acute attacks of MS, especially those involving the spinal cord and brainstem, CSF may contain measurable amounts of myelin basic protein. Oligoclonal bands or abnormal immunoglobulin synthesis is found in about 90% of patients with a diagnosis of MS. Though not specific to MS, these findings support the diagnosis of MS in equivocal cases.

CSF studies may demonstrate elevated protein levels and leukocytes in patients with transverse myelitis; such studies are not generally required for optic neuritis but are usually obtained to help in the diagnosis of MS.

NERVE CONDUCTION STUDIES
Electrodiagnostic testing may demonstrate conduction blocks, differential slowing, or focal slowing. Slowing of conduction over demyelinated segments of axons or over incompletely remyelinated pathways provides a useful marker for identifying additional subclinical lesions in the sensory pathways. Conduction can be measured along the visual, auditory, and somatosensory pathways with the use of summated cortical evoked responses. In these tests, a time-locked recording of

<table>
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<th>Table 98.2 Differentiating Between the Demyelinating Disorders</th>
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<td><strong>MULTIPLE SCLEROSIS</strong></td>
</tr>
<tr>
<td><strong>Epidemiology</strong></td>
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<tr>
<td><strong>Onset</strong></td>
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<td><strong>Duration</strong></td>
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<tr>
<td><strong>Symptoms</strong></td>
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<td><strong>Diagnosis, testing</strong></td>
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<td><strong>Treatment</strong></td>
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<tr>
<td><strong>Disposition</strong></td>
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*CSF, Cerebrospinal fluid; FVC, forced vital capacity; ICU, intensive care unit; IV, intravenous; MRI, magnetic resonance imaging.*
the electroencephalogram over the afferent cortex of interest is obtained after repeated visual, auditory, or sensory stimulation. If the demyelination is significant, conduction is delayed.

Electrodiagnostic testing may demonstrate abnormal motor and sensory conduction, but these signs can take a few weeks to develop.

**MAGNETIC RESONANCE IMAGING**

MRI permits the exclusion of many diseases that mimic MS and identifies certain lesions that are hyperintense on T2-weighted or proton density imaging and hypointense or isointense on T1-weighted imaging. Typical lesions are ovoid and periventricular, with the long axis perpendicular to the ventricle, but lesions may appear anywhere in the white matter. Although MRI is extremely sensitive in detecting white matter lesions in patients with MS, it is not very specific because many other diseases produce multiple white matter lesions. Useful features for increasing the predictive value of MRI for the diagnosis of MS include the presence of three or more white matter lesions, lesions that abut the body of the lateral ventricles, infratentorial lesions, lesions larger than 5 mm in diameter, and lesions that demonstrate gadolinium enhancement.

MRI is extremely useful for confirming the presence of an intramedullary lesion at the level in the spinal cord commensurate with the symptoms of transverse myelitis. The lesions are typically hyperintense on T2-weighted imaging; they involve the majority of the cross-sectional area of the cord over several segments and may be enhanced with contrast agents. The lesions may cause swelling of the spinal cord. Gadolinium-enhanced MRI may show abnormal enhancement of the nerve roots in the region of the conus medullaris and cauda equina.5

MRI is also useful for diagnosing optic neuritis and evaluating for concomitant MS. Gadolinium enhancement may demonstrate optic nerve involvement. In questionable cases, visually evoked potentials may demonstrate prolonged latency.

**TREATMENT**

For all demyelinating conditions, the EP’s goal is to reduce the current demyelinating episode while ensuring that the ABCs (airway, breathing, and circulation) are maintained. Because inflammation is a central component of demyelination, corticosteroids are frequently used; their effectiveness in patients with GBS and optic neuritis is questionable. Preventing and aggressively treating fever are important because an increased core temperature can worsen the demyelination.

