Journal of The Transverse Myelitis Association

The organization advocating for children and adults with acute disseminated encephalomyelitis, neuromyelitis optica, optic neuritis and transverse myelitis

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From the Editor
Sandy Siegel

Change is difficult for a normal person. It is even more of a challenge for me. You aren't going to find a person more attached to a routine than me. Kazu and I really love our routine. In fact, Kazu probably finds me highly comforting because he can count on me to support his routine … by my staying on my routine. While variety might be the spice of life, I'm generally thriving on monotony. Kazu eats exactly the same thing twice a day, every single day. I've never observed a single instance of diminished excitement as the same dry pellets are poured into his bowl. Kazu and I totally get each other.

In 82 days, 5 hours and 4 minutes from now, I am going to experience one of the more dramatic changes in my life. I am going to retire from my job with the state of Ohio that I have held for 35 years. I am blessed in so many different ways. I was engaged in work that made a significant difference in people's lives; I worked for a regulatory agency. My work involved the regulation of the telecommunications and electric industries. I worked with an amazing professional staff that was very well educated and highly motivated. Most of the people I've worked with have doctorates or master's degrees in economics, systems engineering and in the social sciences. We also have our share of MBAs in finance and accounting and we have a large staff of attorneys. My work was recognized and valued by my peers. And over this very long period of time, I developed very close friendships and good relationships even with the people in the industries we regulate. It has been a great job.

For the past sixteen years I have been doing two jobs. The job that I am retiring from took up my time during the week and it paid me a salary and offered wonderful benefits. I've left the house every day at about 6:00 am and left work at 4:00 pm. I stopped to do a workout on the way home every day, because that's what highly neurotic people do, and I got home at around 6:30 pm. The last thing I did before leaving for work in the morning was to walk Kazu and the first thing I did when I returned was to walk Kazu. Kazu only craps for me; it is our special thing. By the time I got home from walking Kazu, I had two hours before my body was done for the day. My mind was already done. That is a significant portion of life routine to have removed from a really neurotic person. It has also been an enormous amount of time getting in the way of the work from the other job.

For those of you who have been paying attention and/or have been around for a while, you know that my second job has been conducted while I am not doing my state job. This is a job for which I do not receive a salary. We can wax philosophic some time about all of the benefits. It has been really difficult over the past 16 years to do both of these jobs and to also have a life. I try. Pauline and my family are my priority. Beyond my family, my life has definitely been significantly sacrificed. My TMA work has always been too important for me to figure out other-than-family life priorities. I receive phone calls from people who have a loved one in the middle of an inflammatory attack. This usually happens at least once a week and some times more often. I receive phone calls from people daily who are not doing well. Either they are not receiving appropriate medical care or they lack even the most basic understanding about what is going on with their bodies and what can be done for them; and what cannot be done for them. In addition to the phone calls, I receive emails of a similar nature all day long. It is impossible for me to ignore people who are in desperate straits. Managing our membership databases, publishing a newsletter and journal and membership directory, helping with the information on our web site, creating materials for new member packets, arranging all of the printing and mailing activities, coordinating and developing and managing support groups around the world, planning and organizing retreat weekends and family camps, assisting with the planning of symposia and other educational opportunities, coordinating work with other advocacy organizations, managing the day to day activities of our organization, trying to keep everyone loving everyone else … the tremendous amount of work that needs to be done that is not being done – it just never ends.

It is no wonder that I can't hit out of a sand trap.

I very well could have gone on doing precisely what I am doing now for a great deal longer (work both jobs) … because I'm definitely neurotic enough to pull it off. I once asked my psychologist during counseling just how neurotic he thought I was. He laughed - way too hard. And then he respond-

ed that he thought I was pretty neurotic, but since I wore it so well, he wouldn't recommend doing anything about it. I took that to mean that my neurosis was one of my more positive traits, like my thick hair and fast typing.

But complicated life circumstances are helping me to make this move and to motivate me to work on getting parts of my life back. I used to be a well-rounded and interesting person. I have great hopes that my retirement is going to allow me to continue my work for the TMA and still allow me to have the life that evaporated 16 years ago. I plan to give Pauline and our children more of my time. I'm truly fascinated with just how much time it is going to take before they are highly overwhelmed by my attention and yearning for those wonderful bygone days of neglect. I'll keep you posted.

I plan to return to reading novels, magazines and newspapers. The past 16 years have been filled with so much reading of medical journals and health advocacy literature that I haven't really had time for the reading I love to do. And now reading involves all of these great electronic toys.

I spent 14 years of my life training to become a cultural anthropologist. I was a very good anthropologist. Complicated life circumstances made a career in anthropology difficult for me to pursue. During my retirement, I plan to publish from the tremendous amount of excellent material that I collected during two years of fieldwork research; I am a Native American expert. I returned from my work with 10,000 pages of hand written field notes on yellow legal pads (no computers in 1976 – 1978) and more than 3,500 slides. Beyond my dissertation, I never had the opportunity to write about the experience or publish any information about the tribes we lived with for two years. I definitely owe that writing to the remarkable people with whom we shared our lives during that time.

I was a pretty decent conga player in my day. I would love to return to playing, if the arthritis in my hands allows. I intend to play enough golf that I'm able to get out of a trap and actually hit off of a fairway every so often. I plan to continue and intensify my suffering as a fan of the Cleveland Browns and Indians. I plan to get Pauline to a beach and under water (in scuba gear) as often as possible. And I need to clean the basement.

And I would love to do some writing … perhaps about all of those complicated life circumstances. What I am not going to do is look for another job; I already face more work every day than I can possibly finish even if I don't have my paying job taking up way too much of my time. I am not going to be bored.

One of the activities I am most excited about pursuing is research on TM, ADEM, NMO and ON. I am a social scientist. I know how to do research. I plan to focus my time, energy and expertise on collecting information from all of you about these disorders. I know how to collect the information, I have good friends who can run the data and perform statistical analyses, and I know how to evaluate the data and information and get the results in front of the physicians who are caring for all of you. This is an area where I believe I can make a significant contribution. And it is no small thing that I work for free.

How is my retirement going to impact the TMA and more importantly, how is this change in my life going to impact all of you? The answers to that question have consumed the greater part of my ruminations as I go through the planning and the emotional process of preparing for my retirement.

My brother started talking to me in 2006 about what kind of succession plan the TMA had in place to deal with the eventuality that I was no longer around to do this work. Of course, my reaction to him was, "And what do you mean not be around?" Winning the lottery and death only happen to other people. Over the years, his approach to these discussions has gone from gentle nudging to characterizing my inactivity in this area as totally irresponsible. "How can you take all of the work you've done in the past couple of decades and gamble that it will totally disappear if you are no longer able to do this work or you are no longer here?"

I've given lots of thought to my brother's words. My brother is a brilliant clinical psychologist who has spent his entire career not offering me any advice whatsoever. My brother's usual reaction to one of my multitude of life dilemmas is something like, "Wow, you are really messed up." I'm sure my brother's patients receive greater depth and clarity from him; I'm also certain they receive the same level of candor. So, if my brother is willing to offer me a substantial reflection on my approach to the organization, I feel compelled to take his admonitions seriously. Okay, now where do I find a crack pot whose wife got TM, is neurotic enough to work his brains out to get things done even when he creates his own self-imposed but like-they-come-from-the-burning-bush deadlines for everything, has enough compassion to force him to pick up the telephone and return every single phone call and return every single email message from everyone … no matter the circumstances; is passionate enough to internalize the cause so that advocacy comes from the depth of his heart and soul and has a value system such that they don't want to be paid for doing any of this work? If I post this on Monster.com, will I get beyond the crackpot part of the position description?

The future of the TMA depends on the work continuing past me. And as we have all learned in such a stark and intimate way, none of us knows what is going to happen tomorrow, let alone at 2:00 this afternoon. I can imagine what the world was like without an advocacy organization for TM, ADEM, NMO and ON – and I don't want to go back to that time. I don't want that for Pauline and I don't want that for all of you and your loved ones. Whether you are actively involved in this work or you don't ever think about it, your life circumstances are seriously diminished by not having an organization exist that makes possible the training of doctors and researchers focused on these disorders, that creates educational opportunities for patients and medical professionals, that provides information and offers support networks and support opportunities (i.e., retreats and family camps), and that advocates for and raises money to fund research. You need an organization that networks with other advocacy organizations that share our concerns and goals. You need an organization that keeps our issues in front of politicians and the government who allocate resources and design and develop programs that impact so much about how we receive medical care. Most of you are aware of why you need The Transverse Myelitis Association and why we cannot risk losing this organization.

The only way I can guarantee that the work will continue is to pay someone to do it. It is very difficult to sustain an entirely volunteer organization. And the person getting paid is not going to be me … remember, I'm the crackpot. We need to bring on new energy, new creativity, and new passion to fill a position that will continue past the person who fills the job. It can't be about Jim, Debbie and Paula – although it will always be about these people so long as they are willing and able to do this work.

One of our first priorities, after my retirement, is going to be to raise the funds to hire an executive director of the TMA.

We need more doctors who specialize in caring for patients with TM, ADEM and NMO, who perform the research, and who become faculty members in departments of neurology who can teach other physicians about these disorders and can attract more physicians into this discipline. We are going to have to raise money to fund the Jim Lubin Fellowship in order to make this happen. We need to have a sense of urgency about attracting more doctors and scientists into our discipline.

The amount of research we can make happen on TM, ADEM and NMO depends directly on our raising money to fund it. The evidence is clear; unless we raise money to fund TM and ADEM research, it isn't going to happen. Victoria Jackson has made the most compelling case that money, sound scientific oversight and passion can change the research landscape for these disorders. That is precisely what she and Bill Guthy have accomplished with NMO. We need to match their efforts for TM and ADEM.

The common denominator in almost everything about our future plans involves raising money. I will need your help to professionalize the TMA and to transform the organization into one that can manage the complexities of focusing our resources in a transparent, responsible, informed manner to achieve our goals. We have a wonderful community of people who have so many skills and talents and expertise. We are going to be calling on those of you who can serve our community and help us to fill these critically important needs.

We are also going to be depending on everyone to get involved in helping us with our fundraising efforts. We have been incredibly gentle with these efforts over the past 16 years. Part of that gentleness was dictated by the absence of time and resources. We can no longer afford to manage our fundraising as we have in the past. We are going to come to you in every way imaginable to make the case as to why you need to support these efforts.

The amount of work that needs to be done is staggering and the risks of not accomplishing this work are frightening. We still don't know what kind of disorders TM or ADEM are – it is ***postulated*** that they are auto-immune. The nomenclature surrounding the medical disorders called ***transverse myelitis*** is a nightmare that significantly complicates diagnosis and seriously impedes research. This nomenclature needs to be fixed even if it means that we become The Transverse Myelitis Association, the advocacy organization for people with ADEM, NMO, ON and TM … and we no longer have a disorder called ***transverse myelitis***. Changing the name of this disorder is a really messy thought (particularly for the person who thrives on no change and compulsive-neurotic order) but it needs to happen. It will give organizational historians lots to talk about … like, where did that logo come from?

At least some headway is being made in the understanding of NMO; and it is occurring almost exclusively from the efforts of the Guthy-Jackson Charitable Foundation.

An acute attack from NMO, TM and ADEM are pretty much treated in the same way; and not necessarily because that's the best way to do it. It is done this way, mostly because we don't know enough to do it differently. There haven't been enough studies or enough scientific evidence to guide the decisions about acute treatment; it remains a judgment call by the physician in charge. We use the nuclear explo-

sion approach to treating an immune attack because we don't understand the immune system well enough to identify specific culprits in the immune system that would allow for a narrow, specific, and less all-encompassing target to focus on. For example, we might be able to cripple the enemy by taking out the water towers, but since we don't know that the water towers are the necessary and sufficient infrastructure for enemy survival, we have to level the entire town. And this is no big deal, unless you happen to live in this town. We manage recurrent cases in the same manner because we also lack sufficient information to target small and more specific targets. There hasn't been a single clinical trial to test a drug to treat any of the symptoms of TM. This is also the case for ADEM and NMO. Hey, if it works for diabetic neuropathy, it will probably work for NMO. Well, okay. And I haven't even touched on the entire issue of how do we repair the nervous system after it has been permanently damaged?

I could go on like this for quite a while … but as I have to get back to my emotional preparations for my retirement (which for the most part looks like, *can I stay home now*), I should end here. One of the great contributions that the TMA can make in this research is that we are the leading advocate in the universe for research across all of the neuroimmunologic disorders. It is our position that we can best come to an understanding of all of these disorders – ADEM, NMO, ON, MS and TM – by studying all of them together. We just don't understand enough about any of them to draw a fence around one and study it in isolation from the others. The overlap of cases and the relationships are far too compelling to ignore any of these disorders in studying one of them. **And we fervently argue that they *all* need to be studied.** That is also why we are so grateful for our relationship with the Accelerated Cure Project. This organization shares precisely this approach to studying these disorders and is likely the greatest fundamental support of this endeavor through their repository.

I'm driven to find the answers by the indignities and suffering I witness in Pauline every day. It totally sucks. I observe it going on all around me … Jim, Paula, Debbie, Maureen, Rachel, Kevin, Ashley, Elliot, Maggie, Jonathan, Maria, Alana, Walter … a day doesn't go by that I don't think about the challenges that so many of you face from bladder and bowel issues, from pain and spasticity, from depression and fatigue and cognitive problems, from sexual dysfunction and from paralysis, including not being able to breathe. I hear it from you on the phone every day, and I communicate with you about it in emails. I need for there to be better answers for Pauline and for all of you. I need for these children to have the best shot at a good quality of life. They need to be able to live their dreams in the same way that all of us had that chance. I need for the 5 month old baby who is going to have an inflammatory attack tomorrow to have a better chance of recovery, because we have good science upon which to base their treatments, as opposed to the expert, educated-guess crap shoot we're employing today.

None of the answers are going to fall from the sky and none of them are going to appear for free. And I've been looking around for the past 16 years, and none of them are going to happen because someone else paid for them. **The bill is ours** and unless we begin the serious process of creating the funds to support all of this research and the physicians who are going to conduct it, our critical needs will remain unmet.

If you can make a difference, now is a great time to start. If you can't, I would encourage you to get involved in fundraising, primarily with your friends and family who best understand how ADEM or NMO or TM has impacted your lives. As these are rare diseases and our numbers are small, we cannot afford to have anyone on the sidelines; we all need to get involved.

I am really looking forward to my rapidly approaching retirement. Pauline is supportive but cautiously concerned, anxious and fearful. *Sandy, do you think a 2500 square foot house is really large enough for you and me? Is a king size bed really the largest bed they make?* Kazu has taken a wait and see approach to all of this retirement conversation. No doubt his final evaluation will be measured by whether my retirement has any impact on his caloric intake and the frequency of his walks.

I hope that my retirement means positive and significant changes for the TMA; changes that move us forward in a professional way to accomplish so many of these important goals that are yet to be fulfilled and that so many of you are depending on.

81 days, 5 hours, 57 minutes … but who's counting?

Please take care of yourselves and each other.

Sandy

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CLINICAL PRACTICE

Transverse Myelitis

Elliot M. Frohman, M.D., Ph.D., and Dean M. Wingerchuk, M.D.

This Journal *feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.*

An otherwise healthy 28-year-old woman presents to the emergency department with progressive weakness that began 3 days earlier. She reports difficulty walking, numbness in the body below her breasts, and urinary urgency, and she notes that neck flexion triggers an electrical sensation that radiates to the coccyx. Physical examination reveals moderate paraparesis with hyperreflexia, a left extensor plantar response, impairment of vibratory and proprioceptive sensation, and a sensory level at T6. Magnetic resonance imaging (MRI) reveals a lower cervical cord lesion that enhances after gadolinium administration, a finding that is consistent with transverse myelitis. How should she be further evaluated and treated?

THE CLINICAL PROBLEM

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The term "transverse myelitis" describes a heterogeneous group of inflammatory disorders that are characterized by acute or subacute motor, sensory, and autonomic (bladder, bowel, and sexual) spinal cord dysfunction (Table 1).[1] The clinical signs are caused by an interruption in ascending and descending neuroanatomical pathways in the transverse plane of the spinal cord, and a resulting sensory level is characteristic of the syndrome. The transverse myelitis syndrome may arise from various causes, but it most often occurs as an autoimmune phenomenon after an infection or vaccination (accounting for 60% of the cases in children) or as a result of a direct infection, an underlying systemic autoimmune disease, or an acquired demyelinating disease such as multiple sclerosis or the spectrum of disorders related to neuromyelitis optica (Devic's disease, a demyelinating disease that is defined by transverse myelitis and optic neuritis).[2-7] However, after detailed evaluation, 15 to 30% of the cases of transverse myelitis are ultimately categorized as idiopathic.[5,8]

Estimates of the annual incidence of idiopathic or postinfectious transverse myelitis range from 1.3 to 8 cases per million. Although the disorder can develop at any age, there is a bimodal peak in the incidence at 10 to 19 years of age and at 30 to 39 years.[9,10] The incidence increases to 24.6 cases per million annually if acquired demyelinating causes, especially multiple sclerosis, are included.[11] There is no clear pattern among cases of idiopathic transverse myelitis with respect to sex, geographic distribution, or familial susceptibility.

The pathological hallmark of transverse myelitis is the presence of focal collections of lymphocytes and monocytes, with varying degrees of demyelination, axonal injury, and astroglial and microglial activation, within the spinal cord.[2] Neuromyelitis optica lesions contain deposits of immunoglobulin and complement around small blood vessels, and necrosis can be observed in severe cases.[7] The observation that systemic infection or immunization precedes many cases of trans-

verse myelitis suggests that mechanisms such as molecular mimicry and the development of autoantibodies may play roles in the pathogenesis of the syndrome.[2,4]

The prognosis after an attack of transverse myelitis is highly variable among both adults and children.[8,11,12] Patients who have an attack of transverse myelitis associated with multiple sclerosis may have a substantial or even complete recovery, but patients with transverse myelitis or neuromyelitis optica associated with other diseases usually have clinically significant residual neurologic deficits. Most of the recovery occurs over the course of the first 3 months after the event, although improvement may continue for a year or longer. In one study of idiopathic myelitis, more than one third of the patients had a rapidly progressive course with a poor outcome (death or inability to ambulate). The combination of severe weakness, hypotonia, and areflexia — "spinal shock" — was the only recognized predictor of a poor outcome.[8]

Table 1. Diagnostic Criteria for Transverse Myelitis.*

Bilateral (not necessarily symmetric) sensorimotor and autonomic spinal cord dysfunction
Clearly defined sensory level
Progression to nadir of clinical deficits between 4 hours and 21 days after symptom onset
Demonstration of spinal cord inflammation: cerebrospinal fluid pleocytosis or elevated IgG index,† or MRI revealing a gadolinium-enhancing cord lesion
Exclusion of compressive, postradiation, neoplastic, and vascular causes

* Clinical events that are consistent with transverse myelitis but that are not associated with cerebrospinal fluid abnormalities or abnormalities detected on MRI and that have no identifiable underlying cause are categorized as possible idiopathic transverse myelitis.

† The IgG index is a measure of intrathecal synthesis of immunoglobulin and is calculated with the use of the following formula: (CSF IgG ÷ serum IgG) ÷ (CSF albumin ÷ serum albumin), where CSF denotes cerebrospinal fluid.

STRATEGIES AND EVIDENCE

DIAGNOSIS

A history of motor weakness, sensory abnormalities referable to the spinal cord, and bladder or bowel dysfunction point to the diagnosis of myelopathy. Symptoms and signs of transverse myelitis typically evolve over the course of hours to days and are usually bilateral; however, unilateral or markedly asymmetric presentations can occur.[1] Transverse myelitis is sometimes manifested as rapid-onset, severe paraparesis or quadriparesis with areflexia, which may lead to diagnostic confusion with other causes of ascending weakness, such as the Guillain–Barré syndrome; otherwise, hyperreflexia and Babinski signs are present, confirming a central rather than a peripheral cause of the muscle weakness. A well-defined truncal sensory level, below which the sensation of pain and temperature is altered or lost, distinguishes myelopathy from cerebral lesions and peripheral neuropathies. Neuropathic pain may occur in the midline (an aching, deep pain) or in a dermatomal distribution (radicular or lancinating pain or a sensation of burning or itching), with the latter pattern providing a clue to the anatomical level of the lesion. Demyelination is responsible for the presence of Lhermitte's sign (paresthesias that radiate down the spine or limbs with neck flexion) and paroxysmal tonic spasms (involuntary dystonic contractions of limb or trunk muscles). Urinary incontinence or retention, bowel incontinence or constipation, and sexual dysfunction are common but vary in severity among patients.

Once the clinical syndrome of myelopathy is recognized, a knowledge of basic spinal cord anatomy and vascular supply helps to guide the differential diagnosis, which includes compressive, vascular, metabolic, neoplastic, and other causes (see Fig. 1 in the Supplementary Appendix, available with the full text of this article at NEJM.org). Figure 1 provides a systematic approach to the evaluation of myelopathy.[1-3] As a first step, MRI is warranted to rule out the presence of structural lesions, especially those amenable to urgent neurosurgical intervention. The entire spinal cord should be imaged so that false negative results that may be caused by misleading localizing signs, such as a thoracic sensory level caused by a cervical lesion, can be avoided. The finding of an intrinsic cord lesion (sometimes more than one) is characteristic of myelitis; in the acute phase, such lesions usually enhance with intravenous gadolinium administration. An assessment of the spinal cord syndrome at presentation, together with an evaluation of the number, size, and shape of lesions detected on MRI, provides the basis for determining whether the lesion is consistent with myelitis. Lesions associated with idiopathic transverse myelitis usually span at least two vertebral segments, as vi-

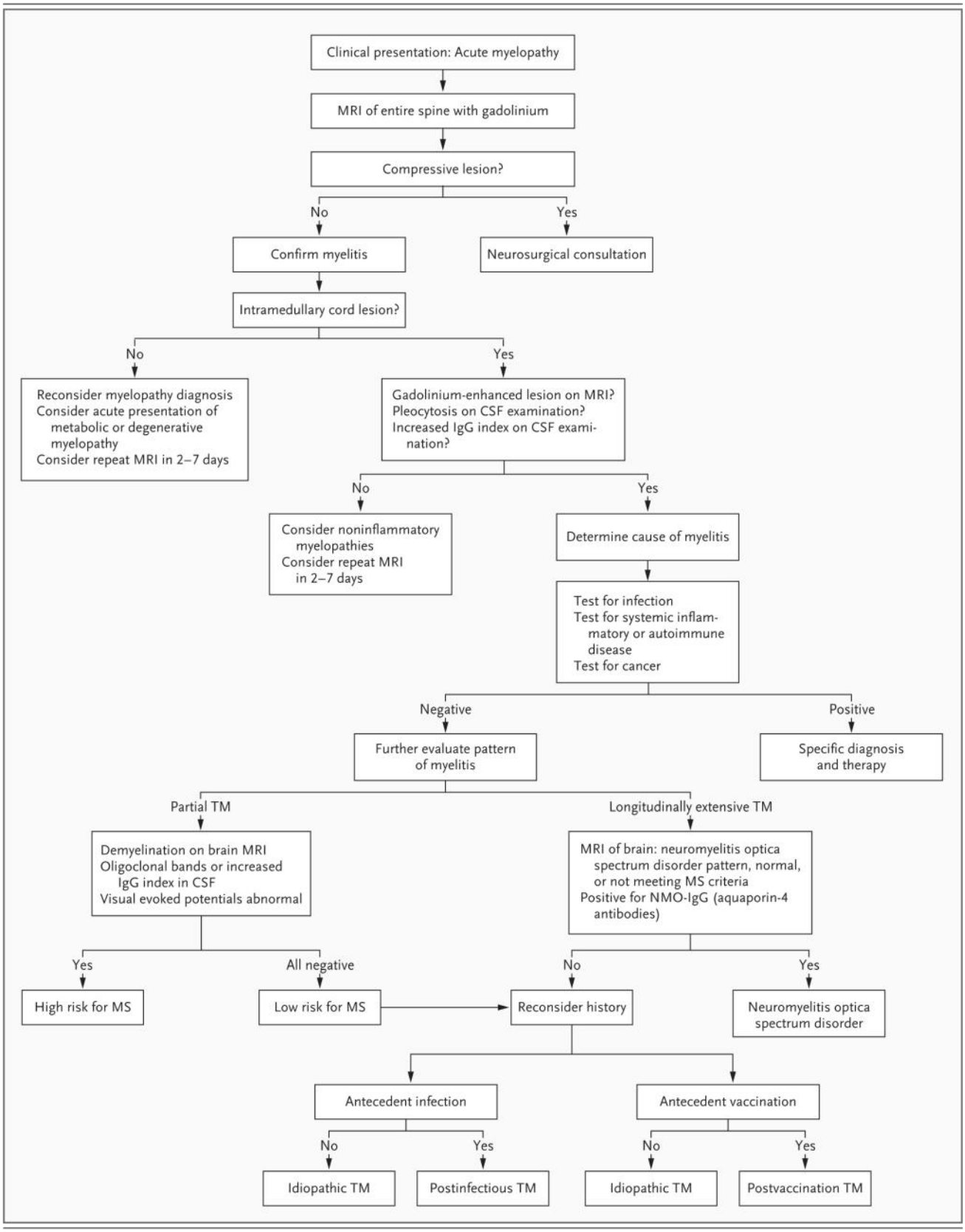
Clinical presentation: Acute myelopathy
MRI of entire spine with gadolinium
Compressive lesion?
No
Yes
Confirm myelitis
Neurosurgical consultation
Intramedullary cord lesion?
No
Yes
Reconsider myelopathy diagnosis
Consider acute presentation of metabolic or degenerative myelopathy
Consider repeat MRI in 2–7 days
Gadolinium-enhanced lesion on MRI?
Pleocytosis on CSF examination?
Increased IgG index on CSF examination?
No
Yes
Consider noninflammatory myelopathies
Consider repeat MRI in 2–7 days
Determine cause of myelitis
Test for infection
Test for systemic inflammatory or autoimmune disease
Test for cancer
Negative
Positive
Further evaluate pattern of myelitis
Specific diagnosis and therapy
Partial TM
Longitudinally extensive TM
Demyelination on brain MRI
Oligoclonal bands or increased IgG index in CSF
Visual evoked potentials abnormal
MRI of brain: neuromyelitis optica spectrum disorder pattern, normal, or not meeting MS criteria
Positive for NMO-IgG (aquaporin-4 antibodies)
Yes
All negative
No
Yes
High risk for MS
Low risk for MS
Reconsider history
Neuromyelitis optica spectrum disorder
Antecedent infection
Antecedent vaccination
No
Yes
No
Yes
Idiopathic TM
Postinfectious TM
Idiopathic TM
Postvaccination TM

Figure 1 (facing page). Diagnostic Algorithm for the Evaluation of Acute Myelopathies and Myelitis.

A systematic approach to the evaluation of acute myelopathy syndromes allows for early identification of cases requiring emergency neurosurgical treatment and provides the highest probability of establishing the specific diagnosis of transverse myelitis, as well as determining the cause of the syndrome. CSF denotes cerebrospinal fluid, MRI magnetic resonance imaging, MS multiple sclerosis, NMO neuromyelitis optica, and TM transverse myelitis.

sualized on MRI of the spinal cord (Fig. 2). Normal MRI results should prompt a reconsideration of the diagnosis of myelopathy in favor of other disorders of the central or peripheral nervous system.[2,3]

The transverse myelitis syndrome has an extensive differential diagnosis. The medical history, medical review of systems, social and travel history, and general physical examination can provide clues that point toward possible infectious or paraneoplastic causes, as well as causes associated with systemic inflammatory or autoimmune diseases such as systemic lupus erythematosus, Sjögren's syndrome, and sarcoidosis.[1-3,13,14] Implication of a connective-tissue disease (e.g., systemic lupus erythematosus) as the cause of transverse myelitis requires evidence of systemic disease, and the diagnosis should not be based solely on the presence of serum autoantibodies (e.g., antinuclear antibody or extractable nuclear antigen), because such antibodies may be present in patients with underlying multiple sclerosis or neuromyelitis optica.[15] Clinical features, laboratory tests, and diagnostic imaging options that are helpful in making the diagnosis are summarized in Table 2, as well as in Table 1 in the Supplementary Appendix. The occurrence of transverse myelitis after infection or vaccination does not preclude the need for further evaluation, since infection or immunizations may also trigger attacks of myelitis in the context of an underlying disease (especially multiple sclerosis or neuromyelitis optica).

Transverse myelitis is a common manifestation or presenting feature of acquired demyelinating diseases of the central nervous system.[16] Among children, it is a frequent characteristic of acute disseminated encephalomyelitis, which typically occurs after an infection or immunization and is associated with clinical and MRI evidence of multifocal cerebral involvement.[4] Multiple sclerosis is associated with short lesions (spanning fewer than two vertebral segments) that are located in the periphery of the cord, affecting mainly white matter ("partial" transverse myelitis) (Fig. 2), and there is usually concomitant MRI evidence of demyelinating brain lesions.[17] Neuromyelitis optica is strongly associated with longitudinally extensive transverse myelitis, defined by a lesion that spans three or more vertebral segments on MRI.[6,7] Such lesions tend to be symmetric and situated centrally within the cord (involving both gray and white matter) and may extend into the brain stem, causing nausea, vomiting, and hiccups (Fig. 2).[18,19] Neuromyelitis optica is specifically associated with the serum autoantibody marker NMO-IgG, which targets the astrocytic water channel, aquaporin-4.[20,21]

Identifying the cause of transverse myelitis facilitates the prediction of the future clinical course and informs the decision about whether to provide prophylaxis against future neurologic events. The postinfectious, postvaccination, and idiopathic forms of transverse myelitis are usually monophasic syndromes, whereas multiple sclerosis and neuromyelitis optica–spectrum disorders are relapsing diseases that are associated with a high risk of future attacks of transverse myelitis and other neurologic events. The presence on MRI of brain lesions that are characteristic of demyelination indicates a high risk of multiple sclerosis after a partial myelitis event (Fig. 2 in the Supplementary Appendix). Seropositivity for NMO-IgG (and to a lesser extent, ssA autoantibody) in a patient with longitudinally extensive transverse myelitis is highly predictive of relapsing disease and, in the case of NMO-IgG, conversion to definite neuromyelitis optica, which can also be associated with abnormalities on MRI of the brain (Fig. 2 in the Supplementary Appendix).[22,23] Factors suggesting that a person who has had a myelitis event is at high risk for clinically definite multiple sclerosis or neuromyelitis optica are summarized in Table 1 in the Supplementary Appendix.

MANAGEMENT

Initial Immunotherapy

The goals of therapy during the acute phase of myelitis (Table 2 in the Supplementary Appendix) are to halt the progression and initiate the reso-

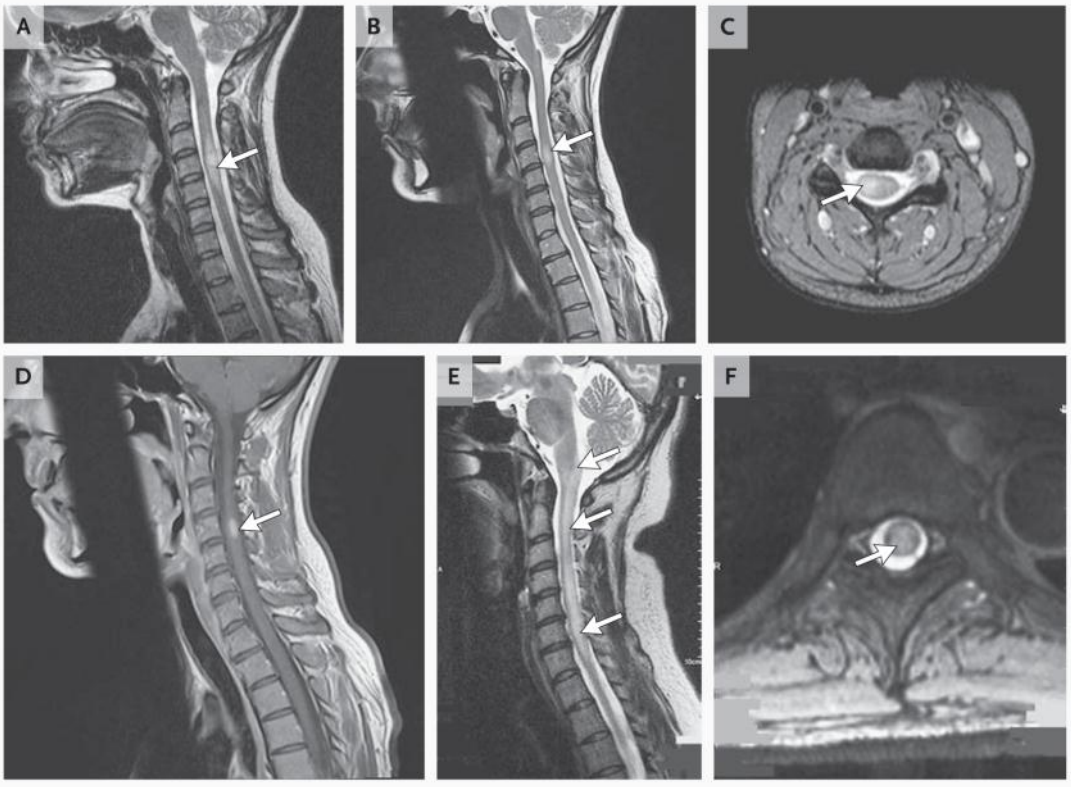

Figure 2. Features of Common Myelitis Syndromes on Neuroimaging.

The findings on MRI are a key component of the diagnostic evaluation of transverse myelitis. Acute myelitis events are associated with a focal lesion within the spinal cord. Panel A shows an example of a lesion associated with idiopathic myelitis (sagittal plane, T_2-weighted sequence). Short-segment lesions, as shown in Panel B (sagittal plane, T_2-weighted sequence), and those that are asymmetric, as shown in Panel C (axial plane, T_2-weighted sequence), are characteristic of multiple sclerosis. Lesion enhancement after the administration of gadolinium, as shown in Panel D (sagittal plane, T_1-weighted sequence), suggests acute inflammation of the spinal cord. In contrast, a longitudinally extensive lesion (i.e., one that spans several vertebral segments), as shown in Panel E (sagittal plane, T_2-weighted sequence), especially if it extends rostrally into the brain stem and is located centrally within the cord, as in Panel F (axial plane, T_2-weighted sequence), is typical of neuromyelitis optica.

lution of the inflammatory spinal cord lesion, thereby speeding clinical recovery. Corticosteroids are the standard first-line treatment; however, because there have been no randomized, controlled trials of corticosteroid therapy of patients with transverse myelitis, supporting evidence for the use of corticosteroids as first-line therapy is derived from case studies or extrapolation from trials involving patients with multiple sclerosis.[1-5,7,17,24,25] Approximately 50 to 70% of patients have partial or complete recovery and are ambulatory with or without aid.[8,24] High-dose intravenous regimens are typically used (e.g., 1000 mg of methylprednisolone daily, generally for 3 to 5 days),[25] although evidence of the superiority of a particular corticosteroid drug, dose, or route of administration is lacking. Oral regimens may be used in the case of patients with relatively mild episodes of myelitis who do not require hospitalization (e.g., in cases associated with established multiple sclerosis).[25,26] Potential adverse effects of pulsed corticosteroid therapy include gastrointestinal symptoms, insomnia, headache, anxiety, mania, hypertension, hyperglycemia, and electrolyte disturbances.

Rescue therapy with plasma exchange may benefit patients who do not have a response to corticosteroids.[27-29] In a randomized, crossover trial involving 22 patients with idiopathic inflammatory demyelinating syndromes (7 of which were cases of myelitis) that did not respond to corticosteroids, 42% of the patients had moder-

Table 2. Concise Differential Diagnosis and Diagnostic Testing for Transverse Myelitis.*

Possible Cause	Diagnostic Tests
Infection	Blood serologic studies; CSF culture, serologic studies, and PCR; chest radiography and other imaging as indicated
Systemic autoimmune or inflammatory disease	Clinical examination; serologic studies; chest and joint radiography; other tests or imaging as indicated by history and examination
Paraneoplastic cause	Chest radiography, computed tomography, or positron-emission tomography; comprehensive serum and CSF paraneoplastic antibody panel
Acquired CNS demyelinating disease (multiple sclerosis, neuromyelitis optica)	Brain MRI with gadolinium enhancement; CSF examination for cell count and differential count, oligoclonal bands, and IgG index; tests of visual evoked potentials; serum NMO-IgG testing
Postinfectious or postvaccination cause	History taking that reveals clear, recent history of infection or vaccination; serologic confirmation of recent infection; exclusion of other causes

* CNS denotes central nervous system, CSF cerebrospinal fluid, NMO neuromyelitis optica, and PCR polymerase chain reaction.

ate to marked improvement during plasma exchange, as compared with only 5.9% of patients undergoing a sham procedure.[27] Hypotension, electrolyte imbalance, coagulopathy, thrombocytopenia, catheter-related thrombosis, and infection are recognized complications of plasma exchange.

In an uncontrolled, retrospective study involving 122 patients with transverse myelitis from various causes, 56 patients with severe impairment that did not respond to corticosteroid therapy were further treated with plasma exchange, cyclophosphamide, or both. Plasmapheresis was associated with an improvement among patients who had some remaining sensorimotor function at the nadir of the attack, but patients who had complete loss of sensorimotor function generally appeared to have improvement only when they were treated with both cyclophosphamide and plasmapheresis. Adjunctive short-term immunosuppressive strategies deserve further investigation.

Among patients with underlying demyelinating disease, long-term immunomodulatory or immunosuppressive therapies have been shown to reduce the risk of future attacks.[30,31] A discussion of these therapies is beyond the scope of this article.

Treatment of Symptoms and Complications

There are several important stabilizing and preventive measures that can be taken to reduce the symptoms and complications of transverse myelitis (Table 2 in the Supplementary Appendix). No data from randomized trials are available to confirm the specific efficacy of therapeutic or prophylactic interventions in patients with transverse myelitis; therefore, recommendations are based on observational studies or clinical experience with patients who have multiple sclerosis or other neurologic diseases. Most patients with new-onset myelitis are hospitalized for observation and management of their condition.

Respiratory and Oropharyngeal Support

Transverse myelitis can cause respiratory failure by involving the upper cervical spinal cord and brain stem[7]; therefore, regular reassessment of respiratory and oropharyngeal functions are required during the evolution of myelitis. Dyspnea, the use of accessory muscles, or a weak cough requires further evaluation with the use of tests of respiratory forces and pulmonary function. Intubation for mechanical ventilation is required for some patients. Dysarthria, dysphagia, or reduced tongue function or gag reflex warrants a formal investigation of the patient's swallowing function to assess the need for the temporary placement of a feeding tube, through which adequate nutrition can be provided while the risk of aspiration pneumonia is minimized.

Motor Weakness and Complications of Immobilization

Administration of low-molecular-weight heparin for prophylaxis against deep-vein thrombosis is warranted for all patients with immobility.[32] Nonambulatory patients benefit from frequent adjustments of their position while they are sitting or in bed to promote comfort and maintain skin integrity. Collaboration with a physical medicine

team should be considered so that multidisciplinary neurorehabilitation can be initiated early.[33] Ambulation can be aided with the use of appropriate devices. An oral sustained-release potassium-channel blocker, 4-aminopyridine, has been shown to improve walking speed in patients with multiple sclerosis, possibly by prolonging the duration of the action potential, although this agent has not been studied specifically in patients with transverse myelitis.[34]

Abnormalities of Tone

Severe myelitis may be associated with hypotonia in the acute phase (during spinal shock), but this is typically followed by the emergence of increased resistance to movement (tonic spasticity), along with involuntary muscle spasms (phasic spasticity). Spasticity is an adaptive response that can facilitate ambulation, but when it is excessive, painful, or intrusive, it may require treatment with physical therapy and medications. Data from controlled trials support the benefits of baclofen, tizanidine, and benzodiazepines for the treatment of patients with spasticity associated with disorders of the brain and spinal cord.[35]

Pain

Pain is common during and after an attack of myelitis and can be caused by direct neural injury (neuropathic pain), orthopedic factors (e.g., pain due to postural derangements or bursitis), spasticity, or some combination of these factors. Neuropathic pain may respond to treatment with anticonvulsant agents, antidepressant medications (tricyclic antidepressants and reuptake inhibitors of serotonin and norepinephrine), nonsteroidal analgesics, and narcotics.[30,36-40]

Fatigue

Reduced mobility, medications, pain, and other factors can contribute to excessive fatigue after an episode of myelitis; systematic evaluation and management of the causes of the fatigue are warranted (Table 2 in the Supplementary Appendix). Pharmacotherapy is reserved for cases in which these causes have been ruled out or treated.[41] Data from randomized, controlled trials have shown the efficacy of amantadine for the treatment of fatigue associated with multiple sclerosis, and in one study[42] — but not another[43] — modafinil was shown to be beneficial. A randomized, blinded, crossover, pilot study showed that acetyl L-carnitine was superior to placebo and amantadine for the treatment of fatigue associated with multiple sclerosis,[44] but larger, controlled trials are required to corroborate this finding. Stimulants such as dextroamphetamine or methylphenidate are occasionally used to treat severe, refractory fatigue that occurs after an episode of myelitis, but the usefulness of these agents in treating patients with myelitis has not been tested in randomized, controlled trials.

Genitourinary and Bowel Dysfunction

The placement of a urethral catheter is usually necessary during the acute phase of transverse myelitis, owing to retention of urine in the bladder. After the acute phase, detrusor hyperreflexia typically develops and is characterized by urinary frequency, urgency, urge incontinence, and the perception of bladder spasms.[45] These symptoms are usually reduced with the administration of anticholinergic agents (e.g., oxybutynin and tolterodine).[30,46] Less frequently, there is inadequate relaxation of bladder sphincters during detrusor contraction (detrusor–sphincter dyssynergia), resulting in retention of urine, with an increased risk of vesicoureteral reflux, infection, and calculus formation. Urinary symptoms are unreliable in differentiating poor bladder compliance (failure to store urine) from bladder retention. Ultrasonographic assessment of residual urine volume in the bladder after voiding is useful to rule out urinary retention, but urodynamic studies may be required to fully characterize the urinary dysfunction.[45] Drugs that inhibit α_1-adrenergic receptors can promote urinary sphincter relaxation and bladder emptying in patients with excess sphincter activity, but some patients require intermittent catheterization to adequately empty their bladder.[47]

In the acute and chronic phases of transverse myelitis, bowel dysfunction is characterized by constipation and the risk of impaction, difficulty with bowel evacuation from the rectal vault, and in some cases incontinence, which is usually associated with an inadequate bowel program to reduce constipation and control the timing of defecation.[30]

Sexual dysfunction is a frequent consequence of transverse myelitis and may be manifested as reduced genital sensation, pain, reduced ability to achieve arousal, or anorgasmia.[48] Therapeutic options are outlined in Table 2 in the Supplementary Appendix.

Psychiatric Considerations

Mood and anxiety disorders are among the most common long-term consequences of transverse myelitis and influence other symptoms, such as pain and sexual function. Pharmacotherapy is commonly prescribed, either alone or in conjunction with psychological counseling.

AREAS OF UNCERTAINTY

Identifying the cause of transverse myelitis is often challenging, and in many circumstances the cause remains unknown. The yield of several tests appears to be low, and there is no consensus regarding the optimal evaluation in terms of cost-effectiveness. Data from randomized trials to guide decisions regarding initial therapy, indications for intensification of treatment, and the optimal management of associated symptoms are scarce.

GUIDELINES

Consensus criteria for the diagnosis of transverse myelitis have been developed.[1] However, to our knowledge, there are no professional guidelines for the management of transverse myelitis.

CONCLUSIONS AND RECOMMENDATIONS

The patient described in the vignette presented with classic clinical and neuroimaging manifestations of acute transverse myelitis. Information obtained from the clinical history, an analysis of the cerebrospinal fluid, the results of other laboratory tests, and an MRI study of the brain, together with an assessment of the characteristics of the spinal cord lesion, allow for a rapid assessment of the likelihood that the episode of transverse myelitis is associated with an infection, an underlying systemic disease, or a demyelinating disease such as multiple sclerosis. Admission to the hospital is warranted for observation of the evolution of the syndrome and for treatment of the patient. Data from randomized trials to inform the treatment specifically for patients with transverse myelitis are lacking; however, on the basis of clinical experience and trials involving patients with other demyelinating diseases, high-dose corticosteroids are considered to be the first-line therapy. Assessments by physical and occupational therapists and treatment of symptoms such as pain and urinary dysfunction are indicated. Counseling about the natural history of transverse myelitis and the prognosis must be given on an individual basis, depending on the cause of the condition (if it is identified), and patients and families should be offered support in managing this debilitating condition.

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Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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REFERENCES

1. Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. Neurology 2002;59:499-505.
2. Kaplin AI, Krishnan C, Deshpande DM, Pardo CA, Kerr DA. Diagnosis and management of acute myelopathies. Neurologist 2005;11:2-18.
3. Jacob A, Weinshenker BG. An approach to the diagnosis of acute transverse myelitis. Semin Neurol 2008;28:105-20.
4. Wingerchuk DM. Postinfectious encephalomyelitis. Curr Neurol Neurosci Rep 2003;3:256-64.
5. de Seze J, Stojkovic T, Breteau G, et al. Acute myelopathies: clinical, laboratory and outcome profiles in 79 cases. Brain 2001;124:1509-21.
6. Wingerchuk DM, Lennon VA, Pittock SJ, Lucchinetti CF, Weinshenker BG. Revised diagnostic criteria for neuromyelitis optica. Neurology 2006;66:1485-9.
7. Wingerchuk DM, Lennon VA, Lucchinetti CF, Pittock SJ, Weinshenker BG. The spectrum of neuromyelitis optica. Lancet Neurol 2007;6:805-15.
8. de Seze J, Lanctin C, Lebrun C, et al. Idiopathic acute transverse myelitis: application of the recent diagnostic criteria. Neurology 2005;65:1950-3.
9. Berman M, Feldman S, Alter M, Zilber N, Kahana E. Acute transverse myelitis: incidence and etiologic considerations. Neurology 1981;31:966-71.
10. Bhat A, Naguwa S, Cheema G, Gershwin ME. The epidemiology of transverse myelitis. Autoimmun Rev 2010;9:A395-A399.
11. Debette S, de Sèze J, Pruvo JP, et al. Long-term outcome of acute and subacute myelopathies. J Neurol 2009;256:980-8.
12. Pidcock FS, Krishnan C, Crawford TO, Salorio CF, Trovato M, Kerr DA. Acute transverse myelitis in childhood: center-based analysis of 47 cases. Neurology 2007;68:1474-80.
13. Kumar N, Frohman EM. Spinal neurosarcoidosis mimicking an idiopathic inflammatory demyelinating syndrome. Arch Neurol 2004;61:586-9.
14. Pittock SJ, Lucchinetti CF. Inflammatory transverse myelitis: evolving concepts. Curr Opin Neurol 2006;19:362-8.
15. Pittock SJ, Lennon VA, de Seze J, et al. Neuromyelitis optica and non organ-specific autoimmunity. Arch Neurol 2008;65:78-83.

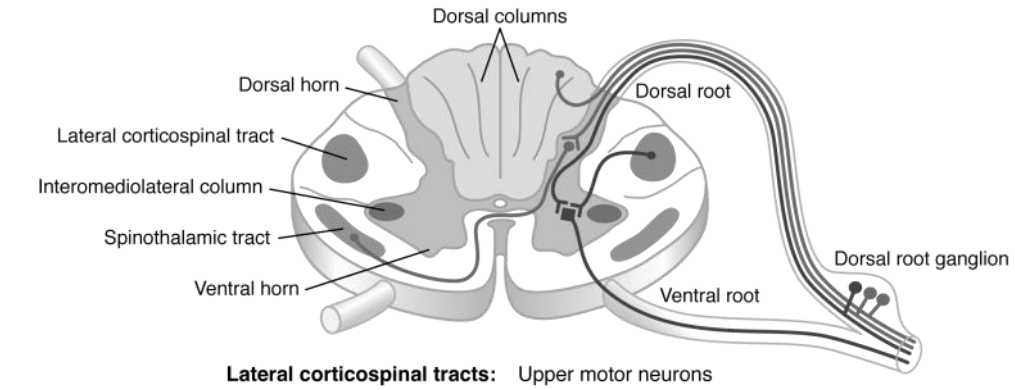
16. Scott TF. Nosology of idiopathic transverse myelitis syndromes. Acta Neurol Scand 2007;115:371-6.
17. Compston A, Coles A. Multiple sclerosis. Lancet 2008;372:1502-17.
18. Wingerchuk DM, Hogancamp WF, O'Brien PC, Weinshenker BG. The clinical course of neuromyelitis optica (Devic's syndrome). Neurology 1999;53:1107-14.
19. Misu T, Fujihara K, Nakashima I, Sato S, Itoyama Y. Intractable hiccup and nausea with periaqueductal lesions in neuromyelitis optica. Neurology 2005;65:1479-82.
20. Lennon VA, Wingerchuk DM, Kryzer TJ, et al. A serum autoantibody marker of neuromyelitis optica: distinction from multiple sclerosis. Lancet 2004;364:2106-12.
21. Lennon VA, Kryzer TJ, Pittock SJ, Verkman AS, Hinson SR. IgG marker of optic-spinal multiple sclerosis binds to the aquaporin-4 water channel. J Exp Med 2005;202:473-7.
22. Weinshenker BG, Wingerchuk DM, Vukusic S, et al. Neuromyelitis optica IgG predicts relapse after longitudinally extensive transverse myelitis. Ann Neurol 2006;59:566-9.
23. Hummers LK, Krishnan C, Casciola-Rosen L, et al. Recurrent transverse myelitis associates with anti-Ro (SSA) autoantibodies. Neurology 2004;62:147-9.
24. Greenberg BM, Thomas KP, Krishnan C, Kaplin AI, Calabresi PA, Kerr DA. Idiopathic transverse myelitis: corticosteroids, plasma exchange, or cyclophosphamide. Neurology 2007;68:1614-7.
25. Frohman EM, Shah A, Eggenberger E, Metz L, Zivadinov R, Stüve O. Corticosteroids for multiple sclerosis: I. Application for treating MS exacerbations. Neurotherapeutics 2007;4:618-26.
26. Martinelli V, Rocca MA, Annovazzi P, et al. A short-term randomized MRI study of high-dose oral vs intravenous methylprednisolone in MS. Neurology 2009;73:1842-8.
27. Weinshenker BG, O'Brien PC, Petterson TM, et al. A randomized trial of plasma exchange in acute central nervous system inflammatory demyelinating disease. Ann Neurol 1999;46:878-86.
28. Watanabe S, Nakashima I, Misu T, et al. Therapeutic efficacy of plasma exchange in NMO-IgG-positive patients with neuromyelitis optica. Mult Scler 2007;13:128-32.
29. Bonnan M, Valentino R, Olindo S, Mehdaoui H, Smadja D, Cabre P. Plasma exchange in severe spinal attacks associated with neuromyelitis optica spectrum disorder. Mult Scler 2009;15:487-92.
30. Courtney AM, Treadaway K, Remington G, Frohman EM. Multiple sclerosis. Med Clin North Am 2009;93:451-76.
31. Wingerchuk DM, Weinshenker BG. Neuromyelitis optica. Curr Treat Options Neurol 2008;10:55-66.
32. Spivack SB, Aisen ML. A comparison of low molecular weight heparin and low dose unfractionated heparin prophylaxis in subacute myelopathy. J Spinal Cord Med 1997;20:402-5.
33. Kesselring J, Beer S. Symptomatic therapy and neurorehabilitation in multiple sclerosis. Lancet Neurol 2005;4:643-52.
34. Goodman AD, Brown TR, Krupp LB, et al. Sustained-release oral fampridine in multiple sclerosis: a randomised, double-blind, controlled trial. Lancet 2009;373:732-8.
35. Groves L, Shellenberger MK, Davis CS. Tizanidine treatment of spasticity: a meta-analysis of controlled, double-blind, comparative studies with baclofen and diazepam. Adv Ther 1998;15:241-51.
36. Namaka M, Leong C, Grossberndt A, et al. A treatment algorithm for neuropathic pain: an update. Consult Pharm 2009;24:885-902.
37. Saarto T, Wiffen PJ. Antidepressants for neuropathic pain. Cochrane Database Syst Rev 2007;4:CD005454.
38. Tzellos TG, Papazisis G, Amaniti E, Kouvelas D. Efficacy of pregabalin and gabapentin for neuropathic pain in spinal-cord injury: an evidence-based evaluation of the literature. Eur J Clin Pharmacol 2008;64:851-8.
39. Saarto T, Wiffen PJ. Antidepressants for neuropathic pain. Cochrane Database Syst Rev 2005;3:CD005454.
40. Wiffen PJ, McQuay HJ, Moore RA. Carbamazepine for acute and chronic pain. Cochrane Database Syst Rev 2005;3:CD005451.
41. Brañas P, Jordan R, Fry-Smith A, Burls A, Hyde C. Treatments for fatigue in multiple sclerosis: a rapid and systematic review. Health Technol Assess 2000;4:1-61.
42. Rammohan KW, Rosenberg JH, Lynn DJ, Blumenfeld AM, Pollak CP, Nagaraja HN. Efficacy and safety of modafinil (Provigil) for the treatment of fatigue in multiple sclerosis: a two centre phase 2 study. J Neurol Neurosurg Psychiatry 2002;72:179-83.
43. Stankoff B, Waubant E, Confavreux C, et al. Modafinil for fatigue in MS: a randomized placebo-controlled double-blind study. Neurology 2005;64:1139-43.
44. Tomassini V, Pozzilli C, Onesti E, et al. Comparison of the effects of acetyl L-carnitine and amantadine for the treatment of fatigue in multiple sclerosis: results of a pilot, randomised, double-blind, cross-over trial. J Neurol Sci 2004;218:103-8.
45. Lemack GE, Frohman EM, Zimmern PE, Hawker K, Ramnarayan P. Urodynamic distinctions between idiopathic detrusor overactivity and detrusor overactivity secondary to multiple sclerosis. Urology 2006;67:960-4.
46. Abrams P, Freeman R, Anderström C, Mattiasson A. Tolterodine, a new antimuscarinic agent: as effective but better tolerated than oxybutinin in patients with an overactive bladder. Br J Urol 1998;81:801-10.
47. Bennett JK, Foote J, El-Leithy TR, et al. Terazosin for vesicosphincter dyssynergia in spinal cord-injured male patients. Mol Urol 2000;4:415-20.
48. Fletcher SG, Castro-Borrero W, Remington G, Treadaway K, Lemack GE, Frohman EM. Sexual dysfunction in patients with multiple sclerosis: a multidisciplinary approach to evaluation and management. Nat Clin Pract Urol 2009;6:96-107.

Supplemental Appendix

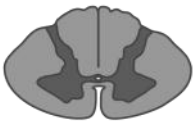
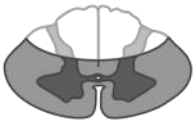
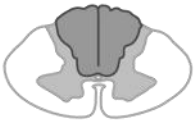
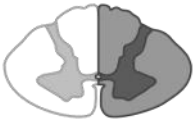
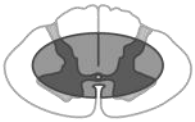
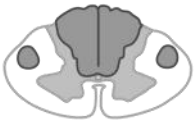
This appendix has been provided by the authors to give readers additional information about their work.
Supplement to: Frohman EM, Wingerchuk DM. Transverse myelitis. N Engl J Med 2010;363:564-72.

Web Figure 1.

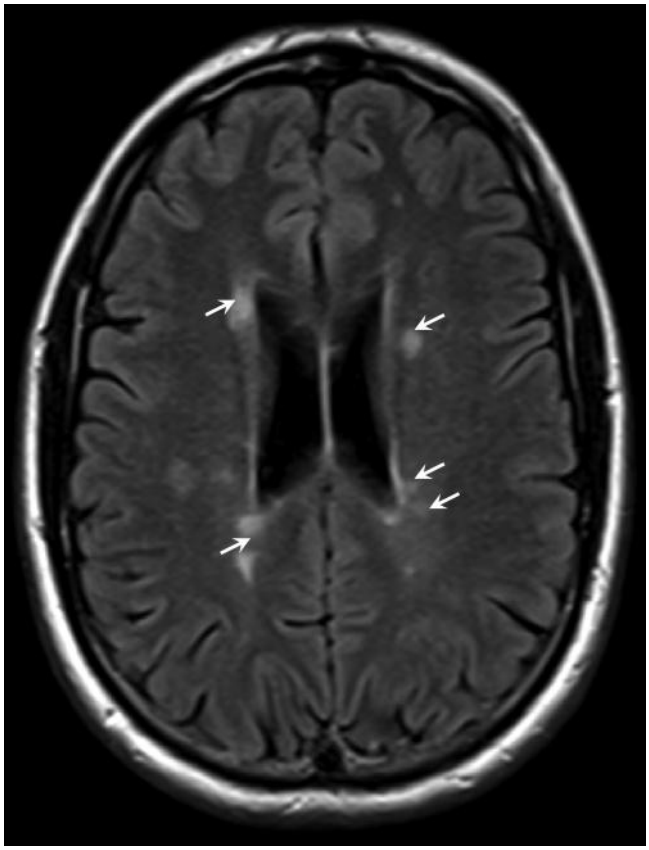
Cross-section of spinal cord



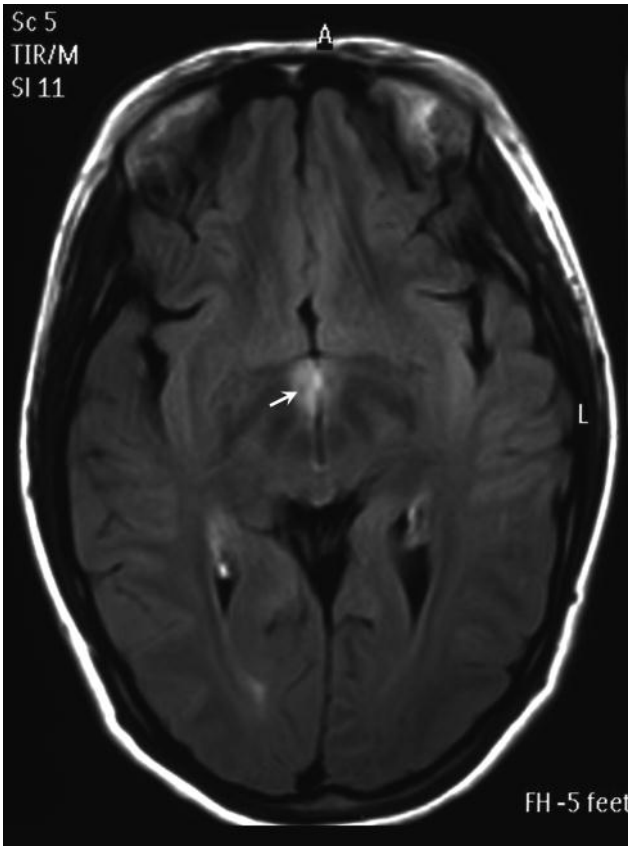
Lateral corticospinal tracts:	Upper motor neurons
Dorsal columns:	Vibration and proprioception Touch and pressure
Lateral spinothalamic tracts:	Pain and temperature
Interomediolateral columns:	Autonomic function
Ventral horns:	Anterior horn cells (lower motor neurons)

	Cord Syndrome	Clinical Signs	Characteristic Etiologies
	Transection	Loss of all motor, sensory, and autonomic function below lesion	Trauma, severe compression, necrotizing myelitis
	Anterior cord	Bilateral flaccid weakness Pain/temperature sensory loss Autonomic dysfunction	Anterior spinal artery occlusion
	Dorsal column	Bilateral reduced vibration, proprioception, touch	Tabes dorsalis Some cases of MS or B12 deficiency
	Brown-Séquard	Ipsilateral pyramidal weakness and vibration/proprioception loss; contralateral pain loss	Multiple sclerosis Compression
	Central	"Dissociated" sensory loss Pain/temperature loss with spared vibration and proprioception	Syrinx, neuromyelitis optica
	Tract-specific	Corticospinal tracts Corticospinal tracts and dorsal columns	Paraneoplastic myelitis B12 or copper deficiency

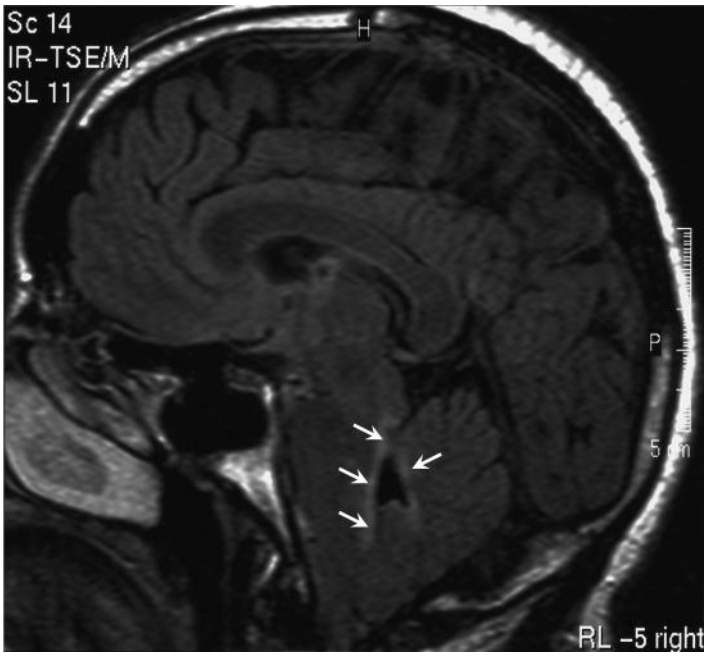
Web Figure 1. Myelopathy: Functional Neuroanatomy, Clinical Signs and Etiologies
The pattern of neurological abnormalities can assist with the differential diagnosis of acute and subacute myelopathy syndromes because of the distinct functions associated with ascending and descending spinal tracts and gray matter structures within the spinal cord (top panel; sinal cord cross-section). The bottom portion of the figure illustrates several examples of clinical cord syndromes and common or characteristic etiologies. In practice, many of these syndromes occur in incomplete forms (e.g., partial or incomplete Brown-Sequard syndrome.



