Multiple Sclerosis

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DR. WILLIAM SCHEIBEL *(Associate Professor of Family Medicine and Practice):* We are very pleased that S.K., who is one of our patients recently diagnosed with multiple sclerosis (MS), is present to discuss her perspective of this disease.

DR. SUSAN ISENSEE *(Third-Year Family Practice Resident):* We'd like to begin with the patient's initial presentation, then progress to how the diagnosis of the disease was made, and finish with the treatment modalities that are currently being used.

S.K. is a 32-year-old, white, married woman with one daughter at home. She first noted the onset of symptoms in April 1984. She was under increased stress at that time because she had just received a diagnosis of carcinoma in situ of the cervix. S.K., would you please tell us about your initial symptoms and how you reacted to those symptoms.

S.K.: The first thing I felt was the sudden complete loss of sensation down the left side of my body. I thought I was having a stroke, because it was as though I could take a pencil and mark a line straight down my body and on one side I had normal feeling, and the other side, I felt nothing. I had no taste on the left side of my tongue, and my vision was doubled. I was scared, but people around me said it was probably just a pinched nerve; so I went to my chiropractor and received an adjustment, but there was no improvement. My left side would not walk with me, as it was numb, and I kept falling over. I then went to my doctor, Dr. Raczek (graduated family practice resident), at the Verona Family Practice Clinic, and he did a physical examination. He told me at first my problem was the result of either a blood clot, a tumor in my head, or MS. We went through some tests and discovered that there was no tumor.

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DR. ISENSEE: On physical examination, Dr. Raczek confirmed a decreased sensation to light touch and pinprick on the left side of her face, the left arm, and the left upper leg. Motor strength was grossly normal, and there were no other specific abnormalities found on the examination. The differential diagnosis was as the patient outlined above. She underwent a computerized tomographic (CT) scan of the head, which was normal. She returned to the clinic the following day and had a lumbar puncture performed, which showed an increased lymphocytosis and positive oligoclonal banding. She also had visual and brainstem evoked response testing, which were normal. She was then seen in consultation by a neurologist. Could you tell us about that visit?

S.K.: Most of my symptoms were gone when I saw him, although I was not able to walk with my eyes closed. He told me that I probably had MS, and that I shouldn't get into hot showers or eat a lot of salty things. He also recommended that I not suntan anymore, and told me to return if I had the recurrence of any symptoms.

DR. ISENSEE: Part of the dilemma was that by the time she saw the neurologist, many of her symptoms had already resolved. She had waited one week from the onset of her symptoms until seeing Dr. Raczek, and it was another week before she saw the neurologist. She did well until January 1985, at which time she had the recurrence of symptoms. I'd like to have her tell us a little more about what this was like.

S.K.: This episode started with my fingertips in both hands going numb one evening. I couldn't feel them at all. When I got up the next morning, I just couldn't get up. It felt like my legs were gone, and there was no sensation from the waist down. My eyesight was fine this time, but I couldn't walk at all. My legs looked perfectly normal, but I couldn't get them to work. Each time I tried to walk, I would fall. These symptoms lasted much longer, nearly two months. I saw Dr. Isensee and the neurologist again and was placed on steroid shots. The shots didn't seem to help at the time, but they may have, because my symptoms did eventually decrease. During these two months, my reflexes were hyperactive. My temperment was the

same way in that I would get real jumpy or real edgy. If my little daughter would do anything wrong, I'd jump on her because my nerves were just like someone was buzzing me.

DR. ISENSEE: Our examination at that time confirmed that she definitely had decreased sensation in her right hand and her right foot. She had an ataxic gait and her reflexes were hyperreflexic. She was started on adrenal corticotropic hormone (ACTH), 40 units intramuscularly every day, along with oral cimetidine. She was continued on the 40 units of ACTH for seven days, which was then gradually decreased over another week. At this time it was felt that she met the criteria for the clinical diagnosis of MS.

DR. SCHEIBEL: Multiple sclerosis can be described by its pathologic definition—a chronic disease of the white matter characterized by scattered foci of demyelination resulting in impulse-conduction abnormalities.[1] The macroscopic lesions that are generally seen are 1 mm to 4 cm and are scattered throughout the white matter. There are several areas that are more commonly affected. The periventricular white matter and medial longitudinal fasciculus of the brain and the myelin of the optic nerves are the most common areas. Microscopically, what is seen is the breakdown of the myelin sheath with relative sparing of the axon.

