MUL
TIPLE SCLEROSIS
THE
GREAT CRIPLER

By
Susan G. Rogers RN, BSN

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To the faculty of Washington State University:

The members of the committee appointed to examine the Intercollegiate College of Nursing research requirements and manuscript of Susan G. Rogers, find it satisfactory and recommend that it be accepted.

Chair: Lorna Schumann, PhD, RN, CS, NP-C, CCRN, ARNP, FNP, ACNP

Billie Marie Severtson, PhD, RN

Laura Hahn, MS, RNCS
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Multiple Sclerosis (MS) has been called “The Great Crippler of Young Adults”. It affects primarily those 20 to 40 years old. Multiple Sclerosis is variable in presentation and in the evolving course of the disease. It is the most common neurological disorder affecting young adults and is characterized by multi-focal plaques along the myelin sheath.

The differential diagnosis for MS is long and varied due to the vast amount of presenting symptoms that differ with each case. Diagnostic tests do not conclude that a person has MS, but are adjunct to assist in diagnosis.

Practitioners are in a unique position to educate both the patient and their loved ones. Patient education is crucial for the family, as well as the patient. It is helpful for patients with MS to have a healthcare team composed of a neurologist, nurse practitioner, nurse, social worker and family physician that can work together to provide the patient with the most up-to-date information, as well as resources available to help them cope with their situation.
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Introduction

Multiple Sclerosis (MS) has been known as “The Great Crippler of Young Adults” (Schapiro, 2001). It is in fact, the most common neurologic disorder affecting those 20 to 40 years old (Gumm, 2000). There are approximately 350,000 cases of MS in the United States (Gilden, 2001). It is believed that there are approximately 200 new cases in the United States every week (National MS Society, 2002).

Multiple Sclerosis is believed to be an autoimmune disease of the central nervous system. It is characterized by multifocal plaque-like lesions on the myelin sheath, the protective covering for the nerve cells (Boyden, 2000). These lesions form scar tissue on the nerves, causing impulses to slow and become more unpredictable, nerve impulses may even be blocked completely (NIH, 2002).

Recent MS research has focused on enhancing agents used with MRI and drug therapies to reduce inflammatory activity. There are more options for treating MS than ever before. As new treatments become available, it is essential for practitioners to keep up-to-date and informed on empirical evidence based medicine that they may share with colleagues, patients, and family members (Swain, 1996).

This article will investigate where MS is most prevalent, and where it is not usually found, how the normal physiological processes are altered by the disease, what signs and symptoms lead to the diagnosis of MS, what other diseases should be considered or ruled out, diagnostic tests and their cost effectiveness, as well as patient education, and treatments. The Health Belief Model (HBM), which originated around 1952, will be examined as a theoretical framework to assist practitioners in determining
which patients would most likely not use interventions, and preventive measures to reduce client reluctance (Stanhope & Lancaster, 1996).

The HBM was originally developed as a systematic method to explain and predict preventive health behavior. It focused on the relationship of health behaviors, practices and utilization of health services (Brown, 1999). The HBM attempts to predict health-related behavior in terms of certain belief patterns. The HBM includes three broad beliefs: 1) general health motivation, 2) perception of the threat-value of a specific disease, and 3) perception of the effectiveness of a specific health behavior for reducing that threat (Mirotznik et al., 1998). In the MS population, the focus is on promoting health, rather than, preventing disease. This holistic focus emphasizes on empowering the client with access to knowledge and resources that will facilitate choosing health behaviors to sustain and enhance their quality of life (Stuifbergen et al., 1999).

History of Multiple Sclerosis

Written descriptions of ailments resembling MS date back, as far as the 14th century. St. Lidwina of Holland, suffered symptoms that appear to be similar to MS. Later in the 19th century Sir Augustus d’Este of England kept a detailed journal of his symptoms, which began shortly after a bout of the measles (MS Ireland, 2002).

In the mid 19th century Robert Carswell found unusual lesions in the brain and spinal cord during a postmortem exam. Carswell recorded their appearance, but did not record any clinical associations with the lesions (MS Ireland, 2002).

Jean Cruveilhier published findings of lesions on the spinal cord. Cruveilhier also described the patient’s clinical symptoms that had progressed over the years. In 1840, Friedrich Theodor von Frerichs elaborated on the findings of Cruveilhier, and was the
first to recognize remissions, as an attribute of MS. He also described nystagmus as a symptom of the disease (MS Ireland, 2002).

Later in the 19th century, almost 40 years after the discovery of the lesions, Jean-Martin Charcot described them as “sclerose en plaques” and MS was established as a disease (MS Ireland, 2002). Charcot developed diagnostic criteria, that includes the now well-known Charcot’s triad, which consists of diplopia (double vision), ataxia (disturbances of balance or co-ordination), and dysarthria (difficulties with, or slurred speech) (Ferri, 1998).

Epidemiology

Although the exact cause of Multiple Sclerosis is not known, there are certain factors that may put one at higher risk for developing the disease. Multiple Sclerosis usually appears between the ages of 20 and 40 (McReynolds et al., 1999). It is twice as common in women as it is in men, which lead some to believe that hormonal influence may make women more vulnerable (Swain, 1996). Multiple Sclerosis is more common in the Caucasian race than in any other, especially in those of northern European descent. Some populations, such as Gypsies, Eskimos, and Bantus (of central Africa), never get MS. Native Indians of North and South America, the Japanese, and other Asian peoples have very low incidence rates (NIH, 2002).

It has been determined that geographic location plays a role in the prevalence of MS. Worldwide, the incidence of MS is twice the rate for those living above 40 degrees latitude, than those living below 40 degrees latitude (Aminoff, 1999). The incidence of MS above 40 degrees is 125/100,000 population, while for those below 40 degrees latitude it is 62.5/100,000 (Schaprio, 2001). Areas considered high risk are Northern
United States, Northern Europe, Canada, New Zealand, and Southern Australia (Swain, 1996). The age of the person living in the high-risk area is also a factor. If the person moves to the high-risk area after the age of 15, it is believed that they will not increase their chances of developing the disease. If the patient moves to the high-risk area before the age of 15, they will be considered to be at higher risk for the development of MS (Barnes, 2000). Multiple Sclerosis is not believed to be hereditary, but there is a genetic component that may increase the chances of getting the disease. The average person has a 1 in 1,000 chance of getting MS. If the person has a close relative (sibling or parent) with MS, their chances of developing MS increase to 1 in 50-100. Identical twins have a 1 in 3 chance of developing MS if their twin has already been diagnosed (Schapiro, 2001).