Web Figure 2. Panel A.



Web Figure 2. Panel B.



Web Figure 2. Panel C.

Web Figure 2. Brain MRI Abnormalities Associated with Multiple Sclerosis and Neuromyelitis Optica

Numerous ovoid periventricular cerebral lesions (examples shown with arrows in Panel A; axial plane, fluid attenuated inversion recovery (FLAIR) sequence) are characteristic of multiple sclerosis and suggest that the patient is at high risk for future clinical events that confirm the diagnosis. Brain imaging in neuromyelitis optica is often normal, but may show patchy subcortical white matter lesions atypical for multiple sclerosis or lesions involving brain regions with high aquaporin-4 density, such as the hypothalamus (arrow, Panel B; axial plane; FLAIR sequence) and adjacent to the third and fourth ventricles (Panel C; axial plane FLAIR sequence show signals change around the fourth ventricle (arrows). Idiopathic transverse myelitis is associated with normal or nonspecific brain imaging results.

Web Table 1. Differential Diagnosis, Clinical Features, and Diagnostic Tests for Acute Myelitis Syndromes[1-4]

Infectious Myelitis		
Etiologies	**Clues to Diagnosis**	**Diagnostic Tests**
Infectious Myelitis Viruses DNA Viruses Herpesviruses Cytomegalovirus Epstein-Barr virus Herpes simplex types 1 and 2 Human herpes virus-6 Varicella zoster virus RNA Viruses Picornaviruses Coxsackieviruses A and B Echoviruses Enteroviruses 70 and 71 Hepatitis Aand C Polioviruses Orthomyxoviruses Influenza A and B Paramyxoviruses Measles Mumps Parainfluenza virus Flaviviruses West Nile virus Japanese encephalitis virus St. Louis encephalitis virus Tick-borne encephalitis virus Dengue virus Retroviruses Human immunodeficiency virus-1 (HIV) Human T-lymphotrophic virus type 1 (HTLV-1) Bacteria Mycoplasma pneumoniae Chlamydia	Meningoencephalitic symptoms Fever Meningismus Encephalopathy/confusion Current systemic infection Immunocompromised status Underlying disease (e.g., malignancy; HIV/AIDS) Immunosuppressive medications Rash Vesicles, dermatomal (Herpes zoster) Vesicles, buccal/hands/feet (Enterovirus 71) Erythema chronicum migrans (B. burgdorferi) Macules/petechiae ankles/wrists (R. rickettsii) Other systemic features of infection Pharyngitis (Epstein-Barr virus) Lymphadenopathy (several viruses) Other historical clues Residence/travel history (parasites) Tick exposure (Lyme disease, Rocky Mountain spotted fever) HIV risk factors Sexually transmitted disease history	Chest radiography and other body imaging as indicated CSF Investigations For viruses: Antibodies and PCR: HSV-2, VZV, EBV, CMV, HHV-6, HSV-1 Antibody (IgM) for West Nile virus Antibody for HTLV-1 Viral culture For bacteria: Gram's stain and bacterial culture Venereal Disease Research Laboratory (VDRL) test for syphilis Antibodies and PCR: B. burgdorferi R. rickettsii For mycobacteria; Acid-fast bacilli smear Mycobacterial culture For fungi and parasites India ink smear Fungal culture Blood Investigations For viruses Serology for HSV-2, VZV, HIV-1, HTLV-1, hepatitis A, B, C For bacteria Blood cultures Serology for Mycoplasma For rickettsia Serology and PCR for B. burgdoferi Serology for R. rickettsii
Streptococcus pneumoniae Legionella Borrelia burgdorferi (Lyme disease) Bartonella henselae (Cat scratch disease) Pertussis Rickettsia rickettsii (Rocky Mountain spotted fever) Fungi Actinomyces Blastomyces Coccidioides Aspergillus Parasites Cysticercosis Schistosomiasis Angiostrongylosis Gnathostoma		For parasites Serology
Myelitis Associated with Systemic Autoimmunity or Inflammatory Diseases		
Etiologies	**Clues to Diagnosis**	**Diagnostic Tests**
Systemic lupus erythematosus	Rash, with or without photosensitivity Inflammatory arthritis or serositis Anemia, leucopenia, or thrombocytopenia	Serological tests: antinuclear antibody, anti-double-stranded DNA, SS-A (Ro), SS-B (La), and others Complement levels Urinalysis with evaluation for hematuria Joint X-rays
Antiphospholipid antibody syndrome	Arterial or venous thrombosis Livedo reticularis Pregnancy loss	Antiphospholipid antibodies and coagulation studies
Sjögren syndrome	Xerophthalmia or xerostomia	Schirmer's test Salivary gland or conjunctival biopsy
Systemic sclerosis	Skin contractures/thickening, capillary telangectasias Raynaud's phenomenon	Anti-centromere and anti-Scl-70 antibodies Nailfold capillaroscopy
Neurosarcoidosis	Adenopathy Erythema nodosum	Chest radiograph or computed tomography Biopsy of mediastinal/other lymphadenopathy
Behcet's disease	Orogenital mucocutaneous aphthous ulcers	Gynecological examination Ophthalmological examination
Paraneoplastic Myelitis		
Etiologies	**Clues to Diagnosis**	**Diagnostic Tests**
Small cell lung carcinoma Breast carcinoma Ovarian carcinoma Other cancers	History of cancer Risk factors for cancer, especially cigarette smoking	Paraneoplastic antibody panel (serum & CSF), including: Anti-Ri, Anti-Hu, anti-Yo, amphiphysin, collapsin response mediator protein-5 (CRMP-5), glutamic acid decarboxylase antibodies, P/Q or N-type calcium channel antibodies, voltage-gated potassium channel antibodies, neuronal and muscle acetylcholine receptor antibodies Chest radiography Chest, abdomen, and pelvis computed tomography Whole body positron emission tomography
Acquired Central Nervous System Demyelinating Diseases		
Etiologies	**Clues to Diagnosis**	**Diagnostic Tests**
Multiple sclerosis	Female:male ratio 2-3:1 Predominantly whites Family history of multiple sclerosis in 15% Partial myelitis syndrome Prior neurological events consistent with CNS demyelination	Brain MRI: periventricular lesions, often with gadolinium enhancement Spinal cord MRI: short lesion length (1-2 vertebral segments), peripheral location in cord CSF: <50 WBC/μL (lymphocytes) and presence of oligoclonal bands or elevated IgG index
Neuromyelitis optica spectrum disorders Neuromyelitis optica Syndromes associated with serum NMO-IgG autoantibodies	Female:male ratio up to 9:1 May be overrepresentation of non-whites Usually sporadic Complete transverse myelitis syndrome Events of intractable nausea/vomiting/hiccoughs Neurogenic respiratory failure Seropositivity for multiple autoantibodies May have multiple co-existing systemic autoimmune diseases	Brain MRI: usually normal or nonspecific white matter lesions; 10% have signature patterns (Figure 3) Spinal cord MRI: longitudinally extensive (>3 vertebral segments) lesion, central location in cord, may extend into brain stem CSF: up to 2000 WBC/μL, may be neutrophilic; oligoclonal bands and IgG index usually normal Serum NMO-IgG (sensitivity 73%, specificity > 90%)

Web Table 2. Acute and Symptomatic Therapy for Transverse Myelitis

Acute Therapy for Transverse Myelitis			
Indication	**Pathophysiology**	**Intervention**	**Safety Considerations**
Acute inflammation	Trafficking of lymphocytes, monocytes, and macrophages into the spinal cord with cytokine and chemokine expression	Corticosteroids: IV methylprednisolone 1gm/day for 3-7 days Alternatives: Oral prednisone 1250mg daily for 3-7 days Oral methylprednisolone 1gm daily for 3-7 days Dexamethasone 160-200mg po/IV daily for 3-7 days	Gastrointestinal side effects Insomnia, headache, anxiety, mania Hypertension, hyperglycemia, Chronic use: Bone loss, cataracts, skin changes
Acute inflammation refractory to corticosteroid therapy	As above; humoral mechanisms of cord injury (autoantibodies, complement) and cytokine and chemokine expression may respond to removal by plasma exchange	Plasma Exchange: ½ to full volume exchange, daily or every other day for a total of 3-7 exchanges	Hypotension, electrolyte imbalance Coagulopathy or catheter-related thrombosis Catheter-related infection or sepsis Thrombocytopenia Iron deficiency

Symptomatic Therapies for Transverse Myelitis and its Complications				
Symptom	**Pathophysiology/Cause**	**Investigations**	**Interventions**	**Purpose of Therapy/Comments**
Respiratory weakness	Neurogenic muscle weakness	Bedside examination Pulmonary function tests Negative inspiratory force	Pulmonary toilet Incentive spirometry Postural percussion and drainage (PP&D) Mucus clearance vest	Prevent hypoxia, aspiration pneumonia, and infection
Swallowing and/or speech dysfunction	Oropharyngeal or lingual weakness with brainstem involvement	Bedside examination Modified barium swallowing study Speech evaluation	Prescribe appropriate diet (e.g. soft mechanical) Avoid thin liquids in those with dysphagia Feeding tube ifhigh risk of aspiration Speech therapy	Prevent aspiration pneumonia and dehydration Optimize nutritional status Enhance communication
Motor weakness	Pyramidal tract dysfunction	Neurological exam	Physical therapy Occupational therapy	Optimize upper and lower extremity voluntary motor function
Gait dysfunction	Pyramidal tract dysfunction: Circumduction, toe drag, pelvic obliquity (tilted pelvis while walking which may cause hip and muscle girdle pain), ataxia, trunk weakness, reduced righting reflexes, orthostasis	Examination	Ankle-foot orthotic (AFO) braces Functional electric stimulation (FES) Heel-cord stretching Assist devices (cane, walker, wheelchair, scooter) 4-aminopyridine (4-AP) increases walking speed in MS	Optimize gait mechanics, prevent falls, avoid deep vein thrombosis, minimize orthostasis Risk of 4-AP is seizures; do not use if prior seizure or significant renal impairment.

Spasticity and phasic motor events	Paroxysmal tonic spasms Hypertonia due to upper motor neuron injury. May be tonic or phasic.	Patient History Examination	Anticonvulsants, especially carbamazepine; baclofen Stretching, baclofen, tizanidine, ice therapy, benzodiazepines, botulinum toxin Intrathecal baclofen pump if severe and refractory	Purpose: enhance function and reduce pain. Risks: Pain during stretching; sedation with virtually all anti-spasticity agents. Pump risks: infection, overdose (causing hypotonia, sedation, and altered mental status), and interrupted dosing from catheter or pump failure or failure to refill (causing withdrawal, including seizures and coma).
Sensory symptoms/ Pain	L'hermitte's sign (symptom) Neuropathic (see text) Orthopedic, myofascial Rectal/genital pain	Patient History Examination	Usually resolves without requirement for therapy; when severe, anticonvulsants Anticonvulsants, muscle relaxants, tricyclic antidepressants, narcotics Physical therapy, acetaminophen, NSAIDS Belladonna & opium (B&O) supprettes, topical analgesics	Risks include sedation, reduced cognition, constipation and urinary retention (tricyclics and narcotics); anticholinergic side effects (tricyclics); also hyponatremia with carbamazepine.
Fatigue	Usually multifactorial: explore sleep hygiene, iatrogenic factors (drugs), depression, spasticity, anemia, hypothyroidism, vitamin deficiency (B12, D)	Patient History Sleep study Laboratory studies	Amantidine Modafinil? Methylphenidate? Acetyl-L-carnitine?	Risks include: livedo reticularis, peripheral edema, neuropsychiatric symptom (amantadine); agitation, elevated blood pressure, and appetite reduction (stimulants). Correct other medical disorders/adjust medications before adding medication.
Bladder	 Detrusor hyperreflexia Detrusor-sphincter dyssynergia (with high post void residual (>100cc)	History Post void urine residual (PVR) Urological consultation	 Anticholinergics Alpha antagonists Clean intermittent self catherization	Purpose: improve patient comfort, prevent infection and hydronephrosis. Risks: dry eyes and mouth, constipation, orthostatic hypotension, urinary retention. Avoid in glaucoma. Risks: hypotension, tachycardia, and bladder incontinence, particularly in those patients with coincident bladder spasms. Risks: urethral injury

	Nocturia Nocturnal enuresis		Antidiuretic hormone (DDAVP) Imipramine	Risks: hyponatremia (DDAVP), anticholinergic side effects (imipramine)
Bowel	Neurogastrointestinal signaling defects Dehydration Drug effects (e.g. anticholinergics) Poor evacuation	Patient History Stool Diary Review drug list	Fluids, fiber bulking, softeners, bowel stimulants Fluids Consider transcutaneous oxybutynin (Oxytrol®) Suppository, minidose enema (Enemeez®) Colostomy for severe cases	Risks: over-aggressive bowel care program can be associated with diarrhea, bloating, abdominal discomfort, and excessively large stools. Use of bowel stimulants, suppository, or minidose enema can be associated with bowel incontinence, particularly in those patients whose mobility limitations affect the ability to get to the toilet in a timely fashion.
Sexual dysfunction	Reduced libido Poor erection Reduced sensation Reduced lubrication	Patient History: Review drug list, depression, relationship issues Exclude non-neurogenic co-morbidities	Men: Eliminate offending agents Proerectile agents Women: High-intensity genital vibrators EROS device to enhance genital blood flow Water-soluble lubricating agents	Headache, lightheadedness, 'blue vision', and priapism have been associated with proerectile drugs
Venous thrombosis	Immobility and hypercoagulable state	Bedside exam Doppler studies	Prophylactic anticoagulation Compression boot Inferior vena cava filter (if necessary)	Risks: heparin-induced thrombocytopenia, hemorrhage
Edema	Immobility and weakness	Bedside exam	Compression socks Pneumatic compression pumps Lymphedema massage Fluid management	Purpose of therapy often cosmetic but may improve mobility in those with paraparesis
Skin Integrity	Immobility	Daily systematic examination of ventral and dorsal body surface	Increase mobility Scheduled repositioning Assess mattress and chair surface needs to avoid skin breakdown	Purpose: prevention of pressure sores which increase risk of sepsis and worsen symptoms such as spasticity

Familial neuromyelitis optica

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ABSTRACT

Background: Detection of aquaporin-4–specific immunoglobulin G (IgG) has expanded the spectrum of neuromyelitis optica (NMO). Rare reports of familial aggregation have suggested a component of genetic susceptibility but these reports mostly antedated the discovery of the NMO-IgG biomarker and recently updated diagnostic criteria.

Methods: We report a case series describing the demographic, clinical, neuroimaging, and NMO-IgG serologic status of 12 multiplex NMO pedigrees with a total of 25 affected individuals.

Results: Twenty-one patients (84%) were women. Families were Asian (n = 5), Latino (n = 4), white (n = 1), or African (n = 2). Apparent transmission was either maternal (n = 5) or paternal (n = 2). In 1 family, 3 individuals had NMO; in the others, 2 individuals were affected. Sibling pairs (n = 6), parent–child (n = 4), and aunt–niece (n = 3) pairs were observed. Nineteen patients (76%) were NMO-IgG positive. Twelve (48%) had clinical or serologic evidence of another autoimmune disease. Familial occurrence of NMO occurs in approximately 3% of patients with well-established diagnosis of NMO.

Conclusions: A small proportion of patients with NMO have relatives with this condition, but familial occurrence is more common than would be expected from its frequency in the general population. Familial NMO is indistinguishable from sporadic NMO based on clinical symptoms, age at onset, sex distribution, and frequency of NMO-IgG detection. One or 2 generations were affected and affected individuals represented a small fraction of family members. Taken together, these data suggest complex genetic susceptibility in NMO. ***Neurology*® 2010;75:310–315**

GLOSSARY

AQP4 = aquaporin-4; **CI** = confidence interval; **HLA** = human leukocyte antigen; **IgG** = immunoglobulin G; **LETM** = longitudinally extensive transverse myelitis; **MS** = multiple sclerosis; **NMO** = neuromyelitis optica; **ON** = optic neuritis; **OR** = odds ratio.

Neuromyelitis optica (NMO) and the NMO spectrum disorders are autoimmune inflammatory diseases of the CNS associated with severe relapses of optic neuritis (ON) and myelitis. A serum IgG autoantibody marker, NMO-IgG,[1] which targets the astrocyte water channel aquaporin-4 (AQP4),[2] and validated clinical diagnostic criteria[3,4] allow sensitive and specific diagnosis of NMO. An NMO spectrum disorder can now be confidently diagnosed even in the face of previously unrecognized clinical phenomena (e.g., symptomatic brain lesions) that precluded a diagnosis of NMO in the past. Furthermore, NMO can now be distinguished from multiple sclerosis (MS) with an initial presentation of ON and myelitis.[5,6]

Although there are few population-based studies, the prevalence of NMO has been estimated by its relative frequency to MS, and a consensus estimate of NMO prevalence is approximately 1 per 100,000.[7,8] Ethnic predilection is controversial; the previously held belief that it is

Supplemental data at www.neurology.org

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a disease predominantly of Asians and perhaps Africans has not been supported in some studies.[7,9] The disproportionate occurrence of NMO in Asians, Latinos, and Africans in some regions may represent a dearth of MS, to which its frequency is compared, in these ethnic groups rather than a true excess of NMO.

NMO-IgG is pathogenic in vitro,[10,11] and can passively transfer NMO-specific brain lesions,[12] but the etiology of NMO remains unknown. We describe the demographic, clinical, neuroimaging, and laboratory profiles of 12 families with NMO, totaling 25 affected individuals, and compare the findings in these NMO cases with those in a series of sporadic NMO cases. The number of multiplex families is unexpectedly large relative to the number expected from the estimated frequency of NMO in the general population, suggesting that heritable factors are involved in susceptibility, although common exposure to an exogenous causative factor within families cannot be excluded. We estimate the frequency of familial aggregation in this disease and evaluated metrics that are potential indicators of the relative importance of genetic susceptibility vs common exposures.

METHODS Standard protocol approvals, registrations, and patient consents. The institutional review board or ethics committee of Mayo Clinic, Rochester, MN; the National Cancer Center, Goyang, South Korea; the University of São Paulo, Brazil; the Legacy Emanuel Hospital, Portland, OR; and the National Hospital for Neurology and Neurosurgery Queen Square, London, UK, approved this study. All patients provided informed consent.

Pedigree ascertainment and assessment. Five families (11 patients) were identified through Mayo Clinic neurology practice. The remaining 7 families (14 patients) were identified by collaborators in South Korea (n = 3), Brazil (n = 3), and the United Kingdom (n = 1). Each index case in this report fulfilled clinical criteria for NMO or an NMO spectrum disorder (e.g., longitudinally extensive transverse myelitis [LETM] or recurrent ON and seropositive for NMO-IgG).[13] The median duration of disease was 4.8 years (range 1–52 years). All patients' sera were tested for NMO-IgG by indirect immunofluorescence on a mouse composite tissue substrate.[1]

We abstracted the following data from the medical record: demographic information, disease duration, age at and year of clinical onset, number and severity of NMO episodes, history and serologic evaluation for evidence of other autoimmune diseases, and MRI results.

We compared intrapair differences in age at onset and calendar year of onset. We used linear regression analysis (age and year of onset) and intraclass correlation of age at onset to evaluate putative common exposures to environmental factors and genetic effects. If susceptibility to NMO was primarily determined by heritable factors, differences in onset age might be smaller between members of pairs relative to the differences between the pairs. If a common exposure to a triggering factor was present, and there was a similar incubation period, affected relatives within pairs might be expected to develop NMO in approximately the same calendar year.

We estimated the frequency of familial aggregation of NMO in families where familial aggregation was not the basis for referral and the family history was determined based on systematic inquiry in patients referred for a diagnosis of NMO at each participating center. We compared the familial cases to a series of patients with sporadic NMO.[3]

RESULTS Demographic characteristics and pedigree structure. Demographic characteristics and pedigree structure are illustrated in table 1. Twenty-one patients (84%) were women. Five families were Asian, 4 Latino, 1 African American, 1 African Caribbean, and 1 white. In 6 families, the affected individuals lived in the same house when they developed initial symptoms of NMO.

In 6 families, 2 generations were involved. One affected pair consisted of half siblings with a common mother. Of the 7 families in which we inferred transmission, 5 (71.4%) had maternal and 2 (28.6%) had paternal line transmission. This difference is likely due to the overrepresentation of women in NMO rather than a sex bias in transmission.

In 11 families, 2 individuals were affected, and in 1 family, 3 individuals were affected. We observed the following pairings: sibling pairs (n = 6), parent–child pairs (n = 3), aunt–niece pairs (n = 2), and a mother–daughter–maternal aunt trio (n = 1). One pair of monozygotic twins was identified. Consanguineous marriages were not reported in any family.

Clinical features. The mean (SD) age at onset was 34.3 years (15.0). The mean difference in age at onset between familial pairs was 17.9 (15.4) years and the mean difference in calendar year of onset was 11.2 (13.4) years. Pearson linear correlation coefficient between the intrapair difference in ages with differences in age at onset was $r = 0.82$ ($p = 0.01$) and with differences in year of onset was $r = 0.16$ ($p = 0.60$) (figure e-1 on the *Neurology*® Web site at www.neurology.org). The intraclass correlation estimate for differences of age at onset was 0, indicating greater variability within relative pairs than across subjects.

The initial syndrome was ON in 14 (51.8%) individuals and LETM in 11 (40.7%) individuals. Intrafamilial concordance for the presenting syndrome (ON or LETM) was seen in 5/12 families (41.6%), which was as expected based on the frequency of these 2 presentations. Two individuals from the same family had both ON and myelitis within a 1-month

Table 1 **Demographic and clinical characteristics of familial NMO patients**

ID (no. of generations)	Relationship	Ethnic background	Age at onset, y	Difference in calendar year of onset	Difference in age at onset	No. of optic neuritis episodes	No. of LETM episodes	NMO-IgG	Other autoimmune disorders/antibodies	Additional autoimmune disease in NMO unaffected relatives
1 (1)	Sister	Asian (Laos)	29.2	16.0	1.1	1	0	Positive	Thyroiditis	Sister with autoimmune thyroid disease
	Sister		28.1			3	3	Positive	Anti-SSA	
2 (2)	Niece	African American	17.0	5.3	17.5	0	2	Positive	None	Father and sister with type 1 diabetes mellitus
	Paternal aunt		34.5			2	1	Negative	None	
3 (1)	Sister	Latino (Mexican)	28.4	12.7	1.9	6	1	Positive	None	None
	Sister		26.5			2	0	Positive	None	
4 (2)	Daughter	Caucasian	54.6	1.9	22.9	1	1	Positive	Polymyositis	None
	Mother		77.5	50.1	43.3	0	3	Positive	Polymyositis	
	Maternal aunt		34.3	42.0	6.4	1	25	Negative	None	
5 (1)	Sister	Latino (Brazilian)	40.7	18.0	15.6	1	4	Positive	RF; anti-β2 glycoprotein	None
	Sister		25.1			3	10	Positive	None	
6 (2)	Father	Latino (Brazilian)	48.3	20.0	31.7	4	6	Positive	None	None
	Daughter		16.6			0	2	Negative[a]	None	
7 (2)	Maternal aunt	Asian (Vietnamese)	52.9	1.2	17.3	0	1	Positive	None	Sister with Sjögren syndrome; nephew with chronic inflammatory demyelinating polyneuropathy
	Niece		35.6			1	1	Negative	None	
8 (1)	Brother	Asian (Korean)	36.5	3.6	5.5	0	2	Positive	None	None
	Sister		30.9			1	4	Positive	ANA	
9 (1)	Daughter	Asian (Korean)	18.6	4.7	32.1	5	2	Positive	None	None
	Mother		50.7			1	1	Positive	RF	
10 (1)	Monozygotic twin sister	Asian (Korean)	33.9	1.8	1.8	0	3	Positive	Sjögren; thyroiditis	None
	Monozygotic twin sister		32.1			3	4	Positive	Sjögren	
11 (2)	Son	Latino (Brazilian)	9.4	3.0	41.8	1	3	Positive	Thyroiditis	Nephew with psoriasis; niece with MS
	Mother		51.2			0	1	Negative[b]	None	
12 (1)	Brother	African Caribbean	22.5	7.9	1.2	6	6	Positive	ANA; RF	Mother and sister with autoimmune thyroid disease; maternal grandmother with type 1 diabetes mellitus
	Sister		23.7			1	1	Positive	Thyroiditis	

Abbreviations: ANA = antinuclear antibody; IgG = immunoglobulin G; LETM = longitudinally extensive transverse myelitis; MS = multiple sclerosis; NMO = neuromyelitis optica; RF = rheumatoid factor; SSA = Sjögren syndrome anti-Ro.

[a] Patient had 2 severe episodes of LETM; brain MRI did not show typical MS lesions.

[b] Serum sample for NMO-IgG collected during course of high-dose IV methylprednisolone.

Table 2 Comparison of sporadic and familial NMO

Feature	Sporadic NMO	Familial NMO	p
No.	48[3]	25	
Median age at onset, y (range)	39 (6–72)	32 (9–77)	—
Patients with other autoimmune diseases (%)	15 (31.2)	7 (28)	0.98
First event (%)			
Optic neuritis	27 (56.2)	12 (44.4)	0.67
Myelitis	20 (41.6)	11 (40.7)	0.85
Myelitis + optic neuritis	1 (2.0)	2 (7.4)	0.56
NMO-IgG positive (%)	33/45 (73.3)[1]	19/25 (76)	0.74

Abbreviations: IgG = immunoglobulin G; NMO = neuromyelitis optica.

interval. The median relapse count was 3 (range 1–26) and the median annualized relapse rate was 0.81 (range 0.12–4.8).

NMO-IgG was positive in 19 (76%), including all 12 index cases. We tested 3 nonaffected relatives: the sister of the index case in family 1 and the father and sister of the index case in family 2. All 3 were NMO-IgG seronegative.

Other autoimmune diseases were documented in 7 affected individuals (autoimmune thyroiditis, n = 3; Sjögren syndrome, n = 2; polymyositis, n = 2). In 6 families, unaffected relatives had other autoimmune diseases (autoimmune thyroiditis, n = 2; type 1 diabetes, n = 2; psoriasis, n = 1; Sjögren syndrome, n = 1; chronic inflammatory demyelinating polyradiculoneuropathy, n = 1).

In family 12, besides the mother and son with NMO, a maternal niece had a diagnosis of MS, with 12 relapses since 1994 involving sensory, cerebellar, and pyramidal systems. Brain and spinal cord MRI were compatible with MS, oligoclonal bands were present in the CSF, and NMO-IgG was negative.

Three additional families with NMO index cases were excluded because it could not be determined with confidence that the relative had NMO or NMO spectrum disorder. In the first family, the mother had relapses of ON (n = 3) and LETM (n = 6) over 15 years of disease and was seropositive for NMO-IgG. Her daughter had 2 episodes of ON over 16 years of disease and was seronegative for NMO-IgG on 2 occasions. In the second family, the NMO-IgG seropositive index case had 2 episodes of ON and 1 of myelitis over 15 months of disease. Her sister died at age 39 due to complications of presumed MS (details not available) and her niece (daughter of a different sister) had multiple episodes of ON and one of transverse myelitis but was seronegative for NMO-IgG and LETM was not documented by MRI. In the third family, the index case had 3 episodes of LETM and was seropositive for NMO-IgG. Her maternal cousin presented with severe bilateral ON and had an episode of hearing loss associated with a lesion in the brainstem 2 years after being documented as seronegative for NMO-IgG.

Familial NMO cases were similar, if not indistinguishable, from sporadic cases, both individually and at a group level in comparison to a reference series of sporadic NMO cases with regard to age at onset, sex distribution, frequency of other coexisting autoimmune diseases, presenting symptoms, and relapsing clinical course (table 2).[2,3]

The frequency of familial aggregation in NMO is 2.8% based on the number of familial index cases in a combined series of 386 consecutive patients with NMO seen at Mayo Clinic, Rochester, MN (n = 5/175; 2.9%), National Cancer Center, Goyang, South Korea (3/88; 3.4%), Medical School of University Hospital of Riberão Preto, São Paulo, Brazil (2/33; 6.0%), National Hospital for Neurology and Neurosurgery Queen Square, London, UK (1/16; 6.3%), and University of São Paulo School of Medicine, São Paulo, Brazil (1/74; 1.4%).

DISCUSSION Analysis of pedigrees of multiplex families with NMO may be helpful in understanding the pattern of transmission and ultimately, combined with molecular genetic analysis, in discovering susceptibility genes. Four multiplex families with NMO (8 affected individuals) have been reported, all but one report from the pre-NMO-IgG[1] era and before formal diagnostic criteria[4] were proposed. Identical twin sisters (24 and 26 years of age at disease onset) with NMO were reported in 1938. Both had severe episodes of transverse myelitis and bilateral blindness and died of NMO-related complications. The autopsy evaluation showed demyelination of the optic nerve and diffuse inflammation and demyelination extending from the cervical to the lower thoracic cord.[14] These reports were followed by those of 2 infant sisters (age 2 and 3 at onset) who had relapses of bilateral ON and thoracic myelitis[15]; of 2 sisters with late onset of NMO (62 and 59)[16]; and of a daughter (aged 29 at onset) with myasthenia gravis and NMO and a mother (62 years at onset) with NMO-IgG seropositive NMO.[17] Recently, 2 sisters, 1 with NMO and 1 with prototypic MS, were reported.[18]

The frequency of NMO in family members of our NMO index cases is greater than expected based on the best estimate of its prevalence frequency in the general population of 1/100,000.[7,8] If the average pedigree size consisted of 100 first- and second-degree relatives, which is likely an overestimate, one would expect 0.38 affected relatives among the 386 sporadic cases from which familial cases were derived at the collaborating institutions; we detected 12 cases

($p < 0.0001$; χ^2). Familial cases accounted for 3% of NMO but considering the cases that were excluded this is a minimum estimate.

In both the historical sporadic and the current familial series, NMO was characterized by acute relapses. The distribution of presenting symptoms (ON, myelitis, or both) was very similar. In both series, ON and myelitis were the dominant clinical manifestations. We had not recognized the extended spectrum of NMO, which includes patients who have recurrent myelitis or recurrent ON, in the sporadic series published in 1999, and NMO-IgG serology was unavailable at that time; therefore, some differences in the characteristics of cases reflects improved understanding of the spectrum of NMO in the familial series. Comparison of outcome data and response to therapy is difficult, because early diagnosis, consistent early treatment with immunosuppressive drugs, and systematic follow-up are much more typical of the contemporary series than it was for the sporadic series.[3]

Only 1 or 2 generations were affected. There was no evidence of bias of transmission from either paternal or maternal line; the excess of maternal line transmission reflects the increased predisposition of women for NMO.

Analysis of age and calendar year of onset in the NMO relative pairs did not strongly support either common environmental exposure or a purely genetic basis for susceptibility. The linear regression analysis of the age at onset with age indicated that age, rather than a specific common exposure, was the principal determinant of age at onset. Furthermore, intraclass correlation of age at onset revealed no excess similarity between members of a pair compared to the interpair differences contrary to expectation if genetic factors solely determined susceptibility to NMO. The lack of intraclass correlation of age at onset and the age dependence of onset may largely be explained by the predilection for NMO to occur in late middle-aged individuals that may obscure pedigree-specific effects on age at onset.[3,19] There was wide variation between members of pairs in years of onset, suggesting that common exposure was unlikely to be an important etiologic factor.

Genetic association studies for this disease are limited. Human leukocyte antigen (HLA) associations have been observed in studies of sporadic cases of NMO. *HLA-DPB1*0501* allele was more frequent in 38 Japanese patients seropositive for NMO-IgG compared to 52 patients with MS.[20] Forty-five French Caucasian NMO cases were compared to healthy controls and patients with MS for HLA class II A and B alleles; no association was found of *DRB1*1501* with NMO (odds ratio [OR] 1.74; 95% confidence interval [CI] 0.97–3.11, $p = 0.06$). HLA-DRB1*03 was associated with NMO-IgG-seropositive NMO (OR 3.08; 95% CI 1.52–6.27, $p = 0.001$).[21] No mutation was found in genes known to harbor the majority of LHON mutations in 32 patients with NMO.[22] No association was found in a study of 7 AQP4 SNPs genotyped in 901 MS trio families, including 69 in which the affected offspring had clinical history of optic-spinal disease.[23] Recently, genome scan was performed with samples of 53 NMO Korean cases and 240 controls. The study was underpowered but the strongest SNP association signals were tested in a cohort of 93 NMO patients and 368 controls. A common promoter SNP in *CYP7A1*, a gene that encodes a member of the cytochrome P450 superfamily of enzymes, had a dose-dependent protective effect against NMO ($p = 0.0004$).[24]

Although certain infections[25] and cancer[26] have been reported to co-occur with NMO, no environmental or disease trigger has been rigorously associated with NMO. In the families we report, we did not find clear indicators of common exposure to infections. One patient had breast cancer a year prior to the development of NMO. We are unaware of NMO occurring in unrelated household members. There does not appear to be any geographic or ethnic restriction of familial occurrence of NMO. Several of the NMO families had members with other autoimmune diseases, suggesting that these individuals may share common genetic risk factors for autoimmunity in addition to factors that lead to AQP4-specific autoimmunity.

The small number of cases within affected pedigrees, the lack of multigenerational pedigrees, the high ratio of sporadic to familial cases, and lack of distinctive characteristics of familial cases, taken together, support the hypothesis that NMO is a complex genetic disease.

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DISCLOSURE

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REFERENCES

1. Lennon VA, Wingerchuk DM, Kryzer TJ, et al. A serum autoantibody marker of neuromyelitis optica: distinction from multiple sclerosis. Lancet 2004;364:2106–2112.
2. Lennon VA, Kryzer TJ, Pittock SJ, et al. IgG marker of optic-spinal multiple sclerosis binds to the aquaporin-4 water channel. J Exp Med 2005;202:473–477.
3. Wingerchuk DM, Hogancamp WF, O'Brien PC, et al. The clinical course of neuromyelitis optica (Devic's syndrome). Neurology 1999;53:1107–1114.
4. Wingerchuk DM, Lennon VA, Pittock SJ, et al. Revised diagnostic criteria for neuromyelitis optica. Neurology 2006;66:1485–1489.
5. Weinshenker BG, Wingerchuk DM, Vukusic S, et al. Neuromyelitis optica IgG predicts relapse after longitudinally extensive transverse myelitis. Ann Neurol 2006;59:566–569.
6. Matiello M, Lennon VA, Jacob A, et al. NMO-IgG predicts the outcome of recurrent optic neuritis. Neurology 2008;70:2197–2200.
7. Cabrera-Gomez JA, Kurtzke JF, Gonzalez-Quevedo A, et al. An epidemiological study of neuromyelitis optica in Cuba. J Neurol 2009;256:35–44.
8. Cabre P. Environmental changes and epidemiology of multiple sclerosis in the French West Indies. J Neurol Sci 2009;286:58–61.
9. Marignier R, de Seze J, Vukusic S, et al. NMO-IgG and Devic's neuromyelitis optica: a French experience. Mult Scler 2008;14:440–445.
10. Hinson SR, Pittock SJ, Lucchinetti CF, et al. Pathogenic potential of IgG binding to water channel extracellular domain in neuromyelitis optica. Neurology 2007;69:2221–2231.
11. Kinoshita M, Nakatsuji Y, Moriya M, et al. Astrocytic necrosis is induced by anti-aquaporin-4 antibody-positive serum. Neuroreport 2009;20:508–512.
12. Bradl M, Misu T, Takahashi T, et al. Neuromyelitis optica: Pathogenicity of patient immunoglobulin in vivo. Ann Neurol 2009;66:630–643.
13. Wingerchuk DM, Lennon VA, Lucchinetti CF, et al. The spectrum of neuromyelitis optica. Lancet Neurol 2007;6: 805–815.
14. McAlpine D. Familial neuromyelitis optica: its occurrence in identical twins. Brain 1938;61:430–438.
15. Ch'ien LT, Medeiros MO, Belluomini JJ, et al. Neuromyelitis optica (Devic's syndrome) in two sisters. Clin Electroencephalogr 1982;13:36–39.
16. Yamakawa K, Kuroda H, Fujihara K, et al. Familial neuromyelitis optica (Devic's syndrome) with late onset in Japan. Neurology 2000;55:318–320.
17. Braley T, Mikol DD. Neuromyelitis optica in a mother and daughter. Arch Neurol 2007;64:1189–1192.
18. Cabrera-Gomez JA, Ramon-Perez L, Saiz A, et al. Neuromyelitis optica and multiple sclerosis in sisters. Mult Scler 2009;15:269–271.
19. Matiello M, Jacob A, Wingerchuk DM, et al. Neuromyelitis optica. Curr Opin Neurol 2007;20:255–260.
20. Matsushita T, Matsuoka T, Isobe N, et al. Association of the HLA-DPB1*0501 allele with anti-aquaporin-4 antibody positivity in Japanese patients with idiopathic central nervous system demyelinating disorders. Tissue Antigens 2009;73:171–176.
21. Zephir H, Fajardy I, Outteryck O, et al. Is neuromyelitis optica associated with human leukocyte antigen? Mult Scler 2009;15:571–579.
22. Cock H, Mandler R, Ahmed W, et al. Neuromyelitis optica (Devic's syndrome): no association with the primary mitochondrial DNA mutations found in Leber hereditary optic neuropathy. J Neurol Neurosurg Psychiatry 1997; 62:85–87.
23. Ban M, Walton A, Goris A, et al. Polymorphisms in the neuromyelitis optica auto-antigen AQP4 and susceptibility to multiple sclerosis. J Neurol 2007;254:398–399.
24. Kim HJ, Park HY, Kim E, et al. Common CYP7A1 promoter polymorphism associated with risk of neuromyelitis optica. Neurobiol Dis 2010;37:349–355.
25. Sellner J, Hemmer B, Muhlau M. The clinical spectrum and immunobiology of parainfectious neuromyelitis optica (Devic) syndromes. J Autoimmun 2010;34:371–379.
26. Pittock SJ, Lennon VA. Aquaporin-4 autoantibodies in a paraneoplastic context. Arch Neurol 2008;65:629–632.

The Transverse Myelitis Association

The membership of The Transverse Myelitis Association includes persons with the rare neuroimmunologic disorders of the central nervous system, their family members and caregivers and the medical professionals who treat people with these disorders. The Transverse Myelitis Association was established in 1994 as an organization dedicated to advocacy for those who have these disorders.

The TMA was incorporated on November 25, 1996 in the state of Washington and became a 501(c)(3) organization on December 9, 1996. The TMA has more than 8,000 members from every state in the United States and from more than 80 countries around the world. There are no membership fees. The TMA is registered with the California Department of Justice, the Maryland Secretary of State, the Ohio Attorney General's Office, and the Washington Secretary of State. The TMA has a large number of support groups across the United States and around the world. Some of our international support organizations have been formally recognized by their governments, including the United Kingdom, Germany, Ghana, Australia and Canada.

The TMA has been registered with the National Organization of Rare Disorders since 1994. The TMA is a member of or has a partnership with the following organizations: Cody Unser Firststep Foundation, the Johns Hopkins TM and NMO Centers and Project RESTORE, the University of Texas Southwestern TM and NMO Centers, the Accelerated Cure Project, Victory Junction Gang Camp, Spinal Injuries Association – Australia, Christopher and Dana Reeve Foundation, National Family Caregivers Association, American Autoimmune Related Diseases Association, Genetic Alliance, Kakkis Everylife Foundation, and the Guthy-Jackson Foundation.

Skin Health: Prevention and Treatment of Skin Breakdown

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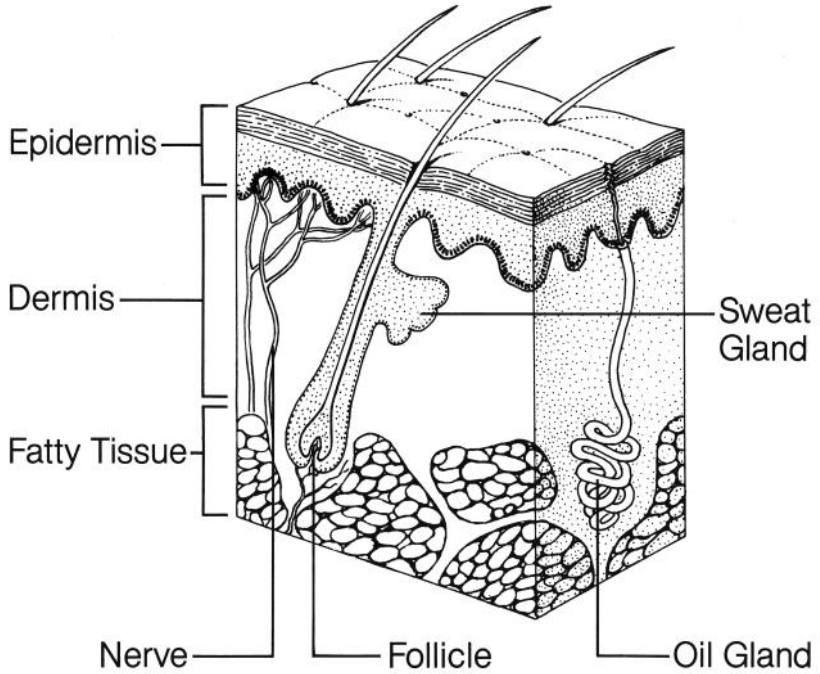
Skin is the largest organ covering the entire outside of the body. It receives one third of the body's blood circulation. Your skin is tough and pliable, forming the body's protective shield against heat, light, chemical and physical action. It plays an active role with the immune system, protecting us from infection. Your skin maintains a stable internal environment and is important in maintaining a proper temperature for the body to function well. In addition to providing protection and internal regulation, your skin gathers sensory information from the environment, allowing you to feel painful and pleasant stimulation. Your skin also stores water, fat, and vitamin D.

The skin consists of three layers: Epidermis, dermis, and subcutaneous tissue. The outermost layer, the epidermis, is composed mostly of dead skin cells that are constantly being shed and replaced. The dermis or second layer has sweat glands, oil glands, nerve endings, and small blood vessels called capillaries, which are all woven together by a protein called collagen. Collagen provides nourishment and support for skin cells. The nerves ending in this layer transmit sensations of pain, itch, touch and pleasure. The hair follicles also originate in this layer. Destruction of either the epidermis or dermis can leave the body open and susceptible to infection. The subcutaneous adipose tissue is the deepest layer of skin and is a layer of fat and collagen that houses larger blood vessels and nerves. This layer is important in controlling the temperature of the skin itself and the body and protects the body from injury by acting as a shock absorber. The thickness of this layer varies throughout the body and from person to person. Underneath the subcutaneous tissue lays muscle and bone.

For the most part, the skin is tough, pliable and resistant to injury. If the skin becomes injured or broken, it is generally very resilient and has an amazing ability to self-repair and heal. Despite this resiliency, the skin is susceptible to breakdown, if subjected to prolonged abuses, such as excessive pressure, shear force, friction or moisture. This is a major concern for persons with transverse myelitis or other neuroimmunologic conditions that cause paralysis and/or decreased sensation.



For people with paralysis, the skin is at increased risk for breakdown for several reasons. Paralysis itself affects the skin and underlying tissue. There is loss of collagen which weakens the skin and makes it less elastic. The lack of muscle function around boney areas of the body leads to muscle atrophy, resulting in less padding, which in turn, adds to the risk of skin breakdown. People with paralysis often have difficulty shifting their weight, repositioning themselves, or transferring without assistance.

Impaired sensation is often present, limiting the ability to sense when to make a weight shift or position adjustment. People with impaired sensation are also vulnerable to injury from many other hazards, such as, heat, cold, sun and trauma. Loss of sensation put an individual at risk for burns from very ordinary activities, such as using a lap top computer sitting directly on your lap or sitting too close to a fireplace. Injury can be caused from things that are too cold such as, ice packs or cold exposure causing frostbite. Ingrown toenails can become infected and sunburn can become severe without feeling it.

When limited mobility is coupled with decreased sensation, a person is more likely to develop a specific type of skin breakdown called a pressure ulcer. According to the National Pressure Ulcer Advisory Panel, a pressure ulcer is defined as a localized injury to the skin and/or underlying tissue usually over a bony prominence, as a re-

sult of pressure, or pressure in combination with shear and/or friction (1). Pressure ulcers are one of the leading causes of complication across the life span of persons with paralysis (2). Up to 95 % of adults with spinal cord injury will develop at least one serious pressure ulcer at some time during their life (3).

Skin breakdown can range from minor scrapes, cuts, tears, blisters or burns to the most serious pressure ulcers with the destruction of tissue down to and even including the bone. A pressure ulcer, especially one that requires surgery, such as a muscle flap or skin graft, can cost thousands of dollars to treat, require lengthy hospitalization, and weeks to months away from family, work, school or community activities. It has been estimated that for persons with spinal cord injury the cost of care for pressure ulcers is about $1.2 to 1.3 billion dollars annually (4).

With a concerted effort, skin breakdown is, for the most part, preventable. It can occur, however, even in people who maintain the most diligent care and use the proper equipment. If skin breakdown is identified early, when still in the minor stages, and if the cause of the breakdown can be identified and eliminated, healing should occur fairly quickly. If it is not identified in its early stages, skin breakdown can rapidly progress from minor to serious.

Skin breakdown is caused in several different ways, including friction, shear, moisture and pressure. These causes can occur individually or in combination. Friction, moisture and sheer are identified as contributing factors to pressure ulcers (5). A friction injury occurs when the skin rubs on surfaces, such as a bed sheet, arm rest or brace and has the appearance of a scrape, abrasion or blister. This type of injury is typically seen on the heels and elbows and may result from repositioning, propping or rubbing due to increased spasticity.

A shearing injury occurs with dragging or sliding of a body part across a surface and has the appearance of a cut or tear. This type of injury can occur from dragging your bottom during a transfer or sliding down in bed when the head of the bed is elevated. With the sliding force, bone is moved against the subcutaneous tissue while the epidermis and dermis remains essentially in the same position; against the supporting surface such as a wheelchair or bed. This action causes occlusion of the blood vessels, decreasing blood flow, oxygen and nourishment to the skin, which eventually leads to breakdown. Sometimes a shear injury will actually tear the tissue over the tailbone and with unrelieved pressure will become a pressure ulcer.

Too much moisture over-hydrates the skin, making it weak and more sensitive to friction, shear and breakdown (think about being in the tub or pool for a long time). Primary sources of excess skin moisture include sweating, bowel and bladder accidents, and drainage from wounds.

Pressure ulcers occur when skin, soft tissue and blood vessels are compressed or squeezed between a bony prominence (such as your tailbone) and an external surface (such as your wheelchair cushion). With compression of these vessels, the blood that nourishes the cells and takes away waste is cut off, starving the tissue of oxygen and vital nutrients. Without food and oxygen, tissue dies and skin breakdown begins. The body tries to compensate by sending more blood to the area. This process results in redness and swelling, places even more pressure on the blood vessels, and further endangers the health of the skin and underlying tissue. Ultimately, a pressure ulcer forms. Increased pressure over short periods of time and slight pressure over long periods of time have been shown to cause equal amounts of damage.

Many factors have been identified as responsible for the development of skin breakdown and pressure ulcer formation. In addition to immobility, impaired sensation and the external factors described above, many internal contributing factors have been identified. These internal factors include poor nutrition and hydration, weight, impaired circulation and oxygenation, impaired cognition or thinking, substance abuse, depression and age (6, 7). Nutritional factors important to prevent or heal wounds include a balanced diet with an adequate intake of protein, vitamin C, vitamin A, and zinc, as well as an adequate intake of fluids (8). When a person is overweight, extra pounds place extra pressure on vulnerable skin areas increasing the risk of compression of blood vessels. Individuals that are underweight often have decreased muscle mass with less fat padding over boney areas leaving them vulnerable to skin breakdown. Smoking, diabetes, anemia and other vascular conditions all lead to decreased circulation, increasing risk for skin breakdown. Individuals who are depressed or have impaired thinking and judgment due to substance abuse are less likely to be vigilant with regard to important self-care issues, such as skin health. Young children generally have more resilient and elastic skin and more baby fat and padding so they often have very little difficulty with skin break down. As children move into adolescence, their skin loses some of its elasticity. They generally have more body weight, putting more stress on pressure areas, such as the ischeal tuberosicties and tailbone with sitting. Teens often begin to have more difficulty with skin breakdown. As we continue to age, our skin becomes increasingly less pliable and resilient. We experience the loss of collagen and muscle mass, as well as decreased circulation, making the skin more vulnerable. The elderly are most prone to skin tears and stripping due to fragile, thin, and vulnerable skin. In addition, incontinence

may become a more frequent issue for bedridden or ill persons, increasing problems with moisture as described above.

HOW CAN I KEEP MY SKIN HEALTHY?

Take responsibility for you own skin care

The first line of defense in keeping your skin healthy is to take responsibility for your own skin care. If you are at risk for skin breakdown, you will need to develop a daily routine for monitoring and caring for your skin. You should do a complete inspection of your skin every day. If you are unable to assess your own skin, you should be knowledgeable about the areas of your body where you are most vulnerable to skin breakdown and be sure that your care givers are checking these areas for you and reporting the status of your skin.

The most common areas for skin breakdown (pressure points) in adults are the sacrum/coccyx (tailbone), heels, elbows, lateral maleollus (outside of the ankle), greater trocater (hip bone) or the bottom of the femur (outside and inside of the knee) and the ischial tuberosities (the bones we sit on). Pressure points for children are different and based on age and development (7). For infants and children less than three years of age, the head makes up a greater portion of the total body weight and surface areas. When they are placed on their backs, the occipital region (back of the head) becomes the primary pressure point. When placed on their side, the ears are also very susceptible. For older children, the sacrum (lower spine) and calcaneous (the heel of the foot) are most at risk (9).

Teach children to take responsibility for their own skin care

Parents of children at risk for skin breakdown need to be sure to check their children's skin every day. This can become more difficult as children enter their teen years, develop more modesty and are interested (or insistent) on being more independent in their own care. This may be an area that parents need to insist on participation as skin breakdown can progress from minor to serious literally overnight in a child (or in an adult for that matter). If you have made daily skin inspection a part of your child's routine since the onset of paralysis, this should be less of an issue. Be sure that they have the equipment, such as a mirror on a flexible wand, to inspect their own skin with your oversight, if at all possible.

Prevent mechanical Injury

Prevent mechanical injury to the skin from friction and shearing forces during repositioning and transfers. Lift, don't slide. Lowering the head of the bed will help minimize sheer and friction from sliding down in bed. Raise the entire bed up to the proper height to facilitate level surface transfers to and from a wheelchair. If necessary, use assistive devices, such as transfer boards or mechanical lifts to help with transfers. Your physical or occupational therapist can help you with training and obtaining the correct equipment. Ensure that clothing fits comfortably and does not have pressure points, such as snaps, thick seams or pockets. Be sure that clothing is smoothed down under the bottom and back so you don't get pressure points from bunched fabric. Keep bed sheets as wrinkle free as possible.

Keep skin clean and dry

Bathe frequently using mild soap. Avoid very hot water as it dries skin. Dry your skin by patting rather than rubbing. Change undergarments or pads as soon as possible after a bowel or bladder accident.

Eat a healthy diet

Eat a healthy diet and drink lots of fluids, especially water. Keep your body weight in a healthy range. People that are overweight or underweight tend to have more problems with skin breakdown. Good nutrition will help make your skin more resistant to breakdown and you will be more likely to heal and fight off infection should it occur. Eat the right kinds of foods. This means a balanced diet with servings from all food groups. For healthy skin it is especially important to get enough of the following nutrients in your diet:

Omega 3 fatty acids (salmon, mackerel, flaxseed)
Vitamin C (citrus fruits, strawberries, broccoli)
Vitamin A (Vegetables that are dark green or dark orange in color)
Zinc (seafood, meat and eggs)
Protein (meat, eggs, cheese, and soy products)

Extra calories, especially from protein, are important for repairing damaged tissues if you do have skin breakdown. If you are concerned that you do not get enough of these foods in your diet, you can speak with a nutritionist or your health care provider about supplementation.

Develop a good home rehabilitation program

A regular daily therapy program will contribute to your overall health and well being, as well as reduce the risk of skin breakdown. A good program should include therapy to increase muscle mass and strength, improve your flexibility, improve your cardiovascular endurance, and increase your circulation. An activity based program that includes components of weight bearing and/or gait training, functional electrical stimulation biking, as well as strengthening and stretching activities are beneficial to help prevent skin breakdown. Use of the Wii gaming system in creative ways for "Wiihab"

can help with improving strength, balance and endurance. Aquatic therapy and horseback riding therapy are also beneficial, in addition to being fun.

Avoid prolonged pressure on any one spot

Reposition frequently. When seated in a wheelchair, do weight shifts every 15 minutes. When lying in bed, reposition every 2 - 4 hours. Use pillows or wedges behind your back and between bony areas, such as knees and ankles. "Float" your heels and ankles off of the bed by supporting your lower leg with a pillow. Keep the head of the bed up less than 30 degrees to prevent shearing of skin from sliding down or the need to be pulled back up. If you use a wheelchair most of the day, avoid lying on your back at night. Instead, turn side to side to give your backside a break. Better yet, sleep on your stomach, if this position is comfortable and you are able to breathe safely. When positioned on your stomach, you have fewer pressure points, and can generally turn less frequently. Being on your stomach gives your backside a break, and allows you to stretch your hip flexor muscles and hamstring muscles, all for the price of one!

Use therapeutic surfaces

Therapeutic surfaces, such as a pressure relieving wheelchair cushion or a pressure relieving mattress will reduce or relieve pressure, promote blood flow to tissues and enable proper positioning. Make sure that you use equipment the way it is recommended and that it fits correctly. When seated in a wheelchair, make sure the cushion is properly positioned and inflated and that you are sitting all the way back in the wheelchair.

Keep muscle spasms under control

Some muscle spasms can be beneficial as they help you change position, if you can't move yourself. Too much muscle spasticity can cause rubbing and friction, especially when you are in bed at night. Talk with your care provider about how to best manage spasticity. Exercise and range of motion are two good ways to reduce spasticity. Make sure orthotics (braces) are fitting properly, that they are worn correctly, and that the straps are fastened properly to prevent friction or pressure. Be sure that your bladder and bowel programs are working well as increased spasticity can be caused by a urinary tract infection or constipation. Spasticity can also increase when you have a burn or skin breakdown.

IDENTIFYING AND TREATING MINOR SKIN BREAKDOWN

What does minor skin breakdown look like?

Skin breakdown starts out as a red or purple spot on fair skin or a shiny, purple, blue or darker spot on dark skin, which does not fade or go away within 20 minutes. When you press on the spot with your finger, it does not become lighter (blanch). It may feel warmer or cooler than the skin around it. The spot may feel hard or squishy under your fingers and may look swollen. If you have sensation, it may be itchy or painful. At first, it may not look like much, especially if the skin is not broken or open, but it can get a lot worse. If your skin becomes blistered, scabbed or has a small open area on the surface, this is more serious, as it indicates that the tissue underneath has begun to die. At this stage, the progression of skin break down is reversible: the skin will return to normal as soon as the cause of the irritation is found and eliminated and the skin is properly cared for. If these steps are not taken, the damage can rapidly progress to a dangerous level where infection can attack the underlying tissue and bone, posing a serious risk to your health.

Treating redness or minor skin breakdown

If the skin is open, contact your health care provider for wound care instructions. These instructions will generally include cleaning the area with soap and water or a saline solution, keeping the area dry, and eliminating the cause of the problem. Do not use hydrogen peroxide or iodine as these products damage new skin cells in the wound. They may prescribe special dressing that will optimize healing. If not, cover the area with a non-stick dressing, such as a Telfa pad to protect it from clothing. Change the dressing one to two times per day or if it gets soiled. Check your skin frequently to be sure the area is not getting worse. Minor burns can also cause blistering and can be treated in the same manner. If the burn covers a large surface area, you should seek care in the emergency room. Once a treatment plan has been established, you must identify and try to remove the source of the irritation to the affected area as much as possible.

Is the damage being caused by pressure during sitting?

Pressure areas caused by sitting often occur on your ischeal tuberocities (sitting bones), lower back, shoulder blades or the back of the heels. If the skin sore is being caused by sitting, check your wheelchair cushion. Do you have a pressure relieving cushion prescribed by your health care provider? Is it inflated correctly? Some cushions require frequent monitoring of inflation and can leak. Could the cushion be placed backward in the chair? Is it in good repair? Some cushions have gel in them that can get hard or squished out of place. If you have a therapeutic cushion and are still having difficulty with pressure on your sitting bones, see if you can try different cushions with pressure mapping. Pressure mapping equipment allows clinicians to visually identify your specific pressure areas when sitting on

different cushions. Then the cushion that works best for you can be ordered. Pressure mapping can also be helpful, if you have a condition that can make seating difficult. Conditions, such as scoliosis or a dislocated hip can make the pressure on you sitting bones unequal. Depending on how bad the skin breakdown is you may need to stay out of your wheelchair for a period of time to allow the area to heal.

Is the damage being caused by pressure from lying on the area?

The areas most vulnerable when lying are the back of the head (in young children), ankles, knees, hips or shoulder blades. If so, avoid positioning on the affected area. If you have difficulty with red areas despite frequent turning, there are various pressure relieving mattresses that can be prescribed by your health care provider to distribute pressure better than a regular mattress. Unfortunately, if you have never had any skin breakdown, it is difficult to get insurance coverage for this type of specialty mattress. If you have had skin breakdown, coverage is often available.

Does the area of damage look more like a scrape or a tear?

