

Second Edition

NEUROLOGY FOR NON-NEUROLOGISTS

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MULTIPLE SCLEROSIS

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The demyelinating disorders are a broad category of diseases of the central nervous system (CNS) in which there is destruction of myelin sheaths with relative preservation of neuronal axons. The primary demyelinating diseases include multiple sclerosis (MS) and its uncommon variants which have been called Devic's optic neuromyelitis, Balo's concentric sclerosis, transitional sclerosis, and Schilder's diffuse sclerosis. The secondary demyelinating diseases include a variety of etiologically specific causes of demyelination such as neoplasm, viral infection, and certain toxic and metabolic disorders. This chapter covers MS in depth because it is the only demyelinating disorder that is frequently encountered by clinicians.

Multiple sclerosis is a chronic neurological disease that typically begins in early adulthood and progresses to significant disability in the majority of cases. An unpredictable course and a wide variety of symptoms and signs are remarkable features of the disease, which is the most common progressive and disabling neurological condition affecting young adults in the United States. (There are approximately 123,000 known MS patients in the U.S.) Because the onset of the illness is usually in early adulthood, family life and job productivity are often seriously disrupted. Current theories favor an immune-mediated pathogenesis of MS secondary to a fundamental defect in the host, with or without the presence of a triggering viral agent.

The mean age of onset of MS is 33 years and the mean age of diagnosis, 37 years. It rarely appears before age ten and only about 10% of cases begin after the age of fifty. MS occurs more frequently in white women, having a female to male ratio of 1.7 to 1 and a white-to-nonwhite ratio of about 2 to 1. It is more common in the cold and temperate climates of the higher latitudes in both hemispheres. The northern latitudes of the continental United States lie well within this "MS belt." Studies of the prevalence of MS in immigrant populations indicate that the chance of a person developing the

disease is correlated with having lived in these higher latitudes in the first 15 years of life.

There is some evidence that genetic factors are involved. MS occurs in 1% to 2% of first-degree relatives (parents, siblings or children) of MS patients, and MS predominantly affects persons of northern European ancestry. Furthermore, recent studies of the distribution of histocompatibility antigen (HLA) genetic marker in MS patients revealed an overrepresentation of A3, B7, and DW2 types. The relation of these factors to the pathogenesis of the disease is unknown.

SYMPTOMS AND SIGNS

No two patients with MS are exactly alike, and the clinical manifestations in a particular person are related to the distribution of lesions within the nervous system. Lesions may be found virtually anywhere within the white matter of the central neuraxis, including the white matter of the cerebral hemispheres, optic nerves, brain stem, cerebellum, and spinal cord. Although some patients have evidence of widespread lesions from the outset, others may present with isolated focal involvement of any of these structures. Symptoms and signs may disappear or may fluctuate in character and intensity. The sometimes bizarre and transient nature of the symptoms may be mistaken for a psychiatric condition.

Muscle weakness and spasticity due to corticospinal tract lesions are among the most frequent symptoms of MS. Spasticity of the lower extremities may be accompanied by painful flexor spasms. Impaired dexterity, slowness of rapid alternating movements, hyperreflexia, extensor plantar responses, and absence of abdominal reflexes may also be noted, along with hemiparesis, paraparesis, quadriparesis, or monoparesis.

Complaints of severe fatigue are common; disabling exhaustion may be brought on by an ordinary day's activities. This symptom is remarkable because in some patients it occurs in the presence of normal strength and without any symptoms generally associated with depression.

Visual disturbances include impaired visual acuity, impaired color vision, central scotoma, diplopia, and uncommonly such visual field defects as homonymous hemianopsia. Symptoms may be unilateral or bilateral. Optic neuritis and retrobulbar neuritis are common in MS. The patient complains of loss of vision which progresses over days and may be mild to severe. Pain in or behind the eye, which sometimes is caused or worsened by movement of the globe, may be an associated complaint. Examination during the episode shows loss of visual acuity, loss of color vision, a central scotoma, and preserved peripheral vision. Examination of the fundus shows a swollen disc if the lesion is close to the optic nerve head (papillitis) otherwise the disc looks

normal (retrobulbar neuritis). Neurological examination following the episode often shows residual deficits of visual acuity and color vision, together with optic atrophy manifested as a pale optic disc, particularly the temporal portion. It must be noted, however, that not all patients with optic neuritis have MS. Only 30% to 60% of previously healthy patients with a first attack of optic neuritis go on to develop symptoms and signs of MS.

The other common visual manifestation of MS is diplopia caused by an internuclear ophthalmoplegia (INO). This is a disorder of conjugate lateral gaze characterized by nystagmus of the abducting eye and weakness of the adducting eye with preservation of convergence. Its presence in a young adult is almost pathognomonic for MS with the demyelinating lesion in the medial longitudinal fasciculus ipsilateral to the eye with the weakness of adduction. It may also be caused by other lesions such as stroke or tumor.

Disturbance of sphincter control is noted in at least two thirds of patients. Urinary dysfunction may be due to a failure to store (hypertonic or spastic bladder), failure to empty (atonic or flaccid bladder), or a mixture of the two. Symptoms include frequency, urgency, incontinence, incomplete emptying, and urinary retention. The major bowel complaint is constipation, although fecal incontinence occurs occasionally.

Complaints of sexual dysfunction are frequent and include erectile and ejaculatory problems in males and loss of orgasmic ability in females. These problems may be compounded by depression or by urinary or fecal incontinence occurring during intercourse.

Lesions in the cerebellar white matter or cerebellar pathways may produce nystagmus, prominent gait and extremity ataxia, and a halting or scanning quality of speech. Severe intention tremor of the upper extremities may make the simplest self-care tasks impossible, and severe ataxia of gait may prevent effective ambulation even when muscle strength is adequate.

Sensory symptoms are diverse and include numbness, tingling, impairment of temperature sensation, and abnormal sense of limb position. Vague sensory complaints in unusual distributions may mysteriously come and go, confounding the diagnosis. Examination may reveal no objective sensory deficit even in the presence of symptoms. Impairment of vibratory perception may be found without any abnormality of other sensory functions. Lhermitte's phenomenon, which is provoked by flexion of the neck, is usually described as an electric shock sensation that radiates down the spine or into the extremities. This unusual symptom is often due to MS, but may also occur with other disorders affecting the posterior columns of the cervical spinal cord. Approximately 5% to 10% of MS patients experience either typical trigeminal neuralgia or a pseudoradicular pain of the extremities or trunk.

Some form of mental disturbance eventually occurs in half of MS patients. Depression is common, and an inappropriate euphoria is seen on occasion. Mild dementia and organic psychosis due to cerebral involvement occur frequently. These disorders may be severe in patients with advanced disease.