At one time a diagnosis of MS resulted in one classification, although some patients simply deteriorated faster than others. In 1996, the US National Multiple Sclerosis Society proposed a reclassification of MS. Consensus has been achieved with the new classification, which has been adopted worldwide (Weinshenker, 2000). Today, MS is categorized as benign, relapsing-remitting (RRMS), secondary progressive (SPMS), primary progressive (PPMS), and malignant.

The benign course is diagnosed, when a patient has had neurological symptoms that have been separated by space and time, and the patient has not had significant neurological disability due to MS, for 10 years after onset (Barnes, 2000). Exacerbations may involve a recurrence of previous symptoms or new symptoms; however, the symptoms generally do not worsen with each successive exacerbation (McReynolds et al., 1999).
The relapsing-remitting course is diagnosed when the patient has clearly defined relapses (with or without remissions) from onset, any fixed disability entirely due to incomplete recovery from relapses, and no evidence of progression between relapses (Barnes, 2000). The psychological effect of this pattern of disease progression leaves the person on constant alert for his or her next relapse (McReynolds et al., 1999).

The secondary progressive course begins initially with the RRMS, then develops into a steadily progressive course with or without superimposed relapses, minor remissions and plateaus (Barnes, 2000). Of the 70 – 75% of people, who start with RRMS, more than 50% will develop SPMS within 10 years; 90% within 25 years (National MS Society, 2002).

The primary progressive course is progressive from the onset, with minor plateaus and temporary improvements allowed, but no clear relapses (Barnes, 2000). The symptoms generally do not remit. Fifteen percent of people with MS are diagnosed with PPMS, although the diagnosis usually needs to be made after the fact – when the person has been living for a period of time with progressive disability, but not acute attacks (National MS Society, 2002).

The malignant course of MS is extremely rare. It is an aggressive, persistent, and life-threatening form of the disease with no known treatment. Malignant MS is characterized by an intense incident of initial symptoms followed by a brief and precipitous progression, which ultimately renders the person incapacitated (McReynolds et al., 1999).

According to Noseworthy et al., (2000), frequent relapses in the first two years, a progressive course from the onset, male sex, and early, permanent motor or cerebellar
findings are independently, but imperfectly, predictive of a more severe clinical course. Women and patients with predominantly sensory symptoms and optic neuritis have a more favorable prognosis.

Pathophysiology

Multiple Sclerosis is the disease prototype for the demyelinating diseases (Waxman, 2000). Myelin is the phospholipid-protein that acts as an electrical insulator over neurons. This insulation increases the velocity of impulse conduction (Thomas, 1997). As inflammation occurs on the myelin sheath, scars or plaques appear, resulting in slowing or even blocking of neuronal current (NIH, 2002). There are three main theories as to how demyelination may develop; autoimmune, viral, and genetic. Many researchers and clinicians believe that these aspects of disease development may all be interrelated (National MS Society, 2002).

Although it has been established that an immune response takes place in MS, the antigen has yet to be identified (Ascherio et al., 2001). In a healthy person, the immune system’s T-cells will cross the intact blood brain barrier when activated by an infectious cause, inspect the central nervous system (CNS), then depart. For some unknown reason, in MS, the T-cells remain in the CNS. The existence of T-cells in the CNS facilitates entry by other leukocytes resulting in an inflammatory process (Boyden, 2000). This inflammation leads to the destruction of myelin and oligodendrocytes, which are the cells in charge of myelin production. The inflammation that destroys the myelin sheath does not damage the axon itself, and a higher than normal number of sodium channels appear spontaneously in demyelinated fibers (Waxman, 2000). This may explain the
of MS patients will have a bout of optic neuritis at some point. This has led to general recognition of optic neuritis as an early sign of MS, especially if tests also reveal abnormalities in the patient’s spinal fluid (NIH, 2002). A research study funded by the National Eye Institute of the National Institutes of Health, assessed the five-year risk of and prognostic factors for the development of clinically definite Multiple Sclerosis following optic neuritis. This study found that optic neuritis, along with a positive MRI were strong predictors of clinically definite MS. Sixteen percent of the patients with no MRI lesions and 51% of patients with three or more MRI lesions had been confirmed with MS after 5 years. Some type of eye movement disorder, such as slowing of adduction, will develop at some time during the disease in 75% of MS patients (Optic Neuritis Study Group, 1997).

Fatigue is also an early and continuing symptom of MS. Many patients describe fatigue that is worse in the afternoon and is accompanied by physiologic increases in body temperature (Noseworthy et al., 2000). The fatigue may develop as a continuous and persistent exhaustion, or it may be triggered by physical effort, and improve with rest. Fatigue may be a result of chronic stimulation of the immune system, infection, medication, extra effort and energy required to compensate for a handicap, disrupted sleep patterns, depression, or nerve fiber activity (Rumrill et al., 1998). Fatigue is the most common cause of disability in those suffering from MS (McReynolds et al., 1999)

Patients with MS experience muscle weakness in their arms or legs (usually the lower extremities) and trouble with coordination and balance at some stage of their illness. These symptoms may be severe enough to keep them from walking or even standing and may eventually lead to paralysis (Barnes, 2000). Patients often confuse
motor and sensory symptoms: they may, for example, say that a limb is ‘numb’ when it is weak, but much more commonly will say a limb is ‘weak’ when, actually it is affected by a sensory disturbance (Barnes, 2000).

Depression is also a common finding in MS. Approximately 5% of MS patients may also encounter episodes of inappropriate euphoria and despair, know as the ‘laughing/weeping syndrome’. This disorder is believed to be due to the demyelination of areas in the brain stem, which control facial expression and emotion. This is usually found only in severe cases (Barnes, 2000). In comparison with other neurological diseases, there is an increased rate of depression in MS patients. The rate of suicide in MS patients is seven times that of the normal population (Taylor & Taylor, 1998). In addition, about 10% of MS patients suffer from more severe psychotic disorders such as manic-depression and paranoia (NIH, 2002).