If the area looks more like a scrape or tear, it may be caused by friction or sheer from sliding down in bed or wheelchair or from dragging your bottom with transfers. If you have had a change in your physical status, consider returning to physical therapy for a "tune up" focusing on increasing your strength, flexibility, and transfer technique. If you are dependent on others for part or all of your transfers, there is equipment that can be helpful to prevent sheer injury. This includes transfer boards, starting with simple slippery wood boards to a b-easy board with a sliding disk, a mechanical lifting device or overhead track lift systems. Your physical therapist and occupational therapist can help you identify equipment that will be most helpful for you and teach you and your care givers how to safely use this equipment. A hospital bed that raises and lowers and has an elevating head and foot rest can be helpful, especially if you require assistance from others for bathing, positioning and transfers. The ability to raise and lower the bed will protect your caregiver's back and often allows for level or "downhill" transfers, avoiding sheer injuries. Manual (hand crank) hospital beds or semi-electric hospital beds (hand crank to raise and lower the bed and electric to elevate the head and foot) are often covered by insurance with a good letter of medical necessity from your health care provider. Ask your health care provider to order a fully electric hospital bed, if you are unable to operate a hand crank independently. Your health care provider should indicate that you require assistance with transfers and bed mobility and require frequent repositioning to prevent costly skin breakdown. If you prefer not to have a hospital bed, you can permanently raise the height of your entire bed so that it is even with your wheelchair using blocks of wood, bricks or bed leg adjusters that can be purchased.

Is the area of damage under a brace?

If redness persists greater than 20 minutes after removing the brace, do not wear the brace. Have your therapist or orthotist evaluate the brace to see if it can be adjusted or whether it will need to be remade. Children may need adjustments or replacement of braces as often as every four to six months during growth spurts. Braces will often require adjustment, if you have lost or gained weight, have increased spasticity, decreased range of motion or worsening scoliosis.

Is the redness or breakdown in the diaper (perineal) area?

Skin breakdown in the perineal area is generally caused by too much moisture often from sweating or irritation from urine and stool. Skin problems in this area start out as redness and swelling (rash) and can progress to vesicles or pimples with oozing, crusting or scaling. Once the skin is open, there is increased risk for infection. Perineal skin care should be done as soon as possible after a bowel and/or bladder accident. Gently wash the area with soap that is indicated for the perineal area. Regular bar soap or antibacterial soap used for routine skin care can dry out this skin. The skin in this area will need moisturization with products such as glycerin, lanolin or mineral oil to replace natural moisture that is lost with frequent cleaning. A skin barrier ointment or creams should be used to protect the skin from moisture or irritation.

If the redness or rash last longer than three days, has areas of multiple red bumps or pimples, or if you have oozing pimples that develop into a honey-colored crusted area, you should be seen by a heath professional as you may need treatment for a yeast infection or an antibiotic. Under-pads or absorbent briefs can be used as long as they wick moisture away, rather than trapping the moisture against the skin. Lastly, try to identify the cause of the skin irritation, especially if from frequent bowel or bladder accidents.

IDENTIFYING AND TREATING SERIOUS SKIN BREAKDOWN

What does serious skin breakdown look like?

Serious skin breakdown occurs when the cause of the problem is not eliminated and tissue has been deprived of oxygen and nutrients for so long that the tissue has died and there is now a large hole or crater. Damage extends at least into the subcutaneous tissue. In the most serious wounds, tissue death includes muscle and extends as deep as the bone. Drainage is almost

always present. If you have fever, see green or yellow drainage, and have a warm temperature around the wound, you may have developed an infection. Wounds such as this, with or without infection, must be evaluated by a health care professional. This care may be obtained through your spinal cord injury provider or you may be referred to a wound center to be evaluated and treated by a professional that specializes in the treatment of serious wounds. When an infection forms in a wound, the surrounding tissue and bone can become infected. If this continues, infection can enter the blood stream causing sepsis; and if untreated, can be fatal.

How is serious skin breakdown treated?

If the wound is not infected, you may be able to be treated at home with bed rest and frequent dressing changes. You should be evaluated by a wound care professional to identify the appropriate treatment plan and dressings. Specialized dressings are important to provide a moist environment for healing. Moist wounds heal faster than dry wounds. It is easier for a wound in a moist environment to granulate or grow new cells and for the cells to move across the wound bed. A moist environment increases the effectiveness of white blood cells in fighting infection, removing waste and dead tissue. Specialized dressings are also important to be sure that the wound heals from the inside out. If the wound heals from the outside first, it can trap infection inside that will later flare up and cause the wound to reopen. Deeper wounds need specialized wound care, including the removal of dead tissue either by special wound dressing or ointments (chemical debridement) or surgical removal of this tissue. This will then be followed by special dressings and packing material that can absorb drainage, remove dead tissue and then help the body heal itself. If a wound is draining heavily, a special dressing should be used to contain the drainage.

The most serious wounds, those that extend to the bone or have signs of infection, will require hospitalization for treatment. This will mean several weeks of bed rest with aggressive wound management and IV antibiotics. Aggressive treatment in a hospital that has a specialized wound care team can help you avoid surgery. Newer wound treatment and dressings can speed healing. Many hospital and wound centers use vacuum assisted closure therapy, hyperbaric oxygen therapy and electrical stimulation either directly in the wound or in the tissue surrounding the wound. Electrical stimulation should not be used if there is any chance that the underlying bone may be infected (osteomyelitis). If non-surgical treatment fails, or the wound is very severe, surgery will be required. Most often this will involve a muscle flap and skin grafting to close the wound and prevent reoccurrence of skin breakdown. This type of surgery generally costs thousands of dollars and requires a period of weeks in the hospital followed by several more weeks at home or in the hospital on a specialized bed. The total process is often 6 - 8 or 10 weeks before you can begin a gradual reseating program.

CONCLUSION

While it is well know that preventing skin breakdown is much easier than treatment and that there are many identified risk factors that can be modified to prevent skin breakdown, we know this is only part of the issue. In the real world, even with the best self-care or the best caregivers, you can develop skin breakdown if unexpected events or changes in your life occur. Remember, the threat of skin breakdown never subsides and, in fact, increases with aging and with the length of time from your diagnosis or injury. It is easy to become over confident or even lax in maintaining prevention habits the longer you have gone without having skin breakdown. Be increasingly vigilant with unexpected changes in your circumstance. Changes, such the loss of a trusted care giver or increasing responsibilities at home, work or school, can increase the risk to your skin. Sudden breakage of equipment also places you at increased risk. Being proactive and assertive in monitoring, maintaining and replacing broken or worn out equipment, such as wheelchair cushions is very important. Lastly, if you have a problem with skin breakdown, take charge immediately. Seek out professional help before it becomes serious.

REFERENCE LIST

1. National Pressure Ulcer Advisory Panel. Updated staging system of the national pressure ulcer advisory panel. February, 2007. *www.npuap.org/pr2.htm*.

2. Krause JS, Carter RE, Pickelsimer EE, Wilson D. A prospective study of health and risk of mortality after spinal cord injury. *Arch Phys Med Rehabil*. 2008;89:1482-91.

3. Jackson J, Carlson M, Rubayi S, Scott MD, Atkins MS, Blanche EI, Saunders-Newton C, Mielke S, Wolfe MK, Clark FA. Qualitative study of principles pertaining to lifestyle and pressure ulcer risk in adults with spinal cord injury. *Disabil Rehabil.* 2010;32(7):567-78.

4. Regan MA, Teasell RW, Wolfe DL, Keast D, Mortenson WB, Aubut JA, Spinal Cord Injury Rehabilitation Evidence Research Team. A systematic review of therapeutic interventions for pressure ulcers after spinal cord injury. *Arch Phys Med Rehabil.* 2009;90(2):213-31.

5. Garber SL, Rintala DH. Pressure ulcers in veterans with spinal cord injury: a retrospective study. *J Rehabil Res Dev.* 2003;40(5):433-41.

6. Jackson J, Carlson M, Rubayi S, Scott MD, Atkins MS, Blanche EI, Saunders-Newton C, Mielke S, Wolfe MK, Clark FA. Qualitative study of principles per-

taining to lifestyle and pressure ulcer risk in adults with spinal cord injury. *Disabil Rehabil*. 2010;32(7):567-78.

7. Butler CT. Pediatric skin care: Guidelines for assessment, prevention, and treatment. *Ped Nurs*. 2006;32(5):443-450.

8. National Pressure Ulcer Advisory Panel. The role of nutrition in pressure ulcer prevention and treatment: National pressure ulcer advisory panel white paper. 2009. *http://www.npuap.org/*

9. Baharestani MM, Ratliff CR. Pressure ulcers in neonates and children: An NPUAP white paper. *Advan Skin & Wound Care*. 2007;20(4):208-220.

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Clinically isolated acute transverse myelitis: prognostic features and incidence

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Abstract

Background Demyelinating acute transverse myelitis (ATM) may be the first presentation of multiple sclerosis or remain a clinically isolated syndrome. North Canterbury, New Zealand provides a well circumscribed population to study ATM.

Objective To identify prognostic features, clinical outcomes and incidence of ATM in North Canterbury, New Zealand.

Methods All patients with ATM as a first neurological presentation diagnosed from January 2001 to December 2005 at a single institution providing all neurological care for North Canterbury were assessed for clinical data, MRI findings, CSF results and clinical outcomes. CHAMPS, Barkhof/Tintore and Swanton criteria were applied to brain MRI.

Results Sixty-one patients were identified with a mean duration of follow-up of 30 ± 17 months. 50% of patients with ATM with brain lesions by CHAMPS criteria converted to clinically definite multiple sclerosis (CDMS). No patients with idiopathic ATM converted to CDMS. There was a strong association with conversion to CDMS and abnormal brain MRI by CHAMPS criteria (hazard ratio, 5.63; 1.83-17.3), Barkhof/Tintore criteria (hazard ratio, 6.43; 2.31-17.9) and Swanton criteria (hazard ratio, 4.53; 1.67-12.3). The age standardised annual incidence of ATM was 24.6 (18.2-31.1) per million, of definite and possible idiopathic ATM was 6.2 (2.9-9.6) per million, and of ATM with brain lesions was 4.7 (1.9-7.6) per million.

Conclusion Patients with idiopathic ATM are at low risk for conversion to CDMS. Abnormal brain MRI by CHAMPS criteria is a sensitive predictor of conversion to CDMS. The annual incidence of ATM in North Canterbury, New Zealand is significantly higher than previously reported.

Introduction

Acute transverse myelitis (ATM) manifests as sensory, motor or autonomic dysfunction due to inflammation of the spinal cord. The etiology of non-compressive acute transverse myelopathy may be classified as delayed radiation effect, spinal cord infarction, parainfectious, systemic autoimmune disease, multiple sclerosis (MS) or idiopathic [1]. Diagnostic criteria for idiopathic ATM were proposed by the Transverse Myelitis Consortium Working Group (TMCWG) [2]. These include bilateral signs or symptoms of spinal cord dysfunction, a clearly defined sensory level, inflammation within spinal cord demonstrated by CSF pleocytosis or elevated IgG index or gadolinium enhancement, and progression to nadir between four hours and twenty-one days.

Increasing use of brain MRI to demon-

strate demyelinating lesions disseminated in time and space led to new diagnostic criteria for MS incorporating these findings [3,4]. Applying these criteria to brain MR imaging to define lesions disseminated in space is predictive of conversion to clinically definite MS (CDMS) [5].

Patients with idiopathic ATM and MS associated ATM are at risk of further demyelinating events. Patients with ATM as a clinically isolated syndrome (CIS) who subsequently develop a second event disseminated in time and place meet criteria for CDMS [3]. Patients at higher risk of conversion to CDMS include those with unilateral signs or symptoms without a clear sensory level, those with spine MRI lesions measuring less than two segments and those with oligoclonal bands identified on CSF examination [6-8].

The incidence of ATM of all causes is between 1.34 to 4.6 per million per year [9,10]. The incidence of idiopathic and MS associated ATM are both approximately 1.0 per million per year in Albuquerque, New Mexico [10]. The incidence of demyelinating disease is known to vary geographically but there are no other estimates of incidence of idiopathic ATM [11]. The aim of our study was to identify predictors and risk of conversion to CDMS in a community based series of clinically isolated ATM patients, and to estimate the incidence of idiopathic and MS associated ATM in a well circumscribed New Zealand population.

Patients and methods

Setting

The Department of Neurology at Christchurch Public Hospital is the only neurology referral centre in the North Canterbury area of New Zealand and provides neurology care to a population of 430,000. The diagnoses of all patients seen by the Department of Neurology are maintained in the prospective Neurology Department Database (NDD). All neurologists practicing in the Canterbury region of New Zealand during the time of this study practiced at Christchurch Public Hospital and they registered all outpatients and inpatients on the NDD.

Patients

All patients diagnosed with acute transverse myelitis by a consultant neurologist between January 1, 2001 and December 31, 2005 were retrospectively identified from the NDD. All patients must have undergone spinal MR imaging. Exclusion criteria were an identifiable compressive, radiation, ischaemic, infectious or systemic autoimmune cause for myelopathy, previous symptoms consistent with demyelination at any site or a progression to nadir less than four hours or greater than 21 days as per the TMCWG criteria [2]. Demographic data, symptoms, signs, CSF examination results, follow-up and clinical outcomes were obtained from the case records. Oligoclonal banding (OCB) was tested by electrophoresis with immunofixation. Further tests including CSF virology and autoimmune screening were performed at the discretion of the treating neurologist. Conversion to CDMS was defined as a second neurological event consistent with demyelination at a different site or spinal level. Further follow-up and clinical information were obtained from primary care physicians and direct patient review.

Classification

Patients meeting the TMCWG criteria, using CSF white cell count greater than five as evidence of pleocytosis, were categorised as definite idiopathic ATM [2]. Patients who had no CSF or MRI evidence of inflammation but otherwise met these criteria were categorised as possible idiopathic ATM. Patients with bilateral signs or symptoms and clearly defined sensory level with abnormal brain MRI by CHAMPS criteria [12] (two or more brain lesions at least three mm in diameter with at least one lesion being periventricular or ovoid) were categorised as ATM with brain lesions. Patients presenting with unilateral signs or symptoms or without a clear sensory level, with normal brain MRI by CHAMPS criteria were categorised as partial ATM without brain lesions. Patients fulfilling criteria for partial ATM with abnormal brain MRI by CHAMPS criteria were categorised as partial ATM with brain lesions. Patients with incomplete records of progression to nadir were categorised as ATM with incomplete data.

MRI acquisition and analysis

MR images were acquired on 1.5 Tesla scanners. Spine and brain MR images were retrospectively reviewed by a neuroradiologist (MH) blinded to the clinical details. Brain MRI findings were classified according to the Barkhof/Tintore criteria [13,14] as used in the McDonald criteria [3] (three of the following four features: one or more gadolinium enhancing lesions or nine T2 hyperintense lesions; one or more infratentorial lesions; one or more juxtacortical lesions; or three or more periventricular lesions), the Swanton criteria [15] (at least one T2 hyperintense lesion in two or more of the following regions; periventricular, juxtacortical, infratentorial), and CHAMPS criteria [12] (as above). Intra-rater reliability (IRR) was assessed by blinded review of 10 randomly selected brain and spine scans with an IRR of 0.97 for reported MRI measures.

Incidence

Identified patients residing outside the North Canterbury region were excluded from the incidence calculation but not from other analyses. Population figures for the North Canterbury region were taken from the 2001 New Zealand census. All incidence results were age standardised to the world population [16].

Statistical analysis

Survivorship functions were obtained using Kaplan-Meier analysis and differences in survival were measured by the log-rank test. All hazards ratios were calculated using Cox proportional hazards models. As all males were OCB -ve we adjusted for age only in these instances. When the outcome-exposure contingency tables contained a zero cell in the multivariable analysis the logistic models did not converge and we reported Fisher exact p□values. Otherwise, all outcomes were adjusted for age and sex and presented with 95% confidence intervals. There was no evidence that the proportional hazards assumption was violated and model adequacy was verified using the May□Hosmer goodness-of-fit test [17]. This test identifies potential problems in fit after using a Cox proportional hazards model by partitioning the data into deciles of risk and comparing observed and expected predicted numbers of events. All analyses were undertaken using Stata version 10.0.

Results

A total of 61 patients were identified with a first diagnosis of ATM made by a consultant neurologist. The clinical characteristics of this group and the subgroups are shown in table 1. Ten patients had incomplete data regarding progression to nadir. Had their progression to nadir met the TMCWG criteria, four would have been categorised as definite idiopathic ATM, one as partial ATM without brain lesions and five as partial ATM with brain lesions. There was no statistical difference in the demographic characteristics between these 10 patients and the remaining group.

Investigations

The investigation characteristics are shown in table 1.

Table 1. Demographic, clinical and investigation characteristics by subgroup

	All patients $n=61$	Definite and possible idiopathic ATM $n=15$	Partial ATM without brain lesions $n=17$	ATM with brain lesions $n=12$	Partial ATM with brain lesions $n=7$
Age in years (mean ± SD)	36.8 ± 11.1	35.6 ± 12.2	35.5 ± 6.9	34.3 ± 10.2	38.7 ± 16.7
Male/female	13/48	2/13	6/11	1/11	2/5
Presenting symptom					
Motor deficit	2 (3%)	0 (0%)	0 (0%)	0 (0%)	1 (14%)
Sensory	34 (56%)	8 (53%)	9 (53%)	6 (50%)	3 (43%)
Sensorimotor	25 (41%)	7 (47%)	8 (47%)	6 (50%)	3 (43%)
Sphincter disturbance	13 (21%)	4 (27%)	2 (12%)	3 (25%)	1 (14%)
Sensory level					
Cervical	7 (11%)	0 (0%)	4 (24%)	1 (8%)	0 (0%)
Thoracic	34 (56%)	12 (80%)	9 (53%)	8 (67%)	2 (29%)
Lumbosacral	10 (16%)	3 (20%)	1 (6%)	3 (25%)	1 (14%)
No sensory level	10 (16%)	0 (0%)	3 (18%)	0 (0%)	4 (57%)
Investigations					
Spine MRI performed	61 (100%)	15 (100%)	17 (100%)	12 (100%)	7 (100%)
Images available for review	55 (90%)	14 (93%)	15 (88%)	12 (100%)	7 (100%)
Gadolinium administered	15 (25%)	3 (20%)	6 (35%)	3 (25%)	1 (14%)
Gadolinium +ve lesion	10 (16%)	3 (20%)	3 (18%)	2 (17%)	1 (14%)
Brain MRI performed	50 (82%)	11 (73%)	12 (71%)	12 (100%)	7 (100%)
Two or more white matter lesions	24 (48%)	0 (0%)	0 (0%)	12 (100%)	7 (100%)
Normal brain MRI	26 (52%)	11 (100%)	12 (100%)	0 (0%)	0 (0%)
CSF examined	41 (67%)	13 (87%)	9 (53%)	9 (75%)	3 (43%)
Protein > 0.4 g/L	17 (41%)	7 (54%)	3 (33%)	5 (56%)	0 (0%)
CSF WCC > 5	25 (61%)	8 (62%)	3 (33%)	7 (78%)	2 (67%)
Oligoclonal bands present	20 (49%)	2 (15%)	3 (33%)	6 (67%)	3 (100%)
CSF index > 0.6 g/L	24 (59%)	4 (31%)	5 (56%)	6 (67%)	3 (100%)

ATM, acute transverse myelitis; CSF, cerebrospinal fluid; WCC, white cell count.

Table 2. Conversion to clinically definite MS and age standardized annual incidence by subgroup

	All patients $n=61$	Definite and possible idiopathic ATM $n=15$	Partial ATM without brain lesions $n=17$	ATM with brain lesions $n=12$	Partial ATM with brain lesions $n=7$
Conversion to CDMS	22/61 (36%)	0/15 (0%)	7/17 (41%)	6/12 (50%)	5/7 (71%)
Follow-up in months (mean ± SD)	30 ± 17	26 ± 18	25 ± 17	34 ± 15	30 ± 19
Age standardized annual incidence					
North Canterbury residents	$n=58$	$n=14$	$n=16$	$n=11$	$n=7$
Overall (95% CI)	24.6 (18.2–31.1)	6.2 (2.9–9.6)	6.6 (3.3–9.9)	4.7 (1.9–7.6)	3.2 (0.8–5.6)
Male (95% CI)	9.7 (4.1–15.3)	1.2 (0–3.4)	4.5 (0.9–8.0)	0.8 (0–2.3)	1.8 (0–4.4)
Female (95% CI)	38.9 (27.4–50.4)	11.0 (4.9–17.2)	8.7 (3.2–14.2)	8.6 (3.1–14.0)	4.5 (0.4–8.6)

ATM, acute transverse myelitis; CI, confidence interval; CDMS, clinically definite multiple sclerosis.

Conversion to clinically definite MS
The conversion to CDMS and mean follow up are shown in table 2. Four patients categorised as ATM with incomplete data converted to CDMS. Had their progression to nadir met TMCWG criteria three would have been categorised as partial ATM with brain lesions and the fourth as definite idiopathic ATM. Overall conversion and conversion by subgroup utilising KM survival analysis are shown in figure 1. The conversion rate of definite and possible idiopathic ATM was different from ATM with brain lesions ($p = 0.003$), partial ATM with brain lesions ($p < 0.001$) and partial ATM without brain lesions ($p = 0.007$). The conversion rates of ATM with brain lesions, partial ATM with brain lesions and partial ATM without brain lesions did not statistically differ.

Incidence
Three patients resided outside the North Canterbury region at the time of diagnosis. For the remaining patients age standardised annual incidence is shown in table 2.

Application of different MRI brain criteria
Univariable analysis of the different criteria are shown in table 3.

Others predictors of conversion
Univariable analysis of MRI spine and CSF characteristics are shown in table 3.

Multivariable outcome analysis
Bivariable and multivariable analysis of the three factors associated with relapse (OCB +ve, abnormal brain MRI, ≥ two spinal lesions) and the three protective factors (normal brain, single spinal lesion, OCB –ve) are shown in table 4.

Discussion

Our annual incidence of idiopathic and MS associated ATM combined, of 10.8 per million, is approximately five times higher than that of Albuquerque, New Mexico and two times higher than previous estimates of ATM of all causes [9,10]. These earlier studies did not include partial ATM with or without brain lesions and so we exclude these patients in this comparison. The incidence of demyelinating disease is known to vary according to latitude and North Canterbury, latitude 43°S, is further from the equator than New Mexico, latitude 35°N, and Israel, latitude 31°N. Our finding of an overall annual incidence of clinically isolated ATM of 24.6 per million is consistent with the known high inci-

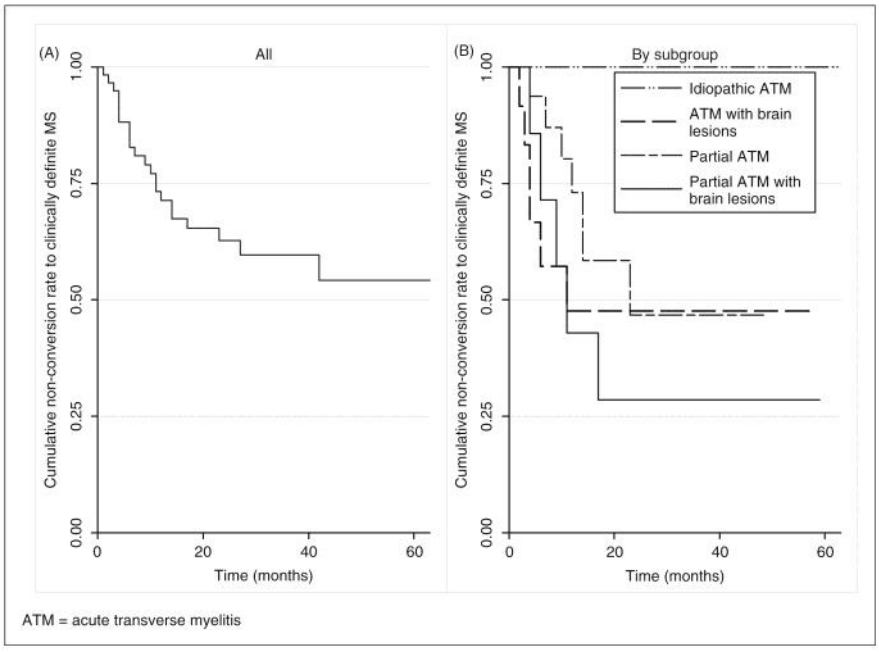

Figure 1. Conversion to clinically definite multiple sclerosis from diagnosis of acute transverse myelitis.

dence of demyelinating disease in Canterbury [18]. A latitudinal gradient of CIS, including ATM, has also been reported from Australia [11].

Idiopathic ATM is identified as a distinct subgroup of transverse myelitis. Earlier studies demonstrate clinical and radiological characteristics different to those of MS associated ATM. The spine MRI appearances of the lesions of idiopathic ATM are longer than those of MS associated ATM [7]. Idiopathic ATM has a conversion rate to CDMS of close to 0% compared with the up to 83% conversion rate of patients with CIS, including ATM, and an abnormal brain MRI [19,20]. However, these early studies of idiopathic ATM used varying definitions. This heterogeneity and the need for further research prompted the TMCWG to propose uniform diagnostic criteria for idiopathic ATM [2]. The criteria emphasise a clinical history and examination consistent with spinal cord inflammation, confirmation of inflammation by CSF or MRI examination and exclusion of other demonstrable etiology. MS associated ATM is differentiated from idiopathic ATM by the criterion "brain MRI abnormalities suggestive of MS". Partial ATM is differentiated by the necessity to demonstrate bilateral signs or symptoms and a clearly defined sensory level.

These criteria identified patients who meet the clinical criteria of idiopathic ATM but without confirmed inflammation [21]. After discussion with the TMCWG these patients were categorised as possible idiopathic ATM and included with definite idiopathic ATM in their analysis. Studies using these new criteria still define a patient population with a low conversion rate to CDMS [21,22]. Our study confirms that patients with idiopathic ATM are at low risk of conversion to CDMS with none of our patients progressing to a second demyelinating event within the timeframe of this study.

Early case series of ATM included only patients with bilateral signs and clearly defined sensory levels [9,23]. Patients with unilateral signs or without sensory levels were classified as partial ATM and excluded. Recent series specifically address partial ATM and demonstrate a strong association between this presentation and brain MRI suggestive of MS [24]. Our study identifies 24 patients with unilateral signs or without sensory levels, of whom 19 had brain MRI performed and seven had abnormalities consistent

Table 3. Outcome analysis*

Variables	Number in analysis	Number positive for variable	Hazard ratio for recurrence	95% CI
MRI brain variables				
Normal MRI brain	50	26	0.18	0.06–0.55
Meet CHAMPS criteria	50	24	5.63	1.83–17.3
Meet Swanton criteria	50	20	4.53	1.67–12.3
Meet Barkhof/Tintore criteria	50	11	6.43	2.31–17.9
MRI spine variables				
Normal MRI spine	55	10	0.53	0.17–1.64
1 lesion	55	23	0.27	0.08–0.98
1 lesion < 2 segments	55	18	0.22	0.05–0.95
1 lesion > 2 segments	55	5	1.31	0.16–10.9
1 or 0 lesions	55	33	0.24	0.09–0.63
2 or more lesions	55	22	4.22	1.59–11.2
Gd +ve lesion	15	10	3.14	0.33–29.7
Cord swelling	55	25	1.23	0.50–3.0
CSF variables				
OCB +ve†	41	20	9.0	1.93–42.3
OCB -ve†	41	21	0.11	0.02–0.51
IgG index elevated	41	24	7.1	1.49–33.4
CSF WCC >5†	41	25	3.5	0.75–16.4

*All outcomes have been adjusted also for age and sex apart from those marked.
†Adjusted for age only.
CI, confidence interval; CSF, cerebrospinal fluid.
Note: six spinal MRIs were unavailable for review.

Table 4. Bivariable and multivariable analysis of factors associated with relapse and protective from relapse

	All factors present relapsed/not relapsed	One or more factors absent relapsed/not relapsed	Hazards Ratio for recurrence	95% CI	Fisher exact *p*-value
Factors associated with relapse					
AB & OCB +ve†	8/5 (62%)	4/16 (20%)	3.10	0.92–10.5	0.027
AB & ≥2CL*	10/2 (83%)	8/29 (22%)	9.81	3.33–28.9	<0.0001
OCB +ve & ≥2CL†	4/2 (67%)	9/22 (29%)	2.56	0.76–8.63	0.157
AB & OCB +ve & ≥2CL‡	4/0 (100%)	8/20 (29%)	NC	NC	0.014
Factors protective from relapse					
NB & OCB -ve‡	0/12 (0%)	12/9 (57%)	NR	NR	0.002
NB & ≤1 CL*	3/17 (15%)	15/14(52%)	0.17	0.05–0.59	0.015
≤1CL & OCB -ve‡	0/14 (0%)	13/10 (57%)	NC	NC	<0.0001
NB & OCB -ve & ≤1 CL‡	0/11 (0%)	12/9 (57%)	NC	NC	0.002

*Adjusted also for age and sex.
†Adjusted for age only.
‡Fisher exact *p*-value reported as models did not converge due to a zero cell. Hazard ratios cannot be calculated in these instances.
NB, normal brain; AB, abnormal brain (at least two lesions consistent with MS lesions); CL, cord lesion; OCB, oligoclonal bands.

with MS by CHAMPS criteria. Previous studies of patients meeting our definition of partial ATM without brain lesions demonstrate a rate of conversion to CDMS between 15-44% [25-27]. Our conversion rate of 41% is similar to these studies. In studies of patients meeting our definition of partial ATM with brain lesions the conversion rate was 44-93% [6,27,28]. Our conversion rate of 71% is similar and confirms that this group has the highest risk of early conversion to CDMS.

The subgroup of MS associated ATM is not well defined. The TMCWG criteria exclude any patient with brain MRI abnormalities suggestive of MS but do not define what these are. The McDonald criteria include the Barkhof/Tintore criteria for brain MRI to demonstrate dissemination in space or, alternatively, the presence of two or more MRI lesions consistent with MS when associated with a positive CSF. Newer MRI criteria were proposed by Swanton et al. in 2006 [15]. We used the simpler CHAMPS definition of two or more white matter lesions to define abnormal brain MRI and demonstrate that these criteria identify more patients at risk of conversion than Swanton or Barkhof/Tintore criteria, with similar hazard ratios. Conversely, excluding patients from the idiopathic ATM group by using the CHAMPS criteria identifies a group at low risk of conversion. The CHAMPS criteria are simple to apply and we demonstrate that their use in brain MRI, along with accurate categorisation using clinical features of ATM, provides strong prognostic information regarding conversion to CDMS.

Presence of oligoclonal banding on CSF examination varies according to category of ATM. Oligoclonal banding was previously demonstrated in 17% of patients with definite and possible idiopathic ATM compared with 62% of patients with partial ATM without brain lesions and 62.5% of partial ATM with and without brain lesions [21,25,26]. Our results confirm these findings, with the lowest percentage in the idiopathic ATM group and higher percentages in the partial ATM without brain lesions and ATM with brain lesions groups. The small number of CSF examinations performed in the partial MS associated group reflects local clinical practice. Oligoclonal banding is associated with increased risk of conversion to CDMS after clinically isolated ATM and our results confirm this association [8,28].

Our findings do not confirm the greater risk of conversion to CDMS associated with spinal lesions less than two vertebral bodies long. Longitudinally extensive cord lesions did not provide a protective effect, but, with only five cases with this finding, the interpretation is limited. None of the patients were thought to have neuromyelitis optica according to Wingerchuk criteria [29]. NMO antibody testing was not performed as the test was not available to us at the time.

Shortcomings of this retrospective study include the variation in investigations, with brain MRI and CSF examinations not performed on all patients. Follow-up MR imaging was not routinely performed and so we have not assessed dissemination of lesions in time to fulfill criteria for MS. The clinical follow-up was not uniform, of moderate duration, and reliant on contact with primary care physicians to identify further neurological events in some cases. However, demyelinating disease is a common disorder

in our community and there is a high level of awareness of the association of spinal cord syndromes with MS among neurologists, neurosurgeons and general practitioners. It is very unlikely that a person with a spinal cord syndrome would not be referred to the specialist neurology services. Due to the lack of standardized follow up we are unable to comment on the degree of recovery from ATM.

This study provides an overview of the clinical outcomes of clinically isolated ATM in a community based neurological service in North Canterbury, New Zealand. It shows the utility of applying CHAMPS MRI criteria to this patient population to predict conversion to CDMS and emphasises the good prognosis in the short term of those with definite or possible idiopathic ATM in relation to conversion to MS. It also demonstrates that ATM incidence may vary geographically as does the incidence of MS.

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References

1. de Seze J, Stojkovic T, Breteau G, Lucas C, Michon-Pasturel U, Gauvrit JY *et al*. Acute myelopathies: Clinical, laboratory and outcome profiles in 79 cases. *Brain* 2001; **124**: 1509-21.

2. Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. *Neurology* 2002; **59**: 499-505.

3. McDonald WI, Compston A, Edan G, Goodkin D, Hartung HP, Lublin FD *et al*. Recommended diagnostic criteria for multiple sclerosis: guidelines from the International Panel on the diagnosis of multiple sclerosis. *Ann Neurol* 2001; **50**: 121-27.

4. Polman CH, Reingold SC, Edan G, Filippi M, Hartung HP, Kappos L *et al*. Diagnostic criteria for multiple sclerosis: 2005 revisions to the "McDonald Criteria". *Ann Neurol* 2005; **58**: 840-46.

5. Tintore M, Rovira A, Rio J, Nos C, Grive E, Sastre-Garriga J *et al*. New diagnostic criteria for multiple sclerosis: application in first demyelinating episode. *Neurology* 2003; **60**: 27-30.

6. Ford B, Tampieri D, Francis G. Long-term follow-up of acute partial transverse myelopathy. *Neurology* 1992; **42**: 250-52.

7. **Bakshi R, Kinkel PR, Mechtler LL, Bates VE, Lindsay BD, Esposito SE** *et al*. Magnetic resonance imaging findings in 22 cases of myelitis: comparison between patients with and without multiple sclerosis. *Eur J Neurol* 1998; **5**: 35-48.

8. Sharief MK, Thompson EJ. The predictive value of intrathecal immunoglobulin synthesis and magnetic resonance imaging in acute isolated syndromes for subsequent development of multiple sclerosis. *Ann Neurol* 1991; **29**: 147-51.

9. Berman M, Feldman S, Alter M, Zilber N, Kahana E. Acute transverse myelitis: incidence and etiologic considerations. *Neurology* 1981; **31**: 966-71.

10. Jeffery DR, Mandler RN, Davis LE. Transverse myelitis. Retrospective analysis of 33 cases, with differentiation of cases associated with multiple sclerosis and parainfectious events. *Arch Neurol* 1993; **50**: 532-35.

11. Lucas R, Taylor BV, Ponsonby A, Chapman C, Coulthard A, Dear K *et al*. Latitudinal variations in incidence of first demyelinating events; descriptive analyses of case participants in the Ausimmune Study. *Mult Scler* 2008; **14(supp 1)**: S190 –S191. Abstract.

12. Jacobs LD, Beck RW, Simon JH, Kinkel RP, Brownscheidle CM, Murray TJ *et al*. Intramuscular interferon beta-1a therapy initiated during a first demyelinating event in multiple sclerosis. CHAMPS Study Group. *N Engl J Med* 2000; **343**: 898-904.

13. Barkhof F, Filippi M, Miller DH, Scheltens P, Campi A, Polman CH *et al*. Comparison of MR imaging criteria at first presentation to predict conversion to clinically definite multiple sclerosis. *Brain* 1997; **120**: 2059-69.

14. Tintore M, Rovira A, Martinez MJ, Rio J, Díaz-Villoslada P, Brieva L *et al*. Isolated demyelinating syndromes: comparison of different MR imaging criteria to predict conversion to clinically definite multiple sclerosis. *Am J Neuroradiol* 2000; **21**: 702-06.

15. Swanton JK, Fernando K, Dalton CM, Miszkiel KA, Thompson AJ, Plant GT *et al*. Modification of MRI criteria for multiple sclerosis in patients with clinically isolated syndromes. *J Neurol Neurosurg Psychiatry* 2006; **77**: 830-33.

16. Doll R, Cook P. Summarizing indices for comparison of cancer incidence data. *Int J Cancer* 1967; **2**: 269-79.

17. May S, Hosmer DW. A simplified method of calculating an overall goodness-of-fit test for the Cox proportional hazards model. *Lifetime Data Anal* 1998; **4**: 109-20.

18. Taylor BV, Richardson A, Mason DF, Willoughby E, Abernethy D, Sabel C. Prevalence of multiple sclerosis in New Zealand. *Mult Scler* 2008; **14(suppl 1)**: S202. Abstract.

19. Al Deeb SM, Yaqub BA, Bruyn GW, Biary NM. Acute transverse myelitis. A localized form of postinfectious encephalomyelitis. *Brain* 1997; **120**: 1115-22.

20. O'Riordan JI, Thompson AJ, Kingsley DP, MacManus DG, Kendall BE, Rudge P *et al*. The prognostic value of brain MRI in clinically isolated syndromes of the CNS. A 10-year follow-up. *Brain* 1998; **121**: 495-503.

21. de Seze J, Lanctin C, Lebrun C, Malikova I, Papeix C, Wiertlewski S *et al*. Idiopathic acute transverse myelitis: application of the recent diagnostic criteria. *Neurology* 2005; **65**: 1950-53.

22. Bruna J, Martinez-Yelamos S, Martinez-Yelamos A, Rubio F, Arbizu T. Idiopathic acute transverse myelitis: a clinical study and prognostic markers in 45 cases. *Mult Scler* 2006; **12**: 169-73.

23. Lipton HL, Teasdall RD. Acute transverse myelopathy in adults. A follow-up study. *Arch Neurol* 1973; **28**: 252-57.

24. Scott TF, Bhagavatula K, Snyder PJ, Chieffe C. Transverse myelitis. Comparison with spinal cord presentations of multiple sclerosis. *Neurology* 1998; **50**: 429-33.

25. Cordonnier C, de Seze J, Breteau G, Ferriby D, Michelin E, Stojkovic T *et al*. Prospective study of patients presenting with acute partial transverse myelopathy. *J Neurol* 2003; **250**: 1447-52.

26. Scott TF, Kassab SL, Singh S. Acute partial transverse myelitis with normal cerebral magnetic resonance imaging: transition rate to clinically definite multiple sclerosis. *Mult Scler* 2005; **11**: 373-77.

27. Sellner J, Luthi N, Buhler R, Gebhardt A, Findling O, Greeve I *et al*. Acute partial transverse myelitis: risk factors for conversion to multiple sclerosis. *Eur J Neurol* 2008; **15**: 398-405.

28. Miller DH, Ormerod IE, Rudge P, Kendall BE, Moseley IF, McDonald WI. The early risk of multiple sclerosis following isolated acute syndromes of the brainstem and spinal cord. *Ann Neurol* 1989; **26**: 635-39.

29. Wingerchuk DM, Lennon VA, Pittock SJ, Lucchinetti CF, Weinshenker BG. Revised diagnostic criteria for neuromyelitis optica. *Neurology* 2006; **66**: 1485-89.

Neurosarcoidosis: Clinical, Pathological and Therapeutic Issues

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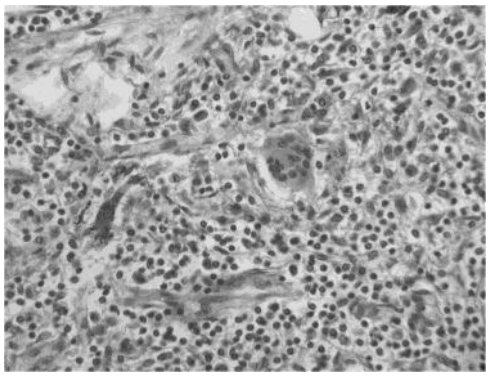
Adapted from a presentation at the Rare Neuroimmonologic Disorders Symposium, July 2006; special thanks to Benjamin Wendt, a medical student at The Ohio State University, who provided significant assistance in editing Dr. Pardo's article.

This paper will describe the Johns Hopkins TM and MS Centers' experience regarding the clinical, pathological, and treatment issues related with neurosarcoidosis. Sarcoidosis is a multisystemic disease. It is important for us to understand Sarcoidosis, because when it affects the brain or the spinal cord, it mimics disorders like multiple sclerosis and transverse myelitis. We are trying to advise physicians that sarcoidosis is a disease that needs to be evaluated carefully before establishing a diagnosis of multiple sclerosis or transverse myelitis.

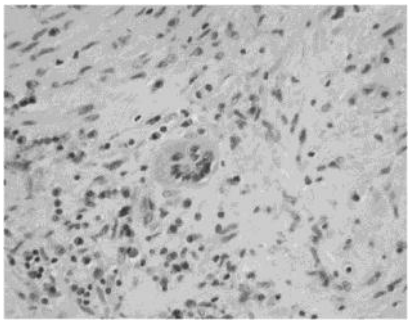
Sarcoidosis: Lung Granuloma

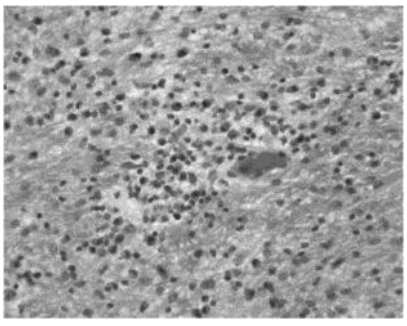


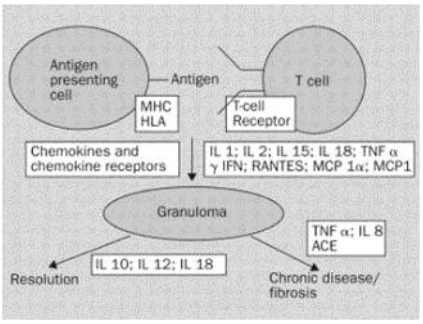
Sarcoidosis is a multisystemic granulomatous disorder. Multisystemic means that it may affect different parts of the body or different organs. It is primarily a disease that affects the lungs and the lymph nodes. The lymph nodes are intimately related with immunological function. The etiology is unknown; at present we do not know what causes this disease. It is a devastating disease in some patients that can produce a multitude of symptoms. Some of the symptoms include chronic coughing, malaise, episodic sweating and fever, fatigue, dyspnea and weight loss.

Sarcoidosis is frequently found when physicians are evaluating x-rays of the chest. Visualization of enlarged lymph nodes in the lungs leads to further assessment by tissue biopsy. Biopsies of the lymphatic tissue result in the identification of granulomas which are the hallmark of Sarcoidosis.

Sarcoidosis: Immune Response







There currently is no direct evidence regarding the factors that might cause Sarcoidosis. There is some speculation and experimental evidence, however, that Sarcoidosis may be an infectious disease caused by microorganisms. It is possible that persistent infections of bacteria may contribute to the symptoms seen in the lymph nodes and the lungs.

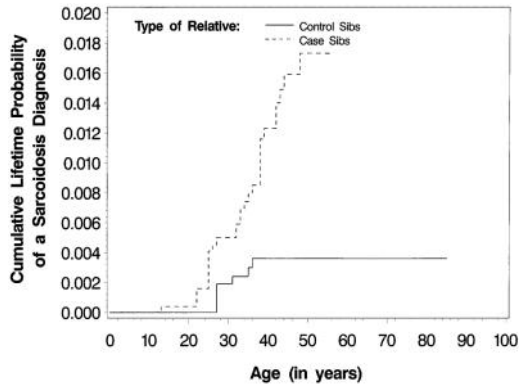
It is very well known that there is some type of genetic susceptibility to Sarcoidosis. It has been demonstrated that there are populations that have major histocompatibility complex (MHC) markers that are associated with a high risk of this disorder and populations that have markers that are associated with a low risk for this disorder. It is also known that polymorphisms in the cytokine family of genes may be involved in genetic susceptibility.

Sarcoidosis: Epidemiology

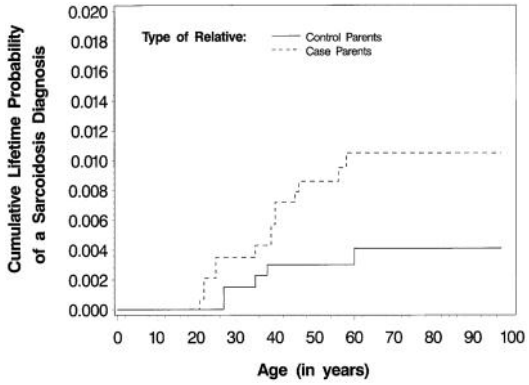
- **Etiological factors**:
  - Mycobacteria (19 studies, 0%-89% positivity rate)
  - *Propionibacterium acnes*
  - *Propionibacterium granulosum*
- **Genetic susceptibility**
  - MHC
    - ☯Risk (HLA DR 11,12,14,15,17)
    - ॐRisk (HLA Dr1, DR4, DQ*0202?)
  - TNFα (The -308)
  - ACE (intron 16, -267)

In the southeast and northeast United States, there are increased numbers of patients with sarcoidosis. Sarcoidosis has also been found to be more prevalent among the African-American population and among Caucasians of Nordic ancestry. We don't understand the increased prevalence of sarcoidosis in these populations, and that raises some concerns about potential issues like environmental risk or the presence of some familial clustering of sarcoidosis.

Risk of Sarcoidosis in Siblings (ACCESS study) Source: ACCESS Study, Am J Respir Crit Care Med 164:1885, 2001

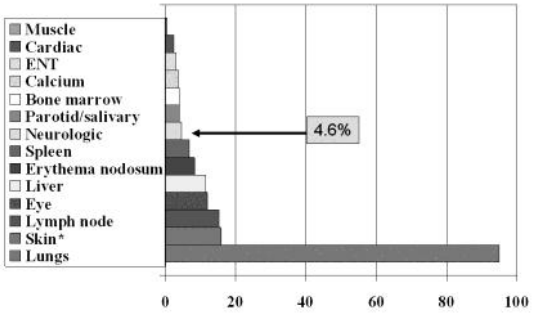


Risk of Sarcoidosis in Parents (ACCESS study) Source: ACCESS Study, Am J Respir Crit Care Med 164:1885, 2001



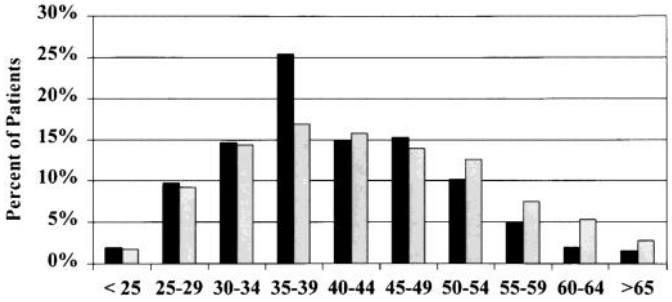
There is a very large longitudinal study on sarcoidosis that was done at the end of the 1990's and was completed in early 2000. The study demonstrated that there is a cumulative risk of sarcoidosis in relatives of patients that have suffered the disease. Interestingly, there is also a potential of sarcoidosis cases in parents of people that have been affected with this disorder.

Sarcoidosis: Organ Involvement (ACCESS: 736 patients 1997 – 1999) Source: ACCESS Study, Am J Respir Crit Care Med 164:1885, 2001

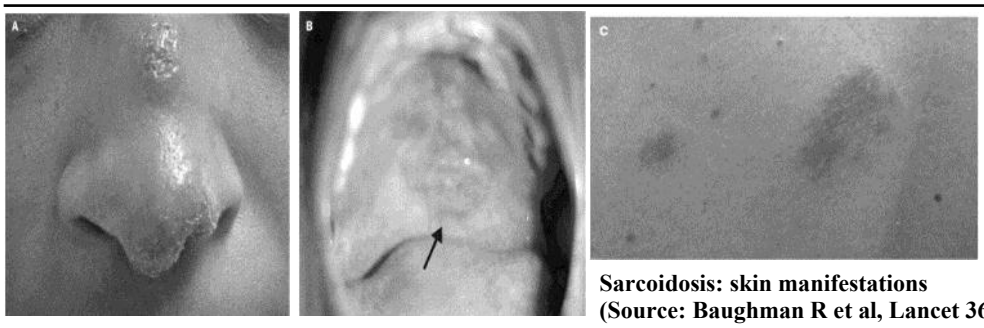


This graphic depicts the group of patients in the ACCESS sarcoidosis study, the largest longitudinal study of sarcoidosis that has been done in the United States, and the population that may be at risk for neurological problems. Neurological involvement was found in 4.6% of the overall population, which is not a high percentage, but the neurological involvement was very aggressive and produced a lot of disability due to the presence of spinal cord involvement, or peripheral nerve involvement, or encephalitis. The graphic makes clear that while Sarcoidosis predominantly affects the respiratory system, it can impact all the areas of the body, including the skin, eyes, liver, and many other organs. This is the reason we call Sarcoidosis a multi-systemic disorder.

Sarcoidosis: Age and sex at diagnosis (ACCESS: 736 patients 1997 – 1999) Source: ACCESS Study, Am J Respir Crit Care Med 164:1885, 2001

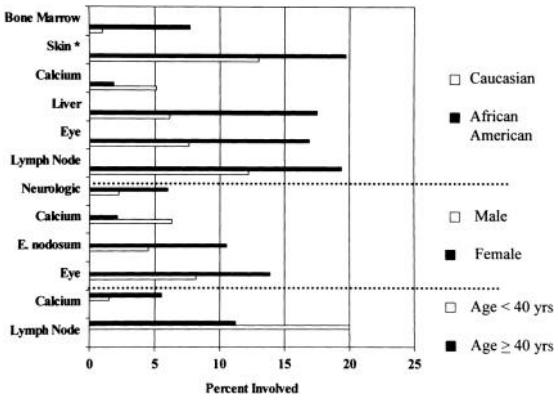


The main peak of onset is between 30 and 50 years of age. In this graphic, the black bar represents the female population and the gray bar is the male population. It can be seen that at peak ages there is a predominance of females impacted by Sarcoidosis.



These images show some of the manifestations of Sarcoidosis on the skin. Sarcoidosis may produce mucosa lesions and skin lesions that appear to be chronic. These lesions are one of the keys to the diagnosis of this disorder.

Sarcoidosis: skin manifestations
(Source: Baughman R et al, Lancet 361:1111; 2003)



Organ involvement – differences between groups: race, sex, age (ACCESS: 736 patients 1997 – 1999) Source: ACCESS Study, Am J Respir Crit Care Med 164:1885, 2001

It is interesting that despite the fact that the percentage of people with neurological symptoms from Sarcoidosis is relatively low (around 5 percent of this population), the female population is more affected by neurosarcoidosis as compared to the male population. The reasons for this susceptibility are not known.

- **Acute**
- Skin: Erythema nodosum, MP rash
- Acute iritis, conjuctivitis, nodules
- Hilar adenopathy
- Facial palsy
- Hypercalcemia/calciuria
- ACE ↑, Gallium +

- **Chronic**
- Skin: Plaques, keloids, ulcers
- Chronic uveitis, glaucoma, keratoconjunctivitis
- Pulmonary infiltration
- Multisystemic involvement
- ACE →, Gallium +/-

Sarcoidosis: Clinical Approach (Acute and Chronic)

Sarcoidosis may manifest acutely, primarily with skin and ocular manifestations, or it may produce a chronic symptomatology that may involve different organs.

Sarcoidosis: Diagnosis

- Clinical approach
- Radiology/Nuclear medicine:
  - Chest X-ray, Chest CT scan
  - Gallium scan
  - FDG PET scan
- Laboratory:
  - Angoitensin-converting enzyme (ACE)
  - Serum/Urine Calcium
- Skin test: Kveim-Siltzbach test

When a clinician sees a patient with suspected sarcoidosis, we do a very thorough evaluation. Since the main area of involvement is the lungs, a great deal of attention is focused on the respiratory system. There are different approaches used that allow us to detect areas of inflammation or the presence of enlarged lymph nodes. These approaches include chest X-Rays and CT scans, as well as nuclear medicine tests such as Gallium scans, and FDG PET-scans. There are also laboratory tests used for diagnosis, such as the use of angiotensin-converting enzyme (ACE) which can give useful information. In the 60's and 70's a skin test was introduced by European physicians called the Kveim-Siltzbach test, but this is no longer available in the United States, because of the potential risk of transferring infections.

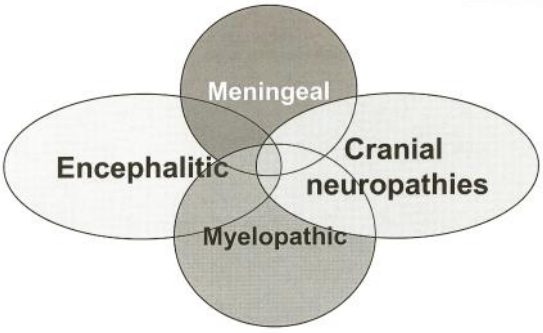
In summary, approximately 5 to 10 percent of patients with sarcoidosis may present with neurosarcoidosis. Interestingly, 50% of Neurosarcoidosis patients have neurological symptoms as the first manifestation of sarcoidosis. Any area of the central nervous system or peripheral nervous system may be affected. This is the reason we raise attention about Sarcoidosis; these patients may manifest with disorders that can mimic multiple sclerosis or mimic meningitis or mimic spinal cord involvement.

Clinical Involvement	Sharma 1997 (n=37)	Chapelon 1990 (n=35)	Stern 1985 (n=33)	Delaney 1977	Wiederholt 1965
Cranial neuropathy	52%	37%	73%	48%	64%
Aseptic meningitis	24%	40%	18%	26%	…
Hydrocephalus	…	40%	9%	17%	…
Hypothalamic	…		15%	26%	25%
Myelopathy	…		6%	9%	4%
Peripheral neuropath	24%/ GBS 5%	40%	6%	4%	14%
Myopathy	8%	9%	12%	9%	7%

Historically, there have been a number of studies that demonstrate the multifocality of neurosarcoidosis in the central nervous system. Sarcoidosis may involve the presence of abnormal-

ities in cranial nerves (cranial neuropathy), such as optic neuritis or facial paralysis. Neurosarcoidosis may also manifest with meningitis or with other problems, such as hydrocephalous, hypothalamic dysfunction, or the presence of myelitis, myelopathies, or the presence of muscle involvement or peripheral nerve involvement. The statistics that have been published in the last 20 years demonstrate the multifocality of involvement of this disorder.

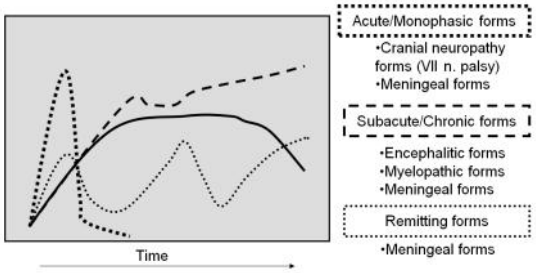
Neurosarcoidosis: Clinopathological Classification



Sarcoidosis is a disease that may affect any compartment of the central nervous system or peripheral nervous system. Patients may have problems associated with encephalitis, myelitis, or they may have problems associated with cranial nerve involvement.

The evolution of this disorder is quite variable. There is a subset of patients in which the primary manifestation of the disease is acute; just one episode of either cranial nerve involvement, or one episode of meningeal involvement (meningitis).

Neurocarcoidosis: Temporal profile



There are other patients for whom the course of the disease follows a temporal pattern that appears to be relapsing and remitting similar to an immunological disorder like multiple sclerosis. There are other groups of patients for whom there is a chronic progression of the disorder that produces a lot of disability and neurological problems.

In general, the most acute manifestation of the disease is the presence of cranial nerve involvement, such as facial paralysis, as Bell's paralysis, or a form of acute meningitis. The chronic encephalitic or myelopathic forms are also frequent.

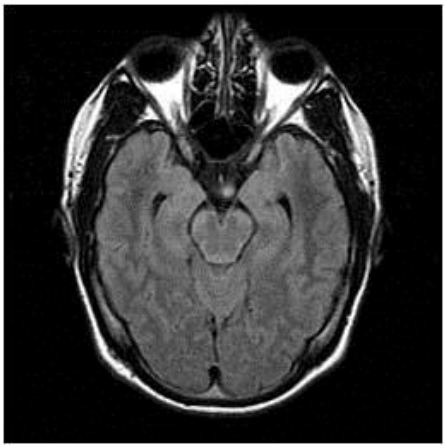
Neurosarcoidosis: Clinical Classification (Meningeal forms)

	Neurological presentation	Clinical profile	Clinical course
Meningeal forms	•Aseptic meningitis •Basal meningitis Chronic meningitis/ •Pachymeningitis •Dural tumor-like sarcoid lesions	Headaches Increase intracraneal pressure Hydrocephalus Papilledema Cranial nerve palsies, mono- or multiple	•Subacute •Relapsing-remitting •Chronic

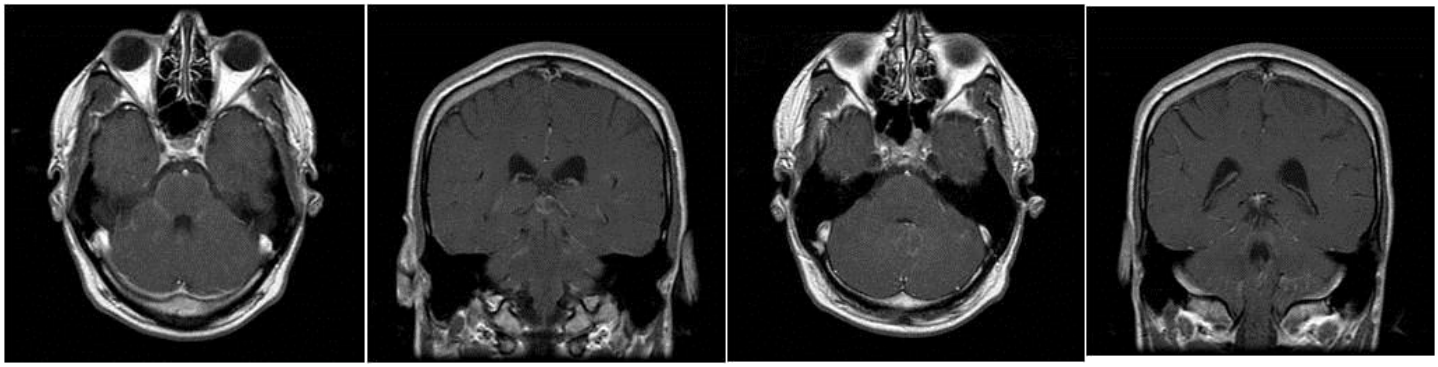
I am now going to present some examples of this disorder beginning with the meningeal manifestation. The meningeal involvement associated with sarcoidosis implies that you may have a patient in which there is aseptic meningitis, meaning that there is a chronic inflammation or acute inflammation that is not associated with bacterial infections or with viral infections.

The patient may manifest with chronic meningitis, or recurrent meningitis. In very aggressive forms, the patient may manifest with a thickening of the dura mater or even the presence of tumor-like lesions of the dura mater that often mimic brain tumors. All of these manifestations and all of these inflammatory processes are going to produce a multitude of symptoms that go from presence of headache, presence of hydrocephalous, or even presence of other problems like cranial nerve paralysis and papilledema and visual loss.

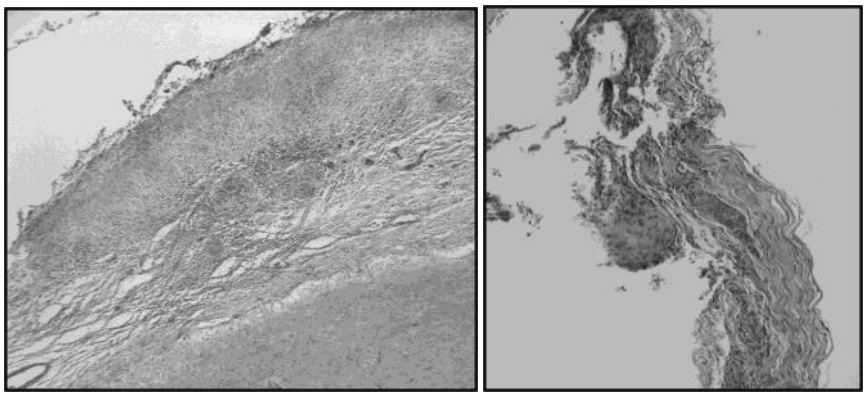
Meningeal MRI's



The brain MRI demonstrates that meningeal involvement means inflammation of the covering of the brain. There is enhancement of the leptomeninges; this is one of the manifestations of the disease. The enhanced white lines or the enhanced areas in which there is a patchiness, are a manifestation of the inflammatory process affecting the meninges.