PRECIPITATING AND EXACERBATING FACTORS

Although the cause of MS is unknown, certain factors sometimes precipitate attacks in known MS patients. Trauma, infection, and surgery have all been associated with worsening of MS. There may be a slight increase in risk of exacerbation during and in the 6 months following pregnancy. There is no evidence that immunization is a precipitating factor.

Elevation of body temperature has a different effect. Fever, heavy physical exertion, hot weather, a hot shower or bath, and exposure to sunlight may all cause a transient and reversible worsening of existing symptoms. For example, weakness may become worse to the point where a normally ambulatory patient is unable to get out of the bathtub after a hot bath. Neurological function then returns to baseline within minutes to several hours of when the patient is helped out of the bath. Similarly, a person who can usually transfer independently may require assistance in this activity during very hot weather. Occasionally a symptom such as visual difficulty in one eye may be present only during exposure to heat. Interestingly, lowering of body temperature by swimming in a cold pool or taking a cold shower may result in a temporary improvement of function.

THE COURSE OF MS

The natural progression of MS is unpredictable. In approximately 40% of MS patients, the disease is initially exacerbating-remitting, with or without complete recovery between episodes. After several years, there is a transition to a slow and relentless chronic progression. In another 20% to 30% of patients, the disease maintains an exacerbating-remitting course. In 10% to 20% of patients, the course from the outset is chronically progressive, a pattern that is seen most often in patients who are older at the time of onset of the illness. Finally, in about 20% of MS patients, the course is benign, the patient suffering only one or two mild exacerbations and no permanent functional disability.

The rate of progression of MS is variable and ranges from the occasional malignant course, with death within weeks or months after onset, to life-long benign disease with minimal symptoms and disability. In general, those who have either chronic progression or frequent severe relapses from the outset of the illness have a less favorable prognosis. Patients who have been in the chronic progressive stage of the illness for a number of years may experience decline in the rate of deterioration.

Over the last half century advances in antibiotic therapy and in the management of complications have increased the lifespans of MS patients. In 1936, only 8% of patients were reported to survive beyond 20 years after the onset. By 1961, survival had increased tenfold, over 80% of patients surviving for 20 years after onset of illness. Of those patients surviving for 20 years or more, approximately 30% remained

gainfully employed. This indicates that the long-term prognosis in MS is more favorable now than in the past.

CLINICAL DIAGNOSIS

The diagnosis of MS remains a clinical one; there is no specific laboratory test for the disease. The clinician generally makes the diagnosis of MS when there is evidence of multiple lesions in time and space. The history includes at least one clear attack of neurological dysfunction which lasted at least 24 hours and had some degree of subsequent recovery. The neurological and laboratory examinations should show evidence of two or more lesions that are not contiguous in space. If the age of onset is between 10 and 50 years, and the patient has lived in the higher latitudes during part of the first 15 years of life, and the symptoms worsen transiently with exposure to heat, the diagnosis is more likely. There must be no better neurological explanation for the patient's symptoms and signs.

Often the diagnosis cannot be made at the time of presentation. In the young person with a single attack which consisted of a single lesion in space, laboratory tests may fail to show evidence of other subclinical lesions. MS can then only be suspected. Similarly, in the older patient with a gradually progressive deficit which can be explained by one lesion at a specific location, MS is a diagnosis of exclusion.

LABORATORY EXAMINATION

There is no specific laboratory test for MS. Laboratory tests are, however, important for ruling out other nervous system diseases that may mimic MS. They are also invaluable for demonstrating evidence of a second lesion which is subclinical and therefore not detectable by history or neurological examination. The demonstration of the second lesion is generally the key to making the diagnosis of MS in the patient with early disease. Laboratory tests are also important in the patient who has symptoms suggestive of MS but who lacks objective signs of nervous system disease. One or more definite lesions of the nervous system may be shown to be present.

The hot bath test may be done in the clinic or on the hospital ward. The patient is immersed in a bathtub of hot water and then examined neurologically for the transient appearance of additional deficits. A patient with subclinical optic neuritis may develop an abnormality of visual acuity or color vision. In other patients, an internuclear ophthalmoplegia, nystagmus, or ataxia may become apparent. Weakness, hyperreflexia, and Babinski signs may also develop.

Visual, auditory, and somatosensory evoked responses are sensitive electrophysiologic procedures that can identify clinically silent lesions. If all three tests are performed, approximately 80% to 85% of patients with a clinically definite diagnosis will have an abnormality on at least one of the tests. The visual and auditory

evoked response tests are especially useful in identifying a second subclinical lesion in patients who have clinical evidence of only a single spinal cord lesion. In this instance, the demonstration of a second lesion remote from the spinal cord may help establish the diagnosis of MS.

The neuroimaging test that is often helpful in the patient with suspected MS is magnetic resonance (MR) imaging of the brain and spinal cord. It may be used to exclude other CNS diseases which may mimic MS. Additionally, it frequently demonstrates characteristic lesions of MS within the CNS white matter. In patients with only one known lesion, the MRI may demonstrate a second lesion, thereby making MS the likely clinical diagnosis. MS lesions appear as areas of increased signal on spin-echo images and decreased signal on inversion-recovery images. Computed tomography (CT) of the brain is of limited usefulness because only 20% of patients with clinically definite MS will have CT abnormalities.

Nonspecific abnormalities present in the cerebrospinal fluid (CSF) are frequently useful in supporting the diagnosis of MS by suggesting the presence of an inflammatory CNS lesion. The CSF cell count commonly shows a modest increase in mononuclear cells. The total CSF protein is mildly or moderately elevated in less than half of MS patients. In approximately 60% to 75% of patients there is an abnormal elevation of the CSF IgG. Furthermore, 85% of patients with clinically definite MS have abnormal oligoclonal bands in the IgG zone on CSF electrophoresis. If IgG and oligoclonal bands are both measured, 90% of such MS patients have abnormalities. One limitation of these tests is that they are less frequently abnormal in early or very mild cases of MS. Additionally, abnormalities of the more sensitive tests may also be produced by other CNS inflammatory processes and by chronic CNS infections.

In summary, the patient with reliable historical or clinical evidence of two separate CNS lesions should have a CSF exam to rule out infection. He should also generally have an MR scan of the brain and in some cases of the spinal cord, to rule out another pathological process that may mimic MS. The patient with only one established lesion and the patient who is an MS suspect without any definite CNS lesions should undergo these tests together with the hot bath test and evoked responses. In these patients, the laboratory tests are done in an attempt to rule out other CNS pathology and to demonstrate the presence of at least two separate CNS lesions required to make the clinical diagnosis of MS.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of multiple sclerosis depends on the syndrome of CNS dysfunction which is present at the time of diagnosis. When an isolated optic neuritis is secondary to local orbital or sinus infection, the causative process can generally be demonstrated with CT or MR. Similarly, serology and spinal fluid examination show whether the optic nerve lesion is due to a meningeal process such as neurosyphilis or carcinomatous meningitis.

