



"The exact cause is not yet known"

Parkinson's Disease

By Alain Delgado, M.D.

First described by James Parkinson in 1817, this disease afflicts about 1% of the population over age 65. Typically beginning after age 50, this disease is characterized by many symptoms. Commonly, patients describe a tremor at rest, stiffness, slowing down, and later in the disease, a problem with falling. Unfortunately, there are no tests that can be performed to make the diagnosis and misdiagnosis occurs in as many as 25% of patients. Therefore, it is important to evaluate for other potential causes while obtaining a good clinical history and examination.

Depending on onset and signs and symptoms, this disease is characterized as either possible, probable, or definite. Once diagnosis is established, the disease severity is classified into 1 of 5 stages.

If it is not Parkinson's Disease then what is it? It may represent medication side effects, strokes, hydrocephalus, toxic substances, metabolic derangements, essential tremors, or tumors. It may be a Parkinson's Plus syndrome that may have atypical features and not respond to typical medications used to treat Parkinson's Disease.

Although the exact cause of Parkinson's Disease is not yet known, it has been established that there is a degeneration of cells in the substantia nigra in the brainstem. This degeneration leads to a depletion in a neurochemical called dopamine.

Most cases of this disease are sporadic, although genetics may play a

role.

Although there is no therapy yet that can slow down or reverse the progression of this disease, there are many symptomatic therapies available. Some medications include levodopa/carbidopa, entacapone, tolcapone, bromocriptine, pramipexole, pergolide, ropinirole, amantadine, selegiline, trihexylphenidyl, and bentsropine mesylate. With all the medications available, one can see there are many options and combinations to be used for the improvement of symptoms.

Should medications fail or be intolerable, there are effective surgical options. One possible option includes implantation of a deep brain stimulator. This implantable device carries a low morbidity and mortality and may provide excellent control of symptoms.

Neurology is an exciting specialty always with new treatments on the horizon. Certainly Parkinson's Disease is one of those neurological diseases that continues to be avidly researched with a wealth of information being discovered.

Peripheral Neuropathy

By Shailesh U. Rajguru, D.O.

Peripheral neuropathy is a disorder involving malfunction of electrical transmission of impulses of the nerves in the extremities.

Nerves are a profoundly complex network of wiring that controls our motor functions (strength), sensations perceived, not perceived, or abnormally perceived. These include sometimes confusing, or difficult to describe symptoms of numbness, yet still feeling hypersensitivity to touch, burning or hot sensation, tingling ("crawling ants"). Peripheral nerves (peripheral nervous system) are the extension of the nerve roots, and surround peripheral structures including blood vessels, muscles and visceral organs such as the intestines, heart and lungs (autonomic nerves). When peripheral neuropathy is diagnosed, involvement is typically of the extremities. The autonomic nervous system is usually preserved with the exception of atypical or severe cases. A nerve is identical to a piece of electrical wire (called an axon) that is covered by a insulatory sheath (called myelin). Malfunction can occur at the level of the axon (nerve) or its sheath (myelin). Depending on the cause, treatment and prognosis can vary based on the type of neuropathy. Symptoms of neuropathy initially begin distally (toes, feet, fingers, hands), and may progress over time to affect the extremity as a whole.

Although numbness or pain symptoms are potentially treatable for relief, the actual treatment of peripheral neuropathy is treatment of the underlying cause. However, despite extensive and thorough evaluations, approximately 10 percent of neuropathy patients remain without a cause identified (termed idiopathic peripheral neuropathy).

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Reflex Sympathetic Dystrophy (RSD)

By Julie Esquinaldo, PA-C

RSD, also referred to as Sudecks Atrophy, minor causalgia, and post traumatic neuralgia, is a painful syndrome which occurs following injury to bone and/or soft tissue. This usually follows trauma, surgery, or nerve injury to a limb or peripheral nerve. It can occur in adults and children. It is often associated with anxiety and depression.