The clinical diagnosis rests on recognizing the intermittent and progressive pattern of multifocal involvement of abnormalities of the motor and sensory function.[2] Our patient gave us a fairly characteristic description of symptoms involving more than one area of the brain and spinal cord, which is an important key to the diagnosis of MS.

There are several ways to classify MS. One useful way is a functional classification: *proven, clinically definite, probable* or *latent,* and *possible MS*.[3] Proven MS requires pathologic proof. Patients with clinically definite MS include those with some physical disablement and a remitting and relapsing course, with at least two episodes that are a month or more apart. The physical dysfunction should reflect predominantly white matter disorders and should involve two or more areas of the central nervous system. The age of onset is usually between 10 and 50 years. Clinically definite MS is still a diagnosis of exclusion because there is not any specific test to confirm it.

Probable or latent MS described patients with slight or no disability but with symptoms that are remitting and relapsing. This group of patients should have more than one abnormal sign commonly associated with MS or have a single episode suggestive of MS with signs of multiple lesions. Our patient did fit into this group after her first episode. Progressive, possible MS refers to patients with some physical disability and a progressive history but no definite evidence of more than one lesion.

The differential diagnosis of MS is quite extensive. It includes ruling out disease related to bone and joint pathology of the spinal cord, which is what our patient initially thought she had. Tumors must be excluded, which is why the CT scan is useful. Metabolic or toxic causes must also be excluded. Transverse myelitis as a result of viruses, bacterial infections, or irritants should also be ruled out. Collagen diseases and motor neuron disease are also in the differential diagnosis.

The cause of MS is still unknown and is currently under intensive investigation. The hypothesis that MS is caused by an environmental agent or agents is supported by epidemiologic findings and is further supported by animal models that show that demyelination can result from viral infections. The protein abnormalities in the spinal fluid and the pathology of lesions of MS support both humoral and cell-mediated involvement. Ongoing studies in the Faeroe Islands are of interest, as this relatively closed population had no history of a case of MS prior to 1940. From 1940 to 1945 the area was occupied by British troops, and 24 cases of MS developed from 1943 to 1960.[4] Geographic studies of MS reveal that there are definite high-incidence areas, while other areas are very low risk.[5] A low prevalence, about five cases per 100,000 people, is found in the continents of South America and Africa and even the warmer climates of North America. The high prevalence areas, 30 to 80 cases per 100,000 people, include most of the northern cities of the United States. Interestingly, S.K. was born in Florida and moved to the north only a year before her disease began.

The autoimmune phenomenon has also been hypothesized as a cause of MS. This theory is supported by some studies and the seemingly beneficial effects of treatment with immunosuppressive agents.[6] There are also a number of studies looking at whether genetic types seem to predispose to the disease. These associations, however, are not the same among various cultures, so that further study is still needed.

S.K. described classic symptoms of MS. They consist of changes in vision, including blurred vision, double vision, and (rarely) even loss of vision. Vertigo, labyrinthitis, altered ambulation, and altered coordination are common. Bandlike paresthesias, numbness, and tingling are seen. Weakness, fatigue, and even dysarthria are frequent. Urinary tract dysfunction is usually a later finding and includes retention, urgency, eneuresis, and impotence. Paresthesias are the most common initial complaint followed by loss of vision or decreased visual acuity, gait difficulties, and then decreased coordination of an extremity.

The signs of MS are the result of demylinated areas that act as short circuits for nerve impulses. Impaired visual acuity is the most common and can be associated with nystagmus, color desaturation, intranuclear opthalmoplegia, and a Marcus Gunn pupil. Color desaturation is confirmed when a red object appears washed out in the affected eye compared with the unaffected eye. A Marcus Gunn pupil is a late abnormality in which the affected pupil is dilated. Intranuclear opthalmoplegia, considered by some to be pathoneu-

monic for MS in young adults if bilateral, is demonstrated when the adducting eye lags behind the abducting eye on testing of extraocular movements. Other signs include asymmetric brisk tendon reflexes, positive Babinski's signs, absent abdominal reflexes, and loss of vibratory sense. Ataxia and incoordination are common. Because many of the signs and symptoms wax and wane with time, repeated observations when appropriate are often the surest aids to diagnosis.

