

Peripheral Neuropathy

A stylized white graphic on a dark red background. It features a human silhouette with one arm raised, overlaid on a white ECG (heart rate) line that flows across the bottom of the page.

U.S. DEPARTMENT OF HEALTH
AND HUMAN SERVICES
Public Health Service
National Institutes of Health



Peripheral Neuropathy

What is peripheral neuropathy?

An estimated 20 million people in the United States have some form of peripheral neuropathy, a condition that develops as a result of damage to the peripheral nervous system — the vast communications network that transmits information between the central nervous system (the brain and spinal cord) and every other part of the body. (Neuropathy means nerve disease or damage.) Symptoms can range from numbness or tingling, to pricking sensations (paresthesia), or muscle weakness. Areas of the body may become abnormally sensitive leading to an exaggeratedly intense or distorted experience of touch (allodynia). In such cases, pain may occur in response to a stimulus that does not normally provoke pain. Severe symptoms may include burning pain (especially at night), muscle wasting, paralysis, or organ or gland dysfunction. Damage to nerves that supply internal organs may impair digestion, sweating, sexual function, and urination. In the most extreme cases, breathing may become difficult, or organ failure may occur.

Peripheral nerves send sensory information back to the brain and spinal cord, such as a message that the feet are cold. Peripheral

nerves also carry signals from the brain and spinal cord to the muscles to generate movement. Damage to the peripheral nervous system interferes with these vital connections. Like static on a telephone line, peripheral neuropathy distorts and sometimes interrupts messages between the brain and spinal cord and the rest of the body.

Peripheral neuropathies can present in a variety of forms and follow different patterns. Symptoms may be experienced over a period of days, weeks, or years. They can be acute or chronic. In acute neuropathies such as Guillain-Barré syndrome (in which the body's immune system attacks part of the peripheral nervous system and impairs sending and receiving nerve signals), symptoms appear suddenly, progress rapidly, and resolve slowly as damaged nerves heal. In chronic forms, symptoms begin subtly and progress slowly. Some people may have periods of relief followed by relapse. Others may reach a plateau stage where symptoms stay the same for many months or years. Many chronic neuropathies worsen over time. Although neuropathy may be painful and potentially debilitating, very few forms are fatal.

In diabetic neuropathy, one of the most common forms of peripheral neuropathy, nerve damage occurs in an ascending pattern. The first nerve fibers to malfunction are the ones that travel the furthest from the brain and the spinal cord. Pain and numbness often are felt symmetrically in both feet followed by a gradual progression up both legs. Later, the fingers, hands, and arms may become affected.

How are the peripheral neuropathies classified?

More than 100 types of peripheral neuropathy have been identified, each with its own symptoms and prognosis. In general, peripheral neuropathies are classified according to the type of damage to the nerves. Some forms of neuropathy involve damage to only one nerve and are called *mononeuropathies*. More frequently however, multiple nerves are affected, called *polyneuropathy*.

Some peripheral neuropathies are due to damage to the axons (the long, threadlike portion of the nerve cell), while others are due to damage to the myelin sheath, the fatty protein that coats and insulates the axon. Peripheral neuropathies may also be caused by a combination of both axonal damage and demyelination. Electrodiagnostic studies can help healthcare providers determine the type of damage involved.

What are the symptoms of peripheral nerve damage?

Symptoms vary depending on whether motor, sensory, or autonomic nerves are damaged. Motor nerves control voluntary movement of muscles such as those used for walking, grasping things, or talking. Sensory nerves transmit information such as the feeling of a light touch or the pain from a cut. Autonomic nerves control organ activities that are regulated automatically such as breathing, digesting food, and heart and gland functions. Some neuropathies may affect all three types of nerves; others

primarily affect one or two types. Doctors may use terms such as predominantly motor neuropathy, predominantly sensory neuropathy, sensory-motor neuropathy, or autonomic neuropathy to describe the types of nerves involved in an individual's condition.

