The controversy over the decision of when to initiate multiple sclerosis (MS) treatment has been the subject of several articles over the last 2 years. The April 2006 issue of the Archives of Neurology printed multiple articles from several authors who took different positions. We have continued that discussion here in this issue of MS Exchange.

The chronic and debilitating nature of MS over a lifetime raises the question in many clinicians’ minds of how to prevent the long-term effects of this disease. Treatment regimens today are aimed primarily at symptom management once the disease has been confirmed. Unfortunately, it’s also recognized that these symptoms are generally progressive, and most patients will experience a loss of function over time as a result of irreversible neuromuscular degeneration. Would early intervention, before the symptoms even fully manifest, avert the damage that leads to disability?

As Frohman et al point out, “natural history studies demonstrate that 20 to 25 years after diagnosis, nearly 90% of patients with MS will have substantial disability.” These authors further explain that a higher frequency of attacks in the early stages is associated with a higher degree of disability later on, a notion that suggests that intervention at the earliest signs of MS can potentially alter the progression of the disease, as well as the level of future disability.

Still, the advantages to early treatment are clouded by several factors. Pittock and colleagues contend that since disease-modifying therapies are aimed at specific types of MS (primary-progressive vs. relapsing-remitting), to initiate therapy before the full diagnosis may be ineffective in many patients. And while these therapies have shown good benefits in the short-term management of MS symptoms, the long-term effects have not yet been fully investigated. As many as a third of patients also do well without any drug therapy at all, and it is difficult to identify which patients will benefit from therapy.

Studies such as the BENEFIT trial investigating the impact of early intervention with interferon beta-1b therapy in a large cohort of 404 newly diagnosed MS patients report 2-year results showing a “robust” effect on disease progression. Follow-up at the third year of the trial with 432 enrolled patients continues to support the benefit of early intervention with disease-modifying therapy. Other long-term studies are underway.

(Continued on page 2)
The approach suggested by Pittock et al is to defer therapy, which can spare many patients the unnecessary expense and possible side effects of treatment, but at the cost of allowing many patients to proceed to a more aggressive course of disease and irreversible damage they might have been spared.

Each clinician, and each facility treating MS patients, develops a standard of care based on one of these two approaches. As of now, no clear advantage to either approach has emerged.

At the same time, advances are being made in methods to better define the criteria for diagnosis at the earliest sign of a clinically isolated symptom (CIS), most often an episode of optic neuritis. The McDonald criteria used by most clinicians as the basis for magnetic resonance imaging (MRI) diagnosis were originally outlined in 2001. They were revised in 2005 to include the appearance of spinal cord lesions as part of the diagnosis. New criteria recommended by Swanton and colleagues have broadened the definition further to facilitate earlier diagnosis without sacrificing accuracy.4

All of these factors combine to suggest that we are moving rapidly toward better answers. As health care practitioners specializing in MS care, we need to keep abreast of the many developments occurring to offer the highest standard of care available to all of our MS patients.

References
Multiple sclerosis (MS) more commonly afflicts women than men, and the first signs are most likely to appear during the childbearing years, complicating the course of a normal pregnancy. Likewise, the hormonal surges of pregnancy have a significant impact on the course of MS, requiring special management of these patients beyond the usual obstetric care.

A. Dessa Sadovnick, PhD, is a genetic specialist in the field of MS at the MS Clinic at Vancouver Hospital and Health Sciences Center, and a professor in the department of Medical Genetics and Faculty of Medicine, Division of Neurology, at the University of British Columbia. In an interview with MS Exchange, Dr. Sadovnick explained the complex interaction of MS and pregnancy.

**MS Exchange:** Does MS present differently in a patient who is pregnant?

**Dr. Sadovnick:** The first onset of MS is only occasionally seen during pregnancy, and the first symptoms are not generally different from those experienced by nonpregnant patients.

**MS Exchange:** Are there any specific symptoms that are particularly difficult to handle during pregnancy?

**Dr. Sadovnick:** Pregnant women with MS are at risk for any of the typical MS symptoms. Symptoms affecting balance and walking may be aggravated during pregnancy given the body’s adjustment to the new center of gravity caused by the growing baby. A normal pregnancy hormone the body produces to relax the ligaments may make falls even more likely in the pregnant woman with MS than other MS patients. Bladder infections, frequency, and urgency may be more common symptoms during pregnancy. Any symptoms affecting “activities of daily living” will be more difficult to handle with the added demands made on the pregnant body. Symptoms of fatigue may also increase.