Spasticity, the increased tone or contractions of muscles causing stiff and awkward movements is a common symptom (Thomas, 1997). Spasticity needs to be approached with caution. Treating spasticity with muscle relaxants may be counterproductive in some patients. Some patients are able to walk with difficulty due to the muscle spasticity (Barnes, 2000). When spasticity is taken away with medication, the weakened muscles may no longer be able to support the body weight, possibly confining the patient to a wheelchair.

Impairment of pain or loss of sensation may occur, or more commonly, the patient may have transient abnormal sensory feelings such as numbness, prickling, or tingling. Most patients learn to ignore the prickling or tingling sensations. Painful sensory symptoms are more distressing (Barnes, 2000).
It is believed that from 30-65% of MS patients also suffer pain. There are several pain syndromes that are more common in the MS patient than in the general population. Trigeminal neuralgia, or Tic douloureux, is 4000 times more common in MS patients (Barnes, 2000). Another pain syndrome common in MS is myelopathic pain, which results from damage to the spinal cord sensory pathways. This can present as a constricted feeling area around a limb or the trunk, producing pain or even making it difficult to breathe (Noseworthy et al., 2000).

Although MS patients may be more prone to certain pain syndromes, they are not exempt from painful conditions that affect the general population. Multiple Sclerosis patients are also more likely to experience an amplitude of everyday aches and pains that result from over stressed joints and poor posture due to the difficulty in maintaining correct stance or gait (Schapiro, 2001). However, they are less capable of coping with this additional stress, making it essential that practitioners inquire about pain, assess and manage it (Barnes, 2000).

Ataxia is defective muscle coordination, which is exhibited when voluntary muscular movements are attempted (Thomas, 1997). Ataxia is a symptom of cerebellar demyelination, which may include, speech, walking and balance. When cerebellar structures are affected, a distinctive gait is produced that is wide-based with erratic staggering (Barnes, 2000).

Tremors are an involuntary movement of part or parts of the body resulting from alternate contractions of opposing muscles (Thomas, 1997). These tremors are uncontrollable by the MS sufferer. The trembling may be fine or coarse, rapid or slow,
and may appear with movement or improve when the body part is voluntarily exercised (Barnes, 2000).

Speech disturbances such as slowed, labored, or slurred speech may evolve (National MS Society, 2002). Swallowing may also be affected, in acute or progressive cases, the patient will eventually become unable to speak or swallow (Barnes, 2000).

Bladder dysfunction is a common symptom of MS. The most common bladder problems encountered by MS patients are urinary frequency, urgency, or incontinence (NIH, 2002). Urinary retention can also be a problem in MS, as it often leads to frequent urinary tract infections, which, eventually may lead to renal complications (Barnes, 2000). Bladder relaxants have been found to be helpful for these patients. Self-catheterization is another treatment that may be useful for this type of bladder problem (Schapiro, 2001).

Bowel dysfunction in the MS patient is usually limited to constipation and generally responds well to routine treatments. There are many pharmaceutical therapies used in MS that cause constipation (Barnes, 2000).

Sexual dysfunction is a common complaint for both male and female MS sufferers. The inability to have an orgasm often has an emotional basis, rather than a physical one (McReynolds et al., 1999). Depression, drug regime, persistent pain or poor self-worth can all contribute. For all these symptoms there are possible treatments. The incidence of male erectile dysfunction in MS is about 60%. There are various treatments for this that can be very effective (Barnes, 2000).

Approximately half (40-60%) of the patients with MS will experience cognitive abnormalities. These may include difficulties with concentration, memory, attention and
difficulty in learning and recalling new information (DeLuca et al., 1998). Often these symptoms are mild and only detected with comprehensive testing. Deficits tend to become more apparent as the information the patient is processing becomes more complex. Usually the mental capacity is unchanged, but the processing speed is found to be sluggish (Taylor & Taylor, 1998). Fatigue may also add to processing difficulties.

The Health Belief Model can be helpful for the primary care provider in understanding how patients perceive their symptoms, and therefore, how they may act upon them. Limitations of time, energy and mobility influence those with chronic conditions, such as MS, to make choices they perceive as most supportive of their quality of life. In order to be effective and enduring, health promotion interventions must be individualized and tailored to the patient's abilities, resources, values and perspective on quality of life (Stuifbergen et al., 1999). The healthcare team can be beneficial in customizing interventions to meet the needs of each patient.

Differential Diagnosis

With the advent of therapies that may alter the progression of relapsing-remitting MS, it is more important than ever to quickly and accurately diagnosis the disease so that treatment may be as beneficial as possible. There is increasing evidence that permanent tissue damage occurs early in the course of MS, making it important that treatment trials include patients in the earliest stages of the disease (Brex et al., 2001). Many ailments can create a medical portrayal that in many cases may look like MS. The differential diagnosis for MS is extremely broad, because the disease has a wide range of severity.

Multiple Sclerosis is diagnosed as “Possible MS”, by clinical symptoms, with at least two episodes, separated by time and anatomic location. The diagnosis is upgraded
to “Probable MS” or “Definite MS” by additional MRI findings that identify plaques or lesions in multiple areas of the central nervous system (NIH, 2000). Several symptoms of MS are difficult at best for practitioners to elucidate, thus delaying diagnosis until substantial evidence presents itself.

There are numerous diseases or ailments that may present similarly to MS. These include; infectious, genetic, autoimmune, metabolic, neoplastic, structural, psychiatric, toxic, vascular, as well as variety of ambiguous illnesses (Noseworthy et al., 2000). Table 2 presents a more complete list of possible diagnoses in the differential of Multiple Sclerosis.

A number of other diseases may produce symptoms similar to those seen in MS. Other conditions with an intermittent course and MS-like lesions of the brain’s white matter include polyarteritis, lupus erythematosus, syringomyelia and tropical leukoencephalopathy (NIH, 2002). These conditions can mimic the acute state of an MS attack. The provider will also need to rule out stroke, neurosyphilis, spinocerebellar ataxia, pernicious anemia, diabetes, Sjogren’s disease, and vitamin B12 deficiency (NIH, 2002).