Motor nerve damage is most commonly associated with muscle weakness. Other symptoms may include painful cramps and fasciculations (uncontrolled muscle twitching visible under the skin), muscle atrophy (severe shrinkage of muscle size), and decreased reflexes.

Sensory nerve damage causes a variety of symptoms because sensory nerves have a broad range of functions. Larger sensory fibers enclosed in myelin register vibration, light touch, and position sense. Damage to large sensory fibers impairs touch, resulting in a general decrease in sensation. Since this is felt most in the hands and feet, people may feel as if they are wearing gloves and stockings even when they are not. This damage to larger sensory fibers may contribute to the loss of reflexes. Loss of position sense often makes people unable to coordinate complex movements like walking or fastening buttons, or to maintain their balance when their eyes are shut.

Smaller sensory fibers without myelin sheaths transmit pain and temperature sensations. Damage to these fibers can interfere with the ability to feel pain or changes in temperature. People may fail to sense that they have been injured from a cut or that a wound is becoming infected. Others may not detect pain that

warns of impending heart attack or other acute conditions. Loss of pain sensation is a particularly serious problem for people with diabetes, contributing to the high rate of lower limb amputations among this population.

Neuropathic pain is a common, often difficult to control symptom of sensory nerve damage and can seriously affect emotional well-being and overall quality of life. Often worse at night, neuropathic pain seriously disrupts sleep and adds to the emotional burden of sensory nerve damage. Neuropathic pain can often be associated with an oversensitization of pain receptors in the skin, so that people feel severe pain (allodynia) from stimuli that are normally painless. For example, some may experience pain from bed sheets draped lightly over the body. Over many years, sensory neuropathy may lead to changes in the skin, hair, as well as to joint and bone damage. Unrecognized injuries due to poor sensation contribute to these changes, so it is important for people with neuropathy to inspect numb areas for injury or damage.

Autonomic nerve damage symptoms are diverse since the parasympathetic and sympathetic nerves of the peripheral nervous system control nearly every organ in the body. Common symptoms of autonomic nerve damage include an inability to sweat normally, which may lead to heat intolerance; a loss of bladder control; and an inability to control muscles that expand or contract blood vessels to regulate blood pressure. A drop in blood pressure when a person moves suddenly from a seated to a standing position (a condition known as postural or orthostatic hypotension)

may result in dizziness, lightheadedness, or fainting. Irregular heartbeats may also occur.

Gastrointestinal symptoms may accompany autonomic neuropathy. Malfunction of nerves controlling intestinal muscle contractions can lead to diarrhea, constipation, or incontinence. Many people also have problems eating or swallowing if autonomic nerves controlling these functions are affected.

What causes peripheral neuropathy?

Peripheral neuropathy may be either inherited or acquired through disease processes or trauma. In many cases, however, a specific cause cannot be identified. Doctors usually refer to neuropathies with no known cause as idiopathic.

Causes of acquired peripheral neuropathy include:

Physical injury (trauma) is the most common cause of acquired nerve injury.

- ***Injury or sudden trauma***, such as from automobile accidents, falls, sports-related activities, and surgical procedures can cause nerves to be partially or completely severed, crushed, compressed, or stretched, sometimes so forcefully that they are partially or completely detached from the spinal cord. Less severe traumas also can cause serious nerve damage. Broken or dislocated bones can exert damaging pressure on neighboring nerves.
- ***Repetitive stress*** frequently leads to entrapment neuropathies, a form of compression injury. Cumulative damage

can result from repetitive, awkward, and/or forceful activities that require movement of any group of joints for prolonged periods. The resulting irritation may cause ligaments, tendons, and muscles to become inflamed and swollen, constricting the narrow passageways through which some nerves pass. Ulnar neuropathy and carpal tunnel syndrome are examples of the most common types of neuropathy from trapped or compressed nerves at the elbow or wrist.

