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From the Editor
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The Doug and Kazu Reunions at the Ross Correctional Institution Monday, June 23, 2008 and Monday, July 20, 2009

Over the past two years, Kazu, Pauline and I have shared in a remarkable and wonderful experience. We have had the honor and privilege of being able to visit the gentlemen who perform the amazing work of raising Canine Companions for Independence (CCI) puppies at the Ross Correctional Institution.

Kazu had been placed with Pauline in August 2007. Kazu is a CCI Service Dog. Kazu was born in California; he is one of Meeko's puppies. Meeko is cared for by a wonderful family in California. The CCI breeding program is located near their headquarters in Santa Rosa, California. This family also cares for Kashi who happens to be Kazu's sister. These kinship relations are pertinent to our story. The puppies from California are delivered to the five CCI regional centers around the country. When Kazu was two months old, he and Kashi hopped a plane to Delaware, Ohio. The regional centers have a legion of volunteer puppy raisers. After the puppies arrive at the regional centers, they are distributed to the puppy raisers.

Denice is an employee at the Ross Correctional Institution (RCI). She is just an incredible human being! She initiated and manages the puppy raising program at RCI for inmates who are in an honors unit. They receive these puppies when they are two months old, and they will have them until they are just over a year old. They teach the puppies about 30 commands, they socialize the puppies and they care for them during this critically important, formative year. There are four or five CCI puppies being raised by four or five handlers at a time at the prison. The puppies live with the inmates in their cells and are with them all day long, every day of the week. Some of the inmates participate in the program as alternates. These men care for the puppies when their handler has some obligation that requires that he be away from his puppy. The puppies are with a handler or an alternate at all times. Occasionally, one of the employees of the prison will take the dogs out for a trip to the store or to a mall or to their homes. The puppies need to be socialized, they need to have experience being out in public and exposed to the different sights, sounds and smells outside of the prison, and they need to develop comfort with being in a car. There is a tremendous amount of work that goes into training and socializing the puppies during the first year of their lives. All of this work and love and care serves as the foundation for the advanced training that occurs when the puppies are turned back into the regional centers when they are from a year old to about 14 months.

Kazu went through advanced training at the regional center in Delaware, Ohio. The dogs learn their advanced commands during this training, which includes turning on and off light switches, pushing and opening an automatic accessible door, tugging open doors and closing them, picking objects up off of the ground, carrying things and many more amazing commands. The dogs know more than 50 different commands by the time they complete advanced training. And the advanced training serves as the basis for additional commands that these service dogs can be taught. There is a CCI graduate liaison who is available to work with the graduates and service dogs for this training and to help with the dogs in a multitude of other ways, including any health or behavioral issues. Since she has had Kazu, Pauline has taught him to bring objects to our recycling bin for her, to empty the clothes dryer and to drag a wagon of clothes into the living room where she can do the folding. She's also taught Kazu "go to Sandy" and "go to Pauline" so that Kazu can find either one of us in the house if we are needed.

In addition to all of the commands, there are very high expectations set for the dogs' behavior. The dogs must exhibit acceptable behavior to be able to receive public access licensing and they must be able to remain focused on their companion while they are working. When Kazu is given a down command, he is expected to remain in this down position for up to four hours without having to give him any other command. And he knows this, because if he looks like he is going to get up, all you have to say is no and he remains down. Only 40% of the dogs that come into the CCI program are able to graduate to become service dogs. The foundation for these behavioral expectations is set while they are with their puppy raisers.

Kazu was raised by Doug. Pauline and I met Denice and Doug's parents at the CCI graduation ceremony. This was a very emotional experience for all of us. Just before Christmas, I wrote a long letter to Doug about Kazu and Pauline. The letter chronicled the advanced training experience and the relationship between Kazu and Pauline after bringing him home and into her life. Doug wrote back and told us all about his incredible year with Kazu. That exchange of correspondence became my editor's column in the TMA Journal Vol. III. Doug and I have been writing to each other regularly and I communicate with Denice by phone and emails. Through our conversations, we decided to visit RCI so that we could meet Doug, see Denice, meet the other people raising CCI puppies, and create an opportunity for a Doug and Kazu reunion.

After reading about Doug's emotional good-bye to Kazu, I wanted for Doug to feel like he didn't need to say goodbye to Kazu at all. My heart moved me to make a personal commitment to have Doug and Kazu remain a part of each other's lives. When I suggested to Pauline that this is what I had in mind, she looked at me like I was asking her to make a visit to a men's maximum security prison. I got the same look when I sent a ballot out with our wedding invitations asking our wedding guests to vote on what they thought we should adopt as our last name. As I thought it was unfair for Pauline to take my last name, the ballot included a variety of symbols such as the one Prince adopted for his name. Fact is, I get this look from Pauline fairly often. Life is short … can't be deterred by ***the look***.

With a lot of very hard work on Denice's part, the arrangements were made for Pauline, Kazu and I to make this visit to RCI on Monday, June 23, 2008.

Denice sets the standards for the men in the program that match the very high expectations that are reflected in the CCI program. She wants men involved in the program who understand the responsibility required in working with these dogs, and who accept that having the puppies is something more than having companionship or having a pet. While it is not possible for every puppy to graduate to become a service dog, Denice expects that all of the puppies coming from RCI are going to have a good opportunity to graduate. There is no doubt that the men's lives are significantly enhanced by having these puppies; the love and companionship between the men and the dogs is just so intense and powerful. The men benefit greatly in so many different ways from working with these dogs, and Denice ensures that the work is channeled into creating a good chance that the puppies from her program will graduate to become service dogs.

Before Kazu, Pauline and I made the trip to RCI in the summer of 2008; no one had ever made the trip to RCI to visit with the inmates and to bring one of the service dogs back to see their puppy raiser. When a student graduates and receives a dog from CCI, they meet their puppy raiser at the graduation ceremony. The students and the puppy raisers have breakfast together before the ceremony. Also, before breakfast, the puppy raiser is given about a half hour to be alone with the dog. There is a framed photograph of the student and service dog that is given to the puppy raiser at breakfast. During the graduation, the puppy raiser ceremoniously hands the leash of the service dog over to the student. It is hard to imagine a more sensitive and caring way for CCI to treat this situation than the way they do. If you are a dog person, I have no doubt that you can empathetically feel the emotions of this situation and of this moment. CCI communicates no expectations whatsoever about any contact or relationship between the student, their service dog and the puppy raiser. The puppy raiser is instructed not to make contact with the student. The choice of contact is entirely up to the student and this decision is very personal and the circumstances are different for everyone. Some people have no contact with the puppy raiser, some people have regular contact, and some people are in contact on only very special occasions. I am making no judgments about how people make this decision. I am making no judgment whatsoever about the comfort level a person might have about making contact with an inmate in a prison.

Before Pauline and Kazu came to prison, the inmates in the puppy raising program had never seen a dog that had completed advanced training. They never had a chance to witness the more than 50 commands that are taught during this training. They never had the chance to watch how the dogs work with a person who has a disability. For the years they had been involved in the program, the men participating in the puppy raising program never had the opportunity to see the very special bond that develops between the service dog and the person with whom they are working.

Denice arranged for us to have a three-hour visit at the prison from noon until 3:00. RCI is located near Chillicothe which is about an hour drive from our house in north Columbus. Pauline spent the morning brushing Kazu and getting him ready for his big day. We had no idea how much walking we would need to do at the prison, so we brought both Pauline's wheelchair and her canes. I got the car loaded, and we headed to Chillicothe. As we got close to the prison, Denice called and told us that she would head to the main entrance so that she would be able to help us through the security process. When we arrived at the prison, I unloaded the car, set up the wheel-

chair, and Pauline took Kazu to hurry. The moment Kazu got out of the car, he knew where he was. He put his nose up into the air and sniffed around and then got a very excited and stressed look on his face. Kazu remained entirely unrelaxed for the next three and a half hours. He was so excited, he could barely contain himself.

We went in the front door and three security guards had us sign in and turn in our driver's licenses. As we were completing the sign-in process, Denice arrived. Kazu saw her and without a "release" he began spinning in circles. He looked like he was about to jump on her and then in a split second, a minor bit of sanity prevailed. But as Denice was petting him, Kazu continued to spin and vibrate. Pauline and I had never seen Kazu this excited. We got Kazu settled down some and headed out of the security area. Denice took us outside into the yard area. The entire prison complex was in front of us. Stone buildings for as far as the eye could see.

A short distance away, we arrived at the visitor's building and went inside. We looked down the short hallway to the large visiting room, and there was Doug. Pauline told him that it was okay for him to call Kazu, which he did. He called Kazu's name twice before Kazu heard him. Kazu had to have been very confused. He knew this room very well. This is the room where Doug worked with him, this was their training room. It was a large room. There were benches to practice

"up" and "jump." It was no wonder that Kazu didn't hear Doug at first. And he was still reeling from his excitement of being with Denice. Finally, he picked up his head, looked down the hallway and saw Doug. He was off. He charged Doug and began spinning wildly and running back and forth in front of Doug.

Pauline said that if the room hadn't been crowded with chairs and tables, Kazu definitely looked like he was going to take off running (like he does at home when he is very excited). Doug was obviously thrilled that he was remembered and that Kazu was pretty out of control with excitement. He left Kazu to do his spinning and running, and then he got down to showing all of the love he has had for Kazu for all of this time and had no way to express. Kazu just loved every single minute of the petting and loving that he was getting from Doug.

The love between this man and this dog was palpable. Denice and Pauline and I just left the two of them alone for a while to be with and to enjoy each other. It was totally awesome!

Finally, Kazu settled down and

Pauline and I introduced ourselves to Doug. We spent some time saying to each other many of the things that we had been communicating to each other about in letters. Pauline thanked Doug for all of his love, dedication and care for Kazu. And Doug thanked us for writing to him, for staying in touch with him and for coming to RCI to visit him. We talked a lot about our experiences with Kazu, the team training, Kazu's time with Doug at RCI and then Pauline's time with Kazu in school and at home. It was a wonderful visit with Doug.

I brought copies of the Journal for Doug, Denice and for Doug's mom. Denice had arranged for us to have about a half hour to visit with Doug before anyone else arrived at the visiting area. The rest of our time was going to be shared with the other handlers, their dogs, and nurses from the prison who help with the puppy raising program.

Before the other puppy raisers arrived, Denice suggested that we take Kazu out into the yard to "hurry" and she also took the opportunity while we were outside to show us the rest of the prison. She pointed out where the cells were located and also the unit where all of the puppy raisers stayed. Denice explained that all of the puppy handlers stay together in cells in the same unit, because she wants them all in the same area where she works. It makes it much easier for her to coordinate their activities. The guys have also bonded with each other and they work together in raising the puppies and teaching them commands. They definitely learn from each other, and as one develops a good technique for training, they all use it. They greatly benefit from their close relationship, working together and living in the same area.

Kazu pooped, and Doug had a bag handy and took care of it. Like old times! We all shared a good laugh about it. As Kazu was finishing his business, a group of guys came walking through the yard with their dogs. The other puppy raisers approached us outside. We introduced ourselves and went into the visiting area. We began talking to each other and the dogs began the process of checking each other out.

Doug and another handler received puppies just the previous week from CCI. Doug had turned in Fancy about two months ago, so he had been without a dog for two months. He was very excited to have Dali, a male who was just over two months old. He was already working on the hurry command (potty training) with Dali. Denice had the guys put a crate up so that the puppies would have a place to stay while there was so much commotion going on in the room. But for most of the time, the puppies were in the visiting area romping around and visiting with everyone. Kazu had not the slightest bit of interest in the puppies. Doug told us that Kazu never showed any interest in puppies.

The inmates all had dogs of different ages and at different stages in the training process. Besides the two puppies, there was Dune and Davida who were both about a year old. Both Dune and Davida were exceptionally well trained. They spent most of the time in the visiting area visiting with everyone and also checking out Kazu. Kazu was so much more interested in Doug and the other people who were paying attention to him; the other dogs weren't getting much notice.

Davida is Kazu's niece. She is the daughter of Kashi who is Kazu's sister. Davida is being raised by Bud. Uncle Kazu pretty much ignored Davida until the very end of our visit. Just before we left, Davida was sniffing Kazu and trying to get him engaged in some kind of play and Kazu went into a play bow. We thought he might take off running, but then he went immediately unenthusiastic about the play idea. By the end of our visit, Kazu was totally exhausted from all of the excitement and all of the attention.

Denice had purchased a beautiful cake for us to celebrate the occasion. It was, of course, a chocolate cake with purple flowers and decoration! It is, after all, all about Pauline. Denice is really a sweetheart!

As we were sitting in the room enjoying the cake, Doug told Pauline that he had a gift for her. He went over to a bag and took out an absolutely beautiful purple quilt that he had made for Pauline. He said that Denice got the pattern for him. Denice told me that he had been working on the quilt for the past two months from the time that he turned in Fancy until he got Dali. Pauline was just totally overwhelmed. And Doug was really moved by Pauline's very emotional reaction, and so pleased. Some of the men are also involved in a quilting program. The quilts they make are donated to nursing homes.

Pauline and I were so impressed with these men. They were kind, sweet, sincere, genuine, and respectful. They had such nice things to say to us. All of them had read the letter that I wrote to Doug and they each took the time to thank me for writing and they let me know how much they appreciated what I had to share.

We next turned our attention to talking about the training program that the handlers are using to work with their

puppies. These guys do an exceptional job of training the dogs. Nate took Dune through all of his commands for us.

As we are very aware of the appropriate way for the commands to be done, through our experience with team training and Kazu, we knew how well Dune was trained. He went through Nate's commands perfectly. It was so impressive. He did each of the commands in the correct way, without any hesitation and exactly the way it was supposed to be done. Pauline told Nate that he did a great job and never saw anything better than what Nate just demonstrated for her — even during team training. Nate was very proud.

Dune was so focused on Nate while they were going through their routine. All of the commands are done with voice. The handlers do not use their hands at all when giving commands. As these dogs are going to be trained as service dogs for people with disabilities, they have to learn to respond to commands by a person who may not be able to use any part of their bodies to communicate. All of the commands begin by saying the dog's name, and the response from the dogs is to lock their eyes onto the handler. When this focus is established, the command is given.

The RCI nurses came to the visitor's center to see Kazu and to spend some time with the new puppies. Of course, Kazu enjoyed another round of attention from the nurses. The nurses are a tremendous help for Denice with the program and provide medical assistance to the puppies that saves some trips back to a veterinarian.

Pauline then described her experiences to the nurses and puppy raisers. She talked about her team training experience, she explained the different things Kazu did for her, and she talked a lot about her experiences with Kazu at her school. The guys asked a lot of questions about her experiences. It was a great exchange.

When Pauline was finished with the discussion and answering questions, she showed the guys some of the things that Kazu learned in advanced training after he left RCI. She had Kazu get a credit card from the floor, she had Kazu do light and switch, she had Kazu open and close the door (tug and push) and she had Kazu tug a bag across the floor for her. She also had Kazu pick up her canes for her. The guys were all very impressed. And Doug was so proud of Kazu!

Kazu had been on his feet for almost the entire time we were in the room. He was exhausted, but too excited to know it. At around 3:00, Denice announced that we were going to have to end our session. So, we took a couple of group photos to remember the occasion, and we said our good-byes to everyone. It was very emotional. I told Doug that we weren't going to say good-bye and we didn't want for him to say good-bye to Kazu either, because we were all going to see each other again.

Denice walked us out to the car and we got everything loaded in just before a rain storm began. The guys returned to their unit. We thanked Denice for an incredible day and a wonderful experience and promised to remain in touch with her.

When Pauline and I first heard that Kazu had been raised in a prison, we both had the same reaction; we were concerned that Kazu didn't have the same puppy experience that he might have had in a family. Our concern was that he didn't have as good an experience; that his life in prison might not have been as comfortable or that he might not have received as much attention or love. As we drove away from the prison, Pauline turned to me and said, "Wow, Kazu had the most incredible and positive life with Doug and with these men. He had such a great life in prison." And I totally agreed with Pauline. He was the focus of Doug's existence for more than a year. He lived with Doug in his cell. He got attention from Doug almost all day long, every single day. He was the center of attention by everyone, everywhere he went in the prison. Everyone knew him and loved him. From the other inmates, to the guards, to the nurses, to the staff, Kazu was the focus of so much care and love. We saw it for ourselves during our visit; everyone loved Kazu! And Doug loved him and took such wonderful care of him. And he trained Kazu as well as a person could train a dog. It is hard to imagine that Kazu would have or could have received this kind of attention in a family. There is no family that I am aware of that has the time or the focus to give to a puppy what these men are able to provide to these dogs in prison. We came home and wrote thank you cards for Doug and Denice, and two days later, we re-

ceived a letter from Doug.

Pauline's and Kazu's visit to RCI was so important for Denice and for the men in this program. By being able to observe the product of their hard work, their dedication, their focus and the love and care they provide to the dogs, they were significantly affirmed in the purpose of their work. Each of these men also observed, first-hand, that the work they were doing was transforming a person's life in the most meaningful and positive and profound ways. Before we left, one of the men approached me and thanked us for coming to visit all of them. He told me that our visit and our communications have totally re-energized him in his work with the dogs.

Denice arranged another visit for Pauline, Kazu and I with Doug and the other puppy raisers in July 2009. The visits always have to be arranged on a Monday because we meet in the prisoner visiting area. There are no visitations on Mondays, so we are able to have this room to ourselves. Denice made contact with another person who had received a service dog who had been trained by Jack at RCI and invited her for a visit at the same time that Pauline and Kazu were coming. I have no doubt that by being able to explain that someone else would be there with them, and that this was a return visit, made this invitation easier for her to consider. We were thrilled that Suzanne accepted the invitation and came with her service dog and with the help of her mother. Suzanne was going to have the opportunity to meet Jack, her dog's puppy raiser. Jack is a veteran in the RCI puppy raising program and has helped Denice by sharing his experience with the other men.

We arrived at the prison just before noon and this time, Denice came outside and met us in front of the prison gate. Again, Kazu recognized Denice immediately and was very excited to see her.

Kazu is just amazingly disciplined. At the time we visited RCI this summer, Pauline had been with Kazu for about two years. Their bond is so strong. When Kazu is with Pauline he is so focused on her; his eyes are often just locked onto her. So, when Kazu is overwhelmed by some outside stimulus, he sometimes becomes confused. Confusion would be a mild characterization of what I observed in Kazu while he was trying to remain composed and focused on Pauline, who had not given him the 'release' command, and thus release him from his work mode, and his being in Denice's presence and being way too excited to remain composed. Finally, Pauline gave Kazu the release command and he spun around in front of Denice while she gave him a full body rub.

We went through the security procedure and once signed in, Denice took us to the visitor's center. We were in the center for only a few minutes when Doug came through the door. Pauline released Kazu and he bolted toward Doug. If anyone has any doubt about what a dog remembers, let me assure you that they have excellent memories. Kazu loves Doug and you can see his connection to this man every time he is in his presence. Doug turned Kazu into CCI for advanced training when Kazu was about 14 months old. He saw Kazu for the first time when Pauline brought him back over a year later. Kazu's reaction to seeing Doug in

July of 2008 and then again this past summer leave no doubt that he remains totally attached to this man. I have no doubt that he has memories of Doug and that they will forever be a part of each other's lives.

After Doug and Kazu had their time together, Doug's puppy, Dali, was brought into the room. Doug had wanted a short visit with Kazu before the two dogs had to share Doug's attention. The little two month old puppy in Doug's arms in the group photograph from 2008 was now larger than Kazu. Dali was scheduled to be turned in to CCI for advanced training in August, so Doug only had another month of time with Dali.

As had been the case with Kazu, Doug was entirely devoted to his training of Dali. He was totally focused on Doug and followed every command when it was given by Doug. There is no doubt that Dali will also become a wonderful service dog after completing advanced training. We talked about Doug's feel-

ings about having to turn in Dali in a short time. Doug explained that we'd definitely helped him with these goodbyes. He said that after Kazu, they remain difficult, but they are not as painful. Because he has been able to stay in touch with us and with Kazu and has a better idea about what these dogs' lives are like with their companions and what the dogs are doing for their companions, it is easier for him to handle the emotions of the separation.

We had a short visit with Doug while Denice went to the security gate to welcome the other family. Suzanne and her mom entered the room with their service dog as the other puppy raisers came into the room. It was a wonderful reunion for her dog and Jack. And there was yet another reunion taking place this day. Dune had gone to advanced training at CCI but did not graduate because of a physical problem. When Dune was released from the program, one of the employees at RCI adopted Dune. Denice asked the employee to bring Dune into work and to bring him to the visitor's center. Nate had the opportunity to spend time with Dune after not having seen him for a year. It was a fantastic reunion for Nate and Dune, as well. As I described from our first visit to RCI, Nate had done an exceptional job

of training Dune and preparing him for advanced training. There was no doubt in either Pauline's or my mind that Dune would have graduated to become a service dog had he not had the physical issue. Nate had worked so hard and had developed such high expectations for Dune, that his being released from the program caused him more disappointment than he could handle. When Nate explained to me that he was no longer in the RCI puppy raising program, we had a long talk about his experience. I told Nate that he really had a gift and that we saw this in his relationship with Dune the previous year and in the discipline and devotion that they both had exhibited when he took Dune through all of his commands. I also expressed to Nate that I made no judgment in regard to his decision to leave the program. I acknowledged all of the emotional issues surrounding being in the puppy program and the separation from the dogs after a year. I also assured Nate that he did all that he could to set the stage for Dune to succeed and that he had no more control over the physical issues that Dune developed any more than I had control over keeping Pauline from getting transverse myelitis. Things just happen that are beyond our control or understanding and we have no choice but to accept these events and try to make the best out of them. I only asked Nate to not totally close the door on his coming back some day. I reminded him about how exceptional his talents were in regards to caring for and training Dune and that his talents could benefit someone like Pauline and Suzanne in the ways he was now able to see for himself.

I received a letter from Nate about a week after our visit to RCI. He thanked me for talking to him about his experiences with Dune and in helping him to find some closure on this difficult and emotional situation for him. I remain hopeful that Nate will find his way back into the program. I know that he greatly benefitted from being in the program, and he does have a gift!

Denice brought in pizzas for everyone, and we shared lunch with Denice, Suzanne and her mother, Nate and the six men from the RCI puppy raising program. Three of the men are raising puppies and three of the men serve as alternates. The three puppies being raised were going to be turned in for advanced training the following month.

As this was our second visit with the men and we have been communicating regularly with Doug by letters, this visit truly did feel like a reunion. There was a lot of conversation about the dogs. Everyone wanted to hear about Pauline's and Suzanne's experi-

ences with their service dogs and there was a lot of conversation about the puppies and the RCI puppy raising program. But we also had a lot of time to talk to Doug about other than dog matters.

Doug had started college and we talked for a long time about his courses and his educational aspirations. We also talked about his relationships with his children and his excitement about his daughter who had recently made a visit to see him. Pauline and I were just thrilled to see Doug's life moving forward in some really positive ways.

And during our conversation, as Doug would periodically hug and stroke Kazu, the thought would enter my mind that Doug's positive directions have no doubt been influenced by his time with Kazu and with Dali and with the incredible opportunities that have been made available to him by Denice.

Dali is exceptionally well trained. Doug has done a fantastic job with Dali. He is completely fixed on Doug and waiting for his next command. His eyes almost never leave him.

Denice bought presents for the dogs; each of them received a hard rubber ball. This ball has become one of Kazu's favorite toys. He loves chasing it around the house, but he also likes to hold it in his mouth and just prance around the house holding it in his mouth, periodically bouncing it off of the floor and catching it back in his mouth. There was an enclosed yard area adjacent to the visitor's room and the dogs went out to run and hurry after we finished lunch. Everyone went outside to visit and to watch the dogs play. And, of course, the dogs insisted on having a group photo. The handsome guy in the middle is Kazu.

It was a wonderful visit with Denice, Suzanne and her mom, and the incredible men of the RCI puppy raising program. We really have come to appreciate and to value our time with Doug, Bud, Jack, Nate and the other men. And we continue to correspond regularly with Doug and Denice during the year by mail with Doug and by emails and phone with Denice.

There was a very poignant moment as we all exited the visitor's center. There are a series of gates that we have to pass through to leave the prison. As Suzanne and her mom and Pauline and I stood at the first gate with Denice, I turned around to see the men walking as a group with their dogs toward their unit inside the prison. As we waved good-bye to each other, I was overwhelmed by the sense of freedom I had in being able to leave this place, and what these men experience every day by virtue of remaining inside.

Kazu and Pauline have been together for about two and half years. They have developed a very comfortable routine and Kazu, has, of course, become a member of our family. In fact, it feels as though we've had Kazu in our lives for decades. Kazu goes to school with Pauline every day and he remains by her side at home during his every waking hour. Even when he is sleeping, he is often nuzzled up against Pauline while she strokes his soft floppy ears. He is as attentive to Pauline's needs as the day he completed his advanced training.

Pauline works with Kazu on his commands every day. She doesn't use all of Kazu's commands regularly so she has to periodically review through all of his commands so that he remembers how to do them. We had a very interesting experience with Kazu that reminded us as to why this practice with the commands is so important. Pauline almost never uses the speak command. Pauline doesn't ever want for Kazu to bark in her school or in a public place, so she doesn't often give Kazu the speak command. For the entire first year she was with Kazu, he never barked and she never gave him the command. One evening, I suggested to Pauline that she might want to give Kazu the command so that he remembers it. We hadn't heard Kazu bark one time since he had graduated from advanced training. She then gave Kazu the speak command, and he looked at her, but didn't do anything. She gave him the command again, and he made some sound from the back of his throat that was really pathetic and not a bark. Needless to say, Pauline and I were both totally freaked out and entirely entertained. Kazu had forgotten how to bark! Pauline gave the command a few more times and while Kazu would open his mouth and make some strange faces, nothing was coming out. Pauline even gave the speak command and would bark herself so that Kazu could hear the sounds coming from her mouth. We finally gave up, because we didn't want to frustrate Kazu. The next day, Pauline gave him the speak command again. Kazu produced a weak bark … but it was a bark. She repeated the command a few more times and Kazu really barked loudly. Kazu has a really manly bark. Doug had explained to Pauline that when he gave Kazu the speak command, he would encourage Kazu and he would continue barking until Doug would tell him to stop. So, Pauline tried this approach and, sure enough, Kazu barked and barked until Pauline told him to stop. Practice. The commands have to be practiced; all of them.

Kazu has a great life. Kazu has a very exciting life and is very well traveled.

In addition to making the trip to visit all of his wonderful friends at RCI this summer, Kazu flew to Washington DC for a meeting of the Paralysis Task Force of the Christopher and Dana Reeve Foundation. He had a great visit with Shelley and Cody Unser at the meeting and even found some time to see all of the monuments and buildings on the mall during his long morning and evening walks.

He next flew to Dallas to visit the new TM and NMO Center at the University of Texas Southwestern that was established by Dr. Ben Greenberg. He thought the Dallas Aquarium was really beautiful and interesting. He spent an evening at Billy Bob's Bar and Rodeo. While he found them to be really big and scary, Kazu was totally appalled at the treatment of the bulls. But the highlight of the Dallas visit was his foray into Dr. Greenberg's swimming pool. That's Kazu in the pool with Paula Lazzeri, her son, Jesse and Pauline. And posed directly in front of Kazu's face is Dr. Douglas Kerr. Dr. Kerr most definitely loves Kazu; everyone loves Kazu.

Kazu then drove down to North Carolina for his first beach vacation. The

big waves were a bit intimidating until he got the hang of running in and out of the surf. Kazu is a mix of lab and golden retriever. We know that he's supposed to love water. Someone is going to have to explain that to Kazu sometime.

Our beach vacation was followed by a week at the TMA family camp at Victory Junction. Kazu had lots of bonding time with Dr. Kerr and he had a wonderful time dancing after every breakfast and dinner at camp. Kazu always loves his time at camp. Victory Junction just totally rocks!

Kazu was exhausted by the end of the summer and was looking forward to beginning the school routine with Pauline. We just love our routine. Kazu and Pauline will have their public access testing in November. I have no doubt that they will pass with flying colors. Kazu does what he's told when Pauline gives him his commands. Kazu and I are equally responsive to all of Pauline's voice commands.

Kazu is just amazing. Kazu's Mom, Meeko, and Dad, Baumann, have created some really wonderful dogs. A large number of Kazu's kin have gone on to become service dogs. He comes from an incredible line of dogs. The CCI breeding program is really exceptional. The breeder caretakers are an amazing and devoted group; these dogs become a part of their families. The advanced trainers are incredibly dedicated and disciplined and focused on their mission, and carry it out with great sensitivity, care and professionalism. And the puppy raisers … they are such a wonderful and generous group of people. They take a two month old puppy and do all of the hard work of leash training, and develop the toileting issues and work through the behavioral issues and teach the puppies about 30 different commands. The puppy raisers create the foundation for the more complex regimen that will come with advanced training. This takes just an amazingly selfless act of kindness. What the puppy raisers have are the wonderful memories of their puppies and the knowledge that they have transformed someone's life in the most positive ways.

What Denice does for these guys is really beyond words to describe. During our visit, one of the guys said to me, "I never thought about helping any one in my entire life. It just never occurred to me to want to help someone else. And then I got involved in this program, and it has changed the way I think. I love that what I'm doing is helping someone who needs this help." Denice has told me on more than one occasion that this program has definitely changed these guys. I can't imagine how it wouldn't. Pauline and I got the softest look at prison from the perspective we had on our visits. It is a very hard place. You can see it on these guys' faces. Prison is a very hard place. The CCI puppy raising program gives them a meaningful and important way to spend their time. Without this program, they would be just doing time. I can't even imagine what is like … but I often think about that moment in time as the men were heading in and we were heading out.

Doug and the other men are so focused on their puppies and what they are trying to accomplish. Their lives are so much better for this program and there is no doubt that the contribution they are making is exceptional. They also got to see that when they met Pauline and Suzanne. Pauline and Suzanne put a face and a real life in front of them to connect their hard work and their dedication and their love and care with their ultimate goals. They work so hard to get a dog graduated. They love their puppies; they really love their puppies. These are good guys. I am so glad that they are involved in the puppy raising program. They are truly

blessed to have someone like Denice in their lives. It was an honor for Pauline and me to meet Doug and the other guys we've met on our visits to RCI. Pauline, Kazu and I are looking forward to our next reunion with Doug, Denice and the other men in the puppy raising program in the summer of 2010!

The Transverse Myelitis Association is proud to be a source of information about Transverse Myelitis and the other neuroimmunologic disorders. Our comments are based on professional advice, published experience and expert opinion, but do not represent therapeutic recommendations or prescriptions. For specific information and advice, consult a qualified physician. The Transverse Myelitis Association does not endorse medications, treatments, products, services or manufacturers. Such names appear in this publication solely because they are considered valuable information. The Transverse Myelitis Association assumes no liability whatsoever for the contents or use of any medications, treatments, products or services mentioned.

Neurorestorative Principles of Rehabilitation

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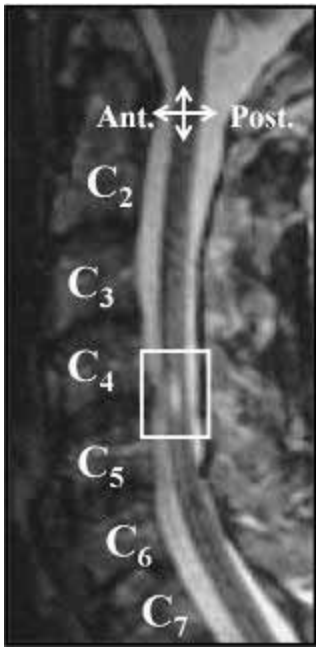
Today's field of neuro-rehabilitation is transforming from a focus on social rehabilitation to life-long restoration of function through regeneration. Many readers likely remember seeing Christopher Reeve's video during Super Bowl XXXIV in 2000. It showed the actor, Christopher Reeve, standing up and walking; intentionally looking real. Despite the uproar in the field of rehabilitation this 30-second video generated, it served its purpose; people started believing that recovery from spinal cord injury is achievable. We feel that there is hope today for recovery for almost everyone with a neurological injury, no matter how long ago the injury occurred.

Traditional rehabilitation is almost exclusively associated with the acute phase of injury. There is very limited rehabilitation available for people living with chronic spinal cord injury. We believe that we now have the tools to optimize recovery of function, through a number of approaches that we will present below. Spinal cord injury is our main focus, however we want to emphasize that these approaches, including regeneration and restoration of function, are going to be applicable to all disorders of the nervous system. It doesn't matter whether it is Alzheimer's disease, Parkinson's disease, stroke, traumatic brain injury, spinal cord injury, or transverse myelitis. Although these conditions have different mechanisms of injury, the end result is similar. Christopher Reeve demonstrated that recovery of function is possible in the worst case scenario, long after an injury.[1] In 2002 we were unable to determine the mechanism that accounts for why this recovery occurred. Here we will discuss today's approaches to regeneration.