Meningeal Pathologies



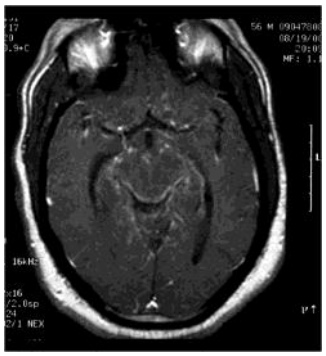
Pathologically, this process is characterized by the presence of inflammation. Inflammation is going to produce infiltration by white cells in the meninges, and is going to produce the presence of granulomas that are the most important hallmark of this disorder.

Neurosarcoidosis: Clinical Classification (Cranial neuropathies)

	Neurological presentation	Clinical profile	Clinical course
Cranial neuropathies	• Facial paralysis • Optic neuropathy • Multiple cranial neuropathies	Mono- or multiple cranial nerve palsies Bilateral Bell's palsy Diplopia Visual blurriness Vestibular symptoms	• Acute • Subacute, • Monophasic • Relapsing-remitting

Cranial neuropathies are another group of manifestations of neurosarcoidosis. These manifestations of the disease are going to affect different cranial nerves, such as the optic nerve or the facial nerve. Cranial neuropathies may produce symptoms such as visual loss or muscle dysfunction. The cranial neuropathies may have different temporal manifestations from acute to sub acute to chronic.

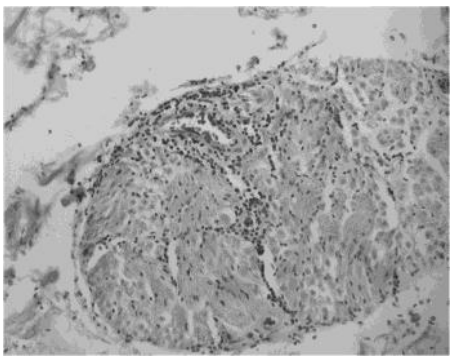
We frequently observe the association of both cranial neuropathy and meningitis. Meningitis may involve the brain stem which would contribute to cranial nerve involvement and subsequent cranial nerve paralysis.



Graphic Neurosarcoidosis Cranial Nerve Involvement+MRI
Series A: Stern et al, 1986 Series B: Chapelon et al, 1990

	I	II	III	IV	V	VI	VII	VIII	IX	X	XI	XII
A 33	3	3	2	1	3	3	11/	1/	1			1
B 35			4		5	2	11	1	1	2	2	2

Cranial neuropathy pathology



Endoneural and perineural Inflammation

- **Ischemic neuropathy**
- **Axonopathy**
- **Myelin loss**

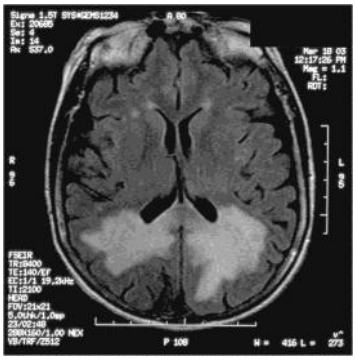
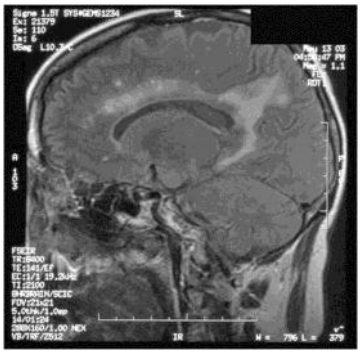
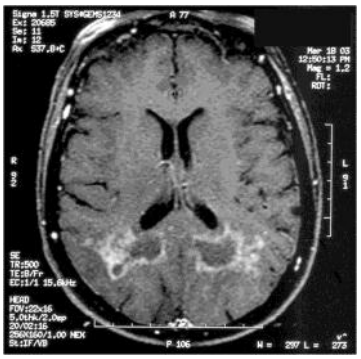
Pathologically, when a cranial nerve is affected, the major impact involves inflammation. This image is from a pathological study from a patient that died after sarcoidosis. This is a cross section of a facial nerve and one may observe the infiltration of that cranial nerve by inflammatory cells.

Neurosarcoidosis: Clinical Classification (Encephalitic forms)

	Neurological presentation	Clinical profile	Clinical course
Encephalitic forms	·Focal encephalitis ·Focal or multifocal leukoencephalitis ·Tumor-like sarcoid lesions	Headaches Psychosis Seizures Neuroendocrine manifestations Increased intracraneal pressure Focal neurological symptoms	·Subacute ·Relapsing-remitting ·Chronic

One of the most aggressive manifestations of these disorders is the encephalitic form of the disease. The encephalitic form means that there is inflammation of the brain parenchyma; that is in contrast with other forms of the disease, in which the inflammation is predominantly the meninges, the covering of the brain. These forms may affect white matter, grey matter or it may produce tumor-like lesions. These forms are going to produce complex manifestations such as headache, psychoses, seizure activity, endocrinological problems, and significant neurological disability. These are the forms that frequently mimic multiple sclerosis, thus, it is extremely important that patients with suspected multiple sclerosis be evaluated to be sure that they do not have neurosarcoidosis. The temporal pattern of these types of manifestations is that the brain parenchyma may be affected in a relapsing-remitting or chronic profile; very similar to what happens in multiple sclerosis.

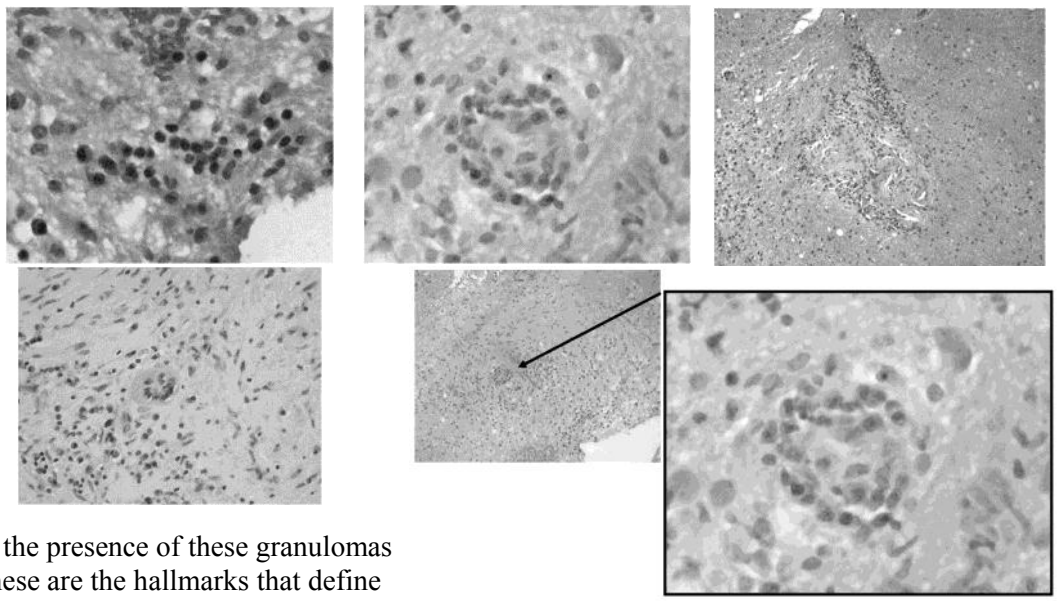
Neurosarcoidosis: Encephalitic form MRI's

These are very dramatic images of the brain of a patient affected by sarcoidosis. We recently evaluated this patient and there was extensive encephalitis predominantly affecting the white matter. Initially, there was concern about the presence of a brain tumor, like a glioblastoma multiform. For this reason, the patient got a biopsy and the surprise for us was that this was no tumor. The tissue biopsy confirmed that this was a manifestation of neurosarcoidosis.

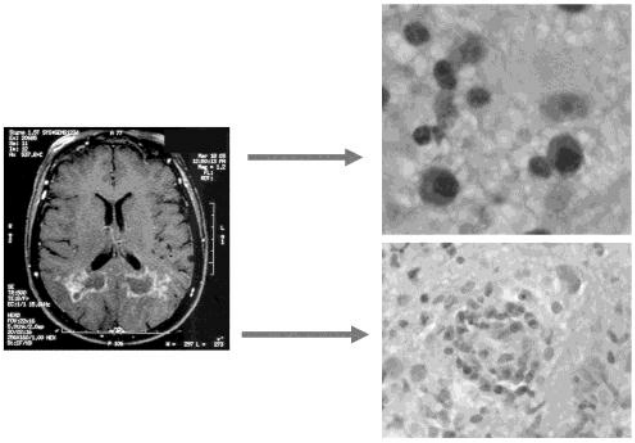
Neurosarcoidosis: Encephalitic forms: Pathological Features

- Lymphocytic infiltrates
- Plasmocytic infiltrates
- Perivascular cuffing
- Demyelination
- Gemistocytic astrocytes
- Parenchymal and leptomemingeal granulomas without necrosis
- Negative AFB staining



The tissue biopsy demonstrated the presence of these granulomas and inflammatory reactions. These are the hallmarks that define the neurosarcoidosis.

Conclusion: Granulomatous encephalitis = Neurosarcoidosis



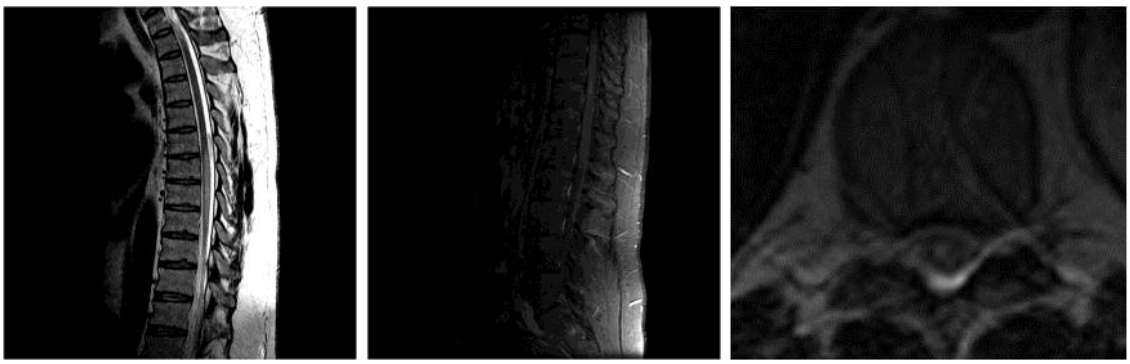
In this particular case, what defined and concluded the overall clinical assessment is that this patient suffered from the encephalitic form of neurosarcoidosis. This was good news because we were able to treat the inflammation with the use of a steroid, IV Solumedrol. Subsequently, the patient received treatment with Prednisone, and is doing much better. During the follow-up investigation, we found that the patient also had sarcoidosis affecting her lungs.

Patients with transverse myelitis also have to be evaluated for neurosarcoidosis. When there is a sub-acute form of myelitis or progressive myelopathy or chronic myelopathy, one of the concerns is the presence of sarcoidosis. We see this with relative frequency in patients with these types of temporal profiles. This is one of the issues in evaluating the differential diagnosis between myelopathy and neurosarcoidosis.

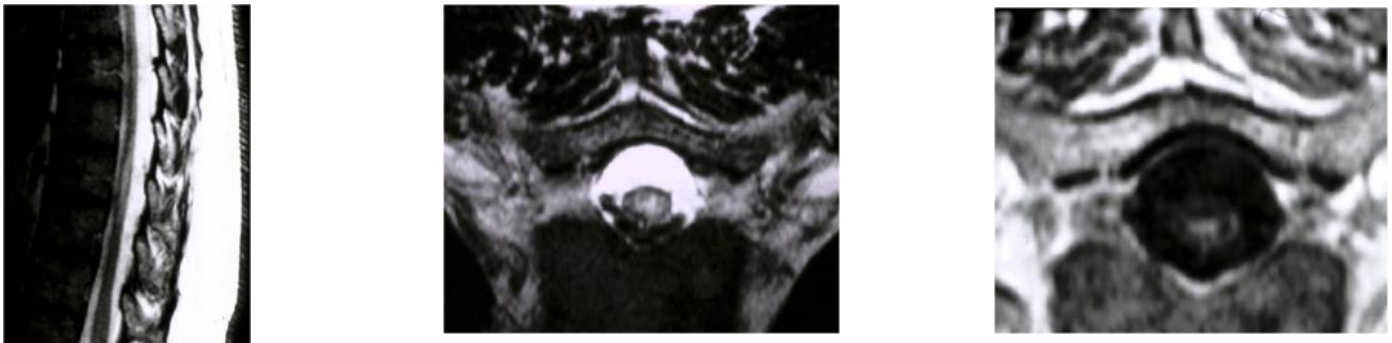
Neurosarcoidosis: Clinical Classification (Myelopathic form)

	Neurological presentation	Clinical profile	Clinical course
Myelopathic form	Subacute or progressive myelopathy	Gait disturbances Paraparesis/ Paraplegia Bladder dysfunction Paresthesias/ dysesthesias Sensory level	Subacute, Monophasic, relapsing-remitting or chronic

Neurosarcoidosis: Myelopathic form MRI's



Neurosarcoidosis in the spinal cord is very aggressive and produces a lot of spinal cord changes. This is a MRI of a patient affected by neurosarcoidosis. The inflammatory granuloma lesion is taking up a large portion of the spinal cord, and producing significant clinical manifestations, such as paraparesis (weakness in the lower extremities) and sensory deficit.



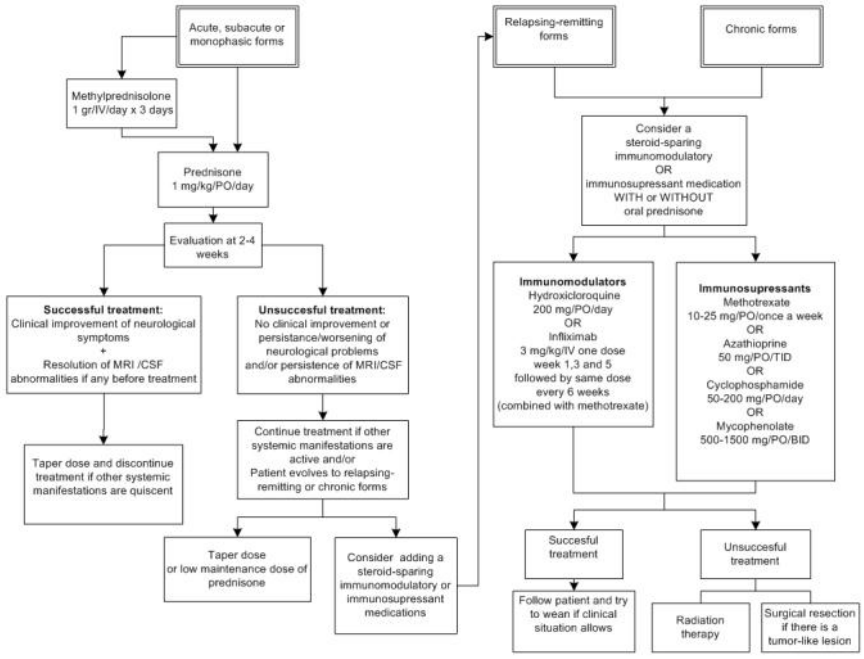
This MRI is of a longitudinal section of the thoracic and lumbar spinal cord. In the middle of the cross-section of the spinal cord, there is an area that appears bright white. This is a manifestation of the inflammatory process in this disorder.

Neurosarcoidosis: Clinical Classification (peripheral nervous system)

Clinical Forms of Neurosarcoidosis: Peripheral Nervous System			
	Neurological presentation	**Clinical profile**	**Clinical course**
Neuropathic form	**Multiple mononeuropathies Polyradiculoneuropaties**	**Multifocal or localized dysesthesias, paresthesias, weakness, mono- or polyradiculopathies**	**Subacute, Relapsing-remitting or chronic**
Myopathic forms	**Focal myositis Polymyositis**	**Weakness, muscle pain**	**Acute, subacute or chronic. Occasionally indolent**

Neurosarcoidosis may produce involvement of any part of the central nervous system, and it may also affect any part of the peripheral nervous system. It may affect the peripheral nerves or it may affect the muscle. This is going to be associated with significant manifestations of numbness, tingling, or the presence of muscle weakness.

This complex diagram identifies the treatment decisions for neurosarcoidosis. The treatment is divided into acute forms, the relapsing-remitting forms and the chronic forms of the disease. The acute treatment is based primarily on the use of IV Solumedrol or Methylprednisolone or Prednisone. These treatments are similar to what we use in patients with acute myelitis or patients with flare-ups of multiple sclerosis. In general terms, the treatment of all of these immunological disorders is very similar in many ways. However, when we complete the treatment of the acute manifestations, we need to decide if the patient needs chronic treatment or whether the treatment of the acute process was enough to control the disease. Unfortunately, in the majority of patients with neurosarcoidosis, we need to continue with chronic treatment. The chronic treatment is based on the use of immunomodulatory medication or the use of immunosuppressant medication. Immunosuppressant medications include Methotrexate, Azathioprine, Cyclophosphamide, or Mycophenolate. All of these medications help to control the inflammatory process which is one of the main mechanisms of the disease in neurosarcoidosis.



There are recent treatment approaches that modulate the presence of some cytokines. The introduction of new medications has produced modulation of the antibody response in sarcoidosis or the reduction of the TNF associated production. All of these immunosuppressant approaches are very helpful in the control of the symptomatology.

The treatment of sarcoidosis depends on the clinical manifestation and the temporal profile. This is extremely important because there are encephalitic forms and myelopathic forms that require more aggressive treatment. This more aggressive treatment means not only the use of steroids, but also the use of immunosuppressant medications. There are forms of sarcoidosis, for example acute forms, in which the use of steroids, such as Prednisone, is good enough to control the disease.

The temporal profile is also extremely important in deciding the type of treatment we are going to use. Whether it is a monofocal disease or a multifocal disease will determine how aggressive our treatment approach will be.

Treatments in women or in elderly people are another important consideration. The majority of approaches involve use of Prednisone or chronic use of steroids. This is very difficult in women that are post-menopausal, because of the high risk of problems associated with osteopenia or osteoporosis. We have to be careful that our treatment approach does not produce more harm than benefit.

In many patients, there is damage of the neuroendocrine function. This damage is produced by the involvement of the hypothalamus and pituitary gland. In these particular situations, the damage is going to produce chronic endocrinologi-

cal problems, such as hypothyroidism, hypogonalism, and other complex endocrinological processes. These problems will require treatment. These problems are not necessarily associated with active inflammation, but rather are the consequence of the damage to the hypothalamus or pituitary gland.

To conclude, Sarcoidosis is a multisystemic disease. It is immunologically mediated. We do not know the etiology of the disorder. In many ways, it appears to be a disorder that mimics a chronic infectious disorder, similar to what we see in tuberculosis. This is one of the reasons we are currently focusing on a search of potential etiological factors, such as micro bacterial infection.

Sarcoidosis is a very heterogeneous disease. Every patient with multiple sclerosis or with myelitis or myelopathies needs to be evaluated very carefully for sarcoidosis. This disease may mimic what we see in these disorders in the brain or in the spinal cord or peripheral nervous system. We are working hard on education about this disorder to get physicians who work in immunological problems and the NIH to pay more attention to this multisystemic disease.

We are trying to modify the standard of care. There are good studies which demonstrate that the CSF H-level is useless. A Mayo Clinic study has detailed that after extensive assessment of the spinal fluid in patients with sarcoidosis, the test was not necessarily reliable and there was no evidence of good sensitivity or good specificity. I think it is useless to request CSF H-level.

Chest X-rays may help, but unfortunately are not good enough. We are trying to encourage physicians who are evaluating sarcoidosis to first request a chest CT scan with contrast. A Gallium scan would be better but, unfortunately, is very expensive. A FDG PET-scan would be even better. The FDG's are flourodioxyglucose PET scans. This is a nuclear medicine-based test that allows us to assess inflammatory activity in different areas of the body. It is a very powerful technique. It is very expensive and the majority of insurance companies deny the use of this test for assessment of sarcoidosis. The cost is close to $5000 versus a chest CT scan that is between $700 and $800. We are trying to tell the insurance company that if we diagnose sarcoidosis early, we can save a lot of money by avoiding more complications in patients. Investing four or five thousand dollars in the FDG PET scan is going to save money in the future. Not too many insurance companies are accepting this position.

If there is suspicion about sarcoidosis, if there is clinical evidence of something going on in the brain and the spinal cord, and if there is a suspicious chest CT scan, it is important to get a pulmonologist involved. They should get a lymph node biopsy or a lung biopsy. If it is demonstrated in a lymph node biopsy or a lung biopsy that there is sarcoidosis, then this patient very likely has neurosarcoidosis. It is very important to do a thorough assessment of the patient at the outset as opposed to giving treatment and then not following up on these other diagnoses. It is possible that the patient can be treated for the neurological problem with steroids or Prednisone, whether it is sarcoidosis or MS and then the opportunity may be missed to establish the definitive diagnosis.

We believe that almost 50 percent of patients with neurosarcoidosis have oligoclonal bands and have increased IgG index. In our pathological findings we have seen evidence of B-cell infiltration in the brain parenchyma or meninges; I think that is the reason we see oligoclonal bands and an IgG increase. We are currently doing this study, and it is not yet published in the literature. So unfortunately, that spinal fluid assessment is not going to help us too much in establishing a definite diagnosis of neurosarcoidosis. But, as I mentioned before, if you have a suspicious chest X-ray or chest CT-scan, you may have the opportunity to do a lymph node biopsy, or a bronchial lavage, or lung biopsy and see if the diagnosis can be established in that way.

I think that that's an approach, and that is absolutely required if the patient is an African-American patient, and you emphasize to the residents and everybody, you have an African-American patient, you are obligated to get a chest CT-scan for problems like optic neuritis, problems like uveitis, problems like encephalitis, meningitis, or anything that resembles sarcoidosis.

One thing we need to emphasize is the care of the patient with sarcoidosis is multi-disciplinary, we need to get the pulmonologist involved, we need to get the endocrinologist involved, because those patients are going to have a lot of problems related to those areas of medicine. Patients are going to have opthamological problems as well, or a patient may have a lot of cardiomyopathies because sarcoidosis also affects the heart and produces difficult problems with cardiomyopathies.

MOOD 24/7

Mood247.com
CO-DEVELOPED BY:
DR KAPLIN OF JOHNS HOPKINS
&
HEALTHCENTRAL

Track, Understand, and Improve Your Quality of Life With This Free Tool

Feeling lonely or blue? Are you battling depression? TM, ADEM, NMO, and ON cause chemical changes in the brain that make you more susceptible to depression and mood disorders. Do you want to improve the quality of life for you and your loved ones? Mood24/7 is the perfect place to start by providing you with an important tool to better understand your mood and help recognize and treat depression if it is playing a role in your life.

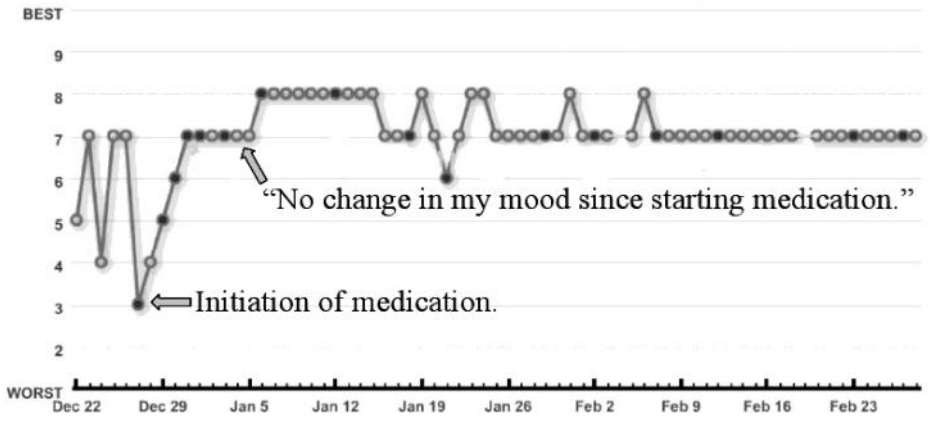
Mood24/7 is a free mood-tracking tool that was co-developed by Dr. Adam Kaplin of Johns Hopkins and HealthCentral. Dr. Kaplin is a TM and MS neuropsychiatrist at the Johns Hopkins MS and TM Centers of Excellence and a physician on the Transverse Myelitis Association Medical Advisory Board. HealthCentral is the third leading online health company. Together, they have developed Mood24/7 to monitor and track mood daily, not only for personal purposes, but also for use in clinical settings.

The service itself is free and it can be highly beneficial to you if you want to monitor your mood and how it changes over time, even if you have not been diagnosed with clinical depression. In addition to monitoring your daily mood, Mood24/7 permits you to enter a daily note of what else is going on of relevance on any given day. Through its use, you may be able to identify certain stressors in your life that lower your mood. People who have used Mood24/7 have said that for personal use, Mood24/7 acts as a "guardian angel" and also raises self-awareness of their daily mood. Because of this, patients are able to actively focus on working to improve their mood.

For some users, using Mood24/7 may lead to the recognition that you may benefit from seeing a mental healthcare provider. It is important to understand that depression affects patients with TM, ADEM, NMO, and ON more than the general population because of chemical changes in the brain due to neuroinflammation (i.e., inflammation affecting the brain); it is not simply a matter of being strong-minded. Multiple studies have shown that depression is the primary determining factor in a patient's self-reported quality of life, with greater bearing than other variables investigated, including physical disability, fatigue, and cognitive impairment. Therefore, getting depression treated is important for your quality of life, which will directly impact the quality of life of your loved ones.

Besides having many personal benefits, Mood24/7 better equips mental healthcare professionals to provide quality care. If you use it and currently see a mental healthcare provider, you are able to share your daily mood charts that can be accessed online with your mental healthcare provider during consults. This will help your healthcare provider have a better appreciation of how you have been doing, track your actual response to different treatments, and help you instantly show your physician your mood in a graphical format instead of forcing you to waste time on trying to remember events that happened weeks ago. By using Mood24/7, Dr. Kaplin has found he is able to visualize his patient's responses to medications. He can now more accurately assess whether a patient should stay on a medication because it is helping or change a medication because it is doing nothing to improve a patient's mood.

Your mood can also be shared with your loved ones so that they can see how you are doing without blatantly asking you your mood. This can help put your loved ones at ease to know that you are doing well or help to alert your loved ones when you may not be in the best mood. Furthermore, Mood24/7 can help you track your child's mood, especially in a time when they may be unwilling to open up about their feelings to you in person. In fact, since the service is free, you can get your whole family to par-



A patient using Mood24/7 who improves on a certain medication might not believe that the medication is doing anything and stop treatment. Mood24/7 not only aids in recognizing stressors in life, but it also tracks response to treatments. Moods are so transient and difficult to remember. The best way to see change is to track your mood.

ticipate in mood reporting if you wanted to.

Mood24/7 is simple to use and requires only seconds a day to use. The user simply chooses a time of day to receive the text and then responds with a number 1 to 10 to symbolize their mood (10 meaning the best mood and 1 meaning the worst mood). After the numerical mood score, users can then enter a space and record whatever other annotations they want, whether it be stressors in their lives or other circumstances. For clinical purposes, medication changes are particularly important to record. The user then sends out the text and all the data gets stored on a secure server where the information can only be accessed through Mood24/7's password-protected site by the user or by other people that the user has to specifically designate to share his or her chart with in his or her trusted circle. Users can select the timeframe over which they want to see their moods graphed out. Again, HealthCentral has plans on keeping Mood24/7 a free service. Your own mobile carrier's text rates or text quotas will still apply though.

Signing up and starting to track your mood is pretty self-explanatory and simple. Just sign on to Mood247.com, click the "get started" button, and fill out the required information. Once you finish that, you will be sent a text to your phone with an activation code to associate your account with your phone. After you insert that code online, your account has been set up. Within your account you can add people to your "trusted circle" so that they can see your mood reports as well as change the time of day that you receive the daily text. There is no obligation to continue receiving texts if you don't want to, and you can stop using Mood24/7 at any time.

Mood24/7 can help you and your loved ones be more aware of your mood and help you to concentrate on the things that can enhance your quality of life. It is a tool designed with both personal use and clinical use in mind. Mood24/7 is a way to revolutionize healthcare by using your own cell phone to help organize your ability to track, understand and improve your daily health. Try it today at Mood247.com.

The Family Information Guide to Assistive Technology and Transition Planning is now available free of charge from the Family Center on Technology and Disability (FCTD). The FCTD is a national organization that produces and distributes information on assistive and instructional technologies. Assistive technology can be anything from a simple device to make holding a pencil easier to a sophisticated computer. The guide is aimed at providing families with the information they need to effectively prepare for and participate in periods of transition in their children's lives. This 50 page guide includes sections on Assistive Technology, Transition Planning, Laws governing accommodations in school settings and a Glossary of assistive technology terms and resources. To order a free copy, go to the FCTD website ***www.fctd.info/show/order_guides***. A pdf version of the guide is also available for download and you can also view the guide online in a fully accessible html version.

Learning about TM and the Other Neuroimmunologic Disorders: Bibliography and Videos on www.myelitis.org

For those of you trying to learn about Transverse Myelitis, Chitra Krishnan has compiled an excellent bibliography about TM. Chitra serves on the TMA Medical Advisory Board. You can find the bibliography by typing this address into your web browser:
http://www.myelitis.org/Bibliography.htm

Jim has created links from the articles in the bibliography to Medline; so when you click on the article citation, you can easily get to a copy of the article to read. Additionally, when you are in Medline, you can link to other recently published articles by clicking on the authors' hotlinks.

Another tremendous resource about TM and the other neuroimmunologic disorders is the streaming video that Jim has posted on the web site. The presentations from the 2010 (Dallas), 2008 (Seattle), 2006, 2004 and 2001 (Baltimore) Symposia, from the Southwest Symposium (sponsored by the Cody Unser First Step Foundation), and from the 2002 children's workshop are available under the link 'Symposia Information' or by typing http://www.myelitis.org/events.htm into your web browser. Jim has the presentations organized as they appeared in each of these symposia program agendas. You can also find PDF files of most of the handouts and PowerPoint presentations. The video presentations are also available by going through the Streaming Video Presentations link from our main web page or by typing ***www.myelitis.org/multimedia.htm*** into your web browser.

You can find The Transverse Myelitis Association on Facebook. It is a great way to support the TMA and it a wonderful way to network with people in our community. Please take the time to become a fan of the TMA and tell your friends and family about your community's page. Our page is a great way for us to raise awareness about these disorders and about your experiences.

Our link is:

http://www.facebook.com/myelitis

The Diaphragmatic Pacemaker: Deserve a Better Quality of Life
Alana Spence

Faster than a speeding bullet! More powerful than a locomotive! Able to leap tall buildings with a single bound! Look, up in the sky! It's a bird, it's a plane -- No, it's... a man in a wheelchair, with a ventilator? Even Superman has his kryptonite.

"Christopher Reeve said he had been hoping for years to be able to breathe without the aid of a ventilator. He demonstrated for Barbara Walters how he was then able to breathe without his ventilator for hours at a time." Christopher Reeve is the third person to receive a diaphragmatic pacemaker. There are only an estimated 500 people, worldwide, who have ever received this device, but what is more important is how many thousands of people need it, but do not yet know about it.

The recently FDA approved diaphragmatic pacemaker provides the best alternative for ventilatory support to people who have little to no function of their lungs, diaphragm, and phrenic nerve. These cases are usually caused by quadriplegia, Multiple Sclerosis, Lou Gehrig's disease, and Transverse Myelitis, as well as a number of other chronic conditions. This device will allow people to have more freedom, safety, and emotional relief.

I am one of the few people in the world to have a diaphragmatic pacemaker, and it has dramatically changed my life in more ways than I could have ever imagined. From only being able to go four hours outside of my house with a ventilator; I now have the freedom to be on my pacer all day, every day.

Based on my own experience, I would like to talk to you about a new medical device called the diaphragmatic pacemaker. I will speak about how and why it works, how it benefits its users, and offer a personal perspective on the significance of this device.

Before we learn about the diaphragmatic pacemaker, you should first understand how the respiratory system works. Many of you may not know how or why you are breathing, just so long as you're breathing. Without breathing, you are dead. Simple enough, right? But what exactly is going on in that body of yours that is making you inhale and exhale? There are three main parts of your respiratory system: the phrenic nerve, the diaphragm, and the lungs. The phrenic nerve is, in simple terms, a nerve that gives an electric signal from the brain to the diaphragm. It is equivalent to a mailman: if you don't have a mailman, the letter from grandma will never be delivered to your house. This electric signal is what allows the diaphragm to contract, and pull air into your lungs. It is an automatic process, in which you don't have to tell the brain that you want to breathe. It simply happens without you knowing it. For those like myself, who cannot breathe by themselves, there is new medical technology that can do that for us -- the diaphragmatic pacemaker.

Diaphragm pacing was developed in Cleveland, Ohio by Dr. Raymond Onders, who surgically implanted my pacer, along with a team of biomedical engineers at Case Western Reserve University Hospital. According to Dr. Onders, the process involves the placement of tiny electrodes onto the diaphragm through laparoscopic surgery. The electrodes are then threaded to a small external battery pack (the size of a TV remote control) that signals the diaphragm to contract, pulling air into the lungs. After a period of conditioning, the diaphragm is strengthened to a point where a person can remain ventilator free 24/7.

According to the article, "History Making Research Technology," Dr. Jeremy Road, the principle investigator of the research trial, explain that "Mechanically ventilated patients almost always have severe impairments for smell, taste, and speech. With diaphragm pacing, they are able to regain their sense of taste and smell, improve speech and live life with a much higher level of independence."

In an interview with Barbara Walters, Christopher Reeve, who underwent surgery in 2003, stated, "While the experimental surgery hasn't given me any more privacy, the 'emotional relief' it has given me is tremendous." Reeve promoted this device when it was unknown to the public and still in the early stages of a clinical trial.

I know first-hand the benefits of having a diaphragmatic pacemaker. When I was on my ventilator, my freedom was limited. While I was in my wheelchair, the batteries of the vent only allowed me to leave the house for a maximum of six hours. The machine was attached to the back of my chair, while the tube was visibly hanging from my neck, drawing attention from the public everywhere that I went.

When I had the surgery in August 2007, when it was still a clinical trial, my life dramatically changed. Now I am able to breathe without the aid of my ventilator, and I am able to breathe with my pacemaker for as long as 24 hours a day, while creating minimal distraction to the public. My parents are much more comfortable leaving the room without having to worry about the ventilator disconnecting. I find it hard to describe to you exactly how this incredible device has given my family and me emotional relief.

The average person doesn't even notice or acknowledge that they're breathing because it comes so naturally. To me, not being able to do something as small as taking a breath; it's

devastating. Having a ventilator pump 12 breaths per minute into my lungs was both uncomfortable and unnatural. The diaphragmatic pacemaker has given me a life that the ventilator never did. The insecurities of wearing a ventilator are long past, and I now have a small taste of normalcy.

The pacemaker, while being extremely successful, and becoming FDA approved in May 2008, has very few drawbacks. For instance, I recently overcame a skin infection from irritation of the wires. Out of the 500+ patients who have had the surgery, less than a handful of cases have gotten an infection. It is not available for those who have more than 50% control of their diaphragm. The surgery can only be given to people who have an active phrenic nerve. Otherwise, the pacemaker will not work. It is an outpatient surgery, and takes about 30 minutes to implant. However there are a few exceptions. For instance, people who have sleep apnea, or who are at extreme risk of soon becoming ventilator independent, should contact their doctors to see if they are viable candidates for the surgery.

Take a deep breath. Now imagine if that was your very last. It happens to thousands of people every year, and they are put on ventilators to keep them alive. One day down the road, you might know someone who knows someone that is put in this situation, and you can tell them about a life-changing medical technology called the diaphragmatic pacemaker. It is an extremely successful practice and the public needs to be made more aware of the technology. This pacer is the only one of its kind. Many potential candidates for this operation do not have the privilege of traveling, so with more exposure, more hospitals will practice this surgery, and give more people the opportunity to have this device implanted. The diaphragmatic pacemaker not only saves lives, it gives people the opportunity for a better quality of life.

From the TM and NMO Centers

The Johns Hopkins Transverse Myelitis Center: One Decade of Work and the Challenges for the Future

Maureen Mealy, RN, BSN and Carlos A. Pardo, MD

The beginning of the 21st century was marked by the establishment of the first center dedicated to the study and research of transverse myelitis (TM). The Johns Hopkins Transverse Myelitis Center (JHTMC) was established in 1999 through the efforts of Dr. Douglas Kerr. In collaboration with Drs. David Irani and Carlos Pardo, these clinicians and researchers created the foundation for a unique center dedicated to facilitate the diagnosis, treatment and research of transverse myelitis. This effort was enthusiastically supported by The Transverse Myelitis Association (TMA), its president, Sandy Siegel, as well as patients and members of the TMA from across the country and around the world. The tireless efforts of other members of the JHTMC, such as Chitra Krishnan and Drs. Adam Kaplin and Ben Greenberg resulted in significant strides being made in our understanding of TM and the initiation of novel research projects that resulted in more rapid and better diagnosis of TM and more effective treatments for TM. The team at the JHTMC has also been dedicated to providing education about TM to the patient community and to clinicians who care for people with TM.

In 2010, more than a decade later, the JHTMC has experienced many changes, including the departure of some of the founding members and the new addition of others. During this period of change, we have taken the opportunity to refresh our focus and to rededicate ourselves to fulfilling our vision of a multidisciplinary and comprehensive approach to patients with transverse myelitis to facilitate early diagnosis and treatment.

Clinical Care at the Johns Hopkins TM Center

We have focused on improving our ability to interact with patients and physicians by facilitating communication with the TM Center. With the support of Maureen Mealy, RN, BSN, Program Manager of the JHTMC, we have established easy access to patient referrals and have expedited the consultation process. By either contacting the JHTMC by phone (410-502-7099, option 2) or by e-mail (***hopkinsTMcenter@jhmi.edu***) patients and physicians may obtain information about TM, make appointments and arrange the coordination of services in a timely manner.

We have established a system to facilitate a comprehensive assessment that involves not only the neurological evaluation, but other types of consultations in an effort to provide patients with long-term plans for management of all health problems associated with TM. These consultations may include specialists in rehabilitation, neuroradiology, neuropsychiatry, neuro-ophthalmology, urology, and spinal neurosurgery at Johns Hopkins Hospital and its affiliates at the Kennedy-Krieger Institute (KKI) International Center for Spinal Cord Injury. Our team at KKI possesses a focused interest in TM.

A pre-visit assessment of needs and review of records allows us to better match each patient to specific doctors. We understand that so many of our patients are coming from across the country and from all over the world, and that their time and resources are valuable. Our assessment and review

process has been put in place to provide patients with the best opportunity to get the absolute most out of their visit to the Center. This process also allows for patients with more urgent needs to be seen in a timely fashion. In fact, for some patients, their visit to the JHTMC may be in conjunction with a several week stay with the rehabilitation team either at Johns Hopkins or at Kennedy Krieger Institute for intensive therapy.

Because of the complexities of transverse myelitis, the JHTMC consists of a team of neurologists within the Neuroimmunology Division, as well as other specialists who have a focused knowledge of this rare and oftentimes difficult disorder. Our neurological team includes:

Dr. Carlos Pardo-Villamizar, Director of the JHTMC, focuses on acute idiopathic transverse myelitis, as well as sub-acute and chronic myelopathies associated with neurosarcoidosis and other etiologies.

Dr. Michael Levy focuses on Neuromyelitis Optica and Neuromyelitis Optica spectrum disorders, including recurrent TM and longitudinally-extensive TM. Dr. Levy is the Director of the NMO Clinic at Hopkins, a clinic that is not only dedicated to the diagnosis and management of NMO, but also focuses its efforts on research into this TM-related disorder.

Dr. Daniel Becker, a neurorehabilitation specialist and also faculty at the Kennedy Krieger Institute International Center for Spinal Cord Injury focuses on evaluation and treatment of long-term effects of TM and neurorehabilitation.

Drs. John Ratchford and Daniel Harrison focus on myelitis, myelopathies, and demyelinating disorders that can present as TM. Both are neuroimmunologists with expertise in clinical trials and neuroimaging technology in neuroimmunologic disorders.

Drs. Peter Calabresi (Director of the Johns Hopkins MS Center) and Scott Newsome focus on assessment of those patients suspected of having demyelinating syndromes that can present as TM. They are also interested in the assessment of novel technologies, such as ocular coherence tomography (OCT), a new non-invasive technique used in the assessment of axonal and retinal damage produced in neuroimmunologic disorders.

Drs. Justin McArthur, Avindra Nath and Arun Venkatesan focus on patients with TM as a result of an infectious disorder. They have a longstanding interest in neuroinfectious disorders, including those that are associated with TM, such as herpes infections or other viruses.

Dr. Julius Birnbaum has the unique expertise of rheumatology and neuroimmunology as he was trained in both specialties. He focuses on patients who have TM as a result of an underlying rheumatologic condition, such as Sjogren's disease or systemic lupus erythematous.

The Neuropsychiatry clinic for TM and MS was established through the pioneering work of Dr. Adam Kaplin. Dr. Kaplin's work continues to be one of the most important facets of the JHTMC. Dr. Kaplin focuses on the assessment and treatment of issues related to depression and cognitive problems in patients affected by TM.

Under the guidance of Dr. Daniel Becker and in partnership with the Kennedy Krieger Institute, we provide focused neuro-rehabilitation assessments, as well as physical and occupational therapy consultations, often times in the clinic while the patients are visiting with their neurologists. Dr. Glenda Bosques, a pediatric rehabilitation specialist, also contributes greatly to the assessment and management of children affected by TM at KKI.

As TM presents as a multi-faceted neurological disorder, other non-neurology specialists are a critical part of clinical care at the JHTMC. Dr. Philippe Gailloud, an interventional neuroradiologist, provides a unique expertise on evaluation of patients suspected to have vascular abnormalities associated with the development of myelopathies and TM such as arteriovenous fistulas and vascular malformations. Dr. Izlam, a neuroradiologist, also provides expertise on imaging studies of TM and related disorders. Dr. Jean-Paul Wolinsky, a neurosurgeon at the Department of Neurosurgery and Spine Center at Hopkins provides important consultation expertise on issues related with spine disorders that mimic TM. Dr. Prem Subramanian, a neuro-ophthalmologist at the Wilmer Eye Institute of Johns Hopkins Hospital provides consultation and assessment of visual problems, such as optic neuritis that some patients with TM may experience. Dr. James Wright from the Department of Urology provides critical expertise regarding the assessment and treatment of bladder and urological problems often associated with TM.

Under the leadership of Dr. Michael Levy and with the support of the Guthy-Jackson Charitable Foundation, we have established a sub-specialty clinic focused on Neuromyelitis Optica (NMO), a very challenging and rare subgroup of transverse myelitis characterized by recurrent longitudinally-extensive transverse myelitis and optic neuritis.

Referrals to the Center are facilitated through The Transverse Myelitis Association and its president, Sandy Siegel, the JHH Hopkins Access Line (HAL, which facilitates inpatient admission to JHH from other hospitals), the JHTMC website, JH Access Services (which facilitates scheduling of outpatients to JHH), and the JH International Office. Additionally, there is

an on-call neuroimmunologist to facilitate triage and admission, as well as to offer advice for the management of TM inpatients outside of the JHH system, in conjunction with the JHH HAL Attending. This allows for prompt attention to meet acute management needs.

Research at the Johns Hopkins TM Center

Research continues to be a major focus at the JHTMC. Studies for which we are presently recruiting include:

Studies of the effect of TM on cognition and depression; and the role of factors, such as cytokines and other immune mediators have been the pioneering work of Dr. Adam Kaplin and his group. Dr. Kaplin was awarded the first NIH grant focused on TM and has continued his efforts to assess the role of other factors that influence the development of cognitive problems and depression in patients with TM.

The Accelerated Cure Project (Johns Hopkins Principal Investigator is Dr. Arun Venkatesan) is a multi-center central blood repository and database which allows us to look into better diagnoses, therapies, and eventually cures for demyelinating disorders of the central nervous system -- transverse myelitis, neuromyelitis optica, acute disseminated encephalomyelitis, optic neuritis and multiple sclerosis. The repository and data are made available in a de-identified manner to researchers from around the world to help each of them with their individual efforts. These researchers then share back their data to the central repository so that we are continuing to learn more about these disorders every day. It requires a questionnaire and blood draw from participants.

A study led by Dr. Neal Halsey at the Johns Hopkins Bloomberg School of Public Health in collaboration with the JHTMC focuses on an epidemiological analysis and assessment of risk factors leading to the development of acute idiopathic transverse myelitis, most specifically, the role that vaccines and acute infections may play in the development of this disorder. Participation entails the response to a questionnaire.

A fourth study involves those patients with Neuromyelitis Optica (NMO). It will actually be a series of studies taking place over several years which build on previous results. This work is supported by the Guthy-Jackson Charitable Foundation and will be performed by a national NMO Consortium that has been created between Johns Hopkins, the University of Texas Southwestern, and Mayo Clinic. Currently, retrospective studies are underway to build the largest database of epidemiologic and demographic information of this patient population in an effort to gain insight to the causes of NMO, the appropriate diagnostic work-up, and the appropriate treatments for the disorder in a de-identified fashion. From this work, we will be generating new studies in the near future, the first of which will involve a comprehensive blood draw every three months at one of the three sites. Our hope is that information generated from this study will help us to gain more insights about the disease, how to predict recurrences and when and how to more effectively treat NMO. In the future, we will be recruiting for treatment studies, as well.

Dr. Kathy Zackowski, PhD is an occupational therapist who possesses the unique expertise on evaluation of gait disorders. She and her group at the Kennedy Krieger Institute have focused research efforts on the assessment of outcome and gait disturbances that result from TM. Future studies on the effects of intervention treatments for TM and outcome measures, including gait, are being designed by Dr. Zackwoski's group.

JHTMC contact Information

The Transverse Myelitis Center
600 N. Wolfe Street
Pathology 627
Baltimore, Maryland 21287

Phone: (410) 502-7099 (option 2)
Fax: (410) 502-6736
Email: hopkinsTMcenter@jhmi.edu
Website: www.hopkinsmedicine.org/jhtmc

If you are a patient or the relative of a patient interested in consultations at the JHTMC, you may expedite scheduling of appointments by sending **ALL** of the following: MRIs (CDs or films), MRI reports, laboratory reports (blood and cerebrospinal fluid), and hospital admission/office notes to the above address or fax.

If you are a physician or health provider interested in urgent referrals or consultations, please contact the phone or e-mail above or call the Hopkins Access Line and request to speak with the neurologist on call (800) 765-5447.

It has been a very eventful year at the JHTMC and we are most grateful for all the support that has been provided to us by patients and families of our patients with TM and the enthusiastic support we receive from the TMA. As we move into 2011 we look forward to meeting the challenges before us and remain committed to offering the best clinical care to people with TM and to the critical research needed to better understand this difficult disorder and to develop better and more effective treatments.

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Treating Neuromyelitis Optica (NMO) in the United Kingdom

Kerry Mutch NMO Nurse Specialist, Liverpool UK

NMO is characterised by relapses affecting the optic nerves and spinal cord. Disability accrues with each relapse and can cause visual problems, including blindness and physical disability, including paralysis of limbs. It is essential for people with NMO to be treated early in the condition to prevent relapses. The National Commissioning Group (NCG) in the UK has acknowledged NMO as a rare neurological condition that requires specialist expertise and has funded two specialist centres in the UK: The Walton Centre in Liverpool sees patients north of Birmingham, Scotland, North Wales and Northern Ireland. John Radcliffe Hospital at Oxford sees patients from south of Birmingham and South Wales. These two centres will develop a national UK diagnostic and advisory service, do active research and collaborate with national and international groups working on NMO. Fortunately for those living in the UK, all this is free of charge and funded by the National Health Service.

NMO Clinic at Walton Centre, Liverpool

The NMO group at the Walton Centre consists of a dedicated NMO multidisciplinary team providing a specialist service for patients with NMO. This group includes Dr. Anu Jacob, Dr. Mike Boggild, consultants, nurse specialist for NMO, orthoptist, physiotherapist, occupational therapist, dietician and psychologist.

The NMO clinic aims in delivering a comprehensive service for patients and includes:

- Accurate diagnosis and management combining clinical, imaging and laboratory assessment
- Relapse management including plasma exchange
- Optimising medication regimes including rituximab
- Multi-disciplinary team assessment
- Symptom management
- Lifestyle issues
- Health promotion
- Provides education, information, knowledge and a greater understanding of the condition
- Liaise and refer on to local services

Each individual has the opportunity to be assessed by all the team members and advised regarding their individual symptoms. The MD team will refer and liaise to local therapists within the patient's own area. We are also lucky to have the support of the only charity dedicated to neurological conditions that provide non-medical advice and information regarding benefits, advice about work and various courses.

How to get an appointment in the clinic?

All that is needed is a letter from the GP to the consultant requesting an appointment. Even patients with suspected NMO or where there are diagnostic difficulties can be referred for evaluation.

Contact the Liverpool NMO Service

Kerry Mutch, NMO Specialist Nurse
0151 529 8357
kerry.mutch@thewaltoncentre.nhs.uk

NMO Clinic John Radcliffe Hospital, **Oxford**

This similar centre is lead by Dr. Jackie Palace and Dr. Isabel Leite, consultant neurologists.

Contact NMO service at Oxford

Jon Revis, NMO Nurse Specialist
01865 231905
ms.advice@orh.nhs.uk|

Antiaquaporin 4 Antibody Testing

NMO has been identified as having an association with a newly discovered antibody, anti aquaporin 4, in about 70 per cent of cases. Professor Angela Vincent's lab provides testing for the UK in addition to research on the antibody and its effects.

From the Desk of Dr. Benjamin Greenberg
Director of the TM and NMO Centers; University of Texas Southwestern, Dallas

After moving from Baltimore to Dallas in 2009, I initiated the TM and NMO program at the University of Texas Southwestern and Childrens Hospital. It has been an incredibly busy time and we have had great success in starting our clinical and research programs. At Childrens we have established a multidisciplinary clinic where patients can come for one day and see neurologists, physical medicine and rehabilitation physicians, neuropsychologists, urologists, social workers, nurses and, if needed, get vision testing. We have strived to provide comprehensive care to our patients and have greatly enjoyed the experience. On both the adult and pediatric side, we have multiple research programs that are ongoing in both TM and NMO and are looking forward to a variety of new intervention trials. During this past year we have aggressively pursued reviews of acute therapies and are working on multiple publications to try and improve the standard of care.

We are available for appointments by contacting the following offices:

For Adults:

Call (214)645-8800 and ask to speak to the Multiple Sclerosis and Transverse Myelitis Clinic for a new patient appointment.

For Children:

Call (214)456-5214 and ask to set up a new patient visit with Dr. Greenberg or Dr. Graves.

2011 promises to be an exciting year for the University of Texas Southwestern Transverse Myelitis and Neuromyelitis Optica program. In the last year and half, we have expanded our care and clinical research programs focused on adults and children with these disorders and are looking forward to a very productive year.

As our consultations and patient referrals increase, we continue to have conversations with patients about maximizing their care close to home. It is difficult for everyone to travel to Dallas for care and while we welcome everyone, it is imperative that we develop ways for patients and families to partner with physicians near their home!

When listening to patients and families, we often are told that their physicians "don't know anything about transverse myelitis" or "have never heard of NMO." This can be incredibly discouraging. For a patient with a rare disorder it is not a surprise that friends or families have never heard of it, but when your doctor doesn't understand the condition, it can be frightening and disheartening. Yet, there are many ways to ensure outstanding care close to home from physicians who are not 'experts' in these conditions. I thought I would take this opportunity to list some of my suggestions for maximizing your long term care close to home.

1. You don't need an expert. You need a physician who is willing to listen, partner and learn.

TM and NMO are not high blood pressure, diabetes or stomach pains – they do not get significant attention in medical school throughout the process of training physicians. It is simple math, 99.9% of physicians will never care for patients with these conditions, so their education doesn't focus on them. What you need is a physician who recognizes what he/she doesn't know and is willing to learn. We at the UT Southwestern TM and NMO program spend a significant amount of time consulting with physicians from around the country. We offer advice, discuss cases and treatment options. It is very rewarding to work with health care providers from all over and help them improve their approach to these rare conditions. Having a physician who is willing to listen and *find* the answers to things they don't know is the most important thing to seek.

2. Doctors like to be helpful, so help them help you!

Suggestion #1 implies that there is more than one kind of physician in the world. While that statement seems obvious, most people assume that all physicians are caring, compassionate individuals who went into medicine as a way to help our fellow humans. This is true for most physicians, but not all. Furthermore, even the physicians who are compassionate individuals at heart have dramatically different ways of conveying it. As noted by Jerome Groopman in his book, "How Doctors Think," a physician will typically interrupt a patient describing symptoms within eighteen seconds. Within those first 18 seconds, a physician is already formulating thoughts about diagnosis and treatment. In a world where the average patient follow-up visit lasts for under 15 minutes, how can we expect our physicians to deal with the litany of problems a TM or NMO patient may be experiencing?

For example, you may come to your physician wanting to discuss your bladder function, fatigue, pain control and immunosuppression medication. I would argue that there is no way to deal with all of those issues in one typical visit AND when you list all of these complex issues at the beginning of a visit, your physician instantly has concerns about running late. How can you help your "compassionate-at-heart" physician deliver appropriate care?

Prioritize. You may need three visits to address all of those concerns. At the first visit, when asked, "How are you," you should indicate that you have several concerns, but are focusing on one for today. For example, "Well doctor, I have several issues that I am looking for guidance on, but I realize we can't cover everything today. I continue to suffer from bladder control issues, fatigue, burning pain and have questions about my medication. While it would be great to talk about everything, the most important issue to me is my pain."

At this point you are informing your physician about the presence of multiple issues, but clearly setting expectations for what you want addressed as a priority. The physician will appreciate this and will also be able to decide if some of your issues need to be addressed in tandem. For example, your physician might say, "I understand. Let's talk about your pain and your fatigue, because sometimes those are related." In any event, you will have clearly outlined your goals for the visit and invited the physician to partner up with you in an effort to fix the issue.

3. Make a plan

A lot of mistakes in care occur when a plan is not clearly formulated. For example, you go to a physician to discuss pain and are prescribed a pain medication. You go home, take it and get no relief. For the next 6 weeks you suffer before coming back to the physician, only to hear that we should have gone higher on the dose…. Frustrating.

When leaving the office with a new prescription, you should ask what to expect, what should be done if it doesn't work and how long could it take before efficacy occurs (some medications require weeks of titrations

before a benefit is seen). Knowing that a plan is in place is very reassuring and ensures that patient and doctor are on the same page.

4. Remember, you are in charge, but success requires a team.

You are the boss, but your employees have egos! There is a great joke about a guy who dies and goes to heaven. While being shown around, he is taken to the great cafeteria in the sky. While waiting in a very long line, he witnesses a man in a white coat with a stethoscope around his neck walk in, cut in line, grab lunch and leave without paying. The man complains to his tour guide, "That's not fair; who does that guy think he is"? The tour guide responds, "Oh, that's G-d. He thinks he's a doctor."

While doctors have had a reputation of developing god complexes, most are nowhere near that narcissistic. They do, however, have egos. They want to help. They want to fix people. They want to be of benefit to the lives of their patients. You are the boss of your health, but guidance from a team of health care providers can be critical for success. You need to engage your physicians in being a team with a common goal. When things go well, be sure to give your physician feedback. It can be helpful for judging what works and what doesn't and it can provide positive reinforcement to the physician that their efforts have beneficial effects.

5. Sometimes things don't go as planned; remember the sandwich technique of feedback

Even with the best of intentions, things don't always go as planned. Sometimes medications don't work. Sometimes a phone call isn't returned in a timely fashion. Sometimes a refill doesn't get called in on time. These are not intentional events, but can occur in any office. How can you let your physicians know about your disappointment, give feedback, but avoid a defensive physician or office staff? Employ the sandwich technique. When giving "negative feedback" or criticism, always "sandwich" it in between two positive comments. For example, despite two phone calls about ongoing pain, you did not receive a call back for 48 hours. How can your concerns be addressed?

"Doctor Smith, I just wanted to say how much I appreciate your efforts to help me get control of my pain. Your willingness to try different things and find a medicine that works has been great. Unfortunately, the last time I tried calling in to let you know I was having a pain crisis, I did not get a call back for 48 hours. I know how busy you and your staff are and the care I get is extraordinary, but in this case, things broke down. Can you tell me what I can do to avoid this from happening again?"

Discussing the issue in this way creates the highest probability of getting the issue resolved. This approach is based on the assumption that your physician has a genuine interest in improving the quality of life for his/her patients. If the physician doesn't, get a new doctor!

In the end, having a rare disorder can be scary and frustrating. Your interactions with physicians should be helpful and not add to your problems! Much of the outcome from these interactions can be dictated by you! Doctors are people; they make mistakes. But in general, they want to help. Finding ways to make that effort easier only improves the quality of your medical care.

UK TM Society

UK TM Conference 1 – 3 April 2011

The UK TM Society is organising a TM Conference for the weekend of 1-3 April 2011 (Friday night to Sunday lunch), at Wyboston Lakes Conference Centre which is 45 minutes north of London by train or car and one hour from London Stansted Airport. All TMA members and professionals interested in TM are invited. Our invitation is extended to TMA members from outside of the UK. Bookings are open in January on the following website:

www.tmsconference.org.uk

Dr. Douglas Kerr, who spoke so well at our previous one-day Conference (which was sold out with 140 delegates) in London in 2007, is returning to UK for our 2011 Conference. Because of the weekend residential format, Dr. Kerr will have more time for separate presentations on Stem Cell and other TM Research, as well as Living with TM and a special session on Paediatric TM.

In addition to Dr. Kerr, we will have Dr. Daniel Becker from the Johns Hopkins TM Center in the USA and several eminent UK speakers on Research and Stem Cells, FES Rehabilitation, Neuropathic Pain, Bladder Management, Depression, Fatigue, NMO (Devics) and Paediatric TM. There will be workshops and equipment demonstrations on Physiotherapy and Balance, Orthotics, Rehabilitation Equipment, Continence, Staying Positive and Becoming an Expert Patient.

Wyboston Conference Centre includes meeting rooms, restaurant and bedrooms all under one roof, so there will be lots of opportunity for socialising with other members and speakers.

The TM Society is subsidising the Conference rates which are UK£150 for single occupancy and UK£300 for double, including all speakers and workshops, dinners, lunches and refreshments. Day visitors can also come for UK£40 with lunch or UK£60 with lunch and Saturday Gala Dinner.

Please look for the details of the conference on our website:
www.tmsconference.org.uk
or contact Lew Gray at:
lewgray@blueyonder.co.uk for further information.

TMA to fund Johns Hopkins Cognitive Impairment Research in Transverse Myelitis

Dr. Adam Kaplin is an Assistant Professor and Clinician in the Departments of Psychiatry and Neurology at Johns Hopkins University. Dr. Kaplin is a clinician in the TM, NMO and MS Centers. Dr. Kaplin has identified cognitive impairment in TM through both pilot studies and clinical observations at the TM Center. The TMA is awarding Dr. Kaplin $25,000 to test a potential treatment for cognitive impairment using animal models of TM. Dr. Kaplin has cared for hundreds of patients with TM at Johns Hopkins. Dr. Kaplin also serves on the TMA Medical Advisory Board. He is a regular participant in our symposia, in our older teen and young adult retreat weekends and in our family camps. He does so much for the TMA and for our community. It is an honor to be able to support his important work. The following is an abstract describing the study.