**Algorithm**

Diagnosis of MS is not well defined, as its presentation varies extraordinarily from case to case. Presenting symptoms are the first clues to the complicated puzzle that is MS. The patient may present with a wide variety of sensory or motor disturbances. Onset may be, as sudden as a few minutes, which may resemble a stroke or as gradual as several months, which may give the impression of a tumor (Noseworthy et al., 2000).
The diagnosis of MS is a clinical one. There are no specific tests that can determine whether or not a person has MS. Evidence must be found showing damage to the central nervous system in more than one area, and on more than one occasion (Barnes, 2000). The objective exam will add another piece to the picture, which helps determine which tests may be needed to rule-out or confirm other possibilities for the patient's symptoms. When the presenting symptoms along with the objective exam indicate the possibility of MS, there are diagnostic tests that may be helpful in making a probable or confirmatory conclusion (Swain, 1996). Figure 1 presents an algorithm of symptoms, clinical findings, and diagnostic tests that may be helpful in the definitive diagnosis of Multiple Sclerosis.

Diagnostic Testing Specificity and Sensitivity

Specificity and sensitivity of current diagnostic procedures will be examined in Table 3. No set of diagnostic criteria can identify every case of MS, because of the enormous clinical variability of the disease (Poser & Brinar, 2001). Magnetic Resonance Imaging (MRI) is currently the most beneficial tool available for confirming the diagnosis of MS in patients or ruling out other possible causes (Filippini et al., 1994). With MRI, lesions of the head, as small as 3-4 mm can be detected, as well as disease load, activity, and progression (Leist et al., 2001). New developments in MRI techniques are currently underway, and undoubtedly MRI will remain an important adjunct in MS diagnosis (Racke, 2001).

Cerebrospinal fluid analysis (CSF) is not routinely performed in all suspected MS cases. Agarose electrophoresis of the spinal fluid may reveal oligoclonal bands, indicating a non-specific immune response in the central nervous system (Ferri, 1998).
The discovery of oligoclonal bands in the spinal fluid is helpful in establishing an inflammatory or immunological etiology. CFS analysis may also show increased gamma globulin, increased total protein, increased white cells, or the presence of myelin basic protein, which is elevated in acute attacks and is indicative of myelin destruction (Ferri, 2002). The CFS is currently considered the best available source of cells infiltrating the central nervous system (Gestri et al., 2001).

Evoked potentials are measured electrical reactions to a certain stimulus. These evoked potentials may be applied to various neurological systems, such as, visual, somatosensory or peripheral nerves (Thomas, 1997). The visual evoked potential is sometimes used to assess nerve fiber conduction. In this instance, conduction velocity will be slow with myelin loss or destruction. The usefulness of evoked potentials in the diagnosis of multiple sclerosis is limited by its relatively low sensitivity to subclinical lesions. However, they are still useful tools in assessing the integrity of afferent and efferent pathways and to quantify the severity of white matter involvement (Leocani et al., 2000).

Blood tests are often performed to rule out other possible causes for the presenting symptoms. A full hematological and biochemical screen, ESR and autoimmune profile should be performed on all patients. Serological tests for syphilis, vitamin B12, folate and protein electrophoresis may be appropriate (Barnes, 2000).

Cost Analysis for Diagnosis of Multiple Sclerosis

Approximate costs for diagnostic tests associated with MS are provided in Table 4. All tests are not mandatory for diagnosis, but early identification of MS enables the patient to begin therapy that may reduce relapses and help retain neurologic function
Magnetic resonance imaging (MRI) is expensive, but its superiority is unquestioned (Swain, 1996).

Patient Education and Preparation

Patients are often in shock and disbelief when confronted with the possibility of having MS. A newly diagnosed patient may experience denial, anger, bargaining, and depression, before they reach acceptance (McReynolds et al., 1999). It is important to educate the patient about their disease, so they understand their options and make choices that will be best for their particular situation. It is helpful for the patient to have a team of health care members such as, neurologist, nurse practitioner, nurse, social worker, and family physician who can work together to help the patient become more informed about their illness and also inform them of resources available to them (Noseworthy et al., 2000).

The Health Belief Model can be a valuable tool to practitioners for both the patients in denial of their disease, as well of those who have accepted the diagnosis. This theoretical framework assumes that development of knowledge and skills to reduce barriers, as well as, enhance resources and self-efficacy, will result in greater participation in health promoting behaviors (Stuifbergen et al., 1999).

Freeman & Thompson (2000), found that 45% of MS patients did not receive any community services other than contact with their general practitioner. Thirty-nine percent of people with moderate and 12% with severe disability failed to receive assistance from community services. The services that were received were with a nurse (17%), care attendant (33%), physiotherapist (23%), occupational therapist (21%), and
social worker (10%). Table 5 provides a list of topics for patient education that may be helpful in understanding tests, assistance, and support systems.

Overview of Treatments

Supportive care includes rest and the treatment of any precipitating illness, such as a urinary tract infection, which may add to the misery of the relapse (Barnes, 2000). There are many therapies offered for the treatment of symptoms, such as spasticity, pain, bladder dysfunction, fatigue, depression and weakness shown in Table 6.

Treatment of MS varies with presentation. Primary progressive MS currently has no treatment for decreasing relapses (Noseworthy et al., 2000). Therapy is presently aimed at treating symptoms. Steroids are used on occasion for acute flares. Because steroids can produce numerous adverse side effects, such as, acne, weight gain, seizures and psychosis, long-term use is not recommended (Schapiro, 2001). Novantrone (Mitoxantrone) has been approved by the FDA for the treatment of secondary-progressive MS (initial period of relapsing-remitting MS, followed by a worsening to a progressive state) (National MS Society, 2002).

Specific Pharmacologic Treatments

There are several therapies available for relapsing-remitting patients such as, interferon beta 1b (Betaseron), interferon beta 1a (Avonex), and glatiramer acetate (Copaxone). While these agents are promising, studies have found a discontinuation rate of 45%, a 26% rate of changing or switching from one agent to another, with over 1/3 of patients rating their experience with these drugs as negative (Holland et al., 2001).

A British study evaluating the cost-effectiveness of interferon beta-1b found that interferon beta-1b produces important, occasional short-term quality-of-life gains, but
because these are infrequent, they translate into small quality-adjusted, life-year gains. With large net costs, interferon beta-1b has a high cost per quality-adjusted life-year gained (Parkin et al., 2000).