There are common goals and mechanisms for both regeneration and activity-based restoration therapy (ABRT). The International Center for Spinal Cord Injury (ICSCI) at Johns Hopkins and The Kennedy Krieger Institute in Baltimore, MD are centers that are dedicated to people living with paralysis. We have a sub-focus on pediatric spinal cord injury. We take care of young children from onset of injury through lifelong care. We design an individualized, lifelong, in-home restoration therapy rehabilitation program. The only realistic way for a person to maintain a therapy program for a long period of time is by doing it at home. There are simply not enough rehabilitation centers in the country, and the information takes years to disseminate throughout the rehabilitation community. The vision of our center is to provide meaningful recovery and life improvement for every person with paralysis.

What is our mission? We are translating today's science to near-term therapeutic applications. We are very interested in what can be done that immediately and positively affects our patients. The ICSCI is developing and applying advanced rehabilitation restoration strategies for optimizing spontaneous recovery in those living with paralysis.

Image 1 illustrates the problem. This is a T2 MRI of someone who had a cervical spinal cord injury from trauma over 20 years ago. Spinal cord injury from transverse myelitis can look identical. The end result is the same



whether you have TM or a trauma to the spinal cord. The cord swells which is followed by a secondary infarct. The spinal cord tissue usually dies from the inside out, and one is left with a donut-like rim of tissue. What is important is the outer rim. That is the part of the spinal cord that carries the fibers to and from all parts of the body. Except for certain areas in the neck, one can lose the entire middle piece of the spinal cord and can have near normal function. That is because those nerves that leave the cord at one level originate from at least three spinal levels. This person, whose MRI is depicted, although completely paralyzed for years, now can do triathlons. This proves that we do not need to cure or repair the entire nervous system. We do not even need to come close. Similar to the data demonstrated in animals, only 5-10% of connections that a person without spinal cord injury has are probably enough for people to run and walk.[2]

Up until a few years ago, we felt that a severe complete or ASIA A spinal cord injury typically had a poor prognosis. This statement was correct in its day. In today's world, this is no longer true. What is different today? We have advanced imaging techniques. For example, diffusion tensor imaging (DTI) can actually visualize the tracts in the spinal cord. This enables us to identify and obtain an index of axons that cross the spinal cord lesion. With the individual in the case above, about 20% of the neural connections across the lesion are functioning. This is a person who can run and walk. The animal models are not as far off as some people have believed, as this phenomenon is exactly what the data from these studies suggested.

A primary goal of restorative therapy used to be to try to fill the cavity in the spinal cord that forms following a spinal cord injury. We now understand that there is plenty of tissue remaining after injury and we do not really need to bridge this gap. A major problem that does need to be addressed, however, is the surrounding scar. As a scar, it expresses chemicals that prevent axons from growing into it. The broken neural connections are constantly trying to regrow. In the scar tissue, there are incurring signals that communicate, "Stop, turn around, and go back the other way." It is as if one had rearranged the road signs. Scientists are using enzymes to break up these signals. They are using multiple molecular approaches to overcome them or to inhibit their development. Another approach being used is the delivery of growth factors to that area that will create favorable conditions for neural cell growth.

We believe that even the scar is not as critical a barrier as once suspected. There are ways to circumvent this problem. Usually, there are connections that exist across the lesion that are not functioning properly, because they are missing their appropriate insulation, myelin, resulting in short circuiting. This is what usually happens in transverse myelitis (TM) or multiple sclerosis (MS). There are many connections that exist across the lesion. Even in ASIA A or complete spinal cord lesions, probably about 3 – 5% of the connections across the lesion sites are functioning; they are just not functioning properly. Scientists, therefore, are trying to promote remyelination. This can be done through transplantation, stimulation of endogenous stem cells, or by delivering growth factors.

It is important to note that successful results from all three of these techniques are highly dependent on activity. One can release brain-derived neurotrophic factor (BDNF) microscopically to the precise appropriate location through neural activity. Oligodendrocyte myelination can be stimulated through activity[3]. We are hypothesizing that loss of activity plays a major role in chronic spinal injury. These neural circuits need to be active. Most of this knowledge is derived from observations in early development. The nervous system needs constant activity to grow and to form the appropriate connections.

Of all the strategies mentioned, we feel that it is most important to ***optimize spontaneous regeneration***. People need to optimize their own physical health to achieve functional recovery. Putting a transplant into a nervous system that has been slowly debilitated because of loss of activity probably will not be very successful. In 1999, our group showed that transplantation of embryonic stem (ES) cell-derived, progenitor cells, in a traumatic SCI model, can produce behavioral recovery in rats.[4] Although their gait patterns were not normal, the animals that received stem cells showed significant improvement of their gait patterns. At that time, we did not have the appropriate markers to identify specific cell activity. Thus, we did not know what the cells were doing. What we saw was that the cells were causing remyelination and we speculated that this was the cause of the improvements. In addition, many neurons that grew from

the transplanted cells sent axons all the way up and down the spinal cord. They grew about one centimeter per week. In fact, these axons preferred to grow in white matter not gray matter; just the opposite of what scientists used to believe.

We know that stem cells have many functions besides simply replacing cells that are lost. They can replace neurons, astrocytes and oligodendrocytes. They can form chimeric blood vessels; meaning they can integrate into normal blood vessels. They are also an important growth factor production and delivery system. They reduce and interact with the inflammatory response following SCI, and they break down inhibitors in the glial scar through enzymatic mechanisms and phagocytosis. There are many mechanisms that contribute to recovery. We do not know which ones are most important, but probably multiple factors are involved.

Is it possible for these cells to create an organ? Yes it is. Organogenesis has already been shown in other organs, such as the pancreas and liver. How can this knowledge be applied to the nervous system? Researchers can get nervous system cells to grow, to connect, and even to appear to function appropriately. However, it does not look like the normal nervous system. It does not form all of the complex structures that the normal nervous system does. So, is the concept of growing or restoring the nervous system achievable? Years ago, we stumbled across something very interesting: neural induced stem cells can spontaneously make embryoid bodies. These amazing little formations contain neural tube-like structures in them, which are almost identical to neural tubes that occur during normal development. These cells can spontaneously become neurons and form circuits in tissue culture dishes. They also form oligodendrocytes that will function appropriately. This tissue is indistinguishable from the immature nervous system at the light and electron microscopy level. In one experiment, we injected the cells as small clumps in the space around the spinal cord of normal animals and then waited 4 months. We saw that the graft grew outside the spinal cord and resembled a highly similar architecture to the spinal cord.[5]

Another exciting topic of neuron-restoration is the concept of activity-based restorative therapies (ABRT). This concept is based on the hypothesis that neural activity is critical for the nervous system to not only maintain itself, but also to support regeneration and for recovery of function. To enhance regeneration, we need to increase neural activity. Activity is dramatically decreased below the level of the injury following SCI. If activity is reduced in the developing nervous system, then development is dramatically impaired. Mechanisms that are important for cell development, migration, path-finding, fate-choice, and myelination, are all dependent on activity. All of these mechanisms would be negatively affected by reducing activity during normal development; this is also the case in the normal, adult nervous system. We used to believe that the nervous system did not turn over or replace itself. We now know that there is significant turnover of cells, mainly of glial cells. Replacement of oligodendrocytes is important and it is regulated by activity. How can we achieve this replacement in individuals with SCI? We can simulate SCI in the laboratory and add functional electrical stimulation (FES) with the prediction that we are going to enhance regeneration. We have been able to show that FES can dramatically enhance regeneration[6]. We measured indices, such as cell birth, migration, myelination and formation of circuits. By adding FES to rats with SCI, we have demonstrated enhanced cell birth and survival selective to the spinal cord area that is stimulated. Adding more activity to a normal area did not show any differences. FES also enhances neural differentiation of embryonic stem cells, shifting differentiation from oligodendrocytes and astrocytes to neurons.[8] Oligodendrocyte myelination can be enhanced by increasing neural activity.

Is it possible to speculate that some of our traditional interventions, for example the use of Baclofen, a drug commonly used in patients with spasticity, may have been inhibiting spontaneous recovery of function? We demonstrated that the use of Baclofen not only inhibited a return of function, it resulted in a worsening of function[7]. By using Baclofen in animals at clinically relevant concentrations following simulation of SCI, using the same standards we would use clinically, we showed that recovery is dramatically impaired.[7] When we took chronically injured animals and started them on Baclofen, they lost function. Even after stopping the drug, the deficit persisted. This implies that whether given early or late in the course of treatment, Baclofen is not necessarily a good thing. We feel that activity is a better treatment of spasticity than any medication.

How can we apply this knowledge to humans? At our center, we are using an FES bike system. With the FES bike, we stimulate muscles of the legs and buttocks using a small computerized control system. The bike is very easy to use and can be used at the patient's home. Patients can use their own wheelchair and can connect themselves very easily. During the early phase, muscles are usually weak. With continued exercise, they become stronger and will require different settings. Changing the program is automated to make it as simple as possible for the patient. The concept that serves as the foundation for ABRT is that the cycling motion results in the

stimulation of the leg muscles. The contraction of these muscles produces patterned neural activation within the spinal cord that goes up the cord and activates a central pattern generator (CPG). The CPG is comparable to a minicomputer within the spinal cord that knows the program for walking. When activated, it sends volleys of activity up the spinal cord. Activity usually cannot cross the injury zone. However, what happens over time is that increased activity in the spinal cord can actually stimulate regeneration. It can stimulate axonal outgrowth, but mostly it stimulates remyelination of those connections that already exist across the lesion but are not functioning properly. Additionally, activity results in the release of growth factors. ABRT can achieve all these things and it does so in largely the appropriate way. The benefits of exercise are well documented for reduction of cardiovascular risk factors, including diabetes, hypertension and hyperlipidemia. People who are paralyzed have an even greater need for exercise because they simply cannot move. Activity in these patients, in addition to the above mentioned benefits, results in increased muscle mass, the maintenance of bone density, enhanced blood flow, and a decrease in the complications that frequently occur in this population.

Ordinarily, one of the limitations of these treatments is the time and effort it takes to come to a rehabilitation center. Few people have the time to go to a center three times a week. We solved this problem by putting the FES bike into the patients' homes. We were able to show that just the health benefits of exercise alone result in long-term cost savings.

Everyone remembers the case of Christopher Reeve. He did not recover any substantial function within his first 5 years following the injury. However, following the initiation of ABRT, he went from no motor function to 20 percent of normal function within three years. Also in those three years, he recovered from 7 percent of normal sensory function to 70 percent normal sensory function. He enhanced his physical body fitness. He had a tenfold reduction in infections and associated use of antibiotics. The reduced major medical complications and improved quality of life gave him hope to live. Initially, he had no hope and was not given any chance of recovery. With therapy, he was on the road to recovery from a worst case scenario injury. Using his example, we want to emphasize that delayed substantial recovery is possible.

At the time, we did not understand the mechanisms that led to these positive changes. We did have a few hypotheses which led to the design of further studies. For example, in a prospective cohort analysis, we looked at the benefits of three hours of FES biking a week. The physical benefits were enormous. Following spinal cord injury, muscle mass shrinks and is replaced by fat tissue leading to an unchanged size of the limb. The loss of muscle causes early diabetes. The increased fat causes the reduction of HDL's (the "good cholesterol"), increasing the cardiovascular risk. Other scientists have already shown that reversing these changes can significantly decrease those complications. FES cycling can do just that following a couple months of therapy. We were able to show in a group of 60 patients who had experienced a spinal cord injury at least 2 years prior that we can reduce fat by 50% and double muscle mass with FES cycling. In this group, 70 percent of people recovered major neurological function.[9] The other 30% might not have gained function, but they also did not lose any. The patients in the control group who did not receive treatment, lost function on an average of 11 points over a period of 3 years on their ASIA scale. Of the 70 percent that did recover, function improved on average 40 points on their ASIA scale. This is a very significant gain. To put this into perspective,: that is a larger gain than the administration of steroids achieves as an acute injury treatment. We made some additional and very interesting observations: 50% of people in the treatment group were able to discontinue use of Baclofen; 90% were able to reduce Baclofen or change from polytherapy to monotherapy for the management of spasticity. There were also significant improvements in functional independence measures.

In summary, we are able to reverse physical deterioration, and enhance muscle volume and strength while reducing spasticity. We are able to reduce complications, achieve functional gains and obtain recovery of neurological function. Our real goal now is to try to understand the molecular mechanisms that are involved and how to optimize this process. We hope to maximize the physical integrity and benefits by using what we know from animal models in terms of stimulation of regeneration and recovery of function. We believe that the analog of regeneration in humans is recovery of function, which is much easier to measure.

FES cycling is just one aspect of ABRT. The FES bike is probably the most effective and efficient first step in ABRT, because it has been designed for that purpose. However, it is just a small part of ABRT. The same concepts can be applied all over the body.

We think that today there really is hope. We believe that the old days of being told that you have a bad spinal cord injury, life is over, and get used to a wheelchair, are over. Currently, there are hardly any treatments for chronic neurological disease. Activity -based restorative therapies may become one of the first.

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Johns Hopkins Center for NMO, Recurrent TM, and Related Neuroimmunologic Disorders: Clinical Care and Research

Michael Levy MD PHD

I have established a new center at Johns Hopkins. My clinical focus is on Neuromyelitis optica (NMO), longitudinally extensive Transverse Myelitis (TM), recurrent TM, bilateral optic neuritis and recurrent optic neuritis. Appointments at my clinic can be made by calling Mary Brown at (410)502-7099.

Neuromyelitis optica (NMO) is a devastating neuroinflammatory disorder targeting the optic nerves and spinal cord leading to blindness and paralysis. The cause of the disease is unknown, but the recent discovery of a serologic antibody against aquaporin-4 (AQP4) in patients with NMO implicates AQP4 as a key player in pathogenesis. AQP4 is the predominate water channel in the brain expressed on the foot processes of astrocytes around blood vessels where it may be important in regulating the permeability of the blood brain barrier (BBB). In humans, there are two isoforms of AQP4. The shorter AQP4 variant, M23, forms organized orthogonal arrays of membrane channels that maintain BBB integrity. In contrast, the longer variant of AQP4, M1, disrupts M23 arrays and destabilizes the BBB. Loss of BBB integrity in NMO patients may permit the influx of humoral immune mediators, including IgG, IgM and complement factors that characterize the pathology of NMO lesions.

The objective of our lab is to understand exactly how AQP4 isoforms are involved in inflammation of the optic nerve and spinal cord of NMO patients. The hypothesis is that an imbalanced expression of AQP4 isoforms (M1 and M23) creates regions in the central nervous system (CNS) that are more susceptible to inflammation in NMO. Our first aim is to understand why NMO inflammation is largely confined to the optic nerve and spinal cord and how AQP4 isoforms confer susceptibility to inflammation. In addition, we are looking at functional effects of inflammation on AQP4 isoforms using cell cultures of human astrocytes exposed to humoral inflammatory mediators. Based on the findings from these experiments, rodent models of NMO will be developed with an imbalance of M1 and M23 to recreate the susceptibility to CNS inflammation.

Since Dr. Eugène Devic first described NMO in 1894, neurologists have been puzzled about the localization of disease to the optic nerve and spinal cord. Our research not only seeks to explain the unique susceptibility of inflammation to the optic nerve and spinal cord, but also delves into the mechanism of disease to yield new information about how AQP4, and the individual AQP4 isoforms, are involved in NMO. Development of an animal model of NMO based on differential AQP4 isoform expression will significantly improve our understanding of the pathogenesis and move the field toward finding new targets for therapy.

The Transverse Myelitis Association

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

The TMA was incorporated on November 25, 1996 in the state of Washington and became a 501(c)(3) organization on December 9, 1996. The TMA has more than 7,500 members from every state in the United States and from more than 80 countries around the world. There are no membership fees. The TMA is registered with the California Department of Justice, the Maryland Secretary of State, the Ohio Attorney General's Office, and the Washington Secretary of State. The TMA has also been registered with the National Organization of Rare Disorders since 1994.

The T M A is honored to have received permission from the authors to reprint this landmark study about transverse myelitis.

Article abstract—There have been few population-based studies of acute transverse myelitis (ATM). Therefore, incidence and population selectivity of this disorder in different regions is not well known. Data on all Jewish patients with ATM throughout Israel were collected for the period 1955 through 1975. Based on 62 patients who satisfied rigid diagnostic criteria, the average annual incidence rate was 1.34 per million population. No significant difference in incidence was noted between European/American-born and Afro/Asian-born populations. There was no significant seasonal or annual fluctuation in frequency. In 37% of the patients, a history of infection prior to ATM was reported, more commonly among younger patients. ATM rarely evolved into multiple sclerosis. More than one-third of the patients with ATM made a good recovery; in another one-third recovery was only fair; 14 patients failed to improve and 3 died. If other population-based studies of ATM were undertaken, comparison with our results might shed further light on the causes of this disorder.

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Acute transverse myelitis: Incidence and etiologic considerations

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Nelly Zilber, Ph.D., and Esther Kahana, M.D.

Although acute transverse myelitis (ATM) is a recognized entity[1,2] little is known about its etiology and even less about its epidemiologic characteristics. Large series of patients with ATM have been reported,[3-5] but the populations from which these patients were drawn were ill-defined. Therefore, these studies tell little about incidence and population selectivity, aspects which are important if a better understanding of the multiple causes of the disorder is to be achieved. The present study was designed to analyze both epidemiologic and clinical aspects of ATM in Israel.

Israel offered unusual opportunities for an epidemiologic study. The population contains immigrants from every continent and many different regions. Detailed demographic data are available. An extensive network of medical facilities staffed by well-trained personnel exists throughout the country. The major population centers have modern hospitals, each with a department of neurology, and all segments of the population have access to these facilities. National health insurance makes care available to all. It is mandatory to submit discharge diagnoses to the Central Bureau of Statistics. These records were used to establish a National Neurological Disease Registry (NNDR), which we have maintained since 1969.

Methods. Records were reviewed from all hospitals in Israel for the years 1955 through 1975 to identify Jewish patients diagnosed as having ATM. We also reviewed data since 1969 in the NNDR to assure inclusion of all hospital cases of ATM. Additional diagnostic categories in our search included myelitis, acute myelomalacia, acute necrotizing myelopathy, paraplegia of unknown origin, spinal lesions of vascular origin, spinal artery thrombosis and hematomyelia. Since ATM is a disease that requires hospital admission in virtually every case, ascertainment of patients was probably complete.

Criteria for the diagnosis of ATM were applied rigorously and included: (1) acutely developing paraparesis affecting motor and sensory systems as well as sphincters; (2) spinal segmental level of sensory disturbance (patients with patchy sensory deficit or Brown-Sequard syndrome were excluded); (3) stable, nonprogressive clinical course;

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Table 1. Acute transverse myelitis (ATM) in Israel (1955-1975): Number of patients ascertained and accepted from hospital discharge diagnoses

Discharge diagnosis	Number Ascertained	Number Accepted
Myelitis[1] or ATM	73	48
Paraparesis, cause unknown	26	11
Myelopathy, myeloradiculopathy	3	2
Spinal lesion, cause unknown	2	—
Myelomalacia	1	—
Encephalomyelitis	1	—
Spinal lesion, vascular	2	1
Total	108	62

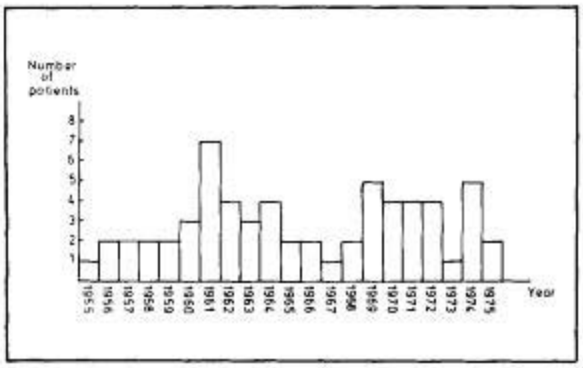

Figure 1. ATM in Israel by year of onset, 1955 to 1975.

Table 2. Annual incidence rate of ATM of the Jewish population in Israel (1955-1975)

Origin	Number of patients	Mean yearly population at risk	Crude incidence rate per 10^6 population ± SD*	Age-adjusted† incidence rate per 10^6 population
Euro-American	20	694,102	1.78 ± 0.35	1.53
Afro-Asian	21	508,476	1.77 ± 0.40	1.53
Israeli	15	918,513	0.78 ± 0.20	0.52
Total	62	2,209,071	1.34 ± 0.17	1.16

SD = standard deviation, calculated assuming a Poisson distribution
† Adjusted to the white population of the US in 1970

(4) no clinical or laboratory evidence of spinal cord compression; and (5) absence of other known neurologic disease, including lues, severe back trauma, malignant disease with metastases, and encephalitis. Patients with irradiation of the spine were also excluded. Most accepted patients were diagnosed as having ATM or myelitis but many had been diagnosed as having paraparesis of unknown origin and would have been missed had we restricted ourselves to a search for cases of ATM or myelitis alone.

Demographic details were obtained from the statistical abstracts published by the Central Bureau of Statistics in Israel.[6] Unless otherwise stated, χ^2 comparisons were used in the statistical analyses.

Results. *Cases studied.* Between 1955 and 1975, 108 patients were identified under the several diagnostic labels from all sources (table 1). Forty-six were excluded, 29 because they did not meet clinical criteria; in 9 patients, the disease developed before immigration to Israel, and in 7 others, ATM developed before 1955 or after 1975. One patient had multiple sclerosis but presented as a transverse myelopathy. The remaining 62 patients (32 men and 30 women) were included in the study.

Annual incidence rate. The number of patients who developed ATM each year ranged between one and seven (figure 1), but these annual fluctuations appeared to be random (χ^2 test, > 0.05). No secular trend was observed in the annual incidence rate (Spearman rank order correlation: $p > 0.05$).

The average annual incidence rate of ATM was 1.34 per million population. The Jewish population was divided into three groups according to place of birth: those born in Europe or America, those born in Africa or Asia (excluding Israel), and those born in Israel. The annual incidence rate in each group was respectively 1.78, 1.77, and 0.78 cases/10^6 population (table 2). Chi-square tests showed that the incidence rate was significantly lower in the Israeli-born than in the Afro/Asian-born ($p < 0.02$) or the European/American-born ($p < 0.01$), but there was no significant difference between Afro/Asian and European/American groups ($p > 0.05$).

Since the age distribution differed in the three populations (table 3), an age-adjusted incidence rate of ATM was calculated. When age-adjusted to the white US population in 1970, incidence rate in each group was 1.53, 1.53, and 0.52, respectively; i.e, the conclusions based upon the crude rates were not changed by age adjustment. Thus, the incidence of ATM was the same in populations born in Europe or America and in Africa or Asia. The apparent significantly lower incidence of ATM in the Israeli-born population requires cautious interpretation because of the small number of Israeli-born individuals who were older than 30 years. Although no case of ATM was found in the Israeli-born population after age 30, had even one such case been found, the age-adjusted incidence of ATM in the Israeli-born group would have increased sharply.

Age distribution. Age of onset of ATM varied between 1½ and 80 years. We investigated whether incidence was increased at any particular age. When age-specific incidence rates (ASIR) were calculated for successive 10-year age groups for the whole population (table 3 and figure 2), there was an apparent peak in the age group 10-19 years

Table 3. Age-specific incidence rates of ATM in Israel (1955-1975)

Age group (years)	European/American-born			Afro/Asian-born			Israeli-born			Total		
	N	n	IR	N	n	IR	N	n	IR	N	n	IR ± SD
0-9	12,647.2	0	—	33,853.8	0	—	434,889.5	4	0.44	481,390.5	4	0.40 ± 0.20
10-19	54,443.5	2	1.75	110,202.3	6	2.59	274,756.6	8	1.39	439,402.4	16	1.93 ± 0.43
20-29	75,561.2	2	1.26	139,303.6	5	1.71	120,968.5	3	1.18	335,833.3	10	1.42 ± 0.45
30-39	105,459.1	3	1.35	113,651.2	2	0.84	48,964.0	0	—	268,074.7	5	0.89 ± 0.40
40-49	147,965.7	5	1.61	84,764.4	3	1.68	20,072.2	0	—	252,802.9	8	1.51 ± 0.53
50-59	147,216.9	6	1.94	60,599.7	3	2.36	9,542.6	0	—	217,359.3	9	1.97 ± 0.66
60-69	97,072.7	3	1.47	31,832.5	2	2.99	5,924.1	0	—	134,829.3	5	1.77 ± 0.79
70+	53,735.3	5	4.43	22,248.4	0	—	3,394.7	0	—	79,378.4	5	3.00 ± 1.34
Total	694,101.7	26	1.78	596,456.0	21	1.77	918,513.1	15	0.78	2,209,070.8	62	1.34 ± 0.17

N = Mean yearly population at risk.
n = Number of patients.
IR = Yearly incidence rate per 10^5 population.
SD = Standard deviation calculated assuming a Poisson-distribution.

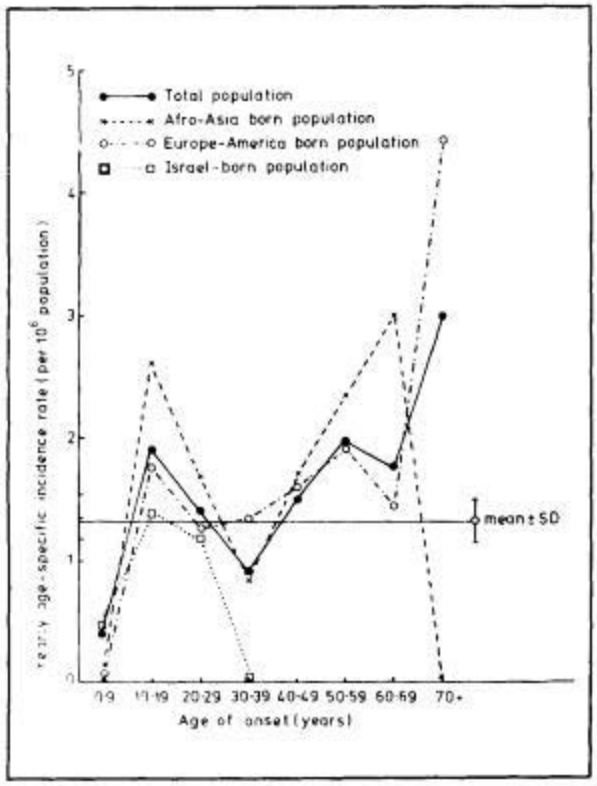

Figure 2. Age-specific incidence of ATM by ethnic origin.

and a second peak after age 40. These peaks were statistically insignificant compared to the mean ASIR for the whole population. However, below age 10 years, the incidence of ATM was significantly lower ($p < 0.02$) than the mean ASIR, and above age 70 it tended to be higher ($p = 0.08$). Comparing larger age classes (for example, younger or older than 40 years), the incidence rates were respectively 1.09 and 1.88 per million population at risk, a statistically significant difference ($p < 0.05$). Thus, incidence rate was higher above age 40 and lower below 40 years of age, with a minimum below 10 years of age.

Seasonal variation. Of the 57 patients for whom date of onset of ATM was available, in 17 symptoms developed in the winter, in 15 in the spring, in 16 in the summer, and in 9 in the autumn. These seasonal differences were not statistically significant ($p > 0.05$).

Preceding infection or trauma. In 25 patients (40%), preceding illness was denied, but 23 (37%) had viral or bacterial infection preceding the onset of ATM by 5 to 21 days. Of these 23, 19 had an upper respiratory infection (possibly viral) and one each had herpes zoster, herpes simplex, otitis media or hepatitis. In two other patients, ATM appeared shortly after smallpox vaccination; three patients gave histories of unusual physical strain before onset of ATM, and one occurred after obstetric delivery. Eight other patients had malaise without fever 3 days to 3 weeks before onset of ATM (figure 3).

Clinical features. The initial symptoms heralding ATM included fever, rash, pain in the back and limbs, muscle weakness, sensory disturbances, and sphincter dysfunction. The interval between the first symptom and greatest neurologic deficit varied from less than 2 hours to 14 days.

At the time of greatest neurologic impairment, muscle weakness was present in 60 patients (97%) and sensory deficit or paresthesia was still present in all cases. In four patients, deep sensation was

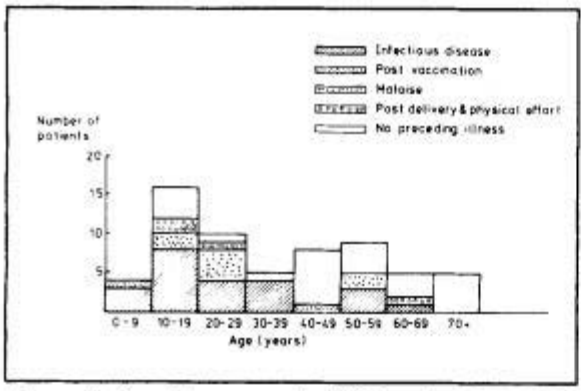

Figure 3. Preceding events in ATM patients by age groups.

Table 4. Acute transverse myelitis in Israel (1955-1975)—spinal cord level below which all sensation was lost, by age of onset

Spinal cord level	Age of onset (years) < 40 N	%	40+ N	%	Total N	%
Cervical	5	14	0	0	5	8
High or midthoracic	12	34	10	37	22	36
Lower thoracic	9	26	11	41	20	32
Lumbar	2	6	3	11	5	8
Unknown	7	20	3	11	10	16
Total	35	100	27	100	62	100

intact. The level of known sensory deficit was in the cervical segments in 5 patients (10%), high thoracic segments in 22 (42%), low thoracic segments in 20 (38%), and lumbar in 5 (10%) (table 4). There was no relationship between the level of the lesion and history of antecedent infection.

Pain in the back, abdominal area or limbs was present in 21 patients (34%). Sphincter disturbances were found in 58 (94%), varying from mere hesitancy to urinary retention and overflow incontinence. Seventeen patients (27%) had fever and eight (13%) had nuchal ridigity.

Laboratory findings. Details of lumbar puncture were available for only 50 patients. During the acute phase, cerebrospinal fluid (CSF) was normal in 19 patients. In 31 patients, the protein content was elevated or pleocytosis was present (or both); 28 patients had CSF protein higher than 40 mg%, the highest being 500 mg%. In those with pleocytosis (20 patients), lymphocytes, polymorphonuclear leukocytes, or both were present.

Myelography was performed on 34 patients and no pathology was found in any of these cases.

Prognosis. No follow-up information was available on three patients. Of the remaining 59, 22 had good recovery, 20 had fair recovery, and 14 had poor recovery according to the criteria of Paine and Byers.[5] Three patients died in the acute stage, two from respiratory insufficiency, and a third from sepsis. There was no correlation between recovery on the one hand and age, sex, ethnic group, clinical picture or preceding illness on the other hand. Recovery took place in most cases between 4 weeks and 3 months after onset. No patient improved later if there were no signs of recovery by 3 months.

Clinical features in different age groups. Infection prior to onset was reported more commonly in the younger than in the older group, 19 (54%) and 4 (15%), respectively. An additional six younger patients and only two older patients reported a prodrome of malaise without fever which could have been due to viral infection. If patients with malaise were included as having antecedent infection, the difference between the younger and older age group in reports of infection would increase even more (71% and 22%, respectively) (figure 3).

The level of sensory deficit was not significantly different in the two age groups ($p < 0.05$) (table 4): Twelve (43% of the cases where this level was known) in the younger group had upper and middle thoracic levels; 9 had lower thoracic levels, 5 had cervical and 2 lumbar sensory levels. The older group had 10 (42% of the cases with known level of sensory deficit) with upper and middle thoracic levels, 11 lower thoracic levels and 3 lumbar levels.

Multiple sclerosis evolving from ATM. Between 1955 and 1975 only one patient who initially satisifed clinical criteria for ATM subsequently developed multiple sclerosis. In the same period, 747 patients were diagnosed in Israel as having suffered from multiple sclerosis. None of these cases of multiple sclerosis had started with ATM.

Discussion. Previous studies of ATM included patients selected from specific hospitals or undefined areas, and no attempt was made to calculate incidence.[3-5,7] In Israel, incidence was calculated for the whole population and for several ethnic groups. No significant difference in incidence was found for the larger ethnic groups, in contrast to multiple sclerosis, which is more common among European- than among Afro/Asian-born groups in Israel.[8]

ATM may be caused by any of a long list of etiologic factors.[4,5,7,9-26] Because the disease is rarely fatal, pathologic data on affected individuals are meager. ATM is rarely the first sign of multiple sclerosis. Altrocchi[3] found that 4 of 67 patients (6%), and Lipton and Teasdall[4] found that 1 out of 29 patients (3%) with ATM later developed multiple sclerosis. In the latter study, autopsies

on eight patients revealed demyelination only at one level; there were no plaques elsewhere.