A presentation which can be entirely explained by a single posterior fossa lesion could be produced by a benign or malignant tumor, basilar impression of the skull, developmental diseases such as Arnold-Chiari malformation, or occasionally by cerebrovascular disease. MR scan generally rules out these conditions. The uncommon chronic meningeal process which began in the posterior fossa would be ruled out by an examination of the spinal fluid for infection or tumor.

If an isolated spinal cord lesion is caused by spondylosis, tumor, or syrinx, it will be seen with MR or CT myelography. When no cause is found for an isolated spinal cord lesion, and the history and spinal fluid are suggestive of a mild acute or subacute inflammatory process, an idiopathic transverse myelitis is the probable diagnosis. Only a few percent of these patients go on to develop MS.

On the rare occasions when MS presents clinically as a subacute or chronic intracerebral mass lesion, low-grade glioma is generally suspected. MRI or, if necessary, brain biopsy will reveal the true cause of the lesion.

In any patient with remitting-relapsing neurological illness, collagen vascular disease, sarcoidosis, and Behçet's syndrome must be considered. All three commonly have associated involvement of other organ systems. MS in contrast, is a strictly neurological disease. In the patient with collagen vascular disease, there may also be abnormal rheumatologic blood tests. Many patients with sarcoidosis will have an abnormal level of angiotensin converting enzyme (ACE).

Finally, neuroimaging tests rule out mass lesions, particularly tumor or spondylosis, in the patient with gradually progressive illness. Neurodegenerative diseases may also present in this fashion and may sometimes be demonstrated by family history or actual examination of family members.

The diagnosis of possible or probable MS is made after ruling out the appropriate conditions in the differential diagnosis. Except for the rare cases where the diagnosis is proved pathologically, the diagnosis is and remains presumptive. For this reason, future changes in neurological status generally warrant reconsideration of the differential diagnosis at a number of times during the course of the disease.

THERAPEUTICS

The management of MS can be divided into four categories: (a) treatment aimed at modification of the disease course, including treatment of the acute exacerbation and treatment directed at long-term suppression of the disease; (b) treatment of the symptoms of MS; (c) prevention and treatment of medical complications; and (d) management of secondary personal and social problems.

The short-term use of either adrenocorticotrophic hormone (ACTH) or oral corticosteroids is the only specific therapeutic measure available for the treatment of the patient with an acute exacerbation of MS. Controlled studies of such therapy have shown that patients with ACTH treatment have a faster recovery, although there is no difference in the final amount of recovery.

ACTH gel, 40 units intramuscularly (IM) twice daily, may be given for 10 days and then tapered at a rate of 10 units every day. Pretreatment evaluation should include a search for tuberculosis, uremia, high blood pressure, diabetes, electrolyte disturbance, and peptic ulcer, which are relative contraindications to the use of ACTH. Because ACTH produces a variable amount of salt and water retention, weight and blood pressure should be checked regularly during treatment, and a low sodium diet with oral potassium supplementation should be prescribed. The occurrence of complications necessitating early discontinuation of therapy are infrequent, although intensification of preexisting depression or euphoria, emotional lability, insomnia, and frank psychosis are among the most common troublesome side effects.

Prednisone may be given orally on an outpatient basis. The total daily dose is determined by weight: 50 mg b.i.d. for weight greater than 180 pounds, 40 mg b.i.d. for patients weighing 110 to 180 pounds, and 30 mg b.i.d. for patients weighing less than 110 pounds. This weight-adjusted dose is given for 7 consecutive days. On the eighth day the total daily amount is given as a single morning dose, and the dose is thereafter gradually tapered to 0 mg at the rate of 10 mg per day.

There is no established treatment that suppresses MS on a long-term basis. Chronic ACTH or prednisone therapies have not proved beneficial in reducing the number of exacerbations or in slowing gradually progressive disability. High-dose cyclophosphamide has recently been shown to have some favorable effect on the course of patients with severe progressive disease. However, the therapy is potentially quite toxic, and retreatment is necessary because the majority of patients regress. For the present, this treatment is reserved for patients with severe MS that is progressing at a significant rate.

Effective symptom management and assistance in coping with the problems of everyday living can improve the quality of life for the MS patient. For example, spasticity may be alleviated by drug therapy. Baclofen, 40 to 80 mg per day in divided doses, is usually of value in reducing severe spasticity as well as involuntary flexor spasms. Mild spasticity generally should not be treated, but most patients with moderate to severe spasticity warrant a trial of therapy. Increased weakness and deterioration in gait are, unfortunately, a limiting side effect of baclofen in some ambulatory patients. Such weakness clears up within 24 to 48 hours after reduction in dose or discontinuation of therapy. This side effect is especially prominent in patients who are relying on their spasticity as a support during standing or walking. Dantrolene sodium is an alternative antispasticity drug that offers no major advantage over baclofen and has the disadvantage of potential hepatotoxicity. Diazepam is also effective in reducing spasticity, but the dosage required for relief of spasticity often produces an unacceptable degree of sedation. In some patients, a bedtime dose of 10 mg of diazepam may quiet nighttime flexor spasms and allow uninterrupted sleep.

Gait difficulty in MS is often due to combinations of weakness, spasticity, and incoordination. Evaluation by a physical therapist with instruction in the use of walking aids and, in some instances, customized braces, may be very beneficial for ambulatory patients.

Bladder dysfunction in MS is common. Referral to a urologist for urodynamic

studies and measurement of post-voiding residual urine are often required to define the type of bladder dysfunction and determine the proper therapy. The failure-to-store bladder may produce simple urinary urgency and occasional accidental incontinence. These symptoms can be effectively managed by intermittent restriction of fluid intake and/or small intermittent doses of oxybutynin chloride. When there is severe urgency and frequent incontinence, the failure-to-store bladder may be converted to a failure-to-empty bladder by the regular administration of oxybutynin chloride (5 mg, two or three times a day). Treatment then proceeds as described below. The failure-to-empty bladder produces frequent overflow incontinence, recurrent infection, or symptoms of urinary retention. The use of chronic indwelling catheters for treatment should be avoided where possible because complicating infection invariably develops. Intermittent self-catheterization is a far safer therapy that is surprisingly well tolerated. It is, however, possible only in patients who have reasonably well-preserved dexterity in the hands. Constipation is frequent in patients with spinal cord involvement and should be treated by conventional methods.

Pain as a direct result of MS lesions within the CNS may occur as a typical trigeminal neuralgia or as pseudoradicular pain, usually in one leg, sometimes in an arm or part of the trunk. The trigeminal neuralgia may be treated with carbamazepine, although such treatment can be associated with undesirable transient weakness similar to that occasionally encountered with baclofen. Surgical procedures such as percutaneous rhizotomy have been employed in refractory cases of trigeminal neuralgia, although the long-term effectiveness of such procedures in MS patients is not well established. When pseudoradicular pain is chronic, it is usually refractory to treatment. Low backache related to weak trunk muscles and poor posture is common in wheelchair patients or in those who have gait disturbance. Conventional therapeutic measures for low back strain are usually effective. For example, patients with spastic, tight hamstring muscles may need physical therapy for stretching of these muscles in order to relieve tension on the lumbosacral spine. A firm bed, proper posture in wheelchair, and regular swimming, along with physical therapy, are also usually beneficial for low back pain.

