Myelitis roughly translates to "spinal cord inflammation", which can result from an infection or inflammatory cause. The term **transverse myelitis (TM)** has been adopted to describe inflammation of the spinal cord due to a misdirected immune response, resulting in varying degrees of weakness, sensory alterations, and autonomic dysfunction (the part of the nervous system that controls involuntary activity, such as the heart, breathing, the digestive system, and reflexes). Reports describing TM date back to the 1880s, but the first known use of “transverse myelitis” is in 1931, where it was used to describe inflammatory changes across the anatomical “transverse” plane seen on autopsy. The Transverse Myelitis Consortium Working Group provided a framework to delineate TM from non-inflammatory spinal cord disorders in 2002.¹
Epidemiology

TM has a conservatively estimated incidence of between 1 and 8 new cases per million per year, or approximately 1,400 new cases each year in the US.\(^2\)\(^-\)\(^6\) Although this disease affects people of all ages, there are peaks between the ages of 10 to 19 years and 30 to 39 years.\(^4\)\(^-\)\(^6\) In addition, approximately 25% of cases are in children.\(^7\) There is no gender or familial association with TM.\(^8\) In 75-90% of cases TM is monophasic, yet a small percentage experience recurrent disease, especially if there is a predisposing underlying illness.\(^9\)\(^-\)\(^11\)

Signs and Symptoms

The spinal cord carries motor nerve fibers to the limbs and trunk and sensory fibers from the body back to the brain. Inflammation within the spinal cord interrupts these pathways and causes the common presenting symptoms. TM generally presents with rapidly progressing muscle weakness or paralysis, beginning with the legs and potentially moving to the arms with varying degrees of severity.\(^12\)\(^-\)\(^14\) The arms are involved in a minority of cases and this is dependent upon the level of spinal cord involvement.\(^12\)\(^-\)\(^14\) Sensation is diminished below the level of spinal cord involvement in the majority of individuals.\(^12\) Pain (ascertained as appreciation of pinprick by the neurologist) and temperature sensation are generally diminished and appreciation of vibration (as caused by a tuning fork) and joint position sense may also be decreased. Many report a tight banding or girdle-like sensation around the trunk, and that area may be very sensitive to touch.\(^12\)

In most cases, a sensory level is documented, most commonly in the mid-thoracic region in adults or the cervical region in children.\(^7\) Pain in the back, extremities, or abdomen is also common while paresthesias (e.g., tingling, numbness, burning sensations) are typical in adults.\(^12\) Sexual dysfunction is also the result of sensory and autonomic involvement.\(^12,15\)\(^-\)\(^17\) Increased urinary urgency, bowel or bladder incontinence, difficulty or inability to void, and incomplete evacuation of bowel or constipation are other characteristic autonomic symptoms.\(^15\)\(^-\)\(^16\) Spasticity and fatigue are other symptoms common to transverse myelitis. Additionally, depression is often documented in TM patients and must be treated to prevent devastating consequences.

In some cases, symptoms progress over hours whereas in other instances, the presentation is over days. Neurologic function tends to decline during the 4-21-day acute phase, while 80% of cases reach their maximal deficit within 10 days of symptom onset.\(^1,18\) At its worst point, 50% of individuals have lost all movements of their legs, 80-94% experience numbness, paresthesias or banding or girdling, and almost all have some degree of bladder dysfunction.\(^12\)

Diagnosis

Diagnosis of TM is based on clinical features, imaging, and laboratory findings. Clinical characteristics of spinal cord injury are bilateral signs and/or symptoms of sensory, motor or autonomic dysfunction attributable to the spinal cord or a clearly defined sensory level. Evaluation for signs of inflammation to distinguish TM from
other spinal cord disorders typically involves a magnetic resonance imaging (MRI) of the spine with contrast and spinal fluid analysis (which requires a lumbar puncture). If a myelopathy is suspected based on history and physical examination, a gadolinium-enhanced magnetic resonance imaging (MRI) of the spinal cord is first obtained to assess if there is a compressive or inflammatory (gadolinium enhancing) lesion, as signs and symptoms can overlap. It is essential to rule out compressive myelopathy (compression of the spinal cord), which can be caused by a tumor, herniated disc, stenosis (a narrowed canal for the cord), hematoma or abscess. Identifying these disorders is critical since immobilization to prevent further injury and early surgery to remove the compression may sometimes reverse neurologic injury to the spinal cord.