**MULTIPLE SCLEROSIS**

Treatment of MS can be discussed in terms of the management of acute relapses, prevention of relapses as a modification of the disease process, and management of symptoms and fixed neurologic deficits. High-dose pulsed corticosteroid therapy is indicated for exacerbations of acute relapses that adversely affect the patient’s function. Intravenous (IV) methylprednisolone at doses of 0.5 to 1 g daily for 5 days reduces the maximal neurologic signs and hastens the resolution of associated fatigue. A controversial study by Sellebjerg et al. supports the use of oral methylprednisolone (500 mg daily for 5 days with a 10-day tapering period).4

In patients with relapsing–remitting MS, disease-modulating drugs reduce the frequency of attacks, the rate of increase in lesions seen on MRI, and the accumulation of disability. In patients with relapsing disease of mild to moderate severity, interferon beta-1b, given subcutaneously every other day, reduced the year-on-year relapse rate by one third and severe attacks by one half. The effect was maintained for up to 5 years.5,6 In another study, interferon β1b reduced MRI contrast-enhanced lesions by 1.6%, as opposed to a 15% increase in those receiving placebo. The number of enlarging or new lesions was also significantly reduced.7 Antibodies developed in 35% of patients taking interferon β1b, but there is a lack of consistent effect of antibodies on clinical outcome. Additionally, these antibody levels were found to have disappeared in the majority of patients after 8 years of treatment.8,9

Interferon β1a produced similar benefits when given intramuscularly three times per week, with a 17% reduction in the relapse rate. When compared with weekly, low-dose interferon β1a, high-dose interferon β1a given three times per week demonstrated a 32% relative reduction in steroid use to treat relapses.10

Glatiramer, a synthetic random compound composed of four amino acids, is found in myelin. Its exact mechanism is unknown, but it is believed that glatiramer acetate binds to the major histocompatibility complex class II antigen and induces organ-specific T helper type 2 cell responses, thus converting proinflammatory T cells to antiinflammatory agents.11 Treatment with glatiramer reduces the relapse rate by 30% and may delay disease progression.12

Natalizumab, a monoclonal antibody against α4 integrins, is effective for relapsing–remitting MS. The reduction in annualized relapse rates with natalizumab is similar to that with glatiramer or interferon β.13 However, it is not a first-line agent because it can cause progressive multifocal leukoencephalopathy in 0.1% of cases.14

Mitoxantrone is an anthracenedione, antineoplastic agent approved for the relapsing–remitting and progressive forms of MS. Cardiotoxicity, acute leukemia, and questionable efficacy limit it to treatment failures or cases of rapidly progressive MS.

Fingolimod, the first oral treatment option for MS approved by the Food and Drug Administration, is a sphingosine analogue that blocks lymphocyte release from lymph nodes through interaction with the sphingosine 1-phosphate receptor. Fingolimod can significantly reduce relapse rates when compared with interferon β1a. However, fingolimod is associated with life-threatening infections, bradycardia, atrioventricular block, tumor development, and macular edema.15 Specific therapies for relief of symptoms are provided in Table 98.3.

**TRANSVERSE MYELITIS**

Corticosteroids and plasma exchange may be beneficial in the treatment of transverse myelitis.16 In a small case series, patients treated with steroids were able to walk after a median time of 23 days versus 97 days for historical controls.17 Plasma exchange is often used for those with more severe disease (e.g., unable to walk) who fail to improve with IV steroid therapy.18 The prognosis for transverse myelitis is variable.
OPTIC NEURITIS

IV methylprednisolone (1 g/day for 3 days) followed by oral prednisone (1 mg/kg/day for 11 days with a 4-day taper) and interferon β1a (30 mcg intramuscularly once a week) for patients at high risk for MS based on MRI has been shown to hasten recovery of vision in patients with optic neuritis.\(^\text{19}\) However, this therapy shows little residual benefit at 1 year. Additionally, oral prednisone therapy alone was found to actually increase the recurrence rate.\(^\text{20}\) Regardless of therapy, most patients recover their vision within a month.

**GUILLAIN-BARRÉ SYNDROME**

Therapy is largely supportive. Most patients recover function if they survive the acute phase. The key therapeutic measure is ventilatory support. FVC should be measured in all patients in whom GBS is suspected. Patients with an FVC of less than 20 mL/kg should be admitted to the intensive care unit because of their high risk for ventilatory insufficiency. For those with FVC lower than 15 mL/kg, intubation is indicated. If intubation is necessary, succinylcholine should be avoided because it can cause hyperkalemia with resultant arrhythmia and hypotension. Dysautonomia can result in severe paroxysmal hypertension, orthostatic hypotension, and arrhythmias. Cardiovascular function should be monitored carefully. Plasmapheresis and IV gamma globulin therapy have both been shown to reduce recovery time by 50%. IV gamma globulin is less expensive and easier to administer, but the risk for viral transmission is greater. IV steroids are frequently administered, but their actual benefit is questionable.