Intention tremor unfortunately responds very little to drug therapy. Surgical cryothalamotomy is reserved for treatment of severe incapacitating intention tremor; useful function in one extremity can sometimes be restored to patients who are otherwise totally helpless. Diplopia is often temporary and can be managed simply by the use of an eye patch. Impaired visual acuity, deafness, and vertigo are often temporary. This is fortunate, since there is no effective treatment for these symptoms. When fatigue and lassitude are severe and disabling, a trial with antidepressant drugs such as imipramine or amitriptyline is worthwhile and may be surprisingly beneficial in some patients. Placement of a penile prosthetic device should be considered in carefully selected patients who are impotent. Penile prostheses should not be implanted in men who have a significant degree of sensory impairment of the penis or perineum lest the penis be painlessly traumatized during intercourse.

All of the major medical complications of MS are either preventable or treatable. These include contractures of limbs, pressure sores, and pulmonary and urinary tract

infections. Every wheelchair and bedridden patient should be involved in a regular program to prevent contractures and pressure sores. Wheelchair patients with good arm function should be taught how to press down on the arms of the chair at frequent intervals in order to relieve pressure on the sacrum and buttocks. Bed-ridden patients require special air or water flotation mattresses, and should be carefully positioned and turned every 2 to 3 hours. Pressure points must be examined frequently, and nursing care efforts must be intensified at the earliest sign of a developing sore. The smallest ulceration should be considered as a potentially life-threatening complication and treated appropriately and vigorously. Patients with progressing pressure sores may require surgical treatment for debridement or skin grafting.

The secondary complications of MS cover a broad spectrum of personal and social difficulties—marital, occupational, psychosexual, recreational, legal, and financial. Most physicians are not traditionally prepared to deal in depth with many of these problems though, ironically, it is often in this area that the most can be done to help some patients. In order to deal effectively with these problems, the physician must become familiar with resources in the community and enlist the help of other professionals such as psychologists, social workers, marriage counselors, vocational rehabilitation counselors, and lawyers. The local chapter of the National Multiple Sclerosis Society may be able to help directly or can recommend referral to people who are qualified and experienced in working with MS patients. The patient can also be encouraged to participate in support groups, which are often sponsored and organized by the local Multiple Sclerosis Society.

The physician's attitude may have a powerful psychological impact on the MS patient. There is sometimes a tendency by both physicians and patients to view the disease as incurable and untreatable. Such a view is excessively negative and unwarranted. A positive but realistic approach by a knowledgeable and sympathetic physician can greatly improve the patient's sense of well-being and perhaps even have a beneficial effect on the course of the disease. Hope is a powerful elixir that should be encouraged. Helplessness should be discouraged. Many patients gravitate toward unproven popular therapies, such as special diets. If the putative therapy is both low-risk and affordable, the physician can be most helpful by taking a tolerant position. An additional benefit of this approach is that the patient thereby learns that the physician is open minded and eager to support the patient in his search for legitimate therapy. At a later date the patient will be more likely to follow the physician's advice against some other therapy which might be unreasonably expensive or potentially harmful.

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evaluation of neurological disorders. Extra-axial posterior fossa lesions involving the auditory nerve, such as cerebellopontine angle tumors, may result in a normal wave I and prolonged latency or absence of all subsequent waves to stimulation of the involved ear. If enough of the auditory nerve has been destroyed, wave I may also be abolished. If a large lesion causes brain stem distortion, the III–V interpeak latency to contralateral stimulation may be prolonged as well.

Intra-Axial Posterior Fossa Lesions. Intramedullary posterior fossa lesions usually do not affect wave I. However, all of the subsequent waves may be delayed or obliterated by direct involvement of the generating structures or by interruption of the pathways to them. The I–III interpeak latency is prolonged in lesions at the pontomedullary level, and the III–V interpeak latency is prolonged by pontine and midbrain lesions. Brain stem auditory evoked potential abnormalities can be caused by tumors, including astrocytomas, pinealomas, fourth ventricle ependymomas, and metastases. Brain stem infarction or hematoma, trauma, infection, degenerative diseases, such as olivopontocerebellar degeneration, central pontine myelinolysis, multiple sclerosis (MS), and other diseases involving the brain stem auditory pathways also lead to BAEP abnormalities. Brain stem auditory evoked potentials are of particular value in the diagnosis of MS, since they may demonstrate subclinical lesions at multiple levels that escape detection by other diagnostic tests, including CT. About one third of patients with possible MS and no clinical evidence of brain stem involvement have abnormal BAEPs; this suggests an additional central nervous system (CNS) lesion and supports the diagnosis of MS. Thus, the BAEP may obviate neuroradiological examinations to exclude other pathological conditions. Magnetic resonance imaging (MRI), very sensitive in the detection of brain stem and cerebellar lesions, appears to have a higher diagnostic yield than BAEPs and may become the test of first choice in these patients.

Supratentorial Structural Lesions. Unless supratentorial lesions distort the brain stem, they do not alter waves I–V. Wave VI and VII abnormalities may be found, but they must be interpreted with caution in view of the normal variability of these waves.

Metabolic and Toxic Encephalopathies. Brain stem auditory evoked potentials are generally not affected by metabolic and toxic encephalopathies such as drug overdose, diabetic ketoacidosis, uremia, and liver disease. Hypothermia, on the other hand, induces reversible BAEP abnormalities.

Coma. Brain stem auditory evoked potentials are useful in the evaluation of comatose patients because they remain relatively unchanged in coma due to cortical damage, metabolic encephalopathy, or drug overdose, whereas they usually display severe abnormalities in coma secondary to brain stem structural damage. Patients fulfilling the clinical criteria of brain death have either no BAEPs or only wave I. On the other hand, normal BAEPs have been found in metabolic or toxic coma, despite clinical findings suggesting brain stem dysfunction, and they may still be present despite anesthetic doses of barbiturates causing electrocerebral silence on EEG.

Clinical Applications

Peripheral Neuropathy. Somatosensory evoked potentials may be used in the evaluation of peripheral nerve sensory conduction velocity. This is particularly helpful in patients with axonal neuropathies affecting large sensory fibers, such as Friedreich's ataxia, when peripheral nerve compound action potentials cannot be obtained. SEPs may be delayed or absent in patients with radiculopathies, plexopathies, or neuropathies.