Interferons have been shown to reduce the relapse rate for MS patients (Tselis & Lisak, 1999). There are certain criteria that are recommended to be a candidate for interferon therapy. The patient should be over the age of 18 and preferably under the age of 55. The patient must have a definite diagnosis of relapsing-remitting MS with no indication of progression. The patient must be able to ambulate without assistance or rest for 100 meters. The patient must have a history of at least 2 relapses in the past year severe enough to warrant therapy, according to a physician (Barnes, 2000). The patient must have no other medical problems contraindicating the use of interferon. The United States National Multiple Sclerosis Society (1998), recommends that initiation of therapy be advised, as soon as possible following a definite diagnosis of MS and determination of a relapsing course.

Interferons are known to have multiple immuno-regulating actions and are given by injection, intramuscularly and subcutaneously (Noseworthy et al., 2000). The injections are generally self-administered, though some patients may require assistance due to disability (Swain, 1996). Common side effects at injection sites include transient redness or minor swelling. Less frequent side effects include persistent induration, discoloration, and skin necrosis (Swain, 1996). Injections into the buttocks seem to be better tolerated than injections elsewhere. Flu-like symptoms have been reported by 52-61% of patients within a few hours of injection. Other complications include increased liver enzymes, headache, weakness, conjunctivitis, nausea, vomiting, diarrhea,
constipation, myalgia, and fever (Barnes, 2000). Interferon beta 1a (Avonex), available since 1993, is given intra-muscular 30 mcg once weekly; interferon beta 1b (Betaseron), approved in 1993, is given subcutaneous 0.25mg (8 million IU) every other day (Gumm, 2000).

Glatiramer acetate (Copaxone) contains synthetic polypeptides, which mimic the structure of myelin protein. It is believed that glatiramer acetate acts as a myelin decoy to block the damaging T-cells in the body (Johnson et al., 1998). Glatiramer acetate is injected SC 20 mg/day. Side effects include allergic reaction, anxiety, chest pain, hypertension, coughing, injection site reactions and weight gain (Teva Pharmaceutical, 2001). Neither interferon or glatiramer acetate therapy is recommended during pregnancy. Each of the drug manufactures, interferon beta 1a (Avonex), interferon beta 1b (Betaseron), and glatiramer acetate (Copaxone) all have adherence support programs and financial support programs that reportedly increase compliance in MS patients (see Table 7).

Future Therapies

There are many new therapies that are being considered as treatment for MS. Investigators are looking at the possibility of a MS vaccine, peptide therapy, protein antigen feeding, cytokine blocking, total lymphoid irradiation, plasma exchange, bone marrow transplantation, bee venom, and remyelination possibilities.

With a MS vaccine, the T-cells are removed, inactivated, and injected back into animals with experimental allergic encephalomyelitis (EAE). This procedure results in destruction of the immune system cells that were attacking myelin basic protein (NIH, 2002).
Peptide therapy is based on evidence that the body can mount an immune response against the T-cells that destroy myelin. Myelin-attacking T-cells are identified with myelin-recognizing receptors on the cells’ surface. A fragment of those receptors is then injected into the body. The immune system “sees” the injected peptide as a foreign invader and launches an attack on any myelin-destroying T-cells that carry that receptor (NIH, 2002).

Protein antigen feeding is similar to peptide therapy. Antigens that trigger an immune response when injected can encourage immune system tolerance when taken orally. Studies have shown that when rodents with EAE are fed myelin protein antigens, they experience fewer relapses (NIH, 2002).

Cytokines are powerful chemicals produced by T-cells. Scientists are studying a variety of substances that may block harmful cytokines, such as those involved in inflammation, or that encourage the production of protective cytokines (NIH, 2002).

Total lymphoid irradiation is a process in which the MS patient’s lymph nodes are irradiate with x-rays in small doses over a few weeks to destroy lymphoid tissue. This tissue is actively involved in tissue destruction in autoimmune diseases. The risk of potentially dangerous side effects dictates further research is necessary to determine what, if any role, this should play in the management of MS (NIH, 2002).

Some studies focus on strategies to reverse the damage to myelin and oligodendrocytes, both of which are destroyed during MS attacks. Scientists now know that oligodendrocytes may proliferate and form new myelin after an attack. Investigators are looking at how drugs used in MS trials affect remyelination. Studies of animal models indicated that monoclonal antibodies and two immunosuppressant drugs,
cyclophosphamide and azathioprine, may accelerate remyelination, while steroids may inhibit it (NIH, 2002).

Plasma exchange or plasmapheresis is a procedure in which blood is removed from the patient, and the plasma is separated from other blood substances, which may contain antibodies and other immunologically active products. These other blood substances are discarded and the plasma is then transfused back into the patient (NIH, 2002).

Bone marrow transplant is a procedure in which bone marrow from a healthy donor is infused into patients who have undergone drug or radiation therapy to suppress their immune system so they will not reject the donated marrow. This therapy carries the risk of potentially severe side effects (NIH, 2002).

Researchers have identified more than 40 active compounds in bee venom. The varied effects that the active compounds in bee venom have on the immune system and inflammatory cascade have led to experimentation with its potential therapeutic use in a number of disorders with strong inflammatory and immunologic components. One disorder of particular interest is multiple sclerosis. The American Apitherapy Society has collected case reports on more than 1300 patients with MS who reported subjective improvement in stability, fatigue, and muscle spasm following bee venom treatment (D'Epiro, 1999).

Dietary factors are believed to contribute to the prevalence of MS. MS has been found to be more prevalent in those people whose diets are high in saturated fats (Schapiro, 2001). Many researchers now believe that a diet low in saturated fats is key to the prevention of MS. Lombard & Germano, (2000) believe that diet, along with various
supplements can be helpful in treating or preventing MS, as well as other ailments. They have what they call a "Cocktail Approach" to treating MS. This combination of vitamin supplements, herbal extracts and prescription drugs should be prescribed and monitored by a health care provider.

In the past there have been ineffective and even potentially hazardous therapies that have been proclaimed as treatments for MS. Some of these include: snake venom injections, electrical stimulation of the spinal cord, removal of the thymus, oxygen chambers, beef heart and swine pancreas extract injections, hysterectomy, removal of silver containing dental fillings and surgical implantation of swine brain into the abdomen (NIH, 2002). None of these therapies have been successful in treating the symptoms of MS.

Conclusion

Multiple Sclerosis is variable in presentation and in the evolving course of the disease. It is the most common neurological disorder affecting young adults and is characterized by multi-focal plaques or lesions along the myelin sheath.