Patients with direct trauma to the spinal cord were excluded in all series, but some patients with unusual physical strain or minimal trauma were included by some investigators.[3] No patient in our series had back trauma, but unusually hard physical work was reported prior to onset by three patients.

Syphilitic endarteritis was probably a common cause of ATM at the beginning of the century. At present, vascular causes resulting mainly from atherosclerosis and collagen disease are probably more common than syphilis.[9-14] Paine and Byers[5] found a dissociation between pain and other sensory modalities in patients with ATM whether they had antecedent infection or not. Dissociated sensory loss is usually attributed to a vascular cause, as in anterior spinal artery thrombosis or syrinx. On examining the level of sensory deficit, Paine and Byers found that high and middle thoracic sensory level was more common in ATM without prior infection. Since blood supply is more limited at high and middle thoracic cord levels, their findings would be compatible with a vascular cause in patients without infection. Another support for vascular etiology in some cases of ATM is that clamping of the aorta or dissecting aneurysm may cause a clinical picture of ATM.[27]

Patients with systemic lupus erythematosus, in which there may be arteritis, sometimes develop ATM, which may be the presenting manifestation.[9-14] A vascular malformation of the spinal cord caused ATM in one of Altrocchi's patients.[3] With most of the older patients in our series (age 40+ years), the disease began with no preceding illness. Higher frequency of involvement at high and middle thoracic level would support the idea of a vascular cause, but we found no significant age-dependent difference in level of the cord lesion. Twelve of 28 (43%) younger patients and 10 of 24 (42%) older patients had high or midthoracic lesions.

Preceding infection was noted in one-third of the patients with ATM in Hoffman's[16] series, in 25% of Altrocchi's[3] series and in at least 37% of the present series (50%, if the eight with a prodrome of malaise without fever are included). We found that postinfectious ATM was more common in individuals under age 40. An autoimmune cause of ATM after infection may be more likely among younger than among older individuals. Two additional patients, one under age 40 and one over age 40, developed ATM after vaccination.

Several reports have mentioned the clinical and pathologic similarity among ATM, acute disseminated encephalomyelitis (ADE), and experimental allergic encephalomyelitis (EAE).[28-31] EAE had been shown to be a cell-mediated, autoimmune, demyelinating disorder. Histologically, both ADE and ATM may show demyelination or necrosis. Basic myelin protein can induce allergic responses that are clinically and pathologically similar to ATM. Abramsky et al[31] investigated sensitivity to basic myelin protein in 10 patients with ATM. In seven, they found a definite in vitro lymphocyte transformation response to purified central nervous system myelin basic protein, and in three of eight there were sensitized lymphocytes to peripheral nerve myelin protein. Five of those with responses to central basic protein were younger than 40 and two were older than 40 years. Their results are compatible with the idea that an autoimmune mechanism explains some cases of ATM, especially in younger individuals. In older individuals another factor, perhaps vascular, may more often be the cause.

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2010 Rare Neuroimmunologic Symposium, September 23 – 26, 2010 Dallas, Texas The TM and NMO Center, University of Texas Southwestern and The Transverse Myelitis Association

The recently established TM and NMO Center at the University of Texas Southwestern and the TMA will co-sponsor the 2010 Rare Neuroimmunologic Symposium on September 23 – 26, 2010. The symposium will present concurrent science and clinical programs. The science program will begin on Thursday evening, September 23rd.

The clinical program of the Rare Neuroimmunologic Symposium is the most comprehensive offered anywhere in the world to people who have acute disseminated encephalomyelitis, neuromyelitis optica, optic neuritis and transverse myelitis and their families and to the medical professionals who offer care to people in this community. The clinical program will take place from Friday, September 24 – Sunday, September 26. The leading researchers and clinicians from across the country will make the educational presentations and will be available to answer your questions.

We strongly urge you to attend the symposium. By educating yourself about your disorder, the cause of symptoms and the most effective treatments for symptom management, you will become a more effective advocate for your medical care. As important, through your participation in the symposium, you will meet others who have shared similar experiences; people who attend our symposia make life-long friendships.

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Quality Care in Transverse Myelitis: A Responsive Protocol

Carrilin C. Trecker, BS, Dana E. Kozubal, Megan Quigg, BA, Edward Hammond, MD, MPH, Chitra Krishnan, MHS, Peter A. Sim, MD, FACEP, and Adam I. Kaplin, MD, PhD

This study was conducted to aid in the development of a multidisciplinary care center for patients with transverse myelitis. We surveyed the parents of 20 children diagnosed with transverse myelitis between the ages of 0.5 and 21 years to understand their experiences in navigating the health care system. We analyzed acute care events and long-term follow-up in relation to patient satisfaction. Results showed satisfactory ratings in the vicinity of 50% in key areas such as the articulation of a treatment plan. A significant disparity was found in the patients' desire for specialty care and their ability to procure such care. In all, 90% of patients expressed a desire to consult with a psychiatrist, but only 25% were successful in making a visit, a 64% deficit. Recommendations and patient opinions regarding the creation of a collaborative care environment are noted. Research with a larger sample will further elucidate the needs in transverse myelitis patient care.

Keywords: pediatric transverse myelitis; multidisciplinary center; patient satisfaction

Introduction

In the Institute of Medicine report "Crossing the Quality Chasm," the authors conclude that the American health care system is fundamentally flawed and in need of radical, systemic change. The Institute recommends health care customized to the needs of the patient, with the patient as the nucleus of the protocol. The Institute of Medicine characterizes this new protocol as "safe, efficient, equitable, timely, patient-centered, and effective."[1]

Consistent with the objectives advanced by the Institute of Medicine, we set out to understand the perceptions of families with children diagnosed with transverse myelitis, an immune-mediated neurological disorder, which results in inflammation of the grey and white matter of the spinal cord. This rare syndrome affects approximately 1 to 8 per million population annually.[2] The onset of the disease is typically sudden, and it progresses to its nadir in hours to days.[3] Transverse myelitis triggers inflammation in the spinal cord and presents clinically with neurological dysfunction in motor, sensory, and autonomic nerves along the inflamed areas. In our experience at Johns Hopkins, we have identified the additional symptoms of depression and cognitive impairment. To manage the diverse symptoms and sequelae, a multidisciplinary treatment team is best suited to follow those with this disease. We envision adapting patient opinions to establish a multidisciplinary care approach, bringing together relevant disciplines such as neurology, psychiatry, urology, gastroenterology, and physiatry. This multidisciplinary design will facilitate collaboration among medical professionals for the benefit of the patient.

To fully appreciate the patients' perspective and incorporate that perspective into the provision of care, the parents of 20 children with transverse myelitis were asked to complete a survey concerning their experiences with the health care system while at the Victory Junction Gang Camp in Randleman, North Carolina, a summer camp for children with special health needs. Parents completed the anonymous questionnaire, which addressed demographics, treatment history, disease management, and opinions and suggestions based on their experiences. This study qualified for Johns Hopkins Hospital institutional review board exemption [under 45 CSF 46, 101 (b) on October 12, 2007 (protocol number NA_00012848)].

Methods

We collated the surveys completed by the parents of the children diagnosed with Transverse Myelitis. The survey

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Trecker CC, Kozubal DE, Quigg M, Hammond E, Krishnan C, Sim PA, Kaplin AI. Quality care in transverse myelitis: a responsive protocol. *J Child Neurol*. 2008;000:1-6.

Table 1. Summary Characteristics of Patient Cohort

Gender	
Female	10
Male	9
Unspecified	1
Average current age / range	
Overall	10.5 (7-23)
Female	11.7 (7-15)
Male	9.6 (7-23)
Average age at onset / range	
Overall	4.9 (0.3-20.8)
Female	6.8 (0.3-10.5)
Male	3.7 (0.5-20.8)
Average age of parents / range	
Mother	40.7 (28-47)
Father	43.8 (31-55)
Number of attacks	
1	18
≥1	2

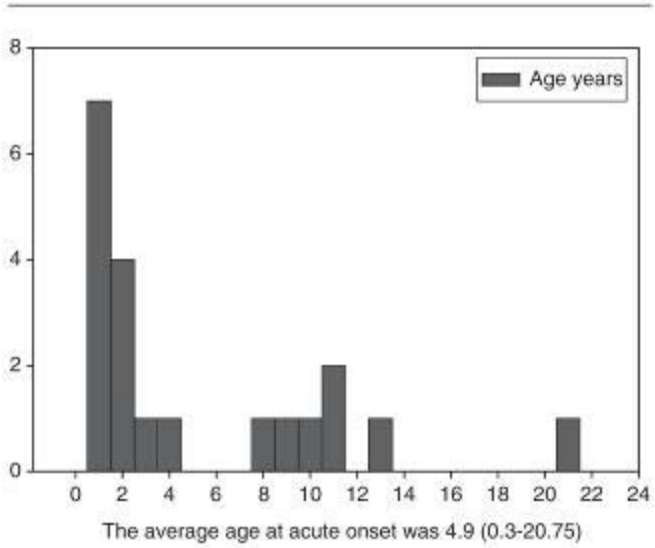

Figure 1. Distribution of age at the onset of acute transverse myelitis.

participants attended Victory Junction Gang Camp during the week of August 19 to 24, 2007. After we articulated the mission and the goals of the transverse myelitis center, the parents of the transverse myelitis campers completed an anonymous questionnaire (see supplemental material). The survey encompassed demographics (age at onset, sex, gender), descriptions of the acute illness (onset of first neurological symptoms, time to diagnosis), and current disease information (current disability, current specialists). The participants also rated their children's care and proffered opinions regarding their experiences in the transverse myelitis health care community. Specifically, participants rated their level of satisfaction on a scale of 1 to 10 in the following 4 areas: time spent by the physician in explaining the diagnosis, physician compassion, time allotted to participants for questions, and manifestation and articulation of a treatment plan. A satisfaction score of 5 or greater was considered "satisfied" and 4 or lower was considered "unsatisfied."

The data entry and analysis were completed using the Statistical Package for Social Sciences (SPSS) version 15.0. Frequencies were calculated, and cross tabulations and parametric tests were performed.

Results

Demographics

The average current age of the patients with transverse myelitis was 10.5 years, with the average age at the time of the child's attack as 4.9 years (Table 1). An analysis of a histogram of the ages at the time of attack demonstrated that the age of onset clustered between 0 to 4 years and 8 to 13 years (Figure 1). This finding is consistent with an analysis of the clinical characteristics of transverse myelitis in children by Pidcock et al,[4] which reported that the onset was clustered between 0 to 2 years and 5 to 17 years.[4] In our sample, 65% (13/20) experienced their initial attack before age 4, whereas 35% (7/20) had their first attack after age 8 (Figure 1).

Acute Phase

Twenty patients with an acute onset of transverse myelitis before age 21 were studied. In all, 35% experienced antecedent flu-like symptoms. Presenting symptoms can be summarized into 4 groups: motor, sensory, cognitive, and autonomic. Eighty-five percent of Patients presented with motor symptoms such as trouble walking, back pain, or paralysis. Sensory issues such as numbness were the presenting symptoms in 20% of children, 5% had cognitive symptoms, and 25% had autonomic symptoms such as bowel or bladder dysfunction (Figure 2, and for specific symptoms see Table 2).

The transverse myelitis diagnosis was made by a neurologist in the majority of the cases, and in other instances by a neurology specialist, a primary care physician, an emergency medicine doctor, and a physician listed as other. When diagnosed by a neurologist or neurology specialist, an incorrect diagnosis was initially delivered in 70% of cases. However, when diagnosed by a primary care physician, emergency medicine doctor, or doctor listed as other, 100% of initial diagnoses were not correct.

Although all patients were ultimately diagnosed with transverse myelitis, 75% (15/20) of the children were initially incorrectly diagnosed. The most common incorrect diagnosis was Guillain-Barre Syndrome, followed by Lyme, acute disseminated encephalomyelitis, and pediatric multiple sclerosis. The average time to the correct

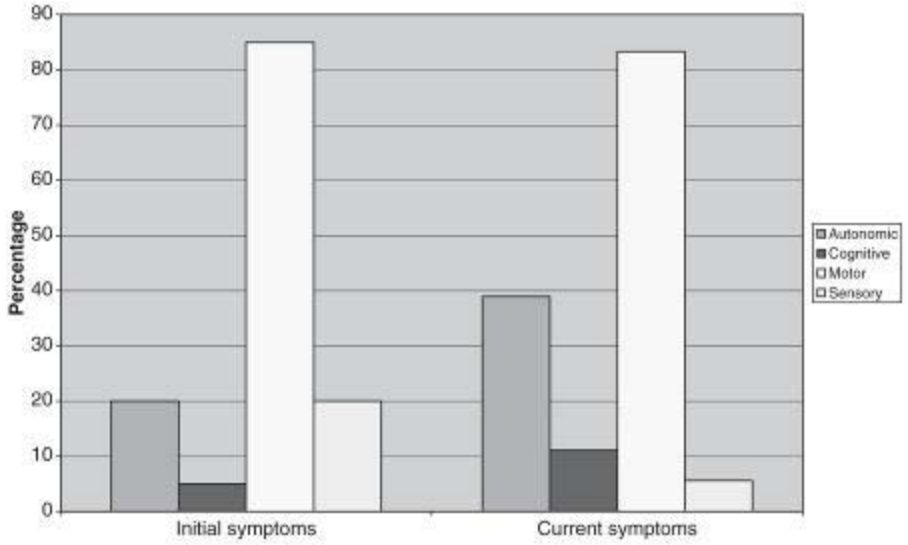

Figure 2. Initial and current symptoms as reported by the parents of the children with transverse myelitis.

Table 2. Initial Symptoms at the Time of First Attack

Symptoms	Number (n = 20)	Percentage
Autonomic	5	25
Urinary	5	25
Bowel	2	10
Cognitive	1	5
Illness	7	35
Flu-like symptoms	3	15
Ear infection	2	10
Fever	4	20
Vomiting	2	10
Motor	17	85
Paralysis	11	55
Trouble walking	6	30
Weakness	3	15
Pain	7	35
Headaches	2	10
Back pain	4	20
Sensory	4	20
Itching	1	5
Rash	1	5
Numbness	2	10
Tingling	1	5

diagnosis among the 20-patient population was 20.6 days (range 0.33-270). Excluding patient #5, whose outlying time to diagnosis was 270 days, the average time to diagnosis was considerably reduced to 7.8 days (0.33-49). When the bimodal age distribution is considered, the sample can be separated into those who experienced their first attack before age 4 (13/20) and after age 8 (7/20). Among the younger age group, the average days to diagnosis was 29.0 (range 0.33-270), and when the 270-day outlier was excluded from this calculation, the average was reduced to 8.9 days (range 0.33-49) among the 0 to 4 age group. In the older age group, the average time to diagnosis was 5.9 days (range 0.4-16).

The majority of the children (75% or 15/20) were diagnosed within a week of symptom onset. However, there were 5 children in the sample whose time to diagnosis exceeded 1 week, with a range of 10 to 270 days. Further scrutiny is required to identify factors, which may have contributed to the extended diagnostic period. Foremost, the average age at the time of presentation was 2.9 years in the cohort, which was diagnosed more than 1 week from the symptoms onset and 5.6 years in the group with time to diagnosis <1 week. Three of the 5 children who were diagnosed more than a week after symptom onset experienced an illness characterized by flu-like symptoms prior to disease onset, whereas only 4 of the remaining 15 children diagnosed within a week of onset reported antecedent illness. Four of the 5 (80%) were initially misdiagnosed: 2 with Guillain-Barre Syndrome, 1 with asthma and allergies, and 1 with a broken bone / a sprain. In addition, if days to diagnosis was >7 days, 80% of the families (4/5) assisted in the diagnostic process, whereas only 33.3% (5/15) helped if days to diagnosis was ≤7 days. There was a significant relationship between the time to diagnosis and the role of the family in the diagnostic process. As the time period between onset and diagnosis increased, the likelihood of the family assisting in the search for diagnostic clues increased ($P < .05$). It is hypothesized that the involvement of the family was

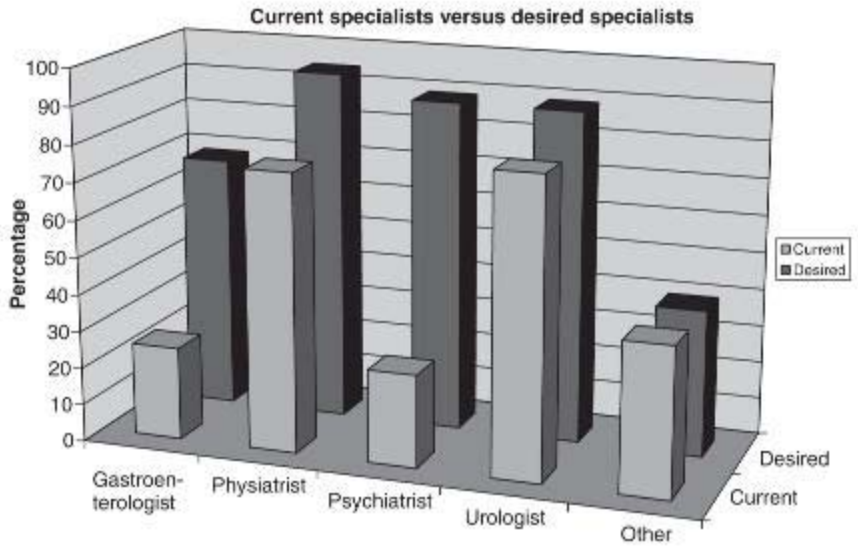

Figure 3.

prompted by the uncertain and seemingly lengthy acute phase. Family help was characterized by information obtained via the internet, other doctors, or other sources.

A second point of significance was found between time to diagnosis and the ease of obtaining a magnetic resonance imaging (MRI). Specifically, patients who experienced a longer time to diagnosis also reported more difficulty in scheduling an MRI ($P < .05$). Given the efficacy of MRI as a diagnostic tool in transverse myelitis, this relationship is expected.

At the time of diagnosis, the greatest number of respondents cited "more compassion" as a key facet for the improvement of the diagnostic process. Other common responses regarded the improvement of long-term treatment options and the need for more hope and more resources. It should be noted that there was also a significant number of parents who did not want anything done differently at the time of diagnosis.

Current Functioning

Following the symptom cluster from the acute phase, motor disability and weakness were the most common sequelae, followed by autonomic dysfunction, cognitive issues, and sensory impairment (Figure 2).

It is also necessary to characterize the type of specialists the patients are currently seeing to address their long-term disabilities and clinical needs (Figure 3). The average number of specialists seen by the patients is 3.2 ± 1.6. The most visited specialist for ongoing care is the urologist, with 80% of the patients (16/20) currently seeing an urologist. Seventy-five percent (15/20) are seeing a physiatrist, 25% (5/20) a gastroenterologist, and 25% (5/20) a psychiatrist. The percentages of those currently seeing the aforementioned specialists can be compared with the percentages of respondents who reported interest in seeing these specialty physicians (Figure 3). Such interest excluded the neurologist. The most sought after specialist is the physiatrist, with 94.4% (17/18) of respondents requesting this service. The urologist and the psychiatrist were also in demand, with 88.9% of patients (16/18) requesting these physicians. Furthermore, 68% (13/19) responded positively to seeking a gastroenterologist.

Satisfaction

To fully understand how to meet the needs of the transverse myelitis community in a multidisciplinary center, we wanted to get a baseline understanding of patient satisfaction; specifically, such information could distinctly elucidate the areas of care that require improvement. In our study, 53% (8/15) of patients were satisfied with the time spent by the physician explaining the diagnosis. Forty-four percent (7/16) were satisfied with the level of compassion displayed by the diagnosing physician. Seventy-one percent (12/17) were satisfied with the amount of time allocated for questions. Lastly, 44% (7/16) were satisfied with the diagnosing physician's articulation of a treatment plan.

Also of note, of those who initially received the correct diagnosis, 100% of patients were satisfied with the time the physician spent in explaining the diagnosis to the patient and family, whereas only 36.4% of patients who

were misdiagnosed were satisfied. Similarly, 100% and 75% of patients who were correctly diagnosed were satisfied with the time available for questions and the level of compassion displayed by the diagnosing physician, respectively, whereas only 55.6% and 36.4% were satisfied with these areas when they initially experienced an incorrect diagnosis.

Discussion

Our findings support the cluster age onset reported by Pidcock et al[4] with 2 peaks observed between ages 0 to 4 and 8 to 21. However, the level of disability in our study patients was less severe. We believe that the difference is attributable to our study design in which the participants were camp attendees, as opposed to a retrospective study in which the hospital records of transverse myelitis children were examined. Children capable of attending a summer camp are more likely to have greater functional capacity than the hospital controls.

Another limitation of our study is the small sample size. However, 20 children should still be considered a significant sample size for such a rare disorder. However, the modest sample size limits our ability to generalize to the transverse myelitis population as a whole. Because the survey was not population based (the participants were attendees at the Victory Junction Gang Camp), it is likely that the sample is not representative of the general transverse myelitis community in the United States. Additionally, because the campers are active participants in the transverse myelitis community, they will presumably use health services at a higher rate than the general population.

One of the main focuses of our analysis was to determine the extent to which the goals set forth in Institute of Medicine report are being met in the pediatric transverse myelitis community. Their specific objective was to provide "safe, efficient, equitable, timely, patient-centered, and effective" care.[1] The satisfaction ratings recorded in our study allowed us to detail the efficacy, timeliness, and effectiveness of providers in diagnosing transverse myelitis. Unfortunately, with satisfaction ratings in the vicinity of 50% for critical activities such as the time spent explaining the diagnosis to the family, time allocated for questions, and the manifestation and articulation of a treatment plan, it is clear that the systemic change is needed. Family assessment of the physician compassion was also identified as an area which needs improvement.

Addressing these needs, Charon proposed a model of "narrative medicine" that brings together empathy, reflection, profession, and trust in medical treatment. She purported a model of patient–doctor communication that includes "diagnostic listening" and could very well have ameliorated low compassion scores accrued by the physicians in this study.[5]

One of our significant and more disconcerting findings was the disparity between the family's desire and perceived need for specialty care for their child and the actual care procured by the family. Parents clearly identified the areas in which they felt they needed specialty care and were unable to procure it. The greatest discrepancy was in psychiatry, where 88.9% of parents expressed a desire or perceived need for care from this specialist and only 25% were successful in procuring it, a 64% deficit. Hinshaw[6] notes that the incidence of provision of mental health care is low for children and adolescents, and this is clearly reflected in our findings; unfortunately, an insidious blanket of shame often overshadows the need for mental health in children. However, our study reported a growing number of parents who acknowledged the need for mental health treatment for their child, possibly indicating a dissipating stigma in the transverse myelitis community.

Our experience leads us to believe that the rates of depression in transverse myelitis are comparable with that in the multiple sclerosis, an autoimmune disease with similar underlying immune mechanisms causing neurological injury (unpublished data). Lifetime prevalence of major depressive disorder among patients with multiple sclerosis is 40% to 60%, 3 to 10 times the rate of depression in the general population.[7] Cytokines secreted during an immune response are implicated in triggering depression in patients with multiple sclerosis and transverse myelitis.[7] We are currently attempting to correlate the presence and severity of depression or cognitive impairment in patients with transverse myelitis, multiple sclerosis, and spinal cord surgery with magnetic resonance spectroscopy abnormalities and elevations in pro-inflammatory cytokines. We hypothesize that the multiple sclerosis and transverse myelitis patient cohort experiencing autoimmune activity will have higher rates of depression than spinal cord surgery patients with motor disability unrelated to immune activation. Consequently, it is imperative that patients with transverse myelitis be cognizant of this increased incidence of depression, its biological underpinning, and the necessity for treatment.

An additional deficit was highlighted between the percentage of those patients seeing a gastroenterologist and those who wanted to see one. Sixty-eight percent of respondents desired to see this specialist, whereas only 25% were successful in doing so, leaving a 43% gap.

To best meet the goals outlined by the Institute of Medicine, transverse myelitis care must be consolidated in a Multidisciplinary Care Center. In our cohort, 65% (13/20) of participants reported that they would be interested in visiting a multidisciplinary center, and 65% (13/20) also indicated that they had visited such a center. Although comprehensive care centers are often perceived

as a popular and highly effective form of care delivery, there is currently a debate about the cost effectiveness of such a system. As the cost of medical care continues to rise, primary care physicians are adopting a gatekeeper role to limit access to more expensive specialty care.[8] However, some studies indicate that primary care physicians have increased the number of rehospitalizations among chronically ill patients.[8] Conversely, research has shown that comprehensive care centers lower hospitalization rates and minimize costly adverse events.[8]Short-term costs may be high for multidisciplinary care centers, but as hospitalizations are reported to be the most costly aspects of multiple sclerosis care in Germany and France, for example, their overall cost effectiveness may be beneficial.[9]

In addition, Sutton et al[10] demonstrated that patients who received multiple sclerosis care at Comprehensive Care Centers had better access to health services and perceived their treatment more positively. A similar trend was seen in a study of patients with amyotrophic lateral sclerosis, in which multidisciplinary treatment reduced mortality.[11] Therefore, the problems of specialty access deficits and low satisfaction ratings can be ameliorated through this approach. One patient noted, "I like when all of the providers for my child talk to each other." Another commented, "I believe as time went out from diagnosis is when we faced greater problems with doctor communication." This communication will be paramount to the functioning of any integrated care center. As previously noted, the proposed multidisciplinary approach will bring together relevant disciplines such as neurology, psychiatry, urology, and physical therapy. This multidisciplinary design will facilitate collaboration among medical professionals for the benefit of the patient.

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Acute Transverse Myelitis and Acute Disseminated Encephalomyelitis in Childhood: Spectrum or Separate Entities?

Eppie M. Yiu, MBBS, Andrew J. Kornberg, MBBS, Monique M. Ryan, MMed, Lee T. Coleman, FRANZCR, and Mark T. Mackay, MBBS

The clinical and radiological features of childhood acute transverse myelitis are compared to those of acute disseminated encephalomyelitis with spinal cord involvement in 22 children with acute transverse myelitis and 12 children with acute disseminated encephalomyelitis with spinal cord involvement. Children with acute transverse myelitis were more likely to have a sensory level (55%) and areflexia. Sixty-eight percent of the children with acute transverse myelitis, and 92% of children with acute disseminated encephalomyelitis had longitudinally extensive transverse myelitis. Demyelination was more extensive in acute disseminated encephalomyelitis (mean 15.6 vertebral segments) than in acute transverse myelitis (mean 8.0 vertebral segments). The outcome was normal to good in 82% with acute transverse myelitis and in 100% with acute disseminated encephalomyelitis. Persistent bladder dysfunction was uncommon in both. Poor prognostic factors in acute transverse myelitis are flaccid paraparesis, respiratory failure, and age less than 6 months. These clinical and radiological differences suggest acute transverse myelitis and acute disseminated encephalomyelitis are separate entities.

Keywords: acute transverse myelitis; acute disseminated encephalomyelitis; magnetic resonance imaging

Acute transverse myelitis, an inflammatory demyelinating myelopathy, presents in childhood with lower limb weakness, back pain, and sphincter disturbance.[1] Diagnostic criteria for adults with acute transverse myelitis have been proposed by the Transverse Myelitis Consortium Working Group,[2] but differences between adult and pediatric acute transverse myelitis are well recognized.[3] Longitudinally extensive transverse myelitis, part of the neuromyelitis optica (NMO) spectrum of disorders,[4,5] is not well studied in children.

Acute disseminated encephalomyelitis is a monophasic inflammatory demyelinating condition of the central nervous system defined by a polysymptomatic presentation and encephalopathy.[6] Spinal cord involvement is reported in 3% to 25% of cases.[7-9] The outcome of acute disseminated encephalomyelitis is generally good, with 57% to 89% of children making a full recovery.[7-10]

It is unclear whether childhood acute transverse myelitis and acute disseminated encephalomyelitis share the same pathophysiology and outcomes, or whether they are separate entities. Both are often preceded by a viral illness or vaccination[8,11,12] and are steroid-responsive demyelinating disorders.[8,13-15]

There have been few large studies of pediatric acute transverse myelitis but none comparing the radiological features of acute transverse myelitis and acute disseminated encephalomyelitis. This study compares the clinical and radiological features of acute transverse myelitis and acute disseminated encephalomyelitis with spinal cord involvement and assesses their response to high-dose intravenous corticosteroid therapy.

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Methods

A total of 34 children admitted to Royal Children's Hospital Melbourne between 1997 and 2004 with idiopathic acute transverse myelitis or acute disseminated encephalomyelitis with spinal cord involvement were identified by an *International Classification of Diseases, Ninth/Tenth Revision*, search of medical records and by searching the radiology department database.

Inclusion and exclusion criteria for acute transverse myelitis were adapted from those proposed by the Adult Transverse Myelitis Consortium Working Group.[2] Diagnosis of acute transverse myelitis required (1) acquired sensory, motor, or autonomic dysfunction attributable to the spinal cord, (2) bilateral signs and/or symptoms, (3) progression to nadir between 4 hours and 21 days, following the onset of symptoms, (4) abnormal spinal magnetic resonance imaging (MRI) and/or cerebrospinal fluid pleocytosis, and (5) age less than 18 years. A clearly defined sensory level was not required for inclusion because this may be difficult to assess in young children. Clinically silent brain lesions were not used as exclusion criteria in this study, as this has been previously described in children with acute transverse myelitis.[16]

Diagnosis of acute disseminated encephalomyelitis with spinal cord involvement was made if there was encephalopathy and multifocal neurologic dysfunction (as per consensus definitions for acute disseminated encephalomyelitis),[6] with symptoms or signs referable to spinal cord dysfunction such as urinary retention or a sensory level. Magnetic resonance imaging confirming spinal cord involvement was also required. Children with radiological evidence of cord demyelination but no clinical features of spinal cord involvement were excluded.

Long-term follow-up was available for 33 children. Participants were followed until complete recovery or for a minimum of 12 months in those with residual deficits. Overall outcomes were graded using a modified Paine global recovery score[1,17]: (1) Normal: full recovery; (2) Good: normal or near-normal gait but mild urinary symptoms, and/or minimal sensory and upper motor neuron signs; (3) Fair: mild spasticity with independent ambulation, urgency and/or constipation, and some sensory abnormalities; and (4) Poor: unable to walk or severe gait disturbance, absence of sphincter control, and sensory deficit. Motor outcomes were graded using the Gross Motor Function Classification System, a 5-level classification system developed to describe motor function at different ages for children with cerebral palsy.[18] Children with no deficit were graded "normal." Urinary sequelae were graded as (1) No sequelae; (2) Mild sequelae: infrequent urgency and urinary tract infections; (3) Moderate sequelae: frequent urgency and recurrent urinary tract infections; and (4) Severe sequelae: no voluntary micturition, urinary incontinence, and postvoid residual urine. Sensory sequelae were recorded as present or absent.

All patients underwent spinal MRI, and 33 scans were made available for review by a neuroradiologist blinded to the clinical presentation and original MRI reports. All but 2 MRIs were performed on a 1.5 Tesla GE Echospeed LX magnet. Multiplanar imaging was performed including T1, T2, and postgadolinium sequences. Brain MRI was performed in 22 patients.

Spinal lesions were characterized according to length, location, and signal abnormalities. Lesions spanning 3 vertebral segments or more were classified as longitudinally extensive transverse myelitis.[19] Axial T2 images were classified as (1) holocord, (2) predominantly gray matter, (3) predominantly white matter, or (4) a combination of these.[20] Gadolinium enhancement, if present, was classified as (1) nodular, (2) diffuse, (3) nerve root, or (4) meningeal.[20] Brain MRI and follow-up imaging were reviewed where possible.

Patients were designated with a favorable outcome if they had a normal or good Paine score at final follow-up or unfavorable if they had a fair or poor outcome. Fisher's exact test was used to compare patient groups for determination of prognostic factors. The unpaired *t* test was used to compare continuous data.

Table 1. Subgroups of Children With Acute Transverse Myelitis and Acute Disseminated Encephalomyelitis With Spinal Cord Involvement

Subgroup	Number
Acute transverse myelitis (ATM)	22
Normal brain MRI	7
Brain MRI not performed	11
MRI showing asymptomatic brain lesions	2
MRI showing brainstem extension but no other brain lesions	1
ATM with nerve root enhancement (brain MRI not performed)	1
Acute disseminated encephalomyelitis with spinal cord involvement	12

Note: MRI, magnetic resonance imaging.