The laboratory is a helpful adjunct to the diagnosis of MS, but the findings are nonspecific. It is essential to examine the cerebral spinal fluid, as it will usually show mildly elevated protein and a mild elevation of the white blood cell (WBC) count (generally less than 20 WBC/mL[3]). Acute exacerbations of MS usually will show a mononuclear pleocytosis in the cerebral spinal fluid (CSF). More specific testing of the CSF will reveal an elevated gamma globulin fraction of protein that is greater than 12 percent. This finding is nonspecific and can be invalidated by a recent myelogram. An immunoglobulin (Ig) G index, felt by some to be more sensitive, can also be calculated by comparing the CSF IgG to the serum IgG. The oligoclonal proteins are discrete bands of gammaglobulins in CSF seen on electrophoresis. Again nonspecific, these markers are the more sensitive for MS. The pattern remains consistent within individual patients throughout the disease, but the distinctive pattern varies among patients. Perhaps there are multiple antigens stimulating this antibody production, but so far absorption procedures have not identified any known or specific antigens.

Detection of myelin basic protein (fragments of myelin) by electronmicroscopy and immunologic methods is also useful. Myelin basic protein levels seem to be elevated when there is an acute demyelination occurring, but return to normal generally within two weeks.

S.K. stated earlier that a neurologist recommended that she avoid hot temperatures, including hot showers and sunbathing. There is a hot bath test that actually makes use of the observation that heat increases the signs and symptoms of MS. Elevation of temperatures 0.5 to 1.5°C can cause conduction blocks in MS patients who have marginally functioning nerve fibers resulting from partial demyelination (subclinical lesions). Examination of patients when they are hyperthermic may bring out lesions that were not otherwise evident, especially diminished visual acuity and nystagmus. The mechanism of action is thought to be the result of shortening the action potential, so it increases symptoms, while cold prolongs the action potential, so it improves symptoms.

One of the diagnostic tests done early in the evaluation of this patient was a CT scan, which was used to rule out tumor or other vascular lesions in the brain. Common findings with the CT on MS patients are decreased densities in the periventricular white matter, which is the area that most commonly harbors the so-called MS plaques. The lesions are usually multiple, and many of them are clinically silent. Contrast is used to enhance the MS lesions, and it does indicate areas of active demyelinization. Generalized atrophy and enlarged ventricles can also be seen nonspecifically in patients with MS. Most of these findings are late, and as was the case with our patient, the initial CT scan was normal.

Evoked responses are becoming increasingly useful in neurology[7] and provide another way of demonstrating multifocal disease to support the diagnosis of MS. An evoked response is a recording of the brain's electrical response to a sensory stimuli. A light flash to the eye, a click to the ear, or a touch to the skin will stimulate a characteristic electrical response in the brain stem or cerebral cortex. A computer connected to electrodes over the brain stem and cortex can produce a wave form of the evoked electrical response by subtracting the random or normal electroencephalographic (EEG) activity. Diseases of myelin will slow transmission of an evoked response and distort the normal wave forms seen for specific sensory stimuli. In this way evoked responses can detect early lesions in myelinated axons before they are clinically apparent.

The three clinically useful evoked response tests are the brain stem auditory evoked response (BAER), the visual evoked response (VER), and the somatosensory evoked response (SSER). The BAER test uses a repetitive click given through headphones to test the integrity of the cochlear nuclei and auditory pathways through the brain stem. The VER test evaluates conduction through the optic nerves and tracts of the lateral geniculate bodies and optic radiation. The stimulus of the VER test is a black-and-white checkerboard screen that switches color rapidly. The SSER test is elicited by stimulation of the median, posterior tibial, or peroneal nerve and is used to test conduction through the dorsal column nuclei, the thalamus, and the corticosensory pathways. These evoked responses, which have a high sensitivity, detect abnormalities of conduction in clinically normal regions of the nervous system.

Multiple sclerosis can follow an acute, progressive, or benign course. In acute disease the neurologic dysfunction progresses rapidly over weeks to months with incomplete remissions followed by severe relapses. This course can be terminal within a number of months. Neurologic dysfunction in progressive MS gradually becomes worse, without well-delineated remissions and relapses. Benign MS is characterized by few exacerbations, often mild, followed by complete recovery. Because of the variety of courses MS can take, it is difficult to give an individual patient a clear prognosis. Two thirds of patients who have benign disease five years after the onset of symptoms will continue with a benign course. Conversely, 99 percent of patients with severe disease at five years will continue with a severe course.[8]

DR. JIM VAWTER *(Second-Year Family Practice Resident):* Have the patients with MS ever been looked at to see whether MS lesions are actually reversed when symptoms have subsided?