Descriptions of MS date back, as far as the 1400's, while it was not established as a disease until the 19th century. Knowledge and treatment modalities have changed dramatically over the past 100 years, and there are more options for those who suffer from MS than ever before.

Many factors are involved that place a person at higher risk for developing the disease. Some of the variables include: age, gender, race, geography, diet, and genetics.

There are several theories as to how MS develops. The most popular theories are autoimmune, viral, and genetic. Most researchers believe that these three theories are
most likely interrelated. As inflammation occurs, oligodendrocytes, which are in charge of myelin production, are destroyed, leaving gaps in the myelin sheath, resulting in MS symptoms.

Clinical manifestation of symptoms vary with the location and severity of the plaques. Common presenting symptoms include: vision disturbances, fatigue, muscle weakness, depression, spasticity, impairment of pain, temperature and touch, pain, ataxia, speech disturbances, bowel/bladder dysfunction, sexual dysfunction and cognitive abnormalities.

The differential diagnosis for MS is long and varied due to the vast amount of presenting symptoms that can differ with each case. Areas to consider include: infectious, genetic, inflammatory, metabolic, neoplastic, structural, toxic and vascular.

Diagnostic tests do not make a definitive conclusion about having MS, but are adjunct to assist in diagnosis. Common tests are MRI, CSF analysis, visual evoked potentials, and blood tests.

The Health Belief Model (HBM) is a framework with a holistic focus to help practitioners put an emphasis on empowering patients with disabling conditions. The HBM assumes that access to knowledge and resources provides patients with the basis for a choice of health behaviors to sustain and enhance their quality of life (Stuifbergen et al., 1999). This framework helps practitioners understand why some people take specific actions to avoid illness, whereas others fail to protect themselves (Stanhope & Lancaster, 1996). In general, behavior depends on how much an individual values a particular goal. If the goal is to avoid a health problem, the individual must feel personally vulnerable to
a problem judged to be potentially serious, and he or she must feel that specific action
will be beneficial in reducing the health threat (Poss, 2001).

Practitioners are in a unique position to evaluate their patients and determine
which of them may be in greater need of education and information about their disease
and their disease process. This intervention may prove invaluable, with greater patient
participation in their own care, along with greater compliance to prescribed therapies.

Patient education is crucial for the family, as well as the patient. It is helpful for
patients with MS to have a healthcare team composed of a neurologist, nurse practitioner,
nurse, social worker and family physician that can work together to provide the patient
with the most up-to-date information, as well as resources available to help them cope
with their situation.

Treatment modalities for MS have progressed rapidly in the last 15 years. There
are more options available than ever before, with the promise of even greater discoveries
in the future.
Table 1

Clinical Features

Possible Symptoms of Multiple Sclerosis

- Vision disturbances
- Fatigue
- Muscle weakness
- Depression
- Spasticity
- Impairment of pain, temperature, touch
- Pain (moderate to severe)
- Ataxia
- Tremor
- Speech disturbances
- Bladder dysfunction
- Bowel dysfunction
- Sexual dysfunction
- Cognitive abnormalities

(Barnes, 2000)
(Noseworthy et al., 2000)
(Swain, 1996)
(Ferri, 2002)
Table 2
Differential Diagnosis

**Infectious**
- Lyme disease
- Syphilis
- Eales’ disease
- CNS infections
- Human immunodeficiency virus
- Herpes zoster
- Leprosy
- Diphtheria

**Genetic**
- Leber’s optic atrophy
- Hereditary paraplegias
- Friedreich’s ataxia
- Amyotrophic lateral sclerosis (ALS)
- Neurofibromatosis
- Charcot-Marie-Tooth disease

**Autoimmune**
- Systemic lupus erythematosus
- Sjogren’s syndrome
- Behcet’s disease
- Sarcoidosis
- Chronic inflammatory demyelinating polyradiculopathy
- Antiphospholipid-antibody syndrome
- Guillain-Barre syndrome

**Metabolic**
- Nutritional deficiencies
  - (thiamin, vitamin B12, folic acid)
- Leukodystrophies
- Myxedema

**Neoplastic**
- Brainstem tumor
- Cerebellar tumor
- Spinal cord tumor
- Central nervous system lymphoma

**Structural**
- Arnold-Chiari malformation
- Ruptured intervertebral disc
- Compression of spinal cord or peripheral nerves

**Psychiatric**
- Malingering

**Toxic**
- Medications (isoniazid, lithium, nitrofurantoin, gold, cisplatin, amitriptyline, amiodarone, metronidazole, dapsone, sulfonamides, diazepam, chloramphenicol, etc.)
- Chemicals (lead, arsenic, cyanide, mercury, organophosphates, etc.)

**Vascular**
- Spinal dural arteriovenous fistula
- Central nervous system vasculitis
- Small cerebral infarctions

**Other**
- Nonhereditary ataxias
- Spondylous myelopathies
- Syringomyelia
- Diabetes mellitus
- Alcohol

(Barnes, 2000)
(Noseworthy et al., 2000)
(Ferri, 2002)
(Swain, 1996)


<table>
<thead>
<tr>
<th>Type of Test</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Magnetic resonance imaging</strong></td>
<td>Accuracy of 80%</td>
<td>96%</td>
</tr>
<tr>
<td>Type of test: Magnetic field study</td>
<td>Having more abnormal criteria</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Strongly predicted conversion to clinically definite MS</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Barkhof et al., 1997)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>81%</td>
<td>96%</td>
</tr>
<tr>
<td>(Offenbacher et al., 1993)</td>
<td>(Offenbacher et al., 1993)</td>
<td></td>
</tr>
<tr>
<td><strong>Cerebrospinal fluid analysis</strong></td>
<td>83%</td>
<td>79%</td>
</tr>
<tr>
<td>Type of test: Fluid analysis</td>
<td>(Richard et al., 2001)</td>
<td>(Richard et al., 2001)</td>
</tr>
<tr>
<td><strong>Evoked potential</strong></td>
<td>Low sensitivity</td>
<td>53%</td>
</tr>
<tr>
<td>Type of test: Electrical stimulus</td>
<td>(Leocani et al., 2000)</td>
<td>(Filippini, et al, 1994)</td>
</tr>
<tr>
<td><strong>Blood tests</strong></td>
<td>Varies</td>
<td>Varies</td>
</tr>
<tr>
<td>Type of test: Blood</td>
<td>with test</td>
<td>with test</td>
</tr>
<tr>
<td>Test</td>
<td>Cost Range</td>
<td></td>
</tr>
<tr>
<td>-------------------------------------</td>
<td>-------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Magnetic Resonance Imaging</td>
<td>~$850.00 - $2000.00</td>
<td></td>
</tr>
<tr>
<td>Cerebrospinal Fluid Analysis</td>
<td>~$119.00 for Analysis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>~$40.00 - $50.00 for Lumbar Puncture</td>
<td></td>
</tr>
<tr>
<td>Evoked Potential</td>
<td>~$250.00 - $300.00</td>
<td></td>
</tr>
<tr>
<td>Blood Tests</td>
<td>Varies with test</td>
<td></td>
</tr>
</tbody>
</table>
Table 5
Patient Education and Preparation

What should the patient know about MRI?

