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Editor's Column
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Many people with TM experience an exacerbation of symptoms. These exacerbations can be quite dramatic and severe, and they can occur at various intervals and may last for different periods of time. For instance, a person may have quite stable symptoms after their recovery from the onset of TM, and then after months and months, or even many years, their symptoms can significantly worsen. Having an exacerbation of symptoms does not mean that you have recurrent TM or Devics or MS. In order to receive a diagnosis of these diseases, you would need to have tests performed that rule in that you have experienced a new demyelinating attack in your central nervous system. Fortunately, it appears that only small percentages of people with TM experience multiple episodes of distinct and different immune attacks. The exacerbations I would like to discuss in this column have to do with a worsening of a person's symptoms that are not brought on by a demyelinating attack. These are exacerbations of symptoms that do not appear to result from any new destruction of myelin or nerve tissue.

So much about TM is not well understood. The issue I am writing about in this column is perhaps one of the least understood issues from among the broad spectrum of little understood issues about TM. And that's probably why I am writing about this issue as opposed to one of the physicians. And I don't blame them. I'm not particularly comfortable writing about it either. Doctors do not want to communicate information that does not have a basis in fact – that means based on peer reviewed, published research and/or well described and published clinical cases. I am not aware of any published literature on this subject and TM. But I know it goes on, and it happens fairly frequently to relatively large numbers of people who have TM and who have not experienced a full recovery – people who have significant residual symptoms from their onset attack and after their recovery.

And this is an important point. I have no idea what happens with people who have completely recovered from TM. I, perhaps, have the most skewed perception of the population of people who have TM. People who have fully recovered from TM are not looking for a TM support group in the same way as people who have difficult residual symptoms. Many of these people are likely not looking for a support group at all. So, every time I read the statement that a third of the people who get TM have a good recovery from it, I'm always thinking that I would love to meet some of them. Thus, my experience with the fully recovered is minimal, and I don't know if they have exacerbations.

One of the most intellectually difficult and emotionally challenging issues I deal with in my many communications with people from the TM community concerns whether a person is having a recurrent episode of Transverse Myelitis. I receive phone calls almost every day from people and many email messages every week from people around the world. From these contacts and requests for information, there is usually one person, and often a couple of people, who are concerned about why their symptoms have worsened after they have had some recovery from their onset episode of TM. Some of these people express a concern that they might have recurrent TM.

These conversations are emotionally challenging because people who have had an episode of TM and think that they might be having another episode are always emotionally distraught. The thoughts and feelings a person has about the possibility of having to relive this experience are so traumatic. On more than one occasion, Pauline has said to me that she wasn't sure she could go through that experience again. It really makes me appreciate what kind of emotional resolve a person with recurrent TM or Devics or MS must possess to handle the issues they have regarding their well being and their potential futures. And I talk to these people all the time, and their emotional and spiritual strength is really so inspiring to me.

These conversations are also intellectually challenging, because I have quite difficult choices to make in what I say to these people and how I say it. First and foremost, I am not a medical doctor, so I am not about dispensing medical advice to anyone. Secondly, even if I were a doctor, I would not be making diagnoses or prescribing treatments over the telephone, without reviewing complete medical histories and performing a comprehensive medical examination.

These people are calling me with their concerns, because they are faced with some interesting dilemmas; they are caught between some difficult choices.

It is not always easy to get in to see a neurologist for an emergency appointment; some physicians are more responsive than others to this type of emergency. And someone who thinks that they are having a new episode of TM does indeed have an emergency situation, because we have learned that a person experiencing a demyelinating attack needs to be treated as quickly as possible.

Many people with TM have not had stellar experiences in the emergency room from their onset episode of TM. The idea that they might have to go to an emergency room and explain that they have TM, which is complicated enough, and then have to explain that they might be having another episode of TM can be a daunting consideration.

Some people communicate that they are being seen by a general neurologist, as opposed to a MS specialist, who has little experience with TM. They are concerned that their having to explain that they might be having another episode of TM may not be well accepted or understood by their physician.

As I have said previously, I know that people are calling me because I answer the phone. I can't tell them what is wrong with them; I would have no idea. I can help them to feel less frantic about their situation and can help them through their decisions about what is a reasonable course of action; all of which entail getting physicians involved as quickly as possible.

I have been dealing with this issue regularly enough that I have finally decided that I needed to write about it. I am concerned about what I say or how much I say, because I am always very sensitive to the fact that I am the idiot standing in my kitchen in Ohio. But I think it is important for me to discuss this issue and get this out there for all of us to think about and talk about. And we need to encourage our physicians to think about this issue. This issue needs to be recognized, because it is happening to significant numbers of people. We need to help people think about what is happening to them, how they might approach it emotionally and what course of action is appropriate and reasonable.

I am going to make a distinction between an exacerbation of symptoms and a new demyelinating attack. This is my terminology; I have no idea how physicians might refer to this differentiation, if they are making it. I know this is complicated; I'll write really slowly. People who have recurrent TM, or Devics or MS have different and distinct episodes of demyelination from a new autoimmune attack. Something triggers the immune system to go haywire … again. In the cases of recurrent TM, there may be new symptoms involved for the person, if the level of attack is above the previous episode or episodes of demyelination. For instance, there may be upper body involvement from the new episode that was not experienced during the first or previous episodes that might have occurred at lower levels of the spinal cord. The only way to know whether a person is having a new episode of demyelination is to go to a neurologist and have him/her perform all of the tests necessary to rule in or out the evidence of a new inflammatory attack. During a new episode of TM, a person who recovered from bladder problems may lose the ability to urinate again. A person who recovered muscle function in their legs, might lose that function again. Any appearance of new symptoms would depend on the level of the spinal cord impacted and the severity of the attack.

Most people who have TM and who have not experienced a complete recovery can have an exacerbation of symptoms. What do I mean by an exacerbation of symptoms? If you have spasticity, the spasticity worsens. If you have nerve pain or paresthesias, the pain or paresthesias can worsen and become more intense. For instance, you might have had tingling in your feet from the onset of TM. Upon experiencing a good recovery, the tingling in your feet might have disappeared for years. With an exacerbation, the tingling in your feet might return. You might have been paralyzed at onset and recovered over time to where you had movement in your legs but had muscle weakness. During an exacerbation, the weakness could become more pronounced; maybe almost to the return of paralysis. Exacerbations can impact any of a person's TM symptoms, but it does not necessarily affect all of their symptoms. For those people who experienced an acute and severe attack from TM at onset, the exacerbations can be similar to the onset of their symptoms or can "feel" similar to that onset. This is the reason that this experience is so traumatic and stressful for people. For many, it involves reliving these most horrible and difficult episodes in their lives. And the fear that this could be happening again is very intense.

The bad news is that these exacerbations are real; the symptoms are real, and this happens to a lot of people with TM. The good news is that the vast majority of these people do not have recurrent TM or MS or Devics and they are not having another demyelinating attack. After some period of time, usually a short period of time, they are going to return to their previous level of recovery; where they were when the exacerbation started. The spasticity will settle down or they will regain the strength in their legs, or their nerve pain or paresthesias will quiet down and improve.

I learned about this experience first from Pauline; and she experienced it in much the same ways that most of you experience it. The first time she got

the flu after her recovery from her onset TM symptoms, the nerve pain she has in her feet and legs became quite severe. Her bladder urgency also worsened considerably. We have since learned that any kind of illness that involves her immune system ordinarily makes her symptoms worse. We know that as her immune system quiets down; her symptoms will also improve, so we do less freaking out about it than we did when it first happened to her. At the same time, there are certain things we monitor and pay attention to in deciding how to manage these events; including when it is time to call and involve the physician. And I'll get to that momentarily.

Differentiating between an exacerbation of symptoms as opposed to having an episode of recurrent TM is critically important. People who have recurrent TM are often treated with the same type of medications as people who have MS; they are treated with the immune modulating drugs due to the possibility of multiple episodes. These medications help to delay future episodes and tend to lessen the severity of episodes should they occur. People who have exacerbations are not placed on these medications.

What triggers an auto-immune attack in MS or recurrent TM or Devics? I haven't the slightest idea. Remember, I'm an idiot. Having said that, I'm not sure anyone else has the answer to that question. But it happens, and I know that there is a lot of research going on to find the answer; there is MS money going into finding the answer or answers, and this research is going to help people who have recurrent TM and Devics disease.

What triggers an exacerbation? I have even less of an idea about exacerbations. Why would some compromise of the immune system make symptoms that are caused by nerve damage worsen – when there is no additional damage being done to the nervous system? Okay, I wrote that really slowly, and I even confused myself.

I'd like to relate an experience I had not too long ago. I have been in contact with a number of people who have recurrent TM; I have asked some people to write articles for the In Their Own Words column so that I can share these experiences and this information with our readers. I received a wonderful article from a gentleman who has had a very difficult lifetime of experience with TM and was given a diagnosis of recurrent TM by a neurologist. I read his article and was really touched by his experiences. This gentleman got TM at the age of ten and was paralyzed from the waist down. He experienced a good recovery from his onset symptoms over a period of time. At the age of 17 he experienced an upper respiratory infection and in the environment of this infection, he once again developed symptoms that were similar to his onset. He was treated with steroids and had a good recovery. He experienced this cycle of infection, recurring worsening of TM symptoms, getting treated with steroids and then having a good recovery a couple of more times in his adulthood. He was eventually given a recurrent TM diagnosis and was placed on one of the MS drugs. Upon completing his In Their Own Words article, I was immediately concerned with what I had read. First, I am not aware of any young child who got TM and then developed recurrent TM. In other words, I am not aware of a case of recurrent TM where the first episode was experienced in early childhood, and the age of ten certainly fit into my thinking about what was childhood. Secondly, seven years was a very long period of time between episodes for recurrent TM. Most people with recurrent TM have a second episode within months of the first episode or at the most a couple of years afterwards. Seven years would have been way outside of any interval I had ever heard about. And, finally, what heightened my greatest curiosity about this person was that every exacerbation of his TM symptoms was experienced in the context of some kind of upper respiratory infection.

I thought about sharing my concerns with this person, particularly because he was prescribed an immune modulating drug when he got his recurrent TM diagnosis, and if he doesn't have recurrent TM, it is probably a really good idea not to be messing with his immune system. But I didn't want to say anything to him without first discussing my concerns with someone who was in a better place to think about this issue; someone who is not an idiot. I presented the basic facts I had to a physician I have great trust in. This person confirmed that my suspicions were at least worth communicating to the person.

So, I called this gentleman, and after beginning the conversation with a reminder that I am an idiot, I shared my concerns. I asked him whether any of his subsequent 'episodes' after the first one when he was ten years old were confirmed as a demyelinating attack with an MRI. He wasn't sure, but he didn't know for certain that they had been. I suggested to him that it would be a good idea for him to discuss this with his physician and particularly with the doctor who gave him the recurrent TM diagnosis. I told him that if he did not have his second, third or fourth episodes confirmed as demyelinating attacks that it was certainly a possibility that he didn't have recurrent TM, and that his exacerbations could be explained as being triggered by his upper respiratory infections.

There was a 12 year old who got TM and had experienced a good recovery from her onset symptoms. She had been paralyzed from the waist down and had lost significant function below her waist. Over a period of a couple of

years, she was able to walk. A year or so later, she got the flu and shortly thereafter, she became paralyzed from the waist down. She was immediately given all of the tests to rule out a recurring episode of inflammatory attack in her spinal cord. It was ruled out. She recovered from her paralysis shortly after she recovered from the flu. And as with the gentleman I described previously, her recovery was similar to the point of recovery she had experienced from her original onset symptoms.

What is the connection between an upper respiratory infection and exacerbations of TM symptoms; how should I know; I'm an idiot. But I know something is going on, because it is happening to people.

Are they experiencing new demyelination and damage to their spinal cords? It doesn't seem like they are; I have not heard of any of these people diagnosed by an MRI with a new inflammatory attack. Why would an infection or the flu or serious cold symptoms trigger the worsening of TM symptoms? Why would a bladder infection or an upper respiratory infection or any kind of illness make a person's muscles become weaker, or make spasticity worse, or bring on bladder or bowel incontinence that may have been fairly stable and improved for a long period of time? Why would these infections and other illnesses trigger tingling and other paresthesias that had been fairly stable and improved for a long period of time? Why would these infections and illnesses trigger a significant worsening of nerve pain? Why would stress to the immune system impact symptoms, if the nervous system is not impacted or there is not more damage being done to the nervous system? Okay, I'll stop.

I really wish someone had the answers to these questions, because then someone could come up with a treatment for it. Or for now, could at least acknowledge for people that it goes on, and get me an extra hour or two in the evenings that I can use to focus on the news so I can aggravate myself about what's going on in the world. Hey, what are all of you people doing out there; please cut it out. Be nice.

It doesn't appear that these illnesses are causing ***new*** nerve damage. And it doesn't appear that these illnesses are triggering the development of ***new*** symptoms; those two statements are consistent and logical. People who experience these exacerbations experience a worsening of symptoms that they had from their onset episode. A person who never had spasticity is not likely going to develop spasticity from an exacerbation. A person who experiences these exacerbations is not likely to develop a new level of sensory deficit from their onset of symptoms. If your onset immune attack was at the T10 level of your spinal cord, the exacerbation will likely involve symptoms that relate back to this same level as the original, and only, demyelinating attack. And it also appears that a person who experiences an exacerbation is likely going to recover to the level of recovery they experienced from the original onset of TM.

I am certain that there are exceptions to what I am writing. How do I know that? Because I never write about anything that doesn't elicit someone writing to me saying, "hey, that's not what happened to me." There are things that generally happen in cases of TM, but there are definitely exceptions to everything. I don't have a handle on what the "garden variety of TM" looks like yet. With just 34,000 cases in the U.S., and with all of the various potential causes, and with the multitude of onset experiences from acute to slowly progressing, and from the differences which seem to be present in cases between adults and children, I'm not sure we're going to get a garden variety of TM.

If you know of a child who got TM and then developed recurrent TM, please get in touch with me and let me know. I really don't want to hear this, but if there is a case, I need to know; and recurrent means that there have been substantiated, diagnosed cases of more than one inflammatory attack in the spinal cord.

When Pauline has a cold or the flu, her nerve pain, bladder urgency and muscle weakness become markedly worse. I know this is going on with people; I hear about it all the time. And to my knowledge, they all recover soon after their cold or flu symptoms are gone.

Okay, while I'm out dangling on this limb, I'm going to crawl out a bit further to where the branch gets really flimsy. I know that people's symptoms worsen under a number of different circumstances besides in the environment of an infection or cold and flu symptoms. I have heard people describe a worsening of symptoms from experiences with high stress and fatigue. Stress is an exceedingly difficult variable to consider, because it is difficult to find a time when stress is not present at some level in our lives. But recognizing the endemic nature of stress in our lives, I know that Pauline's symptoms are worse when she is working on her report cards and preparing for parent-teacher conferences as opposed to the middle of summer vacation when her most significant decisions involve whether she should spend the afternoon making me a pot roast versus ironing all of my shirts.

I am regularly contacted by people who are experiencing high stress at work or have recently experienced a significant tragedy in their family and are concerned, because their symptoms have become much worse. I have no

idea why this happens, but I'm pretty sure it is happening. Is it possible that stress is compromising the immune system in at least an analogous manner to an infection? That is conceivable to me; for whatever that is worth.

We need to acknowledge that this exacerbation thing is going on. And we need to pay attention to the variables that could be triggering these episodes. I have a good sense that infections of all varieties, including those that might result from having the cold or the flu could be involved. I have a sense that inordinately high stress and fatigue can trigger these exacerbations. And I also have a sense that other stuff could trigger these exacerbations. I have heard people talk about changes in the weather or having to deal with extremes of temperature. I would imagine there are all kinds of things that could trigger a worsening of symptoms.

It is also important for me to acknowledge that this discussion about exacerbations should be considered within the context of the very dynamic nature of TM symptoms. People's symptoms change all of the time for a multitude of reasons. The idea of stable symptoms is a relative concept. And that, by the way, is why it is a good idea for people with residual TM symptoms to be examined by a neurologist or a physiatrist once a year, in much the same manner people schedule a physical exam with their family doctor. An exacerbation would represent a change in symptoms that is more significant or more pronounced or more severe than the more day-to-day changes that many people experience with their TM symptoms.

So, what are we supposed to do with this information? Well, one thing you can do with it is relax a bit. You likely don't have recurrent TM and you are not likely to get it. And even if your symptoms get worse on occasion, it is not likely happening from a new inflammatory attack. And if you do have an exacerbation, as I have described in this column, you are very likely going to experience a return to the recovery you experienced from the original onset.

So, let's return to the dilemma. I get a call from someone who thinks they are having a new attack and that they could have recurrent TM. Well, the only way to know for certain is to get to a medical doctor to begin the process of ruling it out. There's no other way to do it; because they are not going to get any great answers from me. But here is what they are going to get from me.

I'm going to ask them if they have a cold or the flu or a urinary tract infection or upper respiratory infection. If they tell me that they do not have any infection or illness, I ask them to describe what their current symptoms are. When they finish their description of symptoms, I ask them if any of these symptoms are different from the symptoms they had when they experienced the first onset of TM and if the level is any different. For instance, is the sensory level or functional level higher than it was at the first onset? I also ask them if there are parts of their body that are impacted that have not been involved previously in their TM symptoms. If they tell me that they don't have any other illness and that they have new symptoms or that they are unable to urinate, or that the sensory or functional level is higher than onset, I suggest they print Dr. Kerr's three articles from our web site, and then head to the nearest emergency room of the hospital that is associated with a medical school that has a MS Center with the articles in hand. And then I ask them to call me as soon as they hear something or if they need some assistance.

If they tell me that they have had the flu or a bladder infection and that the symptoms they have always had are worse than they have been for a while, I suggest that this might not be something they need to be really worried about. I tell them that they should make an appointment and to try to get in to see their neurologist as soon as possible, and let their neurologist know about the change in symptoms. This is something everyone should pay attention to; if you experience a dramatic change in your symptoms, this is something your physician needs to hear about. Telling me about it might make you feel better for a couple of minutes, but I'm not going to do anything highly beneficial for you. Your doctor needs to know this and needs to think about what's going on, and then needs to start ruling some things out. I am woefully inadequate in all of those realms.

Part of this journey with TM involves getting to know your body. It is important to develop some sensitivity, skill and experience about when something minor is going on that can be ignored, versus when something significant is going on that you should monitor for further action versus something really significant that should trigger an immediate contact with your physician or a decision to go to an emergency room. This isn't easy; this isn't easy for any of us. I grew up in a family where there is serious cardiovascular disease; it is a multigenerational problem that is pervasive. My brother and sister and I have all made appearances in emergency rooms with serious cases of heartburn; even from a fairly early age. We laugh about it now, but it is one of those really ironic kinda laughs, because we share the nightmare about gasping desperately for our last breaths while we're chewing on a Tums.

If you have any doubt in your mind about what you should do if your symptoms change, you need to contact your doctor. If you are experiencing new symptoms, severe symptoms, or symptoms that reflect a different or

higher level of impact on your spinal cord, you should contact your physician and if they are not able to see you immediately, you should probably go to the emergency room, with Dr. Kerr's three articles in tow. If you are experiencing sufficient doubt about your symptoms that you think you need to tell me about it, call your doctor, and tell him or her. I'm just going to repeat what I've said in this article. And if you call me right now, you're probably going to have to listen to me complain about my life for an hour before you get a chance to say something to me about your problems. Pauline and I are in the process of remodeling our house to make it accessible. So, every room in our house is in the process of being demolished and renovated. We are living in a construction zone with one toilet, no shower or bathtub, and our lives are sheer unadulterated dirt and chaos. I'm in dire need of a support group. So, call Chitra; I don't think she's sharing her toilet with a construction crew.

While I'm venturing out on the limb, I might as well make my way out to the very end. I have talked to some people who have had exacerbations that occurred in the environment of an infection; there might have been some relationship between their exacerbation and something that could have been going on with their immune systems. Their doctors put them on steroids for a short period of time, and they seemed to improve. I am not prescribing steroids for exacerbations; I am not a doctor and I do not prescribe anything. I am suggesting to you that this is something you might want to talk to your doctor about should you be having the type of exacerbation I have described in this column. It is certainly something to think about.

So, don't get sick and don't have stress. Or be aware that the consequences of being sick are more serious for you today than they were before you had TM, and practice more diligence about avoiding colds and the flu. Wash your hands a lot; practice extra care and cleanliness in the bathroom and kitchen, and use every ounce of common sense you have about these matters. As for stress, lighten up. It is a fact that stress is endemic in our lives and we only have so much control over avoiding it. But we are not passive recipients of every stressful situation that comes our way. We can make choices in our lives which minimize stress, like, hey, Pauline, let's demolish every room in the house and remodel it. And let's live in the house while they do all of the work. And let's share this teeny bathroom with a contractor and his crew and the electrician and the plumber and anyone else who comes into the house. Hey, do we know who all of these people are who are roaming around our house? Okay, you're on your own with the stress thing. I've got way too much stress to be talking to you about stress reduction. Just deal with it the best you can. Hey, Pauline, what happened to the electricity and what's that smell and is that dirt … what is that … is it moving … is it alive?

Please take good care of yourselves and each other.

We Don't Want to Lose You And Finding You Has Become Very Expensive

Please keep us informed of any changes to your mailing address, your phone number and your email address. You can send changes to me via email at ssiegel@myelitis.org; you can send changes to me by mail, you can call me (614)766-1806; or you can fill out a change of information form on the web site: http://www.myelitis.org/memberform.htm – just click on the box indicating that you are changing existing information.

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As a result of this situation, I have to do a separate and special mailing that is devoted to checking the accuracy of the TMA membership database. The process involves a lot of work, a lot of money and about four months of time to complete. And the cost to the Association is substantial, because until we perform a mailing to correct the address information, the materials we are mailing to a bad address just ferment on some post office floor. These are wasted printing and postage costs.

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TRANSVERSE MYELITIS: PATHOGENESIS, DIAGNOSIS AND TREATMENT

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1. ABSTRACT

Transverse Myelitis (TM) is a clinical syndrome in which an immune-mediated process causes neural injury to the spinal cord, resulting in varying degrees of weakness, sensory alterations and autonomic dysfunction. TM may exist as part of a multi-focal CNS disease (e.g. MS), multi-systemic disease (e.g. systemic lupus erythematosus), or as an isolated, idiopathic entity. In this article, we will summarize recent classification and diagnostic schemes (1), which provide a framework for the acute management of patients with TM. Additionally, we will review current concepts on the natural history, immunopathogenesis and treatment strategies for patients with TM.

2. INTRODUCTION

TM is a rare syndrome with an incidence of between 1 and 8 new cases per million people per year (2). TM is characterized by focal inflammation within the spinal cord and clinical manifestations are due to resultant neural dysfunction of motor, sensory and autonomic pathways within and passing through the inflamed area. There is often a clearly defined rostral border of sensory dysfunction and evidence of acute inflammation demonstrated by a spinal MRI and lumbar puncture. When the maximal level of deficit is reached, approximately 50% of patients have lost all movements of their legs, virtually all patients have some degree of bladder dysfunction, and 80-94% of patients have numbness, paresthesias or band like dysesthesias (2-7). Autonomic symptoms consist variably of increased urinary urgency, bowel or bladder incontinence, difficulty or inability to void, incomplete evacuation or bowel constipation and sexual dysfunction (8). Like MS (9), TM is the clinical manifestation of a variety of disorders, with distinct presentations and pathologies (10). Recently, we proposed a diagnostic and classification scheme which has defined TM as either idiopathic or associated with a known inflammatory disease (i.e. multiple sclerosis, systemic lupus erythematosus, Sjogren's syndrome, or neurosarcoidosis) (1). Patients with TM should be offered immunomodulatory treatment such as steroids and plasmapheresis though there is no consensus as to the most appropriate strategy yet. Most TM patients have monophasic disease, while up to 20% will have recurrent inflammatory episodes within the spinal cord (JHTMC case series, 11, 12).

Table 1. Spectrum of Neuroimmunologic Disorders

Disorder	Abbreviation	References
Muscle		
Polymositis	PM	27
Dermatomyositis	DM	107
Neuromuscular Junction		
Myasthenia gravis	MG	25
Peripheral Nerve		
Chronic Inflammatory Demyelinating Polyneuropathy	CIDP	108, 109
Acute Inflammatory Demyelinating Polyneuropathy	AIDP/GBS	13, 79
Spinal Cord		
Transverse myelitis	TM/ATM	1, 3, 19, 36
Tropical spastic paraparesis	TSP/HAM	28, 29
Stiff person syndrome	SPS	24
Spinal Cord and Optic Nerve		
Neuromyelitis optica	NMO	26, 101
Optic Nerve		
Optic neuritis	ON	110
Brain and Spinal Cord		
Multiple Sclerosis	MS	9
Paraneoplastic encephalomyelitis	-	111, 112
Brain		
Acute disseminated encephalomyelitis	ADEM	113
Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection	PANDAS	20, 21
Hashimoto's encephalitis	-	22
Rasmussen's encephalitis	RE	114, 115

3. THE SPECTRUM OF NEUROIMMUNOLOGIC DISORDERS

The clinical, immunologic and pathologic findings in TM patients place this disorder on a continuum of neuroimmunologic disorders (Table 1). In each, there is an acquired alteration in the innate or acquired immune system, resulting in dysfunction and/or cellular injury to cells within the nervous system. Many of the disorders may be post-infectious (TM, ADEM, GBS, PANDAS), suggesting that the infectious agent triggers breakdown of immune tolerance for self antigens. In GBS, a preceding infectious agent (often *Campylobacter jejuni*), may encode a molecular mimic that resembles a host ganglioside expressed on peripheral nerves, resulting in immune-mediated injury (13-15). In ADEM and TM, superantigen-mediated activation of T lymphocytes may be an important event in breaking down immune tolerance (16-19). In PANDAS, evidence to date suggests that the development of anti-neuronal antibodies following infection with Group A beta hemolytic streptococcus (GABHS) results in dysfunction of basal ganglia (20, 21). Similarly, NMO, recurrent TM, SPS, paraneoplastic encephalomyelitis, MG and Hashimoto's encephalopathy have prominent humoral derangements that may contribute to neural dysfunction (for reviews, see (19, 22-26). Other disorders in this group, such as polymyositis, TSP/HAM, Rasmussen's encephalopathy and TM have prominent T lymphocyte derangements (for review, see (27-29). MS has recently been shown to have multiple distinct pathologic subtypes with differing degrees of T lymphocyte, macrophage, antibody and complement contributions (9). In many of the other disorders as well, it is unlikely that a single immune system component is wholly responsible for the clinical disorder, and mixed dysfunction of T lymphocytes, B lymphocytes, macrophages/microglial cells and even NK cells may contribute. Similarly, the mechanisms of neural injury may involve multiple pathways including T lymphocyte killing of neural cells, cytokine injury, activation of toxic microglial pathways, immune-complex deposition, excitotoxic or apoptotic neural injury. Though TM exists on a continuum of neuroimmunologic disorders, the reasons to explain distinct focality of these disorders are unclear. Potential explanations include unique antigen representation within a focal area of the spinal cord, unique trafficking of immune effector cells within a particular region of the spinal cord, or regionally-specific differences in the immune response (for example, differential elaboration of cytokines or efficient antigen presentation to the immune system within the spinal cord).

4. HISTORY OF TM

Several cases of "acute myelitis" were described in 1882, and pathologic analysis revealed that some were due to vascular lesions and others to acute inflammation (30). Subsequently, the occurrence of >200 cases of postvaccinial encephalomyelitis was reported between 1922 and 1923 in England, a complication of smallpox and rabies vaccination (31). Pathologic analyses of fatal cases revealed inflammatory cells and demyelination rather than the vascular pathology noted in earlier reports. Subsequent reports emphasized that TM may be a post-infectious "allergic" response since in many patients, "the fever had fallen and the rash had begun to fade" when myelitis symptom began (32). Several cases were then reported over the next two

Table 2. TM Diagnostic Criteria

Inclusion criteria
1. Development of sensory, motor or autonomic dysfunction attributable to the spinal cord 2. Bilateral signs and/or symptoms 3. Clearly-defined sensory level 4. Inflammation within the spinal cord demonstrated by CSF pleocytosis *or* elevated IgG index *or* gadolinium enhancement (If none of the inflammatory criteria is met at symptom onset, repeat MRI and LP evaluation between 2-7 days following symptom onset)
Exclusion criteria
1. History of previous radiation to the spine within the past 10 years 2. Clear arterial distribution clinical deficit consistent with thrombosis of the anterior spinal artery 3. Extra-axial compressive etiology by neuroimaging (MRI of spine preferred. CT myelography acceptable. X-ray, CT of spine are not adequate) 4. Abnormal flow voids on the surface of the spinal cord c/w AVM 5. Serologic or clinical evidence of connective tissue disease (sarcoidosis, Behcet's disease, Sjogren's syndrome, SLE, mixed connective tissue disorder etc) (Diagnostic of Connective-Tissue Associated TM) 6. History of clinically apparent optic neuritis (Diagnostic of Neuromyelitis optica) 7. CNS manifestations of syphilis, Lyme disease, HIV, HTLV-1, mycoplasma, other viral infection (e.g. HSV-1, HSV-2, VZV, EBV, CMV, HHV-6, enteroviruses) (Diagnostic of Infectious myelitis) 8. Progression to nadir in less than 4 hours from symptom onset 9. Symptom progression continues beyond 21 days from symptom onset 10. Brain and spinal cord MRI abnormalities suggestive of MS and presence of oligoclonal bands in CSF (Suggestive of TM associated with MS. Apply McDonald criteria to definitively define MS)

AVM= arteriovenous malformation; SLE= systemic lupus erythematosus; HTLV-1= human T-cell lymphotropic virus-1; HSV= herpes simplex virus; VZV= varicella zoster virus; EBV=Epstein-Barr virus; CMV= cytomegalovirus; HHV= human herpes virus

decades in which infectious agents, including measles, rubella and mycoplasma, were directly isolated from spinal fluid of patients with TM, suggesting that direct infection of the spinal cord may be involved in some patients (33, 34). It was in 1948 that Dr. Suchett-Kaye, an English neurologist at St. Charles Hospital in London first utilized the term "acute transverse myelitis" (35) in reporting a case of rapidly progressive paraparesis with a thoracic sensory level, occurring as a post-infectious complication of pneumonia.

Acute transverse myelopathy (which includes non-inflammatory causes) and TM have often been used interchangeably throughout the published literature. One report established the following criteria for transverse myelopathy: bilateral spinal cord dysfunction developing over a period of < 4 weeks with a well-defined upper sensory level, no antecedent illness, and exclusion of compressive etiologies (10). Subsequently, these criteria were altered to include only those patients who developed motor, sensory, and sphincter dysfunction acutely over < 14 days, whereas patients with other neurologic disease or underlying systemic diseases were excluded (4). Other authors then defined TM as acutely developing paraparesis (no specification of a time to maximum deficit) with bilateral sensory findings and impaired sphincteric function, a spinal segmental level of sensory disturbance, a stable non-progressive course (to distinguish from progressive spastic paraparesis), and no clinical or laboratory evidence of spinal cord compression (2). Patients were excluded if they had progressive spastic paraparesis, a patchy sensory deficit or hemicord syndrome, syphilis, severe back trauma, metastatic cancer, or encephalitis. To further separate diseases with distinct etiologies, suggested criteria for TM were revised to include only those patients who progressed to maximum deficit within 4 weeks and to exclude other known diseases including arteriovenous malformations of the spinal cord, human T-cell lymphotropic virus-1 (HTLV-1) infection, and sarcoidosis (3). With use of these criteria, cases of TM were classified as parainfectious, related to MS, spinal cord ischemia, or idiopathic.

Most recently, acute noncompressive myelopathies were classified according to an etiologic scheme (36)-1) those related to MS, 2) systemic disease (e.g., systemic lupus erythematosus [SLE], anti-phospholipid syndrome, Sjögren disease), 3) postinfectious , 4) delayed radiation myelopathy, 5) spinal cord infarct, and 6) idiopathic myelopathy. The presence of MS or systemic disease was determined by standard criteria (37-39), whereas parainfectious myelopathies were diagnosed on the basis of positive IgM serology or a fourfold or greater increase in IgG levels on two successive tests to a specific candidate/infectious agent. Delayed radiation myelopathy was diagnosed according to clinical history, and spinal cord infarction was diagnosed on the basis of appropriate clinical and imaging findings in the absence of other likely etiologies. Idiopathic transverse myelopathy was defined in those individuals that could not be otherwise categorized and constituted 16.5% of this series.

5. DEFINITION OF TM

5.1. Criteria of TM

We recently proposed a set of diagnostic criteria which served to distinguish TM from non-inflammatory myelopathies and to distinguish idiopathic TM from TM associated with multifocal CNS and multi-systemic inflammatory disorders. These criteria are summarized in Table 2. A diagnosis of TM requires evidence of inflammation within the spinal cord. Because spinal cord biopsy is not a practical option in the routine evaluation of these patients, spinal MRI and CSF analysis are the only tools currently

available to determine the presence of inflammation within the involved lesion. Gadolinium enhanced spinal MRI and a lumbar puncture are mandatory in the evaluation of suspected TM, and we proposed that abnormal gadolinium enhancement of the spinal cord or CSF pleocytosis or elevated CSF IgG index be required for a diagnosis of TM (1). If none of the inflammatory criteria are met at symptom onset, MRI and lumbar puncture evaluation should be repeated between 2 and 7 days following symptom onset to determine if these inflammatory criteria are met. IgG synthesis rate is a less specific indicator of CNS inflammation than is CSF IgG index (40, 41), and should not be utilized in the diagnosis. Vascular myelopathies can be differentiated from TM by a progression of symptoms to maximal severity in less than 4 hours and the lack of inflammation as defined above. However, these criteria do not completely distinguish vascular myelopathies from TM, since myelopathies associated with venous infarcts or with vascular malformations may be more slowly progressive and may meet the other criteria for TM.

Differentiating idiopathic TM from TM attributed to an underlying disease is also important. Many systemic inflammatory disorders (e.g. sarcoidosis, SLE, Behçet's disease, Sjögren syndrome) may involve the nervous system and TM may be one the possible presentations. Therefore, all patients presenting with TM should be investigated for the presence of systemic inflammatory disease. Important historical information should be obtained from the patient regarding the presence of rashes, night-sweats, oral or genital ulcers, sicca symptoms, shortness of breath, pleuritic pain or hematuria. Examination should attempt to detect the presence of uveitis or retinitis, decreased lacrimation or salivation, skin rash (malar, livedo reticularis, erythema nodosum), oral or genital ulcers, adenopathy, pleuritic or pericardial friction rub, or organomegaly. Laboratory studies should include the following: CBC with differential and smear, ANA, SS-A, SS-B, ESR, and complement. Additional laboratory testing may be required if signs of a systemic vasculitis are detected.

From this evaluation, it may be possible to distinguish idiopathic TM from disease-associated TM (i.e., TM associated with multi-focal CNS disease or systemic inflammatory disease). This distinction is important since patients at high risk of developing MS may be evaluated more closely or may be offered immunomodulatory treatment (42). Similarly, patients with disease-associated TM may need to be closely followed for recurrent systemic and neurologic complications and should be offered immunosuppressive treatment to decrease the risk of recurrence. We routinely offer such patients treatment with azathioprine (2-2.5 mg/kg/d), methotrexate (15-20 mg/week), mycophenolate (2-2.5 g/d) or cyclophosphamide (pulse of 500-1000 mg/m^2 q 4-6 weeks for severe cases) although controlled trials of these interventions are lacking and currently needed.

5.2. Evaluation of patients with acute myelopathies

We recently proposed a diagnostic approach for evaluating patients with acute myelopathies (1). This algorithm is reproduced here as it has been applied to 354 consecutive patients seen at The Johns Hopkins Transverse Myelitis Center since July 1999 (Figure 1). The first priority is to rule out a compressive lesion. If a myelopathy is suspected based on history and physical exam, a gadolinium enhanced MRI of the spinal cord should be obtained as soon as possible. If there is no structural lesion such as epidural blood or a spinal mass, then the presence or absence of spinal cord inflammation should be documented with a lumbar puncture. The absence of pleocytosis would lead to consideration of non-inflammatory causes of myelopathy such as arteriovenous malformations, epidural lipomatosis, fibrocartilaginous embolism or possibly early inflammatory myelopathy (i.e. a false negative CSF). In the presence of an inflammatory process (defined by gadolinium enhancement, CSF WBC pleocytosis or elevated CSF immunoglobulin index), one should determine whether there is an infectious cause. Viral polymerase chain reaction assays should be performed to determine whether there is the presence of viral particles within the CNS (herpes simplex 1 and 2, varicella zoster, cytomegalovirus, Epstein Barr virus and enterovirus). Detection of lyme disease of the CNS typically is based on antibody detection methods (ELISA with confirmatory western blot) and the CSF/serum index is often helpful in determining whether there is true neuroborreliosis (43). Evidence of *M. pneumoniae* infection may be determined by seroconversion, which is defined by a 4-fold increase in titer or a single titer of ≥1:128.