**MS Exchange:** If a patient with MS wants to get pregnant, how do you counsel her?

**Dr. Sadovnick:** Every patient with MS is different. Typically, I will meet with the couple and assess:

a) Concerns (Are they worried about their own health? Parenting abilities? Long-term ability to care for a child? The chance for a child to develop MS?)

b) Family history (of MS or any other health concerns that could impact the health of their children), ethnicity, age

c) Medications (prescription or non-prescription) they are taking that may be teratogenic

d) Personal MS history (age at onset, course, severity, ongoing symptoms)

e) Social support system and financial situation.

In general, if a woman has relapsing-remitting MS with a low Expanded Disability Status Scale (EDSS) score, a good support system, and a desire to have children, pregnancy is not contraindicated. For women with progressive or severe MS or limited social support, having children should be a carefully weighed decision, especially given the unpredictable nature of the condition.

During a preconception counseling session, we discuss the short- and long-term implications of pregnancy on MS symptoms, the impact of MS on pregnancy outcome, the genetic probability of MS occurring again in the family, and MS medications that should be stopped during pregnancy. We also discuss breast-feeding and family history concerns unrelated to MS.

**MS Exchange:** Counterbalancing the additional stress of a pregnancy on MS is the potential benefit the pregnancy may have on the disease course. How significant is this impact?

**Dr. Sadovnick:** Relapse rates are reduced during pregnancy, an effect that may not persist post-delivery. Statistically, about 30% of women will experience an MS relapse in the first 3

(Continued on page 4)
months after delivery. This is important information to know as it enables couples to prepare for this possibility. I recommend that couples try to secure some help around the house to look after older children, prepare meals, and do some housekeeping. Never say no to an offer of help!

We also discuss some strategies for reducing fatigue and stress in the post-partum period. If a woman chooses to breast-feed, she may pump her milk during the day so that her partner can help with night feedings. Alternatively, she may combine breast milk and formula or choose not to breast-feed at all.

**MS Exchange**: Does prolactin permanently repair neurological damage from MS or is it a temporary effect?

**Dr. Sadovnick**: I don’t think we have this answer, especially in humans. Prolactin is proposed as one possible agent protecting against relapse during pregnancy but the mechanism is likely quite complicated.

**MS Exchange**: Does pregnancy alter the course of the disease over the long term?

**Dr. Sadovnick**: Having had a pregnancy does not seem to alter the long-term course of MS. The best predictors of the long-term course are to look at the frequency and severity of relapses experienced in the past.

**MS Exchange**: Does MS pose significant risks to the unborn fetus?

**Dr. Sadovnick**: Maternal MS does not increase the risk of miscarriage, stillbirth, birth defects, or other adverse pregnancy outcomes; however, these risks may be increased by some of the medications used to treat MS or its symptoms.

**MS Exchange**: How do you treat MS during pregnancy and lactation?

**Dr. Sadovnick**: Pregnant and lactating women should ensure that they are not taking medications that could be harmful to a developing fetus. Disease-modifying therapies such as Avonex, Betaseron, Copaxone, Rebif, and chemotherapies (e.g., mitoxantrone) have not been approved for use during pregnancy and while breast-feeding. Symptom-specific treatments, including antibiotics, antidepressants, and anti-epileptics all need to be assessed individually for safety during pregnancy and lactation. The benefit of treating the symptom must be weighed against the potential harm to the fetus/baby in each case. Steroids have been used to treat function-impairing relapse during pregnancy, and do not seem to be associated with an adverse pregnancy outcome.

Women with MS should not be considered “high risk” from an obstetrical point of view unless there are additional complicating features. Epidural use during delivery is not contraindicated. A woman with MS may experience extreme fatigue during labor making an assisted delivery slightly more likely, but Cesarean section is not required based on maternal MS.

**MS Exchange**: Are there any long-term studies on risks to babies born to mothers with MS?

**Dr. Sadovnick**: The background rate for any couple to have a child with a birth defect, handicap, or developmental delay is about 5% at 1 year of age. This number does not seem to increase based solely on the mother having MS. However, some medications used to treat MS symptoms and progression are associated with birth defects, low birth weight, and an increased rate of miscarriage. It is possible that medications used to modify the mother’s immune system may have long-term implications for her children, but this information is not yet available due to the relatively recent development of these drugs.