DR. SCHEIBEL: That question is difficult to answer, because a test to identify individual MS plaques in living patients has not been available. Magnetic resonance imaging may answer this question in the future. Histologically, MS plaques can be differentiated into new and old lesions. Remissions and exacerbations of MS could be better understood if a restorative process were proven to occur in man.

The diagnosis of classical MS is by a history of characteristic symptoms that remit and relapse in a patient less than 50 years old. Symptoms and signs should involve more than one lesion in the central nervous system that cannot be explained by other diseases. The most important laboratory tests are cerebral spinal fluid analysis and electrophysiologic studies.

DR. WILLIAM SCHECKLER *(Associate Professor of Family Medicine):* Is magnetic resonance imaging useful in diagnosing MS?

DR. SCHEIBEL: Magnetic resonance imaging (MRI) is an exciting new tool in MS, as it can pick up lesions in the range of 3 to 4 mm. It should be more sensitive in finding early lesions in patients with symptoms of MS.

DR. WILLIAM SCHWAB *(Assistant Professor of Family Medicine):* I have seen several pictures of MRI scans that show the MS plaques. The scans look impressive, and should be helpful in locating the disease in multiple areas of the brain, a finding that is necessary for definite diagnosis.

DR. ISENSEE: Before discussing treatment, I would like to mention the disability scales that are used to follow patients with MS. These scales are useful because they help evaluate treatment benefits. The most common scale is that used by the National Multiple Sclerosis Society. It is called the Minimal Record of Disability for MS,* and is useful in following patients to determine how much progression there is with their disease.[9]

As Dr. Scheibel mentioned earlier, treatment of MS is difficult because the cause and course of the disease in any specific patient are unknown. There is no curative therapy for MS, and no medications have been shown to alter the long-term course of the disease. Consequently, there are many nonscientific treatments of MS with unreasonable claims for cure. As physicians, we need to help our patients review treatment options and help them to choose legitimate treatment programs. The International Federation of Multiple Sclerosis is available to help both physicians and patients in evaluating treatment options and to provide literature in support of their recommendations when requested.[10]

Symptomatic and supportive treatment is, perhaps, the most important areas of help for MS. Emotional and supportive treatment, paying attention to the family needs and the personal needs of the patient, is extremely important. Our patient can provide us with some insight into the emotional turmoil she and her family experienced with the diagnosis of MS.

S.K.: I am unable to work outside the home now. I am a beautician by trade, but I am afraid to go back to work now because I am unable to stand when I have an attack of my MS. I don't know any beauticians who can afford to be out of work for two months at a time. I fear doing things that healthy people do without a second thought. My daughter has had to learn how to do a lot of things herself because I have not always been able to help her. She has had to grow up fast.

DR. ISENSEE: S.K. also expressed some concern to me that her husband is often gone; his place of work is 45 minutes away from home. Her husband is also concerned that she may hurt herself in a fall and not be able to get help because they live out in the country. We as physicians and health care providers must be aware of the family's day-to-day turmoil in dealing with this disease. It's a frustrating illness, and people go through an adaptive coping mechanism just as cancer patients do. There have been four stages of coping described in the literature.[11] The first one is denial. Patients deny that they have the disease and often change physicians to confirm a diagnosis. The second stage is that of resistance. Patients try to gain control over the disease, and are aware of the diagnosis that has been made but resist it. In this stage the patients' try not to alter their lifestyles because of the disease.

S.K.: During my second MS attack my husband took my skis away from me because he was afraid I was going to break my neck. I refused to stop skiing because it was such a good year for snow. Of course I'd fall a lot, but I'd just laugh and say that I was a terrible skier and try to get up and go again. This concerned other people around me, but I didn't want to give up. I wanted to keep doing certain things because I was afraid one day I might not have the use of my legs any more.

DR. ISENSEE: Following the resistance stage, patients will enter a stage of affirmation or self-confrontation. In this stage the patient faces the disease and begins dealing with it realistically. The final stage, which often takes the longest time to achieve, is the stage of integration, where the patients actually change their lifestyles because of the disease. They accept the diagnosis of the disease but often try not to think much about it. They concentrate on other areas of their lives that they can nourish and make better in view of the fact that their physical symptoms may be disabling.

There is controversy in the literature regarding the psychological changes seen in MS patients and whether they occur because of the disease or whether

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*Copies available from the National Multiple Sclerosis Society, 205 E. 42nd Street, New York, NY 10017.