Spinal Cord Disease. Peroneal nerve SEPs may be of value in patients with spinal cord disease at the thoracic or lumbosacral level, and they complement median nerve SEPs in the evaluation of cervical myelopathies. Recordings from electrodes over the spine provide complementary information to scalp recordings. Patients with clinically complete cord transections have absent SEPs to peroneal stimulation in leads rostral to the lesion, whereas patients with incomplete transections may have delayed or normal responses. The absence of peroneal SEPs in cord-injured patients may, therefore, indicate completeness of the lesion. Abnormal SEPs are frequently seen in patients with myelopathies of various etiologies that cause loss of proprioceptive sense, whereas loss of pain and temperature sensation is associated with normal SEPs.

Posterior Fossa Lesions. In posterior fossa lesions, all waves after P10 may be delayed or absent. The P1–N19 interpeak latency provides an estimate of conduction time in the cervical and brain stem somatosensory pathways from brachial plexus to thalamus. Normal SEPs are characteristic of patients with lateral medullary syndrome, syndrome of the cerebral peduncle, and other brain stem lesions in which sparing of position sense suggests functional integrity of the medial lemniscus. This is in spite of any impairment of pain and temperature sensation resulting from involvement of the spinothalamic system.

Patients with lesions involving the medial lemniscus, as confirmed by impaired joint position sense, display abnormal SEPs. A large number of patients with MS have abnormal SEPs, often in the absence of sensory abnormalities either by history or physical examination. SEPs, may, therefore, contribute to the early diagnosis of MS by demonstrating subclinical lesions.

Supratentorial Structural Lesions. Thalamic and hemispheric lesions spare early potentials of the SEP, but subsequent waves may be delayed or absent. Patients with lesions involving the ventral posterior thalamus, thalamocortical sensory radiations, or somatosensory cortex may have abnormal SEPs. As in spinal cord and brain stem lesions, there is usually a good correlation between impaired joint position sense and SEP abnormalities.

Coma. Somatosensory evoked potentials are useful in evaluating coma following head trauma and coma from structural brain disease, particularly if serial studies are performed. Patients so affected may have prolonged P15–N19 interpeak latency, suggesting brain stem dysfunction, in addition to abnormalities of later waves. Toxic

TABLE 4-9. CSF SYNDROMES

CSF syndrome	Pressure (mm H ₂ O)	Fluid	PMNs (per mm ³)	Lymphs (per mm ³)	Glucose (mg/dl)	Protein (mg/dl)
Bacterial meningitis	200-1000	Clear to cloudy	10-10,000	Occur after treatment	0-40 (normal serum glucose)	40-1000
Viral meningitis	Increased or normal	Clear to cloudy		6-1000	Normal	20-200
Tuberculous meningitis	Increased	Clear to cloudy	0-25	25-500	0-40	40-5000
Fungal meningitis	Normal or increased	Clear to cloudy		0-500	Usually 0-40 sometimes normal	40-600
Cerebral abscess (no meningitis) and subdural empyema	Increased	Clear	0	0-40	Normal	20-300
Brain tumor	Increased	Clear	0	0-100	Normal	20-500
Carcinomatous meningitis	Normal or increased	Clear; cloudy, malignant cells on cytology	0	0-500	0-100	20-500
Multiple sclerosis	Normal	Clear	0	0-100	Normal	20-100
Guillain-Barré	Normal	Clear	0	Occasionally 0-20	Normal	50-1000
Peripheral neuropathies, diabetes, uremia, alcoholism	Normal	Clear	0	0-5	Normal	20-200
Spinal subarachnoid block	Low	Xanthochromic	0	0-50	Normal	500-3000
Pseudotumor cerebri	Increased	Clear	0	0-5	Normal	Normal

CSF of Premature Infants or Neonates. The normal CSF of premature infants and neonates is not the same as adults'. The fluid is typically clear but slightly xanthochromic, contains less than 40 WBC per mm³. RBCs are frequently found, the protein varies between 20 and 150 mg per dl, and the CSF glucose level is approximately equal to that of the plasma.

Patterns of CSF abnormalities found in a variety of neurologic disorders are shown in Table 4-9.

CEREBROSPINAL FLUID PATHWAYS AND HYDROCEPHALUS

Normal Flow and Absorption of CSF

Cerebrospinal fluid is formed by the choroid plexus in both lateral ventricles, third, and fourth ventricles. The CSF volume in adults is estimated to be 90 to 150 ml, 500 ml being formed per day. When CSF flows out of the fourth ventricle, it follows the

TABLE 21-6. CENTRAL CAUSES OF VERTIGO

Vascular disease	Demyelinating disease
TIAs of the vertebrobasilar system	Multiple sclerosis
Strokes of posterior circulation	Postinfectious demyelination
Cerebellar hemorrhage	Progressive multifocal leukoencephalopathy
Cerebellar infarction	Degenerative disease
Subclavian steal syndrome	Friedreich's ataxia
Arteriovenous malformation	Olivopontocerebellar degeneration
Aneurysm	Ophthalmoplegia plus syndrome
Vasculitis	Refsum's disease
Posterior fossa lesions	Temporal lobe epilepsy
Primary brain tumors (glioma, meningioma, epidermoid, sarcoma)	Vestibulogenic reflex epilepsy
Ependymoma	Cranial neuropathies affecting the 8th nerve
Metastases	Sarcoid
Subdural hematoma	Cancer (meningeal carcinomatosis)
Temporal bone cyst	Sjögren's syndrome, Vogt-Koyagnami-Haradas syndrome
Hydrocephalus	Guillain-Barré syndrome
Syringobulbia	Paget's disease
Platybasia	Osteopetrosis
Arnold-Chiari syndrome	Infections
Basilar artery migraine	Meningitis
Trauma	Herpes zoster (Ramsay Hunt)
	Abscess (parenchymal, epidural, subdural)

barometric pressure in an airplane, or examiner induced increases or decreases (Hennert's sign) of pressure. Perilymph fistulas may occur after trauma, infection, and other causes, and can be corrected surgically.

Occlusion of the Internal Auditory Artery

The sudden onset of acute vertigo and deafness without change of consciousness or other neurological signs can occur when the patient suffers an occlusion of the internal auditory artery. This artery either comes directly from the basilar artery or can be a branch of the anterior inferior cerebellar artery. Occlusion occurs in patients with vasculitis, atherosclerosis, or other arterial abnormalities.

Central Causes of Vertigo

Vertigo due to central causes is usually associated with other central nervous system (CNS) symptoms or signs. If a central lesion is suspected from history and examination, audiogram, ENG, BAER, and CT and/or MR are usually indicated. Specific diseases which cause central vertigo are listed in Table 21-6.

VASCULAR DISEASE. *Transient Ischemic Attacks (TIAs).* Vertebrobasilar TIAs are often associated with vertigo and ataxia. Vertigo alone may be the first sign of vertebrobasilar insufficiency. However, most patients will have had one other accompanying sign or symptom of brain stem dysfunction within several months of the

onset of vertigo. In order to make a definite diagnosis of vertebrobasilar insufficiency in vertiginous patients, they must have had nonvestibular brain stem dysfunction combined with vestibular dysfunction at least once. This might be vertigo combined with diplopia, numbness, dysarthria, or dysphagia. Total blindness, numbness or weakness on both sides of the body, and ataxia usually indicate posterior fossa ischemia. TIAs generally last 10 to 20 minutes and resolve. Neurological testing is normal between attacks. Vertigo is seldom a feature of carotid artery disease.