Magnetic resonance imaging is currently the best paraclinical tool for the diagnosis of Multiple Sclerosis (Racke, et al., 2001). A significant advantage of MRI testing is that serial studies can be performed without any risk. The disadvantage of MRI is that any metal (joint replacements, pacemakers, pins from open reduction, etc.) will result in MRI image degradation and may endanger you. Movement during the scan may cause artifacts on the MRI.

You will be asked to remove all metal objects before the scan. The magnetic field can also damage credit cards and watches. During the procedure you may hear a thumping sound; earplugs are available. There are no fluid or food restrictions prior to the MRI. For comfort you will be instructed to empty your bladder before the scan begins. You will be asked to lie very still during the procedure. During the procedure you will be able to talk to the staff through a microphone. Contrast medium may be used during the test, which last from 30 to 90 minutes. There is no special care needed after the procedure (Pagana & Pagana, 2000).

What should the patient know about lumbar puncture?

A needle is placed in the subarachnoid space of the spinal column. Examination of the spinal fluid evaluates the presence blood, bacteria, malignant cells, oligoclonal bands, etc. to be used as an adjunct to diagnosis. Lumbar puncture is contraindicated in people with increased intracranial pressure, severe degenerative vertebral joint disease, or infection near the puncture site. Complications of lumbar puncture include: headache due to persistent CSF leak, introduction of bacteria into the CSF, causing meningitis,
weakness or paralysis of the lower legs due to spinal block, or bleeding, bruising or swelling, due to oozing of blood or cerebrospinal fluid from the puncture site.

There is no fasting required before the procedure, and you must lie still throughout the process. You are usually placed in a fetal position while local anesthetic is injected. A spinal needle is then placed through the subarachnoid space. Three sterile test tubes are filled with 5 to 10 ml of spinal fluid. This procedure is performed by a physician and usually takes about 20 minutes. You are then placed prone with a pillow under the abdomen to increase the intra-abdominal pressure, which will indirectly increase the pressure in the tissues surrounding the spinal cord. You will be encouraged to increase fluid intake and to maintain a reclining position for up to 12 hours to avoid spinal headache (Pagana & Pagana, 2000).

**What does the patient need to know about visual evoked potentials?**

In visual evoked potentials, an electroencephalogram is recorded as you watch a pattern projected on a screen. The characteristics of the waveforms are compared with normal waveforms. In this procedure, important information is obtained regarding the function of the visual operation in transmitting stimuli to the brain. The visual evoked potential is a particularly useful tool for demonstrating lesions of the optic nerve of all kinds (Barnes, 2000).

For the electroencephalography, 21 recording needle electrodes are applied to the scalp in specific clusters, using electric paste to promote conduction. The electroencephalography procedure may take several hours. You will be instructed to avoid caffeine before the test, as stimulants alter the test. A light meal and sufficient fluid intake is also advised, as low blood glucose can also change results. You will be
instructed to wash your hair thoroughly before the test, and that a prickly sensations is felt as electrodes are attached to scalp. You will be asked not to move or talk during the test (Malarkey & McMorrow, 1996).

**What does the patient need to know about blood testing?**

Blood tests are often done to rule out other pathologic causes for your presenting symptoms. Blood draws generally take no longer than a few minutes to perform. Samples are usually taken from an antecubital vein. A tourniquet is usually tied above the vein to be used. Depending upon tests to be drawn, you may be instructed to discontinue all food and fluids for up to 8 hours before the test as eating may change the value of certain tests. There are no post-test considerations (Malarkey & McMorrow, 1996).

**What are my chances of developing MS if I currently have optic neuritis?**

Optic neuritis is a common target site in MS. Fifty-five percent of MS patients will have an attack of optic neuritis at some time or other and it will be the first symptom of MS in approximately 15% those diagnosed with MS (NIH, 2002). In a study funded by the National Eye Institute of the National Institutes of Health, it was found that 16% of the patients, with no MRI brain lesions, developed MS within five years. Of those with three or more MRI brain lesions, 51% developed MS within five years (Optic Neuritis Study Group, 1997).

**What support systems are available to me?**

Your physician, nurse practitioner, neurologist, nurse, and social worker are excellent resources for information regarding MS. The National Multiple Sclerosis Society may have a local chapter office in your area, or they are available by phone or on
the web at www.nationalmssociety.org. The National Multiple Sclerosis Society has information regarding many MS topics, as well as community resources and referrals to local neurologist and counselors. They have programs available for; medical equipment assistance, counseling, self-help groups, exercise programs, in-home assessments, library, educational seminars, long-term care, accessibility and employment, political advocacy, community presentations, professional education, newsletters and MS clinics (National MS Society, 2002).

**What are some important tips to remember?**

**Maintain your health.** Avoid anyone with a cold or the flu. Be sure to get regular checkups and vaccinations as recommended by your health care provider.

**Avoid stress.** Know your personal limits so you can balance the demands and pleasures of home, work, and social events. Don’t try to solve everyone else’s problems.

**Avoid heat.** An enemy of anyone with MS, heat may trigger new neuralgia type symptoms, as your body temperature climbs. Always avoid hot water while bathing, because it increases a feeling of fatigue.

**Avoid fatigue.** Consider adapting your home and work environments to help conserve energy. Alternate heavy housework with light tasks or hire someone else to clean if you can afford to. Ask your primary care provider to recommend an occupational or physical therapist who can give you practical advice and tools to conserve energy.

