

contractures, perhaps certain medications, may have a negative effect on walking. We are not sure, but there are some animal models that say some of the medications that help reduce spasticity may also have the effect of retarding the neural response to certain training. Is that a fair estimation?

Behrman: All are correct. Let me comment on the last, though. Again, that is something that we don't know. We don't know the interactions of some of these spasticity-reducing medications on the training or a lot of the other activities.

Levy: What lessons has this therapy taught us that allows us to be more hopeful than we might have been. Is there any reason to be more optimistic about the potential for functional restoration than we were ten years ago?

Behrman: Yes. I think we talked a little bit about this, but one view that we have had is that the spinal cord is simply a telephone cable that carries messages. But, evidence from scientists indicates that there is circuitry within the spinal cord itself that can contribute to the control of walking. If we can tap this ability in physical rehabilitation, it may further enhance the ability to walk after SCI. Another view has been that the damage after SCI is permanent. This is beyond our current discussion, but there are certainly researchers who are examining regeneration or growth strategies after injury. Growth stimulants or growth factors or transplants may help resolve, somewhat, some of these disconnections or make more appropriate connections in the spinal cord. It turns out that the spinal cord really can learn very similarly to the way that the brain can learn. In the past we assumed that the recovery of walking, as you and I know it, was

not possible based on the anatomy and physiology of the spinal cord. Now there is an abundance of opportunity to facilitate this recovery of walking. Think about other individuals with other pathologies or neurological problems. If I have had a stroke, my spinal cord is still intact. Now, some of the input from the brain is altered but I still have an intact cord itself. Maybe we can use information from the spinal cord to teach the brain something. So, we are kind of working in reverse here. I think that is the opportunity. Perhaps it is not a permanent loss. It may be repairable and if it is repairable, then specific training may stimulate and drive the repair in the right direction.

Levy: So, we are presenting a new hopeful therapy. It is not standard therapy and it is not something that someone is likely to be able to go to their local PT gym now and enroll in. What advice would you give to somebody who has got a disability from a spinal cord injury or a stroke or some other process?

Behrman: Stay in the best shape you can and the best level of fitness that you can. For each person that may be a little bit different. If you have the opportunity to stand, for example, then stand. Standing is a great way to maintain the joint flexibility of your knees and ankles. For some people standing is not available. If not, then a person should make sure to try to lie on his or her stomach to maintain hips flexibility. You never know what advance is coming down the road. Lung capacity is important. Smoking is not beneficial for walking or other activities. Persons who have joint and muscle flexibility and are more fit will be in a better position to accept the opportunity to pursue walking or whatever therapy may be available in the future.

Another important element is to stay informed. There are many other sites or opportunities to communicate with others or learn about research all the way from scientific literature to the science section of the New York Times to websites. There are popular magazines, such as New Mobility or Paraplegia News that will have articles to websites on National Institute of Health and the Veterans Affairs research. So, I would recommend that persons read, stay in touch, and keep fit. Transverse myelitis is a syndrome of spinal cord dysfunction in which most of the functions, namely motor, sensory and sphincter are compromised to some degree below a definable sensory level. In more than 80 % of cases, the peak of

Attempting To Recognize Transverse Myelitis Subtypes

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dysfunction is seen within 10 days. Before the onset of neurological symptoms, non-specific symptoms, such as fever, nausea and muscle pain occur. Initial neurological signs are paresthesias, backache and paresis of legs. Sphincter dysfunction often occurs when weakness and sensory disturbances are present. Thoracic level occurs in 80 %, while cervical and lumbar levels occur in 10% each. In the clinical setting, it is often difficult to distinguish among the various possible causes of TM. MS is often considered a strong possibility, which might not always be the case. Retrospective analyses have provided

useful information regarding the acuity of presentation and its relationship with prognosis, but have not often sorted the various etiologies.

Ropper and Poskanzer studied 52 patients and suggested that patients with acute onset of symptoms tended to have poor prognosis. They reported 4 types of presentation according to symptoms, including paresthesias, pain, weakness and urinary retention. 37 % of patients gave history of previous infection, and 13 % were later diagnosed with MS, but presentation, severity of disease and prognosis were not compared.

Berman studied 62 patients in Israel, reported incidence of 1.34/mill/year and no seasonal preponderance. 37% had a history of previous infections and only one patient developed MS. No attempt was made to characterize clinical differences among groups. Prior to our study, a clinical and laboratory comparison of TM based on presumed cause had not been performed.

We attempted to determine whether clinical differences related to various etiological subgroups. We also presented an estimate of the incidence of TM in a US population.

In our study we found that various types of TM may be distinguishable on the basis of clinical presentation, imaging and CSF studies. In contrast with previous studies, which isolated homogeneous patient populations by studying only the most severe cases, we attempted to classify cases according to cause by means of objective criteria.

Patients with parainfectious-TM (PI-TM) were weaker on initial exam and suffered spinal shock more frequently. They were likely to have more severe and persistent back pain. They have ascending cord dysfunction more

frequently and over a great number of segments.

Patients with MS-TM more often had numbness and paresthesias and tended to show spastic paraparesis on initial exam.

PI-TM had a tendency to show spinal cord swelling, while patients with MS-TM tended to show small plaque-like lesions with gadolinium enhancement. The PI-TM CSF did not contain Oligoclonal bands, while the MS-TM CSF did. The incidence of TM was 4.6/million/year. In the PI-TM 73% of infections were from the upper respiratory tract.

Recurrences were seen also in the PI TM. The second episode followed periods of neurologic stability up to years and were not preceded by URI.

Prognosis for recovery of ambulation and bladder control was variable. Patients with PI-TM were less likely to recover bladder function or independent ambulation at discharge. However, in the long term, patients with MS more likely required catheterizations. Recovery varied. If ambulation was not recovered within 2 months, patients with PI-TM tended to remain wheelchair bound.

While the number of cases in each category was small and the retrospective nature of the study limits the strength of the conclusions, there do appear to be differences between PI-TM and MS-TM. A larger prospective study should provide more conclusive information.

Abstract

This article summarizes the clinical presentation and the available information on the neuropathophysiology of neuropathic pain. A review of the somatosensory nervous system anatomy involved in neuropathic pain is also included. The focus is on the peripheral

The Pathology Of Neuropathic Pain

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nervous system (PNS) because its mechanisms of pain are better defined than in the central nervous system (CNS). Also, the vast majority of neural damage occurs in the PNS, most likely due to its increased exposure to traumatic injury compared with the CNS. Clinicians who recognize some of the cardinal signs of the most common neuropathies can diagnose and treat their patients earlier, reducing the burden of neuropathic pain. Advances in our understanding of the neural mechanisms of chronic pain will hopefully help to define therapeutic strategies in the future.

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There are 2 types of chronic pain: inflammatory nociceptive pain, which is pain associated with tissue damage, and neuropathic pain, which arises from damage to the nerves that carry pain signals from tissue, not damage