Stroke. Infarction of the brain stem may cause vertigo and is invariably associated with other brain stem signs. The most common of these syndromes is a lateral medullary stroke, often caused by occlusion of the vertebral or posterior inferior cerebellar artery.

Cerebellar Hemorrhage. The acute onset of headache, nausea, vomiting, depressed consciousness, and ataxia suggests the possibility of a cerebellar hemorrhage, usually a surgical emergency. This syndrome may mimic acute labyrinthitis except that patients with cerebellar hemorrhage often have a severe headache and become lethargic or comatose. In many cases of cerebellar hemorrhage there may be other signs including diplopia, facial weakness and numbness, pupillary abnormalities, dysarthria, dysphagia, limb ataxia, and long tract signs. Cerebellar hemorrhage requires emergency CT and immediate surgical evacuation of the blood clot in the posterior fossa if it is endangering the patient's life.

Subclavian Steal Syndrome. This rare syndrome is characterized by symptoms of brain stem ischemia produced by exercise or movement of one arm, usually with a decreased or absent peripheral pulse and decreased blood pressure in that arm. This syndrome is caused by a stenosis of the left subclavian artery proximal to the origin of the vertebral artery or a stenosis of the right subclavian artery proximal to the origin of the vertebral artery. Increased blood flow to the left arm, precipitated by various factors, results in retrograde flow down the vertebral artery, causing brain stem ischemia. Patients may experience TIAs with vertigo as one of the symptoms.

Basilar Artery Migraine. Some patients have definite vertigo or other brainstem symptoms which occur prior to, during, or after their otherwise typical migraine headaches. Severe nausea, vomiting, and vertigo may occur. Structural lesions should be ruled out. The syndrome occurs most often in young women and may resolve with treatment for migraine. Rarely, patients have loss of consciousness associated with the syndrome. Dilantin, propranolol, and santsert are useful for treating this disorder.

POSTERIOR FOSSA LESIONS. Any posterior fossa lesion may produce vertigo or dizziness. There are usually cranial nerve or cerebellar symptoms or signs which may be associated with alterations of consciousness. Posterior fossa lesions can press directly on brain stem structures or compress the aqueduct and produce hydrocephalus. Masses around the fourth ventricle can give rise to Brunn's syndrome. This syndrome includes (a) vertigo, nystagmus, and vomiting sometimes associated with loss of consciousness on head turning, (b) freedom from symptoms until the head is turned, and (c) maintenance of the head in a particular position in order to prevent attacks. Most posterior fossa lesions can be diagnosed by contrast CT or MR.

DEMYELINATING DISEASES. Multiple sclerosis can cause isolated vertigo or

vertigo associated with other brain stem findings during an acute exacerbation. Changes of hearing do not usually occur in MS. The vertiginous symptoms usually resolve.

Epilepsy. Some patients have vertigo as the initial manifestation of a generalized seizure or a complex partial (psychomotor or temporal lobe) seizure. It is assumed that the seizure focus in these cases is located in the temporal lobe. Vertigo can be elicited by electrical stimulation of portions of the temporal and inferior frontal lobe in humans. Vestibulogenic epilepsy is a rare type of reflex epilepsy in which vestibular sensory input precipitates a generalized seizure.

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orientation as to place is seen in moderate disturbances of cerebral function, and impairment of orientation as to person is present with severe cerebral dysfunction.

Speech

Speech is produced by the delicate coordination of respiratory muscles, vocal cords, soft palate, tongue, and lips. Dysfunction in any of these produces distinctive speech abnormalities. Partial or complete paralysis of one vocal cord produces hoarseness; paralysis of both vocal cords results in aphonia. Dysfunction of the soft palate produces a distinctive hypernasal speech. The classic pseudobulbar speech associated with bilateral subcortical lesions is characterized by hypernasality, slurring of words, and an apparently great effort with reduced output. Patients with multiple sclerosis (MS) frequently exhibit "scanning" speech, in which each syllable is pronounced with equal strength and the intonation of normal speech is lost. In patients with parkinsonism, speech becomes very soft and may be barely audible. Most speech abnormalities are easily detected during normal conversation with the patient. Lip movements can be tested by asking the patient to say rapidly "mememememememe," tongue movements by "lalalalala" and palatal movements by "gagagagagagaga." In addition, phrases such as "around the rugged rock the ragged rascal ran" may be used. Practically all speech abnormalities except for speech apraxia imply peripheral, brain stem, and/or cerebellar dysfunction.

Language

As opposed to speech dysfunction, impairment of the ability to use abstract language symbols almost always implies cortical damage, usually of the dominant hemisphere. Aphasic patients may have difficulty expressing themselves in speaking or in writing, or may have difficulty comprehending spoken or written language. Although one of these functions may be prominently impaired, more often than not all four functions are disturbed to some degree. Language comprehension is best tested by asking the patient to follow both spoken and written commands, and by determining if the patient comprehends what has been said or written. Language production is tested by asking the patient to talk and write. Eight types of aphasia can usually be recognized. These aphasias and their typical features are presented in Table 2-1.

In Broca's aphasia, the lesion is located in the inferior and posterior portion of the dominant frontal lobe. Patients typically are nonfluent but use substantive words; their speech is slow, produced with great effort, and poorly articulated. There is a marked reduction of language output, but comprehension is usually good. The same mistakes are almost always seen when the patient writes. The ability to name objects is also affected frequently. This type of aphasia is usually accompanied by a hemiparesis on the contralateral side that is worse in the arm than the leg. The patient is aware of the deficit and frequently is frustrated and depressed.

In Wernicke's aphasia, the lesion is located in the posterior and superior portion of the dominant temporal lobe. Language production is fluent, with normal articulation

Clinical Applications

Refractive Errors. Pattern-reversal VEPs can be used to estimate objectively refractive errors and visual acuity in patients who cannot be tested subjectively. This follows, because the amplitude of PRVEPs is related to the clarity of focus of the image in the retina. Astigmatic errors can be detected by performing VEPs to patterns of various orientations.

Retinal Disease. Flash visual evoked potentials (FVEPs) are useful in conjunction with the electroretinogram (ERG) in the evaluation of retinal disease. The ERG is recorded from electrodes placed on or around the eye, and primarily reflects activity in the outer retinal receptor cells. The scalp-recorded VEP depends on the functional integrity of the central visual pathways from the retinal ganglion cells to the visual cortex. Diseases such as Tay-Sachs that affect the retinal ganglion cells with preservation of receptor cells alter the VEP. The ERG in these patients remains unaffected. Conversely, in diseases such as retinitis pigmentosa that affect the outer cells of the retina but do not effect the macula until later stages the ERG is either absent or decreased and the VEP remains unaffected.