Lumbar puncture is used to look for surrogate markers of inflammation in the cerebrospinal fluid (CSF). These include elevated white cell counts, elevated protein or other markers such as oligoclonal bands. While these markers are supportive of TM, it should be noted that they are not present in all individuals.

A series of blood tests are often recommended for patients with spinal cord disorder suspicious for TM. This commonly includes testing for aquaporin-4 and myelin oligodendrocyte glycoprotein (MOG) antibodies, in addition to tests associated with systemic autoimmune disorders such as systemic lupus erythematosus (SLE) and Sjögren's syndrome. If sarcoidosis is suspected based upon history and imaging characteristics, a CT of the chest may be considered to look for lung findings. Other common ordered tests include HIV, syphilis, vitamin B12 and copper levels.

An MRI of the brain is often performed to screen for lesions suggestive of a demyelinating disorder such as MS, NMOSD, or MOG antibody disease. In patients with imaging features of TM that are consistent with MS, brain imaging may be repeated over time to see if characteristic MS lesions develop.

If none of the tests are suggestive of a specific cause, a diagnosis of idiopathic transverse myelitis can be made.

Non-inflammatory myelopathies include those caused by arterial or venous ischemia (blockage), vascular malformations, radiation, fibrocartilaginous embolism or nutritional/metabolic causes. The work-up for suspected vascular spinal cord disorder may include angiograms of the spinal cord vessels and blood testing for a predisposition to developing blood clots.

Potential Causes

There are many disorders that can cause spinal cord inflammation, so TM should be thought of as a group of disorders, and not a single condition. “Disease-associated transverse myelitis” refers to TM that occurs in a patient with an identified autoimmune disorder. Disorders such as neuromyelitis optica spectrum disorder and multiple sclerosis are common causes of TM and may also cause inflammation in other parts of the nervous system. Autoimmune disorders targeting other organs, such
... Diagnosis

as Systemic Lupus Erythematosus,\textsuperscript{20-22} Sjogren’s syndrome,\textsuperscript{20,23,24} and sarcoidosis,\textsuperscript{25,26} are also known to cause TM.

Even after comprehensive medical evaluation, there remains a significant proportion of individuals with spinal cord inflammation that is without a clear identifiable cause. This is a condition called “idiopathic transverse myelitis”.\textsuperscript{19} When a healthcare provider diagnoses someone with ”transverse myelitis”, they are typically referring to idiopathic TM. Even among those labeled as having idiopathic TM, it is likely that there are multiple yet-to-be-identified causes for this inflammation.

TM may develop in the setting of a viral or bacterial infection, even if the symptoms from the infection are mild. Approximately 30-60\% of individuals with TM report a febrile illness (flu-like illness with fever) around the time of neurologic symptoms.\textsuperscript{4-6,14,27-29} Certain infections, such as polio, enteroviruses, and herpes zoster, can directly infect the cells of the spinal cord and cause injury.\textsuperscript{19} In other cases, damage may be mostly due to the immune system’s response to the infection.\textsuperscript{19}

Experts believe that in many cases, an infection triggers a misdirected immune response without directly infecting the spinal cord. This is supported by evidence that infections are an important factor in the development of autoimmune disorders of different types. Infections may trigger autoimmunity through a variety of ways, but one mechanism that has significant evidence is called molecular mimicry.\textsuperscript{13} This theory postulates that an infectious agent may share a molecule that resembles or mimics a molecule in the spinal cord. When the body mounts an immune response to the invading virus or bacterium, it also responds to the spinal cord molecule with which it shares structural characteristics, resulting in TM.\textsuperscript{13}

Although a causal relationship has not been established, TM has been rarely reported following influenza and Hepatitis B vaccinations.\textsuperscript{13,30-32} One theory suggests that it is possible that the vaccination may have excited the immune system, similar to an infection. It is critically important to bear in mind that extensive research has demonstrated that vaccinations are safe, and the potential link to TM may only be coincidental or at worst an exceptionally rare complication.