The next priority is to define the regional distribution of demyelination within the CNS, since several disorders (i.e. multiple sclerosis or acute disseminated encephalomyelitis) may present with TM as the initial manifestation of disease or in the setting of multifocal disease. A gadolinium enhanced brain MRI and visual evoked potential should be ordered to look for these entities. The absence of multifocal areas of demyelination would suggest the diagnosis of isolated TM and lead to appropriate treatment measures (Section 8) (1).

TM is often misdiagnosed as acute inflammatory demyelinating polyradiculoneuropathy (AIDP) or Guillain-Barré syndrome (GBS), because both conditions may present with rapidly progressive sensory and motor loss involving principally the lower extremities. Table 3 illustrates key differential points between these two conditions. A pure paraplegia or paraparesis with a corresponding distribution of sensory loss may favor TM, while GBS may present with a gradient of motor and sensory loss involving the lower extremities greater than the upper extremities. When weakness and sensory loss involves both the upper and lower extremities equally with a distinct spinal cord level, then TM involving the cerival region is more likely. Pathologically brisk deep tendon reflexes are supportive of TM. However, patients with fulminant cases of TM that includes significant destruction of spinal cord gray matter may present with hypotonia and have decreased or absent deep tendon reflexes. Urinary urgency or retention is a common early finding in TM and is less common in GBS. In GBS, dysesthetic pain, involvement of the upper extremity and cranial nerve 7, and absent deep tendon reflexes involving the upper

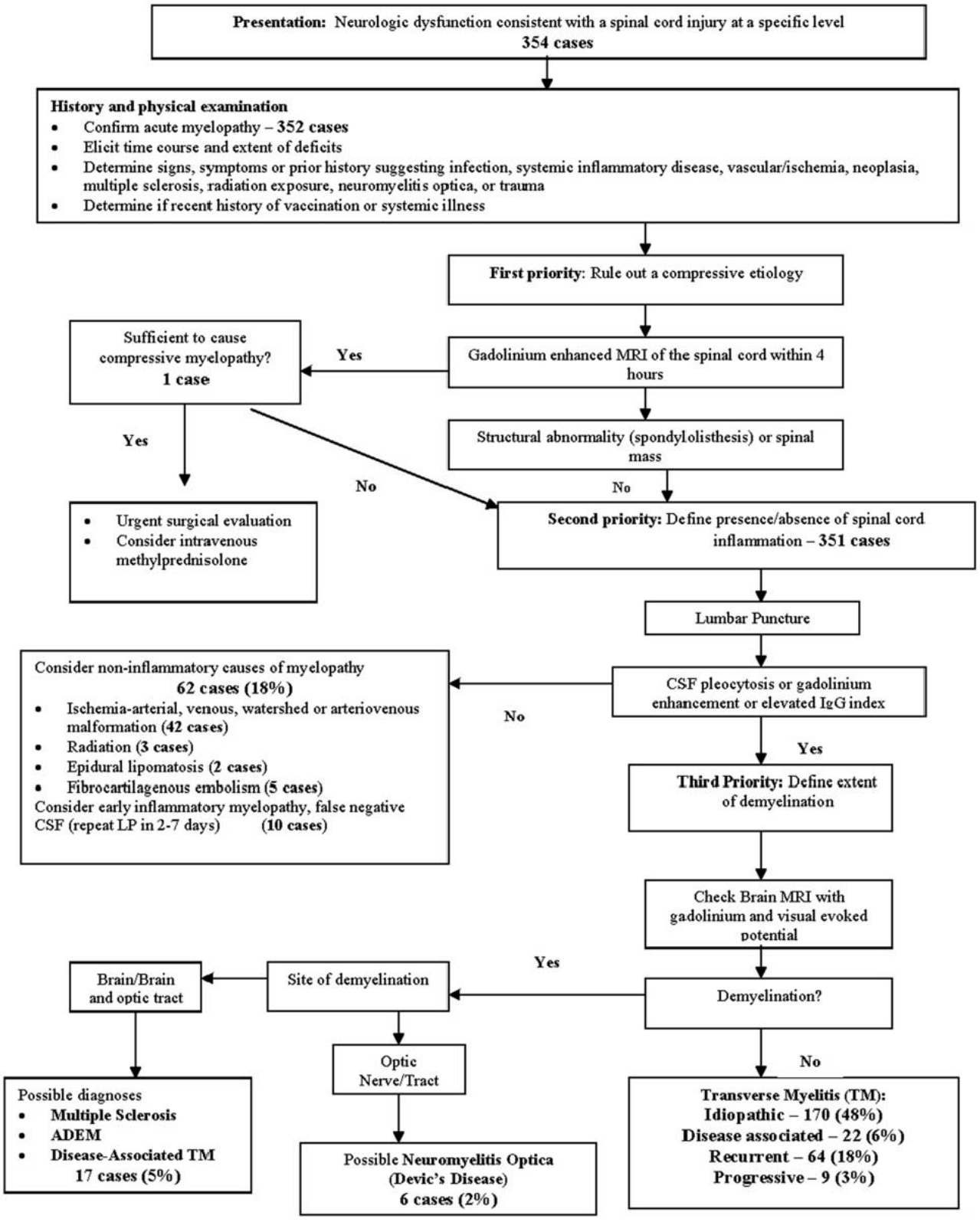

Figure 1. Diagnostic Approach to Acute Myelopathy (JHTMC case series)

extremities are more common findings. An MRI of the spinal cord may show an area of inflammation in TM but not in GBS. Although cerebral spinal fluid findings in TM are not consistent and an elevated cell count may be absent, there is usually a moderate lymphocytic pleocytosis and elevated protein level. This is in contrast to the albumino-cytologic dissociation of the CSF seen in GBS (44).

Table 3. Comparison of TM and GBS

Characteristics	TM	GBS	Distinguishing Feature
Motor findings	Paraparesis or quadriparesis	Ascending weakness LE>UE in the early stages	TM: if UE involvement, often as severe as LE; often no UE involvement GBS: There usually is UE involvement and it is less severe than LE involvement early in the disease
Sensory findings	Usually can diagnosis a spinal cord level	Ascending sensory loss LE>UE in the early stages	TM: sensory level usually identified. Often no arm involvement GBS: no sensory level, usually UE less affected than LE early in the disease
Autonomic findings	Early loss of bowel and bladder control	Autonomic dysfunction of the cardiovascular (CV) system	TM: urinary urgency or retention early and prominent; CV instability only in severe cases higher than T6. GBS: urinary urgency or retention less common; CV instability is more common
Cranial nerve findings	None	EOM palsies or facial weakness	GBS: cranial neuropathies are more common than in TM
Electrophysiologic findings	EMGNCV findings may be normal or may implicate the spinal cord: Prolonged central conduction on somatosensory evoked potential (SEP) latencies or missing SEP in conjunction with normal sensory nerve action potentials	EMG/NCV findings confined to the PNS: motor and/or sensory nerve conduction velocity reduced, distal latencies prolonged; conduction block; reduced H reflex usually present	The lack of peripheral nerve abnormalities in a patient with progressive weakness and sensory loss should suggest evaluation of the spinal cord for pathology Conversely, patients with suspected TM but equivocal clinical, lab or radiologic findings may warrant peripheral nerve examination
MRI findings	Usually a focal area of increased T2 signal with or without gadolinium enhancement	Normal	MRI abnormalities may be helpful in diagnosing a patient who is suspected of having GBS from TM
CSF	Usually, CSF pleocytosis and/or increased IgG index	Usually, elevated protein in the absence of CSF pleocytosis	CSF pleocytosis and elevated IgG index may be helpful in diagnosing a patient who is suspected of having GBS from TM

5.3. Differential Diagnoses/Non-inflammatory myelopathies

As indicated above, the suggested diagnostic algorithm and criteria first distinguish inflammatory from non-inflammatory myelopathies. If the history and evaluation do not suggest a systemic or a CNS inflammatory process, then consideration should be given to ischemic, metabolic, or structural causes of myelopathy. Vascular myelopathy may be fairly easy to recognize in the setting of an anterior spinal artery infarct (sudden onset of symptoms with relative preservation of posterior column function). Or it may be more difficult to recognize in the setting of a venous infarct or in the setting of a vascular malformation. Venous infarct may be suspected when a clinical history and serologic studies are suggestive of a pro-thrombotic state (deep venous thrombosis, pulmonary embolus, livedo reticularis, anti-phospholipid antibodies, factor V Leiden mutation, APC resistance or prothrombin gene mutation). A vascular malformation (dural AV fistula, AVM, cavernous angioma) may be suspected if the imaging suggests the presence of flow voids or bleeding into the spinal cord. A dural AV fistula is most likely to occur in men older than 40 years old and may present with a "stuttering" or progressive myelopathy. Patients with a dural AV fistula may report a postural dependence of symptoms and pain is usually a prominent feature. Spinal angiography is the diagnostic study of choice to define the presence of a vascular malformation. Surgical or endovascular treatment may result in stabilization or clinical improvement in a substantial proportion of patients (45-47).

Fibrocartilagenous embolism is a rare (though likely underreported) cause of acute myelopathy (48-51). In most reported cases, there has been a sudden increase in intrathoracic or intraabdominal pressure prior to the onset of symptoms, and in several autopsies, fibrocartilageneous material was found to have embolized to the spinal cord. The most likely explanation for these findings is that the nucleus pulposis herniated vertically into the vertebral body sinusoids in response to markedly elevated pressure, followed by further herniation through vascular channels into the spinal cord parenchyma. Fibrocartilagenous embolism should be suspected in a patient with a sudden onset of myelopathy that reaches its maximal severity within hours in a patient with an antecedent elevation of intra-abdominal or intrathoracic pressure. Imaging may show acute loss of intervertebral disk height and vertebral body end-plate changes adjacent to an area of T2 signal abnormality within the spinal cord.

Radiation myelopathy may develop at any time up to 15 years following ionizing radiation. Pathologic studies

show preferential involvement of myelinated tissue and blood vessels and it is likely that cellular death of oligodendrocytes and endothelial cells contributes to the clinical disorder (52). Patients may present with slowly progressive spasticity, weakness, hyperreflexia and urinary urgency. There is often a corresponding T2 signal abnormality that is non-enhancing and preferentially affects the more superficial spinal cord white matter. Though anticoagulation (53, 54) or hyperbaric oxygen (55-57) have been proposed as treatment options, neither has been clearly shown to be effective in patients with radiation myelopathy.

5.4. Discrimination from Multiple Sclerosis

TM can be the presenting feature of MS. Patients who are ultimately diagnosed with MS are more likely to have asymmetric clinical findings, predominant sensory symptoms with relative sparing of motor systems, MR lesions extending over fewer than two spinal segments, abnormal brain MRI, and oligoclonal bands in the CSF (36, 58-62). A patient with monofocal CNS demyelination (Transverse Myelitis or optic neuritis) whose brain MRI shows lesions consistent with demyelination (63) has an 83% chance of meeting clinical criteria for MS over the subsequent decade compared with 11% of such patients with normal brain MRI (64).

6. NATURAL HISTORY OF TM

6.1. Epidemiology and Clinical Presentation of TM

TM affects individuals of all ages with bimodal peaks between the ages of 10 and 19 years and 30 and 39 years (2-5). There are approximately 1400 new cases diagnosed in the United States per year and approximately 34,000 people have chronic morbidity from TM at any given time. Approximately 28% of reported TM cases are in children [JHTMC case series]. There is no sex or familial predisposition to TM.

A preceding illness including nonspecific symptoms such as fever, nausea, and muscle pain has been reported in about 40% of pediatric cases within 3 weeks of the onset of the disorder (44, 65)[JHTMC case series]. 30% of all cases of pediatric TM cases referred to an academic center had a history of an immunization with one month of the onset of symptoms [JHTMC case series]. Although a history of an immunization preceding the onset of TM is commonly reported, the relationship to this condition is unclear because of insufficient information.

TM is characterized clinically by acutely or subacutely developing symptoms and signs of neurological dysfunction in motor, sensory and autonomic nerves and nerve tracts of the spinal cord. Weakness is described as a rapidly progressive paraparesis starting with the legs and occasionally progresses to involve the arms as well. Flaccidity maybe noted initially with gradually appearing pyramidal signs by the second week of the illness. A sensory level can be documented in most cases. The most common sensory level in adults is the mid-thoracic region, though children may have a higher frequency of cervical spinal cord involvement and a cervical sensory level (66). Pain may occur in the back, extremities, or abdomen. Paresthesias are a common initial symptom in adults with TM but are unusual for children (67). Autonomic symptoms consist variably of increased urinary urgency, bowel or bladder incontinence, difficulty or inability to void, incomplete evacuation or bowel constipation (8). Also commonly the result of sensory and autonomic nervous system involvement in TM is sexual dysfunction (68 , 69). Genital anesthesia from pudendal nerve involvement (S2-S4) results in impaired sensation in men and women. Additional male sexual problems with parasympathetic (S2-S4) and sympathetic (T10-L2) dysfunction in TM patients include erectile dysfunction, ejaculatory disorders and difficulty reaching orgasm. Corresponding female sexual problems include reduced lubrication and difficulty reaching orgasm.

In addition to the signs and symptoms of direct spinal cord involvement by the immune system in TM, there also appears to be indirect effects manifested as depression that are reminiscent of what has been described in MS (unpublished observations) (70). This depression does not correlate significantly with the degree of physical disability, and can have lethal consequences resulting in suicide in severe cases if left untreated.

When the maximal level of deficit is reached, approximately 50% of patients have lost all movements of their legs, virtually all patients have some degree of bladder dysfunction, and 80-94% of patients have numbness, paresthesias or band like dysesthesias (2-7). In more than 80%, patients reach their clinical nadir within 10 days of the onset of symptoms (44). Although the temporal course may vary, neurologic function usually progressively worsens during the acute phase from between 4-21 days (1).

A spinal MRI and lumbar puncture often show evidence of acute inflammation (2-4, 7, 10, 61, 71). In our case series of 170 idiopathic TM cases, spinal MRI showed a cervical T2 signal abnormality in 44% and a thoracic T2 signal abnormality in 37% of cases. 5% of patients had multifocal lesions and 6% showed a T1 hypointense lesion. This corresponded to the following clinical sensory levels: 22% cervical, 63% thoracic, 9% lumbar, 6% sacral and no sensory level in 7%. The rostral-caudal extent of the lesion ranged from one vertebral segment in many to spanning the entire spinal cord in two patients. In 74% of patients, the T2 lesion also enhanced with gadolinium. 42% of patients had a CSF pleocytosis with a mean WBC count of 38±13 cells (range 0-950 cells). 50% of the patients revealed an elevated protein level (mean protein level 75±14 g/dl). Table 4 lists some of the radiologic features that distinguish various acute myelopathies.

6.2. Monophasic vs. Recurrent TM

75-90% of TM patients experience monophasic disease and have no evidence of multi-systemic or multiphasic disease. Most commonly, symptoms will stop progressing after 2-3 weeks and spinal fluid and MRI abnormalities will stabilize and then begin to resolve. There are several features, however, that predict recurrent disease (Table 5). Patients with multifocal lesions within the spinal

Table 4. Imaging Features of Acute Myelopathies

Imaging feature	Suggested diagnosis
Blood within the spinal cord (bright and dark T1 and T2 signal)	Vascular malformation such as cavernous angioma or dural AV fistula
Flow voids within spinal cord	Dural AV fistula or AVM
Central T2 signal abnormality	Venous hypertension
Ring-enhancing lesion	Infection or tumor (but consider course of IV steroids to rule out inflammatory process before progressing to biopsy)
Acute loss of vertical intervertebral disc height and corresponding T2 signal abnormality	Consider fibrocartilagenous embolism
Fusiform lesion extending over >3 sc segments	Consider Neuromyelitis optica or disease-associated TM
T2 bright lesion in white matter occupying less than 2 spinal cord segments in rostral-caudal extent and less than 50% of the cord diameter	Consider MS
T2 spinal cord lesion adjacent to disk herniation or spondylitic ridge, but lack of spinal cord compression	Consider dynamic spinal cord compression only during flexion or extension (flexion-extension x ray to determine the presence of abnormal spinal column mobility; MRI in flexion or extended position instead of in neutral position)

Table 5. Recurrent Vs. Monophasic TM

Characteristics	Monophasic	Recurrent
Spinal MRI	Single T2 lesion	Multiple distinct lesions or fusiform lesion extending over $\geq$ 3 spinal cord segments
Brain MRI	Normal	T2/FLAIR abnormalities
Blood Serology	Normal	$\geq$ 1 autoantibodies (ANA, dsDNA, phospholipid, c-ANCA)
SS-A	Negative	Positive
CSF Oligoclonal Bands	Negative	Postive
Systemic disease	None	Connective tissue disorder
Optic Nerve Involvement	No	Yes
CSF IL-6	Sustained elevation (>50 pg/ml) (research only)	Declining or normal (<50 pg/ml) (research only)

cord, demyelinating lesions in the brain, oligoclonal bands in the spinal fluid, mixed connective tissue disorder, or serum autoantibodies (most notably SS-A) are at a greater risk of recurrence (72). Preliminary studies suggest that patients who have persistently abnormal CSF cytokine profiles (notably IL-6) may also be at increased risk for recurrent TM, though these findings must be validated before they are utilized clinically (73). At the current time, we do not understand the relative contribution of these factors to gauge whether chronic immunomodulatory treatment is warranted in high-risk patients.

6.3. Prognosis

Some patients with TM may experience recovery in neurologic function regardless of whether specific therapy was instituted. Recovery, if it occurs, should begin within 6 months and the vast majority of patients show some restoration of neurologic function within 8 weeks (67, JHTMC case series). Recovery may be rapid during months 3-6 after symptom onset and may continue, albeit at a slower rate, for up to 2 years (44, 66, JHTMC case series). Longitudinal case series of TM reveal that approximately 1/3 of patients recover with little to no sequelae, 1/3 are left with moderate degree of permanent disability, and 1/3 have severe disabilities (4, 5, 10, 44, 65). Knebusch estimated that a good outcome with normal gait, mild urinary symptoms, and minimal sensory and upper motor neuron signs occurred in 44%. A fair outcome with mild spasticity but independent ambulation, urgency and/or constipation, and some sensory signs occurred in 33%, and a poor outcome with the inability to walk or severe gait disturbance, absence of sphincter control and sensory deficit 23%. The patient cohort we follow at Johns Hopkins is more severe with only 20% experiencing a good outcome by those definitions, likely a reflection of referral bias to a tertiary care center. Symptoms associated with poor outcome include back pain as an initial complaint, rapid progression to maximal symptoms within hours of onset, spinal shock, and sensory disturbance up to the cervical level (67). The presence of 14-3-3 protein, a marker of neuronal injury, in the CSF during the acute phase may also predict a poor outcome (74).

7. IMMUNOPATHOGENESIS OF TM

7.1. General Pathology of TM

The pathology of acute myelopathies reflects the heterogeneous nature of thee disorders. Few studies to date have described the pathology of acute myelitis and the majority of these pathological descriptions are clinicopathological case reports (75-77). Pathological data from autopsies and biopsies from patients with suspected spinal cord lesions later confirmed to be associated with myelitis have been studied at the JHTMC {unpublished data). These data further confirm that TM is an inflammatory condition associated with immune-mediated

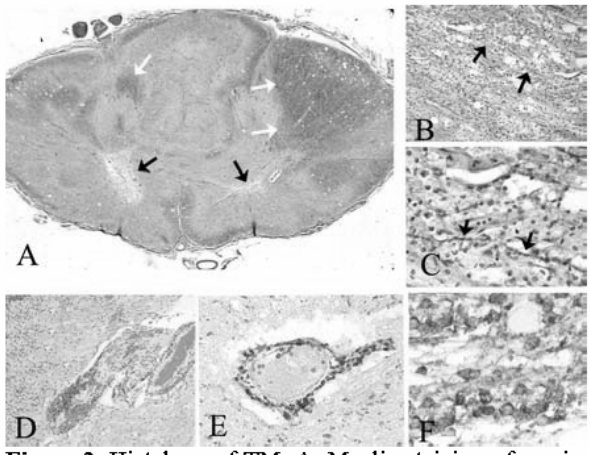

Figure 2. Histology of TM. A: Myelin staining of cervical spinal cord section from a patient who died during a subacute stage of TM. There are a few myelinated areas left (white arrows) and foci of cystic degeneration in the anterior horns (black arrows). The remaining of the spinal cord shows chronic inflammation and demyelination (LFB/HE stain). B: An area of demyelination from the same spinal cord as A that shows areas of active myelin and fiber degeneration (LFB/HE stain). C: High magnification view of few myelinated fibers left in areas of active inflammation (LFB/HE stain). D: Focal area of acute inflammation and perivascular accumulation of inflammatory cells seen in a biopsy obtained from a patient with acute TM (H&E stain). E: Perivascular infiltration by T lymphocytes as demonstrated by immunocytochemistry in an area of active inflammation in a patient with TM (CD3 immunostaining). F: Infiltration by macrophages in an area of myelitis (HLA-Dr immunostaining).

mechanisms. Indeed, all patients who met criteria for the diagnosis of TM and had tissue sampling of the spinal cord (biopsy or autopsy) had inflammatory changes. These pathologic abnormalities invariably included focal infiltration by monocytes and lymphocytes into segments of the spinal cord and perivascular spaces and an invariable astroglial and microglial activation (Figure 2). The magnitude and extension of these inflammatory features vary and are determined by the etiological factors and the temporal profile of the myelopathic changes. The presence of white matter changes, demyelination and axonal injury is prominent in postinfectious myelitis. However, involvement of the central compartment of the cord, gray matter or neurons is also prominent in some cases, a finding that supports the view that in TM, both gray and white matter compartments may be equally affected. In some biopsies obtained during the acute phases of myelitis, infiltration of CD4+ and CD8+ lymphocytes along with an increased presence of monocytes is quite prominent. In biopsies obtained during subacute phases of myelopathic lesions, prominent monocyte and phagocytic-macrophage infiltration is observed. In some cases, autoimmune disorders such as SLE, lead to vasculitic lesions that produce focal areas of spinal cord ischemia without prominent inflammation (78). These immunopathological observations further confirm that TM is an immune-mediated disorder that involves cellular reactions and perhaps humoral factors that injure compartments of the spinal cord.

7.2. Immunopathogenesis of TM

The pathogenesis of TM is believed to be immune-mediated. In support of this mechanism, most patients have CSF pleocytosis and blood-brain barrier breakdown within a focal area of the spinal cord. In 30-60% of idiopathic TM cases, there is an antecedent respiratory, gastrointestinal or systemic illness (2-5, 7, 10, 65).

Therefore, in TM patients, it is likely that there is abnormal activation of the immune system resulting in inflammation and injury within the spinal cord. Several potential mechanisms for this have been proposed (19). A variety of infectious agents encode molecular mimics (e.g. proteins, glycolipids or proteoglycans) that resemble self antigens. Generation of an immune response to the mimic then may result in cross-reactive immune activation against self tissue. This mechanism is well established in Guillain-Barré syndrome, a monophasic, usually post-infectious inflammatory disorder of peripheral nerves (79-81). More recent studies have implicated this mechanism in a variety of CNS inflammatory diseases as well (82-85). The molecular mimic may stimulate T lymphocytes, thus breaking down the immune tolerance of self tissue that had previously existed (82). Alternatively, the molecular mimic may stimulate the generation of antibodies which cross react with self antigens, resulting in immune complex formation and activation of complement-mediated or cell-mediated injury to self tissue (83-85). These autoantibodies may function as an agonist to cellular receptors, altering cellular signaling, metabolism or activity (85).

Another link between an antecedent infection and the development of TM may be the fulminant activation of lymphocytes by microbial superantigens. Superantigens are microbial peptides that have a unique capacity to stimulate a large number of lymphocytes in a unique manner compared with conventional antigens. The stimulation of large numbers of lymphocytes may trigger autoimmune disease by activating autoreactive T cell clones (86, 87). Whereas very little is known definitively about the inciting cause of inflammation in the CNS of patients with TM, nothing is currently understood about the mechanisms of tissue injury in this inflammatory disease.

We recently have carried out a series of investigations that describe immune derangements in TM patients (73, 88). We have found that interleukin 6 (IL-6) levels in the spinal fluid of TM patients were markedly elevated compared to control patients and to MS patients. While relatively low levels of IL6 in MS patients did not correlate with disability, IL6 levels in TM patients strongly correlated with and were highly predictive of disability. IL-6 levels in TM patient's CSF correlated with Nitric Oxide (NO) metabolites, which also correlated with disability. We suggest, therefore, that marked upregulation of IL6 correlates with increased NO production and that this

elevation is etiologically related to tissue injury leading to clinical disability in TM.

8. TREATMENT OF TM

8.1. Intravenous Steroids

Intravenous steroid is often instituted for patients with acute TM. Though there is no randomized, placebo-controlled study that supports this approach, evidence from related disorders and clinical experience support this treatment (89-93). Additionally, there are several small studies which support the administration in patients with TM (94-97). A study of five children with severe TM who received Solumedrol (1g/1.73 meter squared per day) for 3 or 5 consecutive days followed by oral prednisone for 14 days reported beneficial effects compared to ten historic controls (96). In the steroid treated group, the median time to walking was 23 days vs. 97 days, full recovery occurred in 80% vs. 10%, and full motor recovery at 1 year was present in 100% vs. 20%. No serious adverse effects from the steroid treatments occurred.

Other investigations have suggested that intravenous steroid administration may not be effective in TM patients (44, 67, 98). The most significant of these manuscripts (98) compared 12 TM patients seen between 1992-1994 who did not get steroids with 9 patients seen between 1995-1997 who did. Although the authors claimed that there was no statistically significant difference in the outcomes between the groups, it is evident that the TM patients who received steroids were more likely to recover and fewer had a poor outcome on the Barthel Index (33% vs. 67%). Therefore, the available evidence suggests that intravenous steroids are somewhat effective if given in the acute phase of TM. However, these studies did not rigorously define TM and therefore likely included patients with non-inflammatory myelopathies.

At our center, we routinely offer intravenous methylprednisolone (1000 mg) or dexamethasone (200 mg) for 3-5 days unless there are compelling reasons to avoid this therapy. The decision to offer continued steroids or add a new treatment is often based on the clinical course and MRI appearance at the end of 5 days of steroids.

8.2. Plasma Exchange

Plasma exchange (PLEX) is often initiated if a patient has moderate to severe TM (i.e. inability to walk, markedly impaired autonomic function and sensory loss in the lower extremities) and exhibits little clinical improvement within 5-7 days of intravenous steroids. PLEX has been shown to be effective in adults with TM and other inflammatory disorders of the CNS (99-101). Predictors of good response to PLEX include early treatment (less than 20 days from symptom onset), male sex, and a clinically incomplete lesion (i.e. some motor function in the lower extremities, intact or brisk reflexes) (102). It is our experience that PLEX may significantly improve outcomes of patients with severe (though incomplete) TM and who have not significantly improved on intravenous steroids.

8.3. Other Immunomodulatory Treatment

No controlled information currently exists regarding the use of other treatment strategies in patients with acute TM. Some clinicians consider pulse dose intravenous cyclophosphamide (500-1000 mg/m2) for patients with TM that continues to progress despite intravenous steroid therapy. It is the experience at our center that some patients will respond significantly to intravenous cyclophosphamide and this treatment is worthy of consideration while we await double blinded placebo trials. However, cyclophosphamide should be administered under the auspices of an experienced oncology team, and caregivers should monitor the patient carefully for hemorrhagic cystitis and cytopenias.

CSF filtration is a new therapy, not yet available in the United States, in which spinal fluid is filtered for inflammatory factors (including cells, complement, cytokines and antibodies) prior to being reinfused into the patient. In a randomized trial of CSF filtration vs. PLEX for AIDP, CSF filtration was better tolerated and was at least as effective (103). Clinical trials for CSF filtration are currently being initiated.

Chronic immunomodulatory therapy should be considered for the small subgroup of patients with recurrent TM. Although the ideal treatment regimen is not known, we consider a two year course of oral immunomodulatory treatment in patients with two or more distinct episodes of TM. We most commonly treat patients with azathioprine (150-200 mg/d), methotrexate (15-20 mg/week) or mycophenolate (2-3 gm/day), though oral cyclophosphamide (2 g/kg/day) may also be used in patients with systemic inflammatory disease. On any of these medicines, patients must be followed for transaminitis or leukopenias.

8.4. Long Term Management

Many patients withTM will require rehabilitative care to prevent secondary complications of immobility and to improve their functional skills. It is important to begin occupational and physical therapies early during the course of recovery to prevent the inactivity related problems of skin breakdown and soft tissue contractures that lead to loss of range of motion. The principles of rehabilitation in the early and chronic phases after TM are summarized in Tables 6 and 7. During the early recovery period, family education is essential to develop a strategic plan for dealing with the challenges to independence following return to the community. Assessment and fitting for splints designed to passively maintain an optimal position for limbs that cannot be actively moved is an important part of the management at this stage.

The long term management of TM requires attention to a number of issues. These are the residual effects of any spinal cord injury including TM. In addition to chronic medical problems, there are the ongoing issues

Table 6. Chronic Management of Patients with TM

General	• Rehabilitation is critical • Strongly consider inpatient rehabilitation • Daily land based and/or water based therapy for 8-12 weeks • Daily weight bearing for 45-90 minutes. Standing frame if non-ambulatory • bone densitometry: Vitamin D, Calcium • Look for depression and treat if interfering with rehabilitation
Bladder dysfunction	• Assess ability to void spontaneously • Avoid Crede (bearing down to initiate urination) since may be dangerous • Check post void residual. If >80cc, consider clean intermittent catheterization (goal less than 400 cc volumes) • Cystometrogram not required in acute phase • Anticholinergic Rx if sig. urgency • Cranberry juice for urine acidification
Bowel dysfunction	• high fiber diet • increased fluid intake • digital disimpaction • bowel program: colace, senokot, dulcolax, docusate PR, bisacodyl in a water base, miralax, enemas PRN
Weakness	• strengthening program for weaker muscles • Passive and active ROM • PT/OT consultation • Splinting or orthoses when necessary
Pain or dysesthesias	• ROM exercises • gabapentin • carbamazepine • nortriptyline • tramadol • avoid narcotics if possible
Spasticity	• ROM exercises • aquatherapy • baclofen • tizanidine • diazepam • botulinum toxin • tiagabine

Early Rehabilitation Principles (weeks to months)

Table 7. Chronic Management of Patients with TM

General	• Avoid secondary complications • Examine for scoliosis in patients with high/severe lesions. • Serial flexion/extension x-ray of back to follow angle • Skin hygiene to avoid breakdown • Treat fatigue: Amantidine, Methylphenidate, Modafinil, CoQ10 • Bone densitometry: Vitamin D, Calcium, Bisphosphanate therapy • Consider and treat depression
Bladder dysfunction	• Urodynamics study for irritative or obstructive symptoms • Anticholinergic drug if detrusor hyperactive: extended release Ditropan or Detrol • Adrenergic blocker if sphicter dysfunction: Flomax, etc • Clean intermittent catherterization is safe for long term • Cranberry juice/Vitamin C for urine acidifcation • Consider sacral nerve stimulation
Bowel dysfunction	• high fiber diet • increased fluid intake • digital disimpaction • bowel med program: colace, senokot, dulcolax, docusate PR, bisacodyl in a water base, miralax, enemas PRN
Sexual dysfunction	• Phosphodiesterase V inhibitors
Weakness	• Strengthening program for weaker muscles • Passive and active ROM • Splinting or orthoses when necessary • Continued land-based and water based therapy • Ambulation devices when appropriate • Daily weightbearing for 45 –90 minutes. Standing frame if non-ambulatory • Orthopedics evaluation if joint imbalance
Pain or dysesthesias	• ROM exercises • gabapentin • carbamazepine • nortriptyline • tramadol • topical lidocaine (patch or cream) • intrathecal baclofen or opioids
Spasticity	• ROM exercises • aquatherapy • baclofen • tizanidine • diazepam • botulinum toxin • tiagabine • intrathecal baclofen trial

Late Rehabilitation Principles (months to years)

of ordering the appropriate equipment, re-entry into the school for children and community, and coping with the psychological effects of this condition by the patients and their families.

Spasticity is often a very difficult problem to manage. The key goal is to stay flexible with a stretching routine using exercises for active stretching and a bracing program with splints for a prolonged stretch. These splints are commonly used at the ankles, wrists, or elbows. An appropriate strengthening program for the weaker of the spastic muscle acting on a joint and an aerobic conditioning regimen are also recommended. These interventions are supported by adjunctive measures that include anti-spasticity drugs (e.g. diazepam, baclofen, dantrolene, tiagabine), therapeutic botulinum toxin injections, and serial casting. The therapeutic goal is to improve the function of the patient in performing specific activities of daily living (i.e. feeding, dressing, bathing, hygiene, mobility) through improving the available joint range of motion, teaching effective compensatory strategies, and relieving pain.

Another major area of concern is effective management of bowel and bladder function. A high fiber diet, adequate and timely fluid intake, medications to regulate bowel evacuations, and a clean intermittent urinary catheterization are the basic components to success. Regular evaluations by medical specialists for urodynamic studies and adjustment of the bowel program are recommended to prevent potentially serious complications.

9. SPECULATIONS ON FUTURE TREATMENTS OF TM

Work over the last few years has begun to reveal fundamental immune abnormalities in patients with TM and related neuroimmunologic disorders. The generation of autoantibodies and the presence of abnormally elevated cytokine levels in the spinal fluid are likely to be important immunopathogenic events in many patients with TM. Though TM is a heterogeneous syndrome that is associated with distinct pathologies, recent classification strategies have attempted to identify patients with likely similar immunopathogenic events. While current therapies are largely non-specific, future therapies will be more specifically targeted to those critical immunopathogenic events in TM. For example, evolving strategies will more effectively identify autoantibodies and the antigen to which they respond (104, 105), making it possible to develop specific targets to block the effects of these autoantibodies. Additionally, several strategies exist and more are currently being developed that specifically alter cytokine profiles or the effects of these cytokines within the nervous system. However, a cautionary note exists from recent studies examining TNF- α modulation in patients with multiple sclerosis or systemic rheumatologic disease: paradoxical demyelination may be triggered by TNF-α reduction in the blood (106). These findings may suggest that secondary alterations in immune system function may occur in response to blockade of any single pathway and that a "cocktail approach" aimed at halting multiple pro-inflammatory pathways may be ideal.

10. CONCLUSIONS

TM is a clinical syndrome caused by focal inflammation of the spinal cord. Many cases are post-infectious and are thought to be due to a transient abnormality in the immune system that results in injury to a focal area of the spinal cord. Recent studies have emphasized the need to classify TM according to whether there is evidence of systemic disease or multi-focal CNS disease. The importance of this may be that distinct treatment strategies are offered to patients with distinct forms of TM. Though the causes of TM remain unknown, recent advances have suggested specific cytokine derangements that likely contribute to sustained disability. Patients are often left with sustained disability due to injury of motor, sensory or autonomic neurons within the spinal cord. Future research will attempt to define triggers for the immune system derangements, effector mechanisms that propagate the abnormal immune response and cellular injury pathways initiated by the inflammatory response within the spinal cord. Ultimately, this may allow us to identify patients at risk for developing TM, specifically treat the injurious aspects of the immune response, and/or offer neuroprotective treatments which minimize the neural injury that occurs in response to the inflammation.

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NEUROMYELITIS OPTICA: CURRENT CONCEPTS

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1. ABSTRACT

Neuromyelitis optica (NMO; Devic's syndrome or Devic's disease) is a clinically defined disorder consisting of optic neuritis (ON) in combination with myelitis. Its nosological relationship to other central nervous system demyelinating diseases remains uncertain. Advances in understanding of its natural history and pathophysiology may eventually allow determination of whether idiopathic NMO represents a specific disease. This article summarizes recent progress in these areas of NMO research.