Optic Nerve Dysfunction. Visual evoked potentials may be abnormal in a variety of diseases involving the optic nerve. However, VEPs have proven the most useful for evaluating patients with MS. During the acute stage of optic neuritis, when visual acuity is reduced, PRVEPs show prolonged latency and decreased amplitude. The amplitude decrease is sometimes of such a degree that VEPs are undetectable (Fig. 7-3, bottom right trace). If the patient improves, the amplitude gradually increases and may turn to normal levels, but the latency usually remains prolonged even in patients whose visual acuity returns to normal. The PRVEP can thus document optic nerve dysfunction in patients with a history of optic neuritis, even when visual fields, acuity, and funduscopic examinations are normal. Not all patients with a history of optic neuritis, however, have abnormal PRVEPs. Conversely, abnormal PRVEPs are found in at least one third of MS patients who have no history of optic neuritis and who have normal standard clinical neuro-ophthalmological examinations. Thus, PRVEPs may detect subclinical optic nerve lesions and contribute to the early diagnosis of MS.

Chiasmal and Retrochiasmal Lesions. Patients with chiasmal and postchiasmal lesions may have abnormal VEPs. Their value in the evaluation of patients with visual field defects, however, is still not well established. Recording over both sides of the head may allow lateralization of the lesion.

CONCLUSIONS

Evoked potentials provide valuable information for the diagnosis of neurological disorders that cause dysfunction of the auditory, somatosensory, and visual pathways

to about 0.2 cm. Objects that contrast strongly against the background are much easier to discern than those that have a density close to that of surrounding structures. To improve the detectability of a lesion, the radiologist may elect to inject intravenous radiographic contrast medium. Many lesions then are enhanced and show a markedly increased density. This is particularly true of lesions in which the blood-brain barrier is destroyed (e.g., infarct, many tumors, abscesses), where the contrast medium leaves the circulating blood pool and leaks out into the abnormal tissue. Hypervascularity of a lesion is another factor that will contribute to enhancement of contrast medium. Radiographic contrast media may also be injected intrathecally in order to opacify the cerebrospinal fluid and render the extracerebral cisterns clearly visible.

DIAGNOSTIC POWER AND LIMITATIONS. CT is capable of excellent density resolution as well as linear resolution and will consequently reveal both structural and morphological abnormalities. For about 15 years this dual capability has made CT an unsurpassed diagnostic modality for a wide variety of intracranial diseases.

In congenital malformations all major anatomical aberrations, such as abnormally shaped ventricles or underdeveloped parts of the brain are clearly displayed. In aqueductal stenosis, the narrow aqueduct itself may not be visible, but the level of obstruction can be inferred from the appearance of the ventricles.

In trauma to the brain, CT is the preferred modality. Extracerebral and intracerebral hematomas, cerebral edema, and herniation of the brain are accurately shown (Fig. 8-4), as are important sequelae of trauma such as chronic hematomas, porencephalic cysts, and cerebral atrophy.

The yield of CT is also high in cerebrovascular diseases. Intracerebral hemorrhages, visible as areas of increased x-ray attenuation, are consistently demonstrated. The detection rate of cerebral infarcts is not quite as high, probably in the range of 80%. Most infarcts are not detectable during the first 2 to 3 days after the clinical incident but become visible during the ensuing 7 to 21 days. Transient ischemic attacks frequently leave no trace on CT scans because in most instances no organic brain injury of sufficient magnitude has occurred. Arterial aneurysms are detected by most CT scanners, provided that their diameter exceeds 4 to 5 mm. After rupture of an aneurysm, the scanner reveals the extent of the intracranial hemorrhage. The location of the aneurysm may be inferred from the distribution of the hematoma. The presence of an arteriovenous malformation is suggested by curvilinear calcifications and dilated serpentine vascular spaces. Most of the vascular abnormalities diagnosed or suggested by CT require angiography for definitive diagnosis.

Localized inflammatory processes are shown with a high degree of accuracy. They usually evolve from a solid inflammatory focus to an area of cerebritis that later may produce an abscess with a vascular capsule and a necrotic core. The final result is cavitation or resolution. All these stages are recognizable by CT. Diffuse inflammatory processes, such as viral encephalitis, are detectable, although with less accuracy.

Demyelinating diseases, previously not diagnosable by any radiological modality, may be shown by CT, although not consistently. Important examples are multifocal leukoencephalopathy (PML) and multiple sclerosis (MS). In the latter disorder

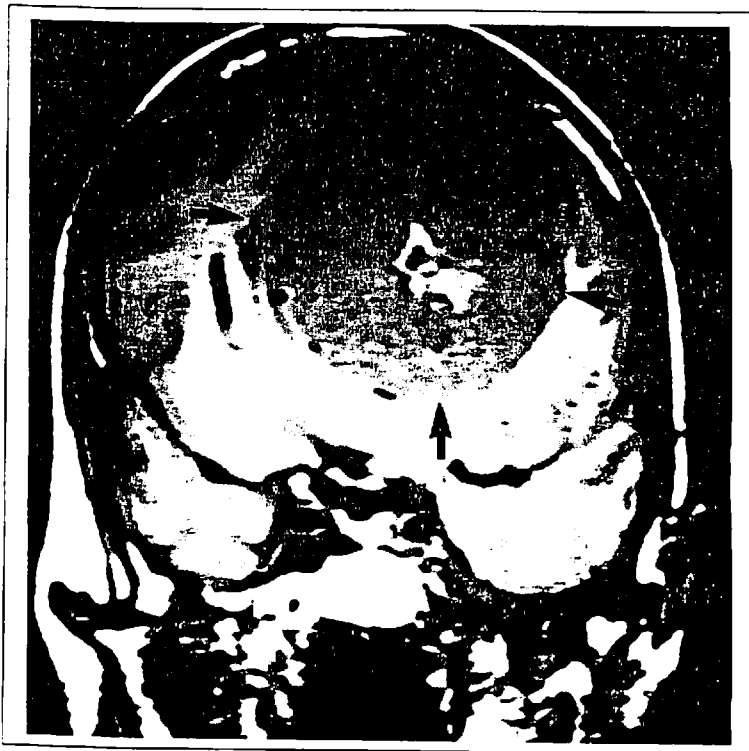


Figure 8-8. MR image scan of brain (coronal plane) showing a large meningioma.

inflammatory foci in patients with AIDS, in whom no abnormalities have been observed on CT. On the other hand, old calcified inflammatory foci are poorly seen by MRI.

Chronic sequelae of head trauma—contusions, hematomas, substance loss—are readily diagnosed by MRI, but acute trauma is at present better examined by CT. Fresh hematomas, fracture lines and depressed bone fragments are poorly seen by MRI. In addition, acute trauma patients may be attached to respirators and monitoring devices; all these factors favor the use of CT rather than MR in the emergency situation.