Myelitis related to cancer (called a paraneoplastic syndrome) is quite rare.\textsuperscript{15,33} When this occurs, the symptoms usually accrue over a much longer timeline (usually several months) than is typical of other causes of myelitis. This is thought to occur due to an immune response to proteins in the cancer cells that are also present on spinal cord cells, resulting in a misdirected immune response.

Vascular causes are noted because they present with the same problems as transverse myelitis.\textsuperscript{34} However, this is really a distinct problem primarily due to inadequate blood flow to the spinal cord instead of actual inflammation. The blood vessels to the spinal cord can close up with blood clots or atherosclerosis or burst and bleed. This is essentially a ”stroke” of the spinal cord.
Acute Treatments

It is extremely important to begin treatments as soon as possible after a rare neuroimmune diagnosis. Treatment in the acute or early stages involves quieting down the immune system as quickly as possible, before damage is done. Time is critical.

The acute therapies most frequently used to treat an inflammatory attack include: high dose intravenous steroids (methylprednisolone), Plasmapheresis (Plasma Exchange or PLEX), Immunoglobulin Therapy (IVIG), and cyclophosphamide. Intravenous Steroids

Intravenous steroid treatment is the first line of therapy often used in acute TM. Corticosteroids have multiple mechanisms of action including anti-inflammatory activity, immunosuppressive properties, and antiproliferative actions. Though there is no randomized double-blind placebo-controlled study that supports this approach, evidence from related disorders and clinical experience support this treatment. The standard of care includes intravenous methylprednisolone (30 mg/kg up to 1000 mg daily) or dexamethasone (120 to 200 mg daily for adults) for 3 to 5 days unless there are compelling reasons to avoid this therapy. The decision to offer continued steroids or to add a new treatment is often based on the clinical course and MRI appearance at the end of 5 days of steroids.

PLEX is often initiated in individuals with motor impairment or who show little clinical improvement after intravenous steroid treatment, but may also be initiated at first presentation for those with significant deficits. It is often given as five treatments, each with exchanges of 1.1 to 1.5 plasma volumes, every other day for 10 days. PLEX is believed to work in autoimmune CNS diseases through the removal of specific or nonspecific soluble factors likely to mediate, be responsible for, or contribute to inflammatory-mediated organ damage. PLEX has been shown to be effective in adults with TM and other inflammatory disorders of the CNS.

Other Immunomodulatory Treatment

If there is continued progression despite intravenous steroid therapy and PLEX, pulse dose intravenous cyclophosphamide (800–1200 mg/m2) is considered. Cyclophosphamide is known to have immunosuppressive properties. From the Johns Hopkins TM Center experience, it has been reported that PLEX provided an added benefit to steroids in patients who were not at a disability level of ASIA A and who did not have a history of autoimmune disease. For those who were classified at a disability level of ASIA A at their nadir, they showed a significant benefit when given combination therapy with steroids, PLEX and IV cyclophosphamide. Cyclophosphamide should be administered under the supervision of an experienced oncology team, and caregivers should monitor the patient carefully for hemorrhagic cystitis and cytopenias.

Another option for treating acute inflammation is intravenous immunoglobulin (IVIG). Immunoglobulin comes from pooled blood that is donated from thousands of healthy people. As the name suggests, IVIG is given intravenously. IVIG is generally well-tolerated. Potential adverse reactions are uncommon, but usually occur during or immediately after an infusion and include headache, nausea, muscle pain, fever, chills, chest discomfort, skin and anaphylactic reactions. Reactions after an infusion can be
... Acute Treatments

more serious and include migraine headaches, aseptic meningitis, renal impairment and blood clots. Like corticosteroids and PLEX, there are no data confirming the value of IVIG in the setting of acute events. While most studies support the use of corticosteroids and/or PLEX in acute demyelinating syndromes, IVIG can be considered in certain circumstances.