Results

A total of 43 children were identified. Four children with disease-associated myelitis due to connective tissue disease, radiotherapy, or NMO were excluded; 1 child had inadequate documentation; 2 had clinical features of acute transverse myelitis but normal neuroimaging (1 with normal cerebrospinal fluid findings, and 1 in whom a lumbar puncture was not performed); and 2 children with clinical features of acute disseminated encephalomyelitis with spinal cord involvement but normal brain MRI (with abnormal spine MRI) were excluded, as they did not meet consensus definitions for acute disseminated encephalomyelitis.[6]

Thus, 34 children met the inclusion criteria. Of these, 22 had acute transverse myelitis. None of these children had encephalopathy or supraspinal signs. Patients with asymptomatic lesions on brain MRI (n = 2) or brainstem extension from a cervical cord lesion (n = 1) were included in this group. Twelve children had acute disseminated encephalomyelitis with spinal cord involvement (Table 1).

Demographic Data and Clinical Features

Of the 34 children, 21 were male and 13 were female. Mean age at presentation in acute transverse myelitis was 7.5 years (median 7.5 years; range, 4 months-15 years)

Table 2. Symptoms at Onset and Nadir in Children With Acute Transverse Myelitis and Acute Disseminated Encephalomyelitis With Spinal Cord Involvement

	ATM (n = 22)		ADEM (n = 12)	
Symptom	Presentation n (%)	Nadir n (%)	Presentation n (%)	Nadir n (%)
Lower limb weakness	21 (95)	22 (100)	11 (92)	12 (100)
Upper limb weakness	–	9 (41)	–	7 (58)
Bladder disturbance	9 (41)	15 (68)	10 (83)	12 (100)
Catheterization	–	13 (59)	–	7 (58)
Sensory symptoms[a]	7 (32)	13 (59)	1 (8)	3 (25)
Sensory level	–	12 (55)	–	1 (8)
Bowel disturbance	–	9 (41)	–	4 (33)
Fever (T > 38°C)	10 (45)	10 (45)	9 (75)	9 (75)
Pain[b]	12 (55)	14 (64)	5 (42)	5 (42)
Headache	3 (14)	3 (14)	8 (67)	8 (67)

Note: ATM, acute transverse myelitis; ADEM, acute disseminated encephalomyelitis with spinal cord involvement.
a. Objective or subjective.
b. Back pain most common.

and 7.2 years in acute disseminated encephalomyelitis (median 6.0 years; range, 2-14 years). One third of cases presented during winter. A prodromal illness was reported in 24 children (71%), 4 weeks prior to presentation: respiratory infections in 14, varicella in 2 (1 with acute disseminated encephalomyelitis and 1 with acute transverse myelitis), and hepatitis B vaccination in 2 with acute transverse myelitis. Mean time to nadir of weakness was 3.1 days in acute transverse myelitis and 4.8 days in acute disseminated encephalomyelitis. Three children reaching nadir of weakness within 12 hours had acute transverse myelitis. Clinical features of both groups are summarized in Table 2.

Lower limb weakness was the most common presenting symptom. Seventy-four percent with acute transverse myelitis and 67% with acute disseminated encephalomyelitis were unable to walk at presentation (excluding those below walking age). Two children with acute transverse myelitis presented with flaccid quadraparesis. At nadir, lower limb weakness was present in all children, and upper limb weakness in 41% and 58% of the acute transverse myelitis and acute disseminated encephalomyelitis groups, respectively. All but 1 child had abnormal lower limb reflexes. Children with acute transverse myelitis were more likely to be hyporeflexic or areflexic while children with acute disseminated encephalomyelitis tended to be hyperreflexic.

Bladder disturbance (retention, or less commonly incontinence) was present in 41% of children with acute transverse myelitis and 83% of children with acute disseminated encephalomyelitis at presentation. However, severe bladder disturbance requiring catherization was present in 59% and 58%, respectively. Sensory symptoms were less common than motor or sphincter dysfunction. A sensory level was present in 55% of children with acute transverse myelitis (cervical in 1, thoracic in 7, and lumbar in 4), compared to 1 child (7%) in the acute disseminated encephalomyelitis group (thoracic level). Another 3 had subjective hyperesthesia or parasthesias (2 with acute disseminated encephalomyelitis and 1 with acute transverse myelitis). Two children (both with acute transverse myelitis) required ventilation for respiratory failure.

Pain and fever occurred in half of the cases. Headache was more common in acute disseminated encephalomyelitis. Supraspinal symptoms in children with acute disseminated encephalomyelitis (in addition to encephalopathy) included ataxia, cranial neuropathies, and mutism.

Investigations

Lumbar puncture was performed in 25 (74%) children. Overall, the cerebrospinal fluid abnormalities were identified in 19 (76%). A predominantly lymphocytic pleocytosis was present in 68% (67% of children with acute transverse myelitis and 78% of those with acute disseminated encephalomyelitis). Cell count mean was 57 cells/μL in acute transverse myelitis (range, 2-348) and 58 cells/μL in acute disseminated encephalomyelitis (range, 0-104). Cerebrospinal fluid protein levels were elevated in 56% (38% of children with acute transverse myelitis and 89% of children with acute disseminated encephalomyelitis). The protein level range was 0.10 to 1.26 g/L, with a mean of 0.41 g/L in the acute transverse myelitis group and a mean of 0.66 g/L in the acute disseminated encephalomyelitis group (normal range 0.20-0.40 g/L). Cerebrospinal fluid oligoclonal bands were negative in 4 children.

Bacterial and viral testing on cerebrospinal fluid for a variety of organisms was negative, except for 1 patient with acute transverse myelitis with positive Epstein-Barr virus polymerase chain reaction testing, and corresponding

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Table 3. Magnetic Resonance Imaging (MRI) Findings of 33 Children With Acute Transverse Myelitis and Acute Disseminated Encephalomyelitis With Spinal Cord Involvement

MRI Findings	Overall (N = 33) (%)	ATM (N = 21) (%)	ADEM With Cord Involvement (N = 12) (%)
T2 hyperintensity	33 (100)	21/21 (100)	12/12 (100)
T1 hypointensity	7/25 (28)	6/20 (30)	1/5 (20)
Gadolinium enhancement	12/24 (50)	8/16 (50)	4/8 (50)
Length (mean)[a]	10.8	8.0	15.6
Continuous lesions	29/33 (88)	20/21 (95)	9/12 (75)
Multifocal lesions	4/33 (12)	1/21 (5)	3/12 (25)
LETM	26/33 (79)	15/21 (71)	11/12 (92)
Cord expansion	18/33 (55)	11/21 (52)	7/12 (58)
Axial T2 patterns			
Holocord	10/31 (32)	7/19 (37)	3/12 (25)
Predominantly gray matter	12/31 (39)	9/19 (47)	3/12 (25)
Predominantly white matter	2/31 (6)	2/19 (11)	0/12 (0)
More than one pattern	7/31 (23)	1/19 (5)	6/12 (50)
Gadolinium axial enhancement patterns			
Nodular	6/12 (50)	4/8 (50)	2/4 (50)
Diffuse	4/12 (33)	2/8 (25)	2/4 (50)
Nerve root	1/12 (8)	1/8 (13)	0/4 (0)
Meningeal	1/12 (8)—with nodular enhancement	1/8 (13)	0/4 (0)

Note: ATM, acute transverse myelitis; ADEM, acute disseminated encephalomyelitis; LETM, longitudinally extensive tranverse myelitis.
a. Length in terms of number of vertebral body segments.

elevated serum immunoglobulin M (IgM) and immunoglobulin G (IgG) titres.

Serological testing was performed in 23 children for a variety of infections. Six children had positive IgM for *Mycoplasma pneumoniae*. Adenovirus was identified on fecal culture in 2 children and rotavirus in 1. *Campylobacter jejuni* was isolated from the child with nerve root enhancement. Influenza A was identified in 1 child on throat swab.

Nerve conduction studies were performed in 3 children and were abnormal in 2 with absent F-wave responses. The 3rd child (with acute transverse myelitis) had gadolinium nerve root enhancement on MRI.

Treatment

High-dose intravenous methylprednisolone was given to all patients except 1 with spontaneous improvement. Most children received 15 mg/kg for 3 to 6 days, at a mean of 5.3 days (median 3.0 days) after symptom onset in those with acute transverse myelitis and at a mean of 5.8 days (median 4.5 days) in those with acute disseminated encephalomyelitis. One child with acute disseminated encephalomyelitis received a second course of intravenous methylprednisolone after 10 days. High-dose intravenous methylprednisolone was followed by a course of oral prednisolone (1-2 mg/kg) in all children, most commonly for 4 weeks. Corticosteroid side effects were uncommon; 4 patients developed transient hypertension. The child with acute transverse myelitis and nerve root enhancement received 2 courses of immunoglobulin after the steroid course.

Magnetic Resonance Imaging Findings

Magnetic resonance imaging was performed at a mean of 4.3 days following onset. A total of 33 magnetic resonance images were available for review. All patients had sagittal T2 and 31 had axial T2 spinal images. Twenty-five patients had sagittal T1 and 20 had axial T1 spinal images. Gadolinium was given in 25 patients, and brain MRI was performed in 22.

Magnetic resonance imaging findings are summarized in Table 3. There were no significant differences between the acute transverse myelitis and acute disseminated encephalomyelitis groups except for the longitudinal extent of cord lesions. All children had T2 hyperintense lesions on spinal magnetic resonance images, best seen on axial images (Figures 1 and 2). Hypointense lesions on T1 imaging were present in only 28%. Cord expansion, most commonly in the cervical region, was present in 55% (Figures 1 and 2). Sixty-eight percent of children with acute transverse myelitis, and 92% of children with acute disseminated encephalomyelitis had longitudinally extensive transverse myelitis. Children with acute disseminated encephalomyelitis had significantly longer lesions, with a mean lesion length of 15.6 segments (median 17.5), compared to 8.0 segments (median 5.0) in children with acute transverse myelitis (2-tailed P value .0018, unpaired t-test). Five children had involvement of the entire spinal

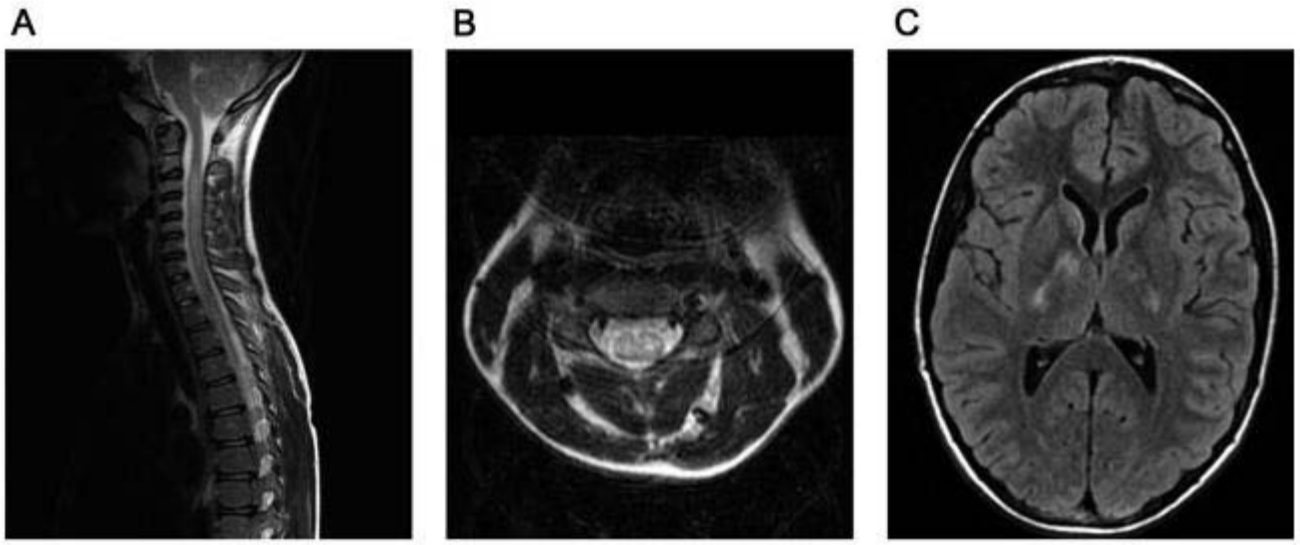

Figure 1. A 14-year-old boy with acute disseminated encephalomyelitis with spinal cord involvement. A, Sagittal T2 image of the spine showing T2 hyperintensity in the cervical region with cord expansion. B, Axial T2 image with holocord pattern of hyperintensity. C, Axial FLAIR image of the brain showing basal ganglia hyperintensity. FLAIR = fluid-attenuated inversion recovery.

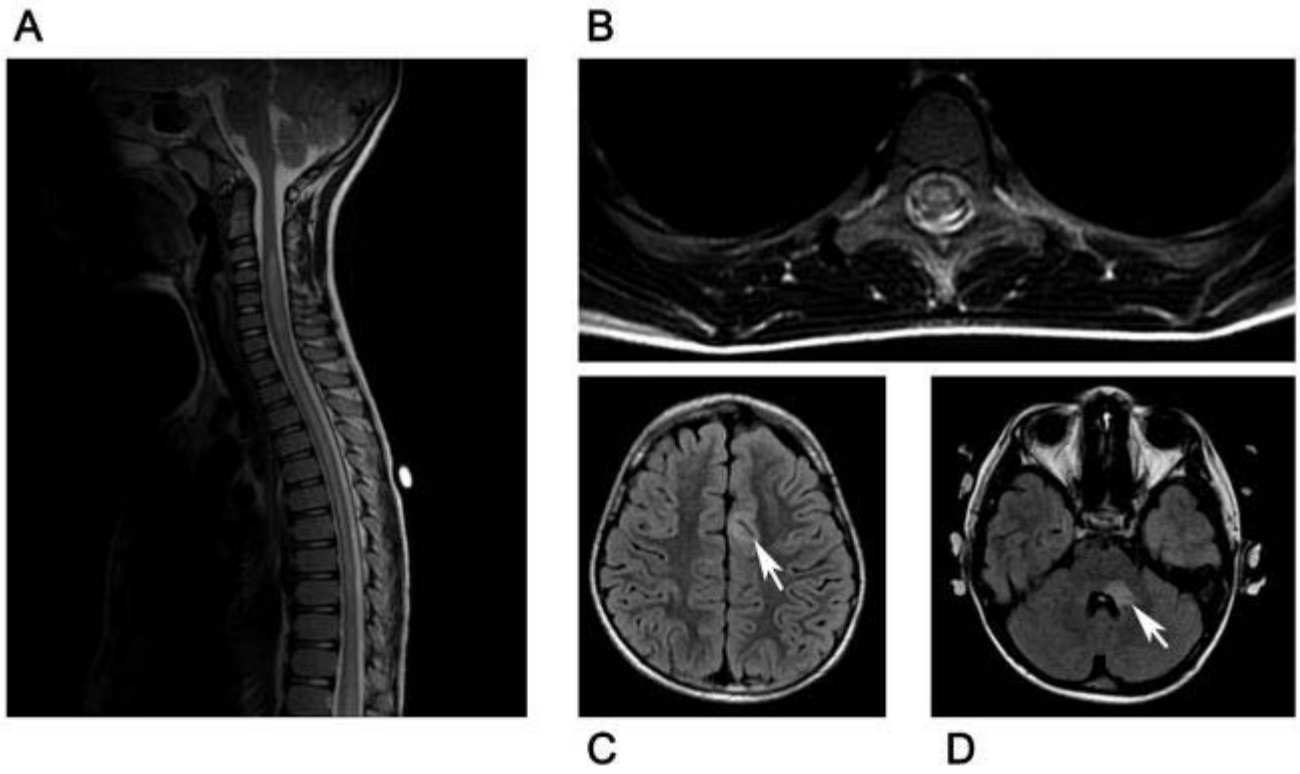

Figure 2. An 8-year-old boy with acute transverse myelitis without encephalopathy or supraspinal signs. A, Sagittal T2 image of the spine showing involvement of the entire spinal cord with cord expansion in the cervical region. B, Axial T2 spinal image showing predominantly gray matter involvement. C, Axial FLAIR image of the brain showing high signal in the left frontal cortical gray matter (arrow). D, Axial FLAIR image of the brain showing high signal in the left superior cerebellar peduncle (arrow). FLAIR = fluid-attenuated inversion recovery.

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cord (2 with acute transverse myelitis and 3 with acute disseminated encephalomyelitis). Most children (88%) had continuous lesions. Four children (14%) had multifocal lesions. Gadolinium enhancement was seen in 50% (Figure 3). Clinical and radiological levels of involvement were disparate in 33%.

Two children with acute transverse myelitis had clinically silent brain lesions, atypical of those seen in multiple sclerosis. These included subcortical white matter, cortical gray matter, brainstem, and cerebellar peduncular lesions (Figure 2). One child with demyelination of the entire cord had extension into the brainstem and cerebellar peduncles. This child had brainstem signs without encephalopathy and so was classified as acute transverse myelitis with brainstem extension rather than acute disseminated encephalomyelitis.

Clinical Recovery

Initial signs of recovery occurred at a mean of 7.2 days from symptom onset. The mean time to independent ambulation was 15 days in 24 documented cases (acute transverse myelitis mean 15.4 days [range, 2-90 days]; acute disseminated encephalomyelitis mean 14.4 days [range, 5-42 days]).

Follow-up data were available in 33 patients for an average of 1.7 years (median 1.0 years; range, 3 weeks-8.5 years), and is summarized in Table 4.

Although differences in outcome did not reach statistical significance between the groups, all children with an unfavorable (fair or poor) global outcome had acute transverse myelitis. Nevertheless, at 12 months, 73% of children with acute transverse myelitis had a normal or good global outcome, corresponding to normal motor function or a Gross Motor Function Classification System score of 1 in 87%. Motor sequelae were permanent if present at 12 months. Of the children with acute transverse myelitis, 77% had normal urinary function at 12 months. Late (after 12 months) recovery of bladder function occurred in 2 cases. Sensory sequelae were present in 2 children.

All children with acute disseminated encephalomyelitis had normal or good recovery by 12 months. One child had mild motor deficits, and 1 had mild urinary sequelae. None had persisting sensory sequelae. No children in either group had further clinical demyelinating episodes.

Follow-up Magnetic Resonance Imaging

Eleven children underwent follow-up MRI scans 2 weeks to 5 years after their initial presentation. All were normal or showed resolving lesions, apart from 1 adolescent with a new asymptomatic right cerebral peduncle lesion at 4 months. Oligoclonal bands were negative. The patient did not develop further clinical symptoms during 2 years of follow-up.

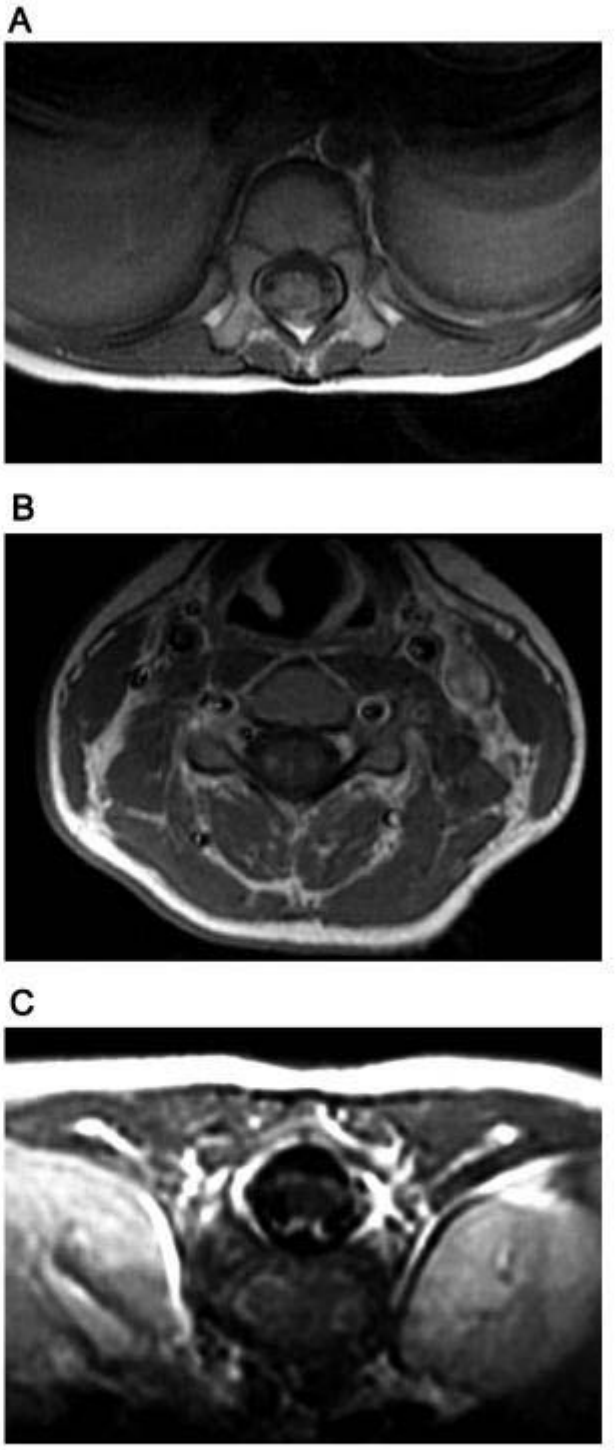

Figure 3. Gadolinium enhancement patterns. A, Diffuse enhancement pattern in a 1½-year-old child with acute transverse myelitis. B, Nodular enhancement pattern in a 15-year-old patient with acute transverse myelitis. C, Nerve root enhancement in a 4-month-old infant with acute transverse myelitis.

Table 4. Long-Term Outcome of 33 Children With Acute Transverse Myelitis and Acute Disseminated Encephalomyelitis With Spinal Cord Involvement

	Overall (33)		ATM (22)		ADEM with Spinal Cord Involvement (11)	
Outcome Measure	At 12 Months n (%)	At Last Follow-up n (%)	At 12 Months n (%)	At Last Follow-up n (%)	At 12 Months n (%)	At Last Follow-up n (%)
Paine global recovery score						
Normal	20 (61)	20 (61)	11 (50)	11 (50)	9 (82)	9 (82)
Good	7 (21)	9 (27)	5 (23)	7 (32)	2 (18)	2 (18)
Fair	2 (6)	2 (6)	2 (9)	2 (9)	0	0
Poor	2 (6)	2 (6)	2 (9)	2 (9)	0	0
Not applicable[a]	2 (6)	–	2 (9)	–	0	–
GMFCS score[b]						
Normal	21 (64)	21 (64)	12 (55)	12 (55)	9 (82)	9 (82)
1	8 (24)	7 (21)	7 (32)	6 (27)	1 (9)	1 (9)
2	2 (6)	3 (9)	1 (5)	2 (9)	1 (9)	1 (9)
3	0	0	0	0	0	0
4	2 (6)	2 (6)	2 (9)	2 (9)	0	0
5	0	0	0	0	0	0
Urinary outcome						
No sequelae	27 (82)	27 (82)	17 (77)	17 (77)	10 (91)	10 (91)
Mild sequelae	1 (3)	4 (12)	0	3 (14)	1 (9)	1 (9)
Moderate sequelae	2 (6)	0	2 (9)	0	0	0
Severe sequelae	1 (3)	1 (3)	1 (5)	1 (5)	0	0
Not applicable[c]	2 (6)	1 (3)	2 (9)	1 (5)	0	0
Sensory sequelae						
Absent	31 (94)	31 (94)	20 (91)	20 (91)	11 (100)	11 (100)
Present	2 (6)	2 (6)	2 (9)	2 (9)	0	0

Note: ATM, acute transverse myelitis; ADEM, acute disseminated encephalomyelitis.
a. Paine score not applicable to children <2 years.
b. Gross Motor Function Classification System.
c. Urinary outcome measures not applied to children <3 years, unless normal continence had been achieved.

Prognostic Factors

As most children in the acute disseminated encephalomyelitis group had a favorable outcome, children with acute transverse myelitis were analyzed separately. Poor prognostic factors included flaccid paraparesis at presentation (4 patients; $P = .0001$), age less than 6 months (2 patients; $P = .03$), and respiratory failure requiring ventilatory support (2 patients; $P = .03$). The child with acute transverse myelitis and nerve root enhancement also had an unfavorable outcome. The longitudinal extent of cord involvement did not affect outcome. Other factors such as time to nadir of less than 24 hours, presence of gadolinium enhancement, T1 hypointensity, cord expansion, and axial T2 hyperintensity patterns were not predictive of prognosis.

Discussion

A comparison of this with previous series of acute transverse myelitis is shown in Table 5. Rates of motor and bladder dysfunction at presentation are similar to previous pediatric series, with lower limb weakness being the most frequent finding. Sensory symptoms were seen in only 32% of children with acute transverse myelitis at presentation in our series, which is lower than in other pediatric and adult series.[21,22] A preceding illness or vaccination was present in 76% of cases, which parallels other pediatric series but is more common than in adult series.[22,23]

T2 hyperintensity on MRI of the spinal cord was a universal finding, best appreciated on axial images. The different axial patterns of T2 involvement[20] did not affect prognosis. This study shows that children have more longitudinally extensive and continuous cord lesions than adults, with adult series reporting mean lesion lengths of 6.7 and 3.3 vertebral segments.[20,24] This is supported by other pediatric series.[25,26] T1 hypointensity and gadolinium enhancement were less sensitive radiological findings, similar to a recent series.[25]

Differences in the clinical and radiological features of acute transverse myelitis and acute disseminated encephalomyelitis with spinal cord involvement were noted. Children with acute disseminated encephalomyelitis were more likely to have headache, while a sensory level and areflexia were more common in children with acute transverse myelitis. Bladder disturbance was marginally more common in acute disseminated encephalomyelitis, but rates of catheterization were similar. Cord lesions were significantly more longitudinally extensive in acute

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Table 5. Comparison of Clinical Features of Adult and Childhood Acute Transverse Myelitis Series

	Our Series (pediatric; ATM, ADEM)	Pidcock et al[25] (pediatric; ATM)	Defresne et al[11] (pediatric; ATM)	Dunne et al[1] (pediatric; ATM)	Ropper and Poskanzer[21] (adult and pediatric)	de Seze et al[22] (adult; ATM)
Publication year	2008	2007	2000	1986	1978	2005
Number	34	47	24	21	52	45
Mean age (years)	7.4	Bimodal 0-2, 5-17 years	8	10	32 (4-83)	38
Prior infection/vaccination (%)	76	75	58	38	33	0
Presenting symptoms (%)						
Lower limb weakness	94	89	67	95	7	27[a] (motor deficit)
Bladder disturbance	56	85	ND	24	6	62
Sensory symptoms	24	91	ND	ND	46	18[a]
Pain (any site including head)	50	75	88	57	33	ND
CSF abnormalities (%)	76	69	62	90	50	–
Pleocytosis	68	50	ND	48	35	20
Increased protein level	56	48	20	52	35	42
Magnetic resonance imaging abnormalities %	100	95	4/6 abnormal	NA	NA	100
Treatment with high-dose intravenous steroids %	Yes	70	No	No	No	Yes
Deaths (n)	0	2	1	0	4	0
Outcomes (Paine) (%)						
Normal	61	[b]	31	38	} 33	} 64
Good	21		25	24		
Fair	6		} 44	19	42	0
Poor	6			19	25	36
Recurrences (%)	0	4	0	0	0	24
Multiple sclerosis (%)	0	2	0	0	13	0

Note: ATM, acute transverse myelitis; ADEM, acute disseminated encephalomyelitis; ND, not documented or insufficient data; NA, not applicable; CSF, cerebrospinal fluid.
a. A further 56% had sensorimotor deficits.
b. 36% of patients able to walk 30 feet independently at follow-up, 80% had residual bladder symptoms.

disseminated encephalomyelitis than acute transverse myelitis. This may be a reflection of lesion load, which is often large in acute disseminated encephalomyelitis. Interestingly, this did not affect spinal cord dysfunction acutely or long term, with the acute disseminated encephalomyelitis cohort tending toward better outcomes.

Asymptomatic brain demyelination was seen in 2 children with acute transverse myelitis, with lesions similar to those typically seen in acute disseminated encephalomyelitis. Clinically silent brain lesions have previously been reported in children with acute transverse myelitis.[16] Another child with acute transverse myelitis had extension of cord changes into the brainstem, which has also been described.[16] Nerve root enhancement, typically described in Guillain-Barré syndrome, was noted in 1 patient. This has also previously been described in children with acute transverse myelitis.[27,28]

In the absence of a biological marker in acute disseminated encephalomyelitis or acute transverse myelitis, it is difficult to say with certainty whether these two entities are on a spectrum or are separate entities. Findings from this study suggest that they are separate entities with some overlapping features, but comparison between larger cohorts is required. In addition, application of consensus-based inclusion criteria for acute transverse myelitis and acute disseminated encephalomyelitis will influence these comparisons.

Outcomes in our series were better than previously published pediatric acute transverse myelitis series, with 73% of children with acute transverse myelitis having a favorable global outcome at 12 months. This may relate to the almost universal use of high-dose intravenous steroids in our patients. Outcomes in our series are also better than adult series, perhaps reflecting a difference in pathogenesis between pediatric and adult acute transverse myelitis.

Factors previously associated with a poor outcome in acute transverse myelitis include complete paraplegia,[11] time to nadir of less than 24 hours,[11,21,29] young age,[25,30] involvement of the peripheral nervous system,[28,31] presentation with spinal shock,[22] requirement for respiratory support,[25] and higher white cell counts in cerebrospinal fluid.[25] Magnetic resonance imaging characteristics including the longitudinal extent of cord involvement[25,31] and T1 hypointensity[25] have been associated with worse outcome. In our series, the only poor prognostic factors were respiratory failure requiring ventilation, age less than 6 months, and flaccid paraparesis at presentation,

although interpretation is limited by the small numbers of patients in these groups. In the 2 infants aged less than 6 months in this study, delayed diagnosis in 1 and concomitant radiculopathy in the other may have contributed to their poor outcomes. However, the immaturity of the developing nervous system at this young age may make it more vulnerable to injury, akin to children with acute disseminated encephalomyelitis affected at a young age who are more likely to have neuropsychological deficits.[32]

The longitudinal extent of lesions in pediatric acute transverse myelitis bears resemblance to those seen in NMO and longitudinally extensive transverse myelitis in adults,[33] in which NMO-IgG antibody rates of 73% and 52%, respectively, have been documented.[5] One study showed NMO-IgG–positive adults presenting with their first episode of longitudinally extensive transverse myelitis had a 56% risk of longitudinally extensive transverse myelitis recurrence or optic neuritis during the subsequent 12 months.[4] Whether adult longitudinally extensive transverse myelitis and pediatric acute transverse myelitis are the same disorder is unclear. The fact that most children in our series had good outcomes without recurrence suggests a difference from adult longitudinally extensive transverse myelitis. This is supported by a recent study that found that no children with monophasic longitudinally extensive transverse myelitis or longitudinally extensive transverse myelitis in the context of acute disseminated encephalomyelitis were seropositive for NMO-IgG.[16] Unfortunately, testing for NMO-IgG antibodies was not possible in this retrospective series.

None of the children reported herein had further clinical episodes of demyelination, consistent with the low rates of recurrent demyelination reported in other series.[1,11,25] This may reflect the relatively short duration of follow-up. The presence of coexistent T2/fluid-attenuated inversion recovery lesions on brain imaging in adult acute transverse myelitis predicts a risk of development of multiple sclerosis ranging from 3% to 21%.[34,35] There are no equivalent pediatric data. We have demonstrated that asymptomatic brain lesions more reminiscent of acute disseminated encephalomyelitis than multiple sclerosis can be present in children with clinically isolated acute transverse myelitis. Whether this finding in children affects the risk of progression to multiple sclerosis is unclear, but it is interesting to note that the risk of progression to multiple sclerosis is higher in acute disseminated encephalomyelitis than in acute transverse myelitis.[36]

Pediatric acute transverse myelitis is characterized by longitudinally extensive lesions on MRI. Its relationship to NMO and adult longitudinally extensive transverse myelitis requires ongoing study. Asymptomatic brain lesions may be seen in pediatric acute transverse myelitis, although its implications for prognosis are unclear. Most children in our series had a normal or good outcome after treatment with high-dose intravenous steroids. Children with acute disseminated encephalomyelitis with spinal cord involvement tended toward better outcomes than children with acute transverse myelitis despite more longitudinally extensive spinal cord changes, suggesting that acute transverse myelitis and acute disseminated encephalomyelitis are separate entities. Limitations of this study are its retrospective nature that limited the outcome assessment and the small number of patients. Future prospective collaborative studies with routine imaging of the brain and spine and testing for NMO-IgG may shed further light on the pathogenesis of these conditions.

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The TMA Newsletter and Journal Archives

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Spectrum of Pediatric Neuromyelitis Optica

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What's Known on This Subject

There have been no descriptive studies of pediatric NMO using current diagnostic criteria. Recent identification of NMO IgG antibody suggests an expanded spectrum of disease, including patients with brain involvement and presentations similar to those of acute disseminated encephalomyelitis or MS.