Diseases or disorders and their related processes (such as inflammation) can be associated with peripheral neuropathy.

- ***Metabolic and endocrine disorders*** impair the body's ability to transform nutrients into energy and process waste products, and this can lead to nerve damage. Diabetes mellitus, characterized by chronically high blood glucose levels, is a leading cause of peripheral neuropathy in the United States. About 60 percent to 70 percent of people with diabetes have mild to severe forms of nervous system damage that can affect sensory, motor, and autonomic nerves and present with varied symptoms. Some metabolic liver diseases also lead to neuropathies as a result of chemical imbalances. Endocrine disorders that lead to hormonal imbalances can disturb normal metabolic processes and cause neuropathies. For example, an underproduction of thyroid hormones slows metabolism, leading to fluid retention and swollen tissues that can exert pressure on peripheral nerves. Overproduction of growth hormone can lead to acromegaly, a

condition characterized by the abnormal enlargement of many parts of the skeleton, including the joints. Nerves running through these affected joints often become entrapped, causing pain.

- ***Small vessel disease*** can decrease oxygen supply to the peripheral nerves and lead to serious nerve tissue damage. Diabetes frequently leads to impaired blood flow to nerves. Various forms of vasculitis (blood vessel inflammation) frequently cause vessel walls to harden, thicken, and develop scar tissue, decreasing their diameter and impeding blood flow. Vasculitis is an example of nerve damage called mononeuritis multiplex or multifocal mononeuropathy, in which isolated nerves in two or more areas are damaged.
- ***Autoimmune diseases***, in which the immune system attacks the body's own tissues, can lead to nerve damage. Sjogren's syndrome, lupus, and rheumatoid arthritis are among the autoimmune diseases that can be associated with peripheral neuropathy. When the tissue surrounding nerves becomes inflamed, the inflammation can spread directly into nerve fibers. Over time, these chronic autoimmune conditions can destroy joints, organs, and connective tissues, making nerve fibers more vulnerable to compression injuries and entrapment. Chronic conditions may alternate between remission and relapse. Acute inflammatory demyelinating neuropathy, better known as Guillain-Barré syndrome, can damage motor, sensory, and autonomic nerve fibers.

Most people recover from this autoimmune syndrome although severe cases can be life threatening. Chronic inflammatory demyelinating polyneuropathy (CIDP) usually damages sensory and motor nerves, leaving autonomic nerves intact. Multifocal motor neuropathy is a form of inflammatory neuropathy that affects motor nerves exclusively. It may be chronic or acute.

- ***Kidney disorders*** may cause neuropathies. Kidney dysfunction can lead to abnormally high amounts of toxic substances in the blood that can damage nerve tissue. A majority of individuals who require dialysis because of kidney failure develop polyneuropathy.
- ***Cancers*** can infiltrate nerve fibers or exert damaging compression forces on nerve fibers. Tumors also can arise directly from nerve tissue cells. Paraneoplastic syndromes, a group of rare degenerative disorders that are triggered by a person's immune system response to a cancerous tumor, also can indirectly cause widespread nerve damage. Toxicity from the chemotherapeutic agents and radiation used to treat cancer also can cause peripheral neuropathy. An estimated 30 to 40 percent of people who undergo chemotherapy develop peripheral neuropathy and it is a leading reason why people with cancer stop chemotherapy early. The severity of chemotherapy-induced peripheral neuropathy (CIPN) varies from person to person. In some cases people may be able to ease their symptoms by lowering their chemotherapy dose or by stopping it temporarily. In

others, CIPN may persist long after stopping chemotherapy.