Recurrence of idiopathic TM is rare and warrants a comprehensive evaluation for known causes of recurrent myelitis. Consultation with a neuroimmunologist should strongly be considered when recurrence occurs, and immunosuppressive treatments may be recommended.

Prognosis and Management

The spectrum of recovery from TM is broad, and ranges from no improvement in symptoms to complete recovery. Some improvement in symptoms can be appreciated during acute treatment, but may not be appreciable until 1-3 months later. Historical data, not controlling for treatment, suggested that approximately 1/3 of individuals recover with little or only minor symptoms, 1/3 are left with a moderate degree of permanent disability and 1/3 have virtually no recovery and are left severely functionally disabled. These data, however, predate a number of more aggressive treatment protocols and are likely inaccurate. In present day experiences the outcomes seem to be better than this distribution, with most persons with TM showing good to fair recovery. Some studies have suggested that certain clinical features (rapid progression of symptoms, back pain) and clinical studies (such as evoked potential tests or markers of injury in the spinal fluid) are often indicators of a less complete recovery. These markers are imperfect and do not assume aggressive rehabilitation or treatment strategies.

TM can be the presenting feature of multiple sclerosis. In individuals with acute partial transverse myelitis and normal brain MRI, about 10-33 percent develop MS over a five to ten-year period. Those who are ultimately diagnosed with MS are more likely to have asymmetric clinical findings, predominant sensory symptoms with relative sparing of motor systems, lesions extending over fewer than 2 spinal segments, abnormal brain MRI, and oligoclonal bands in the CSF.

Long-Term Care

After the acute phase, rehabilitative care to improve functional skills and prevent secondary complications of immobility involves both psychological and physical accommodations. There is very little written in the medical literature specifically dealing with rehabilitation after TM. However, much has been written regarding recovery from spinal cord injury (SCI) in general, and this literature applies. The physical issues include bowel and bladder management, sexual dysfunction, maintenance of skin integrity, spasticity, activities of daily living (i.e., dressing), mobility, and pain.

It is important to begin occupational and physical therapies early during the course of recovery to prevent the inactivity-related problems of skin breakdown and soft tissue
contractures that lead to a decreased range of motion. Assessment and fitting for splints designed to passively maintain an optimal position for limbs that cannot be actively moved is an important part of the management at this stage.

The long-term management of TM requires attention to a number of issues. These are the residual effects of any spinal cord injury, including TM. In addition to chronic medical problems, there are the ongoing issues of ordering the appropriate equipment, reentry into school, re-socialization into the community, and coping with the psychological effects of this condition by the patients and their families. During the early recovery period, family education is essential to develop a strategic plan for dealing with the challenges to independence following return to the community.

Bladder Function

Bladder function is almost always at least transiently impaired in patients with TM. Immediately after the onset of TM, there is frequently a period of transient loss or depression of neural activity below the involved spinal cord lesion, referred to as “spinal shock,” which lasts about 3 weeks. Following this period, two general problems can affect the bladder. The bladder can become overly sensitive and empty after only a small amount of urine has collected, or relatively insensitive, causing the bladder to become over extended and overflow. An overly distended bladder increases the likelihood of urinary tract infections and, in time, may threaten the health of the kidneys. Depending on the dysfunction, treatment options include timed voiding, medicines, external catheters for males (a catheter connected to a condom), padding for women, intermittent internal self-catheterization, an indwelling catheter or electrical stimulation. Surgical options may be appropriate for some people. Common bladder problems include incontinence, frequency, nocturia (frequent urination at night), hesitancy, and retention. Treating incontinence, frequency, and nocturia is often easier than treating hesitancy and retention, where clean intermittent urinary catheterizations are the basic component to success. Working with a good urologist is imperative to prevent potential serious complications, particularly one who understands spinal cord disease. Urodynamic testing is necessary to determine urine retention to check risk for urinary tract infections, particularly if there is a history of UTIs to guide the urologist in terms of the best management.