There are three stages of RSD: the acute stage, dystrophic stage, and the atrophic stage. The acute stage occurs within hours to days of the injury and is characterized by a burning or aching pain occurring over the injured extremity. The limb is exquisitely sensitive to touch. Swelling is usually present as well as temperature change. The skin will become red, warm, and swollen. There may also be increased hair and nail growth. The dystrophic stage occurs within three to six months after the injury and is associated with burning pain radiating both distally and proximally from the injury site. Swelling and sweating can occur. There may also be hypothermia and cyanosis of the extremity. Muscle tremors and spasms can occur along with increased muscle tone and hyperreflexia. The third stage or atrophic stage occurs six months after surgery or trauma. The pain usually spreads proximally at this point. The skin is usually cold, pale, and cyanotic. Atrophy of the muscle and tissue is seen, joints are usually fixed or immobile, and there are contractures of the extremity.

The cause of RSD is unknown, but is thought to arise from a "short-circuit" among the injured nerve fibers. The following injuries more commonly can cause RSD: a crush trauma, burns, frostbite, surgery, Parkinson's disease, stroke, myocardial infarctions, osteoarthritis, cervical and lumbar disc disease, carpal and tarsal tunnel syndromes, diabetes, and hyperthyroidism.

Although there are no specific

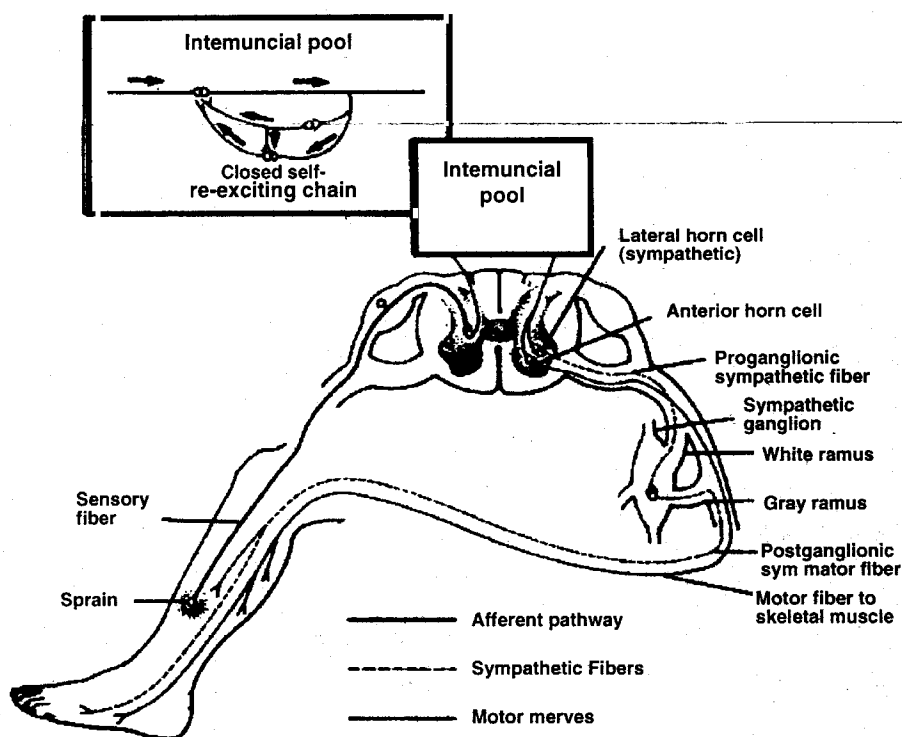
tests to establish the diagnosis, an increased uptake seen on a radionuclide bone scan, bone loss seen on an x-ray, and thermography may be useful confirmatory tests, but are not necessary to make the diagnosis.

Treatment is aimed at relieving pain and improving use and atrophy of the affected extremity. Physical therapy is an important part of treatment. TENS units may be helpful as well as acupuncture. For acute treatment, different medications such as those used for treating seizures, calcium channel blockers, and prednisone may be used. More invasive procedures include use

of anesthetics or sympathetic nerve blocks. Stellate ganglion blocks may be tried, and finally, surgical sympathectomy may be tried for patients unresponsive to conservative measures. These procedures would be done by an anesthesiologist or a pain management physician.