What This Study Adds

To our knowledge, this is the first descriptive study of pediatric NMO spectrum disorders that incorporated the current diagnostic criteria. We describe clinical, laboratory, imaging features, disability, and treatment responses. Potential risk factors for aggressive disease are identified.

ABSTRACT

OBJECTIVE. Our goal was to describe the spectrum of clinical phenotypes, laboratory and imaging features, and treatment in pediatric patients with neuromyelitis optica.

PATIENTS AND METHODS. The study consisted of a retrospective chart review of patients followed in a pediatric multiple sclerosis center with a diagnosis of neuromyelitis optica spectrum disorder.

RESULTS. Nine patients with neuromyelitis optica spectrum disorders were included, all of whom were female. There were 4 black children, 2 Latin American children, 2 white children, and 1 child of mixed Latin American/white heritage. Median age at initial attack was 14 years (range: 1.9–16 years). Median disease duration was 4 years (range: 0.6–9 years). Tests for neuromyelitis optica immunoglobulin G were positive for 7 patients. Eight patients had transverse myelitis and optic neuritis, and 1 patient had longitudinally extensive transverse myelitis without optic neuritis but had a positive neuromyelitis optica immunoglobulin G antibody titer. Cerebral involvement on MRI was found in all subjects, 5 of whom were symptomatic with encephalopathy, seizures, hemiparesis, aphasia, vomiting, or hiccups. Immunosuppressive therapy reduced attack frequency and progression of disability.

CONCLUSIONS. Pediatric neuromyelitis optica has a diverse clinical presentation and may be difficult to distinguish from multiple sclerosis in the early stages of the disease. The recognition of the broad spectrum of this disease to include signs and symptoms of brain involvement is aided by the availability of a serum biomarker: neuromyelitis optica immunoglobulin G. Early diagnosis and immunosuppresive treatment may help to slow the accumulation of severe disability. *Pediatrics* 2008;122:e1039–e1047

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Key Words
neuromyelitis optica, multiple sclerosis, central nervous system, encephalomyelitis, spinal cord

Abbreviations
NMO—neuromyelitis optica
CNS—central nervous system
MS—multiple sclerosis
ON—optic neuritis
TM—transverse myelitis
IgG—immunoglobulin G
AQP4—aquaporin 4
LETM—longitudinally extensive transverse myelitis
SIADH—syndrome of inappropriate antidiuretic hormone secretion
FANA—fluorescent antinuclear antibody
EDSS—Expanded Disability Status Scale
FLAIR—fluid attenuated inversion recovery
Ig—intravenous immunoglobulin
CSF—cerebrospinal fluid
WBC—white blood cell
dsDNA—double-stranded DNA

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NEUROMYELITIS OPTICA (NMO) is a demyelinating disease of the central nervous system (CNS) that is often difficult to distinguish from multiple sclerosis (MS). There is still debate whether NMO is a distinct disease from MS.[1–4] NMO has been described as a more aggressive disease and not responsive to the immunomodulatory therapies used to treat MS.[5]

Diagnostic criteria for NMO were described in 1999 to include optic neuritis (ON), transverse myelitis (TM), and no symptoms or MRI findings implicating other CNS regions.[6] In 2004, an antibody, serum NMO immunoglobulin G (IgG), was found to be a sensitive and specific biomarker for this disease, a discovery confirmed by several independent laboratories.[7–9] This antibody is directed against aquaporin 4 (AQP4), a regulatory water channel with high levels of CNS expression.[10]

Based on preliminary validation studies of this biomarker, the Pediatric Multiple Sclerosis Study Group suggested the following for the diagnosis of pediatric NMO: (1) ON and TM required as major criteria; and (2) either longitudinally extensive transverse myelitis (LETM) with MRI demonstrating involvement of ≥3 spinal segments or NMO IgG seropositivity.[11] It is important to note that these criteria did not incorporate brain MRI findings in NMO, which were subsequently described.[12]

Diagnostic criteria were revised in 2006 by using likelihood ratios to develop several models, including NMO IgG

as well as spinal cord and brain MRI findings.[13] These models were analyzed to determine which provided the greatest sensitivity and specificity. Statistical analysis of a model identical to that proposed for pediatric NMO had a sensitivity of 100% but inadequate specificity of 79%. Additional inclusion into the variables of a brain MRI not meeting Paty criteria for MS demonstrated a sensitivity of 99% and specificity of 90%, suggesting the importance of brain imaging in trying to ensure the diagnosis.

We report our pediatric NMO experience at the Center for Pediatric Multiple Sclerosis in Houston, Texas. This is one of the largest published cohorts of pediatric NMO from a single center and is, to our knowledge, the first reported series focusing on the diverse presentation in the pediatric population using the newly proposed criteria. This report highlights distinguishing characteristics for the disease to include brain MRI findings that should be considered in the diagnosis.

PATIENTS AND METHODS

A retrospective record analysis was performed for patients seen in the Texas Children's Hospital Center for Pediatric Multiple Sclerosis between 2001 and 2007. All patients were seen and evaluated by the principal author (Dr Lotze). Patient information was extracted from an institutional review board-approved database. All patients meeting current pediatric NMO diagnostic criteria were included. One patient who was NMO IgG-seropositive with recurrent LETM without ON was included because her clinical course was otherwise typical for NMO and thought to represent the broader disease spectrum. Information extracted from the database included age, ethnicity, attack history, MRI findings, laboratory values, disability, and response to treatment. NMO IgG-seropositive status was collected at the time of initial evaluation. Repeat testing was performed in 7 patients during the course of disease.

RESULTS

Demographics

Nine patients meeting inclusion criteria for NMO spectrum disorder were identified. Clinical and laboratory characteristics are summarized in Table 1. Selected case histories (patients 1, 2, and 9) are reported in the Appendix demonstrating 3 distinct presentations. All patients were female. Reported ethnicities of the children were black (4 [44%]), Latin American (2 [22%]), white (2 [22%]), and mixed Latin American/white heritage (1 [11%]). Median age at the time of the study was 16 years (range: 6–20). Median age at initial attack was 14 years (range: 1.9–16) with median disease duration of 4 years (range: 0.6–9). Mean relapse rate per year was 2.6 (range: 1–4), and median disability based on the Expanded Disability Status Scale (EDSS) score was 3 (range: 0–8).

Optic Neuritis

ON occurred concurrently with TM in 5 patients and 12 months after an initial presentation of TM in 1 patient (patient 5). In 2 other patients (patients 3 and 8), TM followed the initial presentation of ON, with the 2 attacks separated by 3 and 18 months, respectively. ON was bilateral at onset in 5 patients (Fig 1 A and B) and bilateral sequential in 2 patients (patients 7 and 8), with attacks separated in time by 2 and 12 months, respectively. One patient had unilateral ON (patient 6). Visual impairment varied both between and within each patient. Four patients (patients 1, 4, 7, and 8) had severe vision impairment, with complete vision loss in the affected eye. In the remainder, vision recovered to at least 20/25. Patient 8 has complete vision loss in the right eye after her initial attack of ON, but the vision in her left eye recovered to 20/25.

Transverse Myelitis

All patients had symptoms consistent with TM during their disease course. One patient (patient 7) had a segmental spinal cord lesion involving C2 at initial presentation. She then had an attack of LETM (C1–C6) 4 years later. The remainder of patients had spinal cord involvement greater than 3 spinal segments at onset. LETM on MRI most frequently involved the cervico-thoracic spinal cord (Fig 1C). In 7 patients who had spinal cord imaging performed at the time of acute attacks of TM, enhancement of lesions was found after administration of gadolinium. One patient (patient 9) had recurrent episodes of LETM but did not have clinical, neurophysiologic, or imaging evidence of ON. However, here serum NMO IgG level was positive, and her clinical response to plasmapheresis supported an antibody-mediated disease. In addition, her clinical phenotype was similar to other patients, including a more severe disease course with several attacks per year and significant disability. This patient and 2 others (patients 1 and 4) had atrophy of the spinal cord at segments of previous attacks as measured by progressive decrease in caliber of the cord on serial imaging (Fig 2 A and B).

Brain Involvement

Three patients had symptoms attributed to brain disease. Seizures occurred with the first clinical attacks in 2 patients (patients 4 and 7). Both patients developed epilepsy and required treatment with anticonvulsants. Serial imaging of patient 7 demonstrated atrophy and increased fluid attenuated inversion recovery (FLAIR) signal in the left mesial temporal lobe. Patient 2 developed aphasia as part of her third attack with a lesion in the left temporal lobe. Her initial attack was characterized by syndrome of inappropriate antidiuretic hormone secretion (SIADH) attributed to disease involvement of the hypothalamus on brain imaging (Fig 3A). Patient 7 had aphasia as part of her initial presentation with a thalamic lesion on MRI.

All patients in our cohort had abnormalities on brain MRI from disease onset (Fig 3). Distribution of the lesions demonstrated on brain MRI is summarized in Table 2 and Fig 4. Nonspecific changes (nonovoid, nonenhancing, nonperiventricular deep white matter lesions or too few to satisfy the Barkof criteria for MS) were

TABLE 1 Clinical and Laboratory Parameters of the Pediatric Cohort of Patients With NMO

Patient No.	Age, y/ Ethnicity	Age of Onset, y	Attack Symptoms	No. of Attacks	EDSS Score	Current Impairments	Endocrine-Related Changes	NMO IgG (Age, y)	Other Antibodies	Family History	Current Medicine (s)
1	6/B	2	Vision loss, paraplegia	5	5	BVL, paraparesis		Neg (4.5) Neg (5.4) Neg (6.4)	+APL Ab FANA 1:320	Mother: APL Ab	MM and PRD daily RTM
2	19/W	15	Vision change, encephalopathy, aphasia, paraparesis	>10	0		SIADH, irregular menses	Pos (17.9) Pos (18.2) Pos (18.5) Neg (18.7) Neg (19.7)		Father: Still's disease; First cousin: DM	MM 1 g bid IVIg (1g/kg) and IVMP monthly
3	16/B	14	Vomiting, vision disturbance, quadriplegia	2	2.5	Tonic spasms, left hand weakness, paresthesias	Irregular menses	Neg (15.1) Neg (15.6) Pos (16.1)		Mother: hypothyroid	MM 1 g bid PRD every other day
4	16/LA	7	Quadriparesis, vision loss, seizures	4	8	Encephalopathy, BVL, quadriparesis, epilepsy		Pos (16.8)	+ SSA/Ro; FANA 1:1280 autoimmune hepatitis		MM 1 g bid PRD qD
5	14/B	12	Vomiting, paraplegia	2	1.5	Vertigo, sensory impairment	Catamenial exacerbation	Neg (12.1) Pos (13.8) Neg (14.2)	FANA >1:1280 + anti-ENA with RNAse	Paternal first cousin: MS; Maternal first cousin: DM	MM 1 g bid PRD qD RTM
6	17/W	16	Unilateral ON, paresthesias, leg weakness	3	0			Neg (16.8) Neg (17)	+SSA FANA 1:1280		MM 1 g bid PRD qD
7	20/LA	15	Encephalopathy, seizures, paraplegia, vision loss	>5	5.5	BVL, paraparesis	Postpartum exacerbation	Pos (18)	FANA 1:1280 dsDNA 1:80 + anti-RNP + anti-ENA without RNAse		RTM
8	15/B	12	Bilateral ON, right vision loss, paresthesias	4	3	Right vision loss, paresthesias		Pos (14) Pos (14.2) Pos (14.7) Neg (15.2) Pos (15.4)	FANA 1:320		RTM
9	20/LA/W	16	Paraplegia	>5	7	Paraparesis	Catamenial exacerbation	Pos (18.4) Pos (20.5) Pos (20.7)			AZT, GA, PP monthly RTM

B indicates black, BVL, bilateral vision loss; Neg, negative; APL Ab, antiphospholipid antibodies; MM, mycophenolate mofetil; PRD, prednisone; W, white; Pos, positive; bid, twice per day; DM, diabetes mellitus; IVMP, intravenous methylprednisolone; SSA/Ro, Sjogren's syndrome A/anti-Ro antibody; FANA, fluorescent antinuclear antibody; LA, Latin American; qD, every day; RNP, ribonucleoprotein; ENA, extractable nuclear antigen; RTM, rituximab; AZT, azathioprine; GA, glatiramer acetate; PP, plasmapheresis.

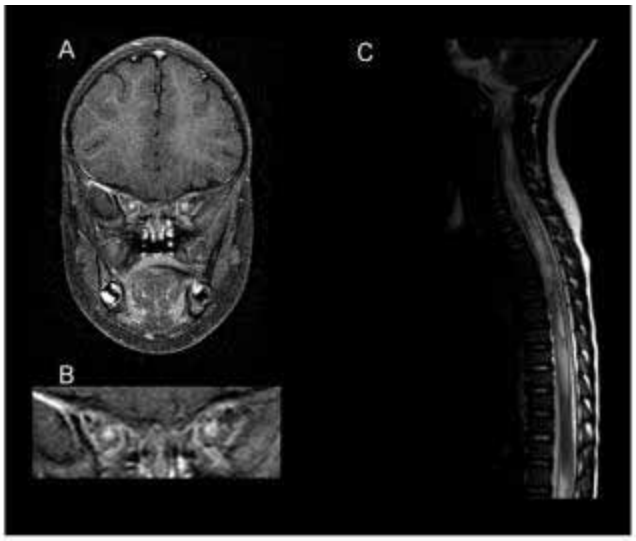

FIGURE 1
Patient 1 with classic clinical presentation. A, Coronal T1-weighted fat-saturated postgadolinium imaging performed through the orbits demonstrating abnormal enhancement returned from both optic nerves. B, Expanded view of the optic nerve enhancement shown in A. C, Sagittal T2-weighted midline imaging of the spine with T2 hyperintensity demonstrating extensive holocord involvement.

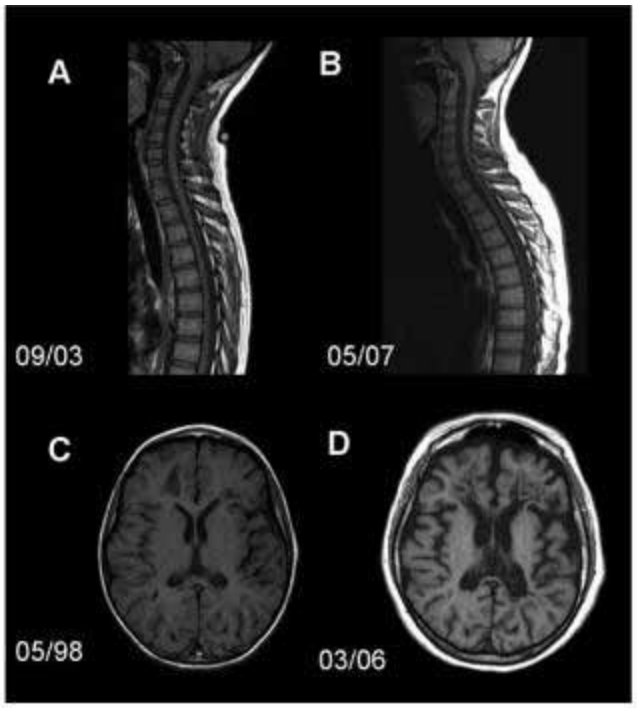

FIGURE 2
Atrophy in individuals with pediatric onset. A and B, Patient 1 with progressive spinal cord atrophy after repeated attacks of LETM as measured by progressive decrease in caliber of the cord on serial imaging over a period of 4 years. C and D, Patient 4 with brain involvement and progressive brain atrophy as measured by sulcal widening and increased ventricular size of serial imaging over a period of 8 years.

found in 8 patients at initial presentation. Atypical lesions (large confluent cerebral or diencephalon lesions) were seen in 1 patient on the initial MRI. With serial imaging during the course of follow-up, 2 patients met Barkof criteria for separation in space on MRI.

Eight patients had periventricular lesions, including the temporal horns of the lateral ventricles, the third and fourth ventricles, and the periaqueductal region. Four patients had lesions involving the corpus callosum, 4 had hypothalamic lesions, and 2 had parahippocampal lesions. Juxtacortical and central white matter lesions were found in 5 patients. Three patient (patients 2, 4, and 7) developed generalized atrophy on serial images as measured by increase in size of the lateral ventricles and sulcal spaces (Fig 2 C and D). All of these patients had a higher lesion load in the central white matter compared with other patients in the cohort.

NMO Serology

NMO IgG was evaluated through the clinical Neuroimmunology Laboratory at Mayo Clinic by using indirect immunofluorescence assay on a substrate of mouse CNS and kidney tissues.[7] Seven patients had NMO IgG seropositivity. Patient 2's NMO IgG seroconverted from positive to negative status during chronic treatment with daily mycophenolate mofetil plus monthly pulse intravenous immunoglobulin and methylprednisolone. Patient 5 was initially seronegative after a course of plasmapheresis, but converted to seropositive during a second attack. Her NMO IgG later returned negative results during treatment with rituximab. Patient 8 seroconverted to a negative NMO IgG during treatment with rituximab. Four months later she returned positive results again.

Menstrual Irregularities

Three patients (patients 2, 3, and 5) had irregular menstrual cycles before their initial attack. Their cycles became regular while receiving immunosuppressive treatment. Catamenial exacerbation of disease occurred in 1 patient, and initiating oral contraceptives corresponded with decreased attacks. One patient (patient 7) had an uncomplicated pregnancy and delivered a healthy term infant. She had no clinical relapses during the pregnancy but 1 month after delivery had an attack of ON and TM.

Autoimmune Disease

Six patients had serologic evidence of other autoimmune antibodies or disease. Patient 1 had positive antiphospholipid antibody titers including IgG anticardiolipin and positive lupus anticoagulant. Patients 4 and 6 were found to have a positive SSA antibody (Sjogren's syndrome A/anti-Ro antibody); however, they did not meet diagnostic criteria for Sjogren disease. Patient 4 developed biopsy-proven autoimmune hepatitis early in her disease course. Six patients had fluorescent antinuclear antibody (FANA) titers of >1:320.

Family History

Six patients had a family history for autoimmune diseases. One parent had rheumatoid arthritis and antiphospholipid antibody syndrome. In another family, 1 parent had adult-onset Still's disease. In a third family, a parent had hypothyroidism. First cousins in 2 families

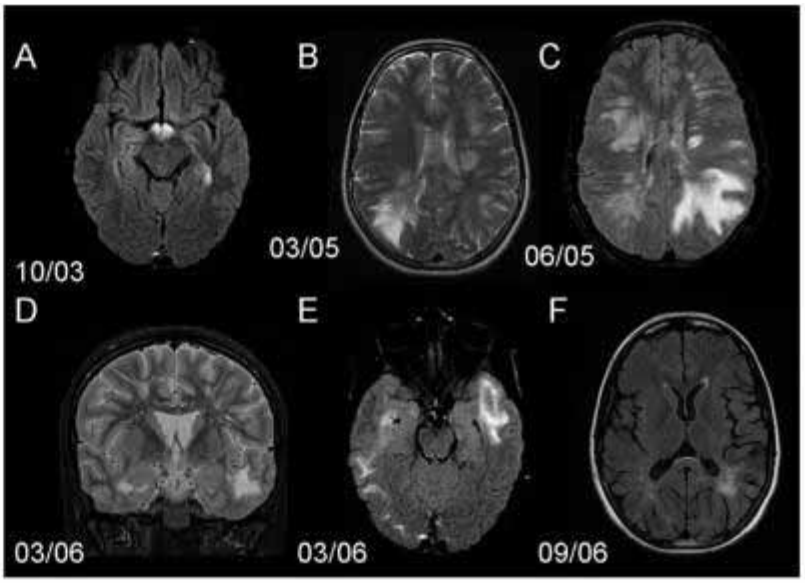

FIGURE 3
Patient 2 with atypical clinical disease. A, Axial T2-weighted FLAIR imaging at initial presentation demonstrating T2 hyperintensity returned from the periventricular gray matter around the anterior end of the third ventricle and hypothalamic region as well as the left periatrial white matter. B, Axial T2-w imaging performed at the level of the body of the corpus callosum showing abnormal T2 hyperintensity in a Dawson-fingers-type pattern of distribution. Large tumefactive lesions are seen in the right periatrial region and the left centrum semiovale. C, Axial T2-weighted FLAIR image demonstrating decreased signal intensity in the right periatrial region and a new tumefactive lesion in the left periatrial region. Also note increased signal abnormality in the right centrum semiovale. D, Coronal T2-weighted STIR (short tau inversion recovery) image demonstrating left-to-right temporal white matter signal abnormality with involvement of the body of the corpus callosum. E, Axial T2-weighted FLAIR image with left temporal lobe involvement again showing left-to-right signal abnormality with possible interruption of the arcuate fibers during an acute aphasic episode. Note additional involvement of the right occipital lobe. F, Axial T2-weighted FLAIR image with residual left periatrial hyperintensity, ex vacuo dilatation of the supratentorial ventricular system, and widened cerebral sulci while on intravenous methylprednisolone for an acute clinical relapse.

had juvenile-onset diabetes mellitus. One patient had a paternal second cousin with MS. This cousin's history included ON, but by report he did not fulfill NMO criteria and was seronegative for NMO IgG.

Disease Course

All patients had a relapsing-remitting course. Median time to second attack was 7 months (range: 3–18 months), the median number of total attacks was 7 (range: 2–11), and the median EDSS was 3 (range 0–8).

Therapy

All patients received treatment for acute attacks with high-dose intravenous methylprednisolone (30 mg/kg per dose). Four patients were also treated with intravenous gammaglobulin and plasmapheresis for acute attacks (patients 1, 2, 5, and 9). Patient 2 was treated with a 6-month course of pulse cyclophosphamide after her third attack.

Median chronic treatment duration was 3.7 years (range: 1–9 years). Six patients were treated with steroids in combination with mycophenolate mofetil (patients 1–6) as part of their regimen. Five patients were treated with rituximab (patients 1, 5, 7, 8, and 9). Patient 9 was additionally treated with azathioprine, glatiramer acetate, and monthly plasmapheresis. Patient 2 was treated with monthly intravenous immunoglobulin (IVIg). On current therapeutic regimens, mean attack rate for the group is 0.5 per year.

DISCUSSION

The definition of NMO has been changing over the past decade. It is now recognized that a significant percentage of the adult patients with NMO have cerebral involvement, and the identification of the NMO IgG biomarker has broadened the disease spectrum. There is increased awareness of pediatric demyelinating disease, including NMO. These changes coupled with the increased effectiveness of therapeutics, have emphasized the need to consider NMO in the differential diagnosis of inflammatory demyelinating diseases.

Although this reported cohort is too small to draw any conclusions concerning the demographics of pediatric NMO, some observations correlate with risk factors identified in other populations. All patients were female, similar to adult NMO populations that reported a fe-

TABLE 2 CNS Distribution of Lesions for Pediatric NMO

Patient No.	Spine	Optic Nerves	Brain						
			Juxtacortical	Central White Matter	Corpus Callosum	Hypothalamus	Periventricular	Brainstem	MRI Appearance
1	C1–T10	Bilateral	+	+			+	+	Nonspecific
2	C1–C2, C4–C6	Chiasm/tracts	+	TD	+	+	+		DIS
3	C3–T1	Bilateral					+	+	Nonspecific
4	C1–T10	Bilateral	+	+	+		+		DIS
5	C2–C5, C7–T2	Optic chiasm			+	+	+	+	Atypical
6	T1–T9	Right				+	+	+	Nonspecific
7	C1–C6, T3–conus	Bilateral	+	+		+	+	+	Nonspecific
8	C2–C4	Bilateral						+	Nonspecific
9	C1–T10		+	+	+		+	+	Nonspecific

TD indicates tumefactive demyelination; DIS, Barkof criteria for dissemination in space.

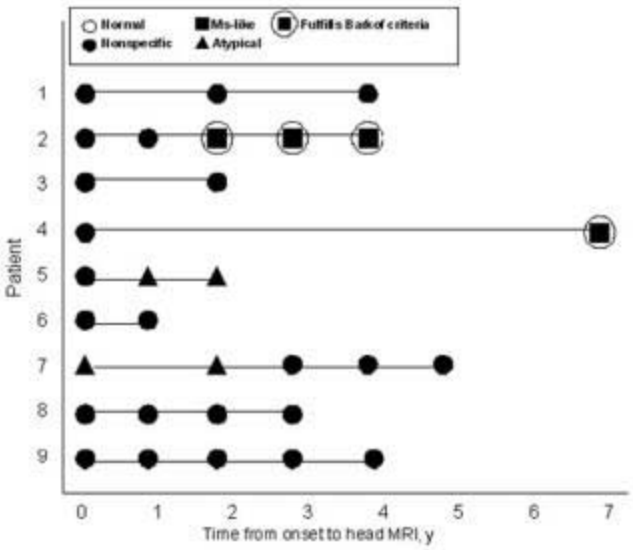

FIGURE 4
MRI brain characteristics in pediatric patients with NMO over time.

male/male bias of 9:1.[5] Similar to observations in pediatric MS,[14] >75% of the cohort had Latin American or African ancestry. This may indicate that Northern European ancestry is less of a determinant in pediatric-onset demyelinating disease than in the adult-onset disease. In this cohort, children with Latin American ethnicity had a higher EDSS (>4.0) compared with non-Latin Americans (Fisher's exact test: $\chi^2 = 5.62$, $P = .047$). There was no significant correlation between age of onset (greater or <10 years) with EDSS ($P = .16$).

In NMO, the lesions in the spinal cord are more longitudinally extensive than MS. We observed that the lesions exhibited a rim-enhancement on post contrast T1-weighted images in contrast to MS lesions, which exhibit a more uniform enhancement. We observed a trend that more longitudinally extensive spinal cord lesions were associated with a greater disability as measured by EDSS (lineal regression: $P = .054$).

There was variable involvement of the optic pathways. Some patients had extensive involvement of the optic nerve, extending along the length of the nerve whereas other patients had minimal involvement limited to the optic chiasm, and 1 patient had no detectable optic pathway involvement. Additional clinical studies including optical coherence tomography and multifocal visual evoked potentials should be considered to better determine the spectrum of optic pathway involvement.[15,16]

Previously, brain involvement was considered to be an exclusion criterion for NMO.[6] However, recent series have reported up to 60% with MRI changes in the brain.[12,17–19] All patients in this study had nonspecific or atypical lesions on initial brain MRI. Two patients had additional evolution of MRI changes during their disease course to eventually satisfy Barkof criteria. The remaining patients continued to show nonspecific or atypical changes.

Brain lesions in our cohort frequently involved the diencephalon, an area of high AQP4 expression along with the ependymal cells around the ventricles and the astrocytic endfeet that form the blood-brain barrier in capillaries and pia of the brain, optic nerves, and spinal cord.[10] One patient had large tumefactive lesions that were nonenhancing, extended up to but not involving the subcortical U fibers, and had finger-like projections that seemed to follow glial tracts. The occurrence of these large lesions in pediatric NMO is important to recognize, because such patients might present with a clinical and radiographic phenotype suggestive of acute disseminated encephalomyelitis. However, in contrast to acute disseminated encephalomyelitis, they are at greater risk for progressive disability without immunosuppressive therapy.

Three patients had generalized cerebral atrophy by MRI. This suggests underlying background disease progression in the brain, as has been described for MS. It is unknown how much brain atrophy will accumulate in NMO, and these patients should be monitored for signs of cognitive impairment with neuropsychological evaluation.

Aphasia is an unusual presentation of demyelinating diseases[20,21]and has not been previously reported in any patient with NMO. Patient 7 initially presented with expressive aphasia, which was attributed to a large lesion in the thalamus. Patient 2 had an expressive aphasia secondary to tumefactive left hemisphere white matter lesions. White matter lesions may produce aphasia through diaschisis, a disruption of the white matter pathways between anatomically related language centers producing deficits indistinguishable from cortical lesions.[22] This is consistent with the magnetic resonance imaging in our patient.

Four patients demonstrated an abnormal T2 signal in the diencephalon with hypothalamic involvement, supporting previous descriptions that this area is commonly affected in NMO.[10] This may account for endocrine changes encountered in 1 of our patients. Case histories of adult NMO have described endocrinopathies related to hypothalamic involvement.[17,23] In our series, 1 patient had symptomatic hyponatremia and with SIADH. Dysfunction of the hypothalamic-pituitary axis should be investigated in NMO because endocrinopathies may complicate the presentation.

NMO IgG titer analysis has been proposed to be a biomarker capable of distinguishing between NMO and MS or other autoimmune CNS diseases.[7] It is important to note that although this antibody test is highly specific, it is not as sensitive resulting in false-negatives. Therefore, a negative NMO IgG test does not exclude the diagnosis. This is demonstrated by patient 1 who had classic clinically defined NMO but never had detectable NMO IgG antibody levels. Treatments such as plasmapheresis or rituximab may affect NMO IgG results, indicating the importance of repeat testing in patients with a clinical course consistent with this disease.

One patient in our cohort did not meet currently criteria for NMO because she did not have detectable optic pathway involvement. However, the aggressiveness of her disease course, NMO IgG seropositivity, and

response to immunosuppressants suggests that recurrent LETM may be part of the NMO disease spectrum and therapy should be modified accordingly.

The relationship between NMO and other autoimmune diseases has been described.[6,24,25] This cosegregation is hypothesized to relate to a similar humoral pathogenesis. NMO may occur in the setting of a clinically evident systemic autoimmune disorder. In our cohort, 6 patients had other autoimmune serologic markers, and 5 patients had a positive family history for autoimmune disease, including 1 family with a history of MS. A recent report of a large cohort of patients with NMO found 4 of 71 had a family history of MS, although there was no reported family history of NMO.[6] Whereas the lifetime risk for MS in first-degree family members is estimated to be 22% to 55%,[26,27] the risk for developing NMO is unknown.

At our center, the current therapeutic approach for pediatric NMO is intravenous methylprednisolone (30 mg/kg per day up to 1 g/d for 5 days) for initial treatment of acute exacerbations. Patients failing to respond within 7 days of treatment are treated with plasmapheresis. If symptoms still persist, IVIg 2 g/kg divided over 2 to 5 days is started. Daily oral prednisone and azathioprine or mycophenolate mofetil are used for chronic therapy. Patients with additional disease progression are changed to rituximab. Rituximab has been shown to be effective in adult NMO[28] and needs additional study in the pediatric populations. The small size of the cohort in this retrospective observational study, as well as the nonstandardized treatments used, limits the ability to assess efficacy of any particular treatment regimen.

CONCLUSIONS

We document one of the largest, to our knowledge, pediatric cohorts of patients with NMO to date and highlights significant clinical points for pediatric patients with an NMO spectrum disorder. It is important for pediatricians to recognize that (1) the current definitions of NMO are expanding, and it is important to consider the diagnosis in the setting of classic disease with negative antibody titers or atypical disease with positive antibody titers, (2) NMO should be considered in the differential diagnosis of children with a central demyelinating event, and involvement of the brain no longer excludes this as a diagnostic possibility, (3) hypothalamic-pituitary axis function should be evaluated in pediatric patients with NMO, and (4) immunomodulatory therapies effective in MS may be of limited benefit; therefore, treatment with immunosuppressive agents may better preserve cognitive, visual, and ambulatory function in the pediatric population.

APPENDIX

Patient 1: Classic Clinically Defined NMO and Negative Serum NMO Antibody Titer

Initial Presentation

A 23-month-old black girl developed quadriparesis, vision loss, vomiting, and irritability 1 week after receiving an influenza vaccination. MRI demonstrated diffuse increased T2 signal in both optic nerves (Fig 1 A and B) and longitudinally extensive T2 bright signal abnormality throughout the cervicothoracic spinal cord with patchy enhancement after contrast (Fig 1C). MRI of the brain and orbits showed a few small foci of increased T2 signal abnormality in the left frontal region (data not shown), and her cerebrospinal fluid (CSF) demonstrated pleocytosis with 19 white blood cells (WBCs) (40% polymorphonuclear neutrophils), elevated protein at 62 mg/dL (normal: 15–45 mg/dL), normal IgG index, and no oligoclonal bands. She was found to have multiple positive antiphospholipid antibodies including anticardiolipin IgG, and a positive lupus anticoagulant test. Her mother was evaluated for chronic arthritic symptoms and was found to also be positive for antiphospholipid antibodies. The patient was initially treated with high-dose intravenous methylprednisolone (30 mg/kg per dose) and had improvement of her vision as well as significant recovery of her arm and leg strength.

Disease Course

Optic neuritis recurred 4 months after the initial attack, for which she again received high-dose intravenous methylprednisolone followed by oral steroids with some improvement of vision. Three months later, while attempting to wean her off the oral steroids, she experienced a third relapse with recurrence of the LETM and gait failure.