Cognitive impairment (e.g., memory and concentration difficulties) occurs in approximately 50% of patients with Multiple Sclerosis (MS), and pilot studies and clinical observations at Johns Hopkins Hospital suggest that such changes are also comparably prevalent in transverse myelitis (TM) patients. Currently there are no treatments for autoimmune cognitive impairment. The hippocampus is a brain region that mediates the generation and recall of memories, and increased cortisol levels cause degeneration of this structure. Hippocampal degeneration has been demonstrated in a variety of disorders that often result in cognitive deficits, including MS. Carbenoxolone (CBX) is a compound that decreases the rate of formation of cortisol in the hippocampus through its inhibition of the enzyme 11b-hydroxysteroid dehydrogenase 1 (11b-HSD1). Preliminary data collected in our laboratory suggest that CBX treatment improves memory as measured by recall in a fear conditioning paradigm in experimental autoimmune encephalomyelitis (EAE), the animal model of autoimmune central nervous system injury like TM and MS. Data demonstrated that CBX-treated animals exhibited a 14% increase in total freezing time in response to the neutral tone compared to untreated controls, suggesting that CBX may improve memory and recall in an animal model analogous to TM of autoimmune cognitive impairment. To confirm this preliminary work, two models of TM and one model of EAE will be employed in experiments designed to test the efficacy of CBX at improving cognitive deficits associated with TM.

TMA Retreat Weekend at Victory Junction 2010

The TMA retreat weekend at Victory Junction Gang Camp took place on October 8 – 10, 2010. There were people with ADEM, NMO and TM who came from across the United States. While at camp, the older teens and young adults who have these disorders are given the opportunity to create friendships which continue after camp. Most of them are in regular contact with each other, including through a very active community on Facebook. Victory Junction is a wonderful camp, and the directors and staff offer the children and families an exceptional experience. The facility is completely accessible, as is the recreation program. While the people with these disorders face significant challenges in their lives, so too, do their siblings and parents. A vibrant support community has developed from these relationships established at camp.

On Saturday morning, an excellent educational program was offered by Drs. Douglas Kerr (Biogen idec), Adam Kaplin (Johns Hopkins), Daniel Becker (Kennedy Krieger), Frank Pidcock (Kennedy Krieger), Michael Levy (Johns Hopkins) and Janet Dean (Kennedy Krieger). The physicians and medical staff, including Dr. Peter Sim, the medical director at Victory Junction, were available to respond to questions and to discuss specific issues throughout the weekend.

Cody Unser presented her documentary, 'Cody: the First Step,' on Saturday afternoon. After showing her incredibly inspirational film, Cody led a discussion with the audience. If you haven't had a chance to see Cody's film, I strongly urge you to watch it. It is playing across the country on public broadcasting stations or the video can be ordered from Cody's web site: www.cufsf.org/

Martha Mann also attended our retreat weekend. Martha is a nurse from the University of Texas Southwestern who also works for the Accelerated Cure Project repository. During the weekend, Martha was able to add nine new people to the repository, and substantially increased our enrollments with ADEM. The logistics of doing these collections during camp were extremely difficult, and we applaud Martha and Betsy Coates, a nurse on the medical staff at Victory Junction, for their efforts. In addition to the actual enrollments, they did a great job of raising awareness of the Accelerated Cure Project repository.

Camp is a life-changing event for the people who come with these disorders, for their families, for the physicians and medical staff and for all of the wonderful volunteers who come from around the country. Some of the volunteers have been coming to our camps since our first retreat weekend in 2006, and have become good friends and supporters of our community. Some of the volunteers have ADEM, TM and NMO and come to support these wonderful teens and young adults. Pauline and I were thrilled to have our son, David, and his wife, Kathryn, attend as volunteers at the retreat.

Alisa Patrone came with her daughter, Jessica, who has ADEM (our camp was likely the largest gathering of people with ADEM we've ever had; at camp, at a symposium, or anywhere else). Alisa and Jessica are from Oregon.

When Jessica was asked how she liked camp, she said, "It was the first time I felt normal since before I was sick." To me that says it all. This girl is such a fighter, but I see a renewed hope and determination in her after the camp. Thank you Sandy, Victory Junction, Kyle & Pattie Petty, and everyone who helped make this possible. We are so blessed to be a part of this.

Julie Rogers Schneider came with her family; their son, Josh, has ADEM. The Schneider family came from Louisiana.

Victory Junction was an amazing time for our family. It is a magical place. The camp is beautiful! We all had so much fun. It didn't take much time for our family to loosen up and start singing and dancing. It helped to meet other families that are going through the same things as our family. We have made life long friendships and have great memories for life. It was so helpful having all the doctors there. They took the time to talk with each family, answering question we all had. Sunday morning arrived too quickly, bringing an end to an awesome weekend. Victory Junction is so special for everything they do. You can feel how much they care for everyone. This has truly changed our lives. THANK YOU!
Brian, Julie, Josh, and Matt Schneider

Many of the teens, young adults and families cannot afford the travel expenses to get to camp. The TMA has a camp fund that we use for the purpose of helping people from our community make it to camp. We want to recognize all of the generous contributions we receive from our members to the camp fund. Without your kindness and support, many of these people would not be able to participate in this wonderful camp experience. We urge you to continue to support this valuable program. Thank you!

And thank you to Pattie and Kyle Petty and to the wonderful staff at Victory Junction for making this opportunity happen for our community.

TMA VJ Family Camp 2011

Our next family camp is scheduled for October 6 (Thursday late afternoon arrival) to October 9 (Sunday at noon), 2011. The camp is for children 6 - 16 with ADEM, NMO or TM, their siblings and parents. Camp will accept applications beginning on June 1. Please plan to have the entire application, including the medical portion, sent in as close after June 1 as possible. The application, as well as information about camp, may be found on the Victory Junction web site:

http://www.victoryjunction.org/

The 2010 Neuroimmunologic Disorder Symposium in Dallas

Susan Daniels

As soon as I walked in the room, I had to walk out again. It was 2008 and I was attending my first Transverse Myelitis Symposium. Technically it was entitled: *Rare Neuroimmunologic Diseases* and I was there for one of those rare diseases. Transverse Myelitis came into my life in the Fall of 2006. One day I was fine and then I wasn't. Lots of medical tests, doctor appointments and physical therapy visits later, things gradually improved, albeit with unpleasant residual symptoms remaining. But I didn't grasp how lucky I was until that day when I walked into the ballroom at the Marriott. For the first time I saw other people with Transverse Myelitis. And along with the TM people were caregivers, service dogs, canes and crutches and wheelchairs. It was more than I was prepared for. I started crying and left.

In the hallway, I could watch from afar. I could hear the talking and laughing, the questions and the answering voices. People were checking in at the desk, meeting each other, finding their places at the tables in the ballroom. I started back in again, this time with more readiness for what my eyes were taking in. I could walk. I could walk without assistance. Here were crowds of people who couldn't. They were there for the same reasons I was. To learn. To share. To get answers for all the difficult times that TM brings with it.

Even though I had had a good experience at the Seattle symposium, two years later, I couldn't decide whether to attend the next symposium in Dallas. I had met many wonderful people in Seattle. Would it be the same? What would I learn? Would it be worth the cost? I decided to attend, then changed my mind. Then very late, I changed my mind again and

made my reservations. The airplane ticket would have been much less had I just made up my mind a week or two earlier. The trip would be expensive. But still, I couldn't stay away.

Getting to Dallas from Seattle is about a four-hour plane ride; a bit longer one direction than the other because of the jet stream. Upon arriving at DFW, I knew the cab ride would be about $50. I opted for the $18 shuttle van. In spite

of the sign, it was a thirty-minute wait, but I was in no hurry. The shuttle guys were friendly and the sun was shining and it was not too hot. All the shuttle drivers seem to know each other well. When the van appeared, I asked to sit in front. We then drove to pick up other riders before leaving the huge airport campus. The driver talked on a cell phone to someone in a foreign language. When he hung up I asked which language. "Amharic," he explained. At least that's what I think he said. Not knowing the language, I asked, "What country?" "Ethiopia." "Do you live in a community together?" I asked. He explained that yes, they lived in mostly one area of Dallas. There are many Ethiopians living near Dallas. In fact, he explained, there are over 80,000 Ethiopians living in Dallas. Many of them fly to Ethiopia once a year. There is even a regular flight from Dallas to Ethiopia that mainly serves their community. Later I found out that there is an Ethiopian American Chamber of Commerce and a very supportive community culture in the Dallas area. Who knew?

The 45-minute drive into Dallas was as expected. Lots of freeways and overpasses with many cars and buildings and more buildings. There were few green trees and no salt water as found in the Pacific Northwest, but it was familiar as home to me. I have been to Dallas many times. In fact, I used to live there. I went to grade school and junior high there, and college just north of Dallas. And yet, it could have been any city for the conference as I would never leave the hotel complex.

I checked in right away and found my room. As I slid my room card out of the slot and opened the door I found the otherwise dark room lit by a spectacular moon. I put down my bags and immediately got out my camera. It was a 'through the window' shot, but the scene was irresistible. A "Welcome to Dallas" scene.

The next morning the meetings began. The hotel furnished a wonderful breakfast. It was a good time to meet new people and greet those I'd seen from two years ago in Seattle. Lew was there from London, Ivan was there from Sri Lanka, and Deborah from California. Later that evening, I was fortunate to have a delightful dinner with Colleen and her daughter, Amanda. We had met in Seattle in the room where we gave blood samples for the Accelerated Cure Project.

Attending one of these symposiums is a cross between attending a regular conference and attending a class reunion. The information is excellent, the presenters knowledgeable, and the take away knowledge can't be beat. Then you have your 'classmates.' We all share a common experience and yet we each experienced it differently. Many of us were hospitalized. Some of us nearly died. Some of us can walk again. Some of us can't. But all of us, every one of us, is different now than before the unexpected happened, before our lives changed forever with TM, ADEM, MS, NMO or whatever specific occurrence got us and brought us to this symposium.

The presentations were made by experts in their fields. I usually sat in the back of the room as I find myself restless and unable to tolerate an hour of sitting. Nevertheless, I found myself staying and staying. The presentations were excellent. I found each presenter I talked with genuinely interested in the topic and its affect on each of us.

After the symposium, I found these factoids in my notes:

One out of a thousand is the risk in the general population for MS, more in the northern European populations. The risk is mutable with migration. You can move away from the risk. The further from the equator you live, the higher the risk. Eighty-four percent of people diagnosed with MS are women. MS is likely not caused by a gene, as diseases such as Huntington's are, although it might be caused by a combination of genes. We will know more as genetic testing becomes more common and more refined. It's rare to find a person with MS who wasn't also exposed to Epstein-Barr. The less the ultraviolet radiation one is exposed to, the higher the risk of MS. Vitamin D deficiency is also an indication of higher risk.

One memory I brought back happened at the banquet Saturday night. Among the people sitting at our table was Bruce Volpe, who spoke in the Basic

Science portion of the symposium. Dr. Volpe commented that the goal of treatment is not to wipe out the armies,

but to aim at the generals in charge. This analogy made sense and caused me to wonder whether we should take out the generals or whether we should try to persuade them to act differently. Later, when I hypothesized that these diseases could be triggered by a mold (due to the higher latitudes of incidence), he was thoughtful. Discussions such as this one are small moments that add up to a good experience.

The thing I remember most about this symposium was the presence of children. In Seattle, there had been no children attending, at least not that I remembered. Probably because the tuition at the Dallas symposium allowed you to bring along family members at no charge, a number of children attended with their families. I was fortunate to meet several of the parents and during a break, spoke quietly with one family about their experience when their son was stricken. As they described their ordeal, tears ran down my cheeks. They were loving, normal parents. Why did this happen? Their son reminded me so much of my grandson, bright and energetic. Their story was not unusual for TM parents: the undiagnosed onset, the treatment, the disbelief, and the coping that continues daily. Heartbreaking.

Meanwhile, the kids, who had not met each other before, had immediate affinity with each other. They talked easily and openly about their own experiences, treatments, and ongoing therapies. It was stunning for me to hear the dramatic stories told with such acceptance. On a break they all watched as Cody Unser, also a TMer, demonstrated how to go up and down an escalator in a wheelchair (not something we recommend for anyone … including Cody!). Cody was delightful with all the kids and their parents.

During the formal presentations, the children gathered in an adjoining room and entertained themselves with crafts, cards, puzzles and just goofing around, as kids will do.

During one of the breakout sessions, the children were gathered in a room and asked about their experiences. The UT Southwestern Medical Center's camera rolled as they each talked frankly about themselves, about being in a wheelchair, about how other kids treat them now, their parent's reactions, and their ongoing treatments. Most of them had onset less than two years. One had had TM for eight years. Being in the room during the filming was the most meaningful part of the symposium for me.

As a photographer, I often experience events through the lens of my camera. This time, not so much. Yes, I took pictures. But I found myself listening

more and shooting less. My TM is still with me. Attending the symposium did not make it better. However, it was for the second time a positive experience for me. I look forward to attending the next symposium.

Susan Daniel lives in Port Orchard, Washington. These and other photos Susan took at the symposium can be found at: ***www.camerabean.com/Events/2010-Rare-Neuroimmunologic***

2010 TMA Distinguished Service Award Douglas A. Kerr, M.D., Ph.D.

Dr. Kerr is the recipient of the 2010 TMA Distinguished Service Award. The award ceremony was held during the Saturday banquet at the Rare Neuroimmunologic Disorders Symposium in Dallas in September. Debbie Capen, Dr. Benjamin Greenberg, Dr. Michael Levy, Paula Lazzeri, Chitra Krishnan and Sandy Siegel participated in the award presentation.

I met Dr. Kerr in 1999. It was the first symposium of The Transverse Myelitis Association in Seattle, Washington. It was the first large gathering of people from the TM community; more than 100 people attended. Dr. Kerr made a number of educational presentations about TM and symptom management. Dr. Kerr and I also conducted a four-hour long question and answer and discussion session with the attendees. It was a remarkable weekend.

When Pauline was diagnosed with TM in 1994, there was no TM specialist in the world and if you reviewed the medical literature, you would have found an amazing paucity of information about this disorder. Almost all of the publications were descriptive and there was virtually no research on TM. No one in the medical community really understood TM, knew what it was, knew effective ways to treat it, and there was an amazing imprecision in regard to its diagnosis. And most frightening and disconcerting of all was that there was not a single medical professional in the world who was either interested in asking the pertinent questions, seeking the answers or caring for people in this community. Dr. Kerr single-handedly changed that entire dynamic; Dr. Kerr forever changed the lives of everyone who has ever received a TM diagnosis and he has changed the future for every person who will ever get the TM diagnosis.

When Dr. Kerr came to Seattle for that first meeting of our community, he was in the final year of his residency at Johns Hopkins Medical School. He was Chief Resident of Neurology with nothing but tremendous potential for a brilliant career in whatever specialization he chose. Dr. Kerr was transformed during the weekend symposium in Seattle. Spending the weekend with people with this little understood disorder stimulated his intellectual curiosity and sharing in the suffering he saw all around him touched his heart in the most intense ways. Dr. Kerr was able to experience the isolation and the fear and the anxiety and the ignorance that people suffered because you will not find a more empathetic person on the face of the earth. Shortly after Dr. Kerr returned from Seattle, he began the process of working through the intricacies of establishing a Center of Excellence in Transverse Myelitis in the Department of Neurology at the Johns Hopkins University Medical School. Dr. Kerr announced his specialization in transverse myelitis. Dr. Kerr made a commitment to offering the best clinical care to people with TM. He made a commitment to focus his research efforts on finding the cause of TM, the most effective acute treatments and the most effective symptom management therapies. And perhaps the most hopeful direction of this research for people who have these disorders, he became committed to finding restorative therapies for people who got TM. Dr. Kerr was also entirely committed to providing education in the broadest context. He was committed to educating physicians about TM. He was committed to collaborative education with scientists to refine and develop the most effective questions to ask and the best methods for arriving at the answers and then translating those results as quickly and responsibly as possible to the provision of patient care. And, perhaps, he was most committed to providing education to the patient community so that they understood their disorder and would be able to become the best advocates for their own medical care.

It is difficult to characterize the impact Dr. Kerr has had on the TM community; through his scientific research, through his patient care and through his efforts to provide the best education. Pauline and I often reflected on just how incredibly amazing it is that someone of Dr. Kerr's caliber, personality, intellect, and character became connected with our Association and our community. Over the years that I have known Dr. Kerr, we have worked very closely on a multitude of projects and the development and achievement of shared goals. Over the years, Dr. Kerr has been a colleague and a mentor. And over the years, Doug has become a good, trusted, caring and loyal friend. Pauline and I have nothing but

the greatest respect and admiration for our friend and we love Doug as a member of our family.

Doug is one of the most remarkable people I've met in my life. He has an amazingly brilliant and creative mind. He thinks of problems in ways that few others think about them and he is so focused and driven to find the solutions to these problems. Doug is one of the most compassionate, sincere, caring, kind and sensitive people. He is accepting of people, he is so amazingly not judgmental and he possesses one of the most positive spirits you are ever going to see in a human being. Doug is charming and warm and so incredibly well spoken. He is totally captivating whether he is talking to a room full of scientists or doctors or patients or his friends or his two daughters. He is wonderfully charismatic and genuine.

Dr. Kerr serves on The Transverse Myelitis Association Medical Advisory Board. He is also very intimately involved in providing guidance for our Association across all of our programs and activities. He is available to serve us in whatever capacity he is asked. It is important to explain the relationship between the TMA and Doug Kerr and what Dr. Kerr derives from this relationship. Between 1994 when the TMA was established until the end of 1997, our Association grew to 187 members. When Dr. Kerr became involved in 1999, we had fewer than 1000 members. Today we have more than 8000 members from more than 80 countries around the world. While we have been able to accomplish a great deal over the past 16 years, we remain a very grass roots organization. Most of the work of the TMA is performed by the four officers who are also the board members of the Association. All of the officers are volunteers; the TMA has no employees.

The international headquarters of the TMA is Pauline's and my home. The address and telephone number identified in all of our publications, on our web site and in the multitude of government and related organization listings and links is our home information. All of the officers work from home. We pay for our own internet access and long distance phone bills and for our own supplies and equipment. We also try to pay for most of our travel expenses. We have almost no overhead. Jim Lubin is the only officer who does not have a full time job. Jim got TM when he was 21 years old and has been a full quadriplegic and ventilator dependent for more than 20 years. Jim is also our IT Director and he does all of our web work by sipping and puffing Morse code into an adaptive device. The Association has no membership fees and we do not charge for any of our publications. We publish newsletters and journals every year. We operate exclusively on the basis of the charitable contributions of our members, many of whom are on disability. We also do not spend any of our resources on fundraising. We have a support network across the country and around the world. All of our support group leaders are also volunteers. I am the only officer who does not have TM.

The give and take between Dr. Kerr and The Transverse Myelitis Association is not symmetrical. The asymmetry of this relationship has been a great disappointment for me, personally, but that is part of the reality of managing a voluntary position with limited time and resources. Raising money has been extremely difficult. Dr. Kerr is not in this for our money, because we usually don't have much. We have managed to raise enough money to support a research position at Johns Hopkins. This person was mentored by Dr. Kerr and she became the first Executive Director of Project RESTORE. Our support of this position was one of the most important investments the Association has ever made. While our support of Doug's work has been very small in comparison to the costs associated with his work, he has been amazingly generous in his public recognition of this support. Doug has never failed to acknowledge our support in every public activity, from symposia to conversations in small groups to mentions in almost all of his publications. The magnitude of his recognition far outmatches the size of the support he has received from us. You will not find a more kind, generous and sensitive person. And he acknowledges us because he is grateful. His recognition is sincere. Doug isn't measuring the dollars in his sentiments, he is measuring the source of the funds. The vast majority of our contributions derive from small contributions and fundraisers that are conducted by our members. One of our members has a small farm and he sells canned vegetables from a roadside stand. He makes monthly donations of $25 that come from his sales. We have children and adults participating in hoop-a-thons, roll-a-thons, hop-a-thons and various other small fundraisers. There are 80 year old people with TM who send me books of stamps for our mailings; because they want to help in some way and just don't have the resources to make financial contributions. Doug knows the source of our funding and is more than gracious in his recognition and gratitude for our support.

When Doug announced his specialization, he began to attract significant numbers of people with TM to his Center of Excellence. He developed a systematic process for collecting data and information and began to study the disorder. This database now contains the experience from more than 800 people with TM. Almost all of this work was funded by Doug personally. He also began to publish about TM. More meaningful work has been published about transverse myelitis in the past 10 years than in all of the time before Doug became involved in studying the disorder. I am acutely aware of

the impact Doug's publications have had on the education of the medical community about TM. There is no one in the world who has spoken to more people with TM than me. I am called every single day by people all around the world who are going through an inflammatory attack (or their family member) or who have been recently diagnosed, or who were diagnosed years ago, but just found our information on the web site. I can easily compare people's experiences before Dr. Kerr became involved to the post Dr. Kerr's involvement era. The differences are dramatic. Dr. Kerr focused his earliest publications on developing an algorithm for ruling in a TM diagnosis. There is absolutely no doubt in my mind that through these publications and his wonderful work in raising awareness that better diagnoses are being made and that they are being made more rapidly. Time is a critical component of treatment, because until the inflammatory attack is resolved, the inflammation is causing irreparable damage to the spinal cord. Dr. Kerr has also published good information about the acute therapies for TM. As there has been almost no good scientific evidence for acute therapies, the publication of this expert experience has been invaluable. Again, all of this work has been personally funded by Dr. Kerr with minimal support from the TMA.

In an effort to create a foundation for further research in TM, Dr. Kerr assembled an impressive team of neuroimmunolgists to develop the diagnostic criteria for transverse myelitis. The work was initiated during a workshop at one of our symposia, and then continued with the development of a paper that was published in 2002. The development of the diagnostic criteria for idiopathic transverse myelitis represents one of the major milestones in the classification of these rare neuroimmunologic disorders. In addition to the importance of this work for TM research, the publication of this work has continued to improve the accuracy and the more timely diagnosis of this disorder. All of the efforts to publish this work were self-funded.

As TM is so little understood, it is extremely difficult for people to receive good medical care. As TM appears to occur in a random fashion across the country and around the world, there are many people who do not have access to physicians who have any experience or knowledge about this disorder. Dr. Kerr has made himself available to every physician who ever calls seeking guidance about diagnosis and treatment. It would be impossible for me to recount every episode in which I have asked Dr. Kerr to offer guidance to a physician who was seeking advice. Over the past ten years, it has happened often, and Dr. Kerr has never been too busy to help.

Beginning in 2001 we started to hold biannual education symposia for scientists who study the rare neuroimmunologic disorders and for the people who have these disorders. These are concurrent sessions. It is hard to imagine that there is a more effective and comprehensive education program for patients held anywhere in the world or a more dynamic science program. One of the really unique aspects of this program is that the scientists who do this work are regularly brought together with a large number of people who have these disorders. It is a highly motivating and inspiring experience for all involved. The amount of work that Dr. Kerr has put into developing, designing, funding and participating in these programs is impossible to describe. The effort is enormous and the benefits that result for the researchers, clinicians, patients and caregivers are immeasurable. In addition to the symposia that have been held in Baltimore, Dr. Kerr has conducted these educational meetings in Seattle, Los Angeles, England, Ireland and Scotland. The fact is that Dr. Kerr has offered these educational programs any place we've asked him to go.

While we had offered educational programs for adults, Doug and I were concerned that we had never offered a program focused on families and an experience for children who have TM and the related disorders. Thus, in 2001, we began the planning for a children's and family workshop in Columbus, Ohio. We raised enough money to bring in families from around the world for a four-day workshop. The parents were provided a four day education program that was offered by the experts in the discipline. The older children were taken on an adaptive recreation adventure and the younger children were taken on trips to the Columbus Zoo and to a children's science museum. The doctors who came to present brought their own families at their own expense and there were no honoraria. Dr. Kerr was instrumental in every aspect of this workshop and as in every case; his involvement set the example for excellence and generosity for all of the physicians participating.

Our children's workshop has evolved into a relationship that The Transverse Myelitis Association has with Victory Junction Gang Camp, one of Paul Newman's Hole in the Wall Gang Camps. Many of the children in our community are severely impacted. It is difficult to find a camp experience for children who are quadriplegic and vent dependent. Our children experience a great range of deficits and difficulties. We have most definitely found a most inclusive and wonderful experience at Victory Junction. We have also been able to work with the camp to fashion a unique experience for our children and their families. Dr. Kerr has, again, been instrumental in planning and developing every aspect of the opportunities that have been offered to us at camp. Every other year

we have a weeklong camp for children from ages 6 to 16 who have TM and their families. And then every other year we have a retreat weekend for older teens and young adults. Dr. Kerr and other physicians from our medical advisory board donate their time at camp. They spend the week and the weekend at camp and make themselves available to the families. They offer individual consults with families to offer guidance to take home with them to their treating physicians. Finally, they offer an education program to the adults who have these disorders and to the parents. The physicians also spend time with the children doing arts and crafts, horseback riding, dancing and singing, playing all sorts of games and swimming in the pool. Again, Dr. Kerr and the other physicians donate their time and come to camp at their own expense.

Dr. Kerr has never said no to me about anything I have ever asked; and I have asked so much from him. Whether to conduct an education program, to write an article for the newsletters or journals, to help with planning camp or conducting our camp program, to provide guidance to our Association, he has always been there for us … without failure. I am regularly contacted by family members of a person in the middle of an inflammatory attack, and they are often not receiving rapid or effective medical care. Dr. Kerr is always on call for me. I have regularly called his home phone, his cell phone, his office phone and his laboratory phone, morning, day and night. The voice on the other end is always gracious and accepting. His reaction is always positive and ready to provide help, if asked.

Shortly after Dr. Kerr established the Center of Excellence and his specialization in transverse myelitis, a very young child developed TM and became his patient. I don't believe that this little boy was even a year old. His attack was virulent and the damage caused to his spinal cord was the most extensive I've ever heard about in my more than 16 years of experience. The attack had virtually destroyed this child's entire spinal cord. This child eventually passed away. This loss was devastating for Doug. Doug and I had the opportunity to talk about this child and this experience on a number of different occasions. I watched this experience change Doug; he carried the memory of this child in his heart and his mind. As focused and motivated as Doug was from the outset of this journey, I witnessed this loss drive him even further. I know that the memory of this child motivates his work every single day of his life, and that he honors this child in the manner he cares for every single one of his patients.

I work with physicians all the time who are brilliant diagnosticians and can provide treatment with great effectiveness, but they lack empathy and compassion. Working with a physician who lacks these qualities is a challenging experience, to say the least. The idea that a doctor for human beings might have difficulty relating to a human being is an interesting proposition. Doug's patients love him; they all do. Everyone knows my home phone number and no one seems particularly reticent about calling me. If people were having a difficult time with Dr. Kerr, I would definitely be hearing about it. I have never heard anyone complain about Dr. Kerr – about anything. He provides excellent care to his patients and he cares! And I have had the opportunity to observe Dr. Kerr's practice of medicine first hand, as Pauline became his patient at Johns Hopkins.

Many of the symptoms of TM are extremely difficult to treat. It is almost never the case that the first medications, at the first dose prescribed, are going to manage neuropathic pain or spasticity or fatigue or depression. Bowel and bladder problems are so difficult for both the patient and the physician. The treatment of these symptoms requires enormous amounts of time and attention; the medical system in America is not about enormous amounts of time and attention. Dr. Kerr is amazingly dedicated to his patients, and he remains committed to finding answers to their issues and devoted to their quality of life. It is not unusual for me to hear from a patient that their doctor has given up on trying to find the answers for their pain or

spasticity or neurogenic bladder. I have never had a patient ever tell me that they have worn out Dr. Kerr. He genuinely cares.

Doug has been amazingly generous about cooperation and sharing. I witnessed this characteristic about Doug repeatedly in so many different situations. He is willing to collaborate, cooperate and to share; with his colleagues at Johns Hopkins, and with his colleagues across the country and around the world. He has been incredibly generous with his students and inclusive in sharing opportunity and credit. I have been in academia for long enough and have been around academia long enough to have observed the competitiveness that often leads to an unwillingness to share and collaborate. I have never met anyone with a more positive spirit. I have witnessed his kindness and his cooperation and his sharing spirit regularly and across every aspect of his work in medicine, science and academics.

In the short time that Doug directed the TM Center at Johns Hopkins he had trained three physicians who have all developed specializations in transverse myelitis and related neuroimmunologic disorders. The TMA works with all three of these physicians and they have developed many of the same wonderful qualities that they learned from their mentor. They are excellent and caring clinicians, brilliant scientists and devoted educators. Each of them has their unique qualities and strengths, but they all bear the very positive characteristics that were developed in their relationship and mentoring by Dr. Kerr.

Everyone Dr. Kerr has worked with and collaborated with and shared life experience with has been made better by their association with this man. Doug made everyone better through his guidance, through his instruction and through his example. He made them better scientists, he made them better clinicians, he made them better teachers and he made us all better human beings.

It was a distinct honor to present Dr. Douglas Kerr with the 2010 TMA Distinguished Service Award.

The Accelerated Cure Project Repository – A Research Resource for Curing Demyelinating Diseases

Scientists working to cure diseases often need access to biological samples and information from people with those diseases. In the case of rare diseases, however, those samples can be hard to come by. So the TMA has teamed up with the Accelerated Cure Project (ACP) to help provide those valuable samples and data to scientists who are studying TM, NMO, ADEM, ON, and MS.

In 2006, ACP established a biorepository to collect blood samples and data from people with MS that could be used in research. Soon afterward, the TMA and ACP joined forces to include samples and data from people with other demyelinating diseases as well. More recently, the Guthy-Jackson Charitable Foundation has provided additional support for enrolling people diagnosed with NMO into the repository. Combining samples from people with these similar-but-different conditions into a single collection not only saves money due to the efficiency of a single operation, but also enhances research because it allows scientists to easily compare different demyelinating diseases in their studies.

So far 2,476 people have enrolled in the repository. This number includes people diagnosed with each of the conditions listed above, as well as friends and family members who serve as control subjects. Scientists working on 43 different research projects have been granted access to samples and data. Most of these researchers are analyzing blood samples for genetics, proteins, gene expression, antibodies, etc., but there are also a few data-only projects. The repository operates under a data-sharing model, meaning that scientists who use the repository must return the results of their experiments to ACP, which can then share those results with any other scientist who is interested in learning from them.

The repository has seen a recent surge in the number of scientists interested in using its samples to learn about NMO. New advances in the understanding of NMO as well as research funding from Guthy-Jackson have motivated scientists to study topics like the genetic origins of the disease and the effects of an NMO-associated antibody on proteins and cells. ACP has also recently received several sample requests from research teams developing assays to distinguish among different demyelinating diseases. These projects may lead to new understanding of disease mechanisms as well as faster diagnoses in the future. Another project involves mining the data contributed by TM and NMO participants to explore the effect of treatment early in the disease.

You can learn more about the research being supported by repository samples and data by visiting:
www.acceleratedcure.org/repository/research.php

Would you like to contribute to this valuable research resource and help scientists who are working to cure TM, NMO, ADEM, ON, and MS? If you have been diagnosed with one of these diseases and have not already enrolled in the ACP repository, we invite you to join and share your samples and da-

ta with researchers around the world. Participation consists of a blood draw and an interview at one of ten clinics (collection sites) located across the US, which are listed below. This is not a treatment study. There are no drugs involved.

If you have NMO and do not live near a collection site but can travel to one, funds may be available through the Guthy-Jackson Charitable Foundation to offset your travel expenses. Additionally, if you have NMO and are unable to travel to one of ACP's collection sites, a nurse may be available to travel to your town to conduct the study visit there.

If you're already a repository participant, you may get a phone call or a postcard asking you to return to the clinic to contribute follow-up samples and data. One of the particularly valuable aspects of the ACP repository is that it is a longitudinal study, meaning that participants are asked to return over the course of their lifetime for follow-up visits. Having participants return for follow-up visits allows ACP to provide valuable samples and data to researchers who are studying disease course and the impact of medications on progression, among other critical topics. Your continued involvement enhances the value of the repository and accelerates research into the causes of ADEM, MS, NMO, ON and TM.

To learn more about participating in the repository, contact the study coordinator at the site of interest; call ACP's repository director, Sara Loud, at (781) 487-0032; or visit the repository section of the ACP website at www.acceleratedcure.org/repository.

Repository Collection Sites

University of Massachusetts Medical School
Multiple Sclerosis Center
Memorial Campus, 119 Belmont St
Jacquith Ground
Worcester, MA 01605
Janice Weaver (Study Coordinator)
508-793-6562
weaveJ01@ummhc.org

Johns Hopkins
600 North Wolfe Street
Pathology #627
Baltimore, MD 21287
Gita Byraiah (Study Coordinator)
410-502-6160
gbyraia1@jhmi.edu

Multiple Sclerosis Clinical Center University of Texas Southwestern Medical Center at Dallas
5323 Harry Hines Blvd.
Dallas, TX 76051
Stephanie Taylor (Study Coordinator - ALL)
Stephanie.taylor@UTSouthwestern.edu
214-645-7949
Martha Mann (Study Coordinator - NMO only)
Martha.Mann@UTSouthwestern.edu
214-645-0543

Shepherd Center, Inc.
2020 Peachtree Road NW
Atlanta, GA 30309
Elizabeth Iski (Study Coordinator)
Elizabeth_Iski@shepherd.org
Erica Sutton (Study Coordinator)
Erica_Sutton@shepherd.org
404-350-3116

The International Multiple Sclerosis Management Practice
Multiple Sclerosis Research Center of New York
521 West 57th Street, 4th Floor
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Sydni Edwards (Study Coordinator)
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dklebus@stanford.edu

TMA Support of the Accelerated Cure Project

The Transverse Myelitis Association made a $10,000 donation to ACP during their annual banquet event in November. The donation was made by Hollie Schmidt, Vice President of Scientific Operations at ACP. Hollie is a tremendous supporter of the TMA; we were honored to have her represent the TMA for this presentation. The donation is designated to support the collection of samples from people with ADEM, NMO, ON and TM into the repository. We are proud of our support of the Accelerated Cure Project and have great hopes that the work they are doing will facilitate research to find better diagnostic tools and answers as to the causes of all of these rare neuroimmunologic disorders.

The Affordable Care Act - *what it means for those with pre-existing conditions*

The following information is provided as a brochure by the Office of Consumer Information and Insurance Oversight in the Department of Health and Human Services. You can also obtain this brochure by going to: ***www.healthcare.gov/center/brochures/pcip.pdf***

Are you uninsured and having a hard time finding health coverage? Have you been turned down for insurance because of a pre-existing condition?

You May Be Eligible for the New Pre-Existing Condition Insurance Plan People with pre-existing conditions face daunting challenges - and high costs - when they go shopping for health insurance. In most states, private insurance companies can refuse to insure you if you have a pre-existing condition.

The Pre-Existing Condition Insurance Plan is a new program created

by the Affordable Care Act to help provide coverage for uninsured people with pre□existing conditions until new insurance market rules go into effect in 2014.

www.HealthCare.gov

The Affordable Care Act helps knock down barriers to coverage for uninsured people with pre□existing conditions The Affordable Care Act helps uninsured people with pre-existing conditions get high quality care, at affordable prices, and get some control back over their own health care. It does so in two ways. Starting in 2014, discrimination based on a pre-existing condition by an insurer will be prohibited in every state, and you will have access to affordable private health insurance choices through a new organization called an Exchange, a competitive marketplace for health plans. Until then, the new law establishes a transitional program called the Pre-Existing Condition Insurance Plan (PCIP) to make health coverage available to you if you have faced barriers to private health insurance because of a pre-existing condition, and have gone without coverage for at least six months.

The Pre□Existing Condition Insurance Plan - an important new option for you The Pre-Existing Condition Insurance Plan, which is administered by either your state or the U.S. Department of Health and Human Services, will provide a new health coverage option for you if you have been uninsured for at least six months, have a pre-existing condition or have been denied health coverage because of your health condition, and are a U.S. citizen or are residing here legally.

How will the Pre-Existing Condition Insurance Plans be set up? The Affordable Care Act provides federal funding to support Pre-Existing Condition Insurance Plans in every state. The program may vary depending on which state you live in. Some states have requested that the U.S. Department of Health and Human Services run their Pre-Existing Condition Insurance Plan. Other states have requested that they run the program themselves. You can go to www.HealthCare.gov and find out how the Plan will work in your state.

As of July 1, 2010, downloadable applications will be available in the states where the U.S. Department of Health and Human Services is running the Pre-Existing Condition Insurance Plan. In those states, coverage will begin on August 1, 2010, if you apply by July 15, 2010. For states running their own programs, application details and coverage dates will vary. Please visit www.HealthCare.gov to find out when your program will begin and how to apply.

What does the new Pre-Existing Condition Insurance Plan cover? Thanks to the new Pre-Existing Condition Insurance Plan, premiums won't cost you more just because of your medical condition. The program will cover a broad range of health benefits, including primary and specialty care, hospital care, and prescription drugs; will include all covered benefits, even to treat a pre□existing condition; will not charge you a higher premium because of your medical condition; will not base eligibility on income.

Who is eligible? To be eligible for the Pre-Existing Condition Insurance Plan, applicants must:
Be a citizen or national of the United States or lawfully present in the United States.
Have been uninsured for at least the last six months.
Have had a problem getting insurance due to a pre-existing condition.
Applicants in most States will need a recent copy of a denial letter from private insurance companies as evidence of having a pre-existing condition.

How do I apply? Log on to www.HealthCare.gov to learn about the Pre-Existing Condition Insurance Plan in your state. If you live in a state where the U.S. Department of Health and Human Services is running your Pre-Existing Condition Insurance Plan, you will be linked directly to our application page. Or you can call 1-866-717-5826 (TTY 1-866-561-1604) for more information.

In order to apply in the states where the U.S. Department of Health and Human Services is running the Pre-Existing Condition Insurance Plan, you will need, at a minimum:
1. A completed and signed application form. 2. A copy of a letter from an insurance company or health plan showing that you have been completely denied individual coverage because of a pre□existing condition, or you were offered individual coverage but were denied certain benefits (for example, by a rider to an insurance policy) because of a pre□existing condition.

If you live in a state that is running its own program, www.HealthCare.gov will help connect you to information about how and where to apply in your state.

Check out www.HealthCare.gov for more information on the Pre□Existing Condition Insurance Plan, and other important health care resources that may be available to you.

WebMD Health Exchange

http://exchanges.webmd.com/transverse-myelitis-association

The WebMD Health Exchange is a place where you can get support and information for health issues from experts and other members who are dealing with ADEM, NMO, ON and TM via discussions, tips and resources sharing.

The TMA Member Exchange connects to WebMD's resources and is also

linked to information from our web site. The Exchange is searchable not only on WebMD, but on the Web, making it easier for users to find the TMA.

Another great feature of the Exchange is that we can include our support groups; each support group can create their own local or regional Exchange and each of these groups will be indexed in the WebMD searchable Exchange directory. By creating local Exchanges, it would make it easier for our members to find local resources. If you are interested in creating a local Exchange for your state or country support group, please get in touch with Jim and he can help you get started.

Caring for Children and Teens with Acute Disseminated Encephalomyelitis, Multiple Sclerosis, Neuromyelitis Optica, Optic Neuritis and Transverse Myelitis

In 2006, the National MS Society established a nationwide network of six Pediatric MS Centers of Excellence to provide diagnosis, comprehensive evaluation and care to children and teens under the age of 18 who have ADEM, MS, NMO, ON and TM. The centers were selected on the basis of having multidisciplinary teams of adult and child specialists; ties to an adult MS center; staff to evaluate and address school and other psycho-social issues; support for families; and the ability to work collaboratively with other institutions in the network. Approximately 60% of the children who are cared for at the pediatric MS centers have ADEM, NMO, ON or TM.

The centers are working together to:

- Improve evaluation and management strategies to enhance diagnosis and care of children with MS and other related disorders
- Develop resources for families, health care professionals and the public
- Collect data that will enable large scale research initiatives

Each Center Offers:

- The latest in comprehensive care and treatment for children with these central nervous system demyelinating disorders, as well as the information and support their families need.
- Evaluation and diagnosis involving both pediatric and adult neurologists
- A team of professionals that offers:
    - Nursing services
    - Cognitive and psychological evaluation
    - Rehabilitation assessment (physical, occupational, speech and language)
    - Vision care
    - Neuroimaging (MRI)
    - Individual case management and social services to ensure proper care and support
    - Information and resources for patients and families
    - School support

Families now have National MS Society-supported resources for evaluation, diagnosis, medical care and support. Children with symptoms suggestive of any CNS demyelinating disorder will be evaluated at one of the centers. A priority of this network is to provide comprehensive care to children with central nervous system demyelinating conditions, regardless of ability to pay. Financial assistance is also available for travel and accommodations according to need.

For information on the Pediatric MS Centers of Excellence or for programs and services available to your child and family call: 1-866-KIDS W MS (866-543-7967) or email: childhoodms@nmss.org.

Additional information can be found at: ***www.nationalMSsociety.org/pediatricms***

The Centers

Center for Pediatric-Onset Demyelinating Disease at the Children's Hospital of Alabama
University of Alabama at Birmingham
CHB 314K
1600 7th Ave South
Birmingham, AL 35233
Center director: Jayne Ness, MD, PhD
Contact person: Sarah M. Dowdy, MPH
Phone: (205) 996-7633
Web: www.uab.edu/cpodd/

UCSF Regional Pediatric MS Center
University of California, San Francisco
350 Parnassus Avenue, Suite 908
San Francisco, CA 94117
Project director: Emmanuelle Waubant, MD, PhD
Contact person: Janace Hart
Phone: (415) 353-3939
Web: www.ucsfhealth.org/pedsms

Partners Pediatric MS Center at the Massachusetts General Hospital for Children
Yawkey Center for Outpatient Care, Suite 6B
55 Fruit St.
Massachusetts General Hospital
Boston, MA 02114
Center director: Tanuja Chitnis, MD
Contact person: Rose Fratarcangeli
Phone: (617) 726-2664
Web: partnersmscenter.org/pediatric

Mayo Clinic Pediatric MS Center
Rochester, MN
200 1st St. SW
Rochester, MN 55905
Center directors: Nancy L. Kuntz, MD & Moses Rodriguez, MD
Contacts: Paula Freitag, MSW
Phone: (507) 538-2555 or (507) 284-2111
Web: www.mayoclinic.org/pediatric-center

Pediatric MS Center of the Jacobs Neurological Institute
State University of New York, Buffalo
219 Bryan St.
Buffalo, NY 14222
Center director: Bianca Weinstock-Guttman, MD
Contact person: Mary Karpinksi, MSW
Phone: (877) 878-7367
Email: PedMS@thejni.org
Web: www.pedms.com/

National Pediatric MS Center at Stony Brook University Hospital
Department of Neurology, HSC-T12-020
Stony Brook University
Stony Brook, NY 11784-8121
Center director: Lauren Krupp, MD
Contact person: Maria Milazzo, MS,CPNP
Phone: (631) 444-7802
Email: info@pediatricmscenter.org
Web: www.pediatricmscenter.org/

Children's Database

The Transverse Myelitis Association has initiated an important project to collect information for a pediatric/ young adult TM (recurrent TM)/NMO/ ADEM/ON data base. The information we are collecting will be used for the following purposes:

1. To develop a contact list that will be used by the TMA to notify and recruit families and older teens and young adults for the family camps and the older teen/young adult retreat opportunities;
2. To develop a contact list to recruit for pediatric studies and clinical trials related to TM/NMO/ADEM/ON; and
3. To develop a directory that can be used by TM/NMO/ADEM/ON families to share information and support between families in similar situations.

This project is being directed by Linda Malecky. Linda's daughter contracted TM at the age of two in 1999. To further the progress of the directory and to provide additional support, Suzie Wedlake, whose daughter contracted NMO in 2005 aged 4, has graciously offered to head up the directory project in Europe, Asia, Africa, and Australia.

If you have a child (25 years old or younger) with one of the rare neuroimmunologic disorders, we are requesting that you send us the following information:

- Parents' names
- Postal address
- Parent's phone
- Parent's email
- Name of child with TM/NMO/ ADEM/ON
- Diagnosis (TM, NMO, ADEM, ON, recurrent TM)
- Child's birth year
- Year child contracted TM/NMO/ ADEM/ON
- Age at onset
- Child's phone and email
- Birth year of brothers and sisters
- Medical facility where child's care given

The TMA is very aware of and sensitive about the short and long-term privacy concerns surrounding the information that we are requesting from you about you and your children, especially as it relates to a directory. We propose the following to address these concerns:

1. The information provided will not be incorporated in the TMA website in any way;
2. Your family will only be included in the directory at your request;
3. The directory will be published and mailed **only** to members who agree to be included in the directory;
4. Only the following information from the data base will be included in the directory:

- Parent's names
- State/Country where living
- Child's diagnosis
- Age (birth year) of child with TM/ NMO/ADEM/ON
- Parent's email
- Parent's phone

The TMA believes that it is extremely important for families (including the children with TM/NMO/ADEM/ ON) to be able to find other families and children for information and peer support, which is why we are collecting information for a directory. However, even with the limited information and distribution we are proposing for the directory, we realize that you or your children, now or in the future, may be concerned about being identified as someone with TM/NMO/ADEM/ON. We will only include those families who specifically indicate that they want to be included in a directory. ***Please provide the data base information regardless of whether you want to be included in the directory or not.*** This will ensure that you are contacted when camp or retreat opportunities arise or if there are studies or trials available that may help your child.

If you have ideas about additional information that we should be collecting for the database and/or including in the directory, please let us know.

If you would like to participate and you live in North or South America, please send your information to Linda Malecky via email: LAMALECKY@VERIZON.NET. If you do not have internet access, you can send Linda the information via the postal service: 107 Tweed Way, Harleysville, PA, 19438. If you live in Europe, Asia, Africa, or Australia, please contact Suzie Wedlake by e-mail at ***wsuziewms@aol.com*** or by postal service at 17 Vicarage Road, Penygraig RCT, South Wales CF40 1HR. The information from the different geographic areas will be combined before mailings are sent.

When you send us your information, please make it clear as to whether you would like to have your information listed in the pediatric TMA directory. ***Parental consent is required for children under the age of 18.***

If you have any questions or concerns about the project, feel free to call Linda (215-855-3488) or myself (614-766 -1806). Linda is hoping to have the first mailing completed by the end of February, 2011. If you sent her your information but do not receive a copy by then or if your information has changed, please contact Linda at ***LAMALECKY@VERIZON.NET***.

We believe that this project will help us better serve the families in our community by making you aware of important opportunities and by facilitating a support network for our families. We are grateful to Linda for her willingness to make this critically important project possible.

The James Timothy Lubin Fellowship in Rare Neuroimmunologic Disorders

In 2008, The Transverse Myelitis Association established the James Timothy Lubin Fellowship in Rare Neuroimmunologic Disorders. The purpose of the Fellowship is to encourage the development of medical specializations in TM, ADEM and NMO. We urge you to make a tax deductible donation to this critically important program by using the link: ***www.myelitis.org/fellowship-donation***

Jim has devoted the past twenty years of his life to helping others. To honor Jim's devotion to our community and to recognize his incredible contributions to people with the neuroimmunologic disorders and their families, The Transverse Myelitis Association has established the James Timothy Lubin Fellowship in Rare Neuroimmunologic Disorders. There is no greater need in our community than the provision of medical care by neurologists who have experience and expertise in these rare disorders. There is also a critical need to foster the development of scientists who are interested in these disorders. What better way to recognize and honor Jim than to establish a fellowship that will ultimately provide the best clinical care to the people Jim has devoted his life to helping and find the causes and cures for TM, NMO, ON and ADEM.

We are going to need your help to raise this money, and this help is going to need to be offered on a continuing basis in order to make this fellowship program a reality. The TMA is committed to an aggressive fundraising effort to create and maintain this fellowship program. More than any other program we have initiated, the James Timothy Lubin Fellowship in Rare Neuroimmunologic Disorders represents the most significant investment in all of our futures.

The purpose of the James Timothy Lubin Fellowship in Rare Neuroimmunologic Disorders is to encourage the development of medical specializations in TM, ADEM and NMO through a year of study under a leading TM, ADEM or NMO specialist. The fellowship is focused on the provision of exceptional clinical care and/or research into these rare neuroimmunologic disorders. Award of the Fellowship will be based on the expectation that the recipient will continue to specialize in ADEM, NMO and/or TM. If the fellowship includes a clinical and basic science research project, the fellowship term may be up to two full academic years.

The fellow will be required to work with a mentor (a TM, NMO and/or ADEM specialist). The mentor must be a faculty member with demonstrated clinical specialization and practice in at least one of the disorders. Preference will be given to medical centers of excellence in the disorders. If the fellowship includes a research program, the mentor must also be a scientist with research experience and publications in these rare disorders.

In order to award one fellowship each year, the TMA will need to raise $100,000. The number of fellowships we can offer will only be limited by the resources we are able to devote to this important program. Most of the people that I speak with for the first time are seeking a TM specialist or a NMO specialist or an ADEM specialist. If you have one of these disorders or if you are a family member or friend of a person with one of these disorders, an investment in this fellowship program will bring you very direct and profound benefits.

We urge you to get involved in this fundraising effort. I know that over the years many people have been inspired by Jim. Please join us in honoring Jim by helping to get this important program started. I can think of no greater legacy for Jim than to have highly motivated, brilliant and skilled physicians enter the discipline of neuroimmunology to provide clinical care to the people Jim has cared for so deeply for the past twenty years. Please make a donation to the TMA for the purpose of funding the James Timothy Lubin Fellowship and then please make your contributions a regular part of your generous giving. If you have been considering starting a fundraising program with your friends and family, this fellowship would be an excellent focus of your efforts. What more pressing or critical issue do you have in your own life or in your child's life than to assure that you or they have the best medical care available and that there are researchers who are interested in understanding TM, NMO, ADEM and ON.

Attracting physicians into research and clinical care focused on ADEM

How many of you with ADEM are being cared for by a physician who feels as though they have a good understanding of your disorder? How many of you with ADEM feel like you are receiving the best medical care for all of your symptoms? How many of you with ADEM are encouraged by the amount of medical research that is focused on your disorder?

To my knowledge, there is no specialist in ADEM anywhere in the world. To my knowledge, there is no researcher focused on trying to understand ADEM, nor how to treat an acute episode of ADEM, nor trying to understand how to most effectively treat the very challenging symptoms that result from an ADEM attack. There is no ADEM Center anywhere in the world.

The future of those impacted by ADEM depends on our ability to at-

tract clinicians and researchers into specializing in our disorder.

Spearheading our fight

Barbara Kreisler is the ADEM Support Group Leader of The Transverse Myelitis Association. She and I are committed to creating greater awareness of ADEM so that those who have ADEM can have a better chance for a good quality of life after an attack. The medical world needs to understand ADEM – Acute Disseminated Encephalomyelitis. Currently, they do not.

We are creating a James Lubin Fellowship focused on ADEM.

Over the years, I have been deeply moved by the people I've met who have ADEM. Some people got ADEM when they were a child, others as a young adult, still others when they were in their 40s, 50s or 60s. ADEM happens to both males and females and there doesn't seem to be any racial or ethnic group more prone to getting ADEM.

There are such compelling stories from people who've been affected by ADEM. Their experiences magnify just how little is understood about the brain. Their symptoms reflect these mysteries of the brain and the seemingly random medical events which befall those whose brains have gone awry from the inflammatory attack and damage to the brain and spinal cord.

Their Personal Stories

It seemed like overnight, I turned from able bodied to disabled. Suddenly, I couldn't read, write, remember my grandchildren's names, walk — and do lots of other things that define who I am. I was hospitalized. My condition confounded physicians. Physical and occupational therapy and sleep filled my days. I had to relearn the alphabet, how to write, read, how to manage the paralysis to my left side. Still no diagnosis. Then a neurologist recalled a case he had years ago. Finally, he gave my condition a name -- ADEM – Acute Disseminated Encephalomyelitis. Now, four years later, my balance is unpredictable so I walk with a cane. My handwriting isn't entirely legible, my computer skills are unreliable. I depend on my husband for everyday tasks. Mental and physical energy has diminished. Otherwise, there are no visible signs that I suffer from the effects of 13 brain lesions. I miss having the energy to write for hours, being able to drive a car, walk independently, the ability to concentrate. Nevertheless, I consider myself as one of the lucky ones who have ADEM. BK

Similarly stricken, others with ADEM are left comatose and paraplegic or quadriplegic or vent-dependent. Total recovery is elusive. ADEM is a devastating disorder.

Rachel still is unable to talk clearly enough to use a phone. However, we can understand her a bit more every day. From what I have read over the years, we believe that she had one of the worst cases of ADEM. Rachel came home from rehab three years in January, at which time she still could not get in and out of bed by herself or use the wheelchair; now, she does all that and even more. She will be starting exercise and writing classes at the local college, and will be getting fitted for a pallet prosthesis to see if we can improve her speech. It took us two years to find a speech therapist who understands this disease. She believes this lift will help. I don't have to tell you how much we searched for ideas and assistance in figuring out what will help Rachel get better. JE (Rachel's mother)

ADEM is a rare disease. ADEM was once considered a childhood disease. We now know that it strikes people of all ages – and in remarkably different ways. That's why it is so baffling.

Stephen was in the hospital for over three weeks; everyday getting worse. Everyday was a different diagnosis. First it was a brain tumor, and then it was West Nile, Autism, MS, Meningitis. He had CAT scans, spinal taps, blood tests, a urine test, EKG, EEG. The last spinal tap was positive for Lyme's Disease. He was scheduled to have surgery to administer his antibiotics at home. The health and neurology departments, however, had conflicting diagnoses. The health department thought it was Lyme's Disease, and neurology thought it was ADEM. Finally, his MRI showed over 20 lesions, but they said that there was nothing to do to treat them. So, together, we agreed to start him on steroids. By this time, he couldn't walk, talk, sit up, go to the bathroom, or swallow very well. The doctors said he might not ever do any of these things again. Within three days he started to eat, walk a little and make more sense when he talked. After weeks of therapy, hospital visits, and doctor visits, he was on the road to recovery. It is two years later, and Stephen is a happy kid, running on his own, laughing and loving school. He has speech therapy, physical therapy and occupational therapy, and he keeps shocking everyone with his progress. I truly believe he was a miracle. I read sad stories of people who are permanently disabled from ADEM and it is horrible to imagine what some go through. Sometimes I feel guilty that he did so well; that you would never know he was ever sick. Stephen's story is a happy one, and there is hope for people with ADEM to live a normal life. KD

An inflammatory attack in the brain leaves people with incredibly difficult symptoms. Finding the causes of ADEM is going to be a very challenging journey. The first step in this journey begins with the recruitment of physicians into this specialization. Without attracting physicians and researchers into this field, there will be no journey at all.

If you have ADEM or if you are a family member of a person with ADEM, we are going to need your help to take this very first step. And the help must come from you. It is exceedingly difficult to raise funds for very rare diseases. Most people support those causes that have directly impacted their lives. The general public is not going to come to our aid on this endeavor; help is going to have to come from you and from your family and friends; the people who best understand how ADEM has impacted your life and the lives of your families.

The James T. Lubin Fellowship in ADEM

Recently launched, the ADEM Fellowship was created to encourage the development of a medical specialization solely focusing on ADEM. The Fellowship will fund one or two academic years of study under a transverse myelitis specialist who has experience with ADEM. The fellowship will encompass both research and clinical care. The mentor will be a faculty member with demonstrated clinical specialization and practice in ADEM or transverse myelitis. Preference will be given to medical centers of excellence focused on transverse myelitis. If the fellowship includes a research program, the mentor must also be a scientist with research experience and publications in these rare disorders.

We need your help to fund the ADEM Fellowship

To award an ADEM Fellowship, we must raise $100,000 for each year. While our initial goal is $200,000, we are going to want to be able to attract a number of physicians and researchers into this discipline. If you have ADEM, or if you have a family member or friend who has ADEM, you understand how the investment in this fellowship program will bring very direct and profound benefits. We urge you to get involved in this fundraising effort.

If you have the ability to make a donation, we strongly encourage you to do so. Please send your contribution to Paula Lazzeri, Treasurer, The Transverse Myelitis Association, 10105 167th Place NE, Redmond WA, 98052-3125. Please be sure to indicate that your contribution is for the James T. Lubin Fellowship for ADEM. Your tax deductible contribution will be appropriately acknowledged.

Even if you don't have the ability to make a donation, you can make a difference. Please send us the names and addresses of friends and family members that we can include in a fundraising campaign. These are the people who have the greatest appreciation for why we need to raise money for the fellowship and for ADEM research. If you do not want to send us this contact information, we would be grateful if you could send the fundraising letter yourself. You can send me an email request, and I would be pleased to send you a copy of the letter for you to mail. We would also encourage you to seek donations from medical specialists, allied health care providers, and medical equipment providers that you have relied on and with whom you have developed relationships. Please also involve any organizations and clubs that you belong to; and please think about sponsoring your own fundraising activities to support the fellowship. We would be glad to help you with ideas.

No one else is going to do this for us. They have their own causes, and their cause is not ADEM. We have to do this for ourselves. Please make a donation if you can. Please consider sending us your contact lists. If you are reticent to do so, we urge you to send the fundraising letter yourself. Thank you for helping us to help you!

Barbara Kreisler and Sandy Siegel

Cody Unser Testifies at US Senate Hearing and Published in The Hill's Congress Blog

Cody Unser, founder of the First Step Foundation and member of The Transverse Myelitis Association, was invited to testify at a US Senate Hearing, called by Senator Tom Harkin, titled "The Promise of Human Embryonic Stem Cell Research," on September 16, 2010. On September 21, 2010, Cody was published in ***THE HILL's Congress Blog.*** Cody is a Public Health graduate student at George Washington University.

We are honored to have Cody's permission to reprint her published blog.

Embryonic stem cell research has become my hope

Words are everything in this world. What you can say is outranked most of the time by how you say it. Unfortunately, this is what has occurred to the promise of embryonic stem cell research. I'm not a scientist, a politician (just yet), nor a lawyer so I won't go into the logistics of the current court ruling halting this research. But what I will disclose is my story and why embryonic stem cells have profound meaning to me, along with millions of Americans, and why people like you should care.

Imagine waking up one morning to a paralyzed body. You can't move or feel your legs. How would you feel? What would go through your mind? Would you panic? Suddenly your life drastically changes. No more chasing your kids around the house, dancing with your wife body to body, eyes to eyes. What if you were a quadriplegic and breathing becomes something done by a machine? What about dressing yourself? What happens to your two-story house? Your car? Your job? Your life? When does it become bad enough for you to do something about it?

February 5, 1999 was when versions of these questions were mine to answer. I was a healthy 12-year-old kid who was very active and had big dreams. I was playing basketball at school and suddenly couldn't catch my breath and my head started bounding with sharp pain. The school I was attending called the ambulance and while lying down in the locker room, my left leg became numb and tingly. I picked it up, put it back down and I couldn't feel the floor.

I was scared out of my mind but I thought that whatever was wrong the doctors could fix. Transverse Myelitis is an autoimmune disorder in which the immune system attacks the spinal cord causing inflammation that damages the cells that control sensory and movement of the body. After staying in the hospital for a couple of months, I went to rehabilitation where I learned how to do everything from a wheelchair, all the while having dreams of my feet imprinting in the sand.

Sometimes hope is all we have to get us through life's hardships. Embryonic stem cell research is hope backed by real and evidence-based science. I have seen it and this has become my life because it is has become my hope. These cells have huge potential to improve how we treat and cure current diseases and those of the future.

Embryonic stem cells are cells that can become a variety of cell types in the body. All of life starts out this way, a cluster of cells that become specialized cell types, which form our tissues that make up who we are. Scientists have been able to use these cells where damaged ones traumatically affect how millions of Americans live life.

"Morality" and "life" are two words I constantly hear when the debate about this research comes up. We give thousands of parents in this country the chance to have the beautiful gift of their own biological children who otherwise would not with in-vitro fertilization. That's life and whether or not we agree with the morality of conceiving children outside "G-d's Plan," lives are created and therefore are given every opportunity to grow and succeed just like the rest of us.

However, because the process of in-vitro fertilization creates more embryos than are implanted, thousands of them are given a different fate. With full consent from the parents, these clusters of cells are frozen for later use, given up for adoption, donated to research or - as most of them are - thrown away. Where do our morals lie if we can easily throw these cells away but do not use them to treat and cure debilitating diseases? This research needs to continue with responsibility and oversight.

The political debate over this research is forcing many of our brilliant scientists to think twice about whether they should stay in this field. I know how dedicated and passionate they are about helping all of us find answers to our pain and suffering. If we keep dragging this debate back here to Washington, in Congress and in the courts, more and more scientists will have no choice but to either find a different research avenue or move to another country where they can pursue the promise that embryonic stem cells possess.