In summary, reflex sympathetic dystrophy is difficult to diagnose and can be difficult to treat. Spontaneous remission can occur within weeks to months. Patients can progress through all stages. Pain is the most disabling symptom and is usually much greater than the actual injury itself.

Complex Regional Pain Syndrome Type 1

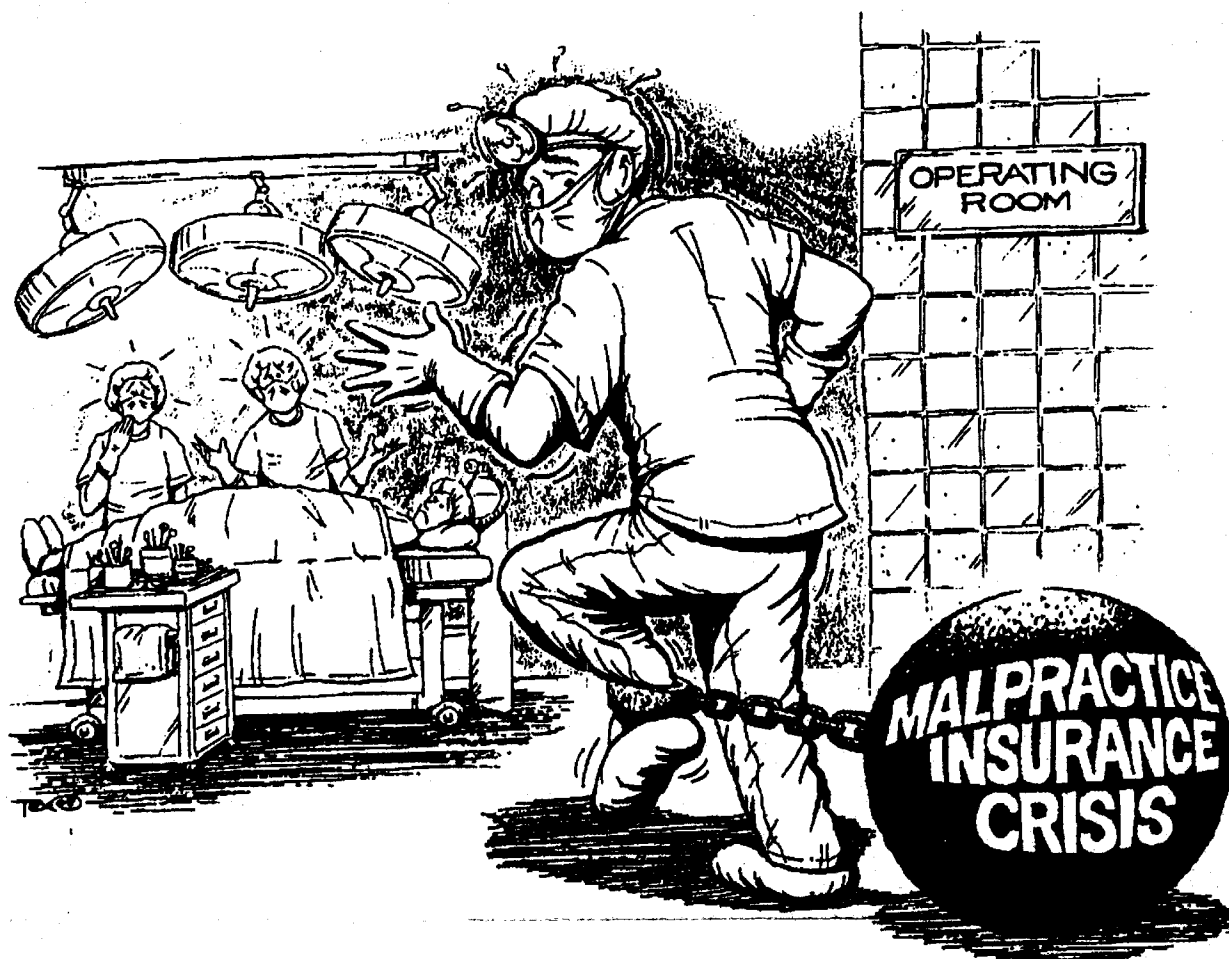


Livingston's Theory of Reverberating Circuits

(Purdy and Miller 1992,p: 1126)