One of the more notable strengths of MRI lies in its ability to reveal abnormalities of the white matter. Small, but numerous, ischemic lesions of white matter are commonplace in older persons; these are difficult to see on CT scans but are quite apparent on MRI. The white matter plaques of multiple sclerosis, only occasionally seen by CT in acute cases, are very obvious on MR scans whether they are new or old (Fig. 8-10) and the diagnosis may be confirmed by the findings of additional plaques deep in the cerebellum and in the spinal cord.

The diagnostic limitation of MRI for diagnosis of CNS diseases is mainly its relative inability to detect calcifications and to demonstrate fresh hematomas. Another dis-

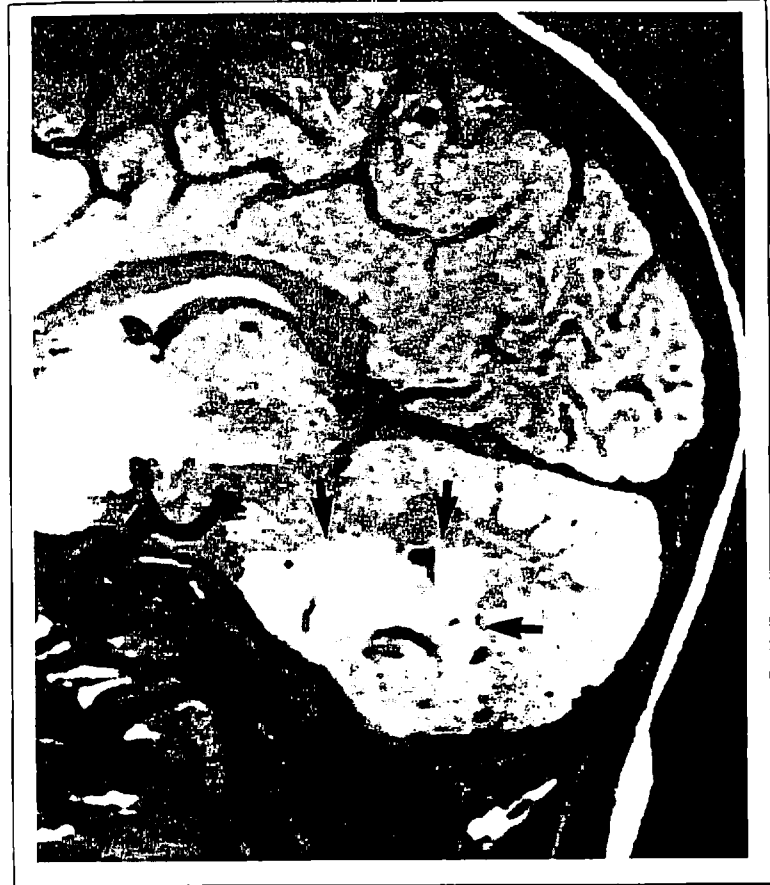


Figure 8-9. MR scan of brain (sagittal plane) showing large ependymoma of cerebellum.

advantage, apart from the high cost of the equipment, is that contraindications to MR examination exist for certain patient categories. Patients with surgically implanted metallic devices should in many cases not be placed in the strong magnetic field of the scanner, and patients with pacemakers cannot be examined. Further, severely ill patients attached to monitors and other equipment with ferromagnetic components cannot be examined. In spite of these shortcomings MR imaging represents a very real advance in the diagnosis of diseases of the brain.

Magnetic resonance has for many years been successfully used for chemical and biochemical analysis. Examination of metabolic abnormalities of the brain by MR spectroscopy is still in its infancy but there is not the slightest doubt that this modality will become a clinical reality within the next few years. The cost of an MR scan of the brain is in the range of \$700 to \$900.

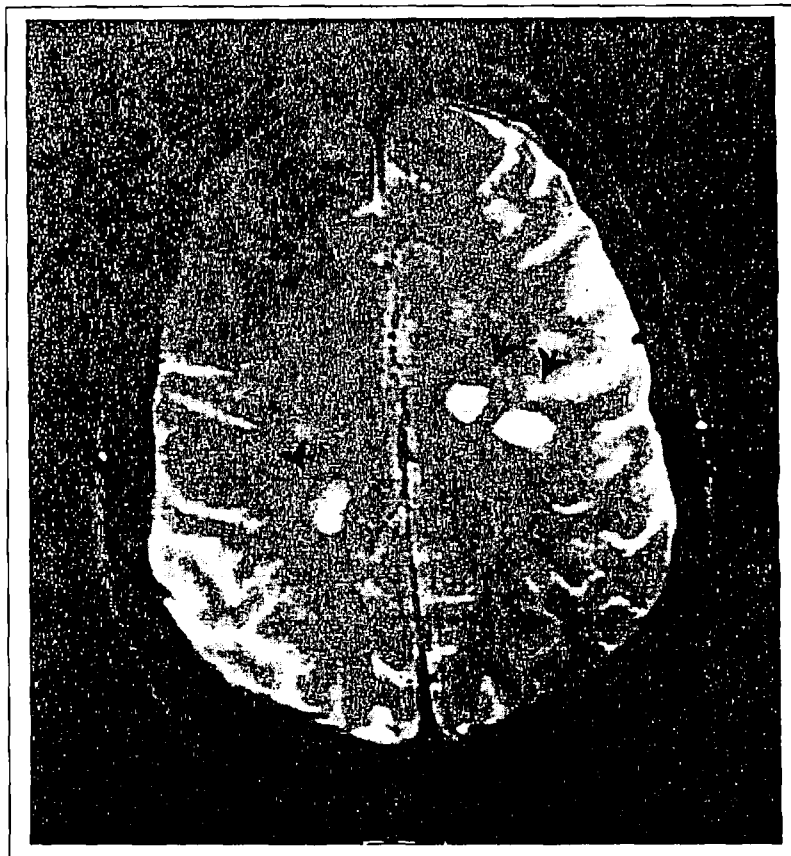


Figure 8-10. MR view of brain showing multiple cerebral plaques of multiple sclerosis.

RADIOLOGY OF THE SPINE AND SPINAL CORD

About 15 years ago, plain film radiography of the spine was frequently the only x-ray examination done on a patient with back pain or other symptoms from the spine and spinal cord. Today, a cornucopia of modalities—spine film, tomography, myelography, angiography, radioisotope scanning, CT, and MRI—are widely available (perhaps with the exception of MRI) and the probability of arriving at a correct diagnosis is greater than ever before. In most patients only one or two of these techniques are used. In the interest of expediency and cost effectiveness it is important to try to select those that are most likely to yield the best information in each patient.

Plain Films of the Spine

Plain films of the spine demonstrate only bony structures and soft-tissue calcifi-