- **Neuromas** are benign tumors that are caused by an overgrowth of nerve tissue that develops after a penetrating injury that severs nerve fibers. Neuromas are often associated with intense pain and sometimes they engulf neighboring nerves, leading to further damage and even greater pain. Neuroma formation can be one element of a more widespread neuropathic pain condition called complex regional pain syndrome or reflex sympathetic dystrophy syndrome, which can be caused by traumatic injuries or surgical trauma. Widespread polyneuropathy is often associated with neurofibromatosis, a genetic disorder in which multiple benign tumors grow on nerve tissue.
- **Infections** can cause peripheral neuropathy. Viruses and bacteria that can attack nerve tissues include herpes varicella-zoster (shingles), Epstein-Barr virus, West Nile virus, cytomegalovirus, and herpes simplex members of the large family of human herpes viruses. These viruses can severely damage sensory nerves, causing attacks of sharp, lightning-like pain. Postherpetic neuralgia is long-lasting, particularly intense pain that often occurs after an attack of shingles. Lyme disease, diphtheria, and leprosy are bacterial diseases characterized by extensive peripheral nerve damage. Diphtheria and leprosy are rare in the United States, but the incidence of Lyme disease is on the rise.

The tick-borne infection can involve a wide range of neuropathic disorders, including a rapidly developing, painful polyneuropathy, often within a few weeks of being infected. West Nile virus is spread by mosquitoes and is associated with a severe motor neuropathy. The inflammation triggered by infection sometimes results in various forms of inflammatory neuropathies that develop quickly or slowly.

- The human immunodeficiency virus (HIV) that causes AIDS is associated with several different forms of neuropathy, depending on the nerves affected and the specific stage of active immunodeficiency disease. A rapidly progressive, painful polyneuropathy affecting the feet and hands can be the first clinically apparent symptom of HIV infection. An estimated 30 percent of people who are HIV positive develop peripheral neuropathy; 20 percent develop distal neuropathic pain.

Exposure to toxins may damage nerves and cause peripheral neuropathy.

- **Medication toxicity** can be caused by many agents in addition to those for fighting cancer. Other agents that commonly cause peripheral neuropathy as a side effect include those used to fight infection such as antiretroviral agents for treating HIV. In addition, anticonvulsant agents and some heart and blood pressure medications can commonly cause peripheral neuropathy. In most cases, the neuropathy resolves when these medications are discontinued or dosages are adjusted.

- ***Environmental or industrial toxins*** such as lead, mercury, and arsenic can cause peripheral neuropathy. In addition, certain insecticides and solvents have also been known to cause neuropathies.
- ***Heavy alcohol consumption*** is a common cause of peripheral neuropathy. Damage to the nerves associated with long-term alcohol abuse may not be reversible when a person stops drinking alcohol, however, doing so may provide some symptom relief and prevent further damage. Chronic alcohol abuse also frequently leads to nutritional deficiencies (including B12, thiamine, and folate) that contribute to the development of peripheral neuropathy.

Genetic mutations can either be inherited or arise *de novo*, meaning they are completely new mutations to an individual and are not passed along by either parent. Some genetic mutations lead to mild neuropathies with symptoms that begin in early adulthood and result in little, if any, significant impairment. More severe hereditary neuropathies often appear in infancy or childhood.

Advances in genetic testing in the last decade have led to significant strides in the ability to identify the genetic causes underlying peripheral neuropathies. For example, several genes have been found to play a role in different types of Charcot-Marie-Tooth, a group of disorders that are among the most common forms of inherited peripheral neuropathies. These neuropathies result from mutations in genes responsible for maintaining the health of the myelin sheath as well as the axons

themselves. Key characteristics of Charcot-Marie-Tooth disorders include extreme weakening and wasting of muscles in the lower legs and feet, gait abnormalities, loss of tendon reflexes, and numbness in the lower limbs.

How is peripheral neuropathy diagnosed?

The symptoms of peripheral neuropathy are highly variable. A thorough neurological examination is required to sort out the cause of the symptoms and involves taking an extensive medical history (covering symptoms, work environment, social habits, exposure to toxins, alcohol use, risk of HIV or other infectious diseases, and family history of neurological diseases). In addition, tests are usually performed to identify the cause of the neuropathy as well as the extent and type of nerve damage.