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they precede the disease.[11] Some people have suggested that there is an increase in hysteria, depression, and hypochondriasis in people who have MS. Depression is most commonly seen. It is unknown whether this is physiologically related to the disease or whether it is an emotional response to a chronic disease. Depression increases when the severity of the disease increases. It is important that health care providers be aware of this and give appropriate support and counseling. We also must remember that the disease causes stress, and stress causes exacerbation of the signs and symptoms of the disease.

Physical therapy and occupational therapy are very important in the treatment of MS. Proper nutrition is also essential, and patients need assistance in this area. Good skin care cannot be ignored. Rapid treatment of acute infection is important, as increased temperature can cause an increase in symptoms. Hot showers alone may increase the patient's symptoms. Primary care physicians can help patients by providing counseling to them in their day-to-day lives.

During an episode of acute symptoms, bed rest without strengthening exercises is recommended. Physical therapy with range of motion exercises, however, is important to prevent flexion contractions or muscle atrophy. Patients often fatigue easily, and they should be assisted in activities that they can perform. Range of motion exercises are recommended three to four times a day. During remissions, active exercises should be increased and encouraged. Strengthening and passive range of motion are very important. Walking is also very good. Running and swimming are acceptable, as long as these exercises do not occur in hot weather or in very warm swimming pools. It is recommended that sunbathing be avoided. Continued mobility is important; consequently, orthotics, weighted shoes, walkers, and canes are often needed. Eventually, wheelchairs may be necessary. The focus then changes to teaching patients how to transfer to and from their wheelchairs. Modification of both the home and workplace may become necessary to allow patients with active MS to remain independent. Occupational therapy may help many patients achieve alterations in their lifestyles so that they may remain productive.[12]

Spasticity is one of the most common problems seen with MS patients. It interferes with activity, comfort, and hygiene. It can cause aching and fatigue. One of the medications used is baclofen, which is a centrally acting gamma-aminobutyric acid agonist. Its side effects include sedation, acute organic brain syndrome, and nausea. It decreases spasticity, but it also decreases muscle tone and it may therefore cause rubbery legs. Patients who are still ambulatory may have more problems with baclofen than those who are wheelchair bound. It must be remembered that abrupt withdrawal can cause seizures. Dantrolene sodium is another antispasmodic medication. It is generally not

recommended long term because it can cause liver toxicity. Diazepam (Valium) is also used to control spasticity, but the dosage that is usually required often causes too much sedation and therefore its usefulness is limited. Experimental and questionable treatments of spasticity include intrathecal steroids and chemical neurectomy with phenol.[13]

Visual symptoms can be treated with an eye patch if diploplia is the main problem. Optic neuritis, with loss of vision, can be treated with steroids. Adrenocorticotropic hormone (ACTH) or prednisone in dosages of 80 to 100 mg/d for two to six weeks with a tapering-off dosage may shorten the duration of visual symptoms.[12,13]

Sensory disturbances including dysesthesias and parathesias are common in the trunk and the extremities. Because this often causes chronic pain, some people have suggested low-dose tricyclic antidepressants. The common other sensory disturbance seen is trigeminal neuralgia. Treatment with carbamazepine (Tegretol) or phenytoin (Dilantin) is recommended here.

Tremors have no definite treatment. Fatigue is often disabling, and patients should rest during the day. If they are active, a noontime rest of an hour or two is often very helpful and will allow them to get through the day and function as normally as possible. One reference suggests that the use of pemoline (Cylert), which is a central nervous system stimulant used in attention-deficit disorders, may be helpful in the morning to combat fatigue.[13]

Bladder dysfunction is common and requires a basic evaluation. The bladder disturbances most frequently seen are urgency, frequency, and incontinence, which often cause vocational and social problems. Urinary tract infections are commonly superimposed on these symptoms. A recommended evaluation of the bladder would usually include a history, urinalysis, postvoid residuals, and cultures and sensitivity tests as needed. Cystometry or urethral electromyography can also be helpful. Once the evaluation has been done, there are various medications that can be used for treatment. One of the more common problems encountered is failure to empty the bladder with a hyporeflexic or hypotonic large-capacity bladder causing overflow incontinence. Treatment here is intermittent catheterization along with the Credé's maneuver and parasympathomimetics. Bethanechol, an anticholinergic agent, can stimulate the parasympathetics, helping micturation in this type of defect. The most common type of bladder dysfunction is failure to store, or the hyperreflexic bladder. This uninhibited bladder has a small capacity for storage. Drugs used to treat this condition would be the anticholinergics. Propantheline is the drug of choice. The mixed bladder dysfunction with hyperreflexia plus detrusor or sphincter dyssynergia is also seen. Medications suggested as possibly being helpful would be anticholinergics along with baclofen. Urinary tract infections (UTIs) should be treated with appropriate antibiotics. Acidifying the urine with ascorbic acid or other agents is sometimes helpful in the prevention of UTIs. Prophylaxis is important if there are recurrent UTIs, and either nitrofurantoin or trimethoprim-sulfamethoxazole can be used here.