Current Therapy and Clinical Status

After this third attack, she was started on monthly IVIg (1 gm/kg per dose) and daily mycophenolate mofetil and continued on daily oral steroids. Two additional relapses included bilateral optic neuritis and transverse myelitis 18 and 21 months later, respectively. At the most recent follow-up 4 years after disease onset, she was ambulatory with the assistance of a walker and has profound vision impairment and urinary incontinence. Bilateral optic atrophy is present by physical examination and on MRI. Her spinal cord has shown progressive atrophy (Fig 2 A and B). However, there remains minimal brain involvement by MRI, and she has exhibited no signs of cognitive impairment.

Patient 2: Nonclassic Clinical Presentation of NMO With Tumefactive CNS Lesions and a Positive NMO Antibody Titer

Initial Presentation

A 15-year-old right-handed white girl initially presented with a 3-week history of fatigue, nausea, blurry vision, and paresthesias in her legs and hands. A sensory level was present at T4, and she had normal mental status and no focal motor deficits. Neuroophthalmologic examination revealed slight thinning of the nerve fiber layer in the superior aspect of the left eye by optical coherence tomography. MRI of the optic nerves demonstrated increased T2 signal in the region of the optic chiasm (data not shown). MRI of the spine demonstrated increased T2 signal at C1–C2 and a second longitudinally extensive lesion at C4–C6. MRI of the brain demonstrated in-

creased T2 signal intensity in the hypothalamus and the periventricular regions (Fig 3A) with minimal gadolinium enhancement. CSF studies showed 1 WBC with slight elevation of protein at 57 mg/dL (normal: 15–45 mg/dL), normal IgG index, and no oligoclonal bands. Hyponatremia with serum sodium as low as 111 mmol/L was found on serum chemistries. Further investigation was consistent with SIADH. She was treated initially with intravenous methylprednisolone for 5 days followed by a 6-week oral steroid taper with resolution of her SIADH and paresthesias.

Disease Course

Four months after initial presentation, she developed nausea and Lhermitte sign (a sudden transient electric-like shock extending down the spine triggered by flexing the head forward). MRI of the spine demonstrated reenhancement of the lesion at C6. She received treatment with intravenous methylprednisolone for 5 days followed by a 3-month oral steroid taper. She remained asymptomatic until 6 months after completing her steroid taper. At that time she started to experience abdominal paresthesias and leg weakness. Her MRI of the spine demonstrated a longitudinally extensive lesion in the thoracic spine (T5–T10). She was treated with intravenous methylprednisolone and started on subcutaneous interferon β-1a for a diagnosis of probable MS.

Four months later she developed severe headaches, disorientation, and right face and arm weakness. An MRI of the brain demonstrated a large right temporoparietal lesion with little enhancement (Fig 3B). Her clinical status improved after intravenous steroid treatment, except for continued frequent headaches. Three months later, her headaches became more severe and she developed an expressive aphasia. Her MRI demonstrated a large left temporal lesion despite immunomodulatory therapy (Fig 3C). Because her monotherapy treatment failed, immunosuppressive therapy was added and included monthly intravenous methylprednisolone 1000 mg and intravenous cyclophosphamide 500 mg/m^2. Cyclophosphamide dosing was titrated to achieve a WBC level of 1500 to 2000. She had another attack 3 months after starting this regimen, with symptoms of blurred vision, back pain, and confusion. MRI of the brain demonstrated multiple large minimally enhancing lesions (data not shown). Five months later, she again developed an expressive aphasia with a new large lesion in the left hemisphere (Fig 3 D and E). Each of these events was treated with intravenous methylprednisolone with good response (Fig 3F).

Repeat studies including antiphospholipid antibodies, ANA, Sjogren's syndrome A/anti-Ro antibody (SSA), Sjogren's syndrome B/anti-La antibody (SSB), double-stranded DNA (dsDNA), cytoplasmic anti-neutrophil cytoplasmic antibodies (cANCA), perinuclear anti-neutrophil cytoplasmic antibodies (pANCA), rapid plasma reagin (RPR), and lupus anticoagulant were negative. Testing for NMO IgG became available and was sent during a later (December 2005) exacerbation with tumefactive lesions and found to be positive (titer = 1:3840 [normal: <1:120]). Her diagnosis was switched from probable MS to NMO with cerebral involvement, interferon was discontinued, and therapy was retargeted to a humoral disease process including monthly intravenous methylprednisolone 1000 mg, monthly IVIg (1 gm/kg per dose), and twice-daily mycophenolate mofetil 1000 mg.

Current Therapy and Clinical Status

After switching to the NMO-directed treatment plan, she had not had any additional relapses for 2 years except for 1 episode of a severe headache. At her most recent follow-up 4 years after disease onset, she had mild fatigue and her MRI showed mild cerebral atrophy. Her EDSS score was 0, her vision was 20/20, and she was attending college with a straight-A average. Her serum NMO IgG was repeated during this period of disease remission, and she has had decreasing titers and eventually seroconverted to negative.

Patient 9: Multiphasic LETM Without ON and With a Positive NMO Antibody Titer

Initial Presentation

A 15-year-old Latin American girl presented with gait failure and urinary retention. MRI demonstrated a longitudinally extensive lesion in the medulla and extending through the cervicothoracic cord (data not shown). Her brain MRI demonstrated a few areas of increased T2 signal change involving the centrum semiovale bilaterally. CSF studies noted pleocytosis with 31 WBCs, elevated protein of 78 mg/dL (normal: 15–45 mg/dL), normal IgG index, and no oligoclonal bands. She was initially treated with high-dose intravenous methylprednisolone followed by plasmapheresis. She had some clinical improvement, but her physical impairments did not return to baseline, and she initially required a cane for ambulation.

Disease Course

After initial improvement, her symptoms recurred 4 weeks later. She received intravenous methylprednisolone for this exacerbation. After a third relapse 2 months later, her clinical picture of multiple small lesions in the brain, transverse myelitis, and no optic nerve involvement placed her initially in the clinical category of MS spectrum disease, and she was started on intramuscular interferon β-1a for maintenance therapy. Repeat CSF studies continued to show pleocytosis with 37 WBCs and no oligoclonal bands.

This patient developed a more severe clinical course with relapses every 1 to 2 months despite starting interferon therapy. After 2 months, she developed leukocytopenia, and interferon was changed to glatiramer acetate with recovery of her WBC count. She continued to have recurrent attacks every other month with symptoms that included gait failure, sensory deficits, arm weakness, dysphagia, and a central Horner syndrome on the right with associated diplopia. Ophthalmologic evaluation, including visual evoked potentials and MRI, did not find evidence for optic neuritis. She was started on monthly plasmapheresis as an adjunct therapy. Over the

next 6 months, exacerbation of her disease was noted to consistently occur 1 week before beginning her menstrual cycle. She was started on an oral contraceptive to help ameliorate these symptoms. She subsequently had only 1 attack over the following 12 months. Antiphospholipid antibodies, ANA, SSA, SSB, dsDNA, cANCA, pANCA, RPR, and lupus anticoagulant were negative. During this period of time, she tested positive for serum NMO IgG. Because she had a better clinical response to humoral targeted immunosuppressant therapy rather than immunomodulatory therapy, azathioprine was added at that time for a working diagnosis of NMO.

Current Therapy and Clinical Status

At her most recent follow-up 4.5 years after disease onset, she had significant weakness in her legs and arms (0/5 in the legs and 4/5 in the arms, bilaterally symmetric). She could only walk a short distance with maximal assistance and used a wheelchair routinely for ambulatory assistance. Serial imaging by MRI demonstrated progressive atrophy of the spinal cord. Brain imaging demonstrated punctate 2- to 3-mm enhancing lesions in the juxtacortical regions of both hemispheres and in the corpus callosum (data not shown). There remained no evidence for optic pathway involvement by MRI or physical examination. She was treated with azathioprine, monthly plasmapheresis, and glatiramer acetate (continued because of patient's perceived benefit), and her attack rate has slowed from every 1 to 2 months to 1 to 2 episodes per year. She was recently started on a trial of rituximab wit the hope of further reducing her relapse rate.

ACKNOWLEDGMENTS

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Drs Lotze and Northrop contributed equally to this work.

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Special Request for People with ADEM

TMA Contributes $20,000 for Enrollments in the ACP Repository

The Accelerated Cure Project is dedicated to determining the causes and accelerating research into finding the cures for NMO, MS, TM, ADEM, and ON. One of ACP's primary initiatives is the building of a repository of blood samples and data from people with these disorders. These samples are distributed to researchers studying the causes of these diseases, thereby accelerating research.

The repository provides a common population of samples useful for a wide variety of studies that enables results from different research perspectives to be easily combined and correlated. The repository contains various types of samples and data that can support scientists working in many fields, such as genetics, nutrition, virology, and more. Researchers gaining access to the repository must return their results to the database to be shared with other researchers; this allows for cross-correlation of their results with all other studies performed using the same samples.

Your information will be handled confidentially. Some of the centers will accept children, while others will not, so please check the center closest to you.

While samples continue to come into the repository through increased enrollment, they also continue to be distributed in support of research into the causes of MS, TM, NMO, ADEM, and ON. You can learn more about the research being supported by the Accelerated Cure Project repository samples and data by visiting: ***www.acceleratedcure.org/repository/research.php***

One of the scientific teams using the ACP repository needs samples from people with ADEM right now! This team has started a company to develop tests that will diagnose diseases and predict their course. They are currently looking at molecules in the blood of people with MS, ADEM, NMO, and ON to see how these diseases are alike and different. Your participation in the repository will help this team carry out its research, providing new insights into the biology of ADEM, and will help other scientists in the future also.

Currently, we have only 13 people with ADEM enrolled in the ACP repository. If we are going to get good research done on ADEM, we are going to need a much larger number of people enrolled. ADEM is rare – you can't count on someone else to do this for you. You hold part of the answer to these most fundamental and critical issues … what happened to you and why? Please help us to find the answers.

In addition to expanding the breadth of the repository through greater collection site access, ACP is also expanding the depth of it through the collection of updated data and samples from already enrolled participants. One of the particularly valuable aspects of the ACP repository is that it is a longitudinal study, meaning that participants are asked to return over the course of their lifetime for follow-up visits. These visits allow for the collection of updated health information, replenishment of blood samples, and provide the opportunity to ask new questions on topics that were not addressed during the first visit. Having participants return for follow-up visits means that ACP can provide valuable samples and data to researchers in support of the study of disease course, the impact of medications on progression, among other critical areas.

ACP has announced that the first follow-up visits are now underway. Participants have enthusiastically returned to provide updated data, new blood samples, and to answer the questions that have been added to the interview related to stress and trauma. If you are a repository participant with a demyelinating disease and you enrolled more than a year ago into the repository, you may be getting a phone call or postcard soon asking you to return and continue your involvement. Your continued involvement enhances the value of the repository and accelerates research into the causes of ADEM, MS, NMO, ON and TM.

ACP, in partnership with the Guthy-Jackson Charitable Foundation, is seeking to enroll people with NMO into our repository. If you have been diagnosed with NMO and have not already enrolled in the ACP repository, we welcome your participation. Participation consists of a blood draw and an interview. This is not a treatment study. There are no drugs involved. If you do not live in close proximity to a collection site, funds may be available to offset travel expenses. Additionally, if you are unable to travel to one of ACP's collection sites, a nurse may be available to travel to your home or office to conduct the study visit on location. If you have been diagnosed with NMO, have not previously participated in the ACP repository, and have an interest in learning more, please contact the repository director as soon as possible for more information.

The TMA made a $20,000 contribution to ACP for the purpose of enrolling people into the repository with ADEM, NMO, ON and TM. The check was presented at a special ACP

dinner event that was held in November. Dr. Benjamin Greenberg represented the TMA and delivered a wonderful speech describing our Association and the importance of our relationship with ACP.

We continue to enroll new subjects at all of the collection sites. To learn more about participating in the repository, contact the study coordinator at the site of interest; call ACP's repository director, Sara Loud, at (781)487-0032, or visit the repository section of the ACP website at ***www.acceleratedcure.org/repository.***

Thank you for your willingness to get involved!

Repository Collection Sites

Barrow Neurological Institute
500 W. Thomas Road, Suite 300
Phoenix, AZ 85013
Principal investigator: Denise Campagnolo, MD
Study coordinator: Breanna Bullock
Study coordinator phone: (602)406-6211
acp-study-barrow0807@acceleratedcure.org

Beth Israel Deaconess Medical Center
Multiple Sclerosis Center
330 Brookline Ave KS 211
Boston, MA 02215
Principal investigator: R. Phillip Kinkel, MD
Study coordinator: Sarah Konkel
Study coordinator phone: (617)667-3726
acp-study-director0807@acceleratedcure.org

Johns Hopkins School of Medicine
Department of Neurology
600 North Wolfe Street Pathology #627
Baltimore, MD 21287
Principal investigator: Arun Venkatesan, MD
Study coordinator: Jana Goins
Study coordinator phone: (410)502-6160
acp-study-hopkins0807@acceleratedcure.org

Multiple Sclerosis Research Center of New York
521 West 57th Street, 4th Floor
New York, New York 10019
Principal investigator: Saud Sadiq, MD
Study coordinator: Lauren Puccio
Study coordinator phone: (212)265-8070
acp-study-msrcny0807@acceleratedcure.org

Shepherd Center, Inc.
2020 Peachtree Road NW
Atlanta, GA 30309
Principal investigator: Benjamin Thrower, MD
Study coordinator: Elizabeth Iski
Study coordinator phone: (404)350-3116
acp-study-shepherd0807@acceleratedcure.org

The Ohio State University Medical Center Multiple Sclerosis Center
1654 Upham Drive, 445 Means Hall
Columbus, OH 43210
Principal investigator: Michael Racke, MD
Study coordinator: Lisa Hafer
Study coordinator phone: (614)293-7877
acp-study-director0807@acceleratedcure.org

Anschutz Medical Campus, University of Colorado Rocky Mountain MS Center
Aurora, Colorado 80045
Principal investigator: Tim Vollmer, MD
Study coordinator: Sydni Edwards
Study coordinator phone: (303)724-2197
acp-study-ucdenver0807@acceleratedcure.org

University of Massachusetts Medical School Multiple Sclerosis Center
Memorial Campus, 119 Belmont Street Jacquith Ground
Worcester, MA 01605
Principal investigator: Peter Riskind, MD
Study coordinator: Janice Weaver
Study coordinator phone: (508)793-6562
acp-study-umass0807@acceleratedcure.org

Multiple Sclerosis Clinical Center
University of Texas Southwestern Medical Center
5323 Harry Hines Boulevard
Dallas, TX 76051
Principal investigator: Elliot Frohman, MD
Study coordinator: Gina Remington
Study coordinator phone: (214)645-8800
acp-study-utsw0807@acceleratedcure.org

Accelerated Cure Project for Multiple Sclerosis
300 Fifth Avenue
Waltham, MA 02451
Repository director: Sara Loud
Repository director phone: (781)487-0032
acp-study-director0807@acceleratedcure.org

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

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

The centers are working together to:
* Improve evaluation and management strategies to enhance diagnosis and care of children with MS and other related disorders;

* Develop resources for families, health care professionals and the public;

* Collect data that will enable large scale research initiatives

Each Center Offers:
*The latest in comprehensive care and treatment for children with these central nervous system demyelinating disorders, as well as the information and support their families need.

* Evaluation and diagnosis involving both pediatric and adult neurologists.

* A team of professionals that offers:

  * Nursing services;
  * Cognitive and psychological Evaluation;
  * Rehabilitation assessment (physical, occupational, speech and language);
  * Vision care;
  * Neuroimaging (MRI);
  * Individual case management and social services to ensure proper care and support;
  * Information and resources for patients and families;
  * School support.

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

for travel and accommodations according to need.

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

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

The Centers:

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

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

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

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

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

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

A National Paralysis and Physical Disability Quality Of Life Action Plan; The Christopher and Dana Reeve Paralysis Act of 2009

The fourth meeting of the Paralysis Task Force of the Christopher and Dana Reeve Foundation was convened in Washington DC, July 21-23, 2009. The task force is composed of a large number of federal and state public health agencies, academic public health specialists, and numerous individuals and organizations involved in health advocacy in the paralysis community. The Congress passed and the President signed into law the Christopher and Dana Reeve Paralysis Act of 2009. The new law mandates the development of "...a national paralysis and physical disability quality of life action plan that promotes health and wellness in order to enhance full participation, independent living, self-sufficiency and equality of opportunity for people with paralysis and other physical disabilities." A primary purpose of the task force meeting was to develop recommendations which would form the basis of the national action plan for paralysis and physical disability. A second goal of the task force meeting involved the population survey that was conducted by the Christopher and Dana Reeve Foundation. The results of this survey research were presented in the TMA Newsletter Volume 9 Issue 1. The workshop included two days of intensive discussions focused on health promotion, care giving, and multicultural outreach. The fourth area of focus was the development of a plan to continue research into the magnitude and composition of the paralysis population in the United States. The meeting organizers were presented with a report at the conclusion of the meeting enumerating the findings and recommendations that were developed during the workshops in each of the subject areas. This information is currently being developed into a draft of the National Paralysis and Physical Disability Quality of Life Action Plan which will be shared with the task force for comment before being finalized.

Cody and Shelley Unser and Pauline and I serve on the Paralysis Task Force. People with acute disseminated encephalomyelitis, neuromyelitis optica and transverse myelitis and their care givers were very well represented in the discussions. We are hopeful that our issues and the issues of the broader paralysis community will be significantly enhanced through the various policies and programs that will be implemented through this plan.

GUTHY JACKSON Charitable Foundation

NMO Roundtable Conference
November 9 – 11, 2009

Pauline and I were honored to be invited to the second NMO Roundtable Conference in Beverly Hills, California. Victoria Jackson and Bill Guthy established the Foundation shortly after their daughter was diagnosed with NMO. As with everyone who receives one of the rare neuroimmunologic disorders diagnoses, they had never heard of NMO and they were entirely confused, frustrated and anxious about their daughter's condition and her future. But unlike most people who get this diagnosis, the Guthy-Jackson family has the resources and the experience to change the future of science

and medicine in regards to our understanding of NMO and the development of effective treatments for this disorder. Beginning just over a year ago, the Foundation began to assemble the leading neuroimmunologic disorder specialists and researchers in the world. The Guthy-Jackson Foundation is funding research at some of the most prestigious medical centers across the country. The Foundation has also created an exceptional leadership and organizational structure that is guiding the directions of the research. They also announced the establishment of a multi-centered consortium that will be funded by the Foundation. The Foundation is committed to scientific collaboration and to the rapid translation of research into clinical care and effective and safe treatments.

During the first two days of the conference, scientists from across the country and around the world presented the results of their research. The studies are amazingly varied, but all of the work is focused on finding ways to stop the inflammatory episodes and to ultimately find a cure for neuromyelitis optica. For the third day of the conference, more than thirty people with NMO were invited to a session, along with their caregivers, where they heard presentations from the leading NMO clinicians. The program included a discussion of the incidence and prevalence of NMO, the diagnostic criteria, the symptoms of the disorder and some of the symptom management strategies. There were also presentations about the immune-suppressant therapies for NMO. The patient session concluded with a question and answer program. Patients and caregivers were provided the opportunity to engage in an exchange with the experts for the entire afternoon. During this session, Dr. Brian Weinshenker offered a very touching and well deserved acknowledgement of the work of Grace Mitchell who leads the Devics Support Network. Grace has tirelessly offered support and information to people who have NMO and participate in this internet community. Everyone in the room appreciated Dr. Weinshenker's wonderful gesture in recognizing Gracie.

Hollie Schmidt from the Accelerated Cure Project described the history and purpose of the ACP repository. Through the efforts of Dr. Benjamin Greenberg and his staff from the University of Texas Southwestern, approximately 20 people with NMO were enrolled into the repository during the conference. The patient session was web cast around the world to people with NMO who were not able to attend.

The Foundation has committed to including the patient sessions as an important part of the NMO Roundtable Conference. The Foundation has also developed a portion of their web site, entitled Spectrum, which they are hoping will become the vehicle through which people with NMO can network, as well as seek support and information about their disorder.

The Transverse Myelitis Association

The James Timothy Lubin Fellowship in Rare Neuroimmunologic Disorders

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

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

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

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

program, the mentor must also be a scientist with research experience and publications in these rare disorders.

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

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

VICTORY JUNCTION GANG

Founded for kids in honor of Adam Petty

August 12-16, 2009
TMA Family Camp
Fabulous 50's Week

Sue Mattis
Sue has TM and is a leader of the Pennsylvania Support Group. She volunteered to come to our camp this summer and served as a crew chief.

Victory Junction! How do you feel! We feel soooooooo good!

Do you ever feel so good about something that you just have to tell someone about it? That's how I feel about my experience as a Crew Chief at Victory Junction Gang Camp this summer. It was ***our kids'*** turn to experience what fun camp can be and just how good they could feel about themselves; maybe for the first time in their lives. For those five days, they could be just kids having fun with other kids; and, of course, with us 'big kids' too!

WOW! What an absolutely incredible facility. Kyle and Pattie Petty have made the dream of their son Adam into a reality. It is far more than what I could have imagined it to be. This is the place where children are able to play and have fun and be themselves regardless of their disabilities. They have every right to be able to experience these things in their lives and because of the generosity of the Petty family, the NASCAR community, and many others who support the Victory Junction Gang Camp, they are able to do just that.

The staff at Victory Junction is the most kind and caring group of individuals you could ever meet. They have such incredible energy, drive, compassion and love for the children who come to camp. The medical staff is second to none and makes sure that all the campers are safe and healthy. Dr. Peter Sim is the Director of the medical staff and is truly a gifted and big-hearted man.

Life in the day of a camper at VJGC is invigorating and exciting. Pole Position Activities were from 7:30 – 8:30 A.M. While these were optional activities, very few wanted to miss out even at this early hour. Campers were up and at em' to participate in mini golf, boating, fishing, the Fab Shop (a fun version of a beauty shop) or Adam's Race Shop (a virtual NASCAR building). Breakfast began in the ***Fuel Stop*** at 8:30; music and dancing followed to get your blood pumping for the day's activities. Two time periods from 9:30 – 10:45 and then from 11:00 – 12:15 gave the campers and their families choices again of the early morning activities, as well as arts and crafts, the waterpark, archery, bowling, the maze, horseback riding or play in the gym. These activities got you geared up for another great meal at the ***Fuel Stop*** at 12:30. **Are you tired yet**? If so, you have time to recharge from 1:30 – 3:00 by watching a movie in the theatre or retreating to your cabin for a little R & R. Back to activity choices from 3:00 – 6:00 and then another time to refuel our bodies with dinner. Each evening after dinner there was something special planned for all the campers and families. Thursday night: ***NASCAR-NIVAL***! How about two race teams who had dinner with the campers then demonstrated their own pit stop with a real race car and changed all four tires in under 20 seconds! How about hot

air balloon rides, popcorn, donuts, games and a visit from Miss North Carolina! Friday night: ***THE CUCUMBER 500***! Is it possible to hollow out a cucumber, make it sea worthy and race it down the Lazy River? Absolutely! Saturday night: ***STAGE NIGHT***! I have to admit that I never saw such an incredibly talented group of campers in my life! We had robots, singers, dancers, joke tellers, comedy routines and musicians just to name a few. An evening of fabulous entertainment!

There was so much to do and so much fun to be had at camp, but I also feel the need to address another aspect of the TMA camp at Victory Junction. Because of the love and commitment that Dr. Kerr, Dr. Kaplin, Dr. Pidcock, Dr. Greenberg and Janet Dean have for their patients and their families, they came to camp with their families and made themselves available to all in attendance for educational programming, as well as informal consultations in regards to medical issues. And truth be told, no one had more fun at camp than these people did! These doctors proved again to our TMA community how dedicated they are to treating their patient's needs and addressing any issues they are facing medically and emotionally. How blessed we are to have this team behind us!

On an individual family basis, it is hard to know exactly what their feelings were about the VJGC experience. I can share my observations. I can't think of anything that could have made these children happier than the experiences they had at camp. Many of these young folks were given the opportunity to do things for the very first time just because the facility is set up to allow them to do so in spite of any disabilities. Being in a wheelchair, or using crutches or a cane doesn't interfere with participation in activities at camp. The children aren't left out of any activity. They are part of everything that happens. No exceptions. You can socialize with others your age who are dealing with the same mobility and medical issues. You are no longer different. You ***belong***. They understand you and you understand them. You can be yourself. Siblings find common ground with siblings in other families. They understand what it is like to deal with someone else in the family seeming to get most of the attention. They can understand what it is like to feel like your family isn't a "normal" family. They understand your feelings, because they have those feelings themselves. What a unique opportunity it is for parents to be able to share with other parents what they deal with on a daily basis as being caregiver to a child with TM or ADEM or NMO. They can talk with other parents and discuss everything from medical issues to emotional issues and what the future holds in store for their children. Where else could you find such a bonding of families than at this camp? We need to hold hope in our hearts that our families will be given this chance to return to Victory Junction Gang Camp.

Barbara Sattler
Barbara has TM and is the leader of the Arizona Support Group. She volunteered to come to our camp this summer and served as a crew chief.

"This place almost makes it worth having TM." I heard this from an eleven year old camper this summer.

I was lucky enough to be able to volunteer at Victory Junction Gang Camp. I had heard about camp from the newsletter and decided I wanted to go. I have to admit my primary motivation was to have a chance to meet Sandy, Pauline and Debbie and to talk with others who had TM.

My first surprise was the application process. I had recently retired as a judge and have TM, so I assumed they would just take me. Instead, to apply, you have to go through a thorough selection process, including a comprehensive application, references, which are checked, pass a background check, a health and medical review and personal interview.

My second surprise was the facility itself. I had been to summer camp before and had an idea of what to expect. Wrong again. The facility is built around a race car lap and the various buildings have model race cars, racing posters, and other references to auto racing. The camp activities include bowling, archery, horseback riding, a water park, fishing, boating, crafts and wood shop. All the activities are accessible to individuals in wheelchairs. Attendance at camp is free to campers and their families.

The most significant difference from my experiences at summer camps was the attitude of every staff member. Each and every one of them is there to make sure the campers and their families have a terrific time. Several times each day, I saw a staff person reach out to motivate a camper, include a camper or compliment a camper on how well they were doing in an activity. I met an older man who was recruited for one weekend five years ago to work in the kitchen, and he came back every year. He told me, "I was wasting away in retirement until I found this camp."

It began as a dream of a young man, Adam Petty, the son of Kyle and grandson of Richard Petty. Most of you probably know Kyle and Richard from their NASCAR fame, but you may be surprised about their charitable accomplishments. Adam, as a child, accompanied his parents to present charitable donations to hospitals and also to visit children. Seeing kids in the hospital unable to do what he could, he conceived of building a camp that was specifically for disabled and sick kids. When he grew older

and started racing himself, he got a loan on his future earnings to build the camp. Unfortunately, at age 18 he died in a practice race. Knowing this was their son's dream, Kyle and Pattie Petty built the camp in honor of their son and have been raising funds to keep it going. It is one of the NASCAR charities.

Mealtime is really special at camp. After each meal, some of the volunteers lead the group in dancing and singing and quickly everyone picks up a few special cheers. While I tended to watch, it was clear to me that this was a very special time for dancers and watchers alike, as it set the mood of togetherness and warmth. You don't have to be either coordinated or have two working arms or legs to be part of the fun.

Included as part of camp staff were the medical people from Johns Hopkins, the University of Texas Southwestern, and Kennedy Krieger Institute. They were not only available for consults to the campers and their families, but participated in daily activities and presented medical lectures. Not only are these doctors brilliant and compassionate, they are great fun. I have no doubt, if awards were handed out, several would have been campers of the week.

As if all of this wasn't enough fun, one night I emerged after dinner to see camp transformed with the presence of two hot air balloons, tattoo booth, singers, a Krispy Crème donut booth, and a NASCAR team racing a car around the lap and then showing the group pit stops.

While the activities were great, for me, the best part of camp was meeting people. I had heard and read about Sandy and Pauline for years. I knew they had to be inspiring, but I was unprepared for how inadequately that term describes them. All I can say is both of them are awesome. Besides being knowledgeable, compassionate and hard working, they are great fun. As an added bonus, Pauline has the most wonderful service dog on the planet, Kazu, who showed us his skills on talent night.

My job at camp was called crew chief. I was assigned to one family to make sure they knew what activities were available, to help out if they needed something and to help them have fun. I was so fortunate to have been assigned to the Harrington family; 21 year old Ashley, who contracted ADEM when she was 19, and her parent's, Jeff and Mary. I heard about Ashley before I met her and knew she was in a wheelchair and couldn't speak. I was concerned that I wouldn't be able to have a relationship with her or be of any assistance. Like many fears, it was unfounded. Because of Ashley's outlook and wonderful smile, it was easy to relate to, connect to and communicate with her. She was willing to try almost everything, although her favorite place was the fab shop which is a combination hair, nail and make-up salon that we visited often. We even acted in a skit together. Because of her attitude, Ashley was clearly a favorite of everyone and was an inspiration to me. It's clear she got many of her great qualities from Jeff and Mary.

While I am sure the campers had a great time, it would be difficult to decide whether they benefit more from camp than the volunteers. You can't help being moved by the courage of the campers and their families and the dedication of the doctors and staff. I know this experience has made me a better person. I would encourage you to apply to be a volunteer at the next TMA retreat weekend or family camp. While it is a wonderful way to give something back to our community, it is also a great way to do something incredible for yourself.

Visit to the TM and NMO Center at the University of Texas Southwestern and the TMA Planning Meeting

Dr. Benjamin Greenberg established the TM and NMO Center at the University of Texas Southwestern. When he moved from Johns Hopkins, Maureen Mealy, Megan Quigg and Stephanie Taylor joined him in Texas. Maureen is his nurse and is involved in Dr. Greenberg's research projects, as are Megan and Stephanie. Megan and Stephanie are also in the process of applying to medical school, and I am already promoting the many virtues of neuroimmunology.

I suggested to the officers that it would be wonderful to make an official visit to the new center, and that we should invite Dr. Douglas Kerr to join us and hold a TMA planning meeting while in Dallas. Dr. Kerr, Paula Lazzeri, her son, Jesse, Pauline, Kazu and I arrived on Thursday, July 9th. We made our official visit to Dr. Greenberg's center on July 10th and also held a planning meeting at the center. Our visit continued throughout the day on Saturday. It was a great meeting and a wonderful visit. We accomplished so much and we had a great time. Our discussions about issues that are central to our Association and our members went on all day and night for three days. These discussions took place in the meeting room at the TM and NMO Center, but they also took place at the Dallas Aquarium, while driving all over the city in cars, trucks and vans, in Ben's and Tasha's living room, kitchen and swimming pool, in hotel rooms, at restaurants and at Billy Bob's Bar and Rodeo.

We planned the education program that was to take place at the TMA Family Camp in August at Victory Junction Gang Camp. I had solicited topics from the parents during the summer that they would like addressed at the education sessions and I pre-

sented these to Drs. Kerr and Greenberg. We discussed the subjects that would be covered by Drs. Kerr, Greenberg, Kaplin, Pidcock and Janet Dean during the three day morning program at camp. In addition to the three two-hour sessions, Drs. Greenberg and Kerr suggested that Janet and the doctors offer to hold informal consultations in the afternoons during the rest times. This turned out to be a wonderful idea and almost all of the families took advantage of this opportunity during our camp.

We discussed plans to hold the next rare neuroimmunologic symposium in Dallas in 2010. Dr. Greenberg and the University of Texas Southwestern offered to take a primary role in the organization and planning of this program. We also discussed including patient education and research and science components to the program as we have done in the past with Johns Hopkins in Baltimore.

We talked about the James T. Lubin Fellowship and how we would raise funds for and structure the fellowship. We are all committed to growing the discipline and attracting TM-NMO-ADEM clinicians and scientists.

We focused the largest amount of time and energy on discussing ways to further research into transverse myelitis and acute disseminated encephalomyelitis. Due to the rarity of these disorders and the wide geographic distribution of cases, research might best be accelerated by establishing a consortium of physicians and medical centers to increase the numbers of patients being seen with these disorders and to conduct multi-centered studies. The development of a multi-centered consortium would make possible research into the development of biomarkers and other ways to define these disorders so that we can better understand what might be causing them, i.e., what is the specific mechanism of disease. These biomarkers would also make it possible to more rapidly diagnose these disorders and develop ways to measure the effectiveness of treatments. Multi-centered studies would produce evidenced-based, scientific results that could guide physicians in their acute therapy decisions.

I have been working with Maureen to compile a list of physicians who have experience treating TM or ADEM or NMO during the acute attack and also who have a clinical practice that can treat the many challenging symptoms of these disorders. This has been a tremendously difficult task as our members are located across all of the United States and including some very rural areas of our country. I am regularly contacted by people who are looking for a physician who has an understanding of these disorders. Our primary objective is to identify

physicians who have the experience to treat the acute episodes. We continued our discussions at the meeting to further develop this list.