Christopher Reeve would have turned 58 on September 25th. One voice has power for he implemented huge changes by using literally all he had, his voice. If I could use mine, my message to the members of Congress is a simple one: If you are not affected by spinal cord injury, Parkinson's, Alzheimer's, diabetes, just to name a few, use your voice on behalf of millions of Americans who are, and who are counting on you. Please pass unambiguous legislation that will once and for all clear up the language of the limits and allowances of this important research.

We live our lives with such great fortune and in a way that allows us to live without even realizing we are. The moment I became paralyzed my quality of life was one of constant struggle. Anything can happen to any of us at any point in time. My hope is that the members of Congress don't need a reason to care about how vital this research is to move forward for future generations. The "Now" has been here, the waiting game is over.

BELIEVE . PROTECT . REACH OUT . SPEAK UP

In 1994, NFCA launched the first ever celebration of family caregiving declaring National Family Caregivers Week. Recognized by President Clinton when he signed the first proclamation in 1997, NFC Month has been proclaimed by an American President annually ever since. Many states, and dozens of local municipalities have proclaimed November, NFC Month.

Day in and day out, more than 65 million family caregivers in this country fulfill a vital role on the care team. No one else is in a better position to ensure continuity of care. Family caregivers are the most familiar with their care recipients' medicine regimen; they are the most knowledgeable about the treatment regimen; and they understand best the dietary and exercise regimen.

NFCA coordinates National Family Caregivers Month as a time to thank, support, educate and empower family caregivers. Celebrating Family Caregivers during NFC month enables all of us to: Raise awareness of family caregiver issues; Celebrate the efforts of family caregivers; Educate family caregivers about self-identification; In-

crease support for family caregivers.

The true strength of the American family finds its roots in an unwavering commitment to care for one another. **President Barack Obama, 2009's NFC Proclamation**

A Message from NFCA President and CEO Suzanne Mintz: *The theme for National Family Caregivers Month 2010 is "Reach Out for Help." National Family Caregivers Association encourages family caregivers to reach out for help all the time, but now the message has extra meaning and is particularly important. This year is the 10th anniversary of the National Family Caregivers Support Program; the very first piece of federal legislation to create a nation-wide program specifically to assist family caregivers. Under the auspices of the Administration on Aging, the program has helped over half a million family caregivers to date. Getting help begins with recognizing that you need it, then asking for it, and accepting it when it is offered. It isn't necessarily an easy thing to do, but it definitely is a really important one for family caregivers wanting a better quality of life for themselves and their loved ones. Another reason for family caregivers to reach out for help now more than ever is because we are all swimming in rough economic waters. Many programs that have existed at the state and community level have either been cut back or eliminated altogether. Family caregivers need volunteer help from their family, friends, and neighbors to ease the burden. The spirit of volunteerism is alive and well. Family caregivers can take advantage of this by reaching out for help with the day-to-day chores of life - making a meal, taking your car in for service, or just picking up a bottle of milk when needed.*

So celebrate National Family Caregivers Month 2010 by helping family caregivers reach out for help and being there to provide it when asked.

Contacting the TMA by Email

When writing email messages to the officers of the TMA or to support group leaders, please use TMA, Transverse Myelitis, TM, ADEM, NMO or ON in the subject header of the message. Please be sure to include a title in the subject header. The volume of emails that we receive and the way spam filters work makes it increasingly difficult to sort through emails to find legitimate messages. Also, if you would like to send an attachment, it is always a prudent approach to send an email notifying the person that you are going to follow up your message with a second email that includes the attachment; and explain the nature of the attachment. If you want to be sure that we see it, save it and open it, please include a subject header in your message and use words that will identify you as a person interested in contacting the TMA. We appreciate your help!

Important Reminder About The Transverse Myelitis Association Membership Directory

In order to receive a TMA membership directory, you must be willing to have your name and contact information listed. Those who have designated that they do not want to be listed in the directory will no longer receive one. The purpose of the directory is to assist our members in finding each other in their local communities, states and countries. As our membership is small and widely scattered around the globe, the directory serves as a way to facilitate the local or regional sharing of information and support. The value of this directory is commensurate with the numbers of our members who are willing to participate in our support network.

It is the expressed policy of the TMA not to share this information for any commercial purposes. The vast majority of our members are listed in the directory. This designation was made when you first completed the membership form on www.myelitis.org or when the original email or telephone contact with the Association was made. If you are not currently listed in the directory, and would like to change your designation so that you can receive the directory, please send an email to ssiegel@myelitis.org requesting that your contact information be listed.

This would also be a good time to check the directory to be sure that your current information is accurate. If your phone number or email address has changed, please notify us. Your membership information will be updated. When you send us any changes, please include all of your information so your membership listing can be easily found and the changes identified.

In addition to receiving the directory, another important benefit of being listed in the directory is having access to local support groups. Over the past several years, our local support groups have been developing around the country and around the world. If you are not listed in the membership directory, we assume that you do not want to be contacted. We do not provide your information to anyone, including the support group leaders who are currently operating in and around your area, or to those who will establish groups in your area in the future.

Due to the increasing size and cost of the TMA Membership Directory, we will be printing and mailing new directories no more frequently than every two years. If you are not currently listed, please consider doing so. We appreciate the willingness of so many of you to make yourselves available to assist others in your communities, states and countries.

Allen Rucker

We are so proud to have Allen as a regular contributor to the TMA Journal. Allen contracted TM in 1996 at the age of 51 and was paralyzed from the attack at the T-10 level. Allen published a memoir about his life after getting TM; "The Best Seat in the House." It is now available in paperback. As his memoir so brilliantly conveys, Allen is on a journey. That journey has taken Allen into a life as a speaker and an advocate for the transverse myelitis and disability communities. Through his many speaking engagements, his appearance on the Montel Williams Show, and as a contributing writer for ABILITY and New Mobility Magazines, Allen is raising awareness about transverse myelitis.

Allen Rucker has an MA in Communication from Stanford University, an MA in American Culture from the University of Michigan, and a BA in English from Washington University, St. Louis. He is the author or co-author of numerous books of humor and non-fiction. "The Sopranos Family Cookbook," one of three books he's written about the Sopranos, was a New York Times #1 bestseller.

As a TV writer-producer, he co-founded the experimental video group, TVTV, and has written numerous network specials, documentaries, and teleplays, including the award-winning cable series, "The History of White People in America," with Martin Mull. He is the recipient of the duPont-Columbia Journalism Award; two Writers Guild Awards, including one for career distinction as a writer with a disability; two CableACE Awards; and two retrospectives at the Paley Center for Media. He is a contributing editor to "New Mobility" magazine, the chair of the WGA Writers with Disabilities Committee, and a frequent public speaker. He lives in LA with his wife, Ann-Marie. They have two sons.

NO COUNTRY FOR OLD MEN IN WHEELCHAIRS
ALLEN RUCKER
12/27/10

I'm 65 years old but I keep being told by The Media that I'm not actually old at all. Betty White is 88 and look at her cavort with twenty-year-olds on SNL. Clint Eastwood is 80, for crying out loud, and he directs a new movie every three weeks. Even the governor-elect of California, Jerry Brown, is 72 and will take office on the 28th anniversary of the end of his ***last*** *term as governor. At 72, he's starting over! I mean, if those are America's role models, then any self-respecting 65-year-old should immediately burn his Medicare card and go clubbing. First you may have to learn what house, open-source, trance, and techno mean, but you're only 65, for pity sakes, so bone up, pop a can of Four Loko, and get to dancing until the break of dawn.*

Speaking strictly for myself, and maybe you, too, I'm 65 with an asterisk. I'm 65 in a wheelchair. That might sound like a prison sentence to some, but soon after becoming permanently paralyzed from TM at age 51 – a complete T-10 para -- I realized I had a leg up, so to speak, on my able-bodied contemporaries. While they were just fearing the distant prospect of old age, I was learning to experience it first hand. I was living the nightmare! I had a good 15-20 years head start on all the indignities of growing old.

Take just one item from the proverbial "Signs of Aging": incontinence. From the get-go, it wasn't, for me, just a periodic embarrassment like it is for those old guys you see in the TV commercials who have to go to the bathroom at the ballgame every five minutes. At 51, I was already at late-stage, full-blown, it-doesn't-get-more-incontinent-than-this incontinence, way beyond some magic pill to make my bladder stop twitching. I could tell those bozos in the ad exactly where they were heading, pill or no pill. They were heading, among other places, to the "Briefs and Undergarments" aisle at their local CVS. They too would soon be learning how to buy hygienic items and feign that they were for poor old Uncle Bert.

Another common symptom of old age: fatigue. Early on in my life with TM, I got tired just putting on my pants in the morning. I started to take daily afternoon naps, proudly, just like grandma used to do. My new idea of "late night" was the ten o'clock news. Which meant that I had less time, and inclination, to keep up with the world of pop culture. A Kardashian or "Snooki" from "Jersey Shore" could walk

in and sit on my lap and I wouldn't know who she was. Being out of touch – another sign of aging.

Plus, I began to be treated like an old codger. Waitresses started calling me "sweetheart" and "dearie" and asking repeatedly if I needed to use the restroom. I was at Costco recently, feasting on the free samples, when a nice lady at the sausage table looked at me and whispered, "Come behind the display, sweetie, I've got something special for you!" Old people are either fussed over like small children or completely ignored. G-d forbid you should try to do anything on your own. "Here, I'll get that banana that is right in front of your nose…oh, you shouldn't have to punch that automatic door button…let me!"

The truth is, you learn to adapt to this stuff pretty quickly and keep going. The way to counter fatigue, for instance, is not to stay at home all day, "resting"; it's to exercise, eat right, and sleep well. The way to deal with being treated like a child is to let it go. Just eat the sausage and roll on. In fact, there is a solution to almost every nuts-and-bolts problem of paralysis, from back pain to being socially marginalized. It just takes practice.

So a funny thing happened on the way to Social Security. As I got older and strove to master the ways of my condition, I started feeling and looking younger! Having done my long stints in the hospital, mostly because of infection, I had no interest in going back. Nor did I look forward to spending the rest of my days lying around, watching catfights on "The View." Because of TM, I became, in that hoary cliché, "pro-active" about my well-being long before a gerontologist ordered me to. In other words, I had to grow up, really grow up, before my time.

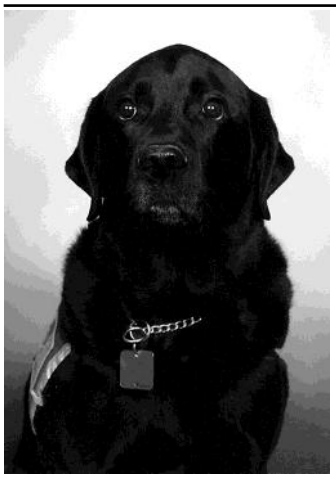
It's all about paying close attention. When someone sticks a gun in your face, it concentrates your mind. When something steals half of your body, same effect. With a serious disability, you always have one eye on your life and one eye on your health. It goes with the territory.

Now I'm at a stage in life where members of my peer group, as it were, many of whom never gave their long-term health more than trendy lip-service, are suddenly starting to break down. Back problems, acid-reflux problems, depression that they have self-medicated for years, and even worst, what the nutritionist Robert Pritiken dubbed the "diseases of affluence" – diabetes, hypertension, kidney stones, osteoarthritis, bowel problems – brought to you by a leisurely, pass-the-mayo, unexamined physical life. They are just heading into the Land of the Sick. I'm already there, waving to them from the shore.

It used to be that I'd go to a dinner party and be the only one who wanted to talk, at times incessantly, about health. Now I go and I'm the only one who doesn't want to carry on about blood thinners or mood enhancers or the do's and don'ts of the Paleo Diet. At the moment my friends are becoming more preoccupied with their biology, I'm feeling pretty good about myself, all things considered, and would rather turn the discussion to something fun, like the economy or that nutty Kim Jong Il. Of course, if I'm having too much fun, I down a third glass of Merlot, lean back, and whoosh – I'm staring at the ceiling.

I don't really know anyone Clint Eastwood's age (80) who lives an active, forward-looking life in a wheelchair. My guess is that they are out there, perhaps directing their own movies. On the other hand, I'm pretty sure my generation of chair users and other mobility-altered, at least the ones who heed the early-warning bell of a chronic disability and adapt to its demands, will not only speed right past the actuarial predictions of their demise – I'm not supposed to live past 70 – but also the term limits of many of their upright, cheeseburger-loving compatriots.

I guess the joke is, wheelchairs and all, we could very well be the last ones standing.



Kazu

We are so proud to have Kazu as a regular contributor to the TMA Journal. Kazu is a Canine Companions for Independece Service Dog. Kazu was matched with Pauline in August 2007 at the Northcentral Regional Center in Delaware, Ohio. This is the center where Pauline went through her intensive training with Kazu. Pauline has had Kazu in her life for about three and half years. Kazu does so much for Pauline. Kazu has totally changed her life. He has totally changed my life, as well. Kazu received a wonderful birthday card from Doug for his 5th birthday this past May. Doug raised Kazu for the first fourteen months of his life at the Ross Correctional Institution in southcentral Ohio. There is an amazing, wonderful and strong bond between Kazu and Doug. Kazu literally inhales all of the cards and letters he receives from Doug. As soon as Kazu received his birthday card, he wrote Doug the following letter.

May 18, 2010

Dear Doug:

Thank you for remembering my birthday! It was so great hearing from you and I loved the card. You are so thoughtful. Great photo of you and Rambo. I hope you are doing well and I hope that all is going well for you in school. I hope you are enjoying your classes. I'm sure you are providing Rambo with a great education. I'm sure your training will make it possible for him to help a person in the same way that my life is devoted to helping Pauline.

It is hard to believe that I'm five years old! Time really flies. It seems like just yesterday that I was with my Mom, Meeko, in California and all of my brothers and sisters. Then I had that scary flight in the dark from California to Ohio – my first plane ride. But the end of the trip was worth the adventure, because then I was handed over to you. We had such a great time together. I learned so much from you. I love you too, Doug. I think about you all the time. I just hope that you are doing okay.

By the way … do you think it's time for me to eat yet?

I have a great life with Pauline. She loves me so much and it is obvious to me and to everyone around her that I am the center of her life. I go everywhere with her. We are together almost 24 hours a day. And now that I'm also in bed with her all night, I can honestly say that we are almost inseparable. I do have my moments, though. There are nights when she can't sleep because of her nerve pain and she's restless. Or it is too hot around all of the covers for me. I sometimes just have to get out of bed and sleep in my crate or on my bed in the living room. I love when Pauline is petting me or rubbing my belly or scratching my ears, but it can get a little overwhelming at times, and I just need to get some sleep so I can be on my toes or paws in the morning when I have to go to work. It sometimes blows my mind that it took her over a year before I was asked to get into bed. What do you think that's about? I know that it sometimes hurts her feelings when I get down off of the bed during the night, but sometimes a guy just needs his space.

By the way … do you think it's time for me to eat yet?

I don't think we have any wild birthday plans for the evening. I've got a late day of work today because Pauline has a meeting until 7:00 tonight. She asked the maid to come pick me up after he was done with work so I wouldn't have to be at school so long. So, I expect that he'll come for me at about 6:00 and then we'll go for our walk up at the school. It has been raining on and off all day and it is nice and cool out. I love the cool weather because I'm a big black dog. I love my walks up at the school. We walk all around the school yard. Last night there were two geese that landed in front of the school and they were just walking around. They were absolutely fascinating and I couldn't take my eyes off of them. I actually walked into the maid a couple of times, because I became so distracted. I think he might have said 'no' a couple of times, but I'm not really sure because it's hard to take the maid too seriously. He is, afterall, the maid. I have to say that when I first moved in with Pauline, it was really difficult to figure out what his purpose was here. He didn't talk to me for over a month. I thought he had a personality disorder. And then I figured out that he lives in the house with us and he does whatever Pauline tells him to do. I thought I was well trained. He takes me for my walks before he leaves in the morning and after he comes home in the evening. I have no idea where he goes all day, but Pauline and I don't seem to mind too much that he's gone. If I'm feeling like I need some affection and Pauline is busy doing school work or is mesmerized by something Oprah is talking about, all I have to do

is walk between the maid's legs, and I get a great massage. I walk in head first and then slowly move through his legs. Then I turn around and come back through his legs the other way. I just go back and forth and back and forth, and I get a full body massage over and over again.

By the way … do you think it's time for me to eat yet?

I love walking around the school and the smells are just amazing. Every dog in the neighborhood is walked at the school. If I was allowed to mark my territory, I'd be a busy busy guy. But I put my nose down into the grass, the maid says, 'hurry' and I empty my bladder. I'm a machine. One of my favorite things to do in the whole world, is to walk down to the bottom of the path at the school. There are five dogs in one yard at the end of the path. I walk down there with the maid, and these dogs start yelping their brains out. Two of the dogs are really big, one is medium sized and two are pretty small, so their barking covers every octave … and they are really loud. I slow down my walk and encourage them into a fevered pitch and then I walk out into the grass right in front of them and go into my squat. I don't look at them at all. There just isn't much more satisfying about life than taking a crap in front of those five yapping dogs running back and forth against the fence in their yard. And then the maid picks my crap up in a plastic bag and takes it home with him. He drops the bag into a large green storage container. Do you think he's saving my crap for some reason? I could devote my entire life trying to understand this very curious creature, if I had nothing else to think about.

By the way … do you think it's time for me to eat yet?

The other thing the maid does, he throws the Frisbee for me. I love chasing after the Frisbee in the backyard when he throws it. When Pauline throws it, it lands in the bushes or it goes behind her and all she does is laugh hysterically while I'm waiting to take off. I have way too much dignity to go chasing off into the bushes. Ya gotta love her. After watching her throw that Frisbee it makes me wonder whether she had a childhood.

Anyway, I'm pretty sure there's not going to be any big birthday celebrations, but I have a sneaking suspicion that I'm going to get a whopper of a birthday present tonight after Pauline comes home from school. When the maid came into the house last night after work, he came in with all kinds of bags. There were bags from the hardware store, from the grocery store and from the pet store. My nose attached itself to the pet store bag as soon as he came in the door. My brain went totally numb and my stomach went into high gear. The smell was completely intoxicating – and the bag looked really heavy (I'm glad he didn't ask me to carry it). I just don't get stuff like that from Pauline. She handles these treats like they are family heirlooms. They do seem pretty neurotic about what I eat and my weight. So, tonight could be one of those very rare occasions when I get to eat something really tasty. I'm just hoping I can chew on this thing until my gums bleed.

We're still in school, but it is getting to be the end of the school year. I'm looking forward to summer vacation as much as Pauline. The summers are just great. The maid gets up at 5:00 and takes me for my walk. Pauline wakes up after we come in and wipes off my paws while the maid is getting me water. Then Pauline feeds me my breakfast. The maid leaves the house and Pauline and I get back into bed for most of the morning. When she gets up, we go into the backyard and while Pauline is drinking her coffee and swinging on the patio chair, I get to smell around in the garden and the grass. I can just inhale the backyard for hours. A rabbit built a nest in one of the gardens about a month ago, and there are babies in the nest. The maid won't let me walk into the garden, but I can hear and smell the babies in there, and I'll stand at the boundary of that garden checking them out. There are all kinds of birds and small critters all around the backyard. And the backyard faces, south, so if I want to lie in the sun, there's plenty of sun, and if I want shade, there are trees all around. The summers are just very quiet and peaceful. I do have my chores, but I just love helping Pauline, so I don't mind at all. In fact, I am happiest and my tail wags the quickest when I am responding to one of Pauline's commands. I spend a lot of time opening and closing doors, picking things up for her, and carrying things around the house. That's what I do the most. We don't do laundry every day, but when we do it, I'm still emptying the dryer and pulling the wagon of clothes into the living room for Pauline to fold. Another thing she has me do is drag all of the throw rugs out the back door so that she can shake them out. Dragging those long and heavy rugs out the back door is really hard work. I've gotten so good at it, I sort of amaze myself. Pauline is always calling me a rock star. I am good. I'm also always hungry.

The only time it gets really loud in the house is when Pauline is watching one of her movies on the gigantic high definition television with the volume cranked up. I'm beginning to think that she's slightly hard of hearing. She loves to watch these movies where a gladiator or some samurai warrior is getting his head cut off with a sword. I'm not sure I understand what she gets out of these movies, but I figure it isn't for me to judge. She doesn't make judgments about places I like to smell, I don't make judgments about her taste in movies. She doesn't watch this kind of stuff after the maid comes home, because he told Pauline that he would prefer not to be watching people having swords poked through their eyeballs or heads rolling down hills into large piles while he is eating dinner at the kitchen table. The dynamic between these two could be a book, and I would write one if I had fingers. Suffice it to say; when the maid gets home, the volume gets turned down on Pauline's television. I can't say I mind; after all I'm a dog and my hearing is dog-like.

By the way … do you think it's time for me to eat yet?

For a couple of old people, Pauline and the maid sure have a lot going on. A few weeks ago, they took me for a ride out to the country to see this great camp. There is a Hole in the Wall Gang Camp being built about 30 miles north of Columbus. This is going to be like the camp we go to in North Carolina – Victory Junction Gang Camp. We picked up Kat and we went to the camp for a tour. Pauline rode in a golf cart and the maid and Kat walked me around the camp. They are still building it, so all of the buildings were still under

construction. And there are a lot of woods all around the camp. I would have considered it a very enjoyable walk and great exercise, if we weren't traipsing around in the middle of a Saturday afternoon while I'm usually taking my nap. The maid did manage to empty my food into a bowl at 4:00 and they fed me outside at the farm … I do have to say that I love a picnic. The maid did take me for another walk after I ate to see if I would do my business at the camp. Not a chance – I made him take me again when we got home. Like I'm going to miss an opportunity to crap in front of the five yelping hounds at the school?

Wow, all that talk about my dinner; do you think it's time for me to eat yet?

Then last weekend, we went to a wedding out at the farm. Pauline's sister, Julie's son, Justin, got married outside at the farm. He and Amiee got married in the late afternoon and then Julie served a fantastic farm dinner … at least it smelled fantastic. I lay in the grass all afternoon – during the service, during the dinner – all afternoon and evening. The sun was out, it was cool; it was just a perfect day. I just love the farm. The smells are to die for – all of the wild animals, and the horses and the cattle. The horses usually come down by the house, so I get to watch them, even though they are on the other side of the fence. They are big, majestic, beautiful animals and totally fascinating to watch. They eat the grass and other plants – just can't get my head around what they are eating. Again, hard to account for people's and horses' tastes. The maid took me for a couple of really long walks which was great. The farm is such a special place. Pauline told me that their first dog was buried out at the farm. She showed me the place she is buried – Pauline and the maid planted a weeping cherry tree on her grave site. This dog's name was Sandy. Very confusing. I've heard Pauline call the maid, Sandy. Makes me very happy that they don't call me, the maid. My name is Kazu. It's bad enough that they've butchered the Japanese pronunciation of the word 'Kazu.' The Japanese family that takes care of my Mom probably should have sent me out to the Midwest with a cassette tape of them saying my name. Canine Companions for Independence spent $40,000 to train me to look into a person's eyes when I hear my name. The next thing that happens is that I get a command. Every time I hear them say my name, the thought goes through my head that I should look up at them and roll my eyeballs. But what would be the point … so I'm Kazu with the accent on the wrong syllable and the wrong vowel sounds. I hear my name pronounced incorrectly 400 times a day; it blows my mind. I suppose I should be happy that they haven't butchered the consonants.

By the way … do you think it's time for me to eat yet?

I love being out at the farm, and I also love being with family. I must be the most lovable big black dog on the face of the earth. After the wedding service, Justin took me under the flowers where the ceremony was held and had everyone take photographs of him squatting down next to me. When we're around family, Pauline tells everyone to stay away from me until she is either sitting down and not needing me or family members lose control over themselves. From what I've observed over the past three years, Pauline's family and the maid's family have developed 'losing control over themselves' into an art form. It's hard to say what sensation they are experiencing while they're petting me, but they sure don't seem like they can get enough of it. Hey, it's pleasant enough for me, so I'm not complaining. I have to say that my favorite toy in the whole world is David. I was just born to be chasing after David. One of the most exciting experiences I have, next to watching my food poured into the bowl, is watching David walk in through our front door. Whenever I see David, I just want to go tearing through the house like a wild banshee, whatever a banshee might be.

I have a great life, Doug. I spend all day long with Pauline and now I'm with her all night long, as well. I help her whenever she asks, and I sometimes help her even when she doesn't ask – I've been known to pick things up for her when they're dropped, before she's even had a chance to ask me to pick it up. And besides all of the helpful things I do for her, we've really become best friends. I love my time with her. She makes me feel so important – like her life wouldn't be nearly as good, if I wasn't there. I can definitely tell that the help I give her makes her life much easier. I've thought many times, with as much as the maid is gone, it's a good thing that I'm around to get things for Pauline or to help her get around in the house or in the school or outside. I'm carrying things around the house or the school or the yard for her while she has hold of her canes. And our companionship is something really special. Just spending time with Pauline is one of my favorite things to do. I love when she is petting me or rubbing my tummy or scratching me – but I also just enjoy lying next to her. I just couldn't have a better friend. I sometimes look up into her smiling face while she is looking down at me and I think to myself, life just doesn't get any better than this.

I would love for every single day to be exactly the same with the same activities built around my two meals. So, why is it that I'm only getting fed twice a day?

By the way … do you think it's time for me to eat yet?

Unfortunately, Pauline and maid think they need to go places. I have to admit that traveling is a bit stressful for me, but if you watch the maid prepare for one of these trips, you'd think he was getting ready to build the Panama Canal. Hey, man, we're just flying to Dallas; there's no need for me to miss a meal. That's just totally insane. I have to become dehydrated because he's freaking out about traveling? Hey, when's the last time you saw me pee on a plane – give me something to drink. We've been doing this travel gig for three years. He's either going to have to get a handle on this and lighten up or one of us is going to need to start taking medication for these journeys.

Hanni's sister is getting married in June and they are going to have the wedding on the beach in Puerto Rico. We all love Abbey and Peter, so making the trip down there was a no-brainer. And with Kat and David and Hanni and Aaron going down there, it will also be a great vacation. Next to chocolate, there's little else Pauline loves more than the beach and ocean in the Caribbean. So, the plan is for us to spend almost a week down there. Fortunately for me, the maid's idea of a beach vacation is a walk on the beach at 5:00 in the morning and then again at 8:00 at night. For all of his irritating neuroses, I'm grateful that he doesn't like being out in the sun. Hey, I'm a big black dog. While Pauline is enjoying the ocean and the surf, the maid and I will be parked in some air conditioned room or under an umbrella catching cool ocean breezes. The crew has been talking about making a trip to the Puerto Rican rain forest. I do what I'm told, but I'm trying to get my head around this rain forest adventure. How is Pauline going to get around in the rain forest and is Pauline going to demand that the maid spray for all of the snakes and spiders in the rainforest before she enters it. I'll have to be working really hard to pay attention and focus on Pauline during this trip, because the sheer entertainment surrounding this rainforest excursion could get to be mighty distracting.

The travel down to Puerto Rico is also an interesting proposition. Pauline needs to take me for an appointment with Dr. Riggs within two weeks of our trip. That is the requirement to bring a dog into the country. We have an 8 hour trip going down and the same coming back and we go through Atlanta. I'm going to be fine, but the maid is going to make Pauline and I into nervous wrecks before we check the luggage. If all goes well, they'll put Pauline and I in the bulkhead and move the maid somewhere back in the plane where he can fret about a hundred different things without our knowing about it. My travel concerns are fairly simple – pack all of my food in a carryon bag in the event the airline loses the luggage.

By the way … do you think it's time for me to eat yet?

At the end of September, we're going to Dallas. We've been to Dallas before. Let's see, what do I remember about Dallas? Oh, yeah, 104 degrees in the shade, bull riding and throwing me into a swimming pool. This trip to Dallas should be much easier – we're going to be at a symposium, so I'll be inside a hotel for three days. I know how these things go, because I attended the symposium in Seattle. The maid goes down to the meetings at 7:00 in the morning after I've been walked and fed. I stay in the hotel room with Pauline until she wakes up and gets herself ready for the meetings. We usually make a grand entry just before lunch. No muss, no fuss. I can handle the symposium schedule. It's irritating to miss my afternoon nap, but I spend the entire afternoon lying on the carpet listening to doctors talk about stuff that no one understands. Give me Oprah. The nice thing about travel to Dallas is that it is a direct flight and the maid always gets us on larger planes that have a sizeable bulkhead. After all, I am a big black dog.

By the way … do you think it's time for me to eat yet?

A few weeks after the symposium, we're going to North Carolina for the retreat weekend at Victory Junction Gang Camp. The maid is all freaked out about not getting direct flights to Greensboro and there only being very small planes that go into this airport. So, he's talking about driving down there. It is a long drive. There are definitely downsides to this drive. Pauline wants to get into the back seat with me so she can put her feet up on the seat and spread out her legs. I understand; she has terrible nerve pain. The only thing worse than delaying my eating is interfering with my sleeping. It's hard enough to sleep when bouncing around on the seat – having to accommodate Pauline's legs is just more than I can deal with. So, by the time we get to the camp, Pauline will be irritated as can be and I will be totally exhausted. The up side to driving to camp is that my meals will be given to me on time, the maid doesn't try to dehydrate me, and I get walked on a regular basis.

I love camp – I love everything about camp. I love being with the kids, it is a totally fun place, and I'm the center of attention. David and Kat are applying to come as volunteers, so if they are able to come, it will be even more fun. I have a lot of work to do there to help Pauline, but she's so happy at camp, it makes my job easy for me. I love it when she's happy. There's just so much fun stuff going on and they dance after meals. And Dr. Kerr is going to be there. Speaking of out of control around me, Dr. Kerr is one of the most out of control people around. Fortunately, I really love Dr. Kerr, so I love his attention and affection. The camp is going to be in October, so the weather down in North Carolina should be really great. It will be a very fun weekend. And we have so many friends down there

and so many friends who come to camp; it will be a very special weekend.

Pauline told me that we are coming to see you on Monday, June 28th. I would tell you how excited I am to see you, if I could talk. Fact is, I can't even bark without getting the speak command. And if Pauline doesn't give me the speak command often enough, I even forget how to bark. It is really quite mortifying. I'm a highly trained service dog that has been bred for my intellect; how can I possibly forget how to do something that the five yapping dogs in the backyard at the school are doing in the most incredibly thoughtless way.

I look forward to our visit with you all year long. It will be so great to see you and the other guys and it will be wonderful to see Denice. I hope to maintain some semblance of control when I see you, but I can't make any promises. I have some really crappy role models around here when it comes to exercising self control. Okay, I'm not even going to try; I'll likely go absolutely bonkers when I see you. Life is short; even an exceptionally well trained service dog needs to be able to express sheer joy.

By the way … do you think it's time for me to eat yet?

I love you, Doug. I think about you all the time and I hope that you are doing well. Please know that I use all of the great training you gave me every single day in helping Pauline. Also please know that I have a great life, that I am very happy, and that I have a maid.

With Great Love,

Kazu

P.S. I got a bone for my birthday – a really big bone; an edible nylabone – the operative concept being edible. After Pauline came home from her school meeting, she brought out this large package that contained the largest bone I have ever seen. She handed it to me, and I immediately took it into my crate to eat. It was 7:15. Pauline told the maid that she didn't think that I was much of a chewer, and that I might not finish the whole thing in one session. I spent the first five minutes licking the bone. Very delicate roast beef flavor. I could have licked it all night long, but my chew instinct kicked in, and before I knew it, I had that bone upright between my paws and my molars were on it. By 7:45, it was history … burp.

In Their Own Words

In each issue of the Journal, we will bring you a column that presents the experiences of our members. Their stories are presented ***In Their Own Words*** by way of letters they have sent us. We are most appreciative of their willingness to share their very personal stories. It is our hope that through the sharing of these experiences, we will all learn something about each other and about ourselves. It is our hope that the stories will help us all realize that we are not alone. It is important to bear in mind that all newsletters and journals are archived on our web site. Should someone do an internet search of your name, your article is likely to be identified in their search results. You may submit your stories by sending them either by e-mail or through the postal service to Sandy Siegel. Please be sure to clearly state that The Transverse Myelitis Association has your permission to publish your article.

Experimental Nerve Transfer

Our son Anthony was diagnosed September of 2008 at the age of 16 months with Transverse Meylitis at St. Joseph's Hospital in Paterson, NJ. He had an ear infection and congestion in his chest and was placed on a nebulizer. Shortly after that he lost full function of his right arm and shoulder. He was hospitalized and received intravenous steroid treatment and regained slight movement in his hand, however the rest of his right arm was paralyzed. He was so young and scared and he had no idea what was going on. The first hospital we went to had misdiagnosed him with a dislocated elbow and now we know how important it is to be educated by researching everything.

After being diagnosed we researched his condition and found that Johns Hopkins had a Transverse Myelitis Center. We quickly emailed Dr. Benjamin Greenberg, Co-director Johns Hopkins Transverse Myelitis Center and he stated he wanted to meet with us as soon as possible. We met with him in November of 2008. He had examined our son and was explaining the diagnosis and that we needed to wait and see if any nerve function would return which can take up to six months. In December of 2008, we were called by Dr. Benjamin Greenberg who stated he was moving to Texas, however he was discussing my son's condition during lunch with other physicians and they wanted us to come down and consider a Nerve Transfer Surgery. So we went and met with Neurosurgeon Dr. Allan Belzberg to discuss what the surgery would entail. Before we proceeded, he had requested other tests to make sure he was a candidate. We also met with Dr. Douglas Kerr, Associate Professor, Neurology and Transverse Myelitis Center Director, who was suggested by Dr. Allen Belzberg to get his opinion about the surgery.

On February 24, 2009 at the age of 22 months, Anthony was the first TM patient to receive the experimental nerve transfer surgery.

The surgery went well and six months after the surgery he was able to bend his arm, shrug his shoulder and regained additional movement in his right hand. The nerve rerouted to his bicep took really well, however the nerve to his deltoid still has no function to date. Anthony's surgery was different than the six year old boy who underwent the same surgery (published in Vol. 9 Issue 2 of the TMA Newsletter). In Anthony's case, the neurosurgeon took the nerves from the same arm that was mostly disabled and rerouted them. So the nerves were not working at full capacity and there was a huge chance he would not regain feeling or movement. We didn't want to wait because after six months the chances of regaining significant function were poor, so we decided on the experimental surgery. We wanted to give our son a chance.

Today at the age of three, he can swing a bat, climb a rock wall, and ride a bike. It is hard to believe he had a non-functional extremity just two years ago.

He is full of life and still keeps up with his seven year old brother. We encourage him to use his "good hand" when he does things. He is always telling me he can do it. His spirit keeps us going.

His pre-school provides him with school-based occupational and physical therapy and he also gets medical based therapy at a facility. Anthony visits the Kennedy Krieger Institute semiannually. He still has a long road ahead of him.

We are so glad he did regain some function and we thank the doctors at Johns Hopkins for coming up with this surgery for our son and others. We would also like to thank Kennedy Krieger Institute for their support and kindness.

We wanted to share our story.
Anna and Brendan McNerney

Learning How to Walk Again

Kimberly Borchert

It has been about 18 months since the most wonderful and the most awful events in my life took place. I'm writing about it here for a few reasons. It is cathartic to write. It's important to share experiences and read about them as well. It helps us to grow, to learn, and to feel less like we are all alone in the world with this illness. Another

important reason was to share information with doctors about experiences directly related to how they sometimes react and treat patients who come down with these sudden disturbing symptoms. I have learned through reading and through my support group that we share common experiences.

My story begins in April 2009. Chris, my husband of eight years and I were about to welcome our first, long awaited daughter. I arrived at the birthing center but soon was sent to the hospital because my temperature was too high. I was devastated that my birth plan began to unravel. My plan was to go all natural, in a tub of water, unmedicated. A lifetime of bad experiences involving doctors and nurses initially led me to the birthing center.

As a child I experienced horrible headaches that sent me to the emergency room. I would cry and scream for hours waiting alone in the hospital room for the doctor. The pain would get so bad that I would begin rhythmically banging my head against the wall screaming for someone to help me. Eventually a doctor would come but it took several hours from the beginning of the pain until I was treated for it. Needless to say, I had bad memories of the hospital and of doctors. I didn't want to have to think about that while experiencing the happy event of my daughter's birth. Now my plan was shattered. I had to suppress my shock and sadness over this sudden change in the agenda. I resigned myself and put my health in the hands of doctors and nurses as opposed to midwives.

At the hospital, I was given an epidural twice. The first one didn't work. Not very long after I was given an epidural that worked, my nurse and the midwife who followed me from the birthing center informed me that my daughter could not survive normal labor. Her heart beat was erratic with each contraction. They told me I had to have an emergency c-section. To say that I was devastated would be a considerable understatement. I had feared even before becoming pregnant with this planned pregnancy that I would have to have a c-section. I began crying uncontrollably. I told my husband that I would die of a pulmonary embolism if I had a c-section. Chris tried to console and to reassure me I wouldn't die. The fear remained and I couldn't stop crying.

As I cried and nurses crowded my room, I was given the proper anesthesia. Almost immediately I was being rolled down the hall in my hospital bed. All of a sudden I couldn't move. The panic that overwhelmed me took my breath away. I felt like I was just a head. This unnerving sensation especially in light of the fact I was not informed it was going to happen caused a feeling of such desperation I couldn't talk. I feared I would choke and be unable to breathe if I gave into it. I pushed everything down and experienced a kind of detachment. Regrettably the fact I was finally going to have my daughter in my arms was something I pushed far back in my mind. This I know for certain would not have happened if I had given birth in the tranquil quiet surroundings, with kind considerate people who still saw me as a person and not just another number, a person passing through.

Chris in surgical scrubs appeared beside me at the operating table. My useless arms were spread out in a way that reminded me of Christ on the Cross. Suddenly Chris said she was here, she was born, Angelea. A doctor or nurse had her in one of those bassinettes examining her. He said she was very pink. My husband asked me if I wanted him to stay or if I wanted him to go with the baby. I told him to go with the baby. I regretted it almost immediately after he left. I began to feel some pain in my chest. I told the anesthesiologist. His demeanor told me I had annoyed him with this information. He quickly told me it was normal.

I was suddenly conscious again being rolled in the bed down the hallway to the hosptial room. My jaw was chattering, crushing my teeth together uncontrollably. I was upset by this symptom and cried. When I was acknowledged, a nurse in an annoyed fashion said this was a symptom resulting from the anesthesia leaving the body. I thought surely every single one of my teeth would be chipped after this. I was given strangely gruff treatment from the doctors and the nurses. I ignored the treatment and anticipated being released.

Chris and I dreamt about having children. We had put off having children so I could finish my degree in social work and also to get into a more agreeable financial situation. I had a miscarriage two years before finally getting pregnant with Angelea. We were unsure whether or not we could get pregnant. It was a time that was hard to live through. At the time I was working for the Department of Children, Youth and Families as a social worker. I was helping children and families and through them, society at large. It was a hard but rewarding job. I had to quit my job as a result of emotional issues surrounding the miscarriage. My goal had been to go back to work as a social worker at some point.

As soon as I stopped shaking, I was able to hold my daughter for the first time. It was unbelievable to hold that little body that had been inside my body for so long. She was truly a little angel and a lucky one I might add. I had a terrible uterus infection. The doctor who opened me up explained that she had never seen anything like it before. Infants born in this situation were usually very ill and ended up in the NICU. Miraculously, Angelea was one hundred percent healthy and I was the one who was ill, the ideal situation under the circumstances.

The next few days in the hospital were filled with frustrating sessions with lactation consultants trying to teach me and my daughter how to breast feed; holding my sleeping daughter; barely handling it when the neonatal nurses took my daughter out of the room for check-ups at too frequent intervals; pumping milk from my breasts with a clumsy machine for 25 or more minutes at a time several times a day; having family members arrive to welcome my daughter into the world; walking with my husband as we wheeled our daughter in her bassinette through the hallways of the hospital; trying to breast feed my daughter which wasn't working; and getting my vitals taken at all hours of the night. Chris and I looked forward to the day we could go home and be with our new baby in peace and comfort.

Four days after my surgery, I came back to my room after a walk and felt unusually out of breathe. I told my nurse I was having trouble breathing and I conveyed my fear of a pulmonary embolism to the nurse. She laughed at me before she dismissed my fear. She told me I would be in really bad shape if I had a pulmonary embolism. She must have told someone about this issue because the next morning, very early, a doctor came in and informed me I would be given a test to determine if I had a pulmonary embolism. She kind of chuckled and said I probably didn't have one but it wouldn't hurt to check; after all I had good insurance.

I came back from the test. Immediately nurses made themselves busy in my room, checking my vitals and hooking another pack of some substance on the IV pole. A doctor came in and informed me I had a pulmonary embolism and I would be receiving blood thinners. I was stunned and a little frightened. Over the next week, my husband and I were sequestered to the hospital. Nurses administered blood thinning shots into my arm. One day another nurse decided to put the medicine into my stomach. I watched her with the needle. I studied the needle. It appeared to be bent. I mentioned it to the nurse who immediately dismissed my claim. Each shot she gave me felt so unusually painful given the small size of the needle. Later on we found out she and other nurses were in fact bending the needles. Bent needles hurt a lot worse than straight needles.

Life for the time being included: mothering my daughter; working hard to breastfeed her; getting little sleep because of having to pump my breasts; being interrupted by vital-signs-taking-personnel; enduring the administration of shots with bent needles; watching my poor husband lose sleep because of the lack of comfort at night on the little couch/seat in the room; overcoming the period of time my daughter had to have her frenulum (tissue under the tongue) snipped because she was tongue tied which prevented her from breastfeeding correctly and the aftermath of not being able to have her in my room for a couple days which made it more challenging to try to breastfeed her; enduring tests to check my lungs which caused a feeling similar to suffocation; MRI's of my legs; CT scans; stressed out nurses who treated me rudely; enduring phlebotomists' attempts to draw blood from my veins after I informed them that I was a hard stick and that was why the nurses on the unit couldn't do it; thanking G-d when they put a PIC line in which was basically a tube like an IV inserted into my chest so nurses could easily draw blood from it saving me from being a human pincushion; waiting every day for the time a nurse would come into my room and tell us it was time to go home because my blood levels were where they should be.

After the hospital social worker set up home health services, we were informed we would be going home. My husband and I began packing things up. The nurse came into our room and began the discharge process. I sat on the couch with her. Abruptly, I began to feel ill. My legs and my back started hurting. I went to my bed to lie down. Pain erupted in my upper thigh in the groin area. It was a type of pain I never felt before. The doctor involved with my c-section came in and saw I was in pain. She told me it was a muscle spasm. The nurse finished the discharge papers. The pain began to get worse shooting down my leg and wrapping itself around my back. My husband and the nurse had to pivot me into the wheelchair from the bed. My legs were becoming weak. I wondered to myself if that was normal.

Before I knew it, the nurse was wheeling me down the hallway at a very fast pace. I thought the wheels were literally going to spin off. My husband was at the car piling everything in and strapping the car seat with my daughter inside in the backseat. My husband had to pivot me from the wheelchair to the car. I could still bear a little weight. The nurse was gone before we finished getting into the car.

The whole day my husband had to help me to the restroom. I spent most of the time in bed. Something was very wrong. He reminded me that the home health nurse was arriving at the house the next morning. Meanwhile, the pain started to become excruciating. I cried. There was the sensation of burning, electrical shocks, the feeling of fire ants burning as they crawled up my leg, the feeling of having spilt ice cold water on my lap, and a feeling of a tight Ace bandage wrapped around my legs.

Nothing took the pain away. We tried ibprophen. When that didn't work we tried the prescribed Vicodin. That also had no effect. The next morning when the home health care nurse arrived, we told her I couldn't walk and I was hav-

ing a lot of pain in my legs and back. My legs hurt to the touch and my pants felt like burning sandpaper rubbing against my skin. Another symptom was bladder issues. The home health nurse said it was up to us if we felt we needed to go to the emergency room. Chris' response was to say something to the affect of: "We just got home from the hospital. I don't want to go back. Do you? We can go if you want to." Of course, my response was to wait. I hated the idea of having to leave my baby. I hoped it would get better. The pain which was already at an intolerable level simply increased and I couldn't bear the slightest amount of weight.

Soon I came to the realization that my sudden loss of my ability to walk warranted emergency care. Chris and I with the baby went to the hospital a couple days after being discharged. The emergency room doctors focused on the fact I had a PE several days earlier. They gave me pain medicines and then sent me for tests to find out if I had blood clots in my legs. The doctor told me I didn't have any clots and sent us home. The pain medicine prescribed had no effect.

We went back to the hospital a day or two later. At that point, it was clear the pain I was experiencing and the burden of not being able to walk was not letting up and forced us to go back for another opinion. This time they admitted me. I was given multiple tests in the hospital. One test was an MRI. It took the medical personnel three attempts to complete the test because I couldn't straighten out my leg without screaming in agony. On the third attempt I had to be completely sedated.

The doctors couldn't tell me what I had for the first few days. Meanwhile, I fell multiple times in the hospital. Being away from my home and my newborn was beyond depressing. At some point, I believe I started to detach myself mentally from the situation. All the while, I futilely believed the doctors would be able to give me a quick procedure and correct the problem immediately.

One day the doctors informed me that I had iliac hematomas. They explained I had internal bleeding which was compressing the nerves of my spine. The c-section doctor commented to the head doctor that I needed to be on nerve pain medicine, Gabapentin. Once they started giving me that medicine I had a substantial decrease in pain. I vegetated in my hospital bed. I informed the doctors I had bladder issues. I also felt like my bladder was going to explode and asked for a catheter. I was finally given one after a machine revealed my bladder was full. I appreciated the catheter. Apparently the fact I had a catheter had infuriated the nurse who came barreling into my room. She had my catheter removed despite my wishes. In the hospital when you are not in the presence of family members, something strange happens. Nurses treat you in a rude callous manner. The same frowning, ill mannered nurse would become a smiling polite angel when a family member was present in the room. I always considered myself a strong willed person; however, I felt utterly powerless and dejected.

I began to have trouble breathing. I would begin to relax when suddenly I would gasp for air. I was given oxygen. I started to fear not being able to breathe. This anxiety followed me to the physical rehabilitation center where I spent a couple weeks. While there I fell several times while learning how to use the walker, the bedside commode, and how to move myself from the hospital bed to the wheel chair. I was released when the social worker had set up follow-up appointments and home health. She explained I couldn't leave the rehabilitation center unless I had a primary care physician to prescribe my much needed medicine. I gave the name of my husband's new doctor who we were planning to have as a family physician.

I was using a walker and a wheelchair when I was taking care of my baby when I left the facility. I had a lot of anxiety, depression, and the feeling of such utter hopelessness over the events of the last 30 or so days. My life had taken an extreme turn. I continued to pump out my breast milk because I wanted to be able to feed my daughter once I was no longer on my pain medicines. It wouldn't hit me until much later I wouldn't ever be able to give my infant breast milk because I had to take these medicines in order to function. Without them, I would be curled up in a ball in bed screaming for someone to make me unconscious. The pain was something no one could live with no matter the size of their will power. I had my daughter to take care of.

I saw many doctors over the course of the next couple weeks. I continued to go to physical therapy after in-home therapy ended. My c-section doctor assured me I would be able to walk and be free of pain once the hematomas were absorbed into my tissues. This gave me a sense of hope. I saw Dr. N., a neurologist. This was an appointment I was anticipating for weeks because I desperately needed answers, a prognosis. When I saw him, he complained at length about medical insurance interfering. He then told me: "If I were you, I would get my tubes tied." These comments were shocking to me. My heart began to sink even lower than it already was. I began to fight back tears as I began to feel a heavy weight being pressed down on my chest. The last flicker of hope went out when he told me: "I don't know why you are here. I am not an expert on iliac hematomas."

I had seen my husband's doctor, our

new family physician, Dr. L. a couple times and was impressed by his friendliness. I was able to take a deep breath and trust in this doctor. One day I went to see him. He came into the examining room. His demeanor was different. He looked uncomfortable and stared at the floor, not at me like he had done before. I sensed immediately there was something wrong. He did not greet me with a handshake and a smile. He sat, his back to me. He told me: "You are on too much pain medicine. We have to start taking you off." I was shocked. At the time, I was still having quite a bit of background pain. My pain levels were out of control and erratic. I was barely getting by with the pain I was still enduring. I responded to him: "Doesn't pain level determine when to go down in dosage? My pain levels are still high."

Dr. L seemed incensed by this answer and looked me in the eye with an expression of pure accusation and insensitivity. He responded: "I'm not sure you're even in pain. You just came in off the street." That comment made this sick feeling erupt in my stomach. I fought the tears I knew were coming. When you are someone with chronic pain issues especially to the extent of the pain I was experiencing, the threat of having your pain medicine dosage being altered was equal to being threatened with being thrown into some kind of torture chamber. I didn't think my experiences and my condition could be questioned in light of everything that had happened to me. I was the last person who would choose this reality of being stuck taking pain medicine. My plan before the birth of my daughter was to have at least two children. Having pain and being on pain medicine meant I could not fulfill my dreams for my family. This continued to devastate me and was the source of extreme emotional pain.

Dr. L. went on to tell me that the amount of pain medicine I was on was equal to twenty-seven Percocet. He told me: "I don't want to be involved in any future addiction." I tried to slow down my rapidly beating heart. My stomach was twisting tighter making me want to vomit. I decided to appeal to reason: "What is the definition of addiction?" He dismissed my question. I went on: "I don't have a history of addiction or abuse. I don't even smoke or drink." Physical addiction is completely different than psychological addiction and abuse. The fact is people like me are less likely to abuse their medicines than the general population. He responded: "You smoke. I thought you told me…." He looked into his paperwork which took him a few minutes. I thought he had mistaken me for a different patient. What else could explain his sudden lack of empathy? He faced me and said: "Come on, you don't really want to use a walker, do you?"

I didn't know how to respond. All I could do was stare bewildered. He was questioning everything I had gone through, the loss of my ability to walk and struggle to use the walker, the loss of my life as I knew it, the loss of my social work career plans. Did he actually think I was using a walker for the heck of it? I felt lucky to be using a walker since I couldn't even do that just weeks earlier. He questioned the fact I had PCOS which is an infertility issue. For some unexplainable reason, I had become a lair, a criminal, a drug abuser in his eyes, and all I had done was become disabled. I wondered what happened to the Hippocratic Oath doctors apparently take. This was "Do no harm"?

I started crying. I felt so small and insignificant. On top of everything else I had to endure, this treatment I was getting was too much. He had me sign a contract that basically said I would only get prescriptions of these narcotics from him. I felt humiliated. I wiped my face with a tissue and signed his paper. He said to me before walking out of the room: "G-d bless."

Even now as I write this, this incident had such a profound effect on me I still discuss it with my therapist and experience anxiety before any doctor appointment. It was an incident that also led me to get a different doctor. After three and a half months after the onset of my symptoms and being misdiagnosed, Dr. H. diagnosed me with transverse myelitis after he reviewed my records and gave me an examination. He assured me the epidural I received in the hospital had nothing to do with what I was experiencing. He also assured me this was never caused by iliac hematomas. He said it could take eighteen to twenty-four months after the onset of symptoms to determine how my life would be like for the duration. I was relieved to have a diagnosis at last. There was speculation the uterine infection I had led to all this mayhem. I did some reading about it and joined the TM support group in my city. Life took on a kind of normalcy, a pattern with no more major, unusual pitfalls. I still experience pain in my leg. I had been so relieved this ailment was no longer a mystery. A named illness felt like a conquerable one.

Learning how to walk again was just a preface to the start of the real journey. One part of that new journey is trying to retain the positive attributes of my personality and resisting the negative attributes of becoming more on edge, hopeless, and depressed. I am still working on trying to rise above the ill treatment I had received from insensitive, heartless doctors to whom I had given my power. I still struggle with trying to reclaim that power. Despite it all, I remain a spiritually-oriented individual. This part of who I am led to my dream of helping people in need. It continues to aide my thinking and reasoning. The other part, the hardest part of learning how to walk again was

learning how to walk on a completely different path than the one I had set out on and had so actively pursued. Learning to come to terms with not having control anymore continues to be a daily lesson on this path. I have to trust in something greater than myself.

The path I was on before consisted of raising a conscientious, healthy female; finding a career as a social worker helping families and children; becoming healthier; having more children and becoming a foster parent with Chris. Now it is simply living on a day-to-day basis. The fact I cannot have children as long as I am sick and on these medicines remains something I greatly struggle with. One shining light remains in my life, my daughter, Angelea. It is for her that I smile and laugh even though sometimes I feel I am dying inside from enduring the pain, feeling sick, and isolated. All but one of the dreams I had before becoming sick had slowly flickered out and disappeared like the sun during sunset. Maybe it won't be like this forever. Those dreams could reappear again like the sun during sunrise after this sudden detour comes to a thankful close. At that time, I can reconnect with a different destiny that resembles the one I had, the one I had worked so hard to achieve, the one that doesn't involve pain.

Mary Lou Qualtrough
Physical Therapist

Sudden Onset

My name is Mary Lou Qualtrough and I had a sudden onset of Transverse Myelitis on September 27, 2004. I was 62 years old and I was working full time in my profession as a Physical Therapist in home health care and living independently in my town house. At approximately 9:30 AM I was working with a total knee replacement patient when I felt a sudden change in the strength in my legs. I knew something was drastically wrong. This was the last day I worked in my Physical Therapy profession. The goal of this article is to show how I went from denial to acceptance by being my own patient advocate to "make it happen" with this life change. I had many trials and treatment options. Some were successful and some were not, but all were worth attempting with the hope that my symptoms could be controlled. I also had many challenges with my health insurance company for coverage of services. I want to share my experiences to encourage and inspire others who have this disease.

By the time I got to the ED at 11 AM on that day I was not able to fully support my own body weight. They diagnosed me with a possible stroke. I remember lying in the ED experiencing the rapid decrease in the strength in my hands and legs and the starting of paralysis. I had severe weakness in my trunk muscles. I was not able to sit without support and I was not able to roll over. I had a band of numbness around my chest extending down to my feet. I could not perform a straight leg raise on the left and could only raise the right leg five inches off the bed. A Magnetic Resonance Imaging scan and neurological evaluation were performed and I was started on IV steroids. A definite diagnosis was not made, but the MRI findings did show abnormal findings in the spinal cord in my neck. My strength and mobility continued to decrease rapidly over a few hours. My left leg and right arm were weaker than the right leg and left arm. I had no strength or movement in the right hand and very limited movement in the 3rd through 5th fingers of my left hand. There was no movement in the left thumb or index finger. It took two people to help me walk and they really were dragging me.

Acute Rehab

I was accepted to the acute rehab spinal cord unit at the University of Rochester, Strong Memorial Hospital on October 4, 2004. Every possible test and evaluation was performed and the Neurologists made a diagnosis of relapsing-remitting Multiple Sclerosis. I refused to accept this diagnosis because I felt I was too old for an initial episode and never had symptoms of weakness or a remission in the past. Of course I was sure I knew everything, but the Neurologists were firm with the diagnosis. I cried for eleven days, then realized I better get going and take advantage of the great rehab care that was available to me. The MD in charge of my case told me not to think of the end, which I knew all too well, but to think of the journey along the way. I will never forget this and her words come back to me often.

I was so overwhelmed when this happened. Even though I was a Physical Therapist, I did not know my elbow from my knee about what was happening to me. I needed all the direction and intervention that I could get. My PT consisted of spinal stabilization exercises, balance exercises on a small trampoline, and every strengthening exercise imaginable. My strength improved in the right leg, but weakness persisted in the left leg. There was no return of motion in my right hand. With the intervention of Occupational Therapy, I was able to dress, feed myself, write and use the bathroom with assistive devices. Transfers from the bed, chair, toilet and car were independent with a walker. I was able to move about unaided with a walker for short distances. The IV steroids were tapered and I was started on zanaflex, an oral medicine to decrease the spasms. Recommendations and modifications were made to my town house and I was discharged home on October 19, 2004.

Definite Diagnosis

The Neurologist at SMH, who specialized in MS, evaluated me two weeks later and felt I did not have MS. He diagnosed me with Transverse Myelitis. He said TM is an autoimmune disorder attacking the spinal cord in my neck and this is probably a one-time event. Treatment is focused on symptomatic therapies. He also said the prognosis of the disease was impossible to predict at this time. I could not believe this was happening to me. I started my appointments in the Urology Department at SMH for treatment and monitoring of my complete bladder incontinence. At about this time a friend of mine gave me Allen Rucker's book, "The Best Seat in the House." His experience with TM was a good source of information and was an inspiration to me. Many of his experiences echoed some of my own.

Drivers Education

I had never been dependent in my life and now I needed someone to take me every place I needed to go. I had just bought a new car one month before this happened and could not even hold onto the steering wheel. In April of 2005 I started going to Rochester Rehab at the Al Sigel Center for driver evaluation and training. After testing, it was determined that the spasticity in my legs was too severe and unpredictable to safely use them on the gas and break pedal. I started training with a hand control lever for the break and gas with my left hand and a brace for my right hand to hold it on the steering wheel to turn the wheel. I was terrified and felt I did not have any control of the car. I had the most patient teacher in the world. He also had nerves of steel. I will never forget the first time he had me drive on the expressway. I credit him for a great deal of my success. My confidence and ability slowly increased and after eight months it was determined that I was safe to drive with hand controls. All of the modifications were made to my little 2005 Toyota Corolla and off I went. Without this ability I would be very homebound and dependent on family and friends for all my appointments. That certainly gets very old for everyone after a while. This was a major accomplishment for me and encouraged me to continue to fight this disease.

Medical Intervention

While I successfully built my confidence with hand-controlled driving, a series of medical conditions proved I had plenty of challenges yet to face. I had been very healthy all of my life and belonged to a fitness center and a hiking club. But during 2005 I was hospitalized twice for extremely low blood pressure. Then, the Physiatrist monitored and increased zanaflex and initiated baclofen, another oral medicine, for the pain and spasms in both legs. My Neurologist ordered MRI scans February, July and October to monitor my condition. And, my Urologist treated recurring urinary tract infections. I started outpatient physical therapy for ongoing strengthening and stretching of my muscles. All told, I had a total of 53 different medical appointments during the year.

In June of 2006, pain was so severe down the left leg that two spinal blocks and botox injections to both hamstrings were given with no decrease in pain. During this time I developed an abnormal curvature in my back, a scoliosis. This was caused by the severe spasms on the right side of my back. I always continued my exercise program to help prevent formation of contractures, but I was losing the battle. I also was given a trial of a series of seven infusions of IV Solu-Medrol, a steroid to help decrease inflammation, but these were discontinued as no benefits were achieved. During the remainder of 2006 oral baclofen and zanaflex were increased. The spasms in my hamstrings and hip flexors were so severe that I developed contractures and was unable to stand up straight and unable to lie on my stomach. My pain was 8-9 on the pain scale when the spasms were at their worst. At night the spasms would cause my heels to draw to my buttocks and would almost throw me out of my bed. I performed my home program of stretching and strengthening exercises as much as I could within the limits of the spasms and pain. The scoliosis on the right side of my back increased and my height decreased from 5 foot 0 inches to 4 foot 9 inches and my posture was very bent over. I needed to use a scooter for most mobility in my home. The oral baclofen and zanaflex were the only treatment I had for the spasms and pain. I felt my doctors had given up on me and they were going the status quo route.

Reaching the Low Point

My safety in my town home was becoming very risky. My town house was a two-story unit with the full bath on the second floor, powder room on the first floor and the laundry room in the basement. I required a home health aid two times a week to assist with bathing, dressing and personal care. I could not safely climb and descend the stairs and had the bed brought down to the living room. During this time I had numerous falls. My niece and her husband invited me to come and live with them to give me time before I would inevitably have to go to a nursing home. They made a wonderful, safe, first floor apartment for me. It was large enough for me to use the scooter for all my mobility. I sold my town house and moved to their home in early February of 2007. This was my lowest point in coping with this disease and the loss of my independence.