**MS Exchange**: Does MS during pregnancy increase the child’s risk of developing MS?

**Dr. Sadovnick**: MS is usually seen only once in a family. However, we see MS affecting more than one family member often enough to know that there must be some genetic factors involved. If you are closely related to someone with MS, your chances of developing MS are higher than if you had no affected family members. Overall, though, these chances are still quite small.

Most women with MS have no other family members with MS. In this case (and if MS does

(Continued on page 10)
his year, the International Organization of Multiple Sclerosis Nurses (IOMSN) was thrilled to sponsor four regional nursing updates across the United States. This project was funded through an educational grant from Biogen Idec, a staunch supporter of MS nursing. We are very grateful to Biogen Idec and our other pharmaceutical partners for their faith and support in helping to promote the importance of MS nursing.

Our goal was to reach nurses in regional areas that have not been well served by MS nursing education. All programs were accredited and we were thrilled to have record turnouts at all events, suggesting a strong interest in our continuing these programs in the future.

Each regional event was chaired and organized by regional nurse leaders who were in charge of finding an appropriate venue and selecting their faculty. A standard set of slides that fulfilled the educational goals were provided by the IOMSN to the speakers to facilitate their preparation. We received excellent evaluations for these programs and will be using this format for future programs.

For their tireless efforts in coordinating these programs, we would like to thank our regional chairs, Elida Greinel from Albuquerque, Pat Loge from Billings, Montana, Beverly Layton from Birmingham, Alabama, and Brant Oliver from Lebanon, New Hampshire. We also want to thank our co-provider, Nurse Practitioner Alternatives, and Laurie Scudder, our brilliant liaison, for their support and input. If you are a nurse eager to facilitate this process in the future, please contact us at the IOMSN office and we will try to make this possible for you.

Colleen Harris
Committee Chair
Education/Mentorship
IOMSN

INTERESTED IN SHARING YOUR KNOWLEDGE WITH THE WORLD? JOIN THE IOMSN!

The IOMSN is the only organization dedicated to the education of MS nurses around the world. If you wish to join the IOMSN, contact the organization at: IOMSN, c/o MS Center at Holy Name Hospital, 718 Teaneck Road, Teaneck, NJ 07666, (201) 837-0727, www.iomsn.org.
**Optic Neuritis and MS: What Does the Connection Mean Clinically?**

Current treatment for MS involves long-term therapy of self-injected drugs taken for an indefinite period of time. While some diagnosed patients may experience a relatively benign course, other patients may progress on to severe disabilities and irreversible tissue damage associated with their disease. The Medical Advisory Board of the National Multiple Sclerosis Society released a consensus statement in 2005 recommending initiating treatment with disease-modifying therapies as soon as patients are diagnosed with relapsing MS, and even in some cases of patients who are not yet diagnosed but experience a CIS and are at high risk for MS. The debate over when to treat is not a new one in MS, and the controversy continues to grow.

Optic neuritis has been strongly linked to the development of MS, presenting the dilemma of whether to treat patients with this single symptom before they have been diagnosed with clinical MS. Robert Shin, MD, Assistant Professor of Neurology and Ophthalmology at the University of Maryland, offered his perception of these same challenges relating to optic neuritis as a predictor of MS at the CMSC.

Optic neuritis is an attack of visual blurring or vision loss in one eye, almost always associated with some pain or eye discomfort on eye movements, caused by demyelination of the optic nerve. By itself, optic neuritis is usually a self-limiting condition. In most cases, the pain and discomfort will resolve and vision will improve within a few weeks to several months. Some primary care physicians, neurologists, or ophthalmologists may decide to treat with intravenous steroids. This does not improve the vision restored to the patient, but does shorten the course of the episode.

Regardless of how the clinician decides to treat the optic neuritis, Dr. Shin strongly recommends that all patients with optic neuritis be sent for a brain magnetic resonance imaging (MRI) scan, with and without contrast, to identify whether they have demyelinating lesions that can signify an increased risk for the future development of MS. Optic neuritis is not the same thing as MS, but it can commonly affect people with MS, or even be the first sign that MS is developing, especially when the initial brain MRI is abnormal.