We discussed the future of the TMA and how to sustain our organization and grow our opportunities into the future. This was the most complex focus of our meetings as much of these discussions concern the transition of the Association from an organization composed entirely of volunteers to one in which people are paid to do some of the work. While no decisions came out of these discussions, it is clear to all of us that we must sustain our organization; regardless of what happens to the people who are currently doing

this work. It is also abundantly clear to everyone that all of the goals we plan to accomplish are going to require that we raise the funds to pay for these programs, and that this will require a level of fundraising that the TMA has not yet managed in our 15 years of existence.

The weekend was incredibly productive. Ben and Doug are so devoted to our organization and to our community. We are thrilled with the establishment of the new TM and NMO Center and we are so grateful to have both Dr. Greenberg and Dr. Kerr thoroughly dedicated to our cause.

National Family Caregivers Month, celebrated every November, is a nationally recognized time to thank, support, educate and empower more than 50 million family caregivers across the country currently providing over $375 billion in "free" caregiving services. Family caregivers provide more than 80% of all home care services.

The TMA is pleased to be an official endorsing organization of NFC Month, an annual event created by the National Family Caregivers Association to bring attention to the needs of family caregivers. "We encourage people to speak up during National Family Caregivers Month," said Suzanne Mintz, NFCA president and CEO. The TMA is pleased to have this opportunity to formally recognize and celebrate the valuable role family caregivers play in our healthcare system.

NFCA is a national nonprofit organization dedicated to empowering family caregivers to act on behalf of themselves and their loved ones, and to remove barriers to their health and wellbeing. NFCA concentrates its efforts in three primary areas: education, building community, and advocacy. For more information, visit www.thefamilycaregiver.org or call (800) 896-3650.

Children's Database

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

1. To develop a contact list that will be used by the TMA to notify and recruit families and older teens and young adults for the family camps and the older teen/young adult retreat opportunities, such as those that are held at Victory Junction Gang Camp;

2. To develop a contact list to recruit for pediatric studies and clinical trials related to TM/NMO/ADEM/ON; and

3. To develop a directory that can be used by TM/NMO/ADEM/ON families to share information and support between families in similar situations.

This project is being directed by Linda Malecky. Linda's daughter contracted TM at the age of two in 1999.

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

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

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

1. The information provided will not be incorporated in the TMA website in any way;

2. Your family will only be included in the directory at your request;

3. The directory will be published and mailed **only** to members who agree to be included in the directory;

4. Only the following information from the data base will be included in the directory:

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

The TMA believes that it is extremely important for families (including the children with TM/NMO/ADEM/ON) to be able to find other families and children for information and peer support, which is why we are collecting information for a directory. However, even with the limited information and distribution we are proposing for the

directory, we realize that you or your children, now or in the future, may be concerned about being identified as someone with TM/NMO/ADEM/ON. We will only include those families who specifically indicate that they want to be included in a directory. **Please provide the data base information regardless of whether you want to be included in the directory or not.** This will ensure that you are contacted when camp or retreat opportunities arise or if there are studies or trials available that may help your child.

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

If you would like to participate, please send your information to Linda Malecky via email:

lamalecky@verizon.net

If you do not have internet access, you can send Linda the information via the postal service: 107 Tweed Way, Harleysville, PA, 19438.

When you send us your information, please make it clear as to whether you would like to have your information listed in the pediatric TMA directory.

If you have any questions or concerns about the project, feel free to call Linda (215-855-3488) or myself (614-766-1806).

We have tried to identify as many children as possible in our community, and Linda has attempted to reach many of you via emails to request this information. We believe that this project will help us better serve the families in our community by making you aware of important opportunities and by facilitating a support network for our families. We are grateful to Linda for her willingness to make this critically important project possible.

Allen Rucker

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

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

As a TV writer-producer, he co-founded the experimental video group, TVTV, and has written numerous network specials, documentaries, and teleplays, including the award-winning cable series, "The History of White People in America," with Martin Mull.

He is the recipient of the duPont-Columbia Journalism Award; two Writers Guild Awards, including one for career distinction as a writer with a disability; two CableACE Awards; and two retrospectives at the Paley Center for Media. He is a contributing editor to "New Mobility" magazine, the chair of the WGA Writers with Disabilities Committee, and a frequent public speaker. He lives in LA with his wife, Ann-Marie. They have two sons.

The Illusion of Control

How much does chance rule our lives? How much does a mysterious confluence of forces that we don't know about, can't anticipate, and have no control over actually dictate how we live?

The answer: probably much, much more than any of us realize.

This is the subject of a fascinating book of a year ago called, "The Drunkard's Walk: How Randomness Rules Our Lives," by a Cal Tech physicist and excellent narrative writer named Leonard Mlodinow. The book uses both real-life anecdotes and the science of probability to argue that, "a lot of what happens to us…is as much the result of random factors as the result of skill, preparedness, or hard work." One reasonably talented actor, for instance, comes to Hollywood, works his tail off for years, and never makes it past a guest role as a dope dealer on "Law and Order." Another reasonably talented actor stumbles into the right audition at the right time and becomes Bruce Willis. We all think that actor #1 is a near-failure because "he didn't have what it takes" and that Bruce Willis must be extraordinarily talented or determined or something to rise so high. We think that Mr. Willis simply took control of his life and made stardom happen.

This is what Mlodinow pegs as "the illusion of control," an illusion we all practice every day in our own lives. He quotes Harvard psychologist Ellen Langer to the effect that, "while people may pay lip service to the concept of chance, they behave as though chance events are subject to control." This is especially true when viewing things in retrospect and when we are assessing our own lives. When things go wrong, in other words, we blame ourselves. We messed up. We should have worked harder, taken more risk,

taken less risk, gone to grad school, not gone to grad school – we create a litany of our own bone-headed missteps and failed expectations to explain our lives.

It's tough to give Chance its due. We love to think things happen for a reason, as in, "Everything happens for a reason, you know," and when we can't come up with one, we often look up and either praise or blame G-d Almighty. Embracing the idea of randomness seems like a cop-out, a ready answer for any failure or shortcoming. "Hey, I didn't ***pick*** *my mother! She was a whiny, controlling you-know-what before I came along!" To simply say that "s**t happens" is the siren cry of the perpetual loser, a person incapable of taking responsibility for his own actions and their consequences.*

And we extend our need for hard answers to the rest of the world. When we see someone else downed by life, like poor people, we intuitively connect their poverty with something they did or didn't do. Our almost patriotic belief that we all can control our own destiny leads to some awful slurs. "Why don't they just get a job? Learn English? Go to college? Move to a better neighborhood?"

When I was first diagnosed with TM thirteen years ago and gently told I was paralyzed for good, I ***knew*** *it was something I had brought on myself. I knew there was a pattern of behavior, or thinking, or both, that had opened the door for TM to come strolling through. I mean, really, how could something as nebulous as "chance" or "bad luck" deal me such a low blow? It seemed so impersonal and so cruel. It was more pathetic than tragic. It made me feel like that poor random house fly that the school kid decides to pull the wings off of. There is a definite explanation here – the callous school kid -- but the fly doesn't know that. To him it's just a simple, life-ending twist of fate.*

The Western-trained doctors treating me generally scoffed at the notion that I was in any way responsible for contracting TM, but a Chinese acupuncturist clearly saw a deep-seated karmic pattern. He said that, upon examining my broken body, that I had "too much wind and too much wetness." This meant, in his view, that I had not let my immune system function properly – I never allowed myself to get sick, for instance – and my system finally snapped, as it were, and caused the paralysis. Self-induced stress, in other words, is what brought this on.

Emotionally, I tend to believe that to this day. It gives me something to hang on to. I wasn't just a bug that flew into the fast moving windshield of life. I played some part in this drama. But, unfortunately, there is no scientific basis to this notion that I know of. And if stress played a factor, there was still a lot of randomness involved. There are a lot of stressed-out people out there and most end up with an ulcer or a drinking problem. Few end up wheeling to work.

I pray that, some day, the people at Johns Hopkins or elsewhere will link TM to something observable and definable – elevated IL-6 antigen levels, or an errant genetic marker, or a traumatic stress reaction, or too much pork fat in the diet – so I could close the book on an explanation and hopefully, remove my own character flaws from the equation. But, most likely, until that happens, I will keep trying to find the "real reason" I'm paralyzed, invariably something I invited and could have avoided.

But I'm working on giving Chance a chance, and I've found that it if you take it seriously as at least a contributing factor in your life, it can be liberating. For one thing, it's ego-deflating, and that's a good thing. You don't yell "Why me!?" every time something bad happens and you don't think you are the king of the world and become an insufferable bore when good fortune comes your way. "You know why they made me Salesman of the Year? I'll tell you why they made me Salesman of the Year…" You'll be less quick to judge others for their perceived failings. You learn humility.

As you begin to see that your malady is an unfortunate biological blimp, and not a punishment for past sins or a true reflection of who "you" are, you tend to give it much less weight in your life. It is no more a precursor or determiner of your life ahead than male-pattern baldness or thick ankles. You are as free to invent your future life in a wheelchair, and roll with all the turns of fortune and misfortune, as you are on two good legs. And keep in mind, if chance played a role in the TM, it probably also lead to some of the many good things in your life – your family, friends, and even the TMA community who is there to support you.

*Chance or no chance, you are still responsible for how you deal with your own circumstances, like tending to your own health, both physical and mental. And Mlodinow of "The Drunkard's Walk" sees the way to move ahead. "One important factor in success," he says, "****is*** *under our control: the number of at bats, the number of chances taken, the number of opportunities." In other words, if you continue engaging with the world, the odds are that something will eventually mesh in some magical way and being in a wheelchair or otherwise impaired will become an afterthought in your life and not the focus.*

In Their Own Words

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

Charlie Green
Ridgeville SC

Hello everyone. Hope everyone is doing well. My name is Charlie Green and I live in Ridgeville, SC. In 1988, I was a fully active boy. I played sports every chance I got and loved hunting and fishing. Up to that point, I was never sick except for a cold here and there. On June 12, my life changed dramatically. I was out of school and went fishing that morning with some friends. That afternoon, I had baseball practice. After warming up and stretching, I went into the dugout and was waiting for the rest of the team to finish loosening up. That's when it hit me.

The first thing that happened to me was the worst pain I have ever felt in my life. My chest felt like it was going to explode. From there, I remember holding out my arms, but not being able to move my fingers. I was able to walk to my father's truck and he rushed me to the emergency room. By the time we made it to the hospital, 15 minutes away, I was completely paralyzed. My whole body shut down and I had to be put on a ventilator.

Three days later, I was transferred to MUSC Children's Hospital. Three weeks later, I was diagnosed with TM. The level was C2 on my spinal cord. I spent three months there with the ability to move only my head and one of my thumbs. I was still dependant on a ventilator. I was then transferred to Shepherd Spinal Center in Atlanta. Within one week, they had me off the ventilator and in a power wheelchair. After three months of PT, I recovered some use of my arms and all of my feeling throughout my body.

I came home and started back to school and continued with my life. I have to say, I have the best family and friends any person could ask for. They have always been beside me and have supported me throughout my life and continue to do so. I went to college and have a degree in Auto-CAD, which is computer aided design. I have been married and now divorced. I have a six year old son. Oddly enough, he was born on June 12th, the same day I became paralyzed. He is fully healthy and the light in every day of my life. I have custody of my son, and he just finished his first year of school and t-ball. I am currently in a relationship with a wonderful woman. We have been together almost two years and she stands behind me 100%.

Since being stricken with TM, I have encountered many obstacles on my life's journey, but none have yet kept me down. My philosophy is, don't focus on what you can't do; focus on what you can do and do it to the best of your ability.

My Daughter, Megan
Lisa Hoersten
Delphos, Ohio

My daughter, Megan, was 10 months old when she was diagnosed with Transverse Myelitis.

It all started on a summer day in June of 2007. One day she was a happy, healthy little girl, and within hours, she was totally paralyzed from her legs down. Her arms were weak and all she would want to do is lie around and be held. We assumed she was constipated from some of the signs we saw. Finally, when she wasn't getting any stronger, we called the doctor. When we called that night, the doctor was not really worried, because she was eating and still drinking. The first thing the next morning, we went to see the doctor. She never hesitated and sent us directly to the ER.

Megan was like a newborn all over again. She could hardly move her arms. It seemed like it took everything she had to hold her head up. My poor little girl was helpless and we had no idea what was wrong with her.

We were at the ER for seven hours. They were running all kinds of tests and were finding nothing. As a parent, that was really frustrating and our emotions were starting to unravel. I am her protector and they are not finding what is wrong with her. I was getting short with the doctors. You want answers now, and they did not have any answers. I have to say, first of all, that I do not have a lot of patience, so being in a situation like this was really difficult.

Finally, we decided to go to a different medical system. So, we headed to Children's Hospital in Columbus, Ohio. It is about two hours away, but it was worth it. We got there and they put her in NICU so that they could observe her more closely. On Saturday, they ran a test on her that ruled a few things out and on Sunday (Father's Day) they diagnosed her with TM.

It was a relief, in some sense, just knowing what she had so that now they could fix it. We soon found out that it was not that easily fixed. We had a long road ahead of us. It has been a road on which we've learned to rely on each other, all of our friends and family; and a road on which Megan's mother has learned some patience.

It has been a year since the onset of TM. Today, Megan is a healthy two year old. She is completely off all of her meds. She is up to par on her occupational therapy, so she is no longer doing that and her Physical Therapy is going well. She is walking with her walker and getting stronger everyday. It also helps that she has her older brother to keep up with.
We look back at pictures we took while she was in the hospital, and it is hard to believe that she has made so much progress within one year. I thank G-d everyday for having given us the strength we've needed to get through this experience. Our road to recovery is far from over as Megan continues to progress. She is a very determined little girl and I thank G-d for that everyday.

Normal

Rob Pall

Normal ... what a simple word! This is a word I took for granted until one week past my 50th birthday. What did normal mean to me? Normal meant being like everyone else; it was being able to run, walk, exercise, climb up stairs, play sports; basically, being able to do everything that "normal" people do without giving it a thought. I guess I figured, as I got older, I would slow down a little; maybe replace basketball with golf. Perhaps I would have to exercise a little longer to stay in shape. No big deal … this was "normal." Sure, like all normal people, I would get sick from time to time and maybe break a bone or two, but I always knew that I would get better. And until one week after my 50th birthday, that was just how life was … normal.

Now, let us go back 11 or so years where in a period of several hours, I went from "normal" to cripple. In a few hours, I had zero feeling from my waist down. That can't be possible. I had played ball all weekend. There had to be a simple answer; maybe a pinched nerve or something like that. The idea that I would never be normal again never crossed my mind. I was sure it would be just a matter of time until I was all better … and "normal again." Even after three MRI's and two lumbar punctures, I was certain that Dr. House would figure out the problem, give me some medicine and I would be all better. I would be normal again … just like everybody else I knew.

Three weeks after being crippled from the waist down, I was told what I have … Transverse Myelitis. What the heck is that? I never heard the words before and had no idea of their meaning. The top neurologist at New York City Hospital explained it to me. He said he was sure I would eventually walk again, but he could not say for sure what assistance I would need. Perhaps a walker (how embarrassing), maybe a quad cane (better but not great) and if I was lucky, perhaps, I could graduate to a straight cane (better but not normal).

After spending three weeks in the hospital, I was transferred to the Kessler Rehabilitation facility in West Orange, New Jersey (the same place where Christopher Reeve did his rehab). Slowly, over a period of three months, I started getting a little better. I went from a walker to a quad cane to, upon leaving the rehab center, a straight cane. I was surely getting better. I would prove all of the doctors wrong. Oh, just one little side note. While it was true that I was learning to walk better, there also came some small side effects. When I first came down with TM, I had no feeling and, therefore, I had no pain or discomfort. But, as some feeling came back, these feelings were so so bad. Where previously I felt nothing, now one of my legs was pins and needles and numb (how is that possible?) Whereas my other leg was numb with excessive banding (tightness) which caused me to walk with a "stiff leg." But at least I was walking and it seemed that I was getting better everyday. Soon I would be all better … I would be normal.

The improvement was constant for the first six months. Then, it continued to a lesser extent over the next six months. Then, I just stopped improving. How can this be? I know, I just have to work harder at getting better; just keep exercising harder and longer. I was so determined to prove the medical profession wrong! But it turned out that they were right, and I was wrong. I hit a plateau where all I could accomplish was abnormal fatigue. I was not getting better and worse yet, I probably never would! However, I would keep all of the pain and discomforts probably for the rest of my life!

Now, for most people, walking with a limp and being in weird discomfort 24/7 would be bad enough, but not for me. For me, not being "normal" was the worst part of the condition. I did not want people to see me as crippled and feel sorry for me. So, I did my best to look normal; even though this hurt and fatigued me more. As far as

my friends and family were concerned, I had made such great strides in getting better. They could not see the unrelenting pain and discomfort that never went away. But I guess I was happy that they still thought of me as normal. After all, is that not what I wanted to act and be treated as - a normal person - and not someone to be pitied?

I don't know! I drive 40 miles each way to work in New York traffic. I work an eight to ten hour day and yet when I get home, my wife still doesn't understand why I am so tired. Just a couch potato! I suppose she just wants me to be normal. I know she tries to understand how I am and what my limitations are, but unless you walk in my shoes, how can one truly understand.

That is probably the main reason I have started the New Jersey Transverse Myelitis Support Group. We had our first meeting in the spring and I was amazed at how many of the attendees had never before met anyone else suffering from TM. It was both enlightening and emotional to be with other people who truly understood what I was saying and what they were saying. I was also amazed by how normal most of them looked. Until they started discussing the horrors in their lives, they looked and acted perfectly normal. In some cases, just from outward impressions, I was jealous; until they spoke, and in no uncertain terms, convinced me they were as bad, if not worse, than me.

Okay … I have come to see being "normal" somewhat differently. The people I have come into contact with in the TM community are probably more normal than most. We are a people who struggle with life, yet embrace it. Most of us do not let our condition define our lives. Instead, we value the little things that normal people take for granted as wonderful gifts.
Maybe, just maybe, being "normal" is overrated!

Untitled

I could beg but oh what for?
My performance convincing ... im not in pain anymore
Im alright just give me a breath
This feeling i have puts all to the test.

I woke up this morning what more can you ask?
I woke up last night and there it was burning quite bright.
I've practiced a face one that can hide this overwhelming feeling inside.

I cannot remember a painless day.
Either i have forgotten or wished them away.
It is a small hell to be trapped within.
This fire stilling roaring stoked time and again.

My soul grows weaker and my mind unclear
I give it my all and in this you must applaud
My performance convincing I'm not in pain anymore.

S. M. Holmes

Sabina Slavin
March 17, 2009
Contracting and Combating T M

On Thursday, October 2, 2008, I was on the treadmill in the morning, as I tried to be every day. In the afternoon, my left leg began to quiver. I went to a dinner party that night and almost fell on my way to the rest room (other guests probably thought that I was sloshed!). The next day, I went to see my PCP. The doctors were perplexed and sent me to the Emergency Room. It was a Friday afternoon, often the busiest day for an ER. A neurologist examined me, looking for a stroke, which I had not had. An x-ray was taken and I was told that I had arthritis in my back and should see an orthopedist.

The next day, I telephoned my PCP to report on my trip to the ER. She told me to come and see her on Monday morning and she would arrange for the orthopedist. But, if I got worse, to go to the ER again. Later in the day, I was unable to use either leg. My husband and I left the house to go to the ER, but I collapsed on the stairs. I told him to call 911. The ambulance arrived promptly and drove me the 12 miles to Danbury Hospital. Again, they checked for a stroke and found nothing. My left leg began to exhibit spasms and I was given an injection to ease my discomfort. I was admitted to the hospital about 10 PM and soon discovered that I could not urinate. A nurse inserted a Foley catheter and I was able to sleep.

At this point, let me say that I am a 67 year old female who has always been in excellent health except for arthritic knees. I swam, scuba-dived, traveled, did about two miles on the treadmill four or five times a week and enjoyed my life in retirement very much.

Danbury Hospital is a teaching hospital and the neurological service always has a neurologist in the house. They began diagnostic tests on Sunday morning. I had MRIs of the cervical and thoracic portions of the spine (with and without contrast) and the neurologist performed a spinal tap. Nothing was found that could have caused my problem.

On Monday, MRI's of the lumbar portion of the spine and the brain were done – again with and without contrast. The neurologist came to me at 2 PM and told me that I had transverse myelitis between T 10 and the first lumbar, L1. No sclerotic lesions were found in my brain. They did find an aneurysm, but they would deal with that later. Treatment with an IV infusion of 1 gram of Solu-Medrol (methylprednisolone) was begun within the hour and would be repeated for four more days. The doctor told

me that I might not see some improvement for two weeks and that he hoped that I would walk again. A side effect of the treatment with steroids was a dramatic elevation in my blood sugar. This was treated with insulin injections until the steroid treatment was finished.

There were numerous discussions as to the cause of my TM, but the conclusion was that it was idiopathic.

The next morning I could lift my legs about 6 inches off the bed! I was delighted and the doctors were amazed. A physical therapist came to see me and got me up into a chair. I also received a visit from an occupational therapist and I was told that I would be moved to a rehabilitation floor the next day. When I arrived there, I was given a tentative discharge date of October 29th. To make a long story short, I was discharged on October 22nd. By that time, I was able to walk with a walker and climb stairs using banisters on both sides. I continued to experience the bladder and bowel problems that other sufferers of TM have reported.

Our home has grab bars at the front and back doors (due to a previous illness incurred by my husband resulting in a bilateral craniotomy. Fortunately, he is fine now). We also have grab bars in the bathroom and the shower and our bedroom and bath are on the ground floor. That made readjustment to life at home easier and safer for me.

I cannot say enough about the quality of the care that I received at Danbury Hospital – from the very early diagnosis that I received through the excellent physical and occupational therapy that was given to me every day. The Hospital also has an out-patient rehabilitation center and I soon was receiving therapy there three times per week. I progressed to a cane and then to walking unassisted. They helped me to become more stable and improved my balance and helped to increase my strength. Now I only use a cane when I go to museums or walk outside in the snow.

A month after I was discharged, we began to address the aneurysm issue. My neurologist sent me to Dr. Philip Meyers, an interventional neurologist at Columbia Presbyterian Hospital in New York City. He advised an angiogram to determine the size and shape of the aneurysm to determine whether he could treat it with a coil technique or if it would need to be clipped. Immediately after the procedure, he told us that clipping was required and made arrangements for us to meet with Dr. Robert Solomon, Chairman of Neurosurgery at Columbia Presbyterian. Surgery was scheduled for December 31st to clip the aneurysm which was about 15 mm in size and oddly shaped. It was located at the bifurcation of the middle cerebral artery, but was not very deep. I was transferred from the operating room to the Intensive Care Unit after about six hours under anesthesia. The next day I got up and walked to the neurosurgery unit and the day after that I went home! Three days later, some 34 staples were removed from my 20-cm incision and I have recovered completely from the surgery! It was truly a miracle that the aneurysm was discovered by accident while the diagnosis was being sought on my TM. I had the surgery while I was healthy, and not as an emergency to repair a ruptured aneurysm.

So how am I today? I was "kicked out" of physical therapy last week and started today in the adjacent supervised gym. I am walking fairly easily, but I do get tired when I stand for a long time, as in a museum. Some days (like yesterday), I feel almost normal. Other days (like several days last week), I am quite uncomfortable. The bowel problem continues, but I am dealing with that. My husband and I went to Switzerland last month for two weeks to visit our daughter and her family who moved there in August of 2008. We had a great time! And next month, we are going to Bonaire for 12 days. We will snorkel, but scuba diving is out for at least six months. I am hoping to continue to improve. I did one mile on the tread mill yesterday in 25 minutes and 1.2 miles today in 30 minutes. As I get more stamina, I want to walk for a longer time. I will probably not go faster than three miles per hour, as I have osteoarthritis in both knees and that limits my walking speed.

I was so fortunate to be treated at Danbury Hospital. My doctors had seen a few cases of TM before, but very few, of course. The care was excellent. The support I received from my husband and family and friends was outstanding. People came to see me in the hospital and said that "I looked great!" I always replied that I was great from the hips up! Thanks also to Sandy Siegel for all of the helpful information that he provided about TM – something I had never heard of before.

If anyone wants to chat with me about my experiences, don't hesitate to contact me at (203)438-3258 or SabinaSlvn@aol.com. I would be delighted to be of any help that I can.

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Mazel Tov, Netta and Ilan!

Netta wrote about herself for the TMA Newsletter in July, 2000. It was a brief introduction to a couple of her beautiful paintings that were included in the Newsletter.

My name is Netta Ganor and I am a c-4/5 quad from Israel. I was struck down with Acute TM on November 25th, 1994, at the age of 15. It all started one Friday afternoon, after I

came back from school. After lunch, I suddenly started to feel a terrible sharp pain in my upper back (behind the shoulders). Gradually, but quite fast, in less than one hour, I lost sensation and the ability to move my hands and finally my legs. My mother immediately called an ambulance, which took me to the hospital. I was taken to the IC department and was diagnosed after 10 days with Acute TM at c-3. Of course, I was ventilated. After three months, more or less, I saw the first improvement; my left arm started to move. Gradually, in the next one and a half years, I got rid of the vent due to hard work in exercising my breathing muscles. After spending more than two and a half years in the rehab hospital, we all had to move to a new house, and that included leaving our city, Jerusalem, in order to be geographically closer to our family.

Today, I still have no sensation from the shoulders down, including my arms. I move the left arm without feeling it. I'm driving a motorized wheelchair, paint and write with my mouth, swim(!) with my head and shoulder movements and do things that people are not supposed to be able to do in my physical condition. I'm very optimistic as for my condition in the future. I don't know what will happen with me or when. I do know that others with TM have had improvements that are measured in years. I've never lost hope and I'm sure that someday in the near future the medicine will find the right cure to our syndrome.

In the meantime, I'm trying to do the maximum I can to make my life as normal as possible. I just don't see any point in sitting around the whole day and mourning. Currently, I'm a second year student for Computer Science at the College of Management in Rishon Letzion. I'm still a fan of sports (especially basketball) and stayed an athlete in my soul :-) I was a tennis player and an athlete in high school.

After one year of being disabled (back in 1994-5), and after watching some other quadriplegic people painting with their mouths, I decided to give it a try myself. It was very hard at first, but as the time went by and with a lot of practice, I learned how to paint this way.

Netta wrote the following in June 2009.

The Happiest Day of My Life

To tell you the truth, I almost stopped believing that this day would come. After years of searching, I almost gave up on love when I finally met Ilan.

We met virtually on an online dating website at the end of May 2006. I remember it was only two days before Shavuot, my favorite Jewish holiday,

and indeed there was something special in the air.

After quite a few failed relationships and dates with guys who couldn't deal with my disability, Ilan was a very unique guy in his own special way of looking at people and his ability to see beyond my disability. That's what made it work from the very beginning. After only a few weeks, it felt like we had known each other forever and my disability just "disappeared" to both of us from the very beginning. We went and still go on trips together, enjoy going out to the cinema, to restaurants, meeting friends and mostly just be with each other.

After one amazing year of being together, Ilan proposed to me and, of course, I accepted. It took us quite a lot of time to pull off our wedding; so much to plan, so much to think of. My wedding dress was the most difficult "project" to execute! But gradually, we figured it all out and had the most

fabulous wedding I could ever imagine – you can see in the pictures!

Now, our next challenge is to go on a trip abroad. I know we'll make it happen!

Netta is a Web Programmer at Applied Materials Israel, she loves designing websites, painting, watching movies, going out and watching sports. Netta loves animals and she loves all of life!

And we love you, Netta. We wish you and Ilan a long life together filled with GOOD HEALTH, happiness and peace!

Contacting the T M A by Email

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

We Don't Want to Lose You

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, 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.

The Association does all of our mailings using the postal service bulk, not-for-profit rate within the United States and our territories and protectorates. We save a considerable amount of money by doing our mailings in this fashion. Unfortunately, when you move and don't provide us with the change, our mail will not be forwarded to you, after your grace period, and this class of mail is not returned to the sender. The cost to the Association is substantial; the materials we are mailing to a bad address just ferment on some post office floor. These are wasted printing and postage costs. Please keep your information current. Your diligence is greatly appreciated.

Tips for those with TM and the other rare Neuroimmunologic Disorders: Patients Helping Patients

Barbara Sattler bsattler@cox.net

As the years go by after receiving my TM diagnosis, I am learning, sometimes the hard way, to cope with a number of my medical problems. I am sure many of you have also learned various ways of dealing with your medical issues, regardless of whether you have an ADEM, NMO, ON or TM diagnosis. I have been frustrated in the past when my doctors have not advised me of a product that might have helped me with a medical issue or offered some other guidance. I considered that it might be very helpful if we began to share some of the things we've learned for ourselves over the years through our experience. I have asked Sandy to please include these tips in the TMA journals and newsletters. As with all medical issues, it is extremely important to bear in mind that you should not try anything without first discussing it with your doctor. Also, it is important to consider that what works for one person may not offer any positive benefit for another. With those important considerations, I am going to offer a list of some things I've learned and some of the things that have worked for me.

I always take someone with me to a doctor's appointment. I need to have another set of ears to remember what was said. Often I will hear something that upsets me or relieves me and then miss something else that the doctor said. After my appointment, I am able to review what I heard and what the other person heard from the doctor.

I bring a list of written questions with me to my appointments. I don't want to have to remember all of my issues at the spur of the moment and I like to keep my thoughts organized. I also try to prioritize my issues on the list.

If I don't understand what my doctor explained to me, I keep asking questions. I certainly don't consider myself to be stupid if I don't understand. I hold my doctors responsible to communicate with me in a way that I can understand. If my doctor doesn't treat me with respect or answer my questions, I am going to change doctors.

Heat is not good for my nerve pain. I avoid going into a sauna, spa or even a hot bath. I, instead, use cold water. I have found that cold ice packs or even packages of frozen vegetables can help to relieve my pain.

Some of my medications can cause dry mouth. I have found a number of products on the market which really work to alleviate dry mouth. I use dry mouth toothpaste and mouthwash which are very easy to find at your local drug store. I use Biotene; there are others.

My primary care doctor has prescribed me Amitizia for my constipation. This has been an excellent medication for me. My neurologist had never heard of it. I have tried many prescription and non-prescription remedies for constipation and this is the only thing that has worked for me.

I have learned not to just "tough it out" when it comes to pain. I have also found that pain medicine works best for me when taken as prescribed. For example, if I am supposed to take it every 6 hours, I don't try to make it to 7 hours. If I wait for 7 hours and I am in pain, it is going to take longer for the medicine to kick in. I take the medication before I hurt.

I am on long-term pain medication 24 hours a day. I hope you are not. If you are, I have found that taking my morning dose an hour before I get up works really well. I set my alarm for an hour before rising, take the pill, and go back to sleep. If I wait to take the medication when I get up, I don't feel very good for the first half hour of my day.

Don't be afraid to try remedies, such as hypnotism, acupuncture, or meditation. During a very painful time, self-hypnosis was very helpful for me. Do be sure to use a credible professional.

If you would like to share your experiences, please send them to me via email and I will compile the suggestions for the TMA publications. Thank you for your willingness to share your suggestions with others.

Medicare Pays for Some Family Caregiver Education

Did you know that Medicare will pay for certain types of family caregiver education when it's provided as part of a patient's medically-necessary face-to-face visit? It's true; although your loved one's physician may not be aware of it.

A physician may be able to bill for family caregiver education as part of the counseling and coordination of care services provided during a patient visit, as long as the caregiver education directly involves the patient and is medically necessary. This education can take place in a doctor's office or other outpatient facility, a patient's home or private residence, or an assisted living facility or other domicile.

Medicare's new publication, "Tip Sheet for Providers: Caregiving Education" can be found at:

www.cms.hhs.gov/Partnerships/downloads/ProviderBillingforCaregiverEducation.pdf.

Download a copy for yourself and be sure to share the information with your loved one's physician.

Support Groups

We Need For You to Get Involved

If you live in Spain, Portugal or France and you have ADEM, NMO, ON or TM, or you are a family member of a person with one of these disorders, we are encouraging you to start a support group in your country. There is also a long list of states in the United States that do not currently have a support group. We need for you to get involved!

What is involved in being a support group leader? The following are the minimum requirements. You must have a telephone and a computer with internet access, and you have to be willing to post your name, telephone number and email address on our web site under the link support groups so that people from your state or country are able to find you. The incidence of TM is 1 – 5 in one million. The incidence of the other disorders is not as well known, but it is likely that the incidence is similar or less. Your phone will not be ringing off the hook.