Sexual problems are often seen in MS. Impotence and anorgasmy must be evaluated and treated appropriately. Men may need a penile implant.

Constipation is the most common bowel dysfunction seen. The customary treatments for constipation are important.

Steroids are the only medications that are recommended during acute exacerbations. They have an anti-inflammatory effect as well as a positive effect on the immunologic system, which some experts feel is important in acute exacerbations of MS. Adrenocorticotropic hormone is recommended in varying dosages, usually starting with 40 units intramuscularly either once a day or twice a day for a week, then decreasing to 20 units for two to four days and tapering off over another two to four days to zero. Oral steroids such as prednisone in a dosage of 60 to 120 mg/d in divided doses for about a week and then gradually tapering off are also helpful. There continues to be a debate as to whether intravenous bursts of methylprednisolone, oral dexamethasone, or intrathecal steroids are useful.

Immunosuppressive therapy has received considerable attention lately because of the belief by some that immunologic mechanisms are involved in the etiology of MS. The side effects of immunosuppressive therapy include myelosuppression, leukopenia, hepatitis, alopecia, stomatitis, and an increased risk for malignancy later. Consequently, these agents are considered experimental, and patients need to be aware of the risks prior to their use. Azathioprine is a purine antagonist that interferes with nucleic acid metabolism. Cyclophosphamide has been receiving more attention lately, as it is classified as an alkylating agent that has been used in combination with ACTH and shown to halt the progression of MS for 6 to 12 months in a number of patients.[6] The long-term benefit has not yet been proven. Other modalities being studied include methotrexate, interferon, plasma exchange, hyperbaric oxygen, and monoclonal antibodies. Dietary measures are also advocated by some. Although one researcher has shown a decrease in the symptoms and progression of MS on a low-fat diet,[14] this has not been substantiated elsewhere in the literature.

In summary, definitive treatments in helping or halting the disease process of MS are not available. Physical and occupational therapy are very important for MS patients, and we can help our patients understand their disease. Our attention to emotional support and dealing with patients' frustrations are invaluable.

DR. DAVID LONSDORF *(Chief Family Practice Resident):* How are you following this patient now? Is she currently being treated with anything? Is she reg-

ularly being followed or just returning whenever she has a flare-up of her symptoms?

DR. ISENSEE: S.K. returns every three months for both re-evaluation of her MS and of her cervical carcinoma (in situ), which was treated with a hysterectomy. She is doing well now and does not have any symptoms that are currently being treated. She is following a prudent diet and resting each day. She is also avoiding sunbathing and hot showers.

DR. KENNETH KUSHNER *(Clinical Psychologist, Department of Family Medicine and Practice):* Is there an MS support group in town, and if so, has S.K. had any contacts with them?

S.K.: I get letters from them, but I don't like to go there because I'm constantly living with my MS. When I go there, there are many people in wheelchairs, and that is very depressing to see. I don't want to see how I might end up. I do have another girlfriend in town with MS who is in a wheelchair, and she attends these support groups all the time.

DR. KUSHNER: Who recommended that you contact them?

S.K.: My girlfriend with the advanced MS—as well as the neurologic consultant.

DR. SCHWAB: I've spoken with people who are doing research in MS using the MRI for diagnosis. What they're beginning to find is that MRI may really change the definitions of the disease in the same way that echocardiography changed our feelings about mitral valve prolapse. When mitral valve prolapse was only a clinical diagnosis, it was not felt to be very widespread, but now that echocardiography is available, we find that almost 10 percent of women have it. The same may be true of MS in diagnosing a large number of patients with clinically silent lesions. There appears to be a specific pattern for MS, and we may continue to find many more people with paresthesias who will show a characteristic spot or two that seems to be consistent with small areas of demyelination on scans. This may drastically change the definitions we currently have of clinical MS.

DR. SCHEIBEL: I would like to thank everyone for participating in this presentation, and special thanks to S.K. for being present and participating actively in our understanding of this disease.

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