Bowel Function

Another major area of concern is effective management of bowel function. A common problem in spinal cord injury is difficulty with evacuation of stool, although fecal incontinence can also occur. The neurologic pathways for defecation are similar to those of the bladder. Many lacking voluntary control of the bowel may still be able to achieve continence by diet, strategic use of stool softeners and fiber, and the technique of rectal stimulation. Other aids include suppositories, anal irrigation, and oral medications. A high-fiber diet, adequate and timely fluid intake, and medications to regulate bowel evacuations are the basic components of success. Regular evaluations by medical specialists for adjustment of the bowel program are recommended to prevent potentially serious complications. There are some surgical options, although this is rarely necessary.
Long-Term Care

Sexual Dysfunction

Sexual dysfunction involves similar innervation and analogous syndromes as those found in bladder dysfunction. Treatment of sexual dysfunction should take into account baseline function before the onset of TM. Of the utmost importance is adequate education and counseling about the known physical and neurologic changes that TM has on sexual functioning. Because of the similarities in innervation between sexual and bladder function, patients with sexual dysfunction should be encouraged to empty their bladders before sexual stimulation to prevent inopportune incontinence. The mainstays of treatment of erectile dysfunction in men are inhibitors of cGMP phosphodiesterase, type 5, which will allow most men with TM to achieve adequate erections for success in intercourse through a combination of reflex and/or psychogenic mechanisms. Although less effective in women, these same types of medications have been shown capable of enhancing a woman's sexual functioning. The most commonly used oral erectile dysfunction drugs are Viagra (sildenafil), Levitra (vardenafil), and Cialis (tadalafil). Although sexual experience is impacted by spinal cord injury, sensual experience and even orgasm are still possible. Lubricants and aids to erection and ejaculation (for fertility) are available. Adjustment to altered sexuality is aided by an attitude of permissive experimentation, as the previous methods and habits may no longer serve.

Skin Breakdown

Skin breakdown occurs if the skin is exposed to pressure for a significant amount of time, without sensation or the strength to shift position as necessary. Sitting position should be changed at least every 15 minutes. This can be accomplished by standing, by lifting the body up while pushing down on armrests, or by just leaning and weight shifting. Wheelchairs can be supplied with either power mechanisms of recline or tilt-in-space to redistribute weight bearing. A variety of wheelchair cushions are available to minimize sitting pressure. Redness that does not blanch when finger pressure is applied may signal the beginning of a pressure ulcer. Good nutrition, vitamin C, and avoidance of moisture all contribute to healthy skin. Pressure ulcers are much easier to prevent than to heal.

Spasticity

Spasticity means stiffness or muscle spasms and is often a very difficult problem to manage. Some stiffness in our muscles is necessary in order to control our movement, but when they become too tight, the result can range from slightly bothersome stiffness (particularly upon wakening) to uncontrollably painful spasms. When the latter occurs, small triggers such as changes in position, temperature, humidity, or presence of infections can cause this painful spasticity. The key goal is to remain flexible with exercise, a daily stretching routine, and a bracing program with splints, as needed. These splints are commonly used at the ankles, wrists or elbows. Also recommended are appropriate strengthening programs for the weaker of the spastic muscles acting on a joint and an aerobic conditioning regimen. These interventions are supported by adjunctive measures that include antispasticity drugs (e.g., diazepam, baclofen, dantrolene, tizanidine), therapeutic botulinum toxin injections, and serial casting. In cases where spasticity is severe, a baclofen pump, which provides the medication directly to the spinal cord, may be considered. The therapeutic goal is to improve the function of the individual in performing specific activities of daily living (i.e., feeding, dressing, bathing, hygiene, mobility) by improving the available joint.
Long-Term Care

range of motion, teaching effective compensatory strategies, and relieving pain. Left untreated, severe spasticity can lead to shortening of the affected muscle or joint called contractures, further impacting mobility, rehabilitation, and independence.