When a person signs up for membership in the TMA, Jim sends their membership form to our support group leaders so that they can contact the new member. Most of these people are recently diagnosed or they are a family member. Think about your experience; what would it have meant to you if you had received a phone call from someone who understood your disorder shortly after you had been diagnosed!

We would ask you to be very familiar with the information on our web site and how to navigate around our web site. Since we do not provide medical advice to people, it is important that we are able to guide people to excellent medical information. There are numerous articles and presentations available on our web site covering all of the disorders and all of the issues surrounding acute therapies, long-term therapies, restorative therapies and symptom management. Also, there are numerous ways for people to share information and support on our web site.

Some of our support groups have meetings; some of them offer support only via the telephone or internet. This is a very individual decision and, while we encourage meetings, we do not require them. Over the years, it has been quite evident to me that the people who benefit the most from the support groups are the support group leaders. Once you have been through such a life-altering experience as receiving an ADEM, NMO, ON or TM diagnosis, or going through this experience with a loved one, there is nothing more transforming as turning this really horrible experience into a positive and beneficial part of your life. Over the years, I've observed this transformation in so many people. I hope that you can relate to the possibility and are open to starting a support group in your state or country. If there is already a support group in your state or country, please understand that your support group leader needs and would greatly appreciate your help. Please get in touch with your support group leader and offer to get involved.

You can change the world … you can help us to heal the world.

Please consider becoming a support group leader if you live in one of the following states: Alabama, Alaska, Connecticut, Delaware, Hawaii, Indiana, Iowa, Kansas, Louisiana, Mississippi, Missouri, Nebraska, New Mexico, North Dakota, Oregon, Rhode Island, South Dakota, Utah, Vermont, West Virginia, or Wyoming.

Paula Lazzeri is currently identified as the support group leader in Washington. As the treasurer of the TMA, Paula is already overwhelmed with the work that needs to be performed for our organization. If you live in Washington, please consider becoming a support group leader.

All you have to do to become a support group leader is to get in touch with me. Thank you!
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Arizona

"It started with flu-like symptoms." "I had nausea, dizziness and I felt – lightheaded." "My new symptom is burning on the back of my thigh." "I hate taking morphine but if I don't, I can't function." "Do you have dry mouth from your meds?" "What about constipation?"

This is the kind of conversation you might hear on the third Saturday of the month when our Arizona support group meets. We started with only three members, two who had TM and the mother of one of us. Now we are up to five, all women.
At first we met at restaurants around town and that worked for awhile; however, some members had concerns about restaurants. A few had problems sitting for long periods of time; others felt uncomfortable discussing bowel and bladder control in public places. Last month, for the first time, we met at a member's house. It worked and we will continue doing that for the near future.

Our group is small so it is easy for us

to be close. At times all of us have felt like staying home because we have been fatigued or not felt well. Instead, we dragged ourselves to the meeting. We are always glad when we make the effort and go.

We almost always discuss how we are feeling, new symptoms, and any concerns about our doctors. Lately, we have been discussing the pros and cons of going on disability as one member is considering that option. I think we all like to tell how we were diagnosed with TM, even though we have heard each person's story before. But there is comfort in retelling it. As we have gotten to know each other, we also talk about the same topics any friends would discuss.

When I started this support group, I thought it would be a way for me to give back and help others because I have had TM for nine years. Instead, I find myself getting so much from the others in the group. For me, the group is the best place to complain about my pain and the feeling I sometimes get of the unfairness of life in giving me TM. Why me? I exercised, didn't smoke, ate right. I was a good person. If that wasn't enough, two years later I got Type 2 diabetes and was told it was likely a result of taking steroids for TM. My friends who don't have TM care but don't really understand. Also, I know if I want to keep their friendship, it is better to keep my complaining to a minimum.

The women in my support group are courageous, awesome and supportive. My life was been enriched by knowing them.

If you are interested in getting involved in our group, please feel free to get in touch with me.

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Southern California

Our group met in October. We were so fortunate to have Shirley Oebel from the American Red Cross who did a wonderful presentation on disaster preparedness for people with disabilities. She explained the steps we need to take to protect ourselves. It was very informative.

As usual we had a great potluck with plenty left over. We had the usual round table discussion. There was a lot of discussion about the H1N1 flu virus and vaccination. There were twelve of us at the meeting. The food was good and the information was invaluable.

On a very sad note, our member, Rick Steele, passed away on September 3rd. Four of our group went to the memorial. He was always very current on medicines and just a joy to be around. He is truly missed.

Our next meeting will be the 3rd Saturday of January 2010.

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Cindy McLeroy
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North and South Carolina

I am asking for help to get a support group more active in North and South Carolina. I have had some personal issues in the past couple of years which have made it difficult for me to get this group going. If you are interested, please get in touch with me.

Also, I would like to share an important resource. There is a group called ASAP (Adaptive Sports & Adventures Program), which offers opportunities to people with disabilities to enjoy life. They offer several different programs with little or no cost. Some of the sports they offer are snow skiing, water skiing, biking, tennis, and basketball. This organization has really been of great benefit to me. They are located in Charlotte. If you live too far away, I would encourage you to seek a similar resource from the rehabilitation department at your local hospital.

I look forward to hearing from people in the Carolinas who are interested in helping me get our support group going.

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Germany

Hello, my name is Ursula Mauro and I'm the support group leader of the German TM support group; since 2006 a registered society named Myelitis e.V. with 119 members.

Each year we hold two meetings, one in South Germany (about 20 people) and one in North Germany (10-15 people). My neurologist, Dr. Weiss, always attends the South Germany meetings and answers questions from the members. It's good to have such a nice, reliable and qualified neurologist who cares for people with such a rare disease as TM.

As in the past, we have applied for and have received grants from the German insurances. These funds are being used primarily to translate medical articles about TM/NMO/ADEM/ON into German for the TMA and German Support Group web sites, to help subsidize the hotel cost for our members to attend meetings and to cover the costs of our office materials and post-

age. As a support group of the TMA, we share the policy of no membership fees. As our members do not support our group in the same way that the US members support the TMA, we depend on these funds to cover almost all of our operating costs. We are grateful to be able to receive this funding and we hope it will remain like this in the future.

Each year I send out two or three small German newsletters to inform people about important news and activities of our society and of the TMA. It's easy to find us on the internet, through the insurances, and through support group networks. Some hospitals and doctors use our leaflets to help us connect to people with these rare disorders in Germany. I regularly receive calls and emails from people who are searching for help and the translated articles are particularly helpful in this regard.

Since October 2008, we are member of the ACHSE, a German society, which is a network of patient organizations with rare diseases. The sponsor of this organization is Luise Köhler, the wife of the German Federal President. ACHSE has 15 people on their scientific advisory board and eight full-time employees. We are very pleased that we were accepted. They really do great work, advocating on behalf of rare diseases, fighting for better possibilities of diagnosis and treatments, and providing us with important information.

I am regularly in contact with Lew Gray from the UK TM Society and Sandy Siegel and Jim Lubin from the TMA. I am very thankful to work with such great people!

When I started to find people with TM in Germany in 2001 who wanted to get involved in a support group, I wouldn't have thought it possible that we would become a society and be as active and successful as we are today. I remember the hardships of the first three years; but I didn't give up and my patience has paid off. I want to encourage people to start support groups in their countries, especially in countries which still don't have TM support groups. Please feel free to contact me if you have questions about how to get started.

If you live in Germany, Austria or Switzerland, please get involved in our support group. We want to get everyone involved and are so pleased with the participation of every new member!

Take care.

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SistaMoon Foundation for Devic's Disease

As with most people, I became aware of Devic's Disease or Neruromyelitis Optica (NMO), because someone I know was diagnosed with the disease. The road to NMO was not an easy one. My daughter, who is a survivor of NMO, was like many others, misdiagnosed. Because of her misdiagnosis, she had an acute attack that has left her a paraplegic. NMO has affected my life, as well. Due to my daughter's disability, I have been unable to return to my career, hobbies or other things that used to bring me joy. However, instead of letting this disease take over my life, I decided to become an advocate for my daughter. I created SistaMoon Foundation to help raise funds for research and support for those affected by NMO. The SistaMoon Foundation is based on the belief that everyone deserves to be educated and well informed when it comes to their health. It is important to me to make sure that information about NMO is disseminated throughout the world.

I created SistaMoon Foundation, a non-profit charitable organization, for many reasons: first, to bring awareness to Neuromyelitis Optica; second, to help raise funds for research and support for individuals diagnosed and affected by this rare disease; third, to advocate for NMO survivors who are unable to advocate for themselves; fourth, my desire to help educate individuals regarding this disease, by providing information from some of the leading researchers and physicians who have the best knowledge of NMO. My daughter, Dawn, was diagnosed in September 2008.

This foundation is necessary because NMO is so rare and under diagnosed, as well as misdiagnosed as Multiple Sclerosis (MS). Although there are many who are misdiagnosed, 35% of African Americans are misdiagnosed with MS when they really have NMO. According to the Walton Centre Medical Services in England, "80-90% of people with Neuromyelitis Optica are women."

I challenge everyone who knows about this disease to tell everyone they know and to encourage them to tell someone else. Before we know it, we will raise awareness and the road to a cure and prevention will be paved.

Shelia Jean-Simone Sheckles-Bennett
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ADEM, NMO, ON, Recurrent TM, TM or NMO with Lupus, Sarcoidosis, Sjogren's and HIV: Finding Each Other to Share Information and Support

We are trying to assist people who have the very rare neuroimmunologic disorders find each other for the purpose of sharing information and support. We are creating the lists identified below for that purpose. If you have one of these neuroimmunologic disorders and would like to be added to the list and then receive a copy of the list, please send us your information. We only share these lists with people who are willing to be added to the lists.

- Recurrent Transverse Myelitis
- Transverse Myelitis or NMO with HIV
- Optic Neuritis

If you are interested in being added to one of these lists and then periodically receiving a copy of the list, you can send me your contact information either by email or through the postal service. Please send me your full name, complete postal address, phone number and email address (if you have one). Be sure you clearly identify to which list you would like to be added.

Sandy Siegel
1787 Sutter Parkway
Powell OH 43065-8806
ssiegel@myelitis.org

Acute Disseminated Encephalomyelitis (ADEM)

The ADEM list is being compiled by Barbara Kreisler. If you would like to be added to the list, please send your information to bkreisler.imprint@verizon.net.
An ADEM Directory will be published and mailed to everyone who is on the ADEM list.

Neuromyelitis Optica (NMO) or Devics disease

The NMO list is being compiled by Grace Mitchell. If you would like to be added to the NMO list, please send your information to gmitchell@myelitis.org.
An NMO Directory will be published and mailed to everyone who is on the NMO list.

TM or NMO and the Rheumatic Disorders (SLE or Lupus, Sjogren's syndrome, Sarcoidosis)

This list is being compiled by Sharon Robinson. If you would like to be added to this list, please send your information to Rufusandchi@yahoo.com.
A directory will be published and mailed to everyone who is on the list.

The TMA Equipment Exchange

Please participate in the TMA Equipment Exchange on www.myelitis.org. You will see the link to the Equipment Exchange on the column of links on the main page of the TMA web site. I have been assisting the TMA Board in developing and offering this program to all individuals affected by TM, ADEM, NMO and ON and their families. The program is intended to assist our community in exchanging surplus equipment with each other for the cost of shipping only. If you are like our family, we have several pieces of equipment that have been outgrown by our son, Jason, who has had TM since ten months of age. We have donated some of his equipment in the past to other organizations, but we are glad to now have another option to share this equipment with others affected with the neuroimmunologic disorders and their families.

We encourage all of you to begin to list your equipment as soon as possible. The more equipment that is listed, the more individuals in our community will be helped. If you have any questions as you begin to use the program, please use the help link on the equipment exchange web site.

Thank you for your support,
Darian Vietzke

TMA Equipment Exchange Instruction Sheet

The TMA equipment exchange is explicitly for exchanging free equipment except for the cost of shipping only. How the cost of shipping is divided is agreed upon by the individual(s) donating the equipment and the receiver (s). Selling of an item is explicitly disallowed.

To list an item(s) to exchange, first follow the on-line instructions to register as a new user and then use the on-line instructions on the Member Area tab to list your item(s) to exchange. Note that several fields can be completed after an item is exchanged. This information is being requested in order to gather statistics to request grant funds to assist in covering shipping costs when exchanging items in the future.

If you are looking for a particular item, follow the on-line instructions to view current ads. Once the item is found, contact the donor (lister) using the on-line instructions to discuss specifics of the item, discuss how to exchange the item if it matches what you are looking for, and how the cost of shipping is to be managed.

Any item inappropriate for exchanging will be removed by the site administrator. To report any item that is inappropriate, please send an e-mail to exchange@myelitis.org

Items exchanged via this site are not tax deductible. Any questions regarding taxes should be directed to your tax accountant.

If you have items you wish to sell and donate a percentage to the TMA, please click on the related link on the front page to use eBay Giving Works.

If you have any comments or questions regarding the TMA Equipment Exchange, please send an e-mail to exchange@myelitis.org. Thank you.

Important Reminder About The Transverse Myelitis Association Membership Directory

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

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

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

The TMA Membership Directory and Privacy on the Internet?

The information we provide on our web site and in our publications to our membership is one of the most important functions of The Transverse Myelitis Association. When you share your information in an In Their Own Words Column, you change lives. I have no doubt about this, because I hear from people every day who are inspired and informed by these writings. The access our support group leaders provide to people in their communities is invaluable. To know that you are not going through this experience alone or to find support and information in your community is truly a blessing for people.

Sharing information in our publications and on our web site is a selfless, kind and generous act, and we are all grateful for your participation. It is also very important to understand and accept that once this information is posted on our web site, it is available to anyone who has a computer and internet access across the globe. This ubiquitous access is the incredible value and also the bane of the information technology age.

So, we want and need for you to be generous about sharing this information, but we also want for you to be informed and judicious about making these decisions to share information. If you do not want to be found in a web search or you do not want for your information to be identified in a web search, please do not write an article for the newsletter or journal and please do not volunteer to be a support group leader. In addition to the information in our publications, it is important to bear in mind that any postings you put on a message board or in a list-serve group can also be accessed through a web search. It is almost always the case that if you are wanting anonymity in your life, the less you put out there electronically, the better, and that includes email messages, because once you hit that send button, you have no control over what the person does with that information on the receiving end.

It is also critically important to bear in mind that The Transverse Myelitis Association does not put membership information on our web site or post it electronically anywhere. We publish the directory in paper copies and we mail these directories only to our members who are listed. We send electronic copies of our member information to the people who do our mailings around the world, but they only receive the information for the people for whom they do the mailings. They do not receive the entire membership database. We expend a great deal of effort in protecting your information and limit to the extent possible, the electronic versions of this database.

If you want privacy, we do what we can to help you achieve that end. Please help us by making informed decisions in regards to what you submit for publication and what you post on the web site on our message boards and in the list serve groups. The TMA functions so effectively as a support network, because so many of you are willing to share and to help others. We urge you to continue to do so; we depend on your willingness to do so. But we don't want for you to participate in this sharing, if this activity is going to compromise any concerns you might have about privacy. Be smart and be realistic about how the internet works and what is private and what is public about the internet.

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Calling the International Headquarters of The Transverse Myelitis Association (614)766-1806

I am compelled to write this article because while I love and value the work I do for The Transverse Myelitis Association, I also value my marriage to Pauline and I enjoy and require some minimum amount of sleep.

Jim Lubin, Debbie Capen and Paula Lazzeri have worked so diligently over the years to create a really professional and wonderful organization. We owe them a tremendous debt of gratitude for their selfless work over many years. Our stationary and business cards look really great, our web site is second to none, we have the most incredible medical advisory board, we

have partnerships with some of the most prestigious organizations, and our publications are filled with great information. No matter how hard the officers work to create a professional organization, when you call the international headquarters of The Transverse Myelitis Association, Pauline or I are going to pick up the telephone in our kitchen. And when we pick up the phone, we're going to say *hello*, because the caller could be my Mom and Dad, or David, or Hanni, or Julie. Sometimes it is a person from the Kidney Foundation wanting to know if I have any clothes or small household items that I would like to donate and sometimes it is a person from Sri Lanka in the middle of an inflammatory attack.

Over the years, I have come to appreciate that among the most important things that I do as President of The Transverse Myelitis Association is to pick up my telephone. Given all of the experiences, and appreciating just how critical many of these phone calls have been for people, it is hard to imagine that I will ever be in a place in my life where I don't pick up the phone. And if answering the telephone is in the job description of the President, I'm pretty sure that I can be President for life, if I so desire.

As this is our home telephone, and we are trying to have a life, it has become time for me to establish some ground rules about the International Headquarters telephone.

1. Please pay attention to time zones. We live in Ohio which is on Eastern Standard Time. Ohio also participates in daylight savings time. Daylight time begins in the United States on the second Sunday in March and ends on the first Sunday in November. On the second Sunday in March, clocks are set ahead one hour at 2:00 a.m. local standard time, which becomes 3:00 a.m. local daylight time. On the first Sunday in November, clocks are set back one hour at 2:00 a.m. local daylight time, which becomes 1:00 a.m. local standard time.

2. If you need to speak to someone who is conscious while they are talking to you, it is best to call me during the day. To reach me during the day, please call me on my cell phone: (614)352-6765. I have my cell phone with me almost everywhere. I don't, however, take my cell with me to meetings at work, so please leave a message, your name and clearly provide your phone number and repeat it. If you are calling from outside of the United States, it would be really helpful if you could provide me with the country and city code when you leave me your number. If you are calling from outside of the United States, I am not going to be able to call you back until I get home, because my international calling plan is on our home phone.

3. When you call and have to leave a message, it is a great idea to give me some sense of the issue you would like to talk to me about and the urgency of my response. If your message indicates that you or a family member is experiencing an inflammatory attack, and I'm awake and screen the call, I won't wait until tomorrow to call you back.

4. When you leave a message and provide your phone number, please repeat it slowly so that I can write it down. If you have a cell phone number, please give me both your home and cell phone numbers.

5. Pauline and I are heavily medicated and we go to bed really early. If you call after 7:00 PM, no one is going to answer the phone. Listening to it ring three times is really irritating and it is even more irritating when we're lying in bed. Please call before 7:00 PM. If you leave a message for me, I am going to return your call the next day. In 15 years, I have never missed returning a phone call the next day.

6. When I am called, I am occasionally asked to speak with the Spanish-speaking person from The Transverse Myelitis Association. After five years of Spanish in high school and college, I only remember, Hola, Paco; que tal, como estas. I can also swear pretty fluently in Yiddish. Otherwise, the international headquarters of The Transverse Myelitis Association only provides an English speaker before 7:00 PM. Please try to find someone who speaks English to be on the phone with you or have them call me.

7. If you call on Saturday or Sunday, please call after 11:00 AM. Pauline and I work Monday through Friday. We have Saturday and Sunday off. I will be awake, because Kazu gets me up every morning at 5:00 AM and doesn't recognize weekends or holidays. I'll be in my office working on responding to TMA emails or working on some other writing assignment for the Association. Pauline will be sound asleep. The ringing is going to wake her up before I can reach the phone, and she is going to be really upset. She isn't going to be upset at you; she's going to be really upset with me, even though I'm not the one who made the phone ring. If you want to talk to someone who is conscious, call before 7:00 PM. If you want to talk to someone who isn't waiting to get yelled at, please call after 11:00 AM on Saturday and Sunday.

Pauline is going to be really mortified that I've disclosed all of this personal business about our lives to people from every state in the United States and from more than 80 countries around the world. I hope that if the telephone stops ringing in the mornings on the weekends that she will forgive me. Thank you for your understanding.

Fundraising and Awareness

Helping to Fund the Work of Your TMA

The officers and board members of the TMA are volunteers; they receive no compensation of any kind for their work. There are no employees in the TMA. There are no offices; the officers work out of their homes. In order to facilitate access to support and information, the TMA does not charge membership fees. As TM, NMO, ADEM and ON are rare conditions and our membership is small, it is extremely difficult to raise funds for our cause. We work most diligently to focus our resources on the direct services to our members. We operate exclusively on the basis of the generous and voluntary support of our members. There are numerous ways for everyone to help support the TMA, even if you are not in a position to make a financial contribution. Please consider getting involved in one of our fundraising efforts.

Search the Internet and Raise Money for the TMA

You can raise money every time you search the web, at iSearchiGive.com. Make it your homepage and use it to find everything from news on the economy, to mood-lifting jokes (we recommend the latter). The Transverse Myelitis Association gets a penny (or more!) every time you search. Believe it or not, it adds up quickly and best of all, it costs you NOTHING!
Start iGiving at:
www.iSearchiGive.com/TMA

Donate your cell phones

You can donate your cell phones to help raise funds for The Transverse Myelitis Association. Go to http://cellphones.myelitis.org

Online Shopping

There are numerous online shopping opportunities, as well as sales on eBay which can be made through the following link: http://www.myelitis.org/store.htm A percentage of the sales are donated to the TMA.

Save Gas. Save Time. Raise Money!

With over 700 stores in the iGive Mall and access to hundreds of exclusive coupons, free shipping deals, and sales, iGive is the smart way to shop. You'll find everything from daily necessities to special occasion and holiday gifts, at stores you know and love. So save a trip to the mall, and avoid the long lines. You'll never pay more when you reach a store through iGive, and up to 26% of each purchase benefits The Transverse Myelitis Association!
Start iGiving at:
www.iGive.com/TMA

Café Press Shop for items with The Transverse Myelitis Association logo to raise awareness and show your support!
http://www.cafepress.com/myelitis

Amazon.com You can shop at Amazon.com for Books, Music, DVDs, Videos, Toys and more.
http://www.myelitis.org/amazon

Music Downloads for any device!

Shop for your favorite song or album from our mp3 store powered by Amazon and download music that works with an Ipod or any mp3 player.
http://www.myelitis.org/shopmp3

eBay

Now you can sell an item on eBay and donate from 10% to 100% of the final sale price to help support the TMA.
http://www.myelitis.org/ebay

If you are a teacher, a student or a parent of a student and would like to establish the Reading for Rachel Program in your school, everything you will need to get the program started can be found on the Reading for Rachel web site: http://www.readingforrachel.org. All funds received by The Transverse Myelitis Association for the Reading for Rachel Program are used exclusively for research to better understand TM, to find treatments for the symptoms of TM, and to ultimately find a cure. If you are interested in starting the Reading for Rachel program in your school, you can also contact Cathy Dorocak, Rachel's Mom and International Chair of the Reading for Rachel Program: cathy@readingforrachel.org; (440)572-5574.

Donations by Check

We always welcome and are grateful for a donation to the TMA. You can download a donation form to include with your check from the link: www.myelitis.org/donation-form.htm Please make a check or money order payable to The Transverse Myelitis Association and mail it to:

The Transverse Myelitis Association
Paula Lazzeri, Treasurer
10105 167th PL NE
Redmond, WA 98052-3125

Thank you!

GoodSearch You Search or Shop... We Give!

Every time you shop online at your favorite stores you could be saving money and earning a donation for The Transverse Myelitis Association.

The new toolbar, developed by GoodSearch & GoodShop takes just a few seconds to download. More than 1,300 top stores including Apple, Best Buy, Gap, PetSmart, and Staples are pitching in and will donate a percentage of each sale to our organization. There's no extra cost to you and you may even save money as the merchants are providing us thousands of money-saving coupons!

The GoodSearch toolbar also includes a search box which is powered by Yahoo! Each time you search the web, about a penny is donated to us!

There's no easier way to help The Transverse Myelitis Association. Please spread the word!

Here's how it works...

1. Download the toolbar at **http://www.goodsearch.com/toolbar/**

2. Search the web directly from the built-in search box. Each search generates a donation (about a penny per search) for our cause!

3. Shop online as you normally would at any of the 1,300+ participating stores. When you're at the store's website, the toolbar will automatically light up displaying the percentage donated and any available coupons. **With the new toolbar, your purchases will generate donations even if you forget to start your shopping at GoodShop!**

We thank you for your support as we change the world one search and one purchase at a time!

Help Raise Awareness with a TMA Wristband

For the past 3 years, thousands of our members have been helping to raise awareness of the TMA by wearing bright blue wristbands. They have been available on the TMA website and at our symposia for purchase. The wristbands are available in a marbled blue/grey in the adult size and solid blue in the youth size. The youth size also fits women with small wrists. These wrist bands are made with 100% synthetic silicone rubber and debossed with the abbreviations "TM-ADEM-NMO-ON" and "www.myelitis.org."

Many families have purchased these wristbands as party favors for birthday celebrations, fundraisers for raising research dollars, and to just proudly wear every day. Several people have sent us photos of themselves displaying their wristbands at known landmarks around the world. All of the money raised through the sale of the wristbands goes towards the cost of printing and mailing out the information that you receive in newsletters like this one, and for mailing out new member packets for those newly diagnosed with TM, ON, ADEM, and NMO.

The wristbands are inexpensive – only $2.00 each – and you can either order them online at our website, making your purchase with a credit card transaction, or you can mail a check to The Transverse Myelitis Association and when we receive your payment, we mail them to you.

To order online, please go to our website at:
www.myelitis.org/wristbands.htm.

For check payments, you would mail your payment along with your order request to:

The Transverse Myelitis Association
Paula Lazzer, Treasurer
10105 167th PL NE
Redmond, WA 98052-3125

Specify "for TMA wrist bands"

Shipping charges:
1-5 $1.00
6-10 $1.50
11-25 $5.00

For quantities more than the above, please send an email. If you would like us to calculate your shipping for you, you can send an email to wristbands@myelitis.org and we will tell you how much to send. You can also call Debbie Capen at (951)658-2689 to get your total cost and more information.

Don't miss out on getting your own one-of-a-kind TMA wristband!

Inkjet and Toner Recycling Program

The Transverse Myelitis Association has partnered with the Funding Factory Recycling Program to collect empty inkjet and toner cartridges. This is an important fund raising effort for the Association. Please go to our web site at http://www.myelitis.org/recycle/. Once you register, you can order prepaid UPS return labels that you put on any box you have. When you fill in the information, use your own name as the

"Organization" name, but also, **PLEASE USE ID NUMBER 63960 AS THE BENEFICIARY.** This ensures that the TMA will be receiving the benefits of the collected cartridges. When filling out the contact information, the form asks for a "title". You can list "other" and put "supporter" for your title. Once the company has your information and you request shipping labels, they will ship them to you to place on the boxes. Once the boxes are filled, you can take them to any place that picks up UPS packages (such as "Mailboxes, ETC.").
We appreciate your participation in this important program!

Honor the Children in Our Community and Support the TMA

The Transverse Myelitis Association held a Children's and Family Workshop in Columbus, Ohio in July, 2002. One of the many activities they participated in during this special weekend involved working with an art therapist from Chicago, Lori Stralow Harris. With the help of Ms. Harris, the children created beautiful paintings which were constructed into a quilt of courage and hope. The original artwork currently hangs in the Johns Hopkins Transverse Myelitis Center where it is appreciated by the hundreds of patients every year who are cared for at the Center.

We are very pleased and proud to be able to offer you the children's artwork through Café Press. The proceeds from the sale of these items will be used to fund the many important programs of The Transverse Myelitis Association. We hope you will take the opportunity to enjoy the children's work and to support the TMA.

http://www.cafepress.com/tmagifts

Share your passion and donate to our cause with your everyday purchases. We've partnered with Capital One® Card Lab Connect to bring you our newest fundraising program, which helps us earn money effortlessly every day! Just carry one of our custom credit cards (it comes with a competitive rate and no annual fee), and 1% of purchases made with the card will be donated to The TMA. We'll also receive a $25 bonus donation when you make your first purchase. And not only will you be donating to our cause with each purchase you make, you'll be helping to spread the word when people see your unique card, which includes our name and logo, as well as Margaret Smith's artwork as backgrounds.

Apply online via a secure web page at http://www.myelitis.org/creditcard

We urge you to only make purchases with this credit card that you are able to pay off each month. We would love to receive the benefits from this program without your paying any interest on your purchases. We greatly appreciate your support!

The TMA Greeting Card Program

http://www.myelitis.org/cards/

We are thrilled to introduce The TMA Greeting Card Program. The cover of each of the cards is a beautiful water color painting of landscapes or flowers. The back of the cards include the TMA logo, our web address and a description of our Association. The inside of each card is blank and perfect for offering your own sentiments. We urge you to use these wonderful paintings as your regular cards for the holiday season, for thank you and everyday notes or for any purpose.

The proceeds from the sale of these items will be used to fund the many important programs of The Transverse Myelitis Association. As the neuroimmunologic disorders are rare and our membership is small, it is extremely difficult to raise funds for our cause. We work most diligently to focus our resources on the direct services to our members. By using these beautiful greeting cards, you will be supporting the important work of the TMA and also raising awareness about acute disseminated encephalomyelitis, neuromyelitis optica, optic neuritis and transverse myelitis.

About the Card Artwork

Sandy and Margaret Smith are members of The Transverse Myelitis Association from Pittenweem, Fife, Scotland. They are active members of the Scotland Support Group led by Margaret Shearer. Sandy has TM. Margaret is an artist. Margaret has created beautiful paintings of landscapes and flowers. She has donated this artwork to the TMA and we are very pleased to be able to offer you these beautiful greeting cards.

The Transverse Myelitis Association 2008 Statement of Financial Activities (in US Dollars) Paula Lazzeri

The following table presents The Transverse Myelitis Association Annual Financial Reports for 2008. The TMA (General) Fund column presents all funds received and expended directly by TMA as recorded in the Association's financial account. The Total Donations and Expenses to Benefit TMA column is presented to help convey the total cost of providing TMA member services during 2008. 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.

INCOME	TMA Funds	Total Donations and Expenses to Benefit TMA
Amazon.com Commissions	261	261
Café Press Commissions	52	52
Donations made by Board of Directors	0	9,974
Endowment Interest	300	300
General Donations	68,240	68,240
iGive.com Commissions	209	209
Interest CD's	3,200	3,200
Interest Savings account	820	820
Makoa Fundraiser	2,536	2,536
MissionFish.com Commissions	298	298
Recycling Commission	1,481	1,481
Research Donations	2,999	2,999
Support Group Donations	200	200
Symposium Fees	79,901	79,901
Wristband/cookbook Fundraiser	97	97
TOTAL INCOME	**160,594**	**170,568**
EXPENSES		
Accelerated Cure Project Support	10,000	10,000
Bank Fees	83	83
Children's Camp Expenses	5,231	5,231
Domain/Web-site/Webhosting	1,274	1,274
Hopkins Research Coordinator Position	8,750	8,750
Internet Service Provider	0	779
Mileage and Parking	0	108
Postage	8,052	8,915
Printing	20,307	20,345
Secretary of State Registrations/Annual Reports	20	20
Computers and computer supplies	0	2,317
Supplies	0	317
Symposiums	126,515	126,515
Telephone	0	1,294
Travel	0	4,258
Wristband Fundraiser	1,125	1,125
TOTAL EXPENSES	**181,357**	**191,331**
NET LOSS	**-20,763**	**-20,763**

Transverse Myelitis Association 2008 Statement of TMA Account Balances

Children's Camp	**10,991**
Operating Fund	**157,265**
Research Fund	**74,492**
Richard Charles Gilmur Endowment Fund	**11,603**
Richard Charles Gilmur Endowment Interest	**869**
Support Group Fund	**2,835**

2008 Donors to The Transverse Myelitis Association

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 2008.

Aetna Foundation
Niyurys Alba
Albemarle Area United Way
Obdulio and Mariana Alfonso
Albert and Janet Aloia
Gloria Allen
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Michael Deyarimin
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Barry and Mary Kercher
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Robert Storbeck
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United Way of Central Maryland
United Way of the Greater Lehigh Valley, Inc.
Jerry and Diane Vechionne
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Nancy Vroom
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Denise Wehrer
Janice Weiner
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Marlene Word
John and Theresa Wrabel
Thomas and Caroline Wrabel
Thomas and Trudy Wrabel
Cheryl Wright
Karen Yong
The Young Family
H. Jay and Rachelle Zukerman

Lucille Assid Memorial:
Karen McGrath
TRC

Milton Ballantyne Memorial:
Fran Garrigan

Brenda Kay Bube Memorial:
Andrew and Carolyn Miller

Clifford C. Camp Memorial:
Merlin and Judith Halverson

Nancy Catalano Memorial:
Leonard and Lynne Martino

Angelo Costanza Memorial:
Flora Costanza

Lewis Evans Memorial:
Claire T. Evans

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Kathleen Junikiewicz
Paul Langer
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Jeffrey and Rose Anne Stern
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TMA Community Photo Album

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The Transverse Myelitis Association

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2010 Rare Neuroimmunologic Symposium
September 23 – 26, 2010 Dallas, Texas

Johns Hopkins Center for NMO, Recurrent TM, and Related Neuroimmunologic Disorders: Clinical Care and Research