2. INTRODUCTION

The term "neuromyelitis optica" (NMO; also known as Devic's syndrome or Devic's disease) describes the coexistence of optic neuritis and myelitis. Recognized for over a century (1), the concept of NMO as a distinct entity has engendered much unresolved controversy. Often diagnosed and classified as a severe form of multiple sclerosis (MS), arbitrary diagnostic criteria that take into account unique clinical, laboratory, and neuroimaging characteristics allow identification of an NMO syndrome that appears to have a natural history and treatment response distinct from "typical" MS (2,3). Recent immunological and immunopathological studies derived from patients with clinical NMO add supportive evidence to the concept of idiopathic NMO as a distinct disease, or at least a distinct entity within the nosological conceptualization of idiopathic demyelinating diseases of the central nervous system.

3. WHAT IS NEUROMYELITIS OPTICA?

3.1. Epidemiology and Disease Associations

The clinical combination of optic neuritis and myelitis has been reported in association with many systemic autoimmune disorders (systemic lupus erythematosus, Sjögren syndrome, mixed connective tissue disease, among others), infectious diseases (pulmonary tuberculosis and a myriad of viral illnesses), and immunizations (3-5). The NMO syndrome, therefore, includes a wide spectrum of para-infectious disorders and complications of systemic autoimmunity. In some instances, the neurological symptoms evolve as a complication of a systemically active connective tissue disorder. Most commonly, however, NMO is the most prominent or only clinically apparent disease and the markers of systemic autoimmunity are found during its investigation. It is not clear whether the same pathophysiological basis is operative in these overlapping syndromes.

Neuromyelitis optica is predominantly a disorder of women (80-90% of cases) and the median age of onset is late in the fourth decade, about 10 years later than for typical MS (6). Most North American NMO patients are Caucasian but there is a distinct overrepresentation of people with Asian and African ancestry compared with typical MS. Although familial cases have been reported, NMO is generally a sporadic disease (7-10). It may differ genetically from typical MS in that most studies do not find an association with the HLA-DRB1*1501 allele that is associated with typical MS (11-13).

Table 1. Various Proposed Diagnostic Criteria for Neuromyelitis Optica

Mandler et al. (16)
• Clinical: Acute involvement of spinal cord and ON, either coincidental or separated by months or years, independent of subsequent progression, but without other clinical features at any time during disease course • Imaging: Normal-appearing brain MRI. Enlargement and cavitation on spinal cord MRI • CSF: Decreased serum/CSF albumin ratio, normal IgG synthesis rate and usually absence of oligoclonal bands
O'Riordan et al. (17)
• Severe (more or less complete) transverse myelitis • An acute unilateral or bilateral optic neuropathy • No clinical involvement beyond the spinal cord and optic nerves • Illness may be monophasic or multiphasic
Wingerchuk et al. (6)
Diagnosis requires all absolute criteria AND one major supportive criterion OR two minor supportive criteria
❑ **Absolute criteria**
• Optic neuritis • Acute myelitis • No clinical disease outside of the optic nerves and spinal cord
❑ **Major Supportive Criteria**
• Negative brain MRI at disease onset (normal or not meeting • radiological diagnostic criteria for MS) • Spinal cord MRI with T2 signal abnormality extending over >3 vertebral segments • CSF pleocytosis (>50 WBC/mm3) OR > 5 neutrophils/mm3
❑ **Minor Supportive Criteria**
• Bilateral optic neuritis • Severe ON with fixed visual acuity worse than 20/200 in at least one eye • Severe, fixed, attack-related weakness (MRC grade 2 or less) in one or more limbs

3.2. Diagnosis and Discrimination from Multiple Sclerosis

Neuromyelitis optica is generally classified as one of the subtypes of idiopathic inflammatory demyelinating diseases of the central nervous system (CNS), of which MS is the prototype. The term NMO is applied to patients who have experienced optic neuritis (unilateral or bilateral) and myelitis without clinical evidence of demyelination affecting brain white matter. It used to be considered a monophasic disorder (for a summarized account of the history of NMO, see Wingerchuk and Weinshenker (3)) but is more often relapsing-remitting. This was recognized early on in Japan, where cases of relapsing NMO were diagnosed as "opticospinal" or "Asian" variants of MS, distinguishing it clearly from "Western" or typical MS. The "opticospinal variant" observed in Asia appears to have the same demographic, clinical, neuroimaging, and pathological characteristics as cases of relapsing NMO reported from North America with the exception that systemic autoimmunity is recognized much more commonly in the West (14). The characteristics of a "pure" subgroup of opticospinal MS (clinical opticospinal disease, normal head MRI except for optic nerve abnormalities, and more than 5 years of clinical follow-up) are virtually identical to cases of relapsing NMO (13).

Classification problems persist because many patients with NMO meet MS diagnostic criteria and some patients with MS may present with ON and myelitis (15). Gradually revised (but still arbitrary and largely unvalidated) diagnostic criteria attempt to discriminate NMO from MS by emphasizing several apparent clinical, laboratory, and neuroimaging differences (Table 1) (6,16,17). Clinically, NMO usually presents with more dramatic acute exacerbations. Optic neuritis attacks are generally severe and exhibit poor recovery; a minority of patients experience simultaneous bilateral ON but the frequency is significantly higher than in MS. Myelitis attacks in NMO are often fulminant, bilateral, "complete transverse myelitis" and accompanied by pain and a greater degree of residual neurological impairment.

Although very severe attacks suggest NMO, there is substantial overlap in clinical severity between NMO and MS; therefore, other factors assume greater diagnostic importance. Laboratory features suggestive of NMO include lack of cerebrospinal fluid (CSF) oligoclonal banding and immunoglobulin abnormalities and seropositivity for multiple autoimmune markers such as antinuclear antibody (ANA), extractable nuclear antigen (ENA), and thyroid autoantibodies (6,17). However, magnetic resonance imaging (MRI) studies of the head and spinal cord appear to have the highest discriminative value. In most cases, head MRI is normal (except for possible optic nerve abnormalities) or demonstrates only a few punctate nonspecific abnormalities that do not fulfill radiological criteria for MS. Over time, brain MRI may reveal an increasing number of cerebral white matter lesions but only in a small minority of cases will these meet MRI criteria for MS and they are rarely symptomatic.

Spinal cord MRI is probably the most useful discriminative test. Most patients with NMO have a contiguous, longitudinally extensive, gadolinium-enhancing cord lesion that spans three or more vertebral segments (Figure 1). Spinal cord plaques in MS uncommonly exceed two segments in length and lesions over more than three segments are exceedingly rare.

The specificity of these criteria for discrimination of NMO from MS is not known. This is in part due to the lack of a gold standard for NMO diagnosis. Prospective studies that evaluate clinical symptoms and signs, CSF and MRI findings at disease onset and correlate them with a gold standard for NMO diagnosis are needed. An objective biological or pathological marker that is associated with NMO but not with MS is needed. A potentially important advance in NMO research was reported in April, 2003 (18,19). Investigators at Mayo Clinic presented data

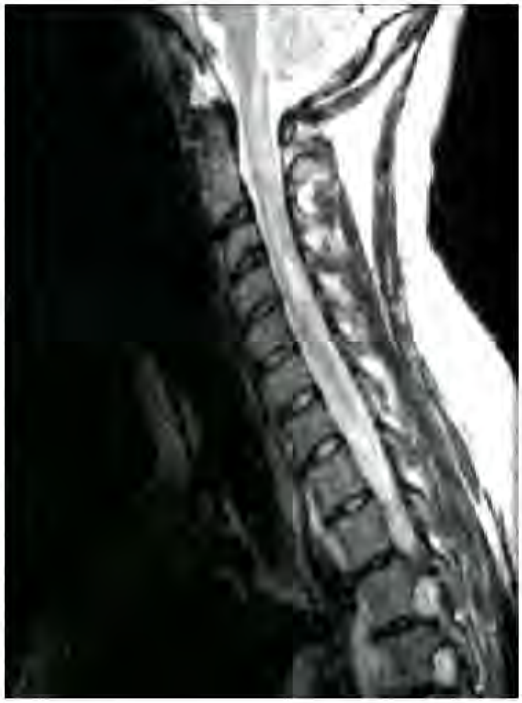

Figure 1. Magnetic resonance imaging of the cervical spinal cord (sagittal T2-weighted images) demonstrates a longitudinally extensive cord lesion extending beyond the cervicomedullary junction.

regarding a serum autoantibody that appears to discriminate NMO from typical multiple sclerosis. This indirect immunofluoresence assay reveals a distinct staining pattern (termed 'NMO-IgG') that appears to selectively bind a target associated with CNS capillaries, pia, and subpia (18). Within a group of 48 patients with definite or probable NMO, 54.2% were seropositive for NMO-IgG compared with none of 20 patients with typical MS (19). This finding represents the first specific biological marker for NMO and, if validated, may be a powerful diagnostic tool available at disease onset.

4. WHAT IS THE NATURAL HISTORY OF NMO?

Neuromyelitis optica remains a construct, i.e., an empirically derived diagnostic entity conceived from clinical experience that suggests that patients with NMO behave differently than those with "typical" MS. Patients fulfill NMO diagnostic criteria if they have presumed demyelinating isolated optic nerve and spinal cord lesions, negative head MRI and CSF oligoclonal banding, and a longitudinally extensive spinal cord lesion of three or more vertebral segments. Patients who fulfill this syndrome definition proceed along either a monophasic or a relapsing course. A monophasic course, which probably occurs in less than 25% of patients, is defined by co-occurrence of either unilateral or bilateral ON and a single episode of myelitis and extended follow-up (several years) that reveals no further exacerbations. Most patients experience a relapsing course, in which the index events of ON and myelitis may be many weeks or even years apart but attacks of ON, myelitis, or both recur over the next months to years. Those patients destined for a relapsing course usually declare it relatively early: after fulfilling NMO diagnostic criteria, 55% have their first optic nerve or spinal cord relapse within one year; this proportion increases to 78% at three years and 90% at five years (6).

For the purposes of prognostication and long-term treatment, clinical and laboratory features that are available early in the disease course and are highly discriminative between the relapsing and monophasic NMO varieties would be very useful. Unfortunately, there are no CSF or imaging variables that independently allow course and severity prediction, but some clinical features are useful. The clinical impression that patients who present with a combination of ON and myelitis simultaneously or in rapid succession (over a few days) are much more likely have a monophasic course was recently confirmed by observational data utilizing a longitudinal database of 80 NMO patients (20). In the largest contemporary series, the median interval between the first clinical event and the development of bilateral ON and myelitis (traditional definition of NMO) was 5 days (range 0 to 151 days) in the monophasic group versus 166 days (range 2 to 730 days) for the relapsing group (6). This single variable is quite powerful; the relative risk of relapsing disease is increased by 2.16 for every month increase in the first inter-attack interval. Other independent predictors include female sex (relative risk = 10.0 female versus male) and less severe motor impairment with the initial myelitis event (relative risk = 0.48 for every point decrease on a motor weakness scale). Complete paraplegia occurs at the clinical nadir of the first myelitis attack in 70% of monophasic patients compared with only 31% of those who later develop relapsing disease. Although the first ON attacks are also typically more severe in those with a monophasic course, with complete loss of light perception occurring in more than 50% versus about 28% of relapsing patients, this is not an independent risk factor for disease course prediction. These prognostic variables may be useful when considering the use of preventative immunosuppressant therapies early in the disease course and in planning epidemiological and therapeutic studies.

Although the ON and myelitis attacks that define monophasic NMO may be more severe, long-term neurological impairment and disability is measurably less in this group than for relapsing disease. This is because most patients with monophasic disease experience some recovery and then remain neurologically stable because no further relapses occur. Although 22% remained functionally blind (20/200 vision or worse) in at least one affected eye, more than 50% of ON episodes demonstrated eventual recovery to 20/30 visual acuity or better. Permanent neurological impairment in monophasic disease is more often related to myelitis; most patients experience at least a moderate degree of both limb weakness and bowel and/or bladder dysfunction. Permanent monoplegia or paraplegia occurs in 31%. Five-year survival of this group is approximately 90% and cause of death is usually unrelated to NMO or a medical complication of immobility (6,20).

Relapsing NMO follows an unpredictable course consisting of clusters of attacks months or years apart. When considering only exacerbation frequency, this disease course resembles its relapsing-remitting "typical MS" counterpart. When attack severity and cumulative disability are considered, however, the natural history of relapsing NMO is markedly different from that of a typical MS cohort (6). A mere five years from disease onset, more than 50% of people with relapsing NMO have at least one eye with less than 20/200 acuity or are nonambulatory due to attack-related paraplegia or quadriplegia. This is in contradistinction to MS, in which most patients recover from early attacks completely or nearly so and suffer mild disability until the secondary progressive phase of the disease takes hold about 10-15 years later. Progressive disease is not a feature of NMO. While the prognosis for most monophasic patients is to maintain some degree of independence (despite moderate visual and motor deficits), those with relapsing disease face early, stepwise accumulation of greater disability.

As in typical MS, relapse frequency is extremely variable in NMO. Several attacks may strike over a few months or remissions lasting a more than a decade may occur. In one series followed over a median of 16.9 years, the median number of relapses was five (range 1 to 18) (6). As in monophasic NMO, a progressive phase of neurological deterioration is uncommon, although there are many patients who seem to have rapid, sometimes stepwise, deterioration when they embark on a taper following a period of continuous corticosteroid therapy.

The long-term survival rate for relapsing NMO is probably the lowest of all relapsing-remitting CNS demyelinating syndromes. Severe, ascending cervical myelitis causing respiratory failure affected as many as one-third patients in one series (though the true incidence is probably lower given improvements in diagnostic accuracy and widening of the NMO spectrum). In one longitudinal series, five-year survival was 68% with all deaths secondary to myelitis-related respiratory failure (6). Improvements in medical and supportive care have likely reduced these estimates but the point is that the disease has the potential for both serious morbidity and mortality. Predictors of mortality in relapsing NMO include a history of systemic autoimmune disease (relative risk = 4.15), greater exacerbation frequency during the first two years of disease (relative risk = 1.21 per attack) and better motor recovery following the first myelitis attack (20).

In summary, the natural history of disease for patients who fulfill NMO diagnostic criteria is markedly different than that of typical MS. Progress has been made in predicting the course and its severity, and this may be useful ultimately in study design and clinical practice.

5. WHAT IS THE PATHOLOGICAL BASIS OF NMO?

The cause of NMO is not known. Its clinical and basic pathological features have led many to conclude that it represents a severe, topographically restricted MS variant. The fact that multiple infectious and systemic autoimmune diseases have been associated with NMO suggests that a single pathological basis for the clinical syndrome is unlikely. The situation is complicated by cases of the NMO phenotype occurring as an apparent complication of classic autoimmune diseases such as systemic lupus erythematosus and Sjögren syndrome (21,22). In addition, serum autoantibodies (antinuclear antibody, extractable nuclear antigen, etc.) associated with those systemic diseases are often found in patients with NMO who do not have clinically evident systemic disease (6,17). These associations suggest that at least some cases may be caused by B-cell mediated processes.

5.1. General Pathology

Pathological studies of NMO optic nerve specimens are limited and generally yield nonspecific demyelination that is more extensive than seen in most MS cases of similar duration (23,24). Brain parenchyma is usually normal but may reveal scattered areas of minor perivascular infiltrates and patchy demyelination or gliosis.

Study of NMO spinal cord lesions at various stages of maturity has been more informative. Acute lesions often expand the cord and are associated with softening, cavitation, and necrosis (24-27). The inflammatory infiltrate is vigorous and includes a high proportion of polymorphonuclear cells. Depending on the severity of the myelitis attack, pathological findings range from modest perivascular inflammatory demyelination to complete hemorrhagic, necrotic destruction of both gray and white matter. In many cases, large numbers of eosinophils are often found in the inflammatory infiltrate (27). The intense eosinophilic and neutrophilic inflammatory response is quite distinct from findings in typical MS. The role of the distinct eosinophilic response in NMO is unknown; it may be a primary response or reflect secondary activation by the C5a component of complement (27).

A potentially discriminative gross pathological abnormality in NMO is the presence of hyalinized medium-sized spinal cord arteries (16,17,27-29). This vascular feature is usually found in conjunction with parenchymal necrosis and a macrophage-predominant inflammatory infiltrate and is not observed in MS. It is not known whether these vascular abnormalities are a clue to the primary immunopathogenesis of NMO lesions or are a nonspecific secondary phenomenon.

5.2. Immunopathology

Immunopathological advances have strengthened the case for humoral immunity underlying the pathogenesis of NMO. Using spinal cord biopsy and autopsy tissue samples, Lucchinetti *et al* discovered prominent IgG and C9 neoantigen (a marker of complement activation) deposits within regions of active myelin destruction and around vessel walls where there was vascular proliferation and fibrosis (27). This finding provides an early indication that the hyalinized vasculature seen on gross examination may be relevant to NMO pathophysiology.

The autoantigen(s) responsible for NMO pathogenesis are not known. The recent discovery of the NMO-IgG serum autoantibody marker (described in Section 3.2) is an important first step in antigen

identification (18,19). This highly specific staining pattern is associated with capillaries in cerebellar cortex and midbrain but the spectrum of CNS regions involved and the precise antigen associated with this marker are not yet known.

Animal models of MS, such as experimental allergic encephalomyelitis (EAE), assist in the understanding of CNS inflammatory demyelination mechanisms. The clinical and pathological phenotype of varies depending upon the genetic strain of the animal and the type, timing, and route of antigen exposure. Induction of EAE using the antigen myelin oligodendrocyte glycoprotein (MOG) can result in inflammatory lesions restricted to the optic nerve and spinal cord (30). MOG-induced EAE can actually produce several other patterns of CNS pathology resembling clinically recognizable MS phenotypes such prototypic MS, optic neuritis, and the acute Marburg variant of MS (31). Therefore, other genetic or environmental factors may be more important in determining the pathological and clinical expression of such idiopathic inflammatory CNS demyelinating disorders.

It is not known why the optic nerves and spinal cord are preferentially affected in NMO. Over years of follow-up, brain MRI can detect dissemination of cerebral white matter lesions, but these are generally asymptomatic, punctate, and subcortical in location. Lucchinetti *et al* postulated that antibody-mediated injury might be more likely in sites of a less effective blood-brain barrier (27); this is indirectly supported by various EAE models that have a predominance of optic nerve and spinal cord lesions.

6. THERAPY

6.1. Acute Exacerbations and Supportive Care

Acute therapy is often the foremost consideration at disease presentation, especially when patients experience rapidly progressive, ascending paraplegia, quadriplegia, or respiratory failure. There are no reports of structured therapeutic trials of first-line treatment for NMO exacerbations. Parenteral corticosteroids are empirically used and appear effective. Many clinicians use intravenous methylprednisolone 1000 mg daily for five consecutive days. I usually begin oral prednisone afterward if the patient is not already taking it as part of an immunosuppressive regimen for NMO attack prevention.

Corticosteroid-refractory attacks are not an uncommon occurrence in NMO. Although various second-line therapies such as intravenous immune globulin have been employed, the only existing controlled evidence supports the use of plasmapheresis. A randomized, double-blind, crossover trial that included many NMO patients demonstrated that compared with sham exchanges, plasmapheresis (seven exchanges, each of ≅55 ml/kg, administered every other day) resulted in clinically meaningful improvement in 42% of patients with various forms of severe, idiopathic inflammatory demyelinating disease (32). A retrospective review found that six of ten NMO patients with severe, corticosteroid-refractory NMO attacks demonstrated moderate or marked clinical improvement shortly after plasmapheresis was initiated (33). Predictors of response included early treatment initiation, male sex, and preserved muscle stretch reflexes. Therefore, if patients with severe attacks worsen during corticosteroid therapy or do not demonstrate improvement very shortly after treatment completion, immediate implementation of plasmapheresis is recommended.

Patients with severe myelitis attacks requiring hospitalization become exposed to the medical risks immobility poses. Prevention of these complications is important and the clinician must be aware of the possibility that an ascending cervical myelitis may cause neurogenic respiratory failure. Patients at risk for this complication require close intensive care unit observation, ongoing monitoring of respiratory and bulbar status, and ventilatory assistance when necessary. Measures to prevent thromboembolic events, decubitus ulceration, aspiration pneumonia, and urinary tract infections are also required.

6.2. Preventative Immunotherapy

Therapy aimed at attack prevention is required for patients with relapsing NMO but not for those with monophasic disease. Perhaps the strongest argument for distinguishing between NMO and MS is that they each appear to require quite different chronic immune-based therapies. The treatments of choice for reduction of MS attack frequency are interferon-beta and glatiramer acetate (34). Although there are some exceptions, such as a report that interferon beta-1b seemed to benefit Japanese opticospinal MS (35) and a case report of clinical stability associated with glatiramer acetate treatment (36), anecdotal evidence from experienced clinicians suggests that these agents fail to significantly alter attack rate for most relapsing NMO patients.

There are no preventative therapies with proven efficacy for relapse suppression in NMO. In the only published prospective treatment study, seven newly diagnosed NMO patients remained stable for at least 18 months after initiation of a combination of azathioprine (2 mg/kg/d) and oral prednisone (1 mg/kg/d) (37). After two months, the prednisone dose was gradually reduced (by 10 mg every three weeks to 20 mg/d, then an even slower reduction to a maintenance dose of 10 mg/d). Most patients received maintenance doses of 75-100 mg azathioprine and 10 mg prednisone daily. During the 18-month follow-up period, no attacks were recorded and neurological impairment scores improved marginally. I usually employ a similar approach for patients with recently active disease, aiming for an initial target dose of 2.5 to 3 mg/kg/d of azathioprine and 40-60 mg/d of prednisone. If clinical stability is achieved and lasts for more than 8 weeks, I slowly taper the prednisone dose on alternate days (e.g., 5 mg every 2 weeks) in an attempt to reduce corticosteroid adverse effects. Once there is hematological evidence that azathioprine is active (mild decrease in leukocyte count and increase in mean corpuscular volume), the dose prednisone is reduced even more slowly (e.g., 5 mg every 4 weeks). Some patients are able to sustain remissions on azathioprine monotherapy but

others will develop steroid dependence, either because of perceived neurological worsening or documented new exacerbations when attempts to reduce the prednisone dose are made.

Various other immunosuppressive drugs have been used but in limited numbers and outside the realm of a structured study. Mycophenolate mofetil may be used in place of azathioprine though its onset of action may be no more rapid. For patients who "fail" the azathioprine and prednisone combination, I consider a trial of mitoxantrone, which is approved for use in rapidly worsening secondary progressive or relapsing-remitting MS. There are no data concerning the use of mitoxantrone in NMO. In some instances, such as when a cluster of attacks has recently occurred, I consider the use of mitoxantrone as first –line therapy to try to achieve a rapid remission. Other therapies aimed at B-cell modulation should be studied in the future based on the recent immunopathological discoveries in NMO. There may be a role for different drugs of this type to induce remission and to sustain it.

The importance of early diagnosis and treatment of relapsing NMO cannot be overstated. The dismal natural history of the disorder, which causes almost exclusively attack-related disability, mandates early and carefully monitored treatment aimed at relapse prevention. Controlled trials, planned and managed by multicenter collaborative efforts, are needed to determine optimal long-term therapy for NMO.

7. CONCLUSION

Significant advances in the last five years have allowed us to better understand the nosology, natural history, and treatment of NMO. We have taken major steps toward unraveling the immunohistochemistry and immunopathology of NMO and solidifying its place as a distinct clinic entity. Relapsing NMO needs to be recognized early in its course to allow early initiation of therapy and to prevent attack-related disability. The recent discovery of a serum autoantibody marker, if validated, may assist in achieving this goal and represents a milestone in the evolution of our knowledge about this uncommon but devastating disorder.

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Spasticity in Transverse Myelitis

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Transverse myelitis (TM) changes the lives of children and adults in many ways. Major motor problems include weakness, increased muscle tone or spasticity, decreased mobility in joints, and fatigue. Muscle spasms and contractures can be painful.

Spasticity is defined as a heightened response to stretch that is velocity dependent. This means that muscle tone increases with movements and usually decreases during sleep. The doctor or therapist evaluates for spasticity through observing the muscle tone during passive movement according to a number of different grading scales. Spasticity is caused by an injury along the spinal cord tract that starts in the brain's motor cortex called the upper motor neuron. The cell body sits in the outer surface of the brain in the area responsible for voluntary movement. The tract comes down through the brain and crosses to the opposite side of the spinal cord then makes a connection to the lower motor neuron. This nerve cell is in the spinal cord and sends a long process out all the way to the muscles of the arms, trunk, and legs. When there has been an injury to the upper motor neuron or its long tract, it results in the inability of the nervous system to produce movement in a relaxed or normal state. When the person tries to move or is passively moved, the tone increases in a specific pattern that limits voluntary movement.

There are other associated signs of upper motor neuron damage. One problem is called **clonus**, in which alternating contractions of opposing muscle groups appear to cause a reverberating circuit and rhythmic contraction of a joint. This occurs most commonly at the ankle when the calf muscles are stretched quickly. The neurologist checks for this by tapping the Achilles tendon with a reflex hammer and watching the response. Clonus can be sustained, and can be uncomfortable and embarrassing. Another associated problem is called the **Babinski** response. This is checked for by scratching the bottom of the foot with a sharp object, such as a key and watching the response of the toes. The great toe (big toe) moves up toward the face, if the reflex is abnormal and the other toes may fan out. This can make wearing shoes less comfortable and cause breakdown of the skin, if the reflex is set off while standing or walking. Spasticity scores can be generated to determine whether a patient is responding to a treatment. One of the most common is called the Ashworth score. It ranges from a score of 0 which is no spasticity up to 4 which is limb rigidity in flexion or extension. This scoring system has been modified, and there are other scales which can detect smaller changes. Spasticity may be helpful to some people for standing and weight bearing. If the spasticity is reduced too much, the patient may not be strong enough to stand for transfers or walking. If spasticity is too high, the patient may not be able to move the arms or legs for function. In the legs, it can make it difficult to move one leg at a time as in walking, and can cause a scissor-like problem where one leg moves forward too close to the midline and is obstructed by the opposite weight bearing leg.

In addition, spasticity that is present most of the time usually leads to limitation of joint movement or contractures. These contractures can become permanent, making daily activities more difficult and less comfortable. An example is that common flexion contractures that limit complete straightening of the hips and knees make lying flat in bed impossible. Turns during the night can be only backlying or sidelying, increasing the risk of skin breakdown (decubitus ulcers). When attempting to stand and walk, hip and knee flexion contractures cause one to expend more energy in weight bearing because it requires constant muscle activity, rather than allowing resting on ligaments. This contributes to muscle fatigue. Another common problem is contracture of the Achilles' tendon causing one to bear weight only on the toes and ball of the foot. This reduces the surface area of weight bearing and decreases stability of the base of support, and again contributes to falls and fatigue. Contractures in the adductor muscles (the muscles that bring the knees closer together) can make dressing and personal hygiene more difficult, resulting in loss of independence. Contractures make orthotics (braces) more difficult to use, resulting in a vicious cycle with progressive loss of range of motion.

Some things can increase spasticity. These include, but are not limited to, bladder infection or distention, bowel distention, ingrown toenails or other painful skin irritants, and deep venous thromboses (blood clots). Excitement and stress can also increase spasticity.

Treatment of spasticity is multifaceted. Range of motion can be maintained through physical therapy or a home stretching program. Strengthening the muscles opposite the spastic muscle groups helps balance the tone and joint positions. The use of orthotics to maintain the desired joint positions is very important, as no one can perform stretching and strengthening exercises throughout the day and night, especially when fatigued. Finally, having specific functional goals, such as maintaining ambulation (walking), wheelchair or driving positioning, transfers, and ability to turn at night prevent some of the dependence caused by TM.

Specialists that can help patients man-

age their spasticity include physical and occupational therapists, orthotists (bracemakers), neurologists and physiatrists, and the wheelchair vendors.

Finally, when these measures are not enough, medications can be used. The most commonly used medication is **baclofen**. This medication is available as a tablet or liquid. It is thought that it reduces the release of excitatory neurotransmitters (chemical messengers). Doses are gradually increased to monitor for side effects, such as somnolence (sleepiness), nausea, paresthesias (abnormal sensations), and even seizures when suddenly withdrawn. Rarely, the liver can be harmed by this medication.

Another medication with similar effects is **diazepam**. It is available as oral tablets, liquid, or rectal gel forms. This class of medicines reduces anxiety, but can also cause sedation. Dependence can develop with this medication, requiring higher doses over time.

Dantrolene works directly on muscles by preventing the release of calcium ions, in turn reducing muscle contraction. It is available as 25 mg tablets, but also as a powder for solution. Some people take very large doses, but there is potential for liver toxicity and requires monitoring blood work.

Tizanidine works by a different mechanism, increasing the inhibitory pathways in the spinal cord. It is available as 4 mg tablets taken every 6-8 hours. It affects the autonomic (automatic) nervous system functions and can reduce blood pressure, cause dry mouth, somnolence, weakness, and can even increase muscle spasms and tone so must be monitored. There are reports of abnormal liver functions, so blood work may be suggested by your doctor.

Other medicines are thought to reduce spasticity, though their main purpose is for other diseases. Examples include **gabapentin**, **amantadine**, and **4-aminopyridine**.

Botulinum toxin is an extremely potent neuromuscular blocker. In its natural form, it is highly toxic. It has been developed as an agent to decrease muscle contraction in dystonia and spasticity by direct injection into the affected muscle groups. The maximum effects are apparent within a few days after the injection. These effects typically wear off after a few months. Children and some adults require sedation for this procedure, and it is most accurate if performed in conjunction with an electromyogram to be sure the injection is into muscle rather than other tissues. While very costly, combining this therapy with rehabilitation maximizes the beneficial effects. Serial casting is used after injection to gradually regain lost range of motion, and ongoing therapy and stretching are important to prevent the loss of motion.

Other treatments of interest include electrical stimulation of peripheral nerves and muscles through the skin, or even through epidural electrodes (around the spinal cord). These are less well accepted. Some therapists use vibration, cold applications, topical anesthetics to reduce tone and some believe that acupuncture is helpful. These are much less studied and difficult to incorporate into a daily routine.

One surgical option is the placement of a baclofen pump under the skin of the abdomen. This is connected to a catheter. The system delivers the medication to the space around the spinal cord, and avoids the central nervous system side effects of the oral form. It requires regular replacement of the medication through a needle. There are rare complications including infection and scar formation. The placement requires anesthesia and the surgery and device are expensive. A test dose is given by injection of baclofen through a spinal needle, with doctor and therapist observing for improvement in muscle tone. Complications of the operation include spinal fluid collections, constipation, headache and spinal fluid leak. There can be kinking of the catheter, and the pump may malfunction if cold.

Another surgical option is called selective posterior rhizotomy. In this operation, the nerve roots bringing sensory information into the spinal cord are exposed and selectively and permanently cut. This reduces the muscle tone in the muscle supplied at that spinal cord root level. It requires anesthesia, and can result in bladder dysfunction and sensory problems.

Patients with spasticity frequently develop osteoporosis, and need to be sure to take in an adequate amount of calcium and vitamin D. Weight bearing is thought to improve bone health, even when support is needed for standing. Physical therapists can do periodic assessment to try to prevent long term problems, such as excessive wear and tear on shoulders from wheeling.

Although spasticity is a big problem in TM, there are a lot of avenues to reduce spasticity and complications. Not all treatments work in every individual, requiring trials and reassessment. Despite their availability, it is up to the individual person to be sure they are receiving these treatments that often increase function and independence. Be your own advocate!

The Right Stuff: Braces, Wheelchairs, and Environmental Modifications

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Introduction

Concentric environments interfacing with the world are a good way to think about the challenges facing someone with transverse myelitis. There is the **immediate environment** that includes the things that come in contact with the individual (e.g., wheelchair, assistive devices for performing daily living tasks, car). The **intermediate environment** is the personal living space and would include one's home. The **community environment** includes adapted public spaces like school and recreational areas. Lastly, the **natural environment** is the essentially unchanged spaces that provide a challenge, but are certainly not "off limits" (e.g., mountain climbing).

This article will review three areas that are essential components of the immediate and intermediate environments. They are lower extremity braces, wheelchairs, and living space modifications.

Ambulation

A major question and challenge for someone with acute transverse myelitis is whether he or she will be able to walk. If so, the questions of how far, how tiring will it be, and what kind of assistance will be needed are important.

Ambulation potential depends primarily on the level of spinal cord injury, but other factors may also play a part. These include energy expenditure related to level of injury, fitness, weight, spasticity, muscle contractures, and pain.

Categories of ambulation are defined as: 1. **Community** in which ambulation is used as the primary mode of mobility; 2. **Household** which includes walking within the home with relative independence and using a wheelchair as the primary mobility in the community; 3. **Exercise** which requires considerable assistance or energy expenditure (it is too great for ambulation to be functional); and 4. **Nonambulatory** which uses a wheelchair entirely.

The neurologic level of injury can be used to predict ambulation category. The level of T11 or below is associated with increased potential for walking. Individuals with complete involvement of the arms and legs do not become community ambulators. Regardless of the level of spinal cord injury, it is important to try for the highest level of weight bearing ambulation possible, because **mobility provides the ability** to overcome functional barriers. It increases self esteem and provides cardiopulmonary exercise. Requirements for ambulation training include the ability to strengthen muscles in the arms and legs, to control the pelvis and trunk, and to stabilize joints for balance.

What are orthotics?

Orthoses or braces are important pieces of equipment that enable mobility. They come in various sizes and shapes, but all share a similar purpose. This is to provide stability around a joint that is lacking due to muscle weakness or imbalance. The type of bracing needed will be different based on the level of spinal cord injury.

For example, someone with a T5 level injury can use a wheelchair and orthotic braces that include the hips, knees, ankles, and feet (HKAFO) for primary mobility. A walker or forearm crutches can be used with a "swing to" or "swing through" gait for shorter distance mobility. Braces that only include the knee, ankle, and foot (KAFO) are not recommended. An ankle foot orthosis (AFO) can be used for positioning, but not for aiding in ambulation. It does not provide enough support. A back brace or thoracolumbosacral orthosis (TLSO) is recommended for trunk support and postural alignment.[1]

If the level is L5, which means there is control of the trunk and some control of the legs, the bracing options may be different. AFOs are used as the primary assist for mobility. A HKAFO is generally not needed, but on occasion may be required, if the muscles that extend the hips or separate the legs are weak. A KAFO may be necessary to control the position of the knee depending on the underlying strength and coordination. Excessive spinal curvature may occur so this should be checked periodically by the medical team. Walkers or crutches may not be needed. A TLSO is not required for ambulation.[1]

Reasons for using braces are to help with standing, exercising, and walking. These activities are important to keep bones and muscles in the leg strong, as well as to relieve pressure on the sitting surfaces of the skin and stretch out tight muscle. Physicians and therapists evaluate a person's abilities and will help to determine the optimal bracing system based on level of spinal cord injury, strength, endurance, and life style.

An excellent web site for understanding the different kinds of braces and their uses is http://spinaltimes.org. It contains a lot of useful information.

Wheelchair Basics

The three basic things to consider when ordering a wheelchair are safety,

comfort, and mobility needs. A "wheeled" mobility system needs to accommodate orthopedic deformities, prevent skin ulcers, promote independence, and provide enough trunk stability to allow the arms and hands to function.

Components of a wheelchair include the frame, seat back and depth, brakes, arm and leg rests, type of cushion, lap belts, wheels, and inserts to provide stability. Each of these parts can be custom made and adjusted for each person.

Frames can be fixed or collapsible with the fixed ones being more sturdy, but not as convenient for transportation. The axle position can affect the stability of the wheelchair. Moving the axle forward and closer to the small wheels in front (casters) increases maneuverability, but decreases the stability of the wheelchair. The reverse is true, if the axle is moved backwards toward the large wheels. For some children, an expandable frame to accommodate for growth is appropriate.

A tilt in space frame maintains a 90 degree angle at the seat even when the chair is tilted backwards. This is to decrease the pressure on the sitting surface and is used for individuals who cannot shift their body weight independently. If the hips do not bend to 90 degrees, then the back has to be "opened" to a greater than 90 degree angle to accommodate this position. The best angle is one in which the pelvis is in a neutral or slightly forward tilt to achieve proper alignment of back muscles. An angle that is too much greater than 90 degrees may result in the need to use back extensor muscles to remain upright.

A guide for the right seat width is to measure across the widest point of the hips including braces and then adding two inches, one for each side. If the seat is too wide, spine curvature may occur from leaning to one side or the other. It also predisposes to unequal weight distribution from leaning to one side. A chair with a seat that is too wide is also harder to self propel, because it is harder to reach the hand rims.