**Pain**

Pain is common following transverse myelitis.

Changes in sensation often occur and can manifest as lack of sensation, or numbness, as well as painful sensations called neuropathic pain. This pain is described in many different ways, including burning, squeezing, stabbing, or tingling. Having the sensation of pain means the nerve signal is getting through, but in an inappropriate way. While this can get better over time, there is a long list of medications to treat these symptoms. The same medication doesn’t work for everyone, so the trial and error of finding the right medication can be frustrating. Alternative therapies such as acupuncture and meditation have also been utilized, with varying success.

While the body is constantly working toward repair, once damage is done to the central nervous system, there will always be evidence of this damage, usually evidenced on an MRI. Clinical fluctuations of old symptoms, particularly in the setting of infection, stress, heat (Uhthoff’s phenomenon), menstrual cycle, or anything that increases core body temperature or throws the body off of its normal course are also possible. It is important to note that this is not inflammatory driven and therefore in no way represents worsening of the condition.

The first step in treating pain effectively is obtaining an accurate diagnosis. Unfortunately, this can be very difficult. Causes of pain include muscle strain from using the body in an unaccustomed manner, nerve compression (i.e., compression of the ulnar nerve at the elbow due to excessive pressure from resting the elbow on an armrest continuously) or dysfunction of the spinal cord from the damage caused by the inflammatory attack. Muscle pain might be treated with analgesics, such as acetaminophen (Tylenol), non-steroidal, anti-inflammatory drugs such as naproxen or ibuprofen (Naprosyn, Aleve, Motrin), or modalities such as heat or cold. Nerve compression might be treated with repositioning and padding (i.e., an elbow pad for an ulnar nerve compression).

Nerve pain can be a significant challenge to find effective treatment. Nerve messages traveling through the damaged portion of the spinal cord may become scrambled and misinterpreted by the brain as pain. Besides the treatments listed above, certain antidepressants such as amitriptyline (Elavil), or anticonvulsants, such as carbamazepine, phenytoin, or gabapentin (Tegretol, Dilantin, Neurontin) may be helpful. Stress and depression should also be addressed since these conditions make pain harder to tolerate.

**Depression**

Individuals with TM should be educated about the effect of TM on mood regulation and routinely screened for the development of symptoms consistent with clinical depression. Warning signs that should prompt a complete evaluation for depression include failure
to progress with rehabilitation and self-care, worsening fixed low mood, pervasive decreased interest, and/or social and professional withdrawal. A preoccupation with death or suicidal thoughts constitutes a true psychiatric emergency and should lead to prompt evaluation and treatment. Depression in TM is similar to the other neurologic symptoms patients endure, which are mediated by the effects of the immune system on the brain. Depression is remarkably prevalent in TM, occurring in up to 25% of those diagnosed at any given time, and is largely independent of the patient’s degree of physical disability. Depression is not due to personal weakness or the inability to "cope." It can have devastating consequences; not only can depression worsen physical disability (such as fatigue, pain, and decreased concentration) but it can have lethal consequences. Despite the severity of the clinical presentation of depression in TM, there is a very robust response to combined aggressive psychopharmacologic and psychotherapeutic interventions. With appropriate recognition and treatment of TM depression, complete symptom remission is standard.

During the early recovery period, family education is essential to develop a strategic plan for dealing with the challenges to independence following a return to home. Ongoing problems typically include ordering the appropriate equipment, dealing with re-entry into school, work, and community, and coping with the psychological effects of this condition on both those diagnosed with TM and their families. Being saddened or demoralized by the diagnosis of TM is appropriate. The inability to move past this grief in a reasonable period of time such that it interferes with relationships and functional living needs to be addressed and treated. Many fear that depression reflects on oneself as an inadequate ability to cope with their diagnosis and feel weak. But it is not a personal strength issue, and depression is very much a physiological manifestation and treatable. Both talking to a psychiatrist/psychologist and medication management can be beneficial, and some studies indicate a synergistic effect of combining the two. Depression can rebound and can at times become more resistant to treatment.