A Ray of Hope and Planning

As I coped with this low point in my life, a ray of hope arrived with an issue of The Journal of The Transverse My-

elitis Association. The Journal was a part of my extensive research on the disease and in it I learned that there was a center for TM at John's Hopkins Hospital in Baltimore and that gave me hope. Patients from all over the world came there for a second opinion from a Neurologist who specializes in Transverse Myelitis. So, in February 2007, I started the process of having all my records sent to the TM Center and I set up an appointment.

In preparation for the appointment, I had to get prior approval from my insurance company for reimbursement for this out of area second opinion. My primary care physician had to provide justification for the evaluation and get a referral. The actual evaluation by the Neurologist cost $675.00 and I was reimbursed $178.50. All the travel costs were at my expense. I thought I would receive more reimbursement because I had prior approval. I challenged this low reimbursement, but I lost this one. Next time I will investigate the rate of reimbursement better so I won't be surprised.

This trip process was difficult to put together because the TM Center did not return my phone calls with answers to my questions, however, on June 1, 2007 off to Baltimore I went with a friend. This trip was frightening for me because it was the first time I was out of a protective environment since the onset of my TM. However, I really thought there was some miracle news there that would make me all better.

Once at the hospital I received a very thorough evaluation and assessment. Essentially the Neurologist confirmed the diagnosis of TM and the only treatment is based on symptom control. I was very disappointed with this information, but I needed this reality orientation to start to adjust to my life change and plan for the long-term management of my symptoms.

Recommendations and Hope

One of the many recommendations that he made for symptom control was a baclofen pump for spasm management that could be used if maximum oral drugs were ineffective. He explained in detail all the risks and benefits of this intervention. Higher doses of baclofen could be given with fewer side effects of drowsiness, dizziness and weakness. I researched and found that the baclofen pump is made by Medtronic and is known as Intrathecal Baclofen Therapy. Lioresal Intrathecal is a liquid form of baclofen and is injected and infused into the fluid filled area surrounding the spinal cord requiring an implanted drug delivery system. I had never heard about this treatment before and was just intrigued with it. The Physiatrist I had at Strong Memorial Hospital discouraged the pump with the rational that it was too difficult to monitor the dosages. She said that I should continue taking the oral medications. However, I was not satisfied with that answer and I pursued what I felt was a better solution. I was referred to a Neurologist in the Neuromedicine Pain Management Center at Strong Memorial Hospital who managed and worked with the pump. He evaluated me on May 19, 2008 and referred me for a standard screening test one month later. At last I received an intrathecally administered test dose of baclofen via a lumbar puncture. I demonstrated a positive reaction with a decrease in spasms and a slight increase in mobility after four hours. I was on my way.

After a series of delays, the pump was surgically placed in late October, some 17 months after the Neurologist at John's Hopkins Hospital first made the recommendation. However, everything was not smooth sailing as I then developed a seroma in my abdomen. When the first pump refill was attempted in December it was determined that the pump had rolled over. A second surgery was performed in early January 2009 correcting the position of the pump. There was actually nothing to this second surgery. When I went to the pharmacy at Strong Memorial Hospital for the first refill kit I was charged $300.00 saying my insurance did not cover the refill kit. I challenged this with several phone calls and letters pointing out that it did not make sense that the Insurance Company would reimburse for the baclofen pump surgery by a Neurosurgeon and then not reimburse for the Leoresal refills. I won this one. They related the mix up was due to a Pharmacy billing code error. If I had not challenged this I would have lost the $300.00 on this refill kit and possibly been charged for every refill kit.

I did have one chuckle with the insurance company. I purchased a rolling walker with a seat and received a reimbursement of $69.71. I cashed that check. I received another check for the same amount two months later. I sent this check with a letter back to the insurance company thinking it was an error. They again sent another check for the same amount. This one I cashed. I also had a disagreement over another small billing error. With a letter and two phone calls, the insurance company ruled in my favor.

Then over a one year period each week, I went for gradual increases in the flow of the Liorsal into the spinal canal. This was non-invasive and all done by computer. During this time the oral baclofen and zanaflex were being tapered. There was the most amazing gradual decrease in spasms and the horrible pain in my hamstrings, hip flexors and hip adductors. The Nurse Practioner who managed the increases was so extremely capable, knowledgeable and encouraging to me. I received a computer print out of each increase, the dosage and date of refill. I also went to a Physical Therapist for myofacial release to the ham-

strings, hip flexors and back muscles and this helped to decrease the contractures in the hips and knees. I was able to perform my stretching and strengthening program at home again and I slowly progressed to ambulating with a walker for long distances and a forearm crutch for short distances. I put the scooter away in the closet.

Improvement Begins

I realized that I did not need to live with my niece and her husband for their assistance in my care and moved to an independent apartment in June of 2009. It is a first floor, two-bedroom apartment with a galley kitchen. The kitchen is small enough for me to move food and dishes safely. I can prepare small meals and have not starved to death. I had several friends who were very against this move away from the support of my family, but I am being successful. I am able to drive my car with my hand controls and strong enough to go to the market for shopping. I even went on a four-day vacation to Charleston South Carolina last July and I am planning another vacation to Boston this April.

I have now been in my apartment seven months with success in this delicate balance. I continue to have a home health aid two times a week for assist in bathing, dressing and help with the laundry. My family and friends are always there for help when I need it, but respect my safety judgment and need for care. In November of 2009 I started volunteering as a receptionist at the American Red Cross one time a week at a blood donor site very close to my home. This is a wonderful fit for me. My baclofen pump levels seem to be just right now to allow me to perform at this level. I have refills to the pump every two months with increases or decreases as needed depending on the spasms. I recently applied to be a volunteer for the Medtronic Ambassador Program to share my experiences with individuals and family caregivers who are considering the Intrathecal Baclofen Therapy. I would never have believed that I could progress to this level after five years since the onset of the Transverse Myelitis.

I had professional Physical Therapy and Occupational Therapy at every level in the course of this disease, either with home care or out patient. I now have a good home exercise program for stretching, strengthening, and positioning exercises and I know when to increase or hold the exercises depending on the spasms. I have every imaginable adaptive device for my hands to help with activities of daily living from very innovative Occupational Therapists. I feel the most beneficial PT for me is hydrotherapy. It is just amazing, even to me, how straight I can walk in the water and how the buoyancy of the water decreases the amount of fatigue I feel with regular exercise. This is just what works for me and may not work for everyone.

Conclusion

I have been asked many times how I was able to keep going through all these changes in my life. The reason is because of the wonderful family, friends and caregiver support that I have. They have encouraged me and praised me for every advance I have made. They also helped pick me up when I was down. This whole disease has been so overwhelming for me that I cannot imagine what it would be like for someone who does not have the basic knowledge that I have as a Physical Therapist and who knows community resources and the workings of health insurance companies. Maybe this review of my experiences can help someone who is going through a similar devastating experience. I want to thank The Transverse Myelitis Association and Sandy Siegel for this opportunity to share my experiences.

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Education and Experience:
BS Degree in Physical Therapy from St. Louis University in 1963; MS Degree in Public Administration in Health from the State University of New York at Brockport in 1998.

Forty-one years of physical therapy experience in the acute care general hospital setting, short-term rehab and long-term care in nursing homes, rehab in a Veterans Administration Hospital, and rehab in the home care setting.

Phillip Gear MD

I wanted to share my story with others as it was through the stories of others I received hope during the early stages of my disease. Further, my family and I were able to realistically look at my illness through another lens and didn't feel so lonely in the struggle of coping with TM.

My life changed on Halloween, October 31, 2002. I was a successful fifty year old Pediatrician in Phoenix, Arizona in what I thought was the peak of my career. I was looking forward to going to Florida the next day to visit my Mom who was fighting ovarian cancer. I woke up that morning with a tingling sensation in my right hand. Hoping there were no circumcisions to be done, I made it through rounds that day and by the time I made it to the office I could hardly sign my name. After a conversation with my physician/friend we agreed I probably had some transient palsy which would disappear after a bust of steroids. That evening both hands were now numb and I was unable to help my kids carry their Halloween bags.

Next morning I woke up with an excruciating pain in the back of my neck. My hands were still numb but, with the help of my eleven year old, I buttoned my pants and headed for the airport. When I arrived at the parking lot both legs were stiffening and I real-

ized I was in trouble. My wife somehow got me into her car and after a full day of tests, blood, spinal tap and MRI, I was given the news I had Transverse Myelitis. My lesion was C4-C8, high up I was told, and I was admitted to the ICU. I didn't recall hearing about that disease in my medical training, but I assumed TM was like Guillan Barre and that I would be practicing medicine again after a two week hiatus. I maintained that thought or state of denial for three weeks until I realized my only highlight during that time was movement of my right big toe.

I was given a short course of IV steroids and sent to rehab. I was no longer a doctor, I was a patient. My therapists jokingly called me Doctor Phil. I stood up for the first time three months later and began walking with assistance soon after. Each achievement was a celebration. Helpful for me was hydrotherapy. Walking in a warm pool took my weight and balance problem out of the equation.

My fears during recovery were numerous and incessant initially. How would I earn a living? Would I be able to walk my girls down the aisle? On and on until I gave up worrying and started accepting my condition. TM and I are at peace now for the most part. There are times when I feel guilty that I may not be able to ski with my children or teach them to swing a golf club, but I don't dwell on that. I look for and find gratitude in what I have, though some days are harder than others. My hands are still spastic. I continue to have numbness from the shoulders down and can only walk short distances, but I enjoy life as a journey. I believe through my experience after resetting my priorities I have come out a hopefully better person able to share my story with my friends, patients and family whose ongoing support I will always appreciate.

Phillip Gear MD
Phoenix AZ

I'm still here

My name is Kaley Connor. I was diagnosed with TM in 2003 when I was just 22 years old and a junior in college. I was unable to finish my degree, not just because of my disability, but because I had three young children to take care of while my husband works his butt off to make ends meet. Being a mother with TM may be the hardest job I will ever have to do, but I wouldn't give it up for anything. My kids have been through a lot with my illness. I've tumbled down the stairs and broken bones from many falls. I once fell through a window trying to make it into the bathroom.

While I wrote this poem … I wrote this for all of us.

I'm still here

When I'm all by myself
I feel the heartbeat of somebody else
With the same ringing in my ear
I count on the things that I always fear
They give me pills to ease my mind
But restless nights I always find

I used to take advantage of the smallest things
And now I fly with broken wings
I feel alone even with a full house
Being "normal," is what I miss most
I once was a tree, I stood very tall
But I've been cut down and now I am small

I've learned I'm lucky though it's been tough
Because now I know that I have more than enough
I never used to stop for a rainbow or a simple sunset
But now I stop so I'll never forget
For all this beauty the earth has to give
There has to be a reason that I was supposed to live

So I'm taking this one day to two
And while I'm here I'm enjoying the view
I know I am never really alone
Because G-d is watching until I come home
And so I live the best I know how
That's really all any of us can do for now.

Kaley Elizabeth Connor

My Struggles as a Consumer of Pain Medication

Barbara Sattler

Before I was diagnosed with TM and became a consumer of pain medication, I didn't pay much attention to the subject. I remember reading articles by people who complained about being in pain and not getting enough meds. Frankly, I thought the person might be a drug addict; or I believed the doctor knew best. I am sure many people today who haven't suffered or seen a loved one suffer feel the same way. I hope they learn the truth, not the hard way.

Before I had TM, I assumed the worst part of a chronic illness was the illness itself. For me, the worst part has been taking narcotic pain meds because of how the world, doctors, pharmacists and people have treated me. Even though I was clueless, I expected others not to be. I'm lucky that I have a supportive husband, son and lots of friends who I can rely on.

A friend of mine who has been an RN for over 20 years and knew I had TM, told me I didn't need morphine. She recounted her story of suffering horrible pain and being prescribed narcotics, which did no good. Finally she got a divorce, realized she was gay and her pain went away. I told her I was happily married and heterosexual and the meds helped. She looked at me as though, if I would just face the facts, I could stop using pain meds. "I have a disease," I wanted to shout at her, "a

horrible chronic disease."

Recently, I visited some relatives who asked a lot of questions about TM and I told them that I took morphine. My cousin, horrified, responded, "What if you get addicted." To myself I said, "Would you prefer that I live in horrible pain?"

My favorite question is, "Do you get high?" Not only do I NOT get high, but the morphine causes side effects such as constipation, dry mouth and fogginess. Some of these can be mitigated with other medication but then you wind up taking a ton of pills.

I am sure that every one of you in my situation has similar stories, if not ones that are worse.

What I Hate About Taking Prescription Narcotics

I hate that a narcotic prescription has to be picked up in person and can't be faxed. I hate that you have to show a picture ID when you pick it up. At many pharmacies there is a big sign: YOU MUST SHOW A PICTURE ID TO PICK UP A NARCOTIC so everyone in line knows why you are there. So much for privacy.

I hate that although the DEA allows 90 day scripts, all the doctors I know will only prescribe 30 days. I hate when I go on vacation, I have had to show my itinerary to get extra meds and I only get enough to cover the trip. What happens if I miss my plane? What happens if an emergency impedes my ability to get home? Luckily I have never lost my pills or had them stolen, but I hate that you need a police report to prove your pills were stolen. I don't have any idea how I would go about demonstrating that they were lost should I lose them.

I hate that if the script is written incorrectly, you can't get your pills. A call from the doctor will not suffice. This happened to me several times. If it was a weekday, I had to go back to my docs, pick up the new script, and take it back to the pharmacy. What about a weekend when the doctor's office was closed? The office recording said that on call docs would not deal with medication issues. I tried anyway. I was told that I should have taken care of RX problems during the week. I told the doc it wasn't my fault, the office wrote the RX incorrectly. He didn't care. His response was, "go to the emergency room." I won't even go into that as an option.

Pain Doctor

About a year ago, I decided I was doing well and wanted to reduce my medication. My neurologist referred me to a pain doctor. After waiting an hour, I saw the doc for five minutes. He advised me to reduce my dose 40 milligrams. I felt this was too much. He said don't worry it will be ok. It wasn't. I had a horrible couple of weeks, suffering both pain and withdrawal.

Second visit. Although I wasn't doing well, he suggested I again reduce my dose 20 more milligrams. When I objected, he threatened to not give me any drugs at all. When I got upset, 'he' told me he had gone cold turkey after taking more narcotics than I was on. Was I there to hear his story or to receive help? This 6 foot 5 inch, 300 pound man became angry with me. Thank goodness my husband was there. Ultimately, the doc gave me a referral to a pain management practice.

Pain Management Doctor

The doc who ran this practice was kind, knowledgeable about TM, and spent time listening to me. He was outraged at the previous dose reduction and said I should go down only 10 percent at a time. Had I found the right place?

In his office, protocol was seeing the doctor occasionally and otherwise a Nurse Practitioner (NP). The first time I saw the NP, he accused me of lying about drugs I was taking. More than anything, I despise this accusation. I never cheated or tried to get additional drugs no matter how much pain I was in. I prided myself on that. I insisted the head doc come in and he verified that I was telling the truth. The NP also accused me of missing an appointment. Ridiculous, because if you don't go, you don't get your meds. No apologies for the accusations.

I avoided NP Rachett and my treatment went well. I had reduced my meds to less than half of where I started, when I began having pain. When it was time for my appointment I was told only Rachett was available. I took my husband. Before you see the doc or NP, you fill out a form concerning your level of pain the last month. I wrote things weren't going well. The NP entered the room by complimenting me on how well I was doing. Had he read the form? He then asked why I wasn't doing well and seemed angry that I didn't know. "It's the disease. That's the way it is," was all I could say. He examined me cursorily and said he saw nothing wrong. How can you see nerve pain? I asked if I could go back up to 10 milligrams for the next month. "NO," he said without any reason or suggestions to deal with the pain. I felt we were in a power struggle and he had all the power. He walked out and refused to talk further. Once again I am looking for another doctor.

I am a competent 62 year old woman who has been a lawyer and a Judge. I have tried and presided over capital murder cases. I have raised a son. I have lots of friends and skills. I have won awards. I have spoken to crowds up to 600 people. Yes, these medical professionals have brought me to tears in a second. They have made me feel incompetent, stupid and unworthy. I keep thinking someone will under-

stand that I have a disease or appreciate how hard I've worked going down on my meds and praise me instead of accusing me of cheating.

The whole atmosphere at the pain doctor's office is one of surveillance and fear. Signs warn you that if you try to go elsewhere to get medicine, you will no longer be seen. Your urine is tested. Your word is never enough. I am not naïve. I know people cheat and that some people don't have a disease other than addiction. The problem is if you have a disease, the atmosphere is not conducive to getting help.

I blame most of this on DEA and the government. Most docs are competent and care about their patients. Without DEA breathing down their backs, they could probably prescribe appropriate meds. I also blame it on our societal values that suffering is good for you, as long as the person saying it isn't the one suffering. We don't give dying folks enough meds because they might become addicted!

I love morphine because without this drug my life would have been hell. I wouldn't have been able to work, go on trips, or live a life worth living.

I have had a couple of wonderful doctors, one a primary care doc and one a neurologist who have been competent and caring. The docs I met at Victory Junction were all special. I only wish they could care for all of us.

Jeff Kause

In 1975, I was a healthy, active, ten year old boy the first time I experienced TM. I was being treated for a sinus infection when one morning I could not get up out of bed. Terrified of what might have happened to me over night, I quickly realized that I was paralyzed from the neck down. I was put in the hospital and tests begun. Our family doctor was quickly joined by a team of neurologists and other experts to figure out what was happening to me. The concern became doubled as I am an identical twin. Would Tony, my twin, be affected? We always seem to suffer the same traumas within days of each other, but Tony never developed any of these problems. Three spinal taps, an EEG, an EKG and enough blood tests to last a lifetime was just a start. Cortisone was the drug of choice then. I ballooned up like the girl from Willy Wonka, but I didn't care, it seemed to be helping me. My paralysis stopped progressing and actually started to go away. After one month in the hospital, I left under my own power, shaky, but able to walk. After several months of home rest, it seemed like it never happened; with only slight nerve damage that no one could see, but I could feel. During countless visits to the team of doctors afterwards, I was told what I had was one of two cases ever reported in Michigan and the only one that had recovered without any permanent paralysis. Several papers were written about my case. There wasn't even a name for it. This was an acute disease and I would never get it again.

Although the experience and concern for a relapse was always there in the back of my brain, over time I gave it less and less thought. After all, the doctors had told me I could never get it again. I was continually healthy and active, playing high school football or riding my ten-speed bike. I worked part time, earned good grades in school and was completely shocked when at age seventeen, I woke up one morning and couldn't get out of bed. I was just starting my senior year of high school and getting over another sinus infection while taking an antibiotic. I remember feeling achy and sore in my back the previous night. This time the paralysis was from the waist down. Once again my family doctor was able to diagnose and treat me quickly, still never giving me a name for the disease. I recovered more slowly this time. I missed the first semester of school and my last year of football. It was a devastating time for me. I had planned on going off to college but that was not meant to be. Even though I was walking fine again, I had more nerve damage in my legs and hands. I also had nerve damage within my bladder. I had almost nightly incontinence and a constant pressure on my bladder to empty it. I was limited in activities I could do, so my social life dwindled away. I tried talking to the doctor and he told me my nerves would improve over time. But they did not improve and I just went on coping the best I could.

I met Dianna ten years later and she married me, despite my health problems. Life went on. At the age of thirty-six, once again I woke up one morning barely able to get out of bed. I had been under a great deal of stress during a seven-month period when my wife endured three major surgeries and nearly died. As she was healing, I became sick with the flu and then a sinus infection. I took an antibiotic but still was feeling really terrible when the pain in my back started. Even to this point, I had never received a diagnosis and I had never heard the words transverse myelitis. Fortunately, my current physician was able to diagnose and treat the TM before I got much worse. A neurologist was called in to help treat me. I still went through a battery of tests while he started the treatment.

Medicine had changed in the eighteen years since my first attack. Now we have MRI and CAT scans, but spinal taps are still around. I also spent two hours in the MRI machine as the technician made a mistake on the first test. Treatments now are steroid Z-Packs and Solu-Medrol instead of Cortisone. After getting a dose of Solu-Medrol via IV treatment for three days followed by high doses of Prednisone, I began feeling much better. I was out of

the hospital in a week. No permanent paralysis, but my bladder became worse. I was on a maintenance dose of Prednisone. Prednisone is a most unpleasant drug to be on because of its many damaging side effects to a person's body, such as causing brittle bones, weight gain, diabetes, high blood pressure and more. Personally, I experienced a great rush of energy. This is bad when you are supposed to be resting and recovering. Several weeks later I felt strong and was tapering off of the Prednisone. I began having prickling in my hands and feet, as well as pain in my back. The neurologist didn't seem to be concerned; she kept ordering Prednisone and even another dose of Solu-Medrol.

I wasn't happy with the answers and treatment I was getting from this neurologist, so I asked my primary doctor for another neurologist. A second neurologist came to the conclusion I should start treatment for multiple sclerosis. The treatment for MS was to take one of the ABC drugs. I was in no hurry to begin such treatment especially when he was not sure of my diagnosis. This was due to the fact that there are two schools of thought to TM. One school believes TM is related to MS and should be treated as such. The other feels TM is separate and responds to different treatments and drugs. My doctor didn't accept the MS theory, and as the neurologists were still unsure, he suggested the Cleveland Clinic. After much discussion with my HMO, I was granted a CONSULTATION ONLY visit to a MS and TM physician at the Mellen Center for Multiple Sclerosis. He told me there were two other people he knew of that had similar experiences with recurrent TM. Those patients had success with taking a low dose of Imuran, an autoimmune suppressant. He went on to explain how there is a growing number of recurrent TM; that there seems to be a trigger to the onset of a TM episode.

When I looked at my bouts with TM I began to see the pattern. I was so rarely ever sick; I must have an awesome immune system. So when my immune system began fighting off the sinus infections, aided by the antibiotics, it was so powerful, it turned on my own body and attacked the myelin sheath. Taking the Imuran made the most sense to me. Besides, I didn't like the idea of giving myself shots every week and spending the weekend recovering from the side effects. My other neurologist assured me Imuran was safe, but they did monitor my liver with blood tests. Imuran has been used in Europe since the 1960's and has been very effective for many people. For the past three years I have been taking 10 mg of Imuran and have been symptom free. I also receive Acupuncture, which seems to help calm my nerves and increase my balance.

Although I am symptom free, the nerve damage to my bladder is a daily reminder that I have been sick. Now with the knowledge that a severe sinus infection could bring on another attack, I wonder every time I get a runny nose. I am very grateful my body responded to treatment and I am able to walk and play with my son. We have just tested and received our first-degree black belts in Tae Kwon Do.

I have recently spoken to Jeff. He told me that he remains symptom free and that he is no longer taking Imuran or any other immune suppressant. Jeff's case is a fairly confusing one. It is not clear that an inflammatory attack was ever identified during his subsequent attacks. Is it possible that Jeff's worsening of symptoms from subsequent 'attacks' are being caused by the sinus infections as opposed to having new inflammatory attacks? It is not uncommon for people to experience a worsening of their symptoms during a bacterial or viral infection. As Jeff's first attack was high on his cord, the subsequent attacks involve areas of his body below the level of damage from this first attack; no new parts of his body are involved. I have encouraged Jeff to seek guidance on his condition from one of the medical centers that specialize in these rare and recurrent neuroimmunologic disorders.

Thomas Wyatt

Valentine's Day 2007. I expected it to be a normal holiday spent at work, then coming home to spend a happy weekend with my wife and 18 month old daughter. Instead, my life changed forever.

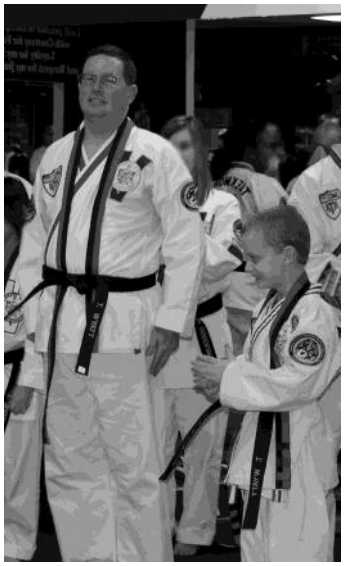
While at my job that day, working as a school custodian, I noticed a pressure and discomfort in my lower back. Perhaps I had slept wrong, I thought, shrugging it off. But driving home, the discomfort persisted and my feet had that tingly sensation you get when they fall asleep. I was also strangely fatigued, barely eating any dinner before I collapsed into bed. Maybe the flu was making a comeback, I thought, since I had suffered a strong case of it just ten days earlier.

The next morning brought no improvement. The pins and needles slowly worked their way up my legs to my knees, then to my waist. My back still ached. I was worn out, spending Saturday on our couch. By Sunday morning, my toes started to feel numb and the pins and needles sensation was moving even higher. I considered going to the ER, but I had just started my job and did not yet have any medical benefits. We were also just recovering from four months of unemployment, and a trip to the ER would set us back financially. But Sunday evening, when I had trouble negotiating the steps upstairs to our bedroom, I told my wife that I would call our family doctor right away the next day.

On Monday morning, just two days after symptoms had first appeared, my legs would barely work. My wife drove me to the doctor's office, and when the nurse called me in to the examining room she had to help me walk, keeping me from falling down. My legs just wouldn't support me anymore, and I could hardly feel anything below my waist. The doctor sent me straight to the ER, where my wife pushed me in with a wheelchair. The next hours were a blur of questions, tests, doctors, more doctors, and more questions. The on call neurologist was summoned. I found out later how fortunate we were to have this particular doctor on call, because he had diagnosed and treated cases of transverse myelitis before. We were in the best of hands, but the doctor could not soften the blow of my probable diagnosis: idiopathic transverse myelitis.

At my lowest point, the first day in the hospital, I had no sensation from the armpits down, and even my face was affected. When the doctor asked me to smile, only half my face moved. I asked the doctor how quickly I would recover, but he couldn't answer. He simply said that time would tell.

The next weeks were long and difficult. My neurologist prescribed gamma globulin, which began to reduce the inflammation in my spine, but I had to learn to use everything again. The process was painful and slow, one small degree of improvement at a time. I felt compelled to recover as quickly as possible, though, knowing that my little family was financially drowning from a lack of income and, soon, medical bills that we had no way of paying. I became obsessed with fighting to recover everything. If the physical therapist wanted me to repeat an exercise fifteen times, I pushed myself to fifty. The therapists wanted to have therapy four hours a day, but I kept on exercising after they were done until the nurses said I was hurting myself. Yet with all the effort, after a month in the hospital, a social worker broke the bad news to us: she did not expect that I would make a full recovery, and she wanted me to apply for vocational rehab.



But four months later, against all odds, I returned to my full time job. I had applied to voc. rehab, but by the time I went walking into the office for my formal evaluation, they said I could not be considered handicapped. I had made too much progress, and recovered almost all functioning.

Two and a half years ago, my family and I (now including my son) decided to join a local taekwondo club. Since my teenage years I had wanted to pursue a black belt in the martial arts, but never had the opportunity. Now, as we put on our uniforms for the first time, I had no idea how this would turn out. To the untrained eye I appear unaffected, but I do have some residual effects from my paralysis. My leg muscles are tight sometimes, making it difficult to jump, and I don't always sense temperatures the way other people do, especially in my legs and feet. I still occasionally have leg spasms at night and sometimes my short term memory also appears affected. Even my bladder sometimes fails me. I would now be asking this body to work out vigorously three or four times a week for at least two years, learning moves and jumps I'd never tried before, and pushing myself to memorize long series of movements. This was without a doubt the hardest thing I've done.

Happily, in October of 2010, I tested for and received my black belt from the American Taekwondo Association. As the teacher gave out the new black belts, he asked if any of us would like to stand and say a few words. I stood and briefly described my history with transverse myelitis and stated how much it has meant to me to be able to accomplish one of my lifelong dreams despite having TM. Yes, TM put me on the sidelines for awhile, and I will probably live with these residual effects the rest of my life. But it was not the end. Transverse myelitis tried to beat me, but I finally feel like I have truly overcome this disease.

Nerve Pain Treatment

Vicki McKie

I want to tell you about a medical treatment that is helping me. I have been having this treatment since December 2009, and it seems to be helping with the constant pain which resulted from my TM.

I was hit with TM in December of 2007. I thought I had ruptured a disc because of sudden severe pain in the thoracic spine with radiating pain and spasms around the ribcage. I woke up the next morning numb and partially (mostly) paralyzed from the chest down. Because it was during Christmas/New Year's holidays, I went through about three weeks of trying and failing to see a doctor. Finally, three months later I saw my neurologist in Augusta, Georgia and was diagnosed with TM.

I had an atypical presentation with a longitudinally extensive lesion beginning at T1 and down through the Cauda. On the MRI it looked like one of those pine trees that had been hit by lightning and all of the bark peeled off in a spiral pattern down to the ground... except it was the lesion that spiraled around my cord. It was transverse in two segments. Somehow, through G-d's grace, hard work, and stubbornness, I have been able to make a remarkable recovery of function since then. I still have partial numbness and weakness which can get better or worse depending on various factors. Resting can make it better. Illness can make it much worse. I still have significant problems with bladder and bowel control, but continue to work towards something resembling normal. The worst symptom has continued to be the constant pain - what I call the three levels of pain.

Level 1 pain is the deep nerve pain such as sciatica that feels like a lightning bolt or electrical current going down both legs, if I make the wrong move, sneeze, cough or overdo it.

Level 2 pain is at the muscular level and can feel soreness or burn like a muscle that has been overworked to the breaking point. This level of pain often results in sustained muscle spasms that just don't let up.

Level 3 pain is at the skin level and can feel like a severe sunburn or at times can feel numb and painful at the same time - like when you play outside in the snow far too long, then come inside and try to wash your hands. The water may be cold, but your hands will feel numb and on fire at the same time. That is how my back feels 90% of the time at the skin level, especially around the thoracic segment where the band of my bra would be. This results in my not being able to wear the correct undergarments because it is just too painful. Add all of these together, and I deal with a lot of pain on a daily basis.

So, on to what is helping me. Since the diagnosis of TM, the weakness in my back has led to a continuing decline in the ability to maintain proper posture. My abdominal muscles are not strong enough to support my back properly. I believe all of this is related to the quickened degeneration of my spinal discs. I have developed six prolapsed discs in the thoracic spine and three degenerated discs in the lumbar spine.

In early 2009, my neurologist referred me to a neurosurgeon for evaluation of possible disc surgery. Two Neurosurgeons said they would not touch my back. The third is a doctor in the nearby medical community in Augusta, Georgia who everyone said is the best Neurosurgeon in our area. It took six months (October 2009) to get in to see him, but I waited because he was "the best." He told me that he would do the surgery if it comes to that (and it may come to that in the end), but first he wanted to try something else. He referred me to another doctor in his group, Dr. Mark Stewart, Board-Certified Anesthesiologist, Fellowship-trained, Board-Certified in Pain Management**.** According to the bio on their website, "Dr. Stewart is an interventional pain management specialist at Augusta Back Neuroscience. He specializes in the nonsurgical relief of pain, including epidural steroid injections, radiofrequency treatments, sympathetic nerve blocks, facet blocks, sacroiliac blocks, peripheral nerve blocks, and many others. A host of pharmacologic agents may also be utilized, but the emphasis is on non-narcotic methods of pain control."

By the time I saw Dr. Stewart in December, I could barely stand up, shuffled when I walked, could not bend forward and could not bend backwards. I had difficulty getting up and down. I could not lie down flat on a bed and had to sleep in a recliner chair. I was in a heap of pain. After a thorough exam he suggested Pulse Radiofrequency Ablation (RAF). He told me it was not to be confused with an earlier radiofrequency treatment that did not include the pulse intervals, and was not as successful. This treatment does not work for everyone, and there can be risks. However, it has given me more relief, for longer periods of time, than anything else we have tried since I began working on my recovery in March of 2008.

I'll try to explain it in my own words, according to my own understanding. This is not a medical explanation, and I haven't found a good explanation for this exact treatment. Although, there is an explanation for a similar procedure, Radiofrequency Neurotomy that mentions the Pulse method at Mayo Clinic's website at: ***http://www.mayoclinic.com/health/radiofrequency-neurotomy/MY00947***

Pulse RFA is done in two steps. The first step, called the diagnostic step, is to identify the nerves that are actually causing the pain. This is done by epidural steroid injections. Sometimes they inject numbing medicine such as Lidocane instead of steroids. They normally inject one to three levels in one procedure. This may be three vertebrae on the right or left side (i.e., L5, L4, L3 Right side), or both sides of one vertebrae. Two weeks later I go

back for an office visit to evaluate the results of the injections. If I receive at least 50% pain relief that may last a day or up to a few weeks, then I qualify for the RAF treatment – which is step two. The RAF is generally scheduled for two weeks after this appointment. I believe the two week intervals are due to insurance processing and obtaining precertification, so this may not be standard in other practices or situations.

In the RAF treatment, Dr. Stewart has a larger team working to help him perform the procedure. I lie on my stomach on the surgical table with a large x-ray machine (Fluoroscope) positioned over the section of my back they will be working on. The operator of this machine takes live images of my spinal area which are projected onto a monitor for the doctor, his team, and me to see what he is doing. I am awake during the entire procedure so I can tell Dr. Stewart what I am feeling. It is imperative that I lie completely still, so medication is available to help me remain calm, if needed. Using special surgical needles, Dr. Stewart inserts a probe into the space where the nerve root ganglia is located outside of the spinal cord. Once the probe is in place, another member of the team delivers radio waves in pulses towards the probes. This heats the tissue surrounding the nerve root, which somehow interrupts the pain signal from the nerve to the brain. I usually feel a slight tingling of energy in the area of my body that the particular nerve supplies. For instance, RAF applied at the left lumbar level may feel like a low electrical current in the calf of my left leg, or my thigh, or my toe depending on which nerve is being treated. After applying a pulse of radio wave, it is turned off for a few minutes to allow the tissue to cool down before another pulse is applied. Dr. Stewart and the team constantly monitor what I am feeling and at the first sign of pain from the radio wave application, we stop to allow it to subside before moving on to the next area to be treated. The whole procedure takes about 30 to 45 minutes to complete.

I know this is not a medically acceptable equation, but I think of it as having a root canal on my spinal cord. However, the goal of RAF is to interrupt the path of the pain signal from the nerve to the brain WITHOUT causing any damage to the nerve itself. They always tell me that it may take anywhere between a few days to a few weeks before I feel the results of the treatment. But the morning after the very first treatment, I woke up and that sciatic nerve pain down my right leg was just flat out GONE! What a miracle I thought that was. What a blessing. That was December 14, 2009. Results of subsequent treatments have not been as dramatic or as immediate. Each procedure has been different, but each one has given me at least 50% or better relief of pain in that segment.

The down side? There can be risks which are listed in the article on the Mayo Clinic's website: ***http://www.mayoclinic.com/health/radiofrequency-neurotomy/MY00947***

The only side effect I've had is neuralgia in the toe next to my big toe, on my left foot. This doesn't bother me all the time, but can flare up for seemingly no reason. I still feel it is a low price to pay for all of the overall relief I've had.

The other down side is that the relief is not permanent. The Mayo Clinic website says the relief typically lasts between three and six months. Dr. Stewart's office told me their experience is anywhere between three months to a year. My first treatment was December 14, 2009 on levels S1, L5, and L4. I am just now beginning to experience more pain in that area again in the last week of September, 2010 – almost ten months after that area was treated. That is ten months that I have not been curled up in a ball, trying to medicate pain which can't be medicated. It's ten months that I have been able to stand straighter and walk with a gait so much more normal that often people who don't know me can't tell there is anything wrong with me. It doesn't mean that I have been pain free. The results are varied and not every treatment gives 100% pain relief. I still experience a lot of pain in the thoracic spine. For some reason the insurance companies won't cover RFA in thoracic levels, claiming it is experimental. However, for the areas that have qualified to be treated, the results have been amazing in my case.

I know that many people with Transverse Myelitis don't have pain. Many can't feel at all below the level of their lesion. However, if anyone else with TM suffers from any of the levels of pain that I mentioned above, I would recommend doing your own research into RFA; find a qualified, reputable doctor who performs this procedure, and talk to him about whether it may help provide relief in your case. I'm glad I was stubborn enough to continue looking for someone to help me.

God bless,

Vicki McKie - South Carolina / Georgia Transverse Myelitis Online Support Group
vickimckie@gmail.com
http://health.groups.yahoo.com/group/CSRATMSupportGroup/
Look for us on Facebook
http://mamamckie.wordpress.com/

A Miracle in Scotland

Sandy Smith
mmsmith30@btinternet.com

Having had TM for almost 22 years, I still find it very difficult to accept what happened to me 6 months ago. I feel as though I experienced a miracle. I hope that my story will give hope to other TM sufferers.

Friday June 3rd 1988; I was painting the cabin of the fishing boat on which I worked, when I suddenly felt a pain in my right leg and it began to feel numb. Within three hours, the numbness had spread throughout the whole of my lower half and I was rushed to hospital. By now the pain in my right side was so bad, I was punching the sides of the ambulance (and to think a doctor had once told me I had an exceptionally high pain threshold)!

During that first week, I had absolutely no control over my lower half. My right leg was totally paralysed but my left leg was liable to shoot out and kick whoever sat on that side of my bed. My wife and sons bullied me into concentrating every ounce of energy into making my toes move and when my big toe finally moved about a ½ inch, I had to be propped up to see it move as I couldn't feel anything and wouldn't believe them. From then on, we were all determined I would walk again.

The next few months were spent doing intensive physiotherapy. As movement gradually returned to my legs, I developed muscle spasms so fierce that I was yanked out of bed at 3 o'clock one morning, pins and needles in my legs, and then several months of the weirdest sensations imaginable. For days on end, my legs would feel like wet concrete setting, then for no reason, my whole bottom half felt like it was encased in broken glass. There were times when any movement made the inside of my legs feel like a wet cloth being very tightly twisted or my feet felt like big sponges. Sounds crazy, I know, but I felt these sensations even though my lower half was completely numb. It was extremely uncomfortable. Then came what was probably the worst time of all, when I was hit with bouts of white hot pain, mostly in my right side and leg, lasting for up to 20 to 25 minutes and which caused me to black out. The strange thing was, when I regained consciousness, the pain had always gone. This was when my physiotherapists refused to treat me as 'they couldn't cope with the pain I was having' so it was back to hospital for another month. But still no answers. Electrical tests showed that the nerves from my brain to my feet were intact, but something appeared to be stopping the messages getting through. No one could explain why.

We were told that Transverse Myelitis was simply an expression used by doctors when there was inflammation in the spinal column and no-one knew why. We were to spend the next 11 years thinking this!

By February 1989 I was able to drag myself along with elbow crutches. A consultant then told me I would probably never walk and would always have to live with **that pain**. We found a private clinic and, mercifully, acupuncture reduced the pain levels to a more manageable level so that I no longer passed out. I still had no feeling in my lower half, but with endless exercise, I slowly began to walk. We got an exercise bike and, at first, my wife had to turn the right pedal until my left leg was strong enough to allow me to pedal unaided. I spent hours trying to walk to the beat of singing nursery rhymes and this helped improve my co-ordination. My son took me swimming, but usually swam close to the edge of the pool with me, as my right leg was liable to suddenly feel like lead and pull me under. There was still no feeling in my lower half, except for pain, which meant that when I sat in a chair, my top half felt like it was floating. Despite this, I was determined to walk.

I was back in hospital after a fairly major relapse in October 1999 when a young doctor asked me to be his 'test' case. Because this condition is so rare, I appeared before about 30 senior consultants. A chance remark by this young doctor resulted in us finding the TM Association in the USA on the Internet and for the first time in 11 ½ years, I realised that TM, although very rare, is a recognised condition and that other people suffer from it. The relief was indescribable. Four years later we met other sufferers for the first time and I no longer felt isolated. We now attend the TM Scotland Support Group which, with the information the TMA sends us, is an enormous support.

My 'miracle' happened in 2010.

Last August I had a mastectomy for breast cancer which was pretty scary at the time. Thankfully, the cancer appears to be gone. Prof Doug Kerr warned us there was a possibility the trauma of the cancer surgery may trigger a TM episode and this is precisely what happened four weeks after the surgery. Within a few hours, my legs seized up completely and felt as solid as steel. I had no idea what was happening, but knew it was serious. The pain was indescribable and I was rushed to hospital. By that time, my bladder had stopped working and was at capacity, so I was catheterised, given morphine and intravenous steroid injections. X-rays were clear and an MRI scan showed the usual area of inflammation, so we knew it wasn't a new attack. Three days later, the pain was reduced with medication and I could walk a few steps with help. Lots of student doctors spent quite a lot time with me as they don't often get the chance to see TM.

Then on day four, during a pin-prick test, I suddenly felt the pin on my legs. The students were shocked when I burst into tears. **It was the first time I had felt my lower half in over 21 years.** I was probably more emotional than when TM first struck. I still had no sensation in the soles of my feet, but everyone was amazed at what seemed like a miracle. My family and friends were completely astounded and

I can still see our youngest granddaughter with tears in her eyes as she said "Oh Di, it's a miracle." (Di is a local name for Grand-dad.) Regaining sensation brought a whole set of new problems. Once again, I was unable to walk and my bowel went into overdrive which I couldn't control. My bladder flow increased, but much to my concern, I couldn't tell the difference between bladder and bowel. I was a psychological wreck, couldn't understand what was happening to my body, and was terrified that I might lose the feeling in my lower half again.

Regular pain medication kept the pain levels manageable. I began learning to walk in a harness, but was still unable to weight-bear. After several days of physiotherapy, sensation returned to the soles of my feet but my bladder stopped working for almost a day. My son put my slippers on for me and I thought my feet were being crushed. Then I realised, I had forgotten what it was to feel 'normal.' I began to have sensations similar to those of the first year of TM but for shorter periods – pins and needles, setting concrete, stretched tendons, toes felt like dough with gravel between them, etc. I began to feel as though the TM had gone into reverse which seems crazy. I continued to have daily physiotherapy to try and get me walking again, but my legs were extremely heavy and very difficult to control. Two weeks later, I found I could sit comfortably in a chair (for short periods) for the first time in years. My toes, which had been clawed for years, began to lie flat again. I stopped throwing my right leg when trying to walk which allowed me to use a walker frame. My bladder and bowel began to slowly improve and after a lot of hard work learning to walk again, I can walk pretty much as I did a year ago.

It has been an emotional rollercoaster and, at times, a psychological nightmare. But six months later, I still have approximately 90% surface feeling in my lower half and a much improved bladder control. Walking is still difficult, but I feel that every day I walk is a bonus and being able to feel my lower half after all these years is wonderful.

Despite having a lumbar puncture, brain scan and gallium scan, no one seems to have an explanation as to why I regained so much sensation after 21 years. I would be very grateful if anyone can let me know if they are aware of anything similar to this happening. I am extremely grateful for all the support I have from the medical staff, my family and fellow TM sufferers and hope that my story will show that no matter how much time passes, it is still possible to improve.

Sandy is married to Margaret, the wonderful artist who graciously and generously donated her beautiful artwork for the TMA greeting card awareness and fundraising program. Sandy and Margaret are active members of Margaret Shearer's very special TMA Support Group in Scotland.

Rachael Garrett

7-31-10 Saturday 9:09 pm

Dear Sanford J. Siegel:

My name is Rachael Garrett and I'm 12 years old. I was diagnosed with Transverse Myelitis on February 16, 2007 when I was nine. February 14 at 3:00 am I woke up with a terrible pain in my stomach. It felt like a ball of needles rolling in my stomach. I walked to my Mom's room to tell her about it and she gave me some Advil and a heating pad. As soon as I lay down, my legs started convulsing. So my Mom had to drag me to the car and take me to the hospital.

When we arrived, I was taken to a room to have blood work done. Within an hour, I was completely paralyzed from the waist down. My Mom had to move my legs for me. As I tried to stand up, I collapsed to the floor. While the nurse tried to help me, the other nurses were drinking Coke and laughing at the front desk.

Right then, my Mom said, "Take her to Children's Hospital in Dallas." They put me in ICU and quickly started to do tests and spinal taps. Two days later, I was diagnosed with TM. My treatment was IV steroids and a lot of blood sugar checking.

I was admitted to the hospital and underwent intensive OT and PT. On the day we tried walking (with a walker), I promised my Mom 20 steps, but gave her 40. All of the nurses cheered me on.

After a week at Children's Hospital, I was transferred to Our Children's House. The normal recovery rate was three months, is what the doctors said, but I was out in one month. I was able to go home on March 9, 2007. It was the greatest feeling in the world to be home.

I soon started outpatient physical therapy at Hunt County Hospital. By the way, I live in Greenville, Texas. I was soon discharged after two months. Then I went to Plano to start PT and was discharged a year later. Just recently I was discharged from Our Children's House at Baylor in Rockwall. In total, I was in PT for three years.

I now live in an apartment with my Mom, Sheryl Holland, in Greenville, Texas.

I am fully recovered and I am one of the 33% that recovered from TM.

Sincerely,

Rachael Garrett

Dear Alana '05

It's me, your future self. I am here to tell you some news that might scare you at first. On November 11, your life will change forever. What you thought would look like your future will not happen. You will not become Mrs. Watson, nor will you have two children, a family dog, and a one-story house with a white picket fence. You will endure something that nobody can really prepare for -- something so much bigger than yourself.

You are a beautiful girl, Alana, who has always looked at life in a very idealistic, and a bit naïve, way. Right now, you feel the wind at your back. You are deeply in love with a young man whom you will never forget, even when you want to so desperately erase him from your memory. I'm very sorry to tell you that one day, you will no longer have him in your life. Many of your friends will disappear, but for every friend that you lose, several more will be gained. You will quickly learn who your family and friends really are -- some people may surprise you -- but that's the least of your issues.

On November 11 of this year, your life will come to a standstill, literally and metaphorically. It will take so much time for you to adjust to a life that nobody would ask for. However, you will learn more about yourself in such a short amount of time than you know about yourself now. You will discover how brave you are, and how strong of a family that you have. You might not see it now, because life is so perfect for you at this moment. The trials you will encounter will be painful, unexpected, and sometimes, heartbreaking. But it is because of these hurdles that will make you an inspiration to people around the world.

I'm sorry, I really don't want to scare you. That is not my intention. What I am really trying to say is, there is nothing to be afraid of. Right now, you are blessed with a wonderful family, a boyfriend who adores you, and friends who, at the moment, are there for you. But this won't last much longer, because soon, very soon, you will face an entirely different chapter in your life. It may be frightening, and it may be extremely unconventional, but this chapter will give you the answer as to why you are alive.

Promise me that you will appreciate every breath that you take. Feel the sand between your toes. Feel the love that is shared through every hug, every kiss, every touch that you encounter. Keep smiling, Alana, because when you do, the sun shines. Don't shy away from your beautiful singing talent, because your voice is very rich and soulful. People would be honored to hear you sing. Take pride in being such a wonderful daughter, sister, and friend, because you have exceeded.

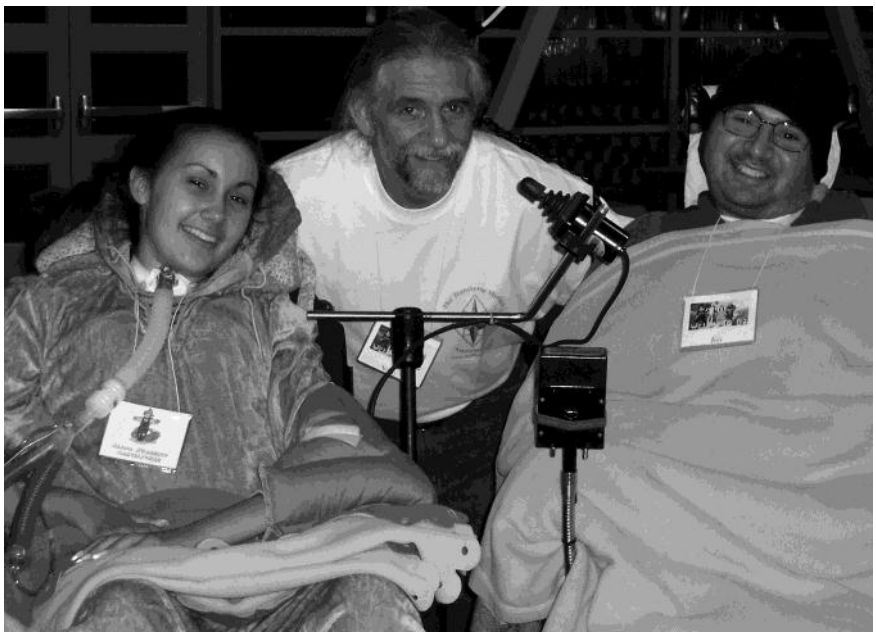
Five years from now, you'll be a completely different person. You will always miss the days when you felt like any dreams can come true. In return, you will learn to love the person that you have come to be. You will understand the importance of family, as well as the importance of hope and faith. You will have the power to inspire those who need guidance, and you will humble people who have more than they truly realize. When I was you, I never would have dreamt of having so much importance. You don't know this now, but in five years' time, you will not only be a better person, but you will make people better.

I know that you've never liked the idea of change, but you will have to get used to it. This is definitely not what you have planned for your future, but I promise you, your destiny is far different and far more incredible than anything you could ever expect. Remember that there is love all around you and people who will always be there for you. Your voice will be the instrument of your destiny. I am very excited for you to go down this terrifying and phenomenal journey. I miss you so much, sweet girl, but I promise that everything will be the way that G-d had planned for you.

Love always,

Alana '10

-- Alana Spence
amspence1989@Gmail.com



Tips for those with TM and the other rare Neuroimmunologic Disorders Patients Helping Patients

Barbara Sattler
bsattler@cox.net

As the years go by after receiving my TM diagnosis, I am learning, sometimes the hard way, to cope with a number of my medical problems. I am sure many of you have also learned various ways of dealing with your medical issues, regardless of whether you have an ADEM, NMO, ON or TM diagnosis. I have been frustrated in the past when my doctors have not advised me of a product that might have helped me with a medical issue or offered some other guidance. I considered that it might be very helpful if we began to share some of the things we've learned for ourselves over the years through our experience. I have asked Sandy to please include these tips in the TMA journals. As with all medical issues, it is extremely important to bear in mind that you should not try anything without first discussing it with your doctor. Also, it is important to consider that what works for one person may not offer any positive benefit for another. With those important considerations, I am going to offer a list of some things I've learned and some of the things that have worked for me.

New Dry Mouth Products
If toothpaste isn't your thing or you need something between brushings, there are two new dry mouth products. Ora Moist is a tiny patch you place on the roof of your mouth. It works for hours and contains no sugar or alcohol. Biotene, the toothpaste maker, also produces a gel you can rub in your mouth. These are available without RX in your drugstore.

Effects of Heat
Maybe because we live in Tucson where temperatures reached 103 degrees on Sept. 20 this year, the effects of heat are often discussed at our support group meeting. Most of us believe hotter weather makes us feel worse. One of our members enlightened us with some information she received from her neurologist. He advised her it isn't heat or humidity that causes increased pain, but anything that raises body temperature, for example having a fever. (Ladies, ovulation also causes the body temperature to rise.) He suggested cold compresses on the neck and head are helpful to alleviate the effects of heat. One person cools off in hot weather by putting ice packs in his hiking vest when going outside for a ride in his wheelchair.

New Medication
For those of you who are taking narcotic medication and want to reduce your dose, ask your doctor about a new medication called Embeda which is a combination of morphine and naltrexone that helps fight withdrawal symptoms. It was developed specifically for those who are reducing their dependence on narcotics. It has helped me reduce my dose in half. It is available by prescription only.

Barbara's Book Reviews

Relaxation Revolution: Enhancing your personal health thru the Science and Genetics of Mind Body Healing by Herbert Benson, MD and William Proctor, JD.

In the l970's, Dr. Benson, a Harvard medical researcher, identified the body's physiologic reaction which he called "relaxation response" that is the exact opposite of stress (fight-flight response). Since then his team has explored how this response, the power of expectation and belief and other mind-body phenomena can produce healing in your own body. Benson and his colleagues established the first therapy to counteract the harmful effects of stress. The book discusses types of self-healing techniques which he believes can treat a wide variety of conditions, including pain. The authors don't claim to cure chronic pain, such as those of us with TM and related conditions suffer, but offer some strategies for pain and medication reduction.

The Pain Chronicles: Cures, Myths, Mysteries, Prayers, Diaries, Brain Scans, Healing and the Science of Suffering by Melanie Thernstrom

The author is a journalist who suffered for years with chronic back pain associated with a degenerative spinal condition. She researched pain studies and reports her conclusions in this book. The book is more historical than self-help, but has some fascinating and disheartening information. She reported on a 19th century study which concluded whites were more sensitive to pain than others, and unfortunately, some doctors may still believe that today. For example, minorities are three times more likely to get insufficient pain relief and have their requests for help seen as drug-seeking behavior. When going to a doctor for pain, women are more likely to get anti-depressants and men opioids. The author also reports on the opioid backlash following the increase in oxycontin abuse in the 90's which led doctors to withhold potentially addictive medication out of fear of prosecution. She also found that patients who believe that pain stems from underlying disease rather than nerve damage will fare worse in dealing with pain than one who understands that although chronic pain feels like an alarm, it is often a false alarm signifying only that the alarm system is broken.

This book's conclusions may make those of us who have had the indignity of being treated like a drug addict when seeking help for chronic pain understand some of the reasons why. This understanding is the first step to bringing about change.

New Times, New Challenges: Law and Advice for Savvy Seniors and Their Families by Kenney F. Hegland and Robert B. Fleming.

I need to tell you that Kenney is my spouse. In this helpful book, a law professor and elder lawyer team up to offer practical and readable advice on problems facing the boomer generation who are getting ready to retire and/or have to deal with aging parents. Although the authors discuss emotional topics such as elder abuse, hospice, living wills, remarriage and death in the family, the book is entertaining and witty. One of my favorite parts of the book encourages you to write a letter to your family about issues relating to death: do you want everything possible done to keep you alive, how much pain medication, how to divide your possessions and your wishes as to a funeral. The letter can be used to start a family conversation. For more information, see the related website: www.heglandlaw.com and blog: heglandlawblog.com

Remember always consult your doctor before you change your medical regime. Send me your tips at bsattler@cox.net

In the previous journal, I asked readers to send me their tips which I would use in future columns. Shortly thereafter, my computer crashed and I lost the tips I received. I apologize to those who sent tips. Please resend them and I will try again. Thank you.

The TMA Equipment Exchange

Darian Vietzke

Please participate in the TMA Equipment Exchange on ***www.myelitis.org***. You will see the link to the Equipment Exchange on the column of links on the main page of the TMA web site. I have been assisting the TMA Board in developing and offering this program to all individuals affected by TM, ADEM, NMO and ON and their families. The program is intended to assist our community in exchanging surplus equipment with each other for the cost of shipping only. If you are like our family, we have several pieces of equipment that have been outgrown by our son, Jason, who has had TM since ten months of age. We have donated some of his equipment in the past to other organizations, but we are glad to now have another option to share this equipment with others affected with the neuroimmunologic disorders and their families.

We encourage all of you to begin to list your equipment as soon as possible. The more equipment that is listed, the more individuals in our community will be helped. If you have any questions as you begin to use the program, please use the help link on the equipment exchange web site. Thank you for your support,

Instruction Sheet

The TMA equipment exchange is explicitly for exchanging free equipment except for the cost of shipping only. How the cost of shipping is divided is agreed upon by the individual(s) donating the equipment and the receiver(s). Selling of an item is explicitly disallowed.

To list an item(s) to exchange, first follow the on-line instructions to register as a new user and then use the on-line instructions on the Member Area tab to list your item(s) to exchange. Note that several fields can be completed after an item is exchanged. This information is being requested in order to gather statistics to request grant funds to assist in covering shipping costs when exchanging items in the future.

If you are looking for a particular item, follow the on-line instructions to view current ads. Once the item is found, contact the donor (lister) using the on-line instructions to discuss specifics of the item, discuss how to exchange the item if it matches what you are looking for, and how the cost of shipping is to be managed.

Any item inappropriate for exchanging will be removed by the site administrator. To report any item that is inappropriate, please send an e-mail to ***exchange@myelitis.org***

Items exchanged via this site are not tax deductible. Any questions regarding taxes should be directed to your tax accountant.

If you have items you wish to sell and donate a percentage to the TMA, please click on the related link on the front page to use eBay Giving Works.

If you have any comments or questions regarding the TMA Equipment Exchange, please send an e-mail to ***exchange@myelitis.org***. Thank you.

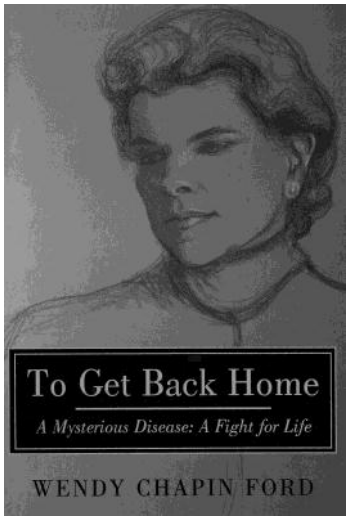
We've made our website talk! ReadSpeaker Added to www.myelitis.org

ReadSpeaker is an innovative program that transforms text into speech. We added ReadSpeaker to our website to facilitate access to information for people who have visual impairment from Optic Neuritis, Neuromyelitis Optica or Multiple Sclerosis. Also, for thousands of people who visit our web site seeking information and support, English is not their first language. Listening to the text could make it easier for people to understand this critically important information.

It is very easy to use; no plug-ins or downloads are required. To activate speech on a web page, all you have to do is look for the "SayIt" icon on the page and click it.

All of the text from the article will be read to you and the speech quality is excellent.

Book Reviews



To Get Back Home
A Mystery Disease: A Fight for Life
Wendy Chapin Ford
iUniverse, Inc. 2009
www.togetbackhome.com

I hadn't met Wendy and she wasn't a member of The Transverse Myelitis Association. I am signed up on Google Alerts to receive any 'hits' for ***transverse myelitis***, ***acute disseminated encephalomyelitis***, ***neuromyelitis optica***, and ***optic neuritis***. I received an alert that Wendy had written a book about acute disseminated encephalomyelitis. I immediately ordered it. I was amazed and thrilled that someone had written about their experiences with ADEM.

To Get Back Home is Wendy's story about getting ADEM, the challenging circumstances surrounding her diagnosis and decisions about her acute treatment and her arduous therapy and difficult recovery. Wendy does a masterful job of chronicling this experience, made even more difficult by the fact that from early in the onset of her disorder until over a month into the acute stage, Wendy was in a coma. She employs the information provided to her by her physicians and other medical professionals, her family members and her many friends to describe and explain this period of time through which she has absolutely no memory.

Wendy does become alert and engaged while she is going through her rehabilitative therapy at Spaulding Rehabilitation Hospital in Boston. Wendy's motivation and determination throughout her therapy emanates from her primary goal 'to get back home.' Wendy is paralyzed from the acute attack. Her therapy is focused on regaining her ability to walk. Wendy is driven by the desire to walk out of the hospital with her husband and children.

Wendy's story is a very personal story, and yet her descriptions of events are quite objective and informative. Her story is very emotional and very inspiring. There is so much that happened to Wendy that I am choosing not to describe for you in this article, because I believe you have to read it for yourself to appreciate what a remarkable person she is; and just how incredibly resourceful, adaptive, and resilient human beings can be.