The depth of the seat can be estimated by measuring from the rear of the buttock to behind the knees then subtract about two inches. This is a guideline and will vary depending on the size of the individual. If the seat is too short, then there may be excessive pressure on the sitting surfaces and the center of gravity will not be appropriately distributed causing the chair to be easier to tip over. An excessively long seat may put pressure on the back of the knees and decrease circulation to the legs.

Seat height should be measured from the bottom of the heel to the beginning of the thigh at the knee then add about two inches to compensate for clearance of the leg rests. The height of the seat will affect the ability for the wheelchair to fit under tables and desks, so this should be checked.

Cushions provide pressure relief over a stable seating base. They can be solid seats made from wood covered in vinyl, foam seats, air filled, or gel filled. Air filled ones have less balance and stability and are subject to leaks. Their main benefit is for pressure relief. The gel ones adjust to the movements of an individual, but they retain heat. Foam ones are cheap and light weight, but have a shorter life span and are harder to keep clean.

Brakes are usually of a push-pull variety to lock. Extension bars can be provided, if it is difficult to bend over to reach the lever. On sports type wheelchairs, the brakes are placed low so that they don't interfere with propulsion.

Arm rests can be fixed or removable. The type will depend on the ability to transfer in and out of the chair. Foot rests can also be either fixed or detachable. The detachable variety can either swing away or be removed by lifting them off. Another type of foot rest is an elevating foot rest which may be used, if leg swelling is a problem. Footplates come in several sizes. Heel loops can be added to better keep the foot on the plate.

There are a variety of trunk supports or "guides," lap belts, and trunk straps to choose from. Avoid "Y" straps or "H" straps connected to the lap belt. These configurations may cause choking.

The best approach to selecting a wheelchair is to form a partnership with your local seating clinic. This is definitely not a "one size" or "one age" fits all situation. Every family will have different needs. Hobbies, fitness, level of spinal cord involvement, and other health conditions will affect the choice of a chair and its components. Be aware that insurance policies have very specific rules guiding the purchase of durable medical equipment like wheelchairs.

Home Modifications

The following are some basic rules about home modifications for wheelchair accessibility. The official American standards are published by American National Standards Institute, 1430 Broadway, New York, NY 10018, (212) 642-4900. There are excellent resources now available on the internet. Although many are sites for companies that actually do the reconstruction, they usually have good information in them. Typing "wheelchair home access" into a search engine is a good way to find these sites. One good example is www.adaptiveaccess.com .

Stairs for ambulatory individuals should have a uniform height of 4 to 7 inches with a depth of 11 inches; handrails on both sides are recommended. Ramp slope must not exceed 1 foot in

length for every one inch increase in height. Width of outdoor ramps should be between 36 to 48 inches. Outside entrances should include a landing with at least a level area of 5 feet by 5 feet. Doorway width of at least 32 inches is mandated, but a width of at least 36 inches is preferred. Doorknobs should be at a height of 36 inches from the floor. A minimum length of 4 ½ feet for clear approach to door is recommended; 10 inch kick plates should be attached to both sides of doors to decrease wheelchair inflicted damage. Hall width should be at least 3 feet with adequate turning space at the beginning and end of the hallway. At least 5 feet is needed for turning or maneuvering a wheelchair. Wood or tile floors are better for wheelchairs than carpets or rugs. Maximum window sill heights should be 2 feet 9 inches. Bedrooms should accommodate a full size double bed. A clear area of 4 feet on one side is needed for the wheel chair and 3 feet on the other side for making the bed.

Some interior designers have specialized in making home access easier. Cynthia Leibrock has written some very detailed books covering this area. She is the founder of "Easy Access to Health" which is a consulting group specializing in barrier free designs.[2]

Good Things to Know

The following list of questions came from www.spinaltimes.org. I think they are very useful when dealing with vendors and issues affecting equipment.

- Who is your vendor?
- Where are they located?
- How can you reach them in an emergency?
- Do they have an emergency number?
- Who is paying for your equipment?
- Are you responsible for part of this payment?
- Who will pay for repairs?
- How do you get repairs done?
- Can you get an update of the purchased item in the future? If so, how?
- Has the vendor made clear to you what they are required to provide in the way of repairs?

References:

1. Molnar GE, Alexander MA. Pediatric Rehabilitation, 3rd edition, page 283. Hanley & Belfus, Philadelphia, PA, 1999.

2. Easy Access to Health, 1331 Green Mountain Drive, Livermore, CO 80536, (970) 219-0212.

The use of erythropoietin in the treatment of acute transverse myelitis

Principal Investigator: Sanjay C. Keswani, MBBS/MRCP

Co-investigators: Douglas A. Kerr, MD PhD; Peter A. Calabresi, MD; Craig Jones, PhD; Chitra Krishnan, MHS; Susumu Mori, PhD

Transverse Myelitis (TM) is a focal inflammatory disorder of the spinal cord, with potentially devastating consequences. Two-thirds of patients are left permanently disabled after TM, being confined to the wheel chair or bed due to limb weakness. Although inflammatory demyelination is the predominant pathological finding in TM, secondary axonal injury and neuronal loss is likely to be more relevant to the development of permanent disability, similar to what has been reported for multiple sclerosis. This study aims to investigate the efficacy of a promising neuroprotective agent, erythropoietin, in the treatment of patients with TM.

Erythropoietin has been shown in numerous animal studies to protect neurons in the central nervous system from injury by a variety of insults, including hypoxia, free radicals and glutamate excitotoxicity, through activation of NF-κB – mediated survival pathways. Of pertinence to this proposal, erythropoietin counteracts secondary injury and markedly enhances neurological recovery from experimental spinal cord trauma, and prevents motor neuron apoptosis and neurological disability in experimental spinal cord ischemic injury.

We propose a prospective, randomized, double-blinded, placebo-controlled six month follow-up pilot study in 30 subjects between the ages of 18 and 70 with newly-diagnosed TM. Patients who consent to enter the study will be randomized to be given either a subcutaneous dose of 40,000 U of PROCRIT (recombinant human erythropoietin) or placebo. They will receive this therapy within 2 weeks of neurological symptom onset. This will be followed by another dose of 40,000 U of PROCRIT or placebo two weeks later. All patients will receive a 5 day course of high dose steroids (1g IV solumedrol qd) which is presently the standard of care, followed by a steroid taper.

The primary aim of this study is to obtain preliminary data on the safety and tolerability of PROCRIT in patients with TM. Secondary outcome measures are changes in neurological function as measured by the Expanded Disability Status Scale (EDSS) and MSFC between months 1 and 6; and assessment of spinal cord axonal loss as measured by Magnetic Resonance Imaging (conventional, DTI and MTw). As recombinant human erythropoietin (rhEPO) has been used for decades to treat the anemia of patients with chronic renal disease and hematological malignancy, and is known to be safe with few side effects, it is expected that rhEPO will also be safe in our patient population. Data generated

from this pilot study will be used to determine if a larger, phase III clinical trial is warranted.

Advocating for Appropriate Educational Services for Students in the United States

Donna M. Owens

Donna M. Owens trained as a special educator and has over 30 years of experience in the field. She has worked as a teacher, administrator, and consultant in education and for mental health, mental retardation and developmental disability agencies. For 15 years she served as an advocate for parents of children with disabilities. She has written articles, training manuals and books for parents to assist them in education advocacy.

There are two laws that address the education of students with special needs. Knowledge of both laws, including their intent and their requirements, will be a help to parents of students with Transverse Myelitis in advocating for appropriate educational services for their children. Both laws provide protection for individuals with disabilities, but they differ in basic intent.

1. Section 504 of the Rehabilitation Act of 1973 was passed to prevent discrimination on the basis of disability; it is civil rights legislation. The non-discrimination requirements of the law apply to employers and organizations that receive financial assistance from any federal department or agency, including schools. Section 504 forbids the exclusion or denial of services to any individual based on disability. It assures the rights of individuals with disabilities to participate in, and have access to, program benefits and services, including educational services.

Section 504 ensures access to individuals with disabilities through the provision of modifications and accommodations. For years Section 504 was thought of as addressing mainly physical access concerns, such as ramps to and inside school buildings, accessible bathrooms, and elevators inside buildings. Access can also refer to the ability of a student to benefit from an educational program and Section 504 is used to ensure that educational modifications and accommodations are provided for disabled students to allow them to benefit from general education instruction. You can learn more about Section 504 at this website: http://www.hhs.gov/ocr/504.html

2. Individual with Disabilities Education Act (IDEA) was passed in 1975 to ensure that all individuals with disabilities have access to a free and appropriate public education. Research at the time showed that over 50% of students with disabilities were NOT being educated in public schools. The intent of the legislation was to bring those students into the public educational system by providing funding to states and establishing specific guidelines for the provision of special education services.

These two laws differ in intent, as described above. They also differ in the areas of funding, eligibility, procedural safeguards, and enforcement provisions. Section 504 has no specific educational protections for students with disabilities unless the argument can be made that a child is being denied access to an appropriate education that that child's non-disabled peers receive. IDEA, however, is very prescriptive regarding educational rights of students with disabilities by: listing types of disabilities to be served, enumerating requirements for evaluation procedures and requirements for the development of an individualized educational program, and by conferring specific rights to parents of children with disabilities to enable them to protect their children's rights. Whereas IDEA is a distinct and whole piece of legislation, Section 504 was one statement included in the Rehabilitation Act of 1973 written to acknowledge the illegality of discrimination on the basis of disability. Therefore, in Section 504 there are no definitions, prescriptions or specified protections.

Funding

No funding is provided with Section 504. Neither states nor programs receive funding for the provision of accommodations or modifications required by the legislation. IDEA does provide funding to states for disbursement to local school districts to support the provision of special education services at the local school district level. Current statistics show that the federal government pays from 10-12% of the cost of special education services provided by local school districts. The funding provided to each state is based on the numbers of students identified under IDEA.

Eligibility

Section 504 does not define "individual with a disability." It is defined in the regulations developed to implement the Rehabilitation Act. These regulations define a person with a disability as anyone who:

Has a physical or mental impairment which substantially limits one or more major life activities, has a record of such impairment, or is regarded as having such an impairment, AND whose disability substantially limits one or more "major life activities."

Major life activities include such activities as caring for one's self, performing manual tasks, walking, seeing, hearing, speaking, breathing, learning, and working.

IDEA is specific to public educational services to children and young adults. Consequently, there is an age requirement related to eligibility. All infants and toddlers (up to two years old) "at risk" of disability are eligible under IDEA, although for this population, services include services to parents and families and may be the responsibility of the state's health department rather than the state's educational agency. All children three to twenty-one years of age are eligible for special education services, if they have a disability that has an adverse affect on the child's education. If a state's law extends the age range for educational services beyond the age of twenty-one, IDEA safeguards follow the state's age limits.

Under IDEA a child with a disability can be defined as one with mental retardation; a hearing impairment or deafness; a speech or language impairment; a visual impairment, including blindness; emotional disturbance; an orthopedic impairment; autism; traumatic brain injury; an other health impairment; a specific learning disability; deaf- blindness or multiple disabilities. All IDEA students are eligible under Section 504, but not all Section 504 students meet the eligibility requirements under IDEA.

Procedures

As mentioned above, procedures governing Section 504 are drawn from the Code of Federal Regulations (CFR) which is prepared by federal employees after the passage of any legislation to guide its implementation. Procedural requirements for Section 504 are established in the CFR, including the definition of persons eligible, the definition of major life activities, identification of the responsible agency, and requirements for making a claim under the law.

According to the CFR, all school districts are required to appoint a 504 Coordinator who has the responsibility for ensuring the district's compliance with the legislation. Districts can develop their own procedures in alignment with the CFR for implementing Section 504, or districts can choose to use procedures outlined in IDEA which would meet those requirements. This means that 504 procedures can be different from school district to school district. It is important to identify your school district's 504 Coordinator and to request a copy of the district's 504 procedures.

IDEA legislation, unlike Section 504, is very prescriptive. The law itself includes a number of fundamental concepts and procedural requirements that parents need to know. First is the **zero-reject** principle, meaning **all** students have a right to a free and appropriate education **(FAPE).** A student cannot be too disabled or service provision too costly to relieve a school district from the responsibility of educating that student appropriately.

Note that a school district is not required to provide the best or ideal education for a student, but an appropriate education. The word appropriate can become a matter of discussion between parents and school district personnel and the courts have been called upon to define appropriate. According to the courts, a child's educational program must provide educational benefit, and the appropriate level of benefit must be determined on a case-by-case basis.

Another fundamental concept reflected in IDEA is the **Least Restrictive Environment (LRE).** This refers to the place where a student receives his/her educational program. At one point in time it was almost understood that students with disabilities needed separate classrooms. Most students were segregated from their nondisabled peers and educated in self-contained classrooms by disability, i.e., students with visual impairments, students with hearing impairments, students with mental retardation, and students with physical disabilities, also referred to as orthopedically handicapped or multi-handicapped. The segregation of students with disabilities was never the intent of IDEA. In fact, the legislation stated from the beginning that students should be educated with their nondisabled peers and provided with the necessary supplementary aides and services to address their unique needs.

During the past ten to fifteen years, there has been an effort to address concerns regarding perceptions and treatment of students with disabilities. Students with disabilities are now considered as students first and students with disabilities second, reversing the trend toward special schools and segregated programs. Another educational trend supporting the inclusion of students with disabilities in regular education classes has been standards-based education, with the assumption that all students can achieve. Students who are separated from the mainstream are less likely to be given access to the same curriculum as those students in regular class, and therefore are prevented from achieving at the same level as their nondisabled peers. Specially designed instruction and supplementary aides and services should be implemented according to the needs of the student with a disability to allow them access to and to benefit from the regular curriculum. The federal government has further mandated through IDEA that the achievement scores of students with disabilities be tracked and reported to ensure that districts are indeed providing IDEA students with an appropriate education aligned with the state-adopted standards.

"Individualized" is the watchword of IDEA. In general, educators approach the teaching of students in groups. Teachers are given responsibility for particular classes and subjects. IDEA,

however, requires that each student be considered individually in regard to their strengths and needs, and that an educational program be developed specifically for the individual student. This program is called the **Individual Educational Plan (IEP).** This plan outlines the school's service plan for the student for each year.

Another fundamental concept under IDEA is **parent participation**. School districts are required to seek out the participation of parents of students with disabilities in planning a child's educational program. The concept is so important in the legislation that districts are required to document their attempts to involve parents in the discussion of their child's individual educational program. Parents must be given notice and give consent if the school wants to evaluate their child for special education services under IDEA. And once a special education program is developed and agreed to by the school and the parents, parents must be notified if there is a significant change in the program.

One important aspect of the procedures mandated under IDEA is the multi-factored evaluation, referred to as the MFE. There are two purposes for the MFE: 1) to determine eligibility for services under IDEA, and 2) to determine the child's strengths and needs to guide the development of the IEP.

The MFE must:

- involve a variety of assessment tools (not just an IQ test),
- be administered by a qualified person(s) using validated tests,
- be conducted by more than one person, or a multidisciplinary team (for example, the school psychologist, the teacher, and related service personnel), and
- address all areas of suspected disability.

IDEA also allows for parents to receive a separate, independent evaluation, at no cost, if they disagree with the results of the initial MFE. Based on the results of the MFE, the IEP team determines the special education services to be provided to the student.

IEPs must be developed each year and must include:

- a statement of the student's present levels of performance,
- annual goals and objectives or benchmarks,
- a statement of the services and accommodations to be provided to the child,
- explanation of the extent to which the child will participate with nondisabled children,
- special education services to be provided (special education is defined as specially designed instruction), and
- modifications for state testing.

This IEP is developed by a team consisting of:

- the teacher(s) of the student, both regular education and special education,
- a person knowledgeable about the student's evaluation results,
- a school district representative who can speak to the resources of the district,
- related service personnel working with the student,
- the parents,
- the student, when appropriate, and
- anyone knowledgeable about the student selected by the school or the parents.

There are procedural safeguards outlined in the IDEA legislation that are directed specifically at parents. According to IDEA, parents must be provided with a copy of these safeguards, including:

- prior written notice about any changes to the IEP,
- parental consent for evaluation,
- an independent evaluation,
- access to educational records,
- opportunity to present complaints,
- mediation, and
- an impartial due process hearing, if complaints cannot be resolved.

When You Have a Concern

There are steps parents should follow when they disagree with their child's IEP as written, or when they question the appropriate implementation of the IEP. These progressive steps are as follows:

1. Discuss the concern with the child's teacher;
2. Request a Case Conference with the IEP team to review the issue;
3. Request an Administrative Review;
4. Request an Impartial Due Process Hearing.

When parents have concerns about their child's educational program, they should first talk to their child's teacher. If their concern is not addressed, parents should request a **case conference** with the entire IEP team to discuss their concern. This request should be in writing and should state the parent's concern. It should be sent to the principal of the child's school.

If as a result of the case conference the concern is not addressed and resolved, then the parents can request, in writing, an **administrative review**. This request should be sent to the school principal. The special education director and the district Superintendent may be copied on this request. A copy of the letter should be kept for your files. This meeting will include the district Superintendent, or designee, and allows the district to review the information regarding the complaint and allows the district the opportunity to address the complaint to the parent's sat-

isfaction.

At this point, if the parents and the school cannot agree, parents have two choices. They can file a formal complaint with the state department of education. This is done by stating the complaint and your contact information in a letter addressed to the State Director of Special Education. You can find the name of the director and his/her address by calling your State Department of Education or by checking their website. By law, this action requires the state to investigate the complaint and make a determination on the issue within a specified period of time (60 days is common). The second option is for the parents to file a request for an impartial due process hearing.

Impartial Due Process Hearing

To resolve conflicts as expeditiously as possible, IDEA requires states to make mediation available to parents and schools when disagreements occur. Mediation must be offered to parents who file for an Impartial Due Process Hearing, and, in fact, mediation can be offered by the school even before a request for a due process hearing is filed. Still, parents always retain the right to an impartial due process hearing to resolve their complaint. All problems are best solved close to the source, and it is advisable for parents to work their way up the system in addressing their disagreements with the school.

There is a great deal of help available to parents in obtaining information about the implementation of IDEA. This includes national, state and local parent and disability organizations, books, magazines and websites. IDEA provides funding for Parent Training and Information Centers in each state specifically designed to support the effort of informing parents about their rights. The following website provides a list with contact information for the parent training and information centers located in each state.

www.pacer.org

The mission of PACER Center is to expand opportunities and enhance the quality of life of children and young adults with disabilities and their families, based on the concept of parents helping parents.

Through its ALLIANCE and other national projects, PACER, a national center, responds to thousands of parents and professionals each year. From California to Minnesota to New York, PACER resources make a difference in the lives of 6.5 million children with disabilities nationwide.

You can also get the name and telephone number of the center in your state by asking your school district or your state department of education. These centers are great sources of information for parents of children with disabilities. You can get printed information, notices about training on IDEA, and individualized technical assistance with a phone call to your state's center.

There are a number of other national and local organizations that provide parent information and advocacy services. These include disability-specific organizations, such as United Cerebral Palsy (UCP), the ARC, and Down's Syndrome Association. Parents of children with disabilities are uncommonly generous when it comes to providing support to other parents. You don't have to talk to someone who has a child with the same disability as your child. The law is not disability specific. In addition, there are local support groups created by parents for parents of children with disabilities where parents can learn from the experiences of others.

Communication Tips

Knowing the law is a critical aspect of advocating for appropriate services for your child with a disability. Knowing good communication techniques is another. Remember that there are a number of factors that can affect your communication with school personnel. So, expect and be prepared to address these issues.

Group size and familiarity with those in the group
As a parent, or one of two parents, it is likely that school personnel will outnumber you in IEP meetings. Also, remember that IEP meetings are held at school, a place where educators are comfortable, but where you will likely feel less comfortable, especially if you have some negative feelings about what is happening (or not happening) at school.

Lack of common experience
Educators know the workings of the school and how things are done. Unless you are also an educator, you don't have the value of that experience. This lack of common experience can be a barrier to good communication in any setting or context.

Listener's preoccupations and speaker's assumptions
These factors can affect what you communicate and what you think is being communicated to you. You can believe that you've stated your case clearly, because your case is so clear to you, but your perception of what you have communicated may not be accurate, and on the other hand, your own assumptions about what you will hear, may affect your perception of what has been communicated to you.

Be aware that clear communication requires care and attention, and not just from you, as parents, but also on the part of the team members you find yourself working with. Give attention to clarifying what is being communi-

cated to you. When you are speaking, check your audience for signs that they understand your message.

Here are some tips to ensure clear communication:

- Take a friend with you to IEP meetings, someone you trust who can be another pair of ears that you can check your perceptions with later, someone who can help you clarify what your concerns are when they sense that the message is not being heard.
- Listen for the content of the message and paraphrase the message back to the speaker: "So what you're saying is…."
- Attend to the speaker's body language and factor that in to your understanding of the message being communicated.
- Check your perceptions: "It looks like you're nervous about including Libby in the 4th grade science class…?"
- Give I messages: "I'm concerned because you say Jeff will have support in completing his work in class, but I haven't heard what support that will be or how it will be provided, or who will provide it."
- Give feedback: "I was worried when I came today because I didn't think you understood how important it is that Laura stay with her class as much as possible throughout the day, but your response has shown me that you do understand." Or "I was worried when you said that individual OT would be limited, but I do understand how Adam's needs for OT can be met in the classroom setting."

There are some common mistakes parents can make in advocating for services for their child:

- Focusing on minor procedural issues, and thinking that "this is the moral equivalent of war." This is not war; this is a process of working with a system to bring about change that will have a positive impact on your child's educational program.
- Being too trusting of educators -- assuming they will always do what's best. This issue of trust is a balance. You should be involved and aware, but also vocal about what you know your child needs to succeed.
- Assuming an all or nothing approach, expecting instant change and becoming angry and vengeful when this does not happen. It does take time to create and support change.
- Not understanding that sometimes educators need to be educated by parents about their child. Education takes time and credible information delivered in a way that the listener can hear it.
- Not even trying programs or services that are suggested by educators, assuming that you know the only way a change for the better can occur.
- Trying to "micro-manage" every event during the school day. Choose the issues that are most critical and focus there.
- Not responding in a timely manner to a proposed IEP. Educators are required by law to develop IEPs for students and not providing them with feedback leaves school personnel in the dark about what your concerns and preferences are. This would be the same as an educator ignoring a letter that you write trying to explain your concerns about an issue.
- Not documenting conversations and meetings with educators regarding issues of concern. Keep a notebook and refer to it: "When we met on the 31st, you said that Scott would have an aide for physical education class, but that hasn't happened."

Remember that clear communication can make the difference between an IEP team that wants to work with you and one that doesn't know how to respond. Educators are a part of a system that is operated by rules and regulations, and they are bound by their contracts to follow those rules. Here are some things to remember about communication with systems:

- Learn the system's rules.
- Seek out the system's resources to help with learning the rules, such as websites and publications.
- Always communicate on paper; date and keep a copy of your correspondence for your own records.
- Follow appeal procedures when agreements cannot be reached.
- Remember that expressions of anger are unproductive. Use your energy to learn more and to plan your next step.

School systems, and the people in them, want to provide the best programs and services they can for their students. Assume that your child's educators have good intentions -- they do. Educators work under constraints of limited resources, fiscal and human, training and time. A part of a parent's job is to find out what the educators serving their son or daughter need, and work with them to obtain it. While you may have different perspectives -- the parent's and the professional's -- the law is designed to include both for the benefit of the student. There really is only one team; try to be a good team member.

Webliography: Information Sources on the Internet

Donna M. Owens

A wide range of assistive technology is available to support individuals with disabilities. Very simply, the purpose of assistive technology is to help people with disabilities to do what people without disabilities do. Assistive technology is anything from a simple tool, like a cane, to a sophisticated electronic device that allows a person with no manual control to move the cursor on a computer with an infrared signaling device. Assistive technology is not defined by what it is, but rather, by what it does. It is anything that assists a person in carrying out an activity that they wouldn't be able to perform, or perform as well, or for as long, without the device. We should consider the possibilities of assistive technology for our children and family members with disabilities and explore resources to pursue it and assess its value for particular activities. The following is a list of web resources on assistive technology with a brief description of content for each.

http://www.mdtap.org/tt/2002.08/art_3.html

This article is found on a page of the Maryland Technology Assistance Program site which was funded through the Assistive Technology Act of 1998. The article provides a description of student's rights to assistive technology both through IDEA and Section 504. While it is written in lay language, its concepts may not be easily understood by all readers. Still, it provides citations to specific court rulings that required the provision of specific assistive technology devices for students, including tutorial software and a laptop for a student with narcolepsy, an assistive hearing device to be used in the classroom, and a text-to-speech scanner for a learning disabled student. Professionals, parents and consumers will find this website to be useful.

http://www.mainecite.org/ptguide

This is the site for the Maine Technical Assistance Project which is funded under the Assistive Technology Act of 1998. It provides access to a comprehensive Parent Guidebook to Assistive Technology which reviews laws and legislation regarding assistive technology. The Guidebook is a thorough review of assistive technology information from advocating for it to finding appropriate assistive technology. In addition to information on law and policy, the site offers information on assistive technology funding, Universal Design, and available training opportunities on assistive technology. This website is useful to parents, professionals, and consumers.

http://www.uiowa.edu/infotech/Legal.htm

This is the website of Infotech, the Iowa Program for Assistive Technology. It provides a comprehensive and understandable review of a child's legal rights to assistive technology under IDEA, Section 504 and the ADA. It conveys the clear message that children have a right to assistive technology and accommodations as a part of a free and appropriate public education It also covers the provision of assistive technology through private insurance and the Medicaid program, including tips for making an appropriate request and what to do in the case of a rejection and the need to appeal. This website is useful to parents, consumers, and professionals.

http://www.katsnet.org/fact4.html

This website, hosted by the Kentucky Assistive Technology Project, provides a wide range of information on assistive technology. This particular fact sheet discusses assistive technology grounded in student rights specifically as assistive technology is related to the IEP. The authors make clear that a parent's request for assistive technology must be related to identified IEP goals. It also makes a point about the student's right to use assistive technology at home, whether it was purchased by the school or not. This website is useful to parents, professionals and consumers.

http://www.nls.org/atiep.htm

This site is hosted by Neighborhood Legal Services, Inc. in Erie County New York. The organization, while providing services to western New York, also houses a state and a National Assistive Technology Project. The materials produced for the project are written in a manner that is parent-friendly and accessible to the lay person. This particular page provides step-by-step directions on IEP development with a focus on including assistive technology in the IEP. There are also example statements of goals and objectives that incorporate assistive technology. The article is an excellent guide to IEP development for parents.

http://ataccess.org/resources/fpic/default.html

This website is hosted by The Alliance for Technology Access (ATA). It is a network of community-based resource centers, developers, vendors and associates dedicated to providing information and support services to children and adults with disabilities, and to increasing the use of standard, assistive, and information technologies. The site contains resources from a variety of AT-focused web pages and hosts a page devoted to addressing commonly asked questions. The site includes a general description of Assistive Technology, as well as frequently used acronyms and abbreviations. Although the site does not focus specifically on legal and IEP issues, it does provide valuable information and links for families.

The Johns Hopkins PROJECT RESTORE

Restoring hope, function, and lives to MS & TM patients

Douglas A. Kerr, MD PhD and Peter A. Calabresi, MD

Multiple Sclerosis (MS) and Transverse Myelitis (TM) are members of a group of neuroimmunologic disorders in which the immune system attacks and injures the nervous system. Although each of these disorders is rare, the worldwide prevalence of the group exceeds 2.5 million people. Since they typically afflict young people, the personal and economic impact of these disorders is staggering. Recent studies have suggested that the total societal costs for MS in the United States are 5.1 billion dollars annually, with the bulk of that cost due to sick leave, premature retirement and loss of income. However, despite the economic importance of these disorders, their rarity makes them difficult to study since few centers can identify and study enough patients. Even within institutions, researchers often study these disorders individually rather than striving to identify common mechanisms and identifying shared treatments. As a result of this, research in these disorders has been slow. Researchers cannot coordinate efforts and cannot rapidly develop new ideas. Perhaps most importantly, there has been little translation of new ideas into clinical therapies.

The implications are that money and effort are wasted, brilliant research is not optimized, and progress is slower than it needs to be. Every day, patients are becoming more disabled, going into or staying in wheelchairs. The solution to this problem is the development of a new way to study these disorders. Researchers must have money to rapidly test ideas, they must collaborate in order to accelerate developments, they must develop model systems to test ideas, and they must have the ability to carry out clinical trials as a prelude to new therapeutic strategies. The Johns Hopkins Department of Neurology is launching a new initiative that will achieve these goals.

Termed the Johns Hopkins Project RESTORE, this multidisciplinary research and clinical collaboration is emerging from The Johns Hopkins TM and MS Centers, and will develop new diagnostic and therapeutic strategies in the treatment of neuroimmunologic disorders, such as multiple sclerosis (MS) and transverse myelitis (TM).

The Johns Hopkins Project Restore: Overview

The Johns Hopkins Project RESTORE is a five year effort that will result in new therapies for neuroimmunologic disorders. The Project will not be a physical space at Johns Hopkins, but rather will be a "new way of doing business." The Project will fund researchers to do collaborative research, will develop novel biomarkers in neuroimmunologic diseases, will develop novel imaging strategies, and will carry out clinical trials to test ideas. At the end of five years, we expect to have dramatically improved our understanding of neuroimmunologic disorders and to have initiated clinical trials testing novel treatments based on this understanding. Funding this sort of project is not within the scope of the National Institutes of Health (NIH) or the pharmaceutical industry at present. The NIH funds established ideas that represent incremental advances from previous work. Funding from the NIH often takes two years to obtain and does not support infrastructure. Further, project funding is finite and a project must cease if the funding lapses. The result of such funding is that researchers spend a great deal of time trying to secure additional funds to support their research. We hope that Project RESTORE will be funded principally by philanthropic support for the first five years for researchers to develop novel, groundbreaking approaches. This funding will allow the rapid testing of ideas and will support the infrastructure to rapidly develop these ideas into clinical therapies. Endowment support, in particular, will protect researchers' time to allow sustained focus on research.

We expect that the Johns Hopkins Project RESTORE will continue efforts at the end of five years, but will have made such substantial advances that a greater share of the funding will be from the National Institutes of Health and pharmaceutical companies. Furthermore, by that point, we believe that Project RESTORE will have marketable and patentable therapies in development, and could share in the royalties from these products.

Project Restore has three principal goals:

RECOVER
- Function
- From acute attacks
- From illness

STOP
- Progression of disease
- Progression of disability

REGENERATE
- Nerve cells
- Myelin

By developing new treatments for TM and MS, we hope to Restore Hope, Restore Function, and Restore Lives to patients and families with these disorders.

Project Restore: Mission And Vision

Although advances have been made in the treatment of MS and TM, current therapies are only partially effective and many patients continue to develop disability despite the therapies. Our mission is as follows:

- To provide advanced care in the diagnosis and treatment of patients with MS and TM
- To develop novel diagnostic strategies to more accurately and sensitively diagnose MS and TM
- To develop new, more effective immunologic therapies that block neurologic injury
- To develop restorative therapies, including the use of stem cells, to restore function to patients with disability from MS and TM
- To be a world leader in education and dissemination of new information about MS and TM
- To facilitate the rapid development and testing of therapeutic strategies
- To provide an optimal environment for training scientists and clinicians

We are poised to make dramatic advances in all of these areas over the first five years of Project RESTORE. In the first two years, we will develop novel imaging strategies that define the pathologic features of MS and TM in order to direct appropriate treatments to these patients. We will also develop novel immunologic and neuroprotective strategies using animal models and cell culture models that will then lead to clinical trials in years three to five. We will further define the ability of stem cells to "rewire" the nervous system and will prepare for neurorestorative clinical trials using stem cells in years four to five. Perhaps, most importantly, we will initiate and carry out clinical trials of novel compounds currently in development.

Conclusion

We believe that we are poised to make major changes in how we treat neuroimmunologic disorders, and have established a formal mechanism for how to achieve this goal. We have leading experts in these diseases. However, the Johns Hopkins Project RESTORE will allow progress to be made at a much faster pace. New discoveries will result in new therapies and will diminish the suffering and disability of millions of patients.

The Transverse Myelitis Association Serves on the Christopher Reeve Paralysis Foundation Paralysis Task Force

The Transverse Myelitis Association was invited to serve on The Paralysis Task Force by the Christopher Reeve Paralysis Foundation (CRPF). The Christopher and Dana Reeve Paralysis Resource Center, funded by the Centers for Disease Control and Prevention (CDC), has established a broad-based Task Force of collaborating partners and stakeholders to create a Public Health Action Plan on Paralysis.

The aims of the plan are to:

Improve the health and quality of life for persons living with paralysis;

Raise awareness among the public and national leaders about the conditions and diagnoses that result in paralysis and the secondary complications and environmental barriers associated with paralysis;

Foster greater understanding of how a public health plan can improve the physical, psychological, social and economic well-being for Americans living with paralysis, their family members and caregivers;

Promote the appropriate management of paralysis and its secondary complications across the lifespan of the individual living with paralysis, including interventions to minimize pain and reduce psychosocial distress;

Support individuals with paralysis in accessing the family, peer, and community resources;

Translate research results into effective clinical practice in community settings; and

Present relevant public health strategies on paralysis for utilization by states, non-profits and other disability organizations.

The Paralysis Task Force Workshop was held from September 26th to 28th, 2004 in Washington DC. The goal of the workshop was to engage in a collaborative process to create bold initiatives which would serve as the foundation of a Public Health Action Plan on Paralysis. The CRPF assembled a task force comprised of public health experts, disability specialists, as well as a broad-based coalition of advocacy organizations representing persons with paralysis resulting from spinal cord injury, spinal cord diseases and birth defects.

The following organizations participated in the Paralysis Task Force Workshop:

Advancing Independence
The ALS Association
American Syringomyelia Alliance Project
American Association on Health and Disability
Brain and Spinal Injury Trust Fund Commission
Capitol Area ADAPT
Centers for Disease Control and Prevention (National Center on Birth Defects and Developmental Disabilities)
Christopher Reeve Paralysis Foundation
The Cody Unser First Step Foundation
Craig Hospital
Department of Health and Human Services (Office on Disability)
Department of Veterans Affairs (Rehabilitation Research and Development)
Easter Seals
Family Support Center of NJ
Friedreich's Ataxia Research Alliance
The Institute for Rehabilitation and Research
Johns Hopkins Transverse Myelitis Center and Project Restore
National Alliance for Caregiving
National Council on Independent Living
National Institute on Disability and Rehabilitation Research
National Institutes of Health (National Center for Medical Rehabilitation Research)
National Multiple Sclerosis Society
National Organization on Disability
National Respite Coalition (Division of the ARCH National Respite Network)
National Spinal Cord Injury Association
National Stroke Association
New Freedom Initiative (Office of Domestic Policy)
New Jersey Division of Disability Services
Paralyzed Veterans of America
Rancho Los Amigos National Rehabilitation Center
Research and Training Center for Independent Living (University of Kansas)
Santa Clara Valley Medical Center (Northern CA TBI/SCI Model Systems of Care)
Shepherd Center
Social Security Administration
Spina Bifida Association of America
The Transverse Myelitis Association
United Cerebral Palsy National
United Spinal Association
University of Kansas Medical Center (Department of Health Policy and Management)
University of New Mexico School of Medicine (Center for Development and Disability Department of Pediatrics)
US Department of Education (Office of Special Education and Rehabilitation Services)
VA San Diego Healthcare System (Spinal Cord Injury Center)
Visiting Nurse Association of America
Volunteers for Medical Engineering
Washington University School of Medicine (Rehabilitation Institute of St. Louis)
YMCA of the USA

In addition to the approximately 45 participants at the workshop, the CRPF was well represented by their staff from New Jersey, Washington DC and California. Those in attendance from the CRPF included, Dana Reeve, Director and Chair of the Quality of Life Committee of the Christopher Reeve Paralysis Foundation, Kathy Lewis, President and CEO, CRPF, Michael Manganiello, Senior Vice President, CRPF and Joseph Canose, Director, The Christopher and Dana Reeve Paralysis Resource Center.