Fatigue

Fatigue is the lack of mental and/or physical energy. Fatigue can be a direct result of a disease process (primary fatigue) or an indirect result (secondary fatigue). In TM, fatigue is more often thought to be a result of secondary fatigue. Examples of secondary fatigue include fatigue from medications, depression, stress, poor sleep patterns, infections, or changes in walking, which increase energy requirements. The key is to try to identify the underlying cause of the fatigue – for example, if one is not sleeping well because of pain, bladder dysfunction, or depression, this needs to be identified and addressed; not getting consistent sleep will worsen every other aspect of TM! If too much energy is exerted due to changes in walking, physical therapy can help identify better body mechanics that will help conserve energy. When nothing else can be identified as contributing to fatigue, REST is recommended! Conserving energy such that activities are planned and paced can allow for these activities to be more enjoyable rather than stressful. Also, reorganizing home and office can help to reduce the amount of wasted energy exerted so that energy can be saved up for activities that are enjoyable. Also, exercise routines incorporated in the day can actually help build stamina and reduce fatigue in the long-run – it’s also...
Rehabilitation and Activities of Daily Living

a great stress reducer! Pilates, yoga, and swimming are great, but the key is to find something enjoyable and not overdo it.

An appropriate strengthening program and an aerobic conditioning regimen are recommended. The effects on mobility as a result of TM can vary widely, however, from paralysis to mild weakness. Either way, physical therapy is instrumental in returning function. Because physical therapists deal with many different types of injuries and diseases, it is ideal to work with one who has a particular interest in spinal cord rehabilitation when possible. Assistive devices may be necessary for weakness – it can be difficult and oftentimes humbling to take the necessary step of using an assistive device, but when faced with the alternative of broken hips, heads, and the downstream effects of lost wages or jobs, it is an important and sometimes indispensable step in maintaining independence. It is also always very important to remember to exercise, as tolerated, in order to maintain physical health and stamina.

Individuals with TM may find ordinary tasks such as dressing, bathing, grooming, and eating very difficult. Many of these obstacles can be mastered with training and specialized equipment. For example, long handled sponges can make bathing easier as can grab bars, portable bath seats and hand-held shower heads. For dressing, elastic shoelaces can eliminate the need to tie shoes while other devices can aid in donning socks. Occupational therapists are specialists in assessing equipment needs and helping people with limited function perform activities of daily living. A home assessment by an experienced professional is often helpful.

Physical therapists assist with mobility. Besides teaching people to walk and transfer more easily, they can recommend mobility aids. This includes everything from canes (single point vs. small quad cane vs. large quad cane) to walkers (static vs. rolling vs. rollator) and braces. For a custom-fabricated orthotic (brace), an orthotist is necessary. Careful thought should go into deciding whether the brace should be an ankle-foot orthosis, whether it should be flexible or stiff, and what angle the foot portion should be in relationship to the calf portion. Some will benefit by a knee-ankle foot orthosis. Each person should be evaluated individually. The best results occur when a physician coordinates the team so that the therapists and orthotists are united on what is to be achieved. The physician best trained to take this role is the physiatrist.

Additional Resources

Myelitis Helpline
srna.ngo/helpline

For questions about our organization and rare neuroimmune disorders, visit the Myelitis Helpline, an online tool developed by SRNA.

Resource Library
srna.ngo/resources

To access up-to-date resources on rare neuroimmune disorders, which include symposium videos, magazines, podcast recordings, published research summaries, information sheets and relevant external resources, visit our Resource Library.
References


... References


19 Krishnan C, Greenberg B. Transverse myelitis. In: UpToDate, Dash JF (Ed), UpToDate, Waltham, MA, 2021.


... References


... References