Livingston adopted the theory of Lorento de No relative to the "intemuncial pool" to explain the vicious cycle of impulses within the spinal cord instigated by the afferent pain stimuli.

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What if this were your surgeon?

The crisis over medical malpractice insurance has reached a fever pitch. Insurers in most states are raising already exorbitant rates. Surgeons and specialists are considering the unthinkable—leaving the profession. And patients are caught in the crossfire—faced with limited access to care or forced to seek treatment in other states.

In more than a dozen states, physicians are staging rallies and work slow downs to bring attention to the crisis over malpractice insurance. Some are abandoning already under-served specialty areas as neurosurgery and obstetrics. Still others are relocating to states that offer medical liability relief.

Patients, too, bear the brunt of runaway medical malpractice costs. The Congressional Budget Office estimates that the threat of suits costs taxpayers as much as \$25 billion a year in needless Medicare costs and other government expenses.

Unless Congress does something NOW to curb excessive damage awards in malpractice cases, the crisis threatens to jeopardize the quality of health care for every American family. Your surgeon or specialist could be the next doctor run out of business.

It's time for both sides of Congress to work with President Bush to limit awards and reduce costs associated with medical malpractice. As the President has made abundantly clear, medical liability reform is an absolute priority of this nation.

Please join us in urging support of the bipartisan HEALTH Act sponsored by Representative Jim Greenwood. Call or write your Senators and Representative and ask them to vote for common-sense medical liability reform—for the good of both patients and physicians.



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Peripheral Neuropathy *(continued from page 1)*

Types of peripheral neuropathy:

Axonal (affecting the nerve itself)

Demyelinating (affecting the insulatory lining of the nerve)

Focal/entrapment neuropathy (carpal tunnel syndrome)

Course of peripheral neuropathy:

Acute (typically following viral infection). Only neuropathy diagnosis that is an emergency! Must be treated immediately in the hospital with intravenous gamma globulin. Otherwise termed AIDP (Acute Inflammatory Demyelinating Polyneuroradiculopathy), or Guillain-Barre Syndrome.

Chronic (CIDP) Chronic form of AIDP, although can occur as an independent diagnosis without preceding history of AIDP. Treatment is intravenous gamma globulin. Chronic forms include metabolic

causes (diabetes mellitus, etc. listed below).

Causes of Peripheral Neuropathy:

Diabetes mellitus

Excessive alcohol use

Hypothyroidism

Vasculitis (rheumatoid arthritis, lupus, paraneoplastic (cancer related), amyloid)

Vitamin B12 deficiency

Excessive pyridoxine (Vitamin B6) use

Certain medications (amiodarone, anti-cancer medications, antibiotics)

Autoimmune (antibody related)

Acquired (viral)

Heavy metals toxicity (e.g. lead)

Evaluations:

Nerve conduction study of affected areas

Needle Electromyography examina-

tion (EMG)

Blood work for cause suspected

Spinal fluid evaluation (acquired neuropathy)

Nerve biopsy

Treatment:

Medications for symptomatic relief (typically anticonvulsants, tri-cyclic antidepressants.)

Opiate analgesics for neuropathy-type pain (neuropathic pain) are of minimal, if any benefit, and likely not suitable for long term use.

Physical therapy for conditioning, strengthening, gait training.

Intravenous gamma globulin (acquired neuropathy, autoimmune neuropathy, proximal diabetic neuropathy)

Prognosis:

Remains dependent on type of neuropathy, severity of neuropathy at time of diagnosis, cause of neuropathy, and response of individual to treatment.