Of the 45 participants, the TM and neuroimmunologic disorder community was very well represented by Dr. Douglas Kerr from the Johns Hopkins TM Center and Project Restore, Cody and Shelley Unser from the Firststep Foundation, and Pauline and Sandy Siegel from The Transverse Myelitis Association. The Task Force members engaged in a two-day process of guided discussion and brainstorming focused on a comprehensive and thorough analysis of health and quality of life issues surrounding paralysis. This process was managed by professional facilitators who elicited a tremendous amount of information and stimulated creative ideas from an amazingly broad range of experts. The participants possessed extensive experience and represented advocacy organizations, government agencies, academic institutions, rehabilitation centers, medical centers, and research facilities.

At the conclusion of the workshop, the participants presented a list of recommendations and each accompanied with a set of specific actions to implement the identified public health policy initiatives. The CRPF is in the process of developing a white paper which will describe the results that were produced from the workshop. Each of the workshop participants will review and comment on the white paper before the final document is completed. The final work will be the Public Health Action Plan on Paralysis. This document will be signed by the participating organizations and will serve to guide the Centers for Disease Control and Prevention (CDC) and the collaborating partners and stakeholders to promote goals to improve the health and quality of life for persons living with paralysis.

October 15, 2004

It is with the deepest sadness that we heard of Christopher Reeve's passing. Christopher Reeve was a wonderful advocate for our community. He created tremendous awareness of our issues in the general public and was a voice of reason before legislators and private and public organizations. He

brought intelligence, diplomacy and dignity to his positions on some very complicated and sensitive issues.

Pauline and I had the opportunity to meet Dana Reeve during the Paralysis Task Force meeting in Washington DC. We were so impressed with her dedication and loyalty to her husband and to her family. It was very obvious from the way she talked about their relationship that the challenges they experienced after Christopher's injury intensified their family bonds and strengthened their love and devotion as a couple. Pauline and I understood this experience very well.

The world has lost a wonderful actor and director. The paralysis community has lost a wonderful advocate. Dana and the Reeve children have lost a wonderful husband and father. We are all so sorry for this tragic loss. We hope and pray that his memory should serve as a blessing for Dana and his family and for all of us.

And the work continues, as Christopher Reeve would have wanted it to continue. The first draft of the Paralysis Health Action Plan will be written and ready for review by early November. We recently received a communication from the CRPF staff. As they wrote in their message to all of us on the Paralysis Task Force, "Now, more than ever, we wish to live up to the legacy that Christopher Reeve left to the world."

Chitra Krishnan to Serve on The Transverse Myelitis Association Medical Advisory Board

The Transverse Myelitis Association is pleased to announce that Chitra Krishnan is the newest member of the TMA Medical Advisory Board. Chitra received her Bachelor of Science in Life Sciences and Biochemistry at St. Xavier's College, University of Mumbai, India in 1997. She obtained her Master's in Health Sciences (MHS) from the Johns Hopkins School of Hygiene and Public Health in 2001.

Chitra Krishnan serves as the Research Coordinator for the Johns Hopkins Transverse Myelopathy Center (JHMTC). Chitra coordinates all basic and clinical science research activities at the JHTMC. She has been trained in the diagnosis and treatment of patients with TM, epidemiology, biostatistics and design and implementation of research studies and clinical trials. While a student at the School of Public Health, she developed a comprehensive health questionnaire and evaluation strategy for following patients with TM. She then created a database for this information and has managed that database for the past three years. This has allowed her to assist with management of TM patients throughout the world as they are diagnosed. As Johns Hopkins has the only TM Center in the world, Chitra has developed a very specialized expertise in TM and recurrent TM. Besides the physicians on our medical advisory board, no one has seen more cases of TM, worked with more patients, or performed more research on TM than Chitra. Chitra is extensively published in the area of the neuroimmunologic disorders, and particularly Transverse Myelitis and Recurrent Transverse Myelitis.

Chitra's title does not accurately convey either the breadth of her work for the Center, nor her commitment and impact on the TM community. In addition to her research, Chitra has become the vital link between The Transverse Myelitis Association and the Johns Hopkins TM Center. She regularly responds to information requests from the TM community. More importantly, Chitra facilitates the critical care for patients who have been recently diagnosed and in the acute phase of a demyelinating episode. As it is not possible for all of the patients with TM to make it to the Johns Hopkins Medical Center, Chitra coordinates care between the physicians at the JHTMC with physicians from around the country and around the world who are treating adult and pediatric cases of TM in the acute phase. Her work in this area has provided many patients with an opportunity for treatment that they might not otherwise receive.

Chitra has also taken on a vital role in assisting the TMA in educating the TM community about their conditions, symptom management practices and novel treatment therapies. As TM and the other neuroimmunologic disorders are quite rare and little understood by the medical community, this education is critical in ensuring that our members become informed advocates for their medical care. Chitra is regularly involved in the development of articles for the TMA newsletters and in the planning of TMA and JHTMC symposia and workshops. Chitra is a regular presenter at the workshops and symposia. She also participates in the important moderated discussions and question and answer sessions that take place at all of the TMA symposia between our membership and our Medical Advisory Board.

Chitra has taken on a very active and critical role in The Transverse Myelitis Association. Chitra regularly assists the TMA in developing its goals and policies and in organizing and planning activities through which these goals may be achieved. Chitra is in frequent contact with the TMA Board of Directors.

Chitra has developed a very personal care and concern for the TM community. She is a member of the TMA and the TM community. Chitra has made herself available to this very special and vibrant community seven days a

week and 52 weeks a year. Her work for the TM community has evolved into an effort that goes well beyond a job or a career. She demonstrates a commitment and motivation that reflects a personal desire to make a difference.

You can pay a person to learn; you can pay a person to perform research and to write reports and apply for grants. You can pay a person to facilitate communications with patients and between physicians. There is no amount of money you can pay a person to make them care. The qualities that Chitra brings to her position have a value that is neither quantifiable nor measurable in dollars and cents. Her commitment to the TM community goes well beyond the responsibilities defined by her job.

Chitra provides a critical service to The Transverse Myelitis Association. Chitra has become an important member of the TM community, and we are proud to have her on our Medical Advisory Board.

In Their Own Words

In each issue of the newsletter, we will bring you a column that presents the experiences of our members. Their stories are presented ***In Their Own Words***. 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. You may submit your stories by sending them either by e-mail or through the postal service to Sandy Siegel.

Recurrent TM and Devic's Disease
Bonnie Brickhouse
Topeka KS

Hi. My name is Bonnie Brickhouse. I have had TM for six years. I had a slow onset of the disease. I was having lower back pain, and I went to see my doctor. He wanted a urine sample, because he thought it might be a kidney infection. I could not get the urine sample he wanted, so he laughed at me and said, "how can I culture your urine, if you don't give me any to culture." I then felt like I needed a new doctor, so I found a new one! In hindsight, I see that he was unable to diagnose my condition. I got a new doctor that was in a group of doctors with a neurologist. I didn't know I'd need one, but I was glad to find out that the neurologist was well versed on TM. She was great.

For a few years, I had been going to college and also working, so I was proud to graduate! I received my BS in Human Services in 1997 and was diagnosed with TM six months later. I used my degree working at the local Public Broadcasting Station in Topeka. I was working when I noticed low back pain would start in the middle of the day and get worse until I went home. With rest at home and a night's sleep, I was okay in the morning. Then the next day, it would start all over. I had that for a little more than a week.

In my job as volunteer coordinator, I got the newsletter ready in the studio for the volunteers to address and code for a mailing. This morning, it was extremely hectic, because I was in charge of getting the food ready for the volunteers. I noticed my right foot wouldn't move when I walked out of the big doors leading to the studio and I fell. Then, my leg started working again, so I didn't think about it anymore. Later, I got a call that my mother was sick and had been air evacuated to a hospital in Wichita. My husband picked me up so we could see Mom in the hospital. That night, we stayed in a motel, and the next day I could hardly walk. I hung onto rails along the halls and my husband for support. We told Mom we had to go and would see her soon. My husband said we better get you home and to a doctor. It was Friday. We went to the emergency room, and the doctor there saw I could hardly walk. He said, "It must be your sinuses." He actually sent me home with sinus medicine!

Besides the lower back pain and not being able to walk, I had bands of muscles which tightened painfully across my chest and waist. Then, they would loosen and tighten again. I had nerve feelings, like burning, prickling, and itching. I had an appointment with my medical doctor on Monday, so we waited over the weekend. I walked at home by hanging on to the furniture. When I went to my appointment, the doctor took one look at me and made arrangements for me to

be admitted to the hospital. The neurologist ordered an MRI, and it told the tale right off the bat. My spine had inflammation in it. She ran all the tests and diagnosed me with TM. She put me in critical care, so they could check on me while I got IV steroids. The neurologist didn't want the TM to affect my blood pressure or breathing. It was close! She took an MRI of my head, and said it showed I did not have MS.

I got better fairly fast and went to the rehabilitation floor. I started to walk with a walker and was able to go home. I had a lot of weird prickling and itching feelings in my arms. I took Baclofen for spasms in my legs. I learned later the herb Wood Betony can be taken for spasms if you need extra spasm control. Also the herb Scullcap is good for TM and MS symptoms.

Three months later, I relapsed. We think it was brought on by a tetanus shot my regular doctor seemed to think I needed. When I got home from the hospital this time, it wasn't as easy to come back. I had a lot of fatigue. It was harder to use the walker, etc. I didn't get a chance to recover from the first relapse before it happened again. I got gastritis which made my system even weaker, and I relapsed again while getting a colonoscopy. There was an infection in my colon, and it was bleeding. I landed in the hospital again. This was the third bout with TM, and it completely took my legs. The neurologist tried plasma pheresis, and it brought me down so far I almost died. I remember there was an angel in my intensive care room that was taller than the ceiling!

They put me in an ambulance, and I was sent to KU Medical Center in Kansas City, KS. I was put under the care of a specialist who was the professor of my neurologist in Topeka. After I got another round of steroids, she put me on Betaseron. It worked really well! I was really on the mend. My daughter and I worked hard for six months, so I could have a physical therapist come to my house two times a week. My daughter and grand daughter came every day, and a friend was with me to give my daughter time off.

Then comes Devics disease. I had low vision in one eye and pain, so I went to my eye doctor. He said optic neuritis was what he suspected. He sent me to an ophthalmologist, and it was optic neuritis. After lots of tests, he gave me some steroid eye drops which cleared it right up, but the inflammation had killed part of my optic nerve. There were places on my optic nerve that were gray where they should have been pink. Then, another problem came later. The steroid drops produced a steroid cataract. I had the cataract removed. That eye doesn't see as bright colors as it should, because it has those dead spots on the optic nerve. I only had one optic neuritis attack.

On my next appointment, my husband and I told the neurologist about my eye, and she said, "I was wondering if your eyes would be affected, you have Devics syndrome." I had no idea the inflammation of my optic nerve was related to TM. If I had known that, I would have seen her about it. I probably would have been warned not to use the steroid eye drops so long.

After two years of hard work, I was walking and going up stairs to the patio with my walker! I was looking forward to the summer! My bladder function came back, and I was going to be rid of the catheter! I had taken Betaseron for almost two years, and it was working so well. It quit working, and I had another relapse right before the summer. Everything was lost. I was numb, weak, and paralyzed again; even my hands. I got better and went home still on the Betaseron. My husband asked the doctor if we should change medicine. She said, no, it will be okay. I relapsed.

Then, she put me on a leukemia-type chemotherapy for almost two years, and I haven't had a relapse since. I am now taking a Copaxone shot once a day and there have been no relapses. It is so nice not to relapse! I am in a wheelchair, and I am on a ventilator at night and oxygen during the day. I have bowel and bladder dysfunction and spasms in my legs and lower torso.

I wish this last demyelination hadn't happened, so I could move my legs. But, I can do so much with my upper torso. It is great to have that much. I am a painter. I love to oil paint and show my paintings. I belong to the Art Guild. I love the computer and e-mail all my friends and family. I talk to my daughter by e-mail every day. I also have friends who are with me every day while my husband goes to work. My e-mail address is bonniebrickhouse@worldnet.att.net Please send me your e-mail address. If you have any questions, I would love to hear from you.

My Journey
Isobel (Bel) Forster
Western Australia

My journey begins in November, 1958. I was 11 years old. Prior to November, 1958, I recall only twice, on different days, (and how far apart I don't remember) shooting pain up and down my spine. I didn't even mention it to my parents. I ignored it, because at school we were practising for the faction sports. I loved running, and I wanted to beat my friend who was always a bit faster than me. I wanted the gold ribbon! On this particular day, after school had finished, my friend who lived next door, asked me to go with her to the local butcher shop for her

mother. It wasn't far, only about 3 blocks, so we walked. I remember getting this terrible pain in the middle of my chest, and then "underneath" each rib cage. Thinking it was just "stitch" or "indigestion," I kept going. In the butcher's I sat down, and remember doubling over; the pain was so bad. When I got home I told my Mum who told me to go and lie down until dinner time and she would see how I felt then. This was now about 4 PM. The next thing I remember is when I was called for dinner and I said, "I can't walk and have no feeling in my legs!" My eldest sister naturally thought that I was playing games, and, unbeknownst to me, pinched my leg. Of course, I didn't respond! I was paralysed from the waist down.

I had no other symptoms, only the odd two pains in the spine, the "stitch," and then the paralysis. My parents called our local doctor who came and advised that I should be taken to The Princess Margaret Hospital for Children as it seemed serious. That night is a bit of a blur. I do remember, however, being given a spinal tap to ascertain whether it could have been poliomyelitis, but as I had the vaccines at school, it was not. The spinal tap was very painful. After that, I remember lying in bed with screens around me, and lots of doctors and nurses looking, talking, asking questions, prodding and poking! It was frightening and horrible. After the first day, because of the paralysis, I couldn't urinate. So, I had to have a catheter. Gosh, how embarrassing that was for an 11 year old! Sometime over the next few days, I had x-rays taken, I guess to see if I had any spinal cord damage or if it was broken. Being totally paralysed from the waist down, the examinations over those first few days and week were continual. I was like a guinea pig on display, with viewing and tests, more viewing, more tests! All they knew was that I had nerve damage. So, they called it "polyneuritis."

I don't recall how many days after the tests, but in their wisdom, the doctors decided to put my legs in half plaster casts from behind the knees to the ankles! Supposedly, this was done to keep me straight and to avoid any spinal damage! This meant, of course, being sponged often to avoid any bed sores. To a young child this was all bewildering, unnecessary, and inconvenient. Who wants plaster casts? I wanted to go home! After a few weeks, the doctors decided that the casts were of no benefit, so they were removed.

The weeks ticked by, and it was heading towards Christmas. I was still paralysed with no feeling in both legs. I still had this "tube" draining into a bottle under the bed and all the embarrassment and inconvenience that comes when you can't use your bowels normally. I wanted to go home, even for the day, but the doctors refused. So, I spent Christmas in the hospital away from my parents and family, and my dog, Suzie. Oh, don't misunderstand me, I remember the staff doing their best to make it a happy and great day, but it wasn't the same! My parents were wonderful though, trying not to show how worried they were. My father never missed a day's visit. He even used to come to see me during his lunch hour, catching the train to the nearby station and walking up to the hospital just to make sure his "baby" was being looked after.

Six weeks had now passed, and then one day, when my school teacher, Miss Fletcher, came to visit, I moved one of my right toes! She had been asking me all these questions, and I said "I can't move" (demonstrating that I couldn't lift my legs). Then, low and behold, I wriggled my toe! Great excitement was instant and the nurse was called. Another demonstration was done! Wow, I had some movement; finally, tiny, but so significant. From what I recall, progress was happening, but slow at first. As the days passed, I tried desperately to wriggle a few more toes. Forcing and willing myself, more toe movement and then very slight movement came in the right leg. Movement in the left toes and leg then began to happen as the days passed. This was encouraging for not only me and my family, but the nursing staff was excited, too. Following that simple movement, I gradually was able to stand for a few moments. But I had no balance; exactly like a baby learning to balance and walk for the first time. I still had lack of temperature sensation, and only the faint feeling of touch to my legs and feet. But this meant now I would start physiotherapy, which would hopefully help towards some sort of recovery and home. Now I longed to go home, to be with my parents, sister and my dog, whom I thought may have forgotten me by now!

Physiotheraphy commenced at the hospital, and after becoming mobile enough to stand unaided and to take a few simple steps, I was allowed to go home. I still had the bladder and bowel problems, but that was nothing we couldn't handle and worry about at home. Let me out of here! Naturally, I had to attend the hospital for regular physiotheraphy. I still had numbness in my legs. I was beginning to get some more feeling back; I could tell the difference between sharp and soft pin pricks, also to touch and some sort of difference between hot and cold. Even to this day, this sensation is not normal and my feet are super sensitive. Weakness of bladder and bowel remains.

My recovery, once I got home, was good. My parents had an old pram, and I used to be taken out in the back yard by my dad. I would use the pram for support and slowly walked up and back the pathway. Strange, this pathway led to the outside toilet we had in those days! Up and back every day un-

til, eventually, I could let go and walk by myself. Progress was so good, that when school commenced in February, 1959, I was able to ride my sister's two-wheeler bike to school! This was close to three months from the onset of the illness to being able to walk unaided; almost normal, if you like, but I couldn't run at all. No gold ribbons! Still had a bit of a gait, but what the heck, I was walking around. However, school had its problems. I was in my last year of primary school (year seven here in Australia). The girl's toilets were so far away and I couldn't always make it. I remember leaving a puddle under my chair one day in class. Of course, the other children thought this was funny. Not me! I also had to leave school on certain days when the hospital bus would pick me up and take me to physiotheraphy. High school was better, because my parents sent me to a private girl's school, and the girls and teachers were more understanding. Recovery by now was good. I went to work, married and had two children. I even got my manual driver's licence.

Strange though, my parents never came forth with any suggestions, nor did any medical person, as how best to handle the bladder and bowel problems. I don't even know if in those days there were incontinence pads. For several months after I went home, my parents toted me from one doctor to another, to see if there was any cure. But, of course, there wasn't. I learnt to live with it, with all its inconvenience, frustrations and embarrassment.

Fortunately, since I made a good partial recovery, I can tell if I need to go to the toilet. Straightaway though; no waiting! Being young, too, perhaps worked to my advantage in my recovery. I could control my bladder for a minute or so, which gave me time to make it most times to the toilet. However, my bowel doesn't work in my favour that way. When the sensation comes, I have to "hightail" it. I have always carried a bucket and loo paper, etc, in the car for emergencies. I used to be able to do "my business" in the back of the car, in the middle of a crowded car park, and no one was the wiser! You learn some tricks and how to cover up when you have lived with it for so long. With my back now, that is not possible.

The biggest achievement came after I was married. We were living in a small country town and I was asked to join the tennis club! I couldn't. I couldn't run; how could I play tennis! The ladies said that's not a problem. As long as I played doubles, they were happy to run all over the place and get the extra exercise! How good was that? So, I got my mum's old wooden tennis racquet and was away! Over time, I became a good tennis player, played on the net and developed quick arm reflexes. I also played badminton until about 8 years ago, but gave both away, because I found being at the back courts too far from the loo! I took up bike riding to stay active and keep my legs going. This activity I enjoyed. But 12 months ago I had to give that away, too, in case I fell off, because my legs are getting weaker all due to my spine.

There has never been another case, as far as I know, here in Western Australia and up to November 2003. I had never come across anyone who had this "polyneuritis." Most people don't have a clue what I am even talking about when they asked me about my illness. In all the 45 years not one of my family doctors looked or queried my illness any further, or suggested I see a neurologist! Also, because I made such a reasonable recovery and knew that there was no cure, I never pursued it further myself. I finally asked to see a neurologist because I was having problems with my arms and my left leg and foot on cold days. They felt like ice. I knew this was from lack of circulation, but I thought there maybe another problem. I have always had the sensory touch syndrome, and the problem with balance. I can trip over my own feet. It's a wonder with all the falls over the years; I did not break my kneecaps or a leg. I was never able to run, and my walk has always been with a gait, because my left leg was weaker than the right and a bit shorter. I suffer with the nerve twinges or pricks; not constant, or every day, but I do have them.

My walking began to deteriorate very slowly about seven years ago. I was having more backaches, too. So, I started to use a walking stick for support and balance. I also use a pronged walking stick for extra balance. Then about five years ago, I found walking long distances was an effort, so I invested in a manual wheelchair. When I needed to go shopping or anywhere which involved long walking, my husband pushed me in the wheelchair. Two years ago, I was finding the pressure of any walking was putting lots of pressure on my lower back and spine, which is now shaped like an "S." So, now to help myself, I use the wheelchair full time. I can only stand for a few minutes and then my legs begin to feel weak and I feel pressure on my back. Sleeping is also disturbed because of my spine. Rolling over in bed is difficult and I have to manually roll myself, because my legs are like dead weight. Due to the lack of circulation, I now have a "dead spot" on my left heel. This has a burning sensation when I lie with my heel on the bed. To stop it developing into a pressure sore, I wear a lambskin half boot. It does relieve the pressure, but because the outside is like "suede," it sticks to the sheet, and I have to manually lift my leg if I am turning over. Satin sheets have been the suggestion, but at this point, we haven't tried them. Satin pyjamas help.

I have now invested in an electric scooter to give me more independence in and out of home. I am fortunate that

I can still do my own personal showering and dressing. I am able to walk up to about 12 steps with the aid of a stick. This means I can get in and out of bed and do personal grooming, etc. Simple manual chores around the home like cooking, sorting laundry, and dusting I do from the wheelchair. We have help with the vacuuming and mopping of the floors. My husband has retired and is a full time carer, doing cooking and what he can to help me in the home and shopping. X-rays of my hip and back show my spine has a bad "S." My spine is deteriorating and basically has "had the bomb." It is tilting forward and in time "may" pinch the nerves. I have a problem now with two discs in my neck, which gives me a burning sensation sometimes. This is an indication that a nerve is being pinched by the discs closing. This is all from years of walking with the unusual gait and the jarring from all of the falls. When I stand or walk, I am like the leaning Tower of Pisa! I swim every week, which is about the only physical activity I can do, and I have physio on a regular basis to prolong my little mobility.

Depending on my arms for support all these years, and using the wheelchair has now created problems. I suffer with sore shoulders and my wrists are showing signs of early carpal tunnel syndrome, which affects my sleep because of the numbness. Fortunately, I don't have this problem during the day! I visited a neurologist for this complaint, and it was during this consultation he examined me further and diagnosed Transverse Myelitis. How grateful for small mercies!

After being diagnosed with Transverse Myelitis (T5), I accepted this new name for my illness and that there now was some easy description and explanation, but no cure. I didn't pursue it any further for about three weeks, until one day I got on the internet and typed in "transverse myelitis." Bingo! There was The Transverse Myelitis Association website and in black and white the description of the illness, the symptoms, the lot! All of these relevant to me! I couldn't believe it and to find that I am now not totally alone with this illness. It was a bit emotional too after 45 years. I am now 56 years old. As yet, I do not take any painkillers, I have never suffered with pain as an ongoing side affect; only infrequent pain in my left hip for a period last year, and backache on and off over the years. The only medication I take at this point is Celebrex for inflammation of the joints. I use probanthene for my bladder and I take that on a need basis. When home I don't take them, but if away or out for the day, I take them to reduce the need to constantly go to the toilet. Ask me where any of the public toilets are in town, and I can tell you! They work well but the side effect leaves you with a dry mouth, (so I carry a bottle of water and sip) and if used regularly, then you end up with constipation! I guess we can't have it both ways!

Now it is a matter of keeping mobile for as long as possible. Taking each day at a time is all we can do. It has been great to make contact with The Transverse Myelitis Association and finally finding other people who have my illness. We all have such different stories and yet have the same illness. All TM sufferers' stories have touched me. Lots of you have problems worse than me.

On a final note, I have received some gold ribbons, but not from running! I took up pastel painting for a hobby, and have received some prizes and sales, too. My new gold ribbon is my 14 month old grandson, watching him grow, and being with my husband, two daughters and son-in-law, who help me all they can.

I thank Sandy, the President, for encouraging me to write my story. I have made contact with Errol White, who is a sufferer himself here in Australia, Steve Alderton, whose baby son has TM, also in Australia. Hopefully, I will have contact with some of the other members, too, in the future. Have courage and do what you can when you can.

Devic's Disease
Cheryl A. Gervase
Cincinnati OH

The first time a physician said, "You may have MS," I automatically said, "I can't have MS. I have lupus. You can't have both." Was I in for a surprise.

I was diagnosed with lupus in 1994, shortly after the birth of my first child. I was experiencing painful joints and began a regimen of low dose prednisone. I had always been very active and healthy. Two weeks after the birth of my second child, I experienced daily vomiting; sometimes up to ten times a day. After insisting on an endoscopy, (my family doctor suggested post-partum depression), I was diagnosed with gastroparesis or paralysis of the stomach. This was my first case of paralysis, and it lasted for four weeks.

I continued to see my rheumatologist, and after about a year, I noticed some numbness in my abdomen and left leg. I also felt intermittent burning on my chest. At times it felt like a painful sunburn. I saw a neurologist and had a MRI, but it was inconclusive.

In 1997, we moved from Texas to Cincinnati. It was a stressful time. The kids were ages one and four, and I was starting a new job. I woke up one morning feeling as though someone had hit me hard in the eye. My vision rapidly deteriorated. Within three days, I was referred to the Cincinnati

Eye Institute and diagnosed with optic neuritis. I remember a doctor saying, "You might not have lupus. You might have MS." I later discovered that a small percentage of lupus patients do develop MS. It was frightening, and I went on my first round of IV steroids. My vision improved slightly, but never did return to normal.

Halloween of 1999 I noticed that my gait was unsteady. I had to hold onto my son while we trick-or-treated just to keep my balance. I was now taking neurontin and prednisone, and seeing both a rheumatologist and neurologist. My rheumatologist decreased my prednisone dosage, and immediately the chest burning and left-sided numbness returned, accompanied by intense pain. My MRI of the brain was normal, but the spinal MRI showed a lesion in my cervical spine. By mid November, I was again on IV steroids and had two spinal taps, both of which were normal. My neck hurt and I was having spasms on the left side of my body which were also very painful.

Over the weekend, I began having intense chest pain. It felt like someone was stabbing an ice pick into my heart. When it subsided, the pain came through my back. By Monday I could no longer walk or move my arms. Another MRI revealed that the lesion was "enhancing," or that inflammation was present in my spinal cord and brainstem. There were still no lesions in my brain, and my spinal tap was normal.

I was admitted to a tertiary care facility and given IV steroids again. I was told that there was little hope for recovery. If I did survive the night, I would not regain the use of my arms or legs. My family gathered at the hospital. My children were brought in to see me. Seeing my kids gave me the determination that I would get through this. My priest visited and anointed me with oil and gave me absolution. As a devout Catholic, I took great comfort in praying the rosary and asking for G-d's grace. I was so scared.

The next morning I awoke and had some movement in my legs. I could not feel them, nor could I feel my arms. I could move my left arm slightly. They call this "altered proprioception," or not knowing where you are in space. I was given a walker and a physical therapist strapped a belt around me to catch me if I fell. I watched my feet and said, "left foot, right foot, walker," as I shuffled down the hallway.

I spoke with my neurologist by phone and she was shocked to hear that I was walking. She gave me another new term: Devic's Disease. This includes optic neuritis and plaques in the spinal cord, but not in the brain. It does not respond to the traditional MS drugs.

My doctors have called my recovery "remarkable" and "miraculous." It took about 18 months to recover my fine and gross motor function. In the beginning, I couldn't feed myself or blow my nose. I couldn't raise my arms to wash my hair, or turn a page in a book. It took 45 minutes just to get dressed. Tying my shoes was so frustrating. After being discharged from the hospital, I spent some time at a rehab facility. After that I had out-patient PT and OT for four months.

I still experience pain in my neck and left shoulder, and stiffness in my left hand. At night my left leg has "burning pins and needles." But I feel very fortunate to be able to walk and have normal bowel and bladder function. My medications include, Neurontin, Imuran, Prednisone, Nortriptylline, and Zanaflex. My biggest battle is with fatigue.

Today, I am able to work part-time as a home health nurse. I am the busy mother of two boys, ages eight and ten. I have a very supportive family who really helped me out when I needed it most. They also knew when to just sit and listen, and when to back off and let me do things for myself. I feel very fortunate to be alive.

Devic's Disease
Jaime Liles
Rockton IL

My name is Jaime Liles. I am 48 years old. When I was 44, I was diagnosed with TM. My initial symptoms included tingling in the hands and legs, and inability to walk properly. I was unable to wash my hair, dress myself and prepare my food due to pain and tingling sensations. I then went to a world-renowned medical center. At the center, they told me I was hysterical and to go home. After a year or so of fear, struggles and pain the symptoms calmed down and I was able to walk almost normally. This lasted for about a year. Then I had a recurring episode and was also diagnosed with Devics. This time my symptoms included more buzzing and needle-like feelings from my feet to my knees and in my hands. Devics is the diagnosis I was given after an eye exam that showed a lesion on the optic nerve. This was done after I mentioned having bouts of blurred vision. At that point, I was petrified. I was so frightened of every new buzz, tingle or pain. I was sure I would become totally incapacitated. I wanted to call my physician every time something changed in my body, every time my eye twitched or I had an increase in symptoms with the tingling or problems walking. I wanted an MRI every week to monitor what was happening with me. I was so scared I would not catch the lesions in time to prevent them from growing so big that I would become paralyzed. It was at this time that my neurologist prescribed intravenous steroids. I took this course of steroids over a period of ten days as an

outpatient. However, I did stay with a friend during these ten days and I recommend this, because it was very traumatic and painful.

After living with one bout of TM, I could not believe I had to live with it again and with Devics on top of it. I could not find much information on Devics or TM until I heard of the 2001 symposium in Baltimore at John Hopkins Hospital. I flew to the symposium and met many wonderful people and physicians. I was so freaked out about the TM and Devics that I was thinking of ending my life. While at the symposium, I met a physician who told me that people with TM have a high rate of suicide. These diseases are just so frightening and life changing. At that point, I received help for my depression and was better able to manage my life.

Now, my TM symptoms have decreased again and the Devics has gotten only a bit worse. I still become scared when the weather changes and my symptoms increase suddenly or if my eyes suddenly get blurry for a day. I get this rush of fear running through my body and have to tell myself to calm down, wait a couple of days, see how the symptoms go. I have to tell myself not to call the physician, not to run and make appointments. Luckily, these flare ups are temporary at this point. I have learned to keep my scheduled appointments, take my medicine as prescribed and keep my depression under control. Now, when my vision gets blurry or I have odd pains in my legs or arms, I just wait it out.

Lastly, I feel it would be disingenuous if I did not mention how important I believe my faith in Jesus Christ has been in my healing and sense of well being. Doctors and medication have served their purpose but beyond that I needed prayer and the ability to turn this over to Christ.

'Do not fear: you are of more value than many sparrows' (Luke 12:7).

'Jesus said, 'Don't be afraid any longer; only believe' (Luke 8:50).

Recurrent Transverse Myelitis
Yolande Major
Lachine, Quebec Canada

When I was first diagnosed with Transverse Myelitis and told there was a 15% chance of reoccurrence, it never occurred to me that it would be my case. But 5½ years have passed since I was offered these odds, and I have since had ten attacks. I can no longer deny that I have Recurrent Transverse Myelitis.

It first started at the end of September 1998. I had had a major backache for about a month. There was no relief from anti-inflammatories, and other symptoms began to appear. First, there was the loss of control of my left knee, and then difficulty voiding and constipation and, finally, numbness began to spread through my left leg to below my breasts.

When I called my family doctor, he told me to go to the emergency. He agreed with my choice of the Royal Victoria Hospital, as the Montreal Neurological Institute (MNI) is right across the street from it. He had assumed it was a neurological problem.

After some time at the emergency, I was transferred to the MNI in the evening. The next morning I had a MRI. The radiologist could see 'a shadow' at the T8-T9 level, but it wasn't clear what it was. In any case, I was treated with IV Solumedrol for five days. The backache went away and I gradually started walking better. I was kept in the hospital for more tests and consultation with other doctors. Some thought it could be a lymphoma or a sarcoid infection. I was checked from A to Z to see if there were any signs of cancer. Finally, I had a biopsy of the spinal cord (major operation) and only after that did I get the diagnosis of Transverse Myelitis. I had insisted on getting the biopsy as I didn't want to go home not knowing what I had. Recovering from the surgery and the burning pain and spasms in my legs took a few more months.

But after that I was walking normally and felt I had almost a complete recovery. Then in May 1999 my left leg started giving me problems again. I was hospitalized a few days later at the MNI. By that time my leg was completely paralyzed. Again I was treated with solumedrol and more tests followed. The MRI showed a shadow from C4 to T7. All other tests came out negative. Ten days after admission, spasms and burning pain started again. But other than that, I gradually regained control of my leg. I should have gone into a rehabilitation centre, but as I had sold my house, I only had three weeks left before moving. Thank goodness I had a lot of help.

I will spare you the details of all of the other reoccurrences. On a few occasions, I was tested to see if it had become multiple sclerosis, but it hasn't. Only on one occasion did I lose control of my right leg. Of course, there is some loss of feeling in both legs, but my right leg is still quite strong.

For about two years now, I have burning pain in both legs and that occurs daily. It's worse by night time. I can still walk with a walker, but my endurance level is low. Stairs are getting more and more difficult to handle. Where I live, there are 15 steps to go to the garage. Since I've had my car adapted, I don't want to deprive myself of that facility. I will be moving sometime in July to a senior apartment as I am 69 years old. This way, I hope to keep my autonomy as long as possi-

ble.

Since my diagnosis has not changed, I am no longer hospitalized at every reoccurrence. I go to the day centre at MNI where I receive the first treatment of solumedrol. For the other days, I can do it at home with the help of a nurse from the local community services.

It's been a while since I've had an MRI, so I can't really tell if my demyelination occurs each time. The last time was in November 2002 and it showed damage from T1 to T6.

Over the past 5½ years, I've tried different treatments to prevent reoccurrences. First, there was Rebif, but I didn't react well to that, so it was stopped after a couple of months. Then, cyclophorphamide, a form of chemiotherapy, once a month, to weaken my immune system. After five treatments, I had another attack. So, that was stopped, too. Since February 2003, I've been on immunoglobuline. It seemed to be working, but at the end of December, I had another attack and another one ten weeks later. So, now I will have a treatment every four weeks.

I'm lucky that I have a good neurologist who is easy to talk to and who will always return a call. And the MNI is certainly the best place in Montreal for this kind of problem. I wish I knew what triggers these reoccurrences. I know stress was involved at the beginning, as well the backache, but certainly now that is not the case. The biggest cause of stress in my life now is this disease.

I get a lot of support from family and friends, as well as from my support group for MS patients. Generally, my morale is quite good. It's lower when I'm too tired.

Will there ever be a cure for this disease and others like it? Certainly, that's what we all hope for!

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Granny - Theresa Ollivierra Guzman Reece
The Surprise Disease
Giselle M. Pierre-Nicholls
Diego Martin Trinidad West Indies

My grandmother was born the fifth of six children, on March 14th 1917, in Belmont, Trinidad. Trinidad is the southernmost Caribbean island and situated off of the coast of Venezuela. She lived her whole life of 86 years in various parts of north Trinidad, Woodbrook, Cocorite, and Diego Martin. She worked as an accounts clerk. She was married and had two children. Her husband died when she was 37. She later remarried and was 58 when her second husband died.

Around May 1986, my grandmother, then 69, started complaining of a weakness in her knees. None of us knew the seriousness of what was in store. Her doctor at the time dismissed it as a sign of getting older. At 15 I did not question this verdict. A few tests were done, but none showed anything was wrong.

In August 1986, she awoke one morning and could not get off the bed. All family members were in disbelief. How was this possible? I remember holding her hand and trying to get her up. The thought of just waking up one morning and not being able to get off the bed seemed so incredible.

By this time we had switched doctors. More tests were done. None were conclusive for any disease we knew. Eventually, her GP diagnosed Acute Transverse Myelitis. "What on earth was that?" The words "no cause ... no cure" are certainly embedded in my memory. I hope I am remembering correctly here; we were told it was a sort of relative to multiple sclerosis, i.e., a nervous system disorder. Later, we learnt that the shingles, which she contracted in early adulthood, may have been a related factor. This was new territory, not just for us, but for our doctor, as well. He had only heard of two cases of TM in Trinidad before my grandmother's case.

Overnight, seemingly with no explanation and little warning, my grandmother had lost all feeling and function from her 10^{th} vertebrae downwards. Basically, from just under her breasts, down to her toes; in effect, rendering her paralysed and incontinent.