Dr. Shin explains that it’s been known for a long time that a certain percentage of people with optic neuritis will go on to develop MS, but one of the challenges has been determining the actual risk in individual patients who have had optic neuritis. “It depends on which study you look at and how long you follow the patients,” says Dr. Shin. “If a patient has optic neuritis and the brain MRI shows some demyelinating lesions, then the risk of MS might be 50% to as high as 80% or 90%, depending on the study and whether you’re following the patient for 5, 10, or 20 years.”

Dr. Shin points out that while most clinicians agree that the opportunity to prevent irreparable damage and long-term disability suggests that MS treatment...
begin as early as possible, this approach needs to be balanced with the realities of the cost of treatment and the hazards and discomforts of injection therapy for a patient who might not need it. “If the risk of developing MS after optic neuritis in high-risk patients is between 50% and 80%, that still leaves 20% to 50% of patients with optic neuritis and an abnormal brain MRI who may not go on to develop MS,” he says. “Should all patients with optic neuritis and an abnormal brain MRI be treated for MS, even when we know that at least some of them will never develop MS?”

“If the risk of developing MS after optic neuritis in high-risk patients is between 50% and 80%, that still leaves 20% to 50% of patients with optic neuritis … who may not go on to develop multiple sclerosis.”

Counter to many MS clinicians today, Dr. Shin does not advocate treating preclinical MS on the basis of CIS or a diagnosis of optic neuritis. “I feel that we should reserve MS treatments for patients who actually have MS,” he says. “If patients present with a CIS or optic neuritis as their only event then, by definition, they don’t have MS, because they’ve only had one event.” If a patient’s brain MRI is abnormal, there is an increased risk that he or she will go on to develop MS, although it is not currently possible to determine which patient will develop MS and would therefore benefit from therapy and which patient will not.

Dr. Shin recommends careful monitoring using MRI at 3- to 6-month intervals for at least a year or two. “We know that for every clinical symptom or attack a patient may have, they can have up to 10 ‘invisible’ events that can be detected by brain MRI. You can detect demyelinating activity even before the patient may have a second symptom on the outside,” he states. “Modified McDonald criteria allow us to use this evidence of new or enhancing lesions on MRI to diagnose MS even before a patient has a second clinical attack.” At that point in time, Dr. Shin suggests, clinicians can recommend starting MS treatment.

If, over time, there are no new clinical attacks and no evidence of new demyelination on brain MRI, then the clinician can refrain from starting MS treatment. “This might be one of the minorities of patients who will not go on to develop MS,” he states.

“This strategy is a compromise,” Dr. Shin says. “If someone presents with optic neuritis, but is going to go on to develop MS, we have a good chance of detecting that within the first year or two using the brain MRI. We haven’t lost much time, but we’re also not running the risk of treating someone with expensive injections that doesn’t have to be on that treatment.”

The alternative, treating all patients with a CIS and evidence of an abnormal brain scan, means that at least some of the patients are being given unnecessary treatment. “To have a patient who does not have MS taking a needle every week or every day for an indefinite period of time with no end point,” he suggests, “does that patient a disservice.” And while numerous studies of MS therapies have shown that, as a collective group, patients with CIS and an abnormal brain MRI can benefit from early intervention with immunomodulating drugs, Dr. Shin concludes, “When I’m sitting down with an individual patient and trying to determine if he or she should be on MS treatment or not, then I have to do what’s best for that individual person.”

DON’T FORGET TO REGISTER FOR THE NEXT EXAMINATION SITTINGS!

The application deadline for the February 2008 Multiple Sclerosis Specialist Certification (MSCS) exam is January 1, 2008.

Information and applications are available at www.ptcny.com.

(Continued on page 8)
Long-Term Data Still Needed To Prove Benefits Of Early Ms Treatment

Patients with a single clinical event and MRI or cerebrospinal spinal fluid (CSF) findings suggestive of MS present a dilemma. In the majority of these cases, a second disease-defining relapse will occur; in others, a new asymptomatic MRI lesion may appear. The clinical picture then fulfills the latest International Panel criteria for diagnosing clinically definite MS (CDMS). Whether to recommend therapy or decide to wait and repeat MRI scans at intervals is an issue of clinical judgment made with education and involvement of the patient.