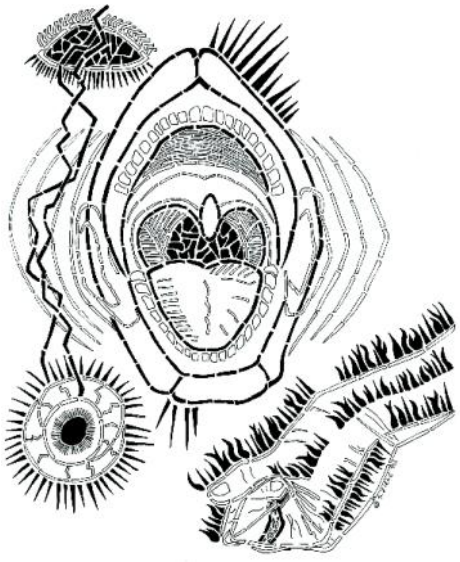
What I will tell you is that I did contact Wendy after reading her book and we have spoken many times since. Getting to know Wendy has only reinforced my sense of what an inspiring and wonderful person she is. ADEM is a really horrible disorder. From my very anecdotal experience, it appears to be particularly horrible for adults who get this disorder. Wendy's first attack was quite severe; she became paralyzed and she was in a coma. Wendy experienced a subsequent attack years after the first episode. Wendy has been told by her physicians that her first attack was ADEM and with her second attack, she was given an MS diagnosis. Her medical professionals have explained to Wendy that her first attack was ADEM and her second attack was MS. In Volume 9 Issue 1 of the TMA Newsletter, I discussed an article published in 2007 which proposes diagnostic criteria for pediatric ADEM and MS. The article does include diagnostic criteria for recurrent ADEM. After Wendy's second attack, her physicians put her on one of the MS drugs. Recently she was taken off of this drug after going for years without another attack. I have no doubt in my mind that if I put Wendy's case in front of a room full of the world's leading neuroimmunologists, I would be entirely entertained for hours by the debate that would ensue about what has happened to Wendy. Does Wendy have MS (both the first and second attack) or does Wendy have recurrent ADEM or did Wendy have an ADEM attack and then a MS attack? This is not an academic discussion about classification. This is a very real concern, as diagnosis determines treatment, and as we have learned from the NMO case, for a person with ADEM, the MS drugs might not be effective.

Beyond Words: An Artful Expression of Chronic Pain and Grief
Susan Zieke

In my more than 16 years of experience in The Transverse Myelitis Association, among the greatest suffering I see in people involves the horrible combination of symptoms that people have with severe depression, nerve pain and fatigue. I often think about this combination as the triumvirate of suffering. Whether one of these symptoms is a cause of the others or that they act on each other to create the

consummate horrible whole is sort of irrelevant to the sufferer. They just want for the suffering to end … however that might happen. Life loses meaning, life loses purpose and the will to live evaporates as each day starts and ends with the same emptiness, despair, hopelessness and suffering. I can hear it in people's voices after being on the phone with them for just 30 seconds. The suffering people experience from these symptoms is something entirely and qualitatively different from what people experience from paralysis or bowel or bladder dysfunction. All of these symptoms really suck; this collection of agony just exists in a horrible realm all its own.

Frustration



My pain

And so, Susan Zieke found her existence in this realm shortly after she got TM. Susan was an artist whose expressions took on the character and magnitude of her suffering and despair. She began to do pen and ink drawings which reflected her journey through this really horrible time and experience.

Susan has collected this artwork into a book she has titled Beyond Words. For those of you who share this experience with Susan, you will see her struggles in each of these drawings. Her feelings are laid bare for all to experience. Through her artwork, Susan found her way to a healing process.

Susan lives in a very small town in northern Wisconsin. Her attack was at the C2-C6 level and she was fortunate that her symptoms came on slowly and that she received appropriate treatment at the onset. She is able to walk but uses a power chair to cover longer distances due to significant motor weakness.

Susan wants to make her book available to others in hopes that it will help validate their feelings. As Susan expressed, "they can show it to others and say this is how I feel." Susan is making a $5 donation to the TMA from every book sold to one of our members. You can order Susan's book by contacting her at: ***beyondwordsartfulexpression@gmail.com.***

The Will to Walk: Journey of Recovery from Paralysis

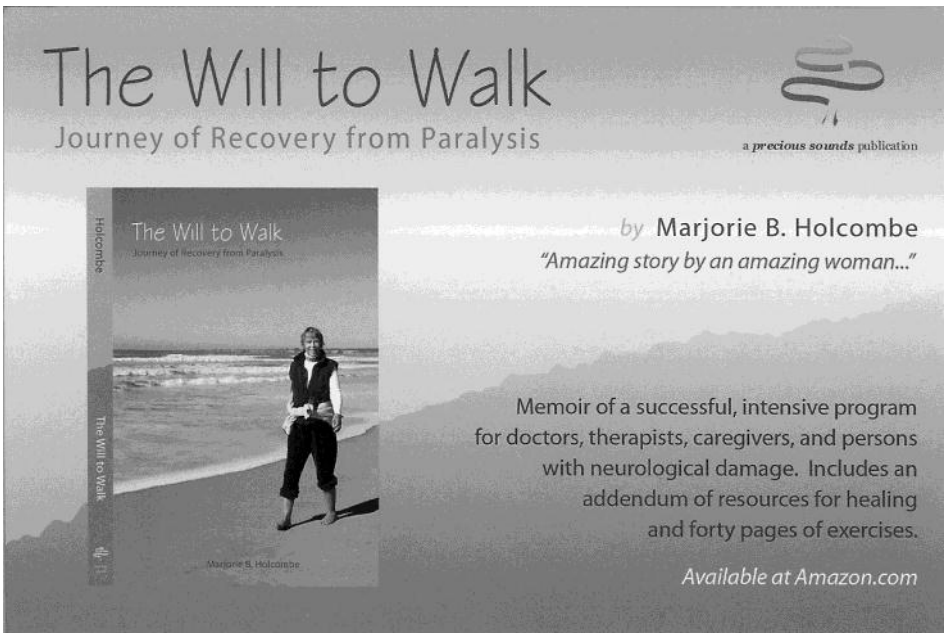
Marjorie B. Holcombe

Marjorie got TM in April 2007 at the age of 68 upon the completion of radiation and chemotherapy for a cancer diagnosis she received the previous January. Marjorie believes that it was these treatments that were the cause of the inflammatory attack in her spinal cord. Marjorie's book chronicles her journey through the onset of TM, her medical treatment and her rehabilitation program. For any reader who has received a TM diagnosis or an ADEM or NMO diagnosis, much of this journey will feel very familiar.

There is much about Marjorie's experience that is, however, quite compelling and unusual. Very early in her writings, she expresses something of her nature and philosophy that serve as a foundation for the directions she will take in her recovery process:

During much of my life I have often felt protected and guided by forces beyond my understanding. Whenever I needed help or felt threatened, events would synchronistically occur for my benefit.

I suppose I had lived under the illusion



that because I sincerely involved myself in a spiritual striving and practices – such as meditation, prayer, study, helping others, loving nature, honoring my body with exercise and nutrition – I was protected from traumatic dangers. That is, in striving to be a 'good' person I had created the foundation for a good life. Misfortune and illness were for others. Not for me.

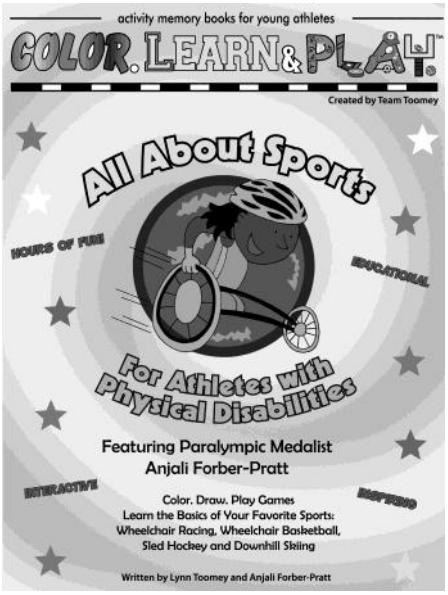
But events were to test the truth of this naive conviction.

While Marjorie experiences many of the same issues most people have during their acute therapies and their rehabilitation which begins as an inpatient in a rehabilitation hospital and then evolves into months and months of outpatient rehabilitation therapy, Marjorie's experience takes an early detour into the world of alternative and complementary medicine. While she becomes increasingly frustrated with her experience with the traditional rehabilitation program, she remains a participant throughout. She does, however, take it upon herself, with the great support of family and friends, to design and enlist the guidance from specialists in alternative, complementary and non-western medicine. Marjorie combines the advice and guidance of these specialists with a program she designs for herself with the aid of professional trainers and caregivers that she hires to live and work with her in her home.

Marjorie systematically chronicles her approach in using these alternative rehabilitation methods. Her book defines each of these methods and offers great detail about their use, as well as references to the specialists who offered their services to her. She also offers an evaluation of the effectiveness of these approaches. Additionally, she includes many pages of description of the specific exercise methods she employed in her very regimented and rigorous rehabilitation program.

Marjorie takes the advice of a Chinese medical specialist very early on in her rehabilitation that if she is willing to devote 6 to 10 hours of intensive physical therapy and exercise every single day that she will recover her ability to walk. Marjorie hires her own personal trainers and caregivers and sets out on a program to engage in this incredibly intensive program. Her devotion, her will, her discipline and her drive are remarkable. The support she receives from her caregivers, both professional and personal, is exceptional. And Marjorie does regain the ability to walk.

Marjorie's book explores the very personal, social and spiritual journey from onset to recovery that she experiences after her TM attack. She explores the complexities of her relationship with her partner, David; the complex social interactions between family, friends and caregivers, and the various and diverse relationships she has with the providers of both traditional and non-traditional medicine. Marjorie's exploration is a wonderful description of the process she engaged in to recover from paralysis. It is also very much a personal attempt at making sense of one of the most challenging and difficult experiences a person is going to have in their lifetime. Her book and the description of her experiences are thoughtful, detailed and inspirational.



Anjali Forber-Pratt
Coordinator of Online Programs,
Leadership & Policy
Doctoral Student,
Human Resource Education
Athlete, US Paralympic Track & Field
Team & University of Illinois Wheelchair
Track & Road Racing Team
Athlete Ambassador for The Hartford

My name is Anjali Forber-Pratt, and I am a member of The Transverse Myelitis Association. I am also a Paralympic medalist and an athlete. I just wrote and launched an educational kids coloring book about disabled sports, called **Color Learn & Play: All About Sports for Athletes with Physical Disabilities.**

I was adopted from Calcutta, India when I was 2.5 months old, arrived in the United States as a healthy baby, and then two months after being here in the states, was diagnosed with transverse myelitis. I was paralyzed from the waist down.

I was often labeled as the kid to be left on the sidelines. Having transverse myelitis as a kid, there weren't other kids with disabilities like me -- the closest I got was finding other kids with spina bifida and spinal cord injury. I remember the disbelief of meeting another adult who had transverse myelitis, because I was so used to saying to other people, well it's kind of like a spinal cord injury, but you've probably never heard of it. Sport taught me that regardless of abilities or disabilities, sport unifies us all. Sport provides an opportunity for individuals to come together regardless of race, political background, ability status or gender. Sport is unique in that it transcends these boundaries and barriers imposed by society and allows for the focus to be on the activity itself, the sportsmanship, the finish line, or the end of the match.

A bit about the project So this coloring book project... most doctoral students get excited about article publica-

tions, and then, there's me, who gets a coloring book published! I've never been one for being "traditional." The coloring book project started about two years ago, and I am so excited to now be able to tell people that they can place their order to learn ***All About Sports: For Athletes with Physical Disabilities.***

As a kid, there were very few books out there that I could relate to, that spoke to me as a disabled athlete. The few that do exist were largely outdated. One of my passions is educating kids about the possibilities that exist in the world, kids with and without disabilities. Obviously, I want to educate kids with disabilities about the sport opportunities that exist for them, but more than that, it is about creating awareness in all kids about the Paralympics, about disabled sports, about this whole movement. And so, the idea was born.

I've always been a firm believer that the universe works in certain ways where things happen for a reason. I had always wanted to do something for kids, but had no idea that it would be an educational coloring book. A few years ago I started working with a publicist in Massachusetts, my home state, and the energy and excitement around my story was contagious. Through a friend, I was put in touch with the woman in charge of this organization called Color, Learn & Play. They specialize in educational coloring books for young athletes and were excited to tap into this niche of disabled sports. We pieced together the activities, co-wrote the story and the idea became a reality. With any project such as one like this, it is easy to get wrapped up in the excitement and to forget that you may fail, that it may not come to fruition.

But for me, failure has never been a good option. With persistence, patience, and sheer determination, the coloring book is actually here!

Why the coloring book? Why not? I hope to educate kids with and without disabilities, educators, therapists, doctors about the Paralympic movement, about the benefits of leading healthy, active lifestyles, and the options available to people with disabilities. I also see this as a way to start to leave my legacy or my mark on the world. I would not be where I am today if it weren't for the tremendous role models, mentors and coaches in my life. This is one small way that I can pay it forward and help to light more fires within other young aspiring athletes.

The coloring book is a 35 page activity book that tells my story, and showcases four sports: wheelchair racing, wheelchair basketball, sled hockey and downhill skiing, as well as educating about the Paralympics and disabled sports. If you are interested, you can order the book on my web site: ***www.anjfp.com***

How does this relate to your other work - in sports, academia, opportunities for kids in developing nations? My vision, which I wrote about several years ago, is a world that is physically and socially accessible and accepting of all individuals with disabilities across all domains of life including education, employment, sport and healthcare. My slogan is: Dream. Drive. Do. Each project I work on has it's own unique mission in and of itself, but keeping this overarching vision in mind is what helps me to see that everything I am doing in my academic work, outreach work, personal life, is connected. Each piece is a different perspective of that. And actually, as I'm sitting here writing this, I think my vision has actually grown beyond just that to truly be a world that is physically and socially accessible and accepting of all individuals including but not limited to disability. I see my work as helping people, people who are left on the sidelines because of "differences" whatever they may be, and helping them to develop and see potential within him/herself. That's my passion, that's my personal mission.

Scrambled Leggs on Kindle and a Facebook site for Women

Sally Franz

Scrambled Leggs...a snarky tale of hospital hooey, the humor book about TM, is now out on Kindle. Just go to Amazon, Kindle Store and type in the book title or Sally Franz.

Are you a woman with TM, or a woman caregiver (boy, is that redundant) for someone with TM? And do you have a wicked sense of humor? There is now a place on Facebook just for you: to laugh, feel safe with your "stuff" about TM and to creatively whine, kvetch and bemoan. It is by invitation only, and YOU are on the list! So, please join us. Just "Friend" Sally Franz and she will bring you into the group.

There are several Sally Franz accounts ... this one has pink, green and blue confetti coming out of her head. And please spread the word to other women in this exclusive club.

Help Wanted: Keeping Our Membership Information Accurate

By doing something as simple as keeping your information accurate in our records, you are helping to save the TMA money; funds that can be used for research or to support symposia or the TMA Kid's Camp.

In addition to asking people to take personal responsibility for keeping address, phone and email information updated and accurate, we are seeking help from our support groups in this important effort. We currently have a number of support groups who regularly contact their membership in order

to confirm the accuracy of their information. For instance, the TM support groups in Canada, India, Germany, Italy, South Africa, Australia and New Zealand, Ghana, Scotland and the UK TM Society regularly check their membership information. Please consider getting involved in this important activity! If you have a flat rate long distance calling plan and internet access, you would be able to easily reach all of the members from your state or country to help verify their information. You would be helping the TMA to save valuable resources, and you would be offered the wonderful opportunity to make connections with the very special people in our community. As our international postage costs are so high, we have a critical need for this work to be done in our support groups outside of the United States.

If you are a support group leader and are involved in a mailing to your state or country members, please be sure to let us know if you are made aware of any information changes. You can send this information to Sandy Siegel at ssiegel@myelitis.org or to: 1787 Sutter Parkway, Powell, OH 43065-8806 USA.

If you are interested in helping us, please get in touch with Sandy Siegel or Debbie Capen at dcapen@myelitis.org or (951)658-2689. Even if you do not have a support group in your state or country, but would like to help us with this work, please get in touch. We would be grateful for your assistance.

The TMA Membership Directory and Privacy on the Internet

The information we provide on our web site and in our publications to our membership is one of the most important functions of The Transverse Myelitis Association. When you share your information in an In Their Own Words Column, you change lives. I have no doubt about this, because I hear from people every day who are inspired and informed by these writings. The access our support group leaders provide to people in their communities is invaluable. To know that you are not going through this experience alone or to find support and information in your community is truly a blessing for people.

Sharing information in our publications and on our web site is a selfless, kind and generous act, and we are all grateful for your participation. It is also very important to understand and accept that once this information is posted on our web site, it is available to anyone who has a computer and internet access across the globe. This ubiquitous access is the incredible value and also the bane of the information technology age.

So, we want and need for you to be generous about sharing this information, but we also want for you to be informed and judicious about making these decisions to share information. If you do not want to be found in a web search or you do not want for your information to be identified in a web search, please do not write an article for the newsletter or journal and please do not volunteer to be a support group leader. In addition to the information in our publications, it is important to bear in mind that any postings you put on a message board or in a list-serve group can also be accessed through a web search. It is almost always the case that if you are wanting anonymity in your life, the less you put out there electronically, the better, and that includes email messages, because once you hit that send button, you have no control over what the person does with that information on the receiving end.

It is also critically important to bear in mind that The Transverse Myelitis Association does not put membership information on our web site or post it electronically anywhere. We publish the directory in paper copies and we mail these directories only to our members who are listed. We send electronic copies of our member information to the people who do our mailings around the world, but they only receive the information for the people for whom they do the mailings. They do not receive the entire membership database. We expend a great deal of effort in protecting your information and limit to the extent possible, the electronic versions of this database.

If you want privacy, we do what we can to help you achieve that end. Please help us by making informed decisions in regards to what you submit for publication and what you post on the web site on our message boards and in the list serve groups. The TMA functions so effectively as a support network, because so many of you are willing to share and to help others. We urge you to continue to do so; we depend on your willingness to do so. But we don't want for you to participate in this sharing, if this activity is going to compromise any concerns you might have about privacy. Be smart and be realistic about how the internet works and what is private and what is public about the internet.

The TMA Newsletter and Journal Archives

The TMA is currently publishing a journal (winter) and a newsletter (summer) every year. When people sign up for membership in the TMA, they receive a packet of information which contains the most recently published TMA Journal or Newsletter. We encourage people to read the previously published newsletters and journals. They are an excellent source of information about the neuroimmunologic disorders, both through articles written by medical professionals

and by people with these disorders and their family members, which describe their personal experiences. Through these publications, you can also learn about research and clinical trials, the TMA, awareness and fundraising efforts, and the support groups around the country and around the world.

All of the newsletters and journals are archived on our web site; you can find them under the link 'newsletters' on the main page of our web site or you can type www.myelitis.org/newsletters/index.html into your web browser. You can view the newsletters and journals as they were published by selecting the PDF files from the column on the right, or you can view them in html format from the column on the left. The html files include an index which makes it very easy to find articles covering specific subjects. Additionally, Jim has installed a search engine for the entire TMA web site, which allows searching for specific subjects. Topics may be searched in the newsletters and journals by using the search engine.

If you have difficulty in finding information about any topic on our web site, and the search engine does not provide you with the results you were seeking, you should always feel free to contact Jim for assistance. You can send Jim a question or a request for help at jlubin@myelitis.org

Medical Advisory Board

Support Groups

About MobileWOMEN.org

We at mobileWOMEN.org believe that women with disabilities steer their own lives. Empowered by knowledge, we are having children, pursuing careers, competing in professional sports arenas, becoming community leaders, and living life to the fullest!

Now at mobileWOMEN.org there is a place for us to unite and ask questions, as well as share ideas, insights, and experiences. Most of all, this site is a place where we can learn from each other.

mobileWOMEN.org is the creation of women in wheelchairs who were having difficulty finding answers to their questions about health, fashion, and other topics. Our mission is to bring together current and accurate information on issues of interest to our community.

mobileWOMEN.org includes a "community" link to an interactive forum where women with mobility disabilities can post questions, comments and communicate with one another. Also, this feature allows women to upload their own personal photos and video clips as a way to educate and inspire others through their stories.

We are ecstatic about providing a site where knowledge will be expanded and success will be celebrated, thereby broadening the horizons for women in wheelchairs all over the world! We are thrilled to be a part of The Transverse Myelitis Association and invite you to visit us and share at ***www.mobilewomen.org***.

Wendy Crawford, a professional model who sustained a spinal cord injury in 1984, resulting in quadriplegia, spent

years of frustration not being able to find answers to what seemed to be simple questions regarding everyday women's issues for individuals in wheelchairs. She decided to take action by creating a website to address these topics. In 2002, her vision became a reality - mobileWOMEN.org was launched and has had loyal readers and interactive participants ever since.

mobileWOMEN.org is a unique online magazine where women in wheelchairs can find answers to questions about health, sexuality, parenting, fashion, and other topics that are not typically addressed in women's mainstream media. The mission of mobileWOMEN.org is to bring together current and accurate information on issues of interest to the disability community and to enable people to share and learn from one another.

The TMA is equally thrilled to establish this wonderful collaboration with Wendy Crawford and the other women from mobileWOMEN.org. Wendy and her organization are also available on the TMA support group page to offer a support network for women and a resource for women's issues.

Support for Women with Rare Neuroimmunologic Disease during Pregnancy

For the past five years I have been involved with the TMA in providing support for women who are pregnant or considering pregnancy while also suffering from transverse myelitis or one of the other rare neuroimmunologic diseases. It has been my pleasure and privilege to communicate with several women via telephone and email and provide information, direction, and general support. I have developed a small network, including neuroimmunologist Dr. Ben Greenberg to try and answer the questions I receive from women all over the world. I am looking forward to continuing this endeavor and hope anyone who feels the need will not hesitate to contact me.

Donna Chattin, RNC-OB, BSN
donna_chattin@comcast.net

For TMA Members who have the ADEM Diagnosis

One of the advantages of your Transverse Myelitis Association membership is that you can connect with others who share your experiences with ADEM. Because ADEM is so rare, that connection can be a vital resource for you. You will discover that there is a community of people who understand your medical condition like no one else possibly can. They can offer you and your family support. They can expand your friendships. And we hope that some day soon, this community can help to connect you to clinical trials and the latest ADEM research.

It's amazing what doors open to you when you become a part of a community.

Are you an adult with ADEM? Does your child have ADEM? We can connect you with others facing the same challenges as you.

It all begins with your TMA membership. If you are new to the TMA or your contact information has changed, it is essential that you provide us with your complete and accurate information.

Please send the following information to Barbara Kreisler:

The name of the person who has ADEM if an adult;
If the person in your family with ADEM is a child, please send the parent's names as the contact;
The street address, state or region, country and zip or postal code;
A phone number, a mobile phone number if you have one and an email address;
The name of the person who has ADEM, their year of birth, their gender, and the year they got ADEM.

You can send the information via email to BarbaraKreisler@verizon.net or you can call Barbara at (703)369-3063 or (571)436-9035 (cell). We look forward to hearing from you.

Thank you!

Barbara Kreisler
ADEM Support Group Leader

http://unanuevaprimavera.blogspot.com/

Why It Is Necessary To Talk About NMO

Amparo T Nisembaum. Alas, MSc
Tiberias Illit, Israel

Neuromyelitis Optica (NMO) syndrome or Devic's disease is an inflammatory and autoimmune illness of the central nervous system, classified within "rare" diseases and affects mainly young people. It is characterized by attacks of optic neuritis and myelitis, being able to produce blindness, great neurological disability and even the short term death.

Currently there is no cure for NMO and there are no treatments to completely and effectively eliminate the possibility of multiple attacks. People are placed on immune suppressants to either increase the chances that an attack can be avoided or to decrease the severity of the attack should it occur. Therapies are centered on the treatment of the acute attacks, the medical prevention of the complications and rehabilitation. For these reasons, the care of Devic's patients is very complicated and challenging for the medical professionals and there are significant challenges for the families that have a loved one with NMO.

My name is Amparo T Nisembaum

Alas. I am 47 years old and was diagnosed with Neuromyelitis Optica in 2008. My first attack of optic neuritis occurred in 1990. I have since had repeated attacks of acute myelitis. I have worked as a researcher in a clinical laboratory since 1986 and have a Master's degree in Biochemistry.

I have created a blog called "Una nueva primavera," because I want to write about this rare disease. ***http://unanuevaprimavera.blogspot.com/*** My blog is in Spanish as I was born and raised in Cuba.

My blog is dedicated to helping people understand this condition and to provide information about the primary signs of this disorder, the recent molecular biology and immunology research on NMO, the possible new treatments, and the many difficult symptoms of NMO, including fatigue and the changes in the behavior of patients.

I also use my blog to talk about and to share my personal experiences with this disease; my diet, my participation in sport, my experiences with depression, the methods I use to control stress, and the treatments I use to treat chronic pain. My blog also includes many of my photographs.

I hope that by sharing my blog with others, it will help people with NMO to better understand their own condition, and I hope that this site is helpful particularly for the many Spanish-speaking peoples of the TMA who have the NMO diagnosis. I also hope that my blog is an inspiration and motivation to the many clinicians and researchers who are devoted to improving the quality of lives of people with this very challenging disorder. So many people with NMO are young; finding the answers to this difficult condition will ensure that all of us are able to make our contributions to future society.

Tucson TM Support Group Meeting and Wedding

Barbara Sattler

Every third Saturday of the month, the five women who comprise the Tucson TM Support group meet for lunch and conversation. The conversations sometimes involve the chatter of daily events and always the recounting of the triumphs and tragedies of the last month. The meeting is not complete without a discussion of our meds changes, our levels of pain and which doctor, insurance company or other health care provider did us wrong.

Our June meeting was much the same. But when our meeting was completed, we adjourned for the marriage of one of our members, Lauren.

Lauren is a very attractive woman who appears younger than her age (which I am not going to reveal). She has lovely multi-colored locks and a pink

wheelchair. I hope someday she will write her story for the TMA Journal as she has endured much pain, horrible medical care and callous treatment from doctors. Like many of us, she has suffered unnecessary pain because of the fear doctors have of prescribing medication we need to get through our day. In spite of this, Lauren is bubbly, sweet and fun to be around. She is also very brave.

When Lauren advised us she wanted to marry, but was having trouble planning the wedding due to logistics and family issues, the group sprung into action. One member volunteered her home which has a lovely outdoor garden area backing up to the desert. Another had credentials to perform the ceremony itself. A cake and photographs were arranged. All supported her decision. A few guests were invited and Lauren's mother heard the ceremony on a speaker phone. Although it was an extremely hot day, as is usual for that time of year, for just the few minutes of the ceremony, there was a light breeze ruffling through the air. A deer walked by just as the ceremony was concluding.

None of us had met Michael until the day of the wedding. As soon as we met him, we all loved him and appreciated his heartfelt thanks for getting the wedding together. We will never forget the way he shared details of how he and Lauren met and the obstacles they overcame to get to this day. Lauren may take some time to forgive him for a little TMI but it was clear to us, that Michael appreciated and loved Lauren. We wish them both much happiness.

Australia and New Zealand

Spinal Injuries ASSOCIATION
Empowering people for 50 years

Hello to all in the TM Community. I am Louise Remilton from Brisbane Australia. I was diagnosed with Transverse Myelitis in December 2004. My story and experience with TM is probably quite unremarkable amongst those who have had to deal with the disease, however on a personal level the pain, disability and loss of function has, without doubt, been the greatest challenge I have ever faced. Despite having the continuing unconditional support and love of my family, there was always an overwhelming sense of isolation and fear that I believe is due to the general lack of knowledge and understanding of the condition in the wider community. A couple of years ago I came across the TMA web-site quite by chance. Since that time, I have become a member of the TMA and have, at last, found a sense of solidarity that has made a positive and lasting impression on my life.

In May 2010 I attended the Transverse Myelitis Awareness Day in Brisbane and for the first time actually met others with this condition. I also saw a presentation by Sandy Siegel and Dr. Benjamin Greenberg via video conference which offered an invaluable insight into the disease, the treatment options and the progress of research on TM.

In September this year, I had the great privilege of attending the 2010 Rare Neuroimmunologic Disorders Symposium in Dallas, Texas. The Symposium combined detailed presentations from a wide range of specialists about all aspects of the disease and the growing awareness and understanding within the medical and research community. I also had the very special opportunity to meet the dedicated team on the TMA board along with many individual members I had come to know as friends on the TMA message forum.

I cannot change the impact TM has had on my life, but I would be honoured to be able to make any contribution I can to assist others who face this disorder. I will never forget the gratitude and sense of relief I experienced when I joined the TMA and have come to know others both here in Australia and overseas who have faced similar challenges. I truly believe that knowledge, support and understanding by others with TM will help to reduce the fear and isolation that most people experience on initial diagnosis. This can't help but contribute to improved outcomes when combined with the professional care and treatments offered by the medical community.

I look forward to continuing my association with the TMA and seeing an improved support network here in Australia now that there is a formal connection between the TMA and the Spinal Injuries Association of Australia.

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Transverse Myelitis Day for Australia and New Zealand

This year's Transverse Myelitis Day was marked by a special event, held at the Association's Brisbane office. About 20 members joined Association president David Riley, CEO Mark Henley and other staff to take part in a video link with the US-based Transverse Myelitis Association (TMA). TMA President Sandy Siegel, whose wife has TM, talked about the history of the organisation, plans for the future and the upcoming "2010 Rare Neuroimmunologic Disorders Symposium" in Dallas and the Retreat weekend for teens and young adults at Victory Junction Gang Camp in North Carolina.

Dr. Benjamin Greenberg from the University of Texas Southwestern Medical Center offered a presentation on the current state of research in these rare neuroimmunologic disorders. Guests had an opportunity to engage in a question and answer session and then enjoyed lunch and a chance to share experiences. Bernice Quinn, the Association's Member Networks Coordinator and organiser of TM Day, said the event was a huge success. "The feedback from those who attended was amazing – it's not often you get the opportunity to hear from a world-renowned expert and be able to ask him questions and also meet with others who have this rare condition," said Bernice. The event was also an occasion to announce the newly-formed partnership between the TMA and the Spinal Injuries Association, which will act as the TMA's affiliate in Australia and New Zealand. Members who would be interested in attending a regular meeting in Brisbane and/or would like a copy of a DVD of the day's presentation (including footage of Sandy's wife Pauline and her service dog Kazu) should contact Bernice via bquinn@spinal.com.au or 07 3391 2044.

Massachusetts Support Group

Cindy Walker

My journey with TM began Feb 7, 1995, when I was 14 years old. It hit me like it does most everyone else that's been afflicted with TM, without warning or hesitation. I was at a dance class and felt what I thought was a pulled muscle in my lower back. The next morning I woke up with no feeling or movement from my belly button down. My mom called an ambulance and we sat in our local hospital emergency room for 12 hours. Finally, the doctors decided to send me to Children's Hospital in Boston which is a 45-minute drive from where we lived. I think I was at Children's for seven days before it was determined I had TM. I was told I would never walk again and that we needed to decide where I would complete my inpatient rehab and where I would learn to live life in a wheelchair.

For a long time I thought that this moment defined who I was. I kept hearing those doctors tell me I would never walk again, and I didn't know what that meant. I had visions of my 20-year-old self, or my 30-year-old self and those visions did not have me wheeling around in a wheelchair. I had a lot of questions that a lot of doctors and rehab specialists could not answer. I spent a lot of time trying to rethink my life, trying to envision where this new person and her wheelchair fit in. I didn't want her here. I didn't want to acknowledge I was "different" or "unique" or "special" or worst of all "disabled."

My rock in all of this was my mother. She was diagnosed with Diabetes when she was 12, after a misdiagnosis and treatment of seizures. She knew what it was like to live with certain restrictions and limitations. Before she had to retire in 1991 due to her health, she was a Social Worker and was in charge of a division of Mass Rehab in Taunton, MA. She believed in me when I didn't believe in myself. She said from the start of all this, that instead of dancing I could play wheelchair basketball. She said I would graduate high school and then college and move on with my life. She made me believe that I could do anything.

While I was in rehab our community of family, friends, and strangers came together to transform our home into an accessible house. It was like the show "Extreme Makeover: Home Edition" but without Ty and the TV cameras. A charity event was held in my name to raise money for my expensive hospital stays and expenditures that insurance refused to pay for, calling things like my shower chair a "luxury" item. Two months after returning home, my mom's health started to deteriorate dramatically. She had always been in and out of hospitals throughout my childhood, but she always bounced back. This time was different. She had her right foot amputated and then her left leg. She had a feeding tube put in, she went blind, and our living room became her bedroom. People used to ask me why I thought I was paralyzed. I would tell them that a community would not come together for a 40-something year old woman like they did for a 14-year-old girl. Our whole house was outfitted to make my life easier, and now it was all there for her.

My mother passed away at the start of my 2nd semester in college and I tried to hold onto the person she saw in me. I had promised her I'd finish college and move to LA to pursue a career in TV/film, and I did. After graduation my boyfriend and I drove out to LA and I got a job at Paramount Pictures working in the network TV finance department. I was elated that in spite of my disability I was accomplishing these goals we had set together. Unfortunately I carried around a lot of anger and sadness. I missed my mom and needed to hear her words of encour-

agement. I missed the sounds of her laughter, and I let the words of those doctors telling me I'd never walk again, weigh me down. After living in LA for four years, I moved back to Massachusetts to try and focus on my life again.

When I meet new people they are curious about why I walk with a crutch and brace. When I tell them about TM, they usually comment on how positive I am. I tell them I was lucky that the paralysis didn't creep higher and that I've regained movement and some feeling in my legs. I can't remember what it feels like to run. I can't remember what my mother's voice sounded like. I can't remember what it feels like to dance, and I can no longer hear my mother's laughter. It feels as though the girl I was never existed and I am finally ready to move on.

The next goal I have set for myself is to enroll in a Master of Social Work program and follow in my mother's footsteps. I want to help people to believe in themselves like my mother helped me to believe in me. I've learned that I'm bigger than this diagnosis. I've stopped waiting for a cure for TM. For a long time I ignored my disability instead of embracing it. I've come to realize that the person I am is not defined by my physical limitations.

I'm happy to be an open ear to anyone that might need it. I know this diagnosis comes with a lot of questions and very few answers!

I hope that others of you in Massachusetts are also interested in getting involved. I look forward to hearing from you.

My email is ***cwalker@myelitis.org***.

Pennsylvania Support Group

Greetings from Pittsburgh, Pennsylvania! My husband, Morgan, and I are listed on the web site for TMA support leaders along with a lovely woman, Sue Mattis, from Erie, PA. We have met a few times over the years and have come to be good friends. Sue has fundraised for us to go to Victory Junction Family Camp and the last time she volunteered at camp and was our volunteer helper. She is one special lady.

Each time I read the journal, I wonder why I don't take the time to say something. So here goes something. I want to take this opportunity to let you know that you are not alone. If you need someone to talk to, please call or email us, we will try to help. Morgan and I are parents of a 13-year old girl, Maria, who got TM at 6 months of age. She was paralyzed from her neck down. We have been through a lot over the years and it has been a lot easier with the support of the TMA Officers, and families we have met along the way. So if we can be of help, don't hesitant to let us know.

In the past, we have offered support group meetings at our home near Pittsburgh with one or two folks in attendance. Once we met folks in the Hershey area for a meeting and presentation from Dr. Douglas Kerr on Stem Cell Research. If you are interested in meeting, please contact us. We will work with you to make this possible.

Just remember you are not alone.
Morgan and Pam Hoge
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Virginia

My name is Lydia and I am very excited to introduce myself as a Transverse Myelitis Support Group Leader for Virginia.

My story is similar to many of the TM survivor's stories I have read. In March of 2007, I was 27 and a high-heel-wearing hairdresser, business owner and single mother of a beautiful little girl. On March 15, 2007, my life's train jumped the tracks. Sudden paralysis, mass confusion, X-ray's, MRIs, EKGs, CAT Scans, blood tests; tubes and bags lodged in several locations. Clueless medical staff from numerous hospitals in Virginia, (as well as one in Philadelphia) scratching their heads at my sudden paralysis. I spent two months in the hospital as the neurologists went back and forth with my results. I have a lesion at the C5-C6 level of my spine and a lovely lesion in my brain. Yes, I know... the question was "Isn't it MS then?" At this point, four years into my TM journey, my neurologist still confirms my diagnosis (written in my medical records) as Idiopathic Transverse Myelitis... with a question mark at the end.

After my onset, I looked for positive and uplifting stories within the TM community about people who really make a difference with what they have to offer now. I was blessed to have found some amazing people, who continue to smile, grab the baton and keep running their race. I hope to use what I have learned, from them, in this new opportunity, as I meet my fellow Virginians who have been diagnosed with Transverse Myelitis. I intend to assist in presenting a positive side to what some may label as a sudden and unfortunate life-altering situation. Yes, we all have bad and good days and I have my share. Concentrating on my daughter, what I have to creatively offer and where I can go with it, has been the driving force behind my slow recovery. I would love to start arranging more opportunities for recently diagnosed patients in their 20's and 30's to meet, share and learn more about where they can go from this moment forward.

Thank you for allowing me to begin this new journey with you and I look forward to meeting you in the future.

Lydia Schoepflin
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ADEM, NMO, ON, Recurrent TM, TM or NMO with Lupus, Sarcoidosis, Sjogren's and HIV: Finding Each Other to Share Information and Support

We are trying to assist people who have the very rare neuroimmunologic disorders find each other for the purpose of sharing information and support. We are creating the lists identified below for that purpose. If you have one of these neuroimmunologic disorders and would like to be added to the list and then receive a copy of the list, please send us your information. We only share these lists with people who are willing to be added to the lists.

- **Recurrent Transverse Myelitis**
- **Transverse Myelitis or NMO with HIV**
- **Optic Neuritis**

If you are interested in being added to one of these lists and then periodically receiving a copy of the list, you can send me your contact information either by email or through the postal service. Please send me your full name, complete postal address, phone number and email address (if you have one). Be sure you clearly identify to which list you would like to be added.

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Acute Disseminated Encephalomyelitis (ADEM)

The ADEM list is being maintained by Barbara Kreisler. If you would like to be added to the list, please send your information to bkreisler.imprint@verizon.net.

Neuromyelitis Optica (NMO) or Devics disease

The NMO list is being maintained by Grace Mitchell. If you would like to be added to the NMO list, please send your information to gmitchell@myelitis.org.

TM or NMO and the Rheumatic Disorders (SLE or Lupus, Sjogren's syndrome, Sarcoidosis)

This list is being maintained by Sharon Robinson. If you would like to be added to this list, please send your information to Rufusandchi@yahoo.com.

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Fundraising and Awareness

Helping to Fund the Work of Your TMA

The officers and board members of the TMA are volunteers; they receive no compensation of any kind for their work. There are no employees in the TMA. There are no offices; the officers work out of their homes. In order to facilitate access to support and information, the TMA does not charge membership fees. As TM, NMO, ADEM and ON are rare conditions and our membership is small, it is extremely difficult to raise funds for our cause. We work most diligently to focus our resources on the direct services to our members. We operate exclusively on the basis of the voluntary support of our members. There are numerous ways for everyone to help support the TMA, even if you are not in a position to make a financial contribution. Please consider getting involved in one of our fundraising efforts.

Search the Internet and Raise Money for the TMA You can raise money every time you search the web, at iSearchiGive.com. Make it your homepage and use it to find everything from news on the economy, to mood-lifting jokes (we recommend the latter). The Transverse Myelitis Association gets a penny (or more!) every time you search. Believe it or not, it adds up quickly and best of all, it costs you NOTHING!
Start iGiving at:
www.iSearchiGive.com/TMA

Donate your cell phones You can donate your cell phones to help raise funds for The Transverse Myelitis Association. Go to http://cellphones.myelitis.org

Online Shopping There are numerous online shopping opportunities, as well as sales on eBay which can be made through the following link: http://www.myelitis.org/store.htm A percentage of the sales are donated to the TMA.

Save Gas. Save Time. Raise Money! With over 700 stores in the iGive Mall and access to hundreds of exclusive coupons, free shipping deals, and sales, iGive is the smart way to shop. You'll find everything from daily necessities to special occasion and holiday gifts, at stores you know and love. So save a trip to the mall, and avoid the long lines. You'll never pay more when you reach a store through iGive, and up to 26% of each purchase benefits The Transverse Myelitis Association! Start iGiving at:
www.iGive.com/TMA

Café Press Shop for items with The Transverse Myelitis Association logo to raise awareness and show your support!
http://www.cafepress.com/myelitis

Amazon.com You can shop at Amazon.com for Books, Music, DVDs, Videos, Toys and more.
http://www.myelitis.org/amazon

Music Downloads for any device! Shop for your favorite song or album from our mp3 store powered by Amazon and download music that works with an ipod or any mp3 player.
http://www.myelitis.org/shopmp3

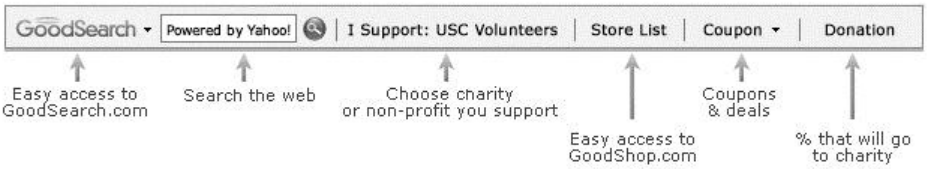
eBay
Now you can sell an item on eBay and donate from 10% to 100% of the final sale price to help support the TMA.
http://www.myelitis.org/ebay

Reading for Rachel
If you are a teacher, a student or a parent of a student and would like to establish the Reading for Rachel Program in your school, everything you will need to get the program started can be found on the Reading for Rachel web site: http://www.readingforrachel.org. All funds received by The Transverse Myelitis Association for the Reading for Rachel Program are used exclusively for research to better understand TM, to find treatments for the symptoms of TM, and to ultimately find a cure. If you are interested in starting the Reading for Rachel Program in your school, you can also contact Cathy Dorocak, Rachel's Mom and International Chair of the Reading for Rachel Program: cathy@readingforrachel.org; (440)572-5574.

Donations by Check
We always welcome and are grateful for a donation to the TMA. You can download a donation form to include with your check from the link: www.myelitis.org/donation-form.htm Please make a check or money order payable to The Transverse Myelitis Association and mail it to:

The Transverse Myelitis Association
Paula Lazzeri, Treasurer
10105 167th PL NE
Redmond, WA 98052-3125

Thank you!



The Goodsearch Toolbar for the TMA

Please add the Transverse Myelitis Association Goodsearch Toolbar. Once added to IE or Firefox, each time you shop at more than 1,300 stores (from Amazon to Zazzle!) a percentage of your purchase will automatically be donated to The Transverse Myelitis Association - at no cost to you (and you may even save money as the toolbar provides coupons and deals as well!). The toolbar also has a search box and each time you search the Internet, about a penny is donated to The Transverse Myelitis Association.

And please pass this along to all of your friends. The two minutes it takes to add this toolbar to your browser can make a lifetime of difference!

Get the toolbar NOW!

http://www.goodsearch.com/toolbar/transverse-myelitis-association

The Transverse Myelitis Association Credit Card Program

Share your passion and donate to our cause with your everyday purchases. We've partnered with Capital One® Card Lab Connect to bring you our newest fundraising program, which helps us earn money effortlessly every day! Just carry one of our custom credit cards (it comes with a competitive rate), and 1% of purchases made with the card will be donated to The TMA. We'll also receive a $25 bonus donation when you make your first purchase. And not only will you be donating to our cause with each purchase you make, you'll be helping to spread the word when people see your unique card, which includes our name and logo, as well as Margaret Smith's artwork as backgrounds.

Apply online via a secure web page at ***www.myelitis.org/creditcard***

We urge you to only make purchases with this credit card that you are able to pay off each month. We would love to receive the benefits from this program without your paying any interest on your purchases.

The Transverse Myelitis Association is an all-volunteer, non-profit organization. We greatly appreciate your support!

Donate your birthday to charity!

Use your birthday to change the world! We all get things we don't need for our birthdays; why not use this special occasion to raise money for a cause you really care about? Facebook has a feature that allows users to create a Birthday Wish and ask their friends to donate to the cause of their choice as a birthday present. Facebook also provides promotional tools, such as emails to friends, status messages, and more. To create your own Birthday Wish, go to ***http://apps.facebook.com/causes/birthdays/new***

In the step "Pick the cause you're raising money for" enter "myelitis" in the search box and select The Transverse Myelitis Association.

Thank you … and happy birthday!

Honor the Children in Our Community and Support the TMA

The Transverse Myelitis Association held a Children's and Family Workshop in Columbus, Ohio in July, 2002. The TMA Workshop focused on children from infancy to their early twenties and included their brothers and sisters and their parents. For most of the parents and children, the workshop represented the first time they had met another child with TM. As TM is a rare disorder, these families often feel isolated in their experiences. The workshop was an incredible opportunity for these families to make connections with others who could offer them emotional support and encouragement.

The workshop offered the children an opportunity to have a fun weekend. One of the many activities they participated in during this special weekend involved working with an art therapist from Chicago, Lori Stralow Harris. With the help of Ms. Harris, the children created beautiful paintings which were constructed into a quilt of courage and hope. The original artwork currently hangs in the Johns Hopkins

Transverse Myelitis Center where it is appreciated by the hundreds of patients every year who are cared for at the Center.

We are very pleased and proud to be able to offer you the children's artwork through Café Press. The proceeds from the sale of these items will be used to fund the many important programs of The Transverse Myelitis Association. We hope you will take the opportunity to enjoy the children's work and to support the TMA.

http://www.cafepress.com/tmagifts

Help Raise Awareness with a TMA Wristband

For the past 3 years, thousands of our members have been helping to raise awareness of the TMA by wearing bright blue wristbands. They have been available on the TMA website and at our symposia for purchase. The wristbands are available in a marbled blue/grey in the adult size and solid blue in the youth size. The youth size also fits women with small wrists. These wrist bands are made with 100% synthetic silicone rubber and debossed with the abbreviations "TM-ADEM-NMO-ON" and "www.myelitis.org."

Many families have purchased these wristbands as party favors for birthday celebrations, fundraisers for raising research dollars, and to just proudly wear every day. Several people have sent us photos of themselves displaying their wristbands at known landmarks around the world. All of the money raised through the sale of the wristbands goes towards the cost of printing and mailing out the information that you receive in newsletters like this one, and for mailing out new member packets for those newly diagnosed with TM, ON, ADEM, and NMO.

The wristbands are inexpensive – only $2.00 each – and you can either order them online at our website, making your purchase with a credit card transaction, or you can mail a check to The Transverse Myelitis Association and when we receive your payment, we mail them to you.

To order online, please go to our website at: www.myelitis.org/wristbands.htm.

For check payments, please mail your payment along with your order request to:
The Transverse Myelitis Association
Paula Lazzer, Treasurer
10105 167th PL NE
Redmond, WA 98052-3125

Specify "for TMA wrist bands"

Shipping charges:
1-5 $1.00
6-10 $1.50
11-25 $5.00

For quantities more than the above, please send an email. If you would like us to calculate your shipping for you, you can send an email to wristbands@myelitis.org and we will tell you how much to send. You can also call Debbie Capen at (951)658-2689 to get your total cost and more information.

Don't miss out on getting your own one-of-a-kind TMA wristband!

The TMA Greeting Card Program

http://www.myelitis.org/cards/

We are thrilled to introduce The TMA Greeting Card Program. The cover of each of the cards is a beautiful water color painting of landscapes or flowers. The back of the cards include the TMA logo, our web address and a description of our Association. The inside of each card is blank and perfect for offering your own sentiments. We urge you to use these wonderful paintings as your regular cards for the holiday season, for thank you and everyday notes or for any purpose.

The proceeds from the sale of these items will be used to fund the many important programs of The Transverse Myelitis Association. As the neuroimmunologic disorders are rare and our membership is small, it is extremely difficult to raise funds for our cause. We work most diligently to focus our resources on the direct services to our members. By using these beautiful greeting cards, you will be supporting the important work of the TMA and also raising awareness about acute disseminated encephalomyelitis, neuromyelitis optica, optic neuritis and transverse myelitis.

About the Card Artwork

Sandy and Margaret Smith are members of The Transverse Myelitis Association from Pittenweem, Fife, Scotland. They are active members of the Scotland Support Group led by Margaret Shearer. Sandy has TM. Margaret is an artist. Margaret has created beautiful paintings of landscapes and flowers. She has donated this artwork to the TMA and we are very pleased to be able to offer you these beautiful greeting cards.

Inkjet and Toner Recycling Program

The Transverse Myelitis Association has partnered with the Funding Factory Recycling Program to collect empty inkjet and toner cartridges. This is an important fund raising effort for the Association.

Please go to our web site at ***www.myelitis.org/recycle/***. Once you register, you can order pre-paid UPS return labels that you put on any box you have. When you fill in the information, use your own name as the "Organization" name, but also, **please use id number 63960 as the beneficiary. This ensures that the TMA will be receiving the benefits of the collected cartridges.** When filling out

the contact information, the form asks for a "title". You can list "other" and put "supporter" for your title. Once the company has your information and you request shipping labels, they will ship them to you to place on the boxes. Once the boxes are filled, you can take them to any place that picks up UPS packages (such as "Mailboxes, ETC."). We appreciate your participation in this important program!

Donating by credit, debit, or gift card

The Transverse Myelitis Association is set up with several companies to securely process donations online using any credit, debit, or gift card with the following logos: Visa, MasterCard, American Express, Discover.

PayPal and Google Checkout are available to process donations from the United States and worldwide. Network for Good is only available to donors residing in the United States.

You can make secure donations online by going to ***http://myelitis.org/donations.htm.*** We greatly appreciate your support!

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We would like to express our deepest gratitude to the persons and the organizations that support the work of The Transverse Myelitis Association. It is through their generosity that we are able to offer the services to our membership; they also make possible the expansion of services to our existing and future members. The following persons and organizations made donations to The Transverse Myelitis Association in 2009 and 2010.

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Kermit and Teresa Kragnes
Marvin and Anne Kurtzman
Andrew and Melissa Lantz
Paula Lazzeri
Beth Lekander
Christopher Lesher Memorial:
- Electric System Operations & Planning c/o Baltimore Gas and Electric
- Patrick and Kelly Flynn
- Charles and Barbara Kleinsmith
- Michael and Kathy Lannon
- Rodney and Mary Pollanck

Bruce Levitt
Jeffrey and Leighann Light
Maureen Littlefield
Gerard and Janet Logan
Richard and Gloria Lombardi
Joe and Jacqueline Long
Charles Lyle
Jim and Helena Lubin
Toni MacIntyre
Melinda Macy
James Maddox
Robert Malecky
Martha Mann
Dana Marcario
Evelyn Marks
Stephen and Mara Marks
Julyette Marshall
Hugh Martin
Albert and Barbara Mast
Joyce Maygers
David and Valerie McCammon

Arlene Messinger
Lawrence and Janet Messinger
June Meyer
Microsoft Matching Gifts
Morton and April Middleman
William Millard
Kelley Miller
Marie Miller
Miller, Monson, Peshel, Polacek, and Hoshaw Law
Paul and Evelyn Miller
Robert and Gail Miller
Charlotte Millford
M.O.M.
Dorothy Monahan
William and Marianne Moore
Geraldine Murray
K. Muston
Domenic and Josephine Narducci
Donald and Marjorie Narducci
Paul and Bonnie Narducci
Nike Matching Gift Program
Herbert and Karen Niles
Niles Family
Stacey Niles
Stephen and Vicki Nye
Lois Osborn
Bruce and Susan Outland
Dexter and Dorothy Packard
Frank and Jo Anne Pajcic
Thomas and Maureen Panattoni
Ronald and Mary Jane Parisi
Pathways Regional School
Kevin and Lori Paul
Mary Ann Pellegrino
Marissa Ona and Edwin C. Pena
Robert and Kristen Penrod
Nancy Penslien
George and Lillie Perdue
Ronnie Perdue
Gary and Susan Perkins
John and Vanessa Pesec
Joanne Petonito
Sally Petterson
James and Laura Pfadt
Bernard and Penelope Pfeister
Doris Phinney
Desire Pignon
Kenneth and Sharon Pipes
John and Shirley Pitts
Robert and Denise Pluhatsch
Irwin and Marcille Pollack
Rob Prentice Memorial:
  Maurice and Patricia Knowlton
Mary Qualtrough
Rebecca Rabalais
Cynthia Hall Ranii
Ronald James Reedy
Johnna Rice
Wilda Rice
Helena Van Rijn
Bruce and Gloria Robertson
C. Darlene and Lynn Rogers
The Roles Family Foundation
Joseph and Lorraine Romangnano
Robert Romps
Paul and Deborah Rosenberg
Kelly Rousseau Memorial:
  Reg and Lynne Rousseau
Lauro and Barbara Rozul
Stephen and Linda Rubarski
Clinton Allen and Ann-Marie Rucker
Edward Ruetz
James and Jenine Ruetz
Robert Ruetz Family Trust
Maria Christou Ruhmel
Grace Sahler
Gary and Marjorie Sanders
Jacinta Dos Santos
Barbara Sattler
Olaseinde and Carolyn Sawyerr
B.D. and K.S. Schechter
Hyman and Perle Schechter
Marc Schechter
Pamela Schechter
Carol Schlegel
Herman and Jeanne Schob
Kim Schob
Eugene and Martha Schramm
Catherine Schuhlein
Katie Hickey Schultz
Doreen Scurlock
Farnaz Sedghi
Ms. Farnaz Sedghi
Renate and Johann Seitz
Kara Seneco
Marvin and Marsha Serota
Betty Shaffer
Jane Shaffer
Helen Short
Dana Campbell-Siegel Memorial:
  Jack and Charlene Kiniyalocts
Daniel and Marcia Siegel
Sandy and Pauline Siegel
Lee and Gail Silver
John and M. Suzanne Simmons
Allan and Astrid Sipos
Richard and Dorothy Skea
Walter and Sabina Slavin
Christine Ann Smith Memorial:
  Gary Benedict
  Leslie and Dorothy Hunter
  Jim and Marilyn Mauldin
  Durward and Cynthia Pariseau
  Jack and Nancy Smith
Cynthia Smith
Robert and Linda Smith
Smithville Emblem Club
Joseph and Jill Snyder
R. Erik and Denise Soderholm
Dawnmarie Souza
David and Kimberly Spach
David and Colleen Spaeth
Robert and Karen Spielman
Eileen Splinter
Eileen Rooney Staab
Michael Stafford
Pat and Harriet Starr
Kenneth and Susan Stachler
Pat Steberl Memorial:
  S.R. Krizan
  Kenneth and Anita Seitz
James and Deborah Stephens
Leo and Phyllis Stevens
Paul and Marjorie I Straley
Theodore and Shirley Sturm
John and Jane Sullivan
Lester and Charyl Suzuki
Jeanette Flemming Sykes
Jim and Jan Tamura
Richard and Virginia Tenney
Terex Corporation
Richard and Anne Thompson
Albert and Patricia Tolle
Doris Trax
Kenneth and Nancy Turbert
Jerry and Diane Vecchione
Patricia Voorheis
Nancy Vroom
Wagner & Zwerman LLP
Calvin and Margherita Wang
Ruth Warren
Charney and Marjorie Weber
Kenneth and Naomi Wegmeyer
Denise Wehrer
Lisa Weiner
James Wells
Wells Fargo Community Support
Western Fire Protection
Steven and Diane White
William Bubba Greene/Greene's Timber Farms
Mark and Maria Williams
Dylan Winesburg Memorial:
  Roger and Trudi Alwine
  Arloa Bell
  Julie Cunningham
  Mark and Teresa Emerson
  Victor and Laura Gran
  Joy Miller
  Kevin and Delenne O'Connor
  Juanita Paulus
  Gerald and Susan Phelps
  Patrick and Kathleen Sapikowski
  Barbara Schmidtendorff
  Dean and Debra Thayer
  William and Kris Whitman
Andrea Winokur
Jerry and Pamela Wiseman
Sheila Woerner
Clarence Virgle Womack Memorial:
  Doris Beard
  Mr. and Mrs. Charles Davis
  Jimmy and Bryan Davis
  Mr. and Mrs. Elmy
  Jerry Hennesse
  Mr. and Mrs. Luke McCormick
  Mr. and Mrs. Wilburn West
  Mr. Mrs. Clyde West
Patricia Woodbury
Henry and Jacquelyn Wray
Helina Hee Jung Yoon
The Young Family
Steve and Connie Yourst
Soheila Zadeh
Richard and Jane Zemba
Ronald Zipfel Memorial:
  Carolyn Polisano
Frank and Geri Zisa
H. Jay and Rachelle Zukerman

The Transverse Myelitis Association 2009 and 2010 Statements of Financial Activities

(in US Dollars) Paula Lazzeri

The following tables present The Transverse Myelitis Association Annual Financial Reports for 2009 and 2010. The TMA (General) Fund column presents all funds received and expended directly by TMA as recorded in the Association's financial account. The Total Donations and Expenses to Benefit TMA column is presented to help convey the total cost of providing TMA member services during 2009 and 2010. This column includes funds/activities reported in the TMA (General) Fund, as well as non-reimbursed expenses paid by members of the Board of Directors. These non-reimbursed expenses also are shown as Donations made by Board of Directors under Revenues.

2009 Statement of Financial Activity

INCOME	TMA Funds	Total Donations and Expenses to Benefit TMA
Amazon.com Commissions	409	409
Children's Camps	5,900	5,900
Donations made by Board of Directors	0	7,436
Endowment Interest	180	180
General Donations	373,064	373,064
iGive.com Commissions	115	115
Interest	5,296	5,296
Jim Lubin Fellowship Fund	6,926	6,926
Mission Fish.com Commissions	205	205
Recycling Commission	914	914
Research Donations	1,100	1,100
Support Group Donations	100	100
Wristband/cookbook/cards Fundraiser	110	110
TOTAL INCOME	**394,319**	**401,755**
EXPENSES		
ACP Support	20,000	20,000
Bank Fees	154	154
Children's Camp Expenses	15,914	15,914
Domain/Web-site/Webhosting	1,249	1,249
Internet Service Provider	0	2,226
Johns Hopkins TM Center Grant	20,000	20,000
Mileage and Parking	0	75
Office Supplies	0	227
Postage	9,159	9,261
Printing	16,671	16,671
Secretary of State Registrations/Annual Reports	140	140
Mailing Services	613	613
Software/Computer/Projector	1,489	1,975
Telephone	0	1,193
TMA Board Expenses	1,260	1,260
Travel Expenses	0	3,127
TOTAL EXPENSES	**86,649**	**94,085**
Net Income/Loss	**307,670**	**307,670**

Transverse Myelitis Association 2009 Statement of TMA Account Balances	
Jim Lubin Fellowship Fund	**500**
Richard Charles Gilmur Endowment Fund	**11,603**
Endowment Interest	**1,030**
Operating Fund	**513,053**
Research Fund	**35,675**
Support Group Fund	**1,654**

2010 Statement of Financial Activity

INCOME	TMA Funds	Total Donations and Expenses to Benefit TMA
Amazon.com Commissions	650	650
CafePress Commissions	51	51
Captial One Commissions	118	118
Children's Camps	4,500	4,500
Donations made by Board of Directors	0	9,637
Endowment Interest	195	195
General Donations	38,857	38,857
iGive.com Commissions	148	148
Interest	1,163	1,163
Jim Lubin Fellowship Fund	10,082	10,082
Mission Fish.com Commissions	67	67
Recycling Commission	1,400	1,400
Research Donations	125	125
Wristband/cookbook/cards Fundraiser	169	169
TOTAL INCOME	**57,525**	**67,162**
EXPENSES		
Bank Fees	30	30
Children's Camp Expenses	5,747	5,747
Domain/Web-site/Webhosting	707	707
Internet Service Provider	0	1,092
Mileage and Parking	0	100
Office Supplies	0	1,306
Postage	3,166	3,760
Printing	20,602	20,602
Mailing Services	1,213	1,213
Software/Computer/Projector	2,777	4,737
Support Group Expenses	459	459
Telephone	0	1,881
TMA Board Expenses	2,404	2,404
Travel Expenses	0	2,704
TOTAL EXPENSES	**37,105**	**46,742**
Net Income/Loss	**20,420**	**20,420**

Transverse Myelitis Association 2010 Statement of TMA Account Balances	
Jim Lubin Fellowship Fund	**10,582**
Richard Charles Gilmur Endowment Fund	**11,603**
Endowment Interest	**1,225**
Operating Fund	**521,479**
Research Fund	**35,800**
Support Group Fund	**1,195**

Officers and Board of Directors of The Transverse Myelitis Association

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The Transverse Myelitis Association

Powell Ohio
43065

Sanford J. Siegel
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Powell, Ohio 43065-8806

UK Conference April 1 - 3, 2011 Wyboston Lakes Conference Centre www.tmsconference.org.uk

Family Camp for Kids with TM, ADEM, NMO or ON and their Families: October 6 – 9, 2011 Victory Junction Gang Camp, Greensboro, NC