Granny did not think she would live much longer. She wasn't told this; it was an assumption she made. She certainly did not think she would be bedridden for 18 years. She never wanted to go out, for a range or reasons, and mostly because of the embarrassment of the catheter. I think, too, she knew it was difficult, as public places are still not disabled-friendly. After 69 years of being self sufficient, she felt that she was a burden to everyone. She received physiotherapy for a number of years. She eventually stopped it after about 10 years due to the expense and the fact that she could not see any concrete results. My mother gave up her job soon after the TM was diagnosed, because nursing care was too expensive. All family members helped with her care. Granny had no feeling in her legs, but they would kick up in the air on occasion. I think it was routine spasms caused by nerves seeking a connection to the brain as described by Christopher Reeve in his autobiography.

I shall refer to my grandmother as 'T' in the rest of this article as that is what her GP fondly called her after years of being his patient.

From about five years before her death in 2003 she stopped writing and watching TV and started forgetting things. She had problems seeing due to an unrelated problem, which was irreversible. Her hearing also deteriorated. All of her friends, brothers and sisters and even her physiotherapist, who were all healthy and walking around, died long before her. In the last two years, conversation was very difficult. Her language turned into what I can only describe as "geriatric language" - the old version of baby language.

Perhaps with more knowledge of the disease at home; advanced facilities to deal with her deteriorating hearing and sight; advanced facilities for persons with disabilities; perhaps with this, her life would not have been as limiting as what she experienced.

Our family always found it interesting that this very rare disease which affects 1 to 5 per million population, affected two people that lived on the same street in a country with a population of about 1.2 million. Reading the TM magazine has been very informative and I think the provision of members' details can be very helpful as it is an opportunity to link up with someone in your area who has the same experience and so share knowledge and support. Thanks to the TMA.

On behalf of my grandmother, thank you to Aunty Elma, Ian, Alison, Tonya, Mark, Shastine, Sharaine, her doctor, who always managed to make her smile, and others who contributed to her care. Thank you most of all, Granny - Theresa Ollivierra Guzman Reece - for showing me how to remain good natured whilst coping with adversity.

Recurrent Transverse Myelitis
Marcille Pollack
Ann Arbor, MI

Well, here I am doing something that I thought that I would never do; sharing my experience with you about recurrent TM. It all started in September 1997 when I had my first episode. I was having back pain which worsened in the middle of the night and I was also bothered by constipation. When I started to lose my right leg, I decided to see an internist. She could not determine what was wrong with me and made an appointment with a neurologist for that same day. After the neurologist examined me, he said that it was urgent that I be checked into the University of Michigan hospital. An MRI was scheduled for that night. The MRI showed lesions at T7-T11.

I was given IV SoluMedrol for three days followed by a prednisone taper. I had three more MRI's on the lumbar and thoracic spine and the brain. I also had three spinal taps. Since I had recently been in New England and there was a possibility of Lyme disease, an infectious diseases doctor was called in and he put me on cepotaxime by IV for two weeks. A neuro-oncologist was also called in and he ordered CAT scans of the chest, abdomen and pelvis to rule out a tumor on my spine. A brain stem auditory evoked potential and a visual evoked potential were normal. The diagnosis was idiopathic transverse myelitis which I had never heard of before. I had walked into the hospital on my own and left two weeks later with a wheelchair, walker, bedside commode and ankle-foot orthotic (AFO). My hospital stay was followed with approximately six weeks of physical therapy. By the end of the therapy, I was able to walk with a cane. I had another MRI in December and was able to walk into the room on my own. I was also driving, swimming and was enrolled in a yoga class for seniors. I thought that I was home free; after all, I was told that TM was monophasic!

In February 2000 my husband and I drove to south Florida to visit my brother. We were there two days when I felt the symptoms reappear in my right leg. I was able to get an immediate appointment with a neurologist at the Cleveland Clinic Florida. After the exam and an MRI, I was checked into the Cleveland Clinic hospital for five days of IV Solu-Medrol and another spinal tap. After three more MRI's, which showed the lesions at the same level as the previous lesions, a tumor was again suspected as being the cause of the TM. After a week, I was transferred to Holy Cross hospital for intensive physical therapy. Most of the patients were recovering from hip and knee surgeries and the PT's had never had a patient with TM. The PT's were super and the food, especially the desserts, was great for hospital fare. I was there a month before returning to Ann Arbor where I continued physical therapy and checked in with a neuro-oncologist.

My third episode occurred barely a month after I returned from Florida. I had another MRI which showed lesions at the T3-T4 levels. I was given another three days of IV Solu-Medrol as an outpatient followed by a prednisone taper. This was followed with a thoracic myelogram, lumbar puncture and a CT myelogram. No abnormal defects were found that would suggest a neoplasm or arteriovenous malformation. The spinal cord and roots were found to be normal. I saw Dr. Kerr at Hopkins in July at which time I had a spinal angiogram to rule out the 5% possibility that a spinal fistula may have been overlooked in the myelogram. No evidence of a spinal fistula was found. So I was back to idiopathic and back to physical therapy.

I have progressed from a wheelchair to

a walker to a cane. I don't use any aids in the house. I use a cane for walking outside. I very seldom use a wheelchair. I use the bedside commode at night as my legs become numb when I sleep and I don't want to stumble going to the bathroom. I have hand controls on my car, but don't drive very often. I leave the driving to my husband as I probably couldn't manage solo trips. I do experience neuropathic pain and have tried various medications, such as baclofen and pamelor (nortriptyline). I have been on neurontin on and off, and I am currently in the on stage. I have seen Dr. Kerr twice. I had a mild setback in August 2000 and Dr. Kerr suggested to my neurologist that I go on imuran (azathioprine) which is an immunosuppressant. I was on imuran from August 2000 to February 2003 with no more episodes. There are some unexplainable small blue cells in my CSF (spinal fluid). Dr. Kerr says that he has not seen this in other TM patients' CSF.

This has been an incredible journey that I would rather not have taken. I am grateful that I live in Ann Arbor, Michigan which has a first rate medical school and teaching hospital. I am also grateful to the Gilmurs' founding the TMA, Jim Lubin, Sandy, Dr. Kerr and all the others who work so diligently for the TMA. What would we have done without them?

Devic's Disease
Margherita Wang
Redlands CA

Like many of my fellow TM patients, I had never heard of this illness before I was stricken in July 1999. I also met with confusion and frustration as my doctors tried to deal with my sudden change from healthy person to one paralyzed at T-7 level. My attending neurologist said I had some form of "myelopathy." He did put me on IV steroids and ran many tests, including MRI, spinal tap, etc. An internist on the staff finally said I had TM.

Meanwhile, my son, who is a physician in Seattle, found out on the internet that there existed a Transverse Myelitis Association and that they were going to have their first meeting in August. He attended the meetings and from what he had learned, thought that a plasma exchange might help me. However, shortly afterwards, I began to regain some movement, so my neurologist decided not to pursue the plasma exchange. I was discharged on August 16th and went home with a walker and wheelchair.

Little did I know then that more troubles were coming my way. In early October, less than two months later, I started losing my vision. It first felt like a veil was being drawn over my left eye. My husband immediately took me to the ophthalmologist who could not find any problem. Within five days, my right eye was also affected, and I woke up blind and terrified on October 13th. My son had been doing his own research, and called to ask my doctors about "neuromyelitis optica." Back to the clinic and hospital for more exams and tests. I was admitted and given IV steroids for three days. My internist felt so sorry for me that he recommended I talk to a psychiatrist. It was comforting but weird talking to someone whose face I could not see.

My son flew down and asked my neurologist if I could have a plasma exchange. On October 18th, I had my first of five daily plasma exchanges. Now my doctors and my son were busy looking up all they could about Devic's as that is what they decided I had. It seemed that the only published study of treatment for Devic's was done by Mandler, Davis, and Kornfield (Ann. Neurology 1993; 34:162-8). The patients in this study were given Azathioprine (Immuran) and Prednisone.

Miraculously, I slowly began to see; first, only shadows, outlines and dark colors, then, shapes, sizes and, finally, subtle color differentiation. The plasma exchange had left me anemic, so my doctors waited until mid-November to put me on Azathioprine (100 mgs/day) and Prednisone (10 mgs/day). Ironically, I had to change neurologists to achieve this new medicine regimen, because my former neurologist still claimed I had "myelopathy." Since then, my new neurologist has tapered the prednisone to five mgs/day and kept the Azathioprine the same as when I started. He thinks I am doing well on this dosage. He examines me every 4-6 months, at which time blood tests (liver function and hemoglobin) are also done.

Although I walk slowly and awkwardly with the help of a cane, and have daily sporadic spasms and nerve pain, I am grateful for the amount of recovery that I have had. And, I am most appreciative of the support of my family, friends and The TM Association whose website and newsletters are full of valuable information and resource material. I would like to take this opportunity to express my heartfelt thanks to Sandy Siegel et. al. for all of their hard work!

Rare Neuroimmunologic Disorders Symposium
The Johns Hopkins Project Restore and The Transverse Myelitis Association
August 19 – 22, 2004 Baltimore, Maryland

The Science Program was held on August 19-20, 2004. The concurrent Clinical Program took place on August 19-22, 2004. The 2004 Rare Neuroimmunologic Disorders Symposium drew the leaders in both the research and the clinical care of patients who have the neuroimmunologic disorders of the central nervous system. The science and clinical program presenters were truly exceptional. There were approximately 100 scientists and physicians who attended the symposium. There were approximately 150 people from the patient population who were in attendance. About three-quarters of the people from the patient community indicated that they were meeting someone else with their condition for the first time during the symposium. For all of these people, people with the conditions and their caregivers, the symposium was an overwhelming emotional experience. The scientists had the opportunity to share their research and clinical experiences. The patient population heard presentations, which summarized this research and directed the information toward a lay audience. Learning about the important strides that are being made in this research is a critical component of the great hope our community is able to feel for the future.

The clinical program was attended by physicians and medical specialists who received CME credits through Johns Hopkins, as well as many people from the patient community and their caregivers. The program represented a comprehensive and thorough discussion of the spectrum of neuroimmunologic disorders, including TM, recurrent TM, ADEM, and Neuromyelitis Optica. Special attention was also given to the differences between adult and pediatric cases and treatments of these disorders.

The presenters provided the audience with an excellent introduction regarding the anatomy of the central nervous system, and also offered an excellent description of the immune system. There were two full days devoted to presentations covering all of the symptoms, which are experienced with the neuroimmunologic disorders. Each presentation provided an explanation of the physiologic cause of the symptom, and then offered the full range of symptom management options that are available for that particular set of symptoms. The presentations also included an extensive treatment of the socioemotional, psychological, and familial issues which surround having these neuroimmunologic disorders.

In addition to the formal presentations and the question and answer segments of these sessions, there were numerous opportunities for the patient population to interact with the physicians and scientists. All of the meals were shared in one large banquet room, and the physicians, researchers, patients and caregivers were mixed at the tables. Our experience has been that these interactions are among the most important events that take place at our symposia. These interactions help to humanize and personalize the diseases for the researchers and physicians. These are highly emotional and poignant experiences for the medical professionals and can also provide tremendous motivation and purpose for their work. The patient community derives tremendous benefit from these interactions, as well. There is wonderful catharsis derived from being able to share their experiences, and they have the opportunity to seek information about their own cases from the professionals who have the greatest understanding about their rare conditions. The patient population receives great emotional support and hope from these discussions with the medical community.

On Saturday afternoon, at the conclusion of the formal clinical program sessions, there were two sets of breakout sessions held in which the patient and caregiver population was provided the opportunity to have group discussions led by a professional expert. The topics covered in these sessions included, returning to work and school, pain management, managing sexual dysfunction, treatment of depression, treatment of spasticity, and positive growth in the face of adversity. One of the physiatrists from our Medical Advisory Board also held a wheelchair clinic in which he offered personalized guidance to patients who attended. Finally, the entire Sunday morning program was devoted to a discussion and question and answer session between the patient and caregiver community and the TMA Medical Advisory Board.

The Transverse Myelitis Association currently has more than 5300 members from every state in the United States and from more than 80 countries around the world. Due to the costs involved in travel and the economic devastation surrounding having a serious illness and disability, most of our membership is not able to attend our symposia. Our Association is devoted to providing information and educational opportunities to ALL of our members around the world. All of the clinical presentations were videotaped during the symposium. The sessions will be posted on our web site as streaming video. In addition to the streaming video, all of the handouts

and the presenter's powerpoint presentations will be available on our web site as pdf files. There is also streaming video available of the welcoming speeches, the keynote speeches during the banquets and the ceremonies and dedications that took place during the symposium.

All of this information may be accessed through our web site by clicking on the link ***symposia and workshops*** from the main page; or you can go directly to:

http://www.myelitis.org/rnds2004

Numerous photographs from the symposium are also available on our web site. We are so grateful for all of the work that Jim does to make this information available to our community and to the physicians and medical professionals who have an interest in the rare neuroimmunologic disorders. We would also like to thank Winston, Michelle and Chris Powell from Tullyvision for the excellent work they performed taping the symposium and preparing the materials for posting on our web site and for distribution to our members.

Ordering Symposium DVDs

TMA members can order DVDs of the symposium from Tullyvision. The TMA is not making any profit from the sale of the DVDs; their distribution is solely for the purpose of offering this critical information to our membership.

The order form has been included with the mailing of this newsletter. The form may be mailed or faxed directly to Tullyvision. You may also fill out the form electronically at our web site (http://www.myelitis.org/rnds2004), and then email the form to Tullyvision.

The 15 DVDs that make up the complete symposium set may be ordered for $100 or the DVDs can be ordered individually for $8 each. The contents of each DVD are identified on the order form. The complete set of DVDs is packaged in an album. Regardless of whether you order the entire set or one DVD, the shipping and handling cost is $6, if you are ordering from within the continental US. If you are ordering from outside of the continental US, please email Tullyvision directly in order to receive your shipping and handling costs. Their email address is identified on the order form. Payment information is also specified on the order form. If you have any questions regarding the ordering or shipping of the DVDs, please get in touch directly with Tullyvision.

Welcome Address: Sandy Siegel

Good morning and welcome to the Rare Neuroimmunologic Disorders Symposium.

The Transverse Myelitis Association made a commitment to bring our members together as often as possible for the purpose of providing information and support. We know that having a rare disease can be a very isolating and frightening experience. There is tremendous emotional benefit from being able to share with others who intimately understand your experiences. No one can quite comprehend what you have been through as well as the other people who are in this room. These meetings also provide us with the important opportunity to offer you information about your condition, as well as the latest management strategies to treat your symptoms. We know that you will leave here on Sunday as better and more informed advocates for your medical care.

I am very grateful for the physicians and scientists who are attending the symposium this weekend. We are very excited about what you are going to be learning from each other through your presentations and your informal discussions. Your interest and your research on the neuroimmunologic disorders give us great hope for the future.

The TMA and Johns Hopkins TM Center made a concerted effort to invite physicians and medical professionals who are treating people with TM, ADEM and Devic's disease. We have also promoted the symposium to emergency physicians. For those who have come to learn, we are appreciative of your being here. We know that you will take away valuable information from both the presentations and from your interactions with the people from our very special community.

On Sunday, July 29th 1994 at 5:30 in the evening, Pauline fell over to the floor with excruciating back pain and was immediately and completely paralyzed from the waist down. Her life, my life, and our children's lives, were changed forever. Our experience with TM was like your experience. We got Pauline to an emergency room. The ER physicians, after hours and hours of tests, concluded that her symptoms were psychosomatic. It wasn't until they finally realized that she was unable to urinate that a neurologist was called in. The neurologist wasn't sure what was going on, but he understood enough to get Pauline on a high course of IV steroids. There was a week of numerous medical tests in the hospital and then the diagnosis of TM. As with all of you, we had never heard of it, and didn't know anyone who had it. Pauline was admitted to Dodd Hall, the rehabilitation facility at Ohio State University. Pauline remained at Dodd for almost two months. She came home in a wheelchair. We still had no information about TM. We were anxious and concerned, primarily because we were ignorant. And being so ignorant made me angry.

A few months after Pauline got home from the hospital, we found Deanne Gilmur. Deanne was interested in starting a support group for people with Transverse Myelitis. Her daughter had developed TM at the age of 18 months. I offered to get involved. That was ten years ago.

The Transverse Myelitis Association is celebrating its 10th anniversary. Our work is dedicated to helping people so that, to the extent possible, they do not have to go through what we went through when Jim, Katie, Pauline, Paula, Debbie and Stephen got TM. Ten years ago, there was no network, there were no support groups, there was no TMA web site, there were no newsletters, there were no symposia and workshops, there was no internet club or bulletin boards, there was no medical advisory board, there was no Johns Hopkins TM Center, and there were no specialists in TM. There was almost no research on TM.

How life has changed for a person with TM in the past ten years. I receive phone calls almost every day from a recently diagnosed person who is searching for information. I am often called by a family member of a person who is still experiencing the acute onset of TM. They are looking for information about treatment and where they can find the best medical care. While I am talking to these people, it is never lost on me just how different our lives would have been if Pauline and I would have been able to make that phone call while she was in Riverside Hospital or Dodd Hall. We had no one to talk to and there was no information available about TM. The physicians often seemed as confused as we were.

When people hang up the phone after our conversations, they aren't feeling great. But I know that the TMA has done a great deal to stem the ignorance and these people know that they are not on their journeys alone.

When I say that the TMA is celebrating our 10th anniversary, I must qualify my use of the word celebrate. My work for the Association is a day-to-day experience filled with sadness, frustration, helplessness and grief. There are so many tragedies and there is so much suffering. I mourn daily for those who have lost their lives or for those who have taken their lives. I grieve for those who have lost the ability to breathe on their own, or have developed pain so horrible that physicians have not found a way to treat it, or who fight to have dignity in the face of losing control of their bladder and bowels. I suffer with those who have lost their ability to move or to walk and have lost some or all of their independence, or with those who have lost the emotional and sexual pleasures they shared with their partner, or who are depressed and can't seem to find a way out of their darkness and are resistant to seeking the help they so desperately need. I grieve for those who have lost their ability to work, or have lost their ability to play, or have lost their homes, or who suffer through their illness without health care insurance or without medication or adequate medical care. And I suffer most with the children who get TM and with their families. There has been so much pain and suffering surrounding what we do. The heartache is sometimes just overwhelming.

In some ways, I feel like this all started yesterday. And in some ways, it feels like we've been at this for 100 years.

But there are also huge triumphs and joys on this journey. And that's just how it is with life. Lots of nachas and lots of tsoris. Joy and sorrow. The grand cosmic balance.

The TMA has accomplished so much in the past ten years. In January 1997 the TMA had 187 members. Today, we have more than 5000 members from every state in the United States and from more than 80 countries around the world. We have support groups all over the globe. The officers of the TMA and the support group leaders are all volunteers. It is just an amazing and dedicated group of people. I want to thank all of the officers of the TMA for the incredible work they perform every day for our community.

Debbie Capen, Paula Lazzeri, and Stephen Miller. Debbie, Paula and Stephen do the work of the Association when they are not working at their jobs, or taking care of their families or attending to their homes and their lives. They also do the work of the Association while they are dealing with all of the complicated and difficult health issues they have from TM.

The only full time worker in the TMA is Jim Lubin. I would like to offer special thanks to Jim. At the age of 21, Jim suffered an inflammatory attack at the C1 C2 level of the spinal cord. Jim is a full quadriplegic and ventilator dependent. Except for some return of sensation, Jim has experienced no recovery. Jim is the heart and soul of the TMA. He works every single day of the year maintaining and expanding the web site, seeking information and finding new ways to share information; finding new and creative ways for us to communicate with each other, and searching for new ways to raise awareness and to raise funds. We have 5000 members because of Jim. Jim is in my mind and heart every day of the year. Even though he will not be in these rooms with us over the next four days, his presence will be felt by me and by everyone who has been so profoundly touched by his goodness, his kindness and his caring. We love you, Jim!

I want to thank the Medical Advisory Board for being here today and for all of the work you do for our community

throughout the year: Chuck Levy, Joanne Lynn, Doug Kerr, Greg Barnes, Leslie Morrison, Jim Bowen, Adam Kaplin, Frank Pidcock, and Chitra Krishnan.

We are so grateful for the counsel and guidance you provide to the Association, but we are most appreciative of the excellent medical care you provide to the people in our community. We have the most wonderful group of physicians in our Association. They are excellent doctors, but they are first and foremost kind and caring human beings.

I also want to offer special thanks to Doug Kerr and Chitra Krishnan. Doug very directly and most profoundly has changed the lives of everyone who has TM or will have TM in the future when he started the Johns Hopkins TM Center.

Thank you, Cody and Shelley Unser. No one has done more to make the world aware of TM than Cody, and she is tirelessly dedicated to improving the quality of life for everyone in our community. Having gotten TM as a teen, it would have been perfectly understandable for Cody and her family to completely focus in on their own needs. Instead, Cody found a way to look beyond her own issues and opened her heart and her spirit to our entire community.

I would like to thank all of the support group leaders from around the country and from around the world. There are so many members who regularly devote their time and energy to holding fundraisers for the TMA – to raise money and to raise awareness about TM. I thank all of you. The parent group is totally amazing! And I want to offer them special thanks. How they find the time and the energy to get involved with fundraising in addition to the challenges they face in meeting the needs of their children is just remarkable to me.

Cathy and Dan Dorocak, Jack and Joanne Callahan, Jeanne and Tom Hamilton, Darian and Amy Vietzke, Pam and Morgan Hoge

When an infant or young child gets TM, these are the parents who I depend on to provide support to the families who are facing the most difficult experience they will ever encounter in their lives. They do this work with such sensitivity, empathy, and care. Thank you so much for being there for me and for being there for other parents and children who so desperately need you.

I want to thank my beautiful wife, Pauline. Pauline is involved in all of the work that I do. When the phone rings, more often than not, Pauline is the person who answers the phone. She gives so much of herself. And she gives all of you me. Thank you, Pauline. I love you!

And I want to remember Joyce Booth. Joyce died this year. Joyce was a dedicated advocate for the TM community and was the support group leader in Mississippi. When we lost Joyce, we also lost our Mississippi TM support group. Joyce has again reminded me just how fragile life is and just how much the TMA depends on the generosity of people to do our work.

So in remembering Joyce and in reflecting on the remarkable accomplishments of the TMA, I do say, "L'chaim – to life." Life is good. There is much to celebrate. What a journey!

We have so much work to do. The journey sometimes seems so daunting. But I never forget - ever - who I am on this journey with. I am so blessed to have all of you in my life. We are all so blessed to have each other.

Keynote Address: Paula Lazzeri

Hello everyone. It is wonderful to be here with all of you. Over the past weekend, it has been great to spend time with my old friends, and it has been wonderful to make new friends with so many of you. We all share a very special bond.

No matter where we come from or our backgrounds, we all share some life changing experiences. If you have Transverse Myelitis or ADEM or Devic's disease or Optic Neuritis, I understand what you have been through. I can relate to the experiences you've had. We can share these experiences in words, and by sharing we can help each other feel better, or we can just sit with each other in silence, and we will both know and understand. We do have a very special bond.

I have had Transverse Myelitis for 25 years. I was a happy, healthy 12 year old girl. I was very athletic and had been involved in gymnastics since I was three years old. I had hopes of one day making the US Olympic team. It was around the time my parents were considering private training that my life was about to change.

One morning in 1979 I woke up with extreme back pain. It was so bad that my parents took me to the emergency room. The doctors said that it was probably just a pinched nerve, and they sent me home later that morning. They said that I would be fine. By the time I got home, my knees began to buckle, my balance was off, and the pain in my back was horrible. No one in my family could believe that anything was seriously wrong with me as we had just returned from the emergency room. I laid down trying to rest and stay calm and I felt a wave of paralysis go from my feet to my neck. In less than one day I went from running around and doing cartwheels to becoming a C 6-7 paraplegic bound to a wheelchair. The same doctors that had

seen me early that morning were there to see me paralyzed that afternoon, and I believe we were all shocked, scared, and confused. It took a month before my parents, family, and I heard the words Transverse Myelitis.

The time before I received the TM diagnosis was agonizing for me and for my family. I was only able to see my family through a glass window, because the doctors were afraid that I had something that was contagious. I endured many tests. I repeatedly asked the doctors and nurses, "Am I going to die?" No one would respond to my question. It was a frightening time for my family. It was a terrifying time for me.

We finally heard the diagnosis, Transverse Myelitis. The diagnosis did not clarify for us anything about what had happened to me. We were just surrounded by uncertainty and we had no idea at all what the future held in store for me. I felt like I was totally alone on a deserted island.

Through the support and encouragement of my family, I decided early on that I was not going to let Transverse Myelitis and my symptoms defeat me. I refused to accept the admonition from my doctors that I would always need help, that I would never walk, or that I would not be able to do anything for myself or that I would lead the life of a normal woman. The struggles were varied and frequent, occasionally painful, always frustrating, and at times filled with tears.

My family was always full of determination not to let these ideas get the best of me. Their love and their nurturing carried me through many difficult times. They believed in me, and they helped me to be able to believe in myself. My brothers and sisters did not treat me like an invalid. I was given no special treatment. My brothers would play football in the living room when my parents weren't home. As I couldn't play with them, I would tell them that I was going to tell our Mom and Dad on them as soon as they got home. My brothers would push me in my chair into the corner of the room, nose first, and would wedge tennis shoes behind my wheels so that I couldn't move. Okay, not very nice but I survived. Plus I knew my Mom would serve her brand of justice when she got home.

I once heard a football coach say, "You are never a loser until you quit trying." I understand that concept and I agree with it wholeheartedly.

I have pushed on through four major spinal surgeries, three of which failed. The fourth was finally a success. I had a full spinal fusion to handle the scoliosis that I had developed after getting TM. I have endured the months and months of separation from my family while I lived at Children's Orthopedic Hospital in Seattle with only short visits to home on the weekends. I tried to keep my determination in tact while I fought for my rights and the rights of other young people in the hospital. We wanted to be treated with dignity and with respect. The staff at the hospital was treating all of us like infants, and it was demoralizing and disheartening. The results of my efforts were that the hospital instituted some major changes regarding its policies impacting young male and female patients and their needs.

I persevered through three years of high school. While pushing my way across the campus, I suffered a collapsed lung. Those were three years of very sore palms and battered feelings. Those were three years of very few friendships and very limited access to school functions. High school can be a difficult experience when you look and act just like everyone else. It can be excruciatingly difficult when you look and act differently than everyone else.

I had it in my head and heart that I was going to walk across the stage to receive my high school diploma at graduation. I worked really hard with my physical therapist. There was blood, sweat and a river of tears, but the final result was that with the aid of a walker, I took 15 steps across the stage and accepted my diploma from the hand of the principal. It was truly one of the most satisfying moments of my life. I went into high school quiet and trying to blend in but left it with a bang! I still see fellow students and their families every now and then who tell me what an impact that made on them that day.

I went on to college and graduated with a degree in business. I have worked as a tax accountant for the past 15 years. Working has also had its ups and downs but by far the experience has been worth it. Not long ago I gained a nick-name from my co-workers. I recently got a new wheelchair and for the first time I had the seat set at an angle. I was not used to the way it was balanced. While delivering files one day, I ran into a cubicle wall and flipped over backwards. Thankfully, a coworker that sat close by came to my rescue. I felt totally helpless and embarrassed, and I shed more than a few tears. I was mortified. A few minutes after the fall, I talked to Sandy about it. He helped me to dust off my ego and we had a good laugh about it. I moved on and tried not to look back. My coworkers now call me "Flipper." I made the mistake of sharing the story with my husband and son and the new nickname has now traveled home and to my church.

Twenty years ago I met the man of my dreams. We have been married for over 17 years. He is an incredible husband who is sensitive, romantic, and devoted to me, always making me feel beautiful and successful. Our decision

to have a family was another milestone in my life. With the help of a high risk OB we forged ahead blindly for there was no information that existed medically on pregnancy and TM. I call my 11 year old son, Jesse, my little miracle baby because he is just that! Motherhood has been so rewarding, not always easy, but I'd never do it any other way.

I have served as the treasurer for the TMA the past ten years, I drive my own van, I take care of my home, and I love to play and watch sports. I sit snow ski but will never be a Picabo Street. I play tennis but will never be a Serena or Venus Williams. I ride a hand-powered bike but will never be a Lance Armstrong.

I believe we can accomplish anything we set our minds and our hearts to do. If you are willing to work hard at it, and to believe in yourself, and maintain a positive attitude, and remain focused on your goals, you can reach for and achieve your dreams. I have succeeded because I won't give up. Picabo Street or the Williams Sisters or Lance Armstrong cannot outdo my determination and perseverance; they cannot outdo my spirit and my heart.

I have come to believe that if this had not happened to me, I would likely have gone on as a healthy, happy human being but perhaps never fully realizing the blessings that were truly mine. Today I am thankful for the use of my hands, my eyes, my ears, my mouth, my mind, and my heart. My parents tell me I had an extrasensory smile before I got sick and that I never lost my smile through it all. People react to my smile. I intend to keep that smile throughout eternity, so get used to it.

That smile reflects my attitude about life. I have had to face many challenges in my life. I have had to endure numerous physical and emotional hardships. But my positive attitude and my sense of hope for today and the future have never been diminished. My life is good, because I will have it no other way. My life is beautiful, because I choose to see life this way. We cannot control all of what happens with our bodies, and we cannot control what goes on in the world around us. But we do control how we think about and feel about ourselves and our families and the world we live in. And it is all good; life is very very good.

My extended family is always supportive and ever close to me, as you see in my sister Donna who is here in Baltimore with me. Love you, Donna. My TMA family has fast become a huge part of my life. There are several people here Sandy, Debbie, Stephen, Doug, Adam, Chitra, Heather, and Drema to name a few who mean the world to me and get me through life with TM. I cannot forget to mention one person who is not here, Jim Lubin, who is an amazing person. His philosophy is "When life isn't the way you like it, like it the way it is, one day at a time." He is so inspiring to me and to everyone who knows him. I am so privileged to be able to see Jim and Helena and to spend time with them.

I am constantly blessed by a heavenly father who took this broken clay vessel and reshaped it into the woman I am today and continues to use me to reach others who need to hear what we can all become with His love and with our own minds and hearts and spirits.

I'd like to end with a favorite verse from the bible. "Consider it pure joy, whenever you face trials of many kinds, because you know that the testing of your faith develops perseverance. Perseverance must finish its work so that you may be mature and complete, not lacking anything."

Children's Art Dedication

The Transverse Myelitis Association made a presentation of artwork to the Johns Hopkins TM Center during the symposium in Baltimore. The following speech was made by Sandy Siegel at the dedication.

The Transverse Myelitis Association advocates for adults and children around the world who have Transverse Myelitis. About twenty percent of the 34,000 people in the United State who have TM are children. Children as young as six months of age get TM. The symptoms of TM are caused by an inflammatory attack and demyelination in the spinal cord; there are multiple causes. Most of the infants who get TM are impacted high on the spinal cord, and their symptoms tend to be severe. The children with TM suffer from paralysis, bowel and bladder dysfunction, and other neurological symptoms. Some of the children with TM are ventilator dependent.

The TMA held a Children's and Family Workshop in Columbus, Ohio in July 2002. For almost all of the children with TM and their brothers and sisters, the workshop was their first opportunity to meet other children who have their condition. Children with TM face special challenges in their lives, as do their families. Having a rare disease can be a frightening and isolating experience. The TMA works diligently to help these children and their families by providing networking, information, emotional support and encouragement. TM is a devastating illness. Many of these children become paralyzed before they have even had a chance in life to take a first step.

While the children suffer significant physical disabilities, they inspire all of us with their intellects, their emotional strength and their positive spirits. These children are incredibly bright and are filled with hope for their fu-

tures. During the course of the workshop, the children created a beautiful piece of art with the guidance from an art therapist, Lori Stralow Harris. This beautiful work of art reflects the creativity, the hopes and the courage of these children. Their minds and hearts serve as a constant reminder to all of us of the strength and resilience of the human spirit. These are truly remarkable and special children.

The TMA is proud to have this wonderful work of art displayed at the Johns Hopkins Medical Center. The Johns Hopkins Transverse Myelitis Center is the only center of excellence in the world dedicated to medical care and research for people who have TM. More people with TM are seen at the JHTMC than at any other medical institution in the world. It is only appropriate that the children's creation should be available for the majority of the TM community to enjoy and appreciate. It is our great hope that the positive and hopeful spirits possessed by these children and reflected in their art serve as an inspiration for the entire TM community and for everyone at the Johns Hopkins Medical Center who sees it.

Chitra Krishnan Awarded The Transverse Myelitis Association 2004 Distinguished Service Award

The award was made at the symposium in Baltimore. The following speech was given by Sandy Siegel at the award ceremony.

A woman in Europe who was diagnosed with transverse myelitis in the mid 1990s is experiencing a serious recurrence of symptoms. She goes to the hospital, but the physicians are uncertain of the cause her exacerbation. They send me their medical records. I send the records to Chitra. Chitra takes over. She has all of her medical records reviewed at the Johns Hopkins TM Center and then initiates a dialogue between Dr. Kerr and the physicians in Europe who are treating this woman. The woman receives a diagnosis of MS. Chitra then follows back with me so that I have information to communicate to the patient and her family.

There is a little boy in the United States; 18 months old and just two days from the onset of TM. He is totally paralyzed from the neck down and on a ventilator. The parents get in touch with me and I call Chitra and give them the parent's cell phone number. Chitra calls the parents who are at the hospital and begins the coordination of care for this little boy between his pediatric neurologists the Johns Hopkins TM Center.

I am getting the newsletter ready for publication and I need articles from three physicians at Johns Hopkins. I call Chitra and ask her to help me extract the articles. Chitra works with these people all day. She sees them working from early in the morning until late at night and seven days a week. They are constantly juggling the demands of caring for patients, teaching, doing rounds and research; and every once in a while, these people try to see their families. I ask Chitra to begin to hound these people for my articles; the articles that they have no time to even think about, let alone write. But she does. In her very diplomatic and charming way, she cajoles and hounds until I have my articles for the newsletter.

I want to publish an article that was previously published in a medical journal; Chitra, contacts the publishers for me so that I can get permission to reprint the articles. I am working on a research project and have hundreds of concepts that need to be categorized for coding. I send Chitra the files to review and to help me develop into meaningful categories. Chitra spends hours and hours doing this review and sorting for me.

Chitra is not employed by me or the TMA, but Chitra is on call for our community seven days a week. I call her at the office all day long. I call her on Saturday nights and I call her on Sunday mornings. She never answers the phone, "Oh, crap, it's you."

Chitra never says no. Chitra does run away to Bombay on occasion, but she never says no.

Chitra serves the TMA and the TM community in so many ways. Her impact and her service to our community are truly extraordinary. I know that when Chitra came to the Johns Hopkins TM Center, she was thinking she was taking employment at a job. The idea that Chitra is working at a job has long since disappeared. Chitra's work is about her ideals, her values and her heart. Chitra isn't about making a living. Chitra has joined the crusade. I can see it in her eyes when we are talking about a sick child, or an adult who is suffering with severe depression or with horrible pain. I hear it in her voice when we are sharing our disappointments about not being awarded a grant for a study or for one of our workshops.

Her enthusiasm for her work is about the people. She loves the people in the TM community – the people who have TM, their families, and the medical professionals who care for this community. It would be difficult to find a person as bright and creative as Chitra. It would be difficult to find another person who is as highly motivated as Chitra. It would be a challenge to find another person who is as organized and as good a communicator as Chitra. But it would be absolutely impossible to find another person who brings the heart and spirit that Chitra brings to her work.

Chitra does work for the Johns Hopkins TM Center. She is a wonderful researcher. She is an exceptional writer and speaker. She is incredibly well organized. She has an amazing ability to communicate with people with sensitivity and kindness.

Chitra does it all. She works tirelessly for the Johns Hopkins TM Center and the TMA.

There is no way to measure her contributions in hours worked, or grant applications prepared, or studies conducted, or articles written, or patients cared for, or symposia coordinated, because while this is all of what Chitra does, what makes her so special is not what she does, but how she does it. Chitra does this work, because she loves what she's doing, and more importantly, she loves who she is doing this work for.

Chitra has worked for over a year to prepare for this symposium. She has been involved in the coordination of every detail of the work from developing the program agendas, to inviting the presenters, to applying for funding, to negotiating the hotel contract, to arranging travel plans, to ordering meals. It is an absolutely enormous job that requires tremendous time and effort to do well. And Chitra only does things well.