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION**

All patients with a first episode of a demyelination disorder merit neurology consultation. Although outpatient therapy is possible for a subset of these patients, this decision is best left to the neurologist. As discussed previously, the typical course of MS is a relapsing-remitting pattern. During an acute exacerbation, the patient is frequently admitted for IV steroid therapy, but oral treatment is possible.

Hospital admission is required for GBS, and FVC measurement typically guides selection of the appropriate level of care. The majority of patients are hospitalized for a month or

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**Table 98.3 Symptomatic Treatment of Multiple Sclerosis**

<table>
<thead>
<tr>
<th>SYMPTOM</th>
<th>TREATMENT OPTIONS</th>
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<tbody>
<tr>
<td>Fatigue</td>
<td>Amantadine, pemoline, methylphenidate, modafinil, or selective serotonin reuptake inhibitor</td>
</tr>
<tr>
<td>Weakness</td>
<td>Steroids, potassium channel blocker</td>
</tr>
<tr>
<td>Loss of balance or coordination, tremor, ataxia</td>
<td>Clonazepam for tremor, steroids for balance</td>
</tr>
<tr>
<td>Sexual dysfunction</td>
<td>Sildenafil, intracavernosal prostaglandins (for erectile dysfunction)</td>
</tr>
<tr>
<td>Vertigo</td>
<td>Meclizine, prochlorperazine, diazepam, metoclopramide</td>
</tr>
<tr>
<td>Paroxysmal symptoms (itching, burning, twitching, Lhermitte sign)</td>
<td>Carbamazepine, phenytoin, tricyclic antidepressants, low-dose antipsychotics, gabapentin</td>
</tr>
<tr>
<td>Bladder urgency</td>
<td>Oxybutynin, tolterodine, imipramine, hyoscymamine, propantheline</td>
</tr>
<tr>
<td>Bladder dyssynergia</td>
<td>Phenoxybenzamine, clonidine, terazosin</td>
</tr>
<tr>
<td>Bladder retention</td>
<td>Intermittent catheterization, bethanechol</td>
</tr>
<tr>
<td>Spasticity (commonly increased tone in the lower extremities)</td>
<td>Baclofen, diazepam, tizanidine, clonazepam, clonidine (adjunctive to baclofen), dantrolene</td>
</tr>
<tr>
<td>Paresthesias</td>
<td>Amitriptyline, carbamazepine, gabapentin, corticosteroids if disabling</td>
</tr>
<tr>
<td>Optic atrophy, blurred vision, central scotomata</td>
<td>Intravenous methylprednisolone for acute optic neuritis</td>
</tr>
<tr>
<td>Intranuclear ophthalmpoplegia</td>
<td>Corticosteroids</td>
</tr>
<tr>
<td>Ataxia</td>
<td>Clonazepam, gabapentin</td>
</tr>
<tr>
<td>Paroxysmal pain</td>
<td>Carbamazepine, phenytoin, misoprostol (trigeminal neuralgia)</td>
</tr>
<tr>
<td>Dysesthetic pain</td>
<td>Amitriptyline, phenytoin, gabapentin, valproic acid, carbamazepine, baclofen</td>
</tr>
</tbody>
</table>

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**PATIENT TEACHING TIPS**


Patients are at risk for exacerbation of symptoms and progression of disease from an elevated core temperature. Aggressive fever control and determination of its cause are important.

There is no cure for demyelinating disorders, but current therapies can reduce their frequency and severity.

Patients with optic neuritis have a worse prognosis with oral steroid therapy. If steroids are used, they must be given intravenously.

Patients with symptoms of progressive weakness and sensory symptoms must be assessed rapidly for Guillain-Barré syndrome. With aggressive therapy, respiratory failure can be avoided.
longer, and with careful attention to respiratory function, mor-
tality is now less than 5%.

Patients should undergo ophthalmology and neurology
evaluation for manifestations of optic neuritis. If steroid
therapy is needed, hospital admission is necessary because
steroids may worsen the clinical outcome.

**SUGGESTED READINGS**

Panitch H, Goodin D, Francis G, et al. EVIDENCE (Evidence of Interferon
Dose-response: European North American Comparative Efficacy) Study Group and
the University of British Columbia MS/MRI Research Group. Benefits of
high-dose, high-frequency interferon beta-1a in relapsing-remitting multiple
sclerosis are sustained to 16 months: final comparative results of the EVIDENCE

**REFERENCES**

References can be found on Expert Consult @
REFERENCES