Clearly, treating CIS with MRI lesions is treating MS at the earliest stage possible for most, but not all, patients. Three-year data from the BENEFIT study of interferon beta-1b shows a persistent difference in disability favoring initiating therapy at the CIS stage rather than waiting for the development of CDMS. Of course, this result applies best to groups of patients and may not apply directly to an individual. Some patients make the decision easier, either by wanting to be as aggressive as possible and having no qualms about starting therapy, or, in contrast, being unsure of their commitment to what may be life-long treatment and needing time to adjust to the probability of MS. Those latter patients may prefer to wait for a new MRI lesion or a second clinical event before deciding that immunomodulators are a better choice than untreated MS.

In the final analysis, we have no data proving that the clinical outcome at 20, 30, or 40 years will be altered by delaying treatment of CIS. These patients will continue to look to their neurologists for advice and remind us of our responsibility to make the best possible judgments with imperfect information. MS

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not occur in their partner’s family history), the chance for a child to develop MS during his or her lifetime is 3%-5%. I stress that there is a 95%-97% chance that the child will never develop MS. Also, onset during childhood is exceedingly rare, so these recurrence risks don’t come in to play until the child is in the “at risk” age group (i.e., adulthood) for MS. At this point, there is no predictive testing (genetic testing) available to determine whether a child will or will not develop MS at any point.

The second possibility is that the mother (or her partner) does have a family history of MS. In this case, the chance for MS to happen again in a family increases as the number of close relatives who are affected increases. Recurrence risks are estimated based on the pattern observed in the family. As an example, if a woman and her mother both had MS, there is a 15%-20% that it will affect the next generation—but that is still an 80%-85% chance that it will not affect the next generation.

New DNA Vaccine for MS Shows Promise

In October 2007, the results of a study printed in the Archives of Neurology of a newly developed DNA vaccine for MS that uses encoding myelin basic protein showed that it is both safe and beneficial. The lead investigator, Dr. Amit Bar-Or of the Montreal Neurological Institute, and his colleagues reported that the use of the BHT-3009 vaccine in 30 patients with relapsing-remitting or secondary-progressive MS showed a general “trend toward a decrease in the number and volume of contrast enhancing lesions” compared with those who received a placebo. Patients were given intramuscular injections of placebo or BHT-3009, with or without atorvastatin, a drug shown to improve autoimmune conditions, at four intervals over a 13-week period. Those who received the vaccine showed no increase in lesions visible on magnetic resonance imaging, clinical exacerbations, disability, or adverse events associated with therapy, compared with those who received the placebo.

The results of this study have strong implications for the potential to prevent or delay the onset of MS symptoms. A Phase IIb randomized trial is currently underway to evaluate the effects in a larger cohort of 290 patients.


New MRI Criteria Make Diagnosis of MS Easier

The McDonald criteria from 2001 and the revised criteria from 2005 provide the basis for current magnetic resonance imaging (MRI) diagnosis of multiple sclerosis (MS). The key determinants are dissemination in space (DIS) and dissemination in time (DIT) of lesions in the brain and the spinal cord. These criteria were established based on retrospective studies, although later studies confirmed their superior specificity and sensitivity to those previously in use.

A new study by Swanton et al, reported in The Lancet/Neurology in August of 2007, softens the definitions of DIS to include at least one T2 lesion in at least two of the four neurological regions considered characteristic for demyelination. The Swanton criteria also call for DIT to be defined simply as a new T2 lesion on a follow-up MRI scan, irrespective of the time since baseline. The current study compared these modified guidelines
to McDonald’s 2001 and 2005 criteria in predicting the risk of conversion from clinically isolated syndrome (CIS) to clinically definite multiple sclerosis (CDMS) in a cohort of approximately 500 patients who had two MRI scans within 12 months of CIS onset.

The specificity for the new set of criteria was nearly as high as for the previous versions of the McDonald 2001 and 2005 criteria: 87% vs. 91% and 88%, respectively. The Swanton criteria showed a superior sensitivity (72%) compared with the McDonald 2001 (47%) and McDonald 2005 (60%) criteria. Patients had a higher risk of conversion across all three criteria if both DIS and DIT were evident on two MRI scans rather than either DIS or DIT alone, according to Cox proportional hazards model analysis. Only the new criteria had an independent significant effect on conversion risk.

The new criteria, which are referred to in Table 1 as the “Swanton criteria,” were shown by the authors to be simpler to comprehend and easier to use than the previous McDonald criteria, while still demonstrating a high degree of specificity and accuracy. The new criteria also allow for diagnosis without the use of gadolinium-enhanced MRI. MS Swanton JK, Rovira A, Tintore M, et al. MRI criteria for multiple sclerosis in patients presenting with clinically isolated syndromes: A multicentre retrospective study. Lancet Neurol. 2007;6:677-686.