The Transverse Myelitis Association is proud to have Chitra on our Medical Advisory Board. We appreciate Chitra's very generous contributions of her creativity, skill, experience and intellect to the very challenging medical, social and emotional issues that surround our community. And most of all we are grateful for Chitra's devotion and care and love for our community.

We love you, Chitra, and we are pleased to award you with the first and the 2004 TMA Distinguished Service Award.

The Transverse Myelitis Association Named Recipient of Christopher Reeve Paralysis Foundation Quality of Life Grant

The Christopher Reeve Paralysis Foundation (CRPF) awarded to The Transverse Myelitis Association a $10,000 Quality of Life Grant. CRPF awarded a total of $779,321 in Quality of Life Grants to 126 organizations nationwide as part of its 2004 first funding cycle of the Quality of Life Program.

The Quality of Life grants program was conceived in 1999 by Dana Reeve. Reeve, Director of the Christopher Reeve Paralysis Foundation and founder of the Christopher and Dana Reeve Paralysis Resource Center, started the program with the goal of helping people living with disabilities in the 'here and now.' Grants are awarded to programs or projects that improve the well-being of people living with paralysis, particularly spinal cord injuries. Funding is awarded twice annually in thirteen categories, including: accessibility, advocacy, arts, assistive technology, children, counseling, education, employment, health promotion, independent living, practical services, sports and recreation, and therapeutic riding.

"For the first time in the history of this program, we have awarded over $775,000 in one cycle which will directly improve the quality of life for not only those living with disabilities, but their loved ones and caregivers as well," said Dana Reeve. "When we first launched this program, I was thrilled to be able to award 20 nonprofit organizations. I never imagined that within five years we would be able to fund over 125 groups."

Health Promotion grants, a special category of Quality of Life grants, are funded through a cooperative agreement with the Centers for Disease Control and Prevention. Funding is awarded to non-profit organizations that address the needs of people living with paralysis caused by spinal cord injuries and other injuries, diseases and birth conditions that result in paralysis. Health promotion grants strive to remove societal and environmental barriers that limit the abilities of individuals living with paralysis to participate in life activities. Participation in these activities improves physical and emotional health and prevents secondary conditions for persons living with paralysis. Health promotion grants totaled $386,421 and were awarded to 50 organizations.

The Transverse Myelitis Association and the Johns Hopkins Transverse Myelitis Center co-sponsored the Rare Neuroimmunologic Disorders Symposium. The Health Promotion Quality of Life grant was used to support this symposium. The primary goal of the symposium was to bring together patients with rare neuroimmunologic disorders, professionals who provide medical treatment and care to this population, and basic and clinical scientists who specialize in research on these disorders. The symposium provided a unique approach and an exceptional opportunity to foster communication and the sharing of information between the significant members of the neuroimmunologic community.

According to Dr. Siegel, President of The Transverse Myelitis Association, "There are considerable benefits to be derived from bringing together these groups who possess a variety of perspectives and experiences but who share very fundamental and common goals: a better understanding of these rare disorders, the improved quality of life for this community of people, im-

proved treatments for these disorders, and ultimately a cure for these neuroimmunologic disorders. This symposium represents a major step in achieving all of these goals."

The Christopher Reeve Paralysis Foundation (CRPF) is committed to funding research that develops treatments and cures for paralysis caused by spinal cord injury and other central nervous system disorders. The Foundation also vigorously works to improve the quality of life for people living with disabilities through its grants program, paralysis resource center, and advocacy efforts. For more information about the CRPF Quality of Life Program or to read about the 2004 grant recipients please visit www.ChristopherReeve.org.

Share the Caring As We Celebrate National Family Caregivers Month

National Family Caregivers Month is celebrated in November to recognize more than 50 million family caregivers across the country who provide services valued at $257 billion annually. Family caregivers form an invisible healthcare workforce and literally underpin our healthcare system by providing approximately 80% of all homecare services. Family caregivers sacrifice time and wages to care for loved ones, and over their lifetime can lose an average of $660,000 in combined Social Security benefits, pension benefits, and wages. National Family Caregivers Month, sponsored by the National Family Caregivers Association, is designed to focus on the many challenges facing family caregivers, draw support for stronger public policy to address family caregiving issues, and raise awareness about community programs that support family caregivers. The Transverse Myelitis Association encourages our members to celebrate National Family Caregivers Month 2004.

The ongoing theme for NFC Month is 'share the caring,' but each year NFCA focuses on different issues relevant to the family caregiving community. This year NFC Month will provide information and education about three timely and important issues including:

1. How public policy affects a family caregiver's day-to-day life
2. Why a family caregiver's good health is essential to their loved one's wellbeing
3. Things family caregivers can do to help keep their loved one safe

National Family Caregivers Association (NFCA), whose mission is to improve the overall quality of life of family caregivers, is a grass roots organization that educates, supports, empowers and speaks up for the millions of Americans caring for chronically ill, aged or disabled loved ones. NFCA is the only organization dedicated to family caregiving that reaches across the boundaries of different diagnoses, different relationships, and different life stages to address the common needs and concerns of all family caregivers. NFCA espouses a philosophy of self-advocacy and self-care that is predicated on the belief that caregivers who choose to take charge of their lives, and see caregiving as but one of its facets, are in a position to be happier and healthier individuals. They are then able to have a higher quality of life and to make a more positive contribution to the well being of their care recipient, all of which has a positive impact on society and health care costs. Through its services in the areas of education and information, support and validation, public awareness and advocacy, NFCA strives to minimize the disparity between a caregiver's quality of life and that of mainstream Americans.

To learn more about how you can become a member, support NFCA, or purchase educational and promotional NFC Month materials, please call NFCA at (800)896-3650 or visit www.thefamilycaregiver.org.

Important Information About The Transverse Myelitis Association Membership Directory

Beginning with the publication of the next TMA membership directory, if you would like to receive the 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 more people who are willing to be listed in the directory to make themselves available to share information and support, the greater is the value of the directory for our members. As the directory is becoming more and more expensive to print and mail, we want to ensure that it maintains this value into the future.

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 continue to receive the directory, please call (614)766-1806 or 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.

The membership directory will no longer be mailed with a newsletter publication. The TMA is unable to receive a bulk not-for-profit discount on any mailings that weigh more than 16 ounces. The increasing size of the directories and the newsletters and the substantial difference in postage costs necessitate that we send the directory as a separate mailing. We will still attempt to publish a new directory annually.

If you are not currently listed, please consider being listed in the directory. We appreciate the willingness of so many of you to make yourselves available to assist others in your communities, states and countries.

The TMA Children's Camp

The Transverse Myelitis Association would like to hold a camp for children during the summer 2007. Shannon O'Keefe is our TM support group leader from Western New York State and has volunteered to spearhead our camp planning efforts. The camp would be for those who have TM who are 25 years old and younger and their brothers, sisters and parents. We will also be inviting our Medical Advisory Board and their families. The purpose of the camp is to provide the children and their families an opportunity to have a fun recreational vacation. If you had the chance to be in Columbus in 2001, you know how wonderful and important it is for these children and the parents to be able to spend time with each other and share experiences. The camp will offer these children and the families an opportunity to create friendships and support that will last a lifetime.

We are not planning to make the camp weekend a four-day seminar for parents. The focus of the camp experience is going to be on camp activities, whatever those might be. I think that usually involves stuff associated with dirt, bugs and a lot of sweating; all of which I try to regularly avoid. We will try to get as many of our Medical Advisory Board folks to attend and might offer one or two evening presentations or discussions. The MAB might also be available to answer your questions. Being able to spend time with the physicians and their families, and having the opportunity to bond with these wonderful folks is always a good thing.

We are currently in the process of researching adaptive adventure camps that have the facilities and the staff in place to meet our needs. Our goal is to find a camp that is centrally located in the United States and that is affordable. It would be our intention to make this camp experience affordable for everyone who wants to attend. You can translate that intention into: "we are going to need to raise a lot of money to do this, and I am going to be knocking on your door for help."

If you are in your early 20s, please consider participating. The camp would be a great opportunity for you to meet others who have TM who are about your age. You could also make a tremendous contribution to our community by getting to know the younger children who would greatly benefit from role models such as yourselves! Please consider getting involved in the planning of the camp, the fundraising, and definitely plan to attend. We need you to participate; and you are going to love the experience!

We need for everyone to get involved in the planning and fundraising. The more people we can get involved, the less one person or a few people are going to have to do. And, hey, we've got a lot going on.

If you can get involved, please get in touch with Shannon as quickly as possible.

Shannon O'keefe
sjokeefe79@yahoo.com
(585)330-1125

Whether we are able to hold the camp is going to depend on the response we receive from families regarding their interest in participating and the willingness our community has in raising the money to make this happen. I know that the children need this opportunity! Please get involved and please help us to make the TMA children's camp a reality.

Support Groups

The Americans with Disabilities Act: A General Discussion
Pamela Schechter

When the Americans with Disabilities Act was signed into law on July 26th, 1990 by President Bush, it became the first comprehensive civil rights law for people with disabilities. The law granted far-reaching protections for disabled Americans in employment, public accommodations, public transportation, housing and education. According to Percy (1989), passage of the Americans with Disabilities Act came at a time when many Americans had come to recognize the extent of disability in America and the harmful aspects of disabilities on the lives and opportunities of a large segment of the population.

The history of the ADA, prior to its enactment in 1990, was guided by many factors, including: the changing role of the federal government and the enactment of social welfare programs during the 1960s; the changing demographics of the disabled; the civil rights movement; and the emergence of a strong and effective advocacy coalition. Of all the other precursor developments, these four factors emerged as critical.

As recently as the 1960s, the flow of federal financial assistance to states and through the states to other entities, was limited, and targeted to those programs that were already in place during the 1950s. All this changed in 1965, under the presidency of Lyndon Johnson, when programs such as Medicaid, Medicare, and the Elementary and Secondary Education Act were initiated and resulted in significant expansion of federal support for social services. With these enlarged entitlements, Congress began to set conditions of participation. For example, in 1973, with the enactment of Section 504 of the Rehabilitation Act, those who received federal funding were obliged to practice non-discrimination against people with disabilities towards their employment and allow them access to public transportation. Implementation of this law paved the way for the ADA and the broader application concerning the disabled.

Well into the seventies, many policy makers viewed disability primarily as a form of premature aging, justifying early retirement with pension rights. The picture changed when the shift from the disabled older person changed to the disabled young adults. Young men and women, disabled in childhood, or those in mid-career, and who sustained the debilitation of chronic diseases were not accepting a life deemed appropriate for the disabled. This younger generation represented the changing demographics of the disabled and the emergence of a significant number of highly visible, highly articulate leaders, who became a powerful political voice and one of the decisive factors preceding the passage of the ADA.

The rallying cry for ADA was based on equal justice and equal protection, using the model of the Civil Rights Act of 1964. The strategies for achieving the ADA's enactment proceeded along similar lines. A decade of disability-related litigation began in the late seventies and was based on constitutional issues. The litigation focused on the lack of access for the disabled, discrimination in employment, the provision of inferior services and the use of institutional segregation for people with mental disabilities. The parallel paths of the two movements were further intensified by the active support of the ADA by well known leaders of the civil rights movement. And finally, as a result of a growing advocacy movement, a broadened sense of community among people with disabilities and the emergence of advocacy organizations, local, state and federal officials began to listen and to learn about the issues of the disabled. The core issues were to minimize their dependency and maximize their access to a productive and participatory life.

A brief overview of the ADA law, as outlined by Pardeck (1998), includes the following major components: employers who employ fifteen or more employees, are prohibited from discriminating against a qualified individual with a disability in all terms and conditions of employment; the law prohibits discrimination and increases the accessibility of persons with disabilities to programs run by state and local governments, such as public social work agencies, colleges and universities, it requires public transportation to be accessible to people with disabilities; private businesses serving the public must make their goods and services available to people with disabilities; telephone services must be available to people with hearing and speech impairments by providing a relay service that uses an operator as an intermediary. The law prohibits retaliation against an individual because of actions related to the act and requires the provision of information about the implementation of the ADA, Rehabilitation Act of 1973 and other state laws relating to disabilities.

With passage of the ADA, the move to ensure the rights of persons with disabilities shifted to the implementation arena. The law did not provide clear direction as to the appropriate strategies necessary for its implementation. In the period since the passage of the law, efforts have focused on interpre-

tation and guidelines for administrative action. Some of the efforts to implement the law are becoming noticeable, such as handicapped parking spaces proximate to commercial enterprises (shopping malls, restaurants and public buildings). Many commercial establishments have built special entry ramps and door entrances, sidewalk curb cuts and elevators with braille floor designations. These modifications are necessary to improve the mobility and access to commercial and public buildings for the disabled. The law also requires the systematic review of social practices and public facilities in order to ensure that discriminatory actions can be identified and eliminated.

The law has generated substantial controversy and has raised significant questions during the course of its implementation. For instance, what are reasonable accommodations to meet the needs of the disabled in public transportation and how much should be spent for these accommodations? For employers who are bound by Section 504, what is a reasonable accommodation of handicapped workers? What are appropriate structural changes to government facilities in order to meet accessibility requirements? What must public and private educators do to offer the types of special services that are mandated for the education of handicapped children, and how should these services be funded? Another controversy surrounds the criteria to be used to define disability and who is eligible for protection under the law.

Current research and comprehensive findings (Percy, 2001) indicate that progress is being made in the implementation of the ADA and in moving the nation forward to a goal of eliminating discrimination based on disability. There is discontent about how quickly the implementation is moving, as well as the number of entities who are regulated by the ADA and are not fully aware of nor in full compliance with its mandate.

According to Percy (2001), we do see enforcement agencies like the Equal Employment Opportunity Commission investigating disputes, seeking resolutions and commencing prosecutions where appropriate. And we do see new buildings being constructed according to guidelines that incorporate accessibility for disabled persons. However, significant challenges and obstacles remain that slow ADA implementation and frustrate people with disabilities.

One of the major obstacles concerns the substantial costs involved in meeting some of the accommodations for the disabled. Where compliance requires substantial expenditures of funds, implementation has been slower. Public transit systems have been struggling, for example, to comply with ADA requirements for accessible buses and responsive systems that provide door-to-door transportation. The transit authority's response in New York City has been "Access-A-Ride," a program that receives half of its funding from the federal government. The delays that are caused by the cost of these programs require a close monitoring of compliance. There needs to be a better understanding of the extent to which the financing of these programs impede their implementation. There is also a significant need for the development of new and more cost-effective programs and technologies.

Another sticking point of the law concerns questions about who is covered and what constitutes a disability for coverage. The framers of the law preferred language that related to conditions that limit one or more major life functions. As a result of the vague language, a number of court cases have focused on what conditions represent a limitation of life functions. It is quite likely that these coverage issues will continue to find their way into the federal courts where interpretations of the law will set precedents for future litigation.

As Percy notes, the challenge of mental disabilities looms large in the interpretation of the law. Protecting individuals with mental disabilities has been relatively more difficult than protecting physically handicapped persons, because our knowledge and understanding of mental disabilities, both within the medical community and in the general population, continues to change, making it difficult to understand mental disability and how it can be appropriately accommodated.

The listing of settlements and current agreements secured through the Department of Justice is impressive and illustrates the impact of the ADA. The list includes many nationally recognized companies, including hotels, restaurants, recreation facilities, and rental car companies. Each of the companies agreed to take action to address the needs and concerns registered by people with disabilities.

Current findings on administrative compliance with the ADA by municipal governments were focused on at least ten cities across the country. Once the data had been compiled and analyzed, the cities were categorized in one of three groups based on their compliance with the seven major administrative requirements of the law. Progressive cities are those that have clearly made an attempt to comply with the components of the law in a timely fashion, even though they have not consistently met the statutory deadline for each administrative requirement. They have also included the ADA in almost all elements of municipal planning and programming. The majority of the progressive cities approach the law as civil rights legislation, train and direct staff members to ensure that the provisions of the law

are fully implemented, and have adopted a grievance procedure.

In contrast, reluctant and non-compliant cities are those that have not met the majority of the law's requirements, although some have been forced to do so years after the statutory deadline has passed. The law is generally viewed as an unfunded mandate and becomes a low priority in municipal governance. Reluctant cities tend to under staff the ADA program and offer few resources for program management.

The study reveals that implementation of the law is most successful in cities where an administrator has recognized the importance of the ADA as a civil rights law and the potential legal exposure it represents. Implementation is slower in cities ranked as reluctant and non-compliant, as a result of a belief that the law's provisions are vague and unlikely to be enforced. This attitude could prove legally expensive for a municipality choosing non-compliance.

In studies done at Cornell University on ADA implementation in the federal and private workplace, researchers have found that while many of the 43 million adults with disabilities are eager for employment, these individuals still face substantial barriers in the recruitment, retention and career advancement processes. According to principal investigator, Susanne Bruyere, Ph.D., director of Cornell's Program on Employment and Disability, barriers that impede applicants and employees with disabilities exist mainly because of negative workplace attitudes and supervisors' lack of information and training on ADA legislation. The research also indicates that these barriers exist in both the federal and private sectors. Dr. Bruyere concluded that attitude issues are most effectively addressed when they are integrated into an organization's staff development training and when managers and supervisors are trained on the various accommodations necessary for different disabilities, as well as how to effectively supervise employees with disabilities.

On January 13th, 2004, the Supreme Court heard oral arguments in a case that raised questions about the constitutionality of the ADA. While Tennessee v. Lane did not directly challenge ADA's provisions governing building design and construction, some of the justices' questions focused on architectural barriers that the law addresses. The case stems from a 1998 lawsuit filed by George Lane and Beverly Jones, both paraplegics, against the state of Tennessee and alleging violations of the ADA's Title II. Title II bars government entities from denying public services to individuals with disabilities and allows those harmed to seek damages. Lane claimed that while attending a hearing, he had to crawl up two flights of stairs in Tennessee's Polk County Courthouse. He sued the state for what he characterized as humiliating treatment that violated the disability act. On May 17th, 2004 in a 5-to-4 ruling, the justices agreed that George Lane and other disabled persons could sue, if states ignore the ADA law that protects their rights.

Although the Americans with Disabilities Act has brought about many positive changes for people with disabilities, there is still much to be done to ensure integration into mainstream society. It is important for all of us to become effective self-advocates for our rights under the law. To become an effective advocate, we must know the law, our rights and entitlements, and we all need to have sufficient knowledge in order to recognize when these rights have been violated.

Because I am one of the thirty-five or forty million Americans with a disability, writing about the ADA is both a personal and professional experience. It is personal, because the law has enabled me to attend school when I could not use the conventional public transportation system. By providing me with paratransit services, an alternative system for persons with disabilities, I was able to attend school on a regular basis. I will eventually obtain an M.S.W. and realize my professional goal of becoming a social worker and helping others with disabilities obtain the protection and benefits of the law.

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Optic Neuritis Support Group of the TMA
Alma A. Hallock
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Hello. My name is Ali Hallock. I joined the TMA in July of 2004. I learned of the TMA through a neurologist at Mayo Clinic. He thought the association was a great way to meet other people suffering from rare conditions.

Over the past three years, I have been battling Optic Neuritis. Optic Neuritis is the inflammation of the optic nerve. It has taken all sight in my left eye. ON even tried taking the right eye, but it was saved with lots of medication, faith and strength.

As frustrating as ON is, it is not as frustrating as not being able to find support of others with the condition. Through the TMA, I am going to be heading up a forum for people with ON. I am looking for others with ON to hop online and participate in the discussion. If you know anyone with this blinding condition, please direct them to the TMA website.

It was nice to meet those of you that attended the TMA symposium in Baltimore. I thought the lectures and discussions were very thorough and insightful. Today I have hope that we will be healed, because of the perseverance and persistence of the doctors that are working 'round the clock' for all of us.

Ali's Optic Neuritis Forum can easily be found by going to www.myelitis.org and clicking on the link 'About Optic Neuritis.'

TM Support Group of Ireland
Ann Moran

Three generations of Morans are having a family reunion. They come from Ireland, England, USA, Australia, and Zimbabwe. My husband's family travelled long distances from their homeland; as did many people in Ireland in the 1950's.

Well, now down to the TMA business!

I have had three new people diagnosed with TM contact me through logging on to the TMA site, which is great. I keep battling on with creating awareness. I am hoping to get some funding through the Health Board to design a three fold information leaflet that can be distributed throughout the country to all Health Boards, hospitals, clinics, and general practitioners.

Thank you to all who sent me information on Social Security/Disability payments. I made out an information sheet and took it with me when we met with our government members. This meeting was organised through "Disability Federation Ireland." I also made several copies from information on the TMA website and gave them to almost everyone who came up and spoke to me. Some found it very interesting. I was also wearing a badge with Transverse Myelitis Support Group Ireland on it, and this seemed to make people ask questions, not having heard of TM before. I was very dry by the evening time from all my talking! Two of the national papers took statements from me regarding funding and community employment schemes and regarding Transverse Myelitis awareness.

Before I end, I want to say a sincere thank you to all of our officers of the TMA. If I remember rightly, when I received my first newsletter from Sandy back in 1997, there were only about 140 people on our membership list. Look at it now; how the TMA has grown. All of this would not be possible without Sandy and his family. It would not be possible without Deanne and Dick Gilmur who made it their business to bring us all the information they could find in the early days, while trying to find answers for themselves and their daughter. Where would we all be today without them. Thank you so much for sharing all of your findings with us and for making the TMA a reality.

I know I felt such a relief in my life to find that there were others out in the world who were stricken with this TM nightmare. For seven years I was working in a fog; no one could tell me what was really wrong with me.

We must not forget Jim Lubin for his dedication and hard work with the TMA web site. If you are lucky enough to meet him, and I have in Seattle, you will see what a remarkable person he is.

Thanks also to Debbie and Paula, who keep the wheels turning on the administration front. All these people work very hard for the TMA, totally voluntarily, giving their time; and they also have jobs and families to care for.

Best wishes,
Ann Moran,
Westport, Southern Ireland

TM Support Group of Egypt and the Middle East
Mahmoud Abdel Moety Hassan

As English is not my first language, I would ask for your understanding in reading my article. My name is Mahmoud Abdel Moety Hassan. I live in Alexandria, Egypt. I am 23 years old and I am a B.Sc. student in the marine

engineering department in the faculty of engineering Alexandria University.

My story with TM started on Tuesday, June 24th 2003. It was half past eleven in the morning. We had just finished the third year exams and we were gathering at the college (me and a group of my friends) to discuss some issues related to the fourth year (B.Sc.) graduation projects. It was during this meeting that I started to feel an intense and strange pain in my left foot. I suspected that something was very wrong. The pain in my left foot then decreased, but moved to my right foot. The intensity of this pain was greater and so I asked my friends to immediately take me to the hospital.

Unfortunately, the doctors in that hospital failed to understand what was going on with me. I stayed in this hospital for an hour during which time I lost all feeling in both legs from below my knees. I was only able to stand by being supported by my friends. I finally decided to go to another hospital. I entered the other hospital in a wheelchair. I was nearly paralyzed below my waist. At this hospital they diagnosed what I had as a psychological case. I was so disappointed and frustrated.

My friends and my brother decided to take me out of this hospital. One of my friends suggested that they take me to a brain and neurology specialist. They took me to this doctor about an hour and a half after I left the second hospital. The doctor examined me and said that it may be psychological or it may be something in my spinal cord. As I am a student, he moved me to the student's hospital.

During a period of about a week in this hospital and with the necessary MRI and scans, I was diagnosed with TM. I've been in the hospital ever since. I am able to leave the hospital at times to attend some of the lectures and sometimes to be with my friends. My family, friends, and professors and instructors at college have never made me feel that I am sick. They treat me as if I am perfectly normal.

One of the strange things that occurred before I got TM is that I felt that something was going to happen to me. I recall that I told my mother that I feel that something really great will bring me down, but that God will help me in the end; even though I had no reason to suspect anything at the time.

Having TM has been very difficult. I have been communicating with Sandy for the past few months, and have decided to start a support network for the countries of the Middle East. If you are interested in getting involved, please get in touch with me. I have set up a web page for our support group under the link 'support groups' on the TMA web site. Having others to share our experiences with will be a great help for all of us. We can also help each other find resources in our countries, help each other find physicians who understand TM and who understand how to treat the symptoms of TM. Please feel free to get in touch with me.

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Dyllice Eastwood and Jennifer Murray

My name is Dyllice Eastwood. I live in Auckland, New Zealand. I have had TM since 5 February 2001. Although I have been in contact by telephone and internet with other TM people, both here in New Zealand and overseas, early this year is the first time I actually met another TM person. Her name is Jennifer, and she lives only ten minutes away from where I work. We had been in the same hospital and rehab centre, although Jennifer got TM six months after me, and we both agreed that if we had been told about another person who was going through the same trauma as ourselves it would have helped us a lot. So we decided that we would like to start a TM support group in New Zealand.

My TM started very fast. Suddenly, on the morning of the 5th, I developed a very bad back pain. When I tried to stand up about an hour later, my right leg wouldn't move. The pain in my back got worse all that day and my left leg started weakening too. I told myself it would be better in the morning, but, of course, it wasn't. So, my husband took me to our doctor. He had to almost carry me into her surgery. Even now, she says she will never forget how I looked when I arrived at her surgery. She immediately arranged for us to go to Auckland Hospital. By this time, I was unable to walk. I had an MRI, CAT scan, and lumber puncture,

which showed spinal cord lesion at T8 and T9 consistent with transverse myelitis. The neurologist ordered another MRI the next day, because they were unsure of a blood clot at T10.

I was treated with IV Methylprednisolone and a reducing dose of Prednisone for three weeks. One of the worst feelings for me was the tight banding across my stomach and severe discomfort of clothes rubbing against my skin. I still suffer from banding, but it is not so severe now or as frequent.

I stayed in the hospital for 11 days. I began to walk a bit, at first with a walker. The hospital staff encouraged me to move about as much as possible, and I must say that just getting to the toilet on time was an effort in itself. I feel lucky that the bowel and bladder issues have not lasted, although there are still a few problems there. The walker was replaced with crutches, and from then on I never looked back. They gave me much more mobility. I was then taken by ambulance to Rehab Plus, a rehab centre closer to home, where I was taught to walk up stairs – still with my crutches, to walk in a straight line – that seemed very difficult. I had pool therapy which I loved very much, and gave me hope that I might be a bit normal again.

Rehab Plus had a great physio regime, and they worked strengthening my legs and torso. I was also allowed to go out for a drive with my husband the first weekend I was there, and allowed to go home for the following weekend. That was bliss. They discharged me the following Thursday, because my son was getting married on the Saturday, and I was improving. Because I had been in the hospitals for one month in total, I hadn't got my "Mother of the Groom" outfit, so my son arranged for the lady making the wedding dress to come to Rehab Plus and make me an outfit too. I said what colour I wanted and the style, and she arrived armed with different materials for me to choose from. The nurses and patients got involved a bit, too, adding their say. Looking back, I really must have looked a sight. I was hunched over a bit, wobbling on crutches, and my hair suffered with all the medications I was taking.

However, I really wanted to walk down the aisle (without crutches) behind the bride and groom, and this made me determined to keep up walking as much as possible. Up and down the corridor I walked, and I can remember phoning my husband to say that I had walked 19 steps without any aids at all. Well, I did manage to walk down the aisle, clinging onto my husband's arm. I cried my eyes out when we got outside; the effort was more than I realised.

Rehab Plus booked me into another rehab centre, Waipareira Trust, until there was a vacancy at Rehab Plus outpatients. I went there twice a week, where we did pool therapy one day and exercises, stationery cycling, etc. the other. I was there about six weeks. Then back to Rehab Plus. On the first day, my physio asked me the three things that I couldn't do now that I would really like to do. That was easy, walk better, drive the car, and get myself up off the floor without looking like a beached whale! So, she did leg strengthening by putting weights on my legs and I would lift my legs up and down. We used a wobble board for balance and the parallel bars. By the time I left there, I could walk better. Today, I have to concentrate when I walk or else I stagger at right angles, and especially if I am carrying anything. I can drive again, but I still can't get off the floor. So, two out of three isn't bad.

I still battle fatigue and memory loss, and have a lot of pain, especially my waist and legs. My toes feel like they are turning under, although they aren't.

Through all of this, my family has been wonderful. I know that without their support and encouragement, I would never have improved as much as I have. I am so proud of them; especially my husband, who would spend hours at the hospital because I needed him so much. My blood pressure was always lower when he was there, because he helped me to relax.

The worst thing was that if I had a pain or problem, no one really said anything. If the doctors or nurses had said, "Oh yes, that is a symptom of TM," it would have been alright. But I started to feel they were beginning to think I was making things up. I felt very alone. So, I look forward to the support group and meeting new people, so that they don't feel so isolated.

Kind Regards,
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My name is Jennifer Murray and on 17th November 2001 my life was changed by TM. The onset was very quick, going from a sore back in the morning to numb legs in the afternoon and, finally, paralysis from the waist down by 10:00 PM.

My TM was diagnosed very quickly. From my first MRI that night, I was told it was probably TM (lesions at T10-12). I was also tested for various other things, and a spinal tap was done. I was started on IV steroids the next morning. Unfortunately, the steroids didn't bring much change. After five days I could only just wriggle the toes on my left foot. So, I had to undergo Plasmapheresis. No one could give me any useful information on TM. All I was told was that I would have to wait and see as far as recovery went.

Four weeks later, I was transferred to Rehab Plus. I was still in a wheelchair, but I had some movement in my left leg and I could wriggle the big toe on my right foot. I still had no sensation from my waist down and I was still on a catheter.

I spent three months at Rehab as an inpatient. During that time, I had to learn how to dress myself, how to self cath and try and regain some of the dignity I felt I had lost. Having someone wipe your bottom for you is about as low as you could go in my book. The hardest work of all was to strengthen the muscles required to walk again. I loved the pool therapy. You could do things in the water that you couldn't do on land. I'll never forget the day my Phsyio said 'now take a step.' I was so excited! So, I stood there with a person on each side holding me up and I went to take a step. But nothing happened! Then I realized, it was because I didn't know how to. Walking was never something you had to think about. It was something that just happened. I had to ask how to take a step. So, they told me what I had to do and I did it! That was eight weeks from the onset of TM.

By the time I left Rehab, I was using a walking frame. I continued with Rehab for another four months as an out patient, going in three times a week for physio and occupational therapy. I was using two crutches by the time I finished there.

I was also under the Otara Spinal Unit. They tried to transfer me there, but no beds were available at the time. I see the urologist and consultants as an out patient. I also received physiotherapy for another 12 months.

I now use one crutch around the house and two when I go out. I have ongoing bowel and bladder problems, muscles spasms, and other symptoms. But, hey, we make the most of the good days and do what we have to do to get through the bad ones.

One of the hardest things through all of this was dealing with the unknown. No one could answer my questions. Finding the TMA site was wonderful. At long last, I found some answers. I also found a friend in Dyllice. It's nice to have someone who knows what you mean when you say things like "I can't feel anything on the outside of my legs, but I can feel things on the inside." If only Rehab had said something to me about Dyllice having been there six months earlier with TM, and helping me get in contact with her. To see someone 'walk' through the door who had TM would've given me so much hope. I look forward to meeting new people through this support group, sharing our experiences and maybe even helping each other with tips that we've learnt along the way to make our lives a little easier.

Kind Regards,
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TM Support Group in Scotland
Margaret Shearer

My name is Margaret Shearer and I live in Prestwick, a small seaside town on the west coast of Scotland in the United Kingdom.

I awoke on the morning of Monday 31st December 2001 with terrible chest pains. I convinced myself that I had heartburn; and, by the way, I had never previously suffered from heartburn. I took appropriate meds to no avail. I was, at this time and for the previous seven months, living and working in Miami Beach, Florida. During the next hour, the pain became so severe that I found I was having difficulty taking a breath and I knew I would not be fit for work that day. As I had been feeling fine up to retiring the previous evening, my boss took me to the emergency department at Mount Sinai Medical Center on Miami Beach.

By the time we made it to the ER, I was aware that my left leg was not capable of bearing weight. The medics felt, due to the extreme pain in my chest area, I should be admitted with a suspected heart attack.

Tests came back negative. I asked if I could be allowed to go the toilet, as I had not gone since the previous evening. The fact that I could not pass urine was not as concerning to me as the realization that I could not walk properly. When trying to get back onto the trolley, my legs were collapsing from under me. I was then reexamined and told that further tests would be done on what was thought to be a stroke. That evening, despite all tests being negative, I was admitted due to the increasing pain being experienced in my breathing.

As midnight approached for the start of 2002, my thoughts were with my three sons and my extended family back home in Scotland. I was hoping that all would be okay within a few days, and that they would never know I had celebrated the New Year in the hospital! During the early hours, I called in a nurse to ask if I could go to the bathroom and she assisted me. I was still not able to urinate, and decided to return to my bed. I fell onto the floor trying to get up from the toilet. My legs were now both totally incapable of holding me up and my right arm did not have the strength to pull myself back up onto the toilet.

I was concerned that the paralysis was spreading and aware that my trunk area was also numb. My chest felt as though it was being squeezed so tightly that I could not breathe properly.

At this point, I insisted on being catheterized thinking it would resolve the discomfort. Of course, that wasn't so. In the morning of January 1st, I was told that further tests would have to be done. A stroke was still being considered, especially as my condition was deteriorating despite my taking painkillers. I had four spinal taps done and several MRIs in the next four days. At one point, I was told that I would have to go for a surgical procedure to remove a growth on my spinal column. A second opinion was sought and further scans revealed that I had a lesion from T1/2 through to T7 and they were considering TM and, possibly, MS.

Only my left arm was functioning normally. I finally broke the news to my family about what was going on with me. I insisted that I would be back to normal and at work again in a few weeks. I was led to believe this by the consultant. I was also told that I was the first person at the hospital to have been admitted with these symptoms and concerns. Suggestions were made that perhaps I had caught a virus while I had been in Scotland a few weeks previously. This was later discounted as the lumbar punctures were returned with no evidence of a virus or bacteria.

On the 4th January 2002, I was officially diagnosed as having Acute Idiopathic TM. I was placed on intravenous steroids, gabapentin, amitryptilin and antibiotics. I continued to remain at Mount Sinai and was receiving excellent treatment, including physio and occupational therapy. My sons, who were studying Neuroscience and General Medicine in Scotland, convinced me that it would be in my best interest to remain in the States, as there were no TM specialists in the UK. My insurance company was also paying all of the medical bills and receiving weekly updates from the consultants on my progress from the rehab department that I was attending for six hours every day.

By March, I still had not reached a state of self-care. When the insurance company suggested I go into a supportive care home, I decided that I should return to Scotland. My hope was that I would continue to make progress to a stage where I could go back to work. In Scotland, I was given fulltime, around-the-clock care in my own home and attended the Douglas Grant Neuro Rehab Centre in Ayrshire where the staff were so caring and have continued to support me these last two years. I have discarded my body brace and leg splints and learned to walk again over short distances with the aid of a walking frame and an ankle splint. I have continued to need all of the meds that I was taking in the States.

On the whole, I no longer need constant care. I have superb support from my GP, the Urology Department and all of my family and friends. In 2002, I met my challenge of learning to drive again and now have a specially adapted car that takes me and my wheelchair, as well as passengers. Last year I read of Cody Unser going scuba diving and decided that would be my challenge for 2003. I started training with a physio at a local swimming club for people with disabilities and increased my stamina so that I could swim the 25 lengths of the pool. I then had to convince my consultant to allow me to try out the scuba equipment, as I needed a medical certificate. In August, I completed all of my ocean dives in the Mediterranean to gain my Open Water Scuba Diving License. The fact that I was in a wheelchair did not make a difference to my instructors and I managed to enter the boat at the harbour side on each dive.

At the present time, I am still in constant pain which permeates my upper back area. It is very sensitive to touch. I receive a course of acupuncture at various times which helps to reduce the pain to a point of acceptance and allows me to continue with exercises. I can walk for short distances with my walker, but use my wheelchair outdoors as I constantly fall without any prior warning. My left leg has regained mechanical function, but remains numb to touch. My right leg has no feeling in it at all. My right arm has regained 6/20 strength, but goes into spasms when I use it too much. I have had numerous UTI's, which knock me back a bit. I experience daily spasms, shooting pains, tingling sensations, headaches that range from crushing and throbbing to aching and feelings of nausea. I am on a lot of medication, including herbal and also have become a vegetarian since reading a book 'Foods That Fight Pain.' I have learned to self-cath and need suppositories for my bowel to empty. I still have the banding feeling in my trunk area and, at times, the spasms there still take my breath away. Recent urology tests show no improvement in that area, but I tell myself that it could be worse. I have had no accidents!