**Exercise.** Moderate exercise will help improve and optimize your muscle strength, but know your limits and avoid fatigue by resting frequently. Swimming or water therapy is a great choice, especially because it eliminates overheating.
Eat a balanced diet. Eating sensibly helps decrease the chance of contracting other illnesses that could trigger an exacerbation.

Foster enriching relationships. Join a support group. Enjoy the quiet times that nourish supportive relationships, and avoid negative people.

Maintain your independence in a sensible way. Independence is great, but everyone needs help at times. Be willing to admit when you reach your limits, and ask for help when you need it.

Use assistive devices. Canes and walkers are your tools; use them to conserve energy.

Accept yourself as you are. Accept who you are now and know that you may face changes later (www.springnet.com).
### Table 6

Therapies for Symptoms

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spasticity</td>
<td>Baclofen (Lioresal)</td>
</tr>
<tr>
<td></td>
<td>Tizanidine (Zanaflex)</td>
</tr>
<tr>
<td></td>
<td>Diazepam (Valium)</td>
</tr>
<tr>
<td></td>
<td>Clonazepan (Klonopin)</td>
</tr>
<tr>
<td></td>
<td>Dantrolene (Dantrium)</td>
</tr>
<tr>
<td>Optic neuritis</td>
<td>Methylprednisolone (Solu-Medrol)</td>
</tr>
<tr>
<td></td>
<td>Oral steroids</td>
</tr>
<tr>
<td>Speech</td>
<td>Clonazepam (Klonopin)</td>
</tr>
<tr>
<td></td>
<td>Tizanidine (Zanaflex)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Antidepressants</td>
</tr>
<tr>
<td></td>
<td>Amantadine (Symmetrel)</td>
</tr>
<tr>
<td></td>
<td>Pemoline (Cylert)</td>
</tr>
<tr>
<td></td>
<td>Tricyclic antidepressants or SSRIs</td>
</tr>
<tr>
<td>Pain</td>
<td>Aspirin or acetaminophen</td>
</tr>
<tr>
<td></td>
<td>Antidepressants</td>
</tr>
<tr>
<td></td>
<td>Codeine</td>
</tr>
<tr>
<td></td>
<td>Mexiletine (Mexitil)</td>
</tr>
<tr>
<td>Trigeminal neuralgia</td>
<td>Carbamazepine, other anticonvulsants</td>
</tr>
<tr>
<td>Sexual dysfunction</td>
<td>Papaverine (Cerespan) injections</td>
</tr>
<tr>
<td></td>
<td>Sildenafil (Viagra)</td>
</tr>
<tr>
<td>Bladder disorders</td>
<td>Oxybutynin (Ditropan)</td>
</tr>
<tr>
<td></td>
<td>DDAVP nasal spray (Desmopressin)</td>
</tr>
<tr>
<td></td>
<td>Prazosin (Minipress)</td>
</tr>
<tr>
<td>Ataxia/Tremors</td>
<td>Carbamazepine (Tegretol)</td>
</tr>
<tr>
<td></td>
<td>Isoniazid (INH)</td>
</tr>
</tbody>
</table>

(Barnes, 2000)
(NIH, 2002)
(Swain, 1996)
**Table 7**

Support Programs

Betaseron (Pathways) 800-788-1467 or [www.betaseron.com](http://www.betaseron.com)

Avonex (MS Active Source) 800-456-2255 or [www.avonex.com](http://www.avonex.com)

Copaxone (Shared Solutions) 800-887-8100 or [www.copaxone.com](http://www.copaxone.com)
Multiple Sclerosis Algorithm

**EARLY PRESENTING SYMPTOMS**
(One or more)
- Vision Disturbances
- Fatigue
- Muscle Weakness
- Depression
- Spasticity
- Impairment of Pain, Temperature, Touch
- Pain (moderate to severe)

**INITIAL OBJECTIVE EXAM**
(One or More)
- Optic Neuritis, Decreased Visual Acuity, Nystagmus
- Decreased Motor Strength
- Abnormal Reflexes
- Positive Hoffman's Sign
- Positive Babinski Sign
- Lhermitte's Sign
- Charcot's Triad
- Decreased Awareness of Vibrations
- Decreased Awareness of Position

**DIAGNOSTIC TESTS**
- Magnetic Resonance Imaging
  - Abnormal brain or spinal cord scans found in ~88%
- Cerebrospinal Fluid Analysis
  - Abnormal WBCs, abnormal protein levels, oligoclonal bands, or elevated IgG found in ~83%
- Visual Evoked Response
  - ~85% have abnormally prolonged latencies
- Blood Tests
  - (R/O other causes)

**SECONDARY PRESENTING SYMPTOMS**
Any of Early Symptoms - Plus
- Ataxia
- Tremor
- Speech Disturbances
- Bladder Dysfunction
- Bowel Dysfunction
- Sexual Dysfunction
- Cognitive Abnormalities

**SECONDARY OBJECTIVE EXAM**
Any of initial exam
- Plus
- Speech Impairment
- Cognitive Impairment

**CATEGORIES OF MULTIPLE SCLEROSIS**
- **Benign** - No significant disability due to MS 10 years after onset.
- **Relapsing-Remitting** - Clearly defined relapses from onset. Any fixed disability entire due to incomplete recovery from relapses. No evidence of progression between relapses.
- **Secondary Progressive** - Initially relapsing-remitting then develops into steadily progressive course with or without superimposed relapses, minor remissions and plateaus.
- **Primary Progressive** - Disease progressive form onset with minor plateaus and temporary improvements allowed, but no clear relapses. The most common initial symptom was weakness.

**Treatments**
- Interferon Beta 1a (Avonex), Interferon Beta 1b (Betaseron), Glatiramer Acetate (Copaxone), Mitoxantrone (Novantrone)
- These treatments are available to slow the progression of the disease. It is important for the patient, along with their provider to carefully consider which treatment is best in each individual case.
- Other treatments are available to treat accompanying symptoms, such as, fatigue, bladder/bowel dysfunction, sexual dysfunction, spasticity, depression, pain, speech problems, trigeminal neuralgia, ataxia, tremors, optic neuritis, etc.

(Barnes, 2000) (Anderson et al., 1999) (Ferri, 2002) (Swain, 1996)
References


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Psychiatry, 68, 114-158.