Table 1: Comparison of McDonald MRI Criteria to New Swanton Criteria

<table>
<thead>
<tr>
<th>Detection of DIS Abnormalities:</th>
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<tbody>
<tr>
<td><strong>McDonald 2001 Criteria</strong></td>
<td>Include at least three of the following:</td>
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<tr>
<td></td>
<td>- 9 T2 lesions or 1 GD-enhanced lesion</td>
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<tr>
<td></td>
<td>- ≥3 periventricular lesions</td>
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<td></td>
<td>- ≥1 juxtacortical lesion</td>
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<tr>
<td></td>
<td>- ≥1 posterior fossa lesion</td>
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<tr>
<td></td>
<td>A single spinal cord lesion can replace 1 brain lesion</td>
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<tr>
<td><strong>McDonald 2005 Revised Criteria</strong></td>
<td>Include at least three of the following:</td>
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<tr>
<td></td>
<td>- 9 T2 lesions or 1 GD-enhanced lesion</td>
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<td></td>
<td>- ≥3 periventricular lesions</td>
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<tr>
<td></td>
<td>- ≥1 juxtacortical lesion</td>
</tr>
<tr>
<td></td>
<td>- ≥1 posterior fossa lesion</td>
</tr>
<tr>
<td></td>
<td>A spinal cord lesion can replace an infratentorial lesion and any number of spinal cord lesions can be included in the final count</td>
</tr>
<tr>
<td><strong>Swanton 2007 Criteria</strong></td>
<td>≥1 lesion located in at least two of the following sites:</td>
</tr>
<tr>
<td></td>
<td>- periventricular, juxtacortical, posterior fossa, or spinal cord</td>
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<td></td>
<td>- All lesions in symptomatic region excluded in brainstem and spinal cord syndromes</td>
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<tr>
<th>Detection of DIT Abnormalities:</th>
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<tbody>
<tr>
<td><strong>McDonald 2001 Criteria</strong></td>
<td>1 GD-enhanced lesion ≥3 months post-CIS</td>
</tr>
<tr>
<td></td>
<td>The appearance of a new T2 lesion compared with a previous scan ≥3 months after CIS</td>
</tr>
<tr>
<td><strong>McDonald 2005 Revised Criteria</strong></td>
<td>1 GD-enhanced lesion ≥3 months post-CIS</td>
</tr>
<tr>
<td></td>
<td>The appearance of a new T2 lesion compared with a previous scan ≥30 days after CIS onset</td>
</tr>
<tr>
<td><strong>Swanton 2007 Criteria</strong></td>
<td>Any new T2 lesion appearing on MRI without time reference to a baseline scan</td>
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</tbody>
</table>

CIS=clinically isolated syndrome; DIS=dissemination in time; DIT-dissemination in space; GD=gadolinium; MRI=magnetic resonance imaging.
Continuing Education Conference Calendar

**JANUARY 18-20, 2008**
American Academy of Neurology 2008 Winter Conference  
Location: Miami, FL, USA  
Contact: Lori Strachota, Registration Services  
   Senior Administrator  
   Tel: 651-695-2706  
   Email: lstrachota@aan.com  
   Website: www.aan.com

**APRIL 12-18, 2008**
American Academy of Neurology 60th Annual Meeting  
Location: Chicago, IL, USA  
Contact: Member Services  
   Tel: 651-695-2717  
   Email: memberservices@aan.com  
   Website: www.aan.com

**MAY 28-MAY 31, 2008**
22nd CMSC Annual Meeting  
Location: Denver, CO, USA

**JUNE 23-24, 2008**
LEAD Summit 2008: Center for American Nurses Educational Conference and Annual Meeting  
Location: Washington, DC, USA  
Contact: American Nurses Association (ANA)  
   Tel: 800-274-4ANA  
   Website: http://nursingworld.org

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We want to hear from you. We welcome your comments and suggestions, as well any information on meetings and studies.

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**Teva Neuroscience** is dedicated to the MS nurse community and has supported scholarships for nurses, educational programs such as monographs, CE programs, IOMSN dinners, the *MS Exchange*, and *MS Nurse Counseling Points™*.  
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