During my initial stay in Mount Sinai, my neuro told me that I would be able to find out more about my condition on the Internet. On going in to the TMA web site, I found that there was a UK contact and I emailed Geoff Treglown that I was returning home and would be interested in any information from him. Within a few days of my arrival here, Geoff contacted me on the phone and has been supportive ever since.

In October 2003, I took up his invitation to attend a support group meeting in Manchester. For the first time, I met people with the same condition as me, including Sandy from Fife in Scotland who, like me, had driven more than five hours to attend this meeting. I was thrilled to hear so many stories from such a courageous group of fellow human beings. Aware that no such group existed in Scotland, I asked

Geoff to see if any people in Scotland were interested in a group meeting.

With the wonders of email and the telephone, the first Scotland Support Group for TM met in November in Glasgow. We had ten people who have TM come from all areas of Scotland to attend that meeting with several caregivers. There were seven people who could not attend, but hoped to do so at the next meeting in January 2004. Since then, our numbers have grown to twenty people. They range in age from 22 to 68 years and the members of our group have been diagnosed with the condition for time spans of three months to 54 years. There are 15 females and five males in the group. Two of our members have Reccurent TM, one has Devics, one has ADEM and 16 of us have TM. All of us have attended various hospitals in the UK, take a variety of medications and are at different levels of recovery.

Our meeting lasted for over four hours. We each introduced ourselves and discussed how the condition had affected our lives, both personally and professionally. Like Sandy and I, this was the first time that any of them had met another person with TM. We discussed the benefits of our various medications and what types of treatments we were getting from the various medical personnel and departments. At the close of the extended meeting we agreed to meet again after the holidays.

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TM has changed my life in so many ways, but the people I have had the privilege to meet because of it has been both gratifying and joyful. No words could express the level of kindness, care and support that has been shown to me these past two years. I hope that our Support Group will be the beginnings of getting the word out to our medical community and to encourage them to perform research on TM. We will continue to share our experiences, showing empathy and compassion, and making others aware we are here to assist anyone who contacts us.

Future meetings will be held in the Neuro and Spinal Rehab Centre at Southern General Hospital, Glasgow. Announcements of our meetings and other information about our support group will be posted on our web page; please check our page regularly. (www.myelitis.org; click on the link 'support groups;' click on the Scotland Support Group page). If you are interested in the Scotland TM Support Group and want to know more, please feel free to contact me.

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TM Support Group of Alaska
Jennifer LeMay

I was 27 years old, had been married eight years, adored my three year old son who was the pride and joy of my life, and loved my fast-paced job working as an engineer in Anchorage, Alaska. On 17 September 2002, my life changed drastically when I woke up with severe pain in my lower back. The pain was unlike anything I'd ever experienced, and I went to a doctor that day. She thought I had a possible kidney infection, kidney stones, or some other kidney disorder. I felt reassured that there was a reason for the pain and that things would get better soon. However, life would be far from normal, as my condition deteriorated rather dramatically from September to December.

For the next six weeks, my pain focused in my lower back as a burning feeling. Laying down made it more bearable, but the pain was there 24/7, and I was not able to do anything but lay flat on my back 98% of the time. Pain pills didn't seem to help. The pain worsened, my blood pressure increased, and my reflexes were very brusque. Tests determined my kidneys were fine. In early November, I began a terrifying, rapid downhill spiral. There were new symptoms appearing every day. My mom flew up from Montana to help us. The pain radiated downward from my back to the tips of my toes. My legs began having weird sensations that lasted hours at a time. I couldn't stand very well, and my legs were threatening to give out on me. I had blurry vision, and it sometimes felt like a camera flashbulb was flashing repeatedly in front of my eyes. My bladder and bowel were giving me problems. I lost my appetite and didn't feel very human anymore since I was living my life lying down and on pain medications that didn't seem to work. I went through numerous tests and rounds of blood work, but nothing could pinpoint a cause.

The pain became worse, which I didn't think was possible, and more of my body was going numb or having crawling sensations. My joints ached, and my muscles from head to toe were in constant pain. The only parts of my body that didn't hurt were my nose, tongue, and teeth. I became hypersensitive to touch so that taking a shower or walking across the carpet felt like needles pricking my skin. Any touch of others hurt so badly; the texture of the buttons on the phone dug into my skin. My son was no longer able to sit on my lap, and my husband couldn't hold my hand. I felt useless, fragile, and breakable and no one had a clue what was happening to me.

The feeling in my legs began to diminish, and both legs were feeling like dead weight. My balance was next to nothing, and I would lean into the walls when my mom or husband helped me walk. I was getting electri-

cal jolts up and down the backs of both legs. Some of them were sharp enough to make my legs jump forward. I began to travel in a wheelchair and got a handicapped parking permit. My neurologist prescribed baclofen in November which stopped the muscle spasms and seemed to strengthen my legs slowly. A SSEP showed delayed response from my spinal cord to my legs. I tried physical therapy, but the therapy made my heart race and legs go completely numb.

On 2 December, I was able to wiggle my toes for the first time in weeks. I could now sit up for eight to ten minutes at a time and would get chest pains from the exertion. My hands, legs, and feet were numb all the time. I had lost 26 pounds by this point. My baclofen dose was increased, and I was able to start walking on my own again. The hypersensitivity left. This was the first encouragement I had had in 11 weeks. I was even able to stand up one night and brush my teeth next to my son for the first time since this started. Talk about exciting!

I had a spinal tap in mid-December, and the results showed a slightly elevated protein level. I continued to make some progress. For the first time out of my three visits with the neurologist, I was able to walk into the office (not in a wheelchair!), sit in the waiting room (for previous appointments, I'd have had to lie on the floor, because I was in too much pain to sit), and sit on the exam table rather than lay down. The test results were still not leading to a conclusive diagnosis, although the most likely possibility that explained all of my symptoms was transverve myelitis of the cervical spinal cord. On 19 December, I actually had a diagnosis of TM. Finally, this disease had a name!

From January through September of 2003, I felt like a mountain climber. I'd have periods of time where I would feel like I was making baby steps forward, and then I'd feel like I was sliding backward in giant leaps. I had extreme fatigue. My hands felt really crippled and stiff, and it was very painful to write with a pen. Typing was easier, but left my fingers burning. The burning in my lower back continued. My feet still went numb every now and then, and my ankles and knees throbbed. My reflexes continued to be very brusque, but I learned how to walk again in physical therapy, and the spastiscity in my legs stopped by September. My mom went home in mid-January, but it was too much for us to manage on our own so she came back after two weeks and stayed with us until March (five months total). I tried a wide variety of medicines, but with the exception of baclofen, nothing helped.

Eventually, the pain began to dissipate in my entire body and centralized itself into my lower back, hands, feet, wrists, and ankles. By September, I was feeling pretty decent (not normal); but compared to the last year, life was pretty good again. I went back to work a few hours a day and spent most of my time at home resting. My physical therapy visits ran out, so I joined a gym. In the beginning, I could only work out on the elliptical trainer for three minutes. I'd be so exhausted, I'd have to rest so I could drive home. With time, I was able to lengthen my workout time and level of intensity. However, I had a relapse in October. A lot of the symptoms from the previous year returned. My progress literally went back to where I had been three months before. It was so scary to have wobbly legs again, numbness in my hands and feet, burning in my thighs, increased intensity in my back pain that radiated downward, pain in ankles and wrists, and spots in my eyes. My activities came to almost a complete standstill. One night, my son (then four years old) went to bed and laid there sobbing. I went in to see what was wrong, and he said he didn't like me hurting so much more. He prayed that night that G-d would make me feel as good as he did.

I had another spinal tap in November. The plan had been to wait until we received all the results in three weeks before we decided the next step. However, I was finally able to convince the neurologist that if I were a sinking ship, I'd rather have a life raft while the boat is sinking rather than a salvage attempt weeks after the boat sank. He prescribed three days of IV solu-medrol. The two most noticeable differences from the steroids were that my hands felt better within a few days (not crippling up again; numb, constant burning pains along the outlines, and sore wrists which made any hand movement difficult) and there was now no more burning along the sides of my legs. Everything else was pretty much the same (throbbing ankles, burning back, fatigue). By the middle of December, I estimated I was back to where I had been in the middle of September. I continued to be in about 75% less pain, and my ankles quit throbbing all together! The spinal tap showed increased protein again.

In 2004, February was a good month. March, April, and the first few weeks of May were difficult and painful. I tried a five day treatment of IV solumedrol in early March, but this seemed to make things worse. Things were still better than a year ago, but it was hard to feel like I was going backwards again. I woke up one Sunday toward the end of May and felt noticeably better. This was as memorable a turning point as 2 December 2002 had been. There is no medical explanation, and my neurologist was so surprised at my next appointment. I've never seen him so smiley and he repeated several of the office tests we typically run through, because he couldn't believe the progress. I believe G-d touched me in a mighty way and made the nerve cells remyelinate.

Fatigue is still something I deal with, but it is better than a year ago. If I overdo something, my back will burn (ibuprofen 800 helps). My hands stiffen up sometimes, but baclofen is helpful (I've even been able to decrease the dose 50%). I have gained a new perspective on life through this roller coaster ride and have become a better person through this experience. I rely on G-d for my strength, because I can do nothing on my own. I wanted to give up so many times permanently (especially during the first year), but G-d always had His hand on me, and I kept going when I thought I could no longer do so. I appreciate the small things, take time to smell the roses, answer my son's never-ending questions, and feel blessed to wake up and be able to feel pretty good. I'm so thankful that I am able to be sturdy on my feet and can park away from the handicapped spots and feel strong enough to kick a soccer ball with my son. There is no guarantee of the future. I don't know if I'll have another relapse and will start sliding down the mountain again. I am scared sometimes at the unknown, but generally, I am able to keep my eyes focused ahead. I have really enjoyed the last four months and will continue to live day by day.

It was great to be able to go to the Rare Neuroimmunologic Diseases Symposium in Baltimore last month. My family and I have never met a more accepting group of people in our lives. People came to gather information and gain support from others who know what they are going through. I enjoyed being able to laugh about some of my oddball symptoms with others that have had the same things. Considering the trials we have faced and continue to face, we are an upbeat group of people, and the symposium was so positive and encouraging to attend. My husband and I volunteered at the symposium to start a support group in Alaska for people with TM. We want to encourage, listen, laugh, cry, and provide support for others in our shoes and those that are going through endless tests and specialists and still do not have a diagnosis.

Our motto is "pain is inevitable, misery is optional."

Jennifer LeMay
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(Please put TM in the subject line so we don't think your message is spam. Thanks!)

Welcome to the Connecticut TM Support Group!
Jean Giordano

I feel like I'm coming out of the closet.

Hi; my name is Jean Giordano. I got TM in 1986, but wasn't diagnosed until 1999. Before that it remained a mysterious, unknown illness. It was a relief to finally know what had happened to me. But I was still alone. Though I did a little research at the library and on the Internet, I wasn't ready to look too deeply into it. I mentioned my 'discovery' to my few friends and family. It did not make much of an impression on them; probably because of my own fears.

Recently, I got an inner nudge to browse the Web again. This time I found the TMA site. I read about other people with TM. I saw that there are support groups and resources, people working to help find a cure, and exploring ways to function better in daily life. I felt a surge of heat in my body, both exciting and uncomfortable. Something in me was aroused from a long slumber.
During all of those years, I searched for healing; by finding the spiritual purpose for having this rare condition, and by using alternative therapies. Now, my quest has brought me to the point of acknowledgment: joining the TMA and starting a support group in Connecticut.

This group is for people who have any form of TM, and their family, friends and care givers. It's so important to know that there are others out there like you and me who actually understand our experience.

If you live in Connecticut and would like to participate in this group in any way, or have questions and want to talk, please contact me:

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TM Support Group of Maryland / Washington DC
Alan and Kelly Connor

The MD/DC support group has had two meetings since our inaugural meeting in March 2003. We met in September and again in January 2004. The meeting in September was held at Snyder's restaurant. Dr. Adam Kaplin, Consulting Psychiatrist, JHTMC, was our guest speaker. He spoke on TM and Depression. A detailed article is available on this subject in the October 2003 issue of the TMA newsletter. We had 12 people at this meeting.

In January, our meeting was held at Gunning's Seafood restaurant. Our guest speaker was Dr. Stephen Wegener, a psychologist at the Good Samaritan Rehab Hospital in Baltimore. Dr. Wegener is also a consultant for the JHTMC. Dr. Wegener addressed the psychological aspects of recovery and living with a chronic illness. In summary, there are three things that people who do well after TM have in common. The person feels supported, they are flexible, and the individual has an activity or some-

thing to look forward to. We also discussed some of the ways TM'ers can improve their outcome: Be pro-active in your care, by finding doctors that will listen and work with you, control depression, control pain, and have an exercise program to keep yourself as active and healthy as possible. We had 14 members at this meeting.

Our next meeting is scheduled for May 15, 2004, and our guest will be Julie Farace, RN from the JHTMC. Ms. Farace will be discussing what a typical visit to the TM center is like, as well as what services are provided there. We will also be discussing some of the common residual effects of TM and the associated treatments. I know some of our regular members will not be able to make this meeting and we will miss you. We hope to also see some new faces.

We have been participating in some fundraising. (Fundraising is strictly voluntary and not required to join us for meetings). We are selling raffle tickets that will raise $300 for the TMA. This raffle takes place in mid-May.

Take care,
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Joyce Booth

TM Support Group of Mississippi

Hello, TM Family. My name is Joyce and I have had TM since September 1992. I had just turned forty-two years old when it caught me. I have begun a support group in Mississippi for all of the TMers, their families, caregivers, and friends to come together and be able to discuss our problems and needs with someone that really understands.

My journey started on September 17, 1992….

I do not let myself become discouraged. Life is too short to waste time worrying about what one cannot do. I have a good sense of humor and a strong faith in G-d. I feel these two traits have helped me to get to where I am today. I have learned that if I cannot do something the way I used to, I should try to find another way. If it is something I just cannot do at all, then it cannot be that important. It can wait till someone else can do it.

I may have to give in to some of the symptoms I have from time to time, but I will never give up. Everyone has a purpose in life. We each need to find it and live our lives to the fullest.

Joyce lived her life to the fullest, she had a purpose, and she never gave up.

It is with the deepest sadness for me to write that the TMA and the TM community have lost a dear friend. The world has lost a champion for goodness and kindness. Joyce passed away on Wednesday, December 31, 2003.

Joyce was going to college to become a social worker. It was a passion for her and her dream. Joyce may not have earned the degree; but she was already doing the work. She was passionate about her work for the TMA and her support of the TM community in Mississippi. Joyce had monthly meetings of her support group. For those of you involved in leading support groups, you understand the dedication and effort and energy required to hold these meetings on a monthly basis. Joyce was always reaching out to her group and offering support and encouragement. She had a clear understanding of the need of support and encouragement and she was generous and kind in offering this support to others. She was on the telephone regularly with people from her community. So many of us had regular communications with Joyce via email. Joyce often met with people personally in her community when there was a need for help. Joyce was a steady source of strength for so many of us.

The TM community is spread out all over the country and all over the world. My relationships take place on the telephone, in letters and in email messages. So it was with Joyce and me. I communicated with Joyce regularly through emails and on the telephone. Joyce was just a really sweet and dear person. Joyce had so many of her own physical and emotional challenges to deal with and yet she was able to find the strength and the courage to reach beyond her own difficulties to help others. Joyce wanted to help people. She took the experiences from her own life and she offered these as gifts to others who learned from Joyce and were buoyed by Joyce's wonderful spirit.

There is a void in my world; a deep and sad void. I will personally miss this very sweet person. I have lost a good friend. The TM community has lost a friend and a great supporter. Joyce was tireless in her work for our community. She will be deeply missed by all who knew her; she will be missed by everyone in our community who benefited from her kindness, her caring nature and her positive energy.

We love you, Joyce. Her memory should serve as a blessing for all of us.

TM Support Group of New York
Pam Schechter

Since the publication of the last Transverse Myelitis newsletter, the New York Support Group, which focuses on Transverse Myelitis awareness and education, held its milestone meeting, the tenth on August 23, 2003 and the eleventh on January 24, 2004. We hold our meetings at Ben's Delicatessen and Restaurant in Bayside, Queens, because the location is in a shopping mall adjacent to a major highway and is accessible to members who live outside the city.

Our anniversary meeting, the tenth, was chaired by Paula Goldstein, C.S.W. who is a licensed social work practitioner. She is currently group coordinator at the Center For Coping, which offers individual and group counseling and teaches coping skills to help people deal with a variety of medical problems, stress, family problems, or other difficult life situations. She is also the director of patient services for the Long Island/Queens Chapter of the Lupus Foundation. She specializes in working with people with chronic medical conditions and is a frequent guest speaker for many organizations. She is the co-author of several books, including "Coping With Breast Cancer" and "Successful Living With Breast Cancer," published in 1999 and 2002, respectively. Because of her impressive credentials and experience, we were delighted that Ms. Goldstein had kindly consented to be the principle speaker and to lead the discussion at the meeting.

At the beginning of the meeting, Ms. Goldstein distributed handouts to the members and their guests. It was entitled, "Coping With Transverse Myelitis" and listed fifteen statements as points of discussion during the meeting. Ms. Goldstein based the statements on her prior professional experience with treating people with chronic illnesses. Some of the statements discussed included: every person deals with Transverse Myelitis (TM) in the same way (this statement generated a very lively discussion that ranged from outright opposition to, surprisingly, some members who agreed with it. Other statements that led to spirited discussions included: once diagnosed with TM, people can resume some of their regular physical activities without any problems; everyone with Transverse Myelitis complains about fatigue; family members are usually able to take TM in stride; although nutrition is important, there is no diet that helps people with TM; for people with TM, coping strategies must be used consistently, and finally, people are still afraid to work with a mental health professional to deal with problems.

From this diverse mix of controversial statements (selected by Ms. Goldstein), the ensuing discussions provided the members and their guests with a forum to dispute or agree with each one based on their own personal experiences. As the meeting progressed and the discussions intensified, Ms. Goldstein listened carefully to the members who contributed their thoughts and feelings and she made many perceptive and supportive comments when appropriate. At the conclusion, the members agreed that the meeting was a productive, insightful and supportive session and members looked forward to similar support sessions.

On January 24, 2004, Mr. Richard Sabel was our principle speaker and discussed a wide range of comprehensive alternative, as well as conventional and traditional methods to physical therapy and rehabilitation for individuals with spinal cord involvement. Mr. Sabel holds an M.P.H. (Master of Public Health) and O.T.R. (Occupational Therapy and Rehabilitation), and is currently the Program Manager at Beth Israel's inpatient rehabilitation program and is adjunct instructor at Long Island University, Division of Occupational Therapy. His approach included integrated western therapeutic practice with an eastern philosophy for personal development, prevention and therapeutic function. This subject was of primary importance, because it provided a powerful broad-based method in addressing the mind-body-spirit connection that could alleviate some of the physical and mental pressures of TM.

Additionally, Mr. Sabel lectured on integrative rehabilitation, restorative yoga and breathing exercises as a means to promote the occupational performance and functioning of the members. Halfway through his lecture, Mr. Sabel commenced a series of exercises for the members to perform while seated that included body movements of arms, legs and torso, deep breathing and other exercises to promote serenity and a feeling of well-being. The consensus of the members and their guests was unanimous: they felt better mentally and physically. At the end of the meeting, they recommended that we invite Mr. Sabel to return at a future time.

TM Support Group of Virginia
Pamela New

Our group has been very busy this past year. We held our very first state-wide meeting and had an attorney who specializes in social security disability law as our guest speaker along with a retired social security intake specialist who now works with her practice. It was nice to hear from the attorney side of the disability process that we are, indeed, doing things the "right" way!

I have been working really hard to get a teen chat room working for the teens

in my group! They seem very interested in the idea and we have had a couple of meetings about the concept. I have spoken with their moms, set up chat room etiquette, and informed the parents that the room is open to anyone with internet access. Meeting with the kids has been totally awesome. They are the most amazing kids. There are six "kids" in my group; four of them are in wheelchairs. Edward is now 21 years old, having contracted TM at the age of seven. He is mobile, works, attended college and has a smile that will melt your heart! He is at a point where I want to be one day. He made a comment at our first meeting that I remember still: "I just like to forget about TM. It doesn't control me anymore." I want to be like Edward! He is on board to help me with the younger kids with the chat room. He is an awesome role model. He still has some residual TM problems, but you would never know if not for the splint on his left hand. Courtney is my other role model for my kids. She is now 23 and has been in a wheelchair since she was 16. She drives her customized van, attends college, and is totally awesome, as well. Between these two young adults, I know that the other kids in my group will come to realize that having TM is not the end of the world. Brandon, age 15, Emmanuel, age 14, and Casey, age 13 are all in wheelchairs. Mia is 16 and has some mild mobility difficulty with her left leg, some residual from her initial TM attack.

The chat room is there for the teens; adults will not be there participating or "watching." This is important. If this works well, others, internationally, will become involved. TM is not just in Virginia, not just in the US, but exists around the world. We are working through Geoff, and the UK support group, to get teens from the UK involved. This is an important project and would like to get as many teens involved as possible. It will work, because it is important to all of them! Ron Hutton (VA), Stephen Miller (OH), Paula (WA), and Debbie (CA) have all been instrumental in this project.

Fundraising! My children are still working on a bowling fundraiser. The name of the fundraiser is Virginia Bowls Over TM. I will keep you posted on the details. The plan is to coordinate it state-wide so that all of the children and grandchildren of support group members can participate in the fundraiser in bowling alleys around the state on the same day. I cannot take credit for the name of the fundraiser. It goes to Greg Seiler who also came up with the fundraiser premise. It will be fun and will raise money. This I promise!

If you have any ideas for other fundraisers, please let me know!

Fundraising and Awareness

Help us find a cure for Transverse Myelitis

Let me introduce myself. My name is Cathy and I am the mother of three wonderful young children: Matthew, Kevin and Rachel. Our lives were changed forever on October 9, 1999 when Rachel (who was 6 1/2 months old at the time) woke up and was paralyzed from the neck down. She spent 18 days in the hospital where she was diagnosed with Transverse Myelitis. In Rachel's case, doctors feel the virus she had at the time caused her immune system to attack her spinal cord, causing her spinal cord injury. Thankfully, Rachel has regained the use of her arms, but she remains totally paralyzed from the waist down. The prognosis for Rachel is uncertain; as it is for everyone who contracts TM.

Rachel's birthday is March 24th. In honor of her birthday, her brothers, Matthew and Kevin, launched the inaugural Reading for Rachel Program in March 2000. Matthew and Kevin are starting a tradition in honor of their sister, and we are hoping that you will become a part of this tradition by participating in this wonderful learning experience. All funds received by The Transverse Myelitis Association for the Reading for Rachel Program will be used exclusively for research to better understand TM, to find treatments for the symptoms of TM, and to ultimately find a cure.

Matthew and Kevin are asking other students to help raise funds to support TM research by finding sponsors who will pledge and pay for each of the books they read during the month of March. Not only does this project provide research funds for The Transverse Myelitis Association, but it provides wonderful lessons about life and educational opportunities for all who participate.

The Reading for Rachel Program raised more than $9,000 in 2003 for TM research. We are very grateful for everyone who participated in our program and for making such a wonderful difference for so many deserving people. 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 our web site:

http://www.readingforrachel.org/

Reading for Rachel will take place in Worthington Estates Elementary School beginning on November 17th this year. Pauline Siegel is a second grade teacher in this school near Columbus, Ohio. Rachel and I help to kick off the program each year at Worthington Estates, and we will do so again this year. It is wonderful to meet all of the students and teachers. It is a great opportunity for us to help bring awareness about TM and to help these young people better understand something about paralysis.

My husband, Dan, and I are so proud of our sons for starting this wonderful annual project in honor of their sister, Rachel. It gives them a real sense of purpose as we patiently wait for Rachel's uncertain recovery. We are most appreciative of your support of the Reading for Rachel Program and The Transverse Myelitis Association. Your generosity and caring offers hope to Rachel and to the many other children and adults with TM.

Cathy Dorocak
Rachel's Mom
And National and International Chair of the Reading for Rachel Program
cathy@readingforrachel.org
(440) 572-5574

Kevin's Cause Fundraiser to Benefit TM Research
Jeanne Hamilton

Our son, Kevin, was afflicted with TM in May of 2000. A group of friends who are concerned about how TM continues to affect Kevin and our family helped us to put on the second Kevin's Cause Fundraiser to benefit TM research.

There were four elements to our fundraiser last September:

1. Raffle of a "Free" 2 year leased Subaru (I have a brother who works for Subaru).
2. Raffle of $1000.00 cash.
3. Donation to attend BBQ Event
4. Silent Auction tables

The Subaru lease we secured through my brother. (We chose a lease instead of a car, because we would have had to purchase a car at cost for something like $15,000.00. Also, there would have been tax consequences for the winner. The lease was totally free and a lot less risky although somewhat less desirable). The $1000.00 cash came from the proceeds of the raffle, and tickets were very easy to sell. We gave raffle tickets to sell to everyone who offered to sell them. Family, friends, and TMA members from the Illinois support group and the parents online chat group were very helpful selling raffle tickets.

For everything, we solicited friends and family: Does anyone have printing connections? We were able to print the 10,000 raffle tickets and 1000 invitations for a total cost of $50.00. Can anyone make a donation to the silent auction directly or know someone who could make a donation? Can you sell raffle tickets? While I primarily wrote the invitation, I have a friend who is a graphic designer who put it together for me.

As far as the mailing list, I started with my address book, then added my husband's business contacts, then asked friends and family to give additional addresses for their friends and acquaintances who would be willing to possibly come to the event or make a donation. I believe we sent out around 450 invitations.

Having the BBQ gives the fundraising a focal point. Both raffle winners were announced at the BBQ and people attending participated in the silent auction. For the BBQ, my friend opened up her very large house. Additionally, we used two outdoor tents. We provided the main course, a limited selection of bottled beer, wine, margaritas, and some violinists and a singer for entertainment. We also asked close friends and family to bring desserts and appetizers. Overall, the night was a lot of fun.

We have used this formula twice and it has been very successful, although a lot of work. Between our two parties, we have raised over $100,000.00 for TM research.

I would be happy to assist anyone else trying to raise money for the TMA. If you have any questions that I can answer, please call me at (847)670-9457 or e-mail me at jeannemarieh@msn.com.

TMA Cookbook
Alan Connor

We finally finished the cookbook, and it went to the publisher on April 12th, 2004. I want to thank everyone who contributed recipes, stories, words of wisdom, and art. The book has 146 recipes, along with TMA information, and cooking hints. The cost for the book is $10 plus $2 for shipping and handling in the continental US. We are still trying to determine the cost to ship outside the US, and all of this information will be posted on the website. We have ordered 200 books to start and we need to sell 82 to break even with the publisher. Everything we sell after the 82 books will be a contribution to the TMA, and we can order more as we go along. It is a very nice book. It is a great little gift. You can start your holiday shopping early, or buy it as a way to say 'thank you' to someone that has been especially sup-

portive of you or your loved one with TM. To purchase, make your check or money order payable to the TMA, and send to:

Alan Connor
117 Foxhound Drive
Glen Burnie, Maryland 21061

Allow 2-4 weeks for delivery.

http://www.myelitis.org/local/md/cookbook.htm

The Independent Riders of Ohio Poker Run for the TMA

The Independent Riders of Ohio held a poker run and an auction to raise money for The Transverse Myelitis Association. The fundraiser was held on September 6th, 2003 to honor Theresa "Giggles" Large, the Independent Riders Club Secretary. Giggles has TM and is a member of the TMA. Members of the Ohio TM Support Group of the TMA were also in attendance at the fundraiser. The poker run covered central Ohio and then an auction and dinner were held at J.R. Buzzard's Roadhouse. Live entertainment was provided by Cameltoe. A great time was had by all. It was a fitting honor for Giggles and the Independent Riders of Ohio raised over $1600 for the TMA. A special thank you to Diann "Wiggles" South for all of her hard work in putting on this event for the TMA and her wonderful friend, Theresa!

Sell on eBay and Support The Transverse Myelitis Association!

Looking for a fun and unique way to raise money for The Transverse Myelitis Association?

Now you can sell an item on eBay and donate from 10% to 100% of the final sale price to help support educational and research efforts for a spectrum of rare neuroimmunologic diseases of the central nervous system. Disorders in this spectrum include, Transverse Myelitis, Acute Disseminated Encephalomyelitis (ADEM), Optic Neuritis, and Neuromyelitis Optica (Devic's disease).

Clean out that closet or basement or attic. You can sell practically anything on eBay and donate a percentage of your net proceeds to The Transverse Myelitis Association.

How Does It Work?
MissionFish, a nonprofit organization specializing in online charitable auctions, developed Giving Works for eBay. Although items will be posted through MissionFish, they will appear with all other listings on eBay. Giving Works items are marked with a charity ribbon icon so buyers know they are supporting a good cause. Buyers tend to trust charity sellers more and these items often sell for more than non-charity listings. In addition, Giving Works items appear in three categories for the price of one: the category you select, the eBay Giving Works category and on the MissionFish nonprofit homepage.

To start:
1. Visit www.MissionFish.org
2. Get registered
3. Choose an item to sell
4. Write an item title and description
5. List your item through MissionFish and indicate that it will benefit The Transverse Myelitis Association.
6. Complete your transaction; MissionFish collects your donation and passes it on to us.

We appreciate your continued support!

Jim Lubin
Information Technology Director

Donations to The Transverse Myelitis Association using Paypal

It has been a fundamental policy of the TMA from its inception that we would not charge a fee for access to support and information. Consequently, the TMA does not have a membership fee. Regardless of whether you have one of the rare neuroimmunologic diseases, or you are a caregiver or family member, or you are a physician, scientist or medical professional, membership in the TMA is free.

Unfortunately, our services come at a cost. For those of you who have been involved in the TMA, you know that we have no overhead or administrative costs and you also know that we do not use our resources to raise money. The officers pay for most of their own supplies, internet access, and long distance phone bills. The officers and support group leaders are all volunteers; the TMA has no employees. And we all work out of our homes. The money we raise goes exclusively to providing services to our members, and most of our resources are used for postage and printing and to offer educational opportunities to our members.

Our operations depend entirely on donations from our members. If you are able to make a contribution to the TMA, we need for you to do so. At the present time, donations to the TMA are almost exclusively made from our membership in the United States. Having an international membership is very important to the TMA, and it is also very expensive. We need for our international members to assist us with donations when it is possible for you to do so.

International contributions to the TMA can be made through Paypal. Paypal is a service that allows you to securely send and receive money via bank or credit card to anyone worldwide. Ac-

counts are easy to setup and they are free. Please visit www.paypal.com for more details or to set up a free account. You can use your PayPal account to make donations in U.S. Dollars, Canadian Dollars, Euros, Pounds Sterling or Yen.

We are grateful for your willingness to support your TMA.

The Most Successful Method for Raising Money for Research

The most successful fundraising activities we engage in are those that focus on our friends and family members. I know, I'm sounding like a broken record. Try to get used to it, because I don't plan on quitting. If we want a cure for myelin and nerve damage, we need more research, and if we want research focused on the damage caused by the neuroimmunologic disorders, we need to raise the money. No one else is doing this fundraising and no one else is going to do it.

If we are going to raise this money, the vast majority of it is going to come from you and from your friends and family members. Why? Because they are the only people in the universe who know about these conditions and they are really the only people who care!

I am not comfortable asking my family for money. I am less comfortable asking my friends for money. I am no different than you. But I have learned to do it, because I have come to accept that this is the only way I can make the difference for Pauline and the so many others of you who I have come to love and care about so deeply.

I have an idea which I think might make this easier for you to accomplish. I have written a letter and have posted it on our web site. You can find it from the 'donations' link under the title ***Fundraising Letter for Christmas Cards***. I have included important information about TM and the neuroimmunologic disorders, about the TMA, and why it is important for the TMA to succeed in raising money for research. The letter is created in Word. Since most people have MSWord on their computers, I am encouraging you to personalize this letter. Please include information in this letter about how TM and the other neuroimmunologic conditions have impacted your lives, and why you need for this research to be done.

When you send your Christmas cards this year, and every year, please include a copy of this letter in your card. Just fold it and put it into the card; it won't cost you a penny more in postage, unless you are sending really fancy shmancy and heavy cards or your letter becomes a book. So, what's a little more postage? And, if you don't celebrate Christmas, you can include the letter with any regular correspondence you have with your family and friends. You can include it in all of the cards you send to people! *Happy Birthday, Uncle Harold; and oh, by the way ….* Or use the letter as a good excuse to communicate with your family and friends. Send all of them a letter to let them know how you are doing; and include this letter with your mailing.

Do it for yourselves and do it for the other children and adults in your community who need this research and the great hope that this research brings for all of us.

The Transverse Myelitis Association 2003 Statement of Financial Activities
(in US Dollars)
Paula Lazzeri

The following tables present The Transverse Myelitis Association Annual Financial Report for 2003. 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 costs of providing TMA member services during 2003. 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. The Donations made by Board of Directors line item presents the amount of funds spent by members of the Board of Directors that were not reimbursed by the TMA (General) Fund.

The Transverse Myelitis Association 2003 Treasurer's Annual Report

	TMA Funds	Total Donations and Expenses to Benefit TMA
INCOME		
2003 Strategic Planning Meeting Donations	26,093	26,093
2004 TM Symposium	50	50
amazon.com Commissions	58	58
CafePress Commissions	116	116
Donations made by Board of Directors	0	12,253
Endowment Donations	265	265
Endowment Interest	65	65
General Donations	71,526	71,526
iGive.com Commissions	124	124
Interest	1,563	1,563
Research Donations	64,855	64,855
Support Group Donations	604	604
Video Sales	300	300
TOTAL INCOME	**165,619**	**177,872**
EXPENSES		
Bank Fees (cashier checks, merchant svcs)	80	80
Board Meeting Expenses	0	4,795
Domain/Web-site/Webhosting	599	792
Internet Service Provider	0	1,606
Johns Hopkins Coordinator Research Position	25,000	25,000
Membership Fees	0	215
Mileage and Parking	0	192
Office Equipment	0	2,496
Office Supplies	0	764
Postage	14,984	15,647
Printing	9,243	9,293
Software	40	189
Secretary of State Registrations/Annual Report	230	230
Strategic Planning Meeting Expenses	14,289	14,289
Support Group Expenses	235	349
Telephone and Fax	0	1,018
Workshop Expenses	1,000	1,000
TOTAL EXPENSES	**65,700**	**77,953**
Net Income	**99,919**	**99,919**

Transverse Myelitis Association 2003 Statement of TMA Account Balances

Operating Fund	**115,987**
Research Fund	**128,989**
2004 TM Symposium	**50**
Endowment Fund	**10,007**
Endowment Interest	**465**
Support Group Fund	**369**

The Transverse Myelitis Association 2003 Donors

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 2003. The donations made by members of the Board of Directors include non-reimbursed expenses.

$5-25

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Edna Alvarez
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Willis and Dorothy Wurdeman
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Richard and Jane Zemba

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Milton and Carol Howe
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Delores Cain
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Paul and Marianne Marchionna
United Way of King County
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Joyce Mackiewicz
Robert and Dorothy Paulson
Gregory and Rose Marie Karoly
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Nils and Min Wickstrom
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Gerald and Hazel Zimbric

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David Argetsinger
Nevin and Donna Armstrong
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RJ Bartholomew
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Morgan and Pamela Hoge
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Indiana Lions Speech & Hearing
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Jay and Coille Putman
Nancy Reed
Donna Segal
Sunset Software c/o Richard Katz
Ray Ogle and Gail Whitelaw
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$1,500-1,800
Michael and Debbie Capen
Elaine Chapin
Independent Riders of Ohio
Darian and Amy Vietzke

$2,000-2,300
Dick and Deanne Gilmur

$3,000-4,000
Walter and Maureen Hallagan
Myk and Paula Lazzeri

$4,000-5,000
The Gardner Grout Foundation

$5,000-5,500
Claddagh Foundation, Inc.
Diane Lynn Family Foundation, Inc.
Sandy and Pauline Siegel

$8,500-9,000
Reading for Rachel

$25,000-27,000
TMA Strategic Planning Meeting fundraisers (Includes Chafetz, Lazzeri fundraisers, and quilt raffle)

$50,000+
Kevin's Cause Fundraiser

TMA Medical Advisory Board

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The Transverse Myelitis Association serves on the Christopher Reeve Paralysis Foundation Paralysis Task Force