A physical examination and various tests may reveal the presence of a systemic disease causing the nerve damage. Tests of muscle strength, as well as evidence of cramps or fasciculations, indicate motor fiber involvement. Evaluation of the person's ability to sense vibration, light touch, body position, temperature, and pain reveals any sensory nerve damage and may indicate whether small or large sensory nerve fibers are affected.

Blood tests can detect diabetes, vitamin deficiencies, liver or kidney dysfunction, other metabolic disorders, and signs of abnormal immune system activity. An examination of cerebrospinal fluid that surrounds the brain and spinal cord can reveal abnormal antibodies associated with

some immune-mediated neuropathies. More specialized tests may reveal other blood or cardiovascular diseases, connective tissue disorders, or malignancies. Genetic tests are becoming available for a number of the inherited neuropathies.

Based on the results of the neurological exam, physical exam, patient history, and any previous screening or testing, the following additional tests may be ordered to help determine the nature and extent of the neuropathy:

- ***Nerve conduction velocity (NCV)*** tests can measure the degree of damage in large nerve fibers, revealing whether symptoms are caused by degeneration of the myelin sheath or the axon. The myelin covering is responsible for the very fast speed of nerve conduction. During this test, a probe electrically stimulates a nerve fiber, which responds by generating its own electrical impulse. An electrode placed further along the nerve's pathway measures the speed of impulse transmission along the axon. Slow transmission rates and impulse blockage tend to indicate damage to the myelin sheath, while a reduction in the strength of impulses at normal speeds is a sign of axonal degeneration.
- ***Electromyography (EMG)*** involves inserting a fine needle into a muscle to record electrical activity when muscles are at rest and when they contract. EMG tests detect abnormal electrical activity in motor neuropathy and can help differentiate between muscle and nerve disorders.

- **Magnetic resonance imaging (MRI)** can show muscle quality and size, detect fatty replacement of muscle tissue, and can help rule out tumors, herniated discs, or other abnormalities that may be causing the neuropathy.
- **Nerve biopsy** involves removing and examining a sample of nerve tissue, most often from the lower leg. Although this test can provide valuable information about the degree of nerve damage, it is an invasive procedure that is difficult to perform and may itself cause neuropathic side effects.
- **Skin biopsy** is a test in which doctors remove a thin skin sample and examine nerve fiber endings. This test offers some unique advantages over NCV tests and nerve biopsy. Unlike NCV, it can reveal damage present in smaller fibers; in contrast to conventional nerve biopsy, skin biopsy is less invasive, has fewer side effects, and is easier to perform.

What treatments are available?

Address underlying conditions

The first step in treating peripheral neuropathy is to address any contributing causes such as infection, toxin exposure, medication-related toxicity, vitamin deficiencies, hormonal deficiencies, autoimmune disorders, or compression that can lead to neuropathy. Peripheral nerves have the ability to regenerate axons, as long as the nerve cell itself has not died, which may lead to functional recovery over time. Correcting an underlying condition often can result in the neuropathy resolving on its own as the nerves recover or regenerate.

The adoption of healthy lifestyle habits such as maintaining optimal weight, avoiding exposure to toxins, exercising, eating a balanced diet, correcting vitamin deficiencies, and limiting or avoiding alcohol consumption can reduce the effects of peripheral neuropathy. Exercise can reduce cramps, improve muscle strength, and prevent muscle wasting. Various dietary strategies can improve gastrointestinal symptoms. Timely treatment of injuries can help prevent permanent damage. Smoking cessation is particularly important because smoking constricts the blood vessels that supply nutrients to the peripheral nerves and can worsen neuropathic symptoms. Self-care skills such as meticulous foot care and careful wound treatment in people with diabetes and others who have an impaired ability to feel pain can alleviate symptoms and improve quality of life. Such changes often create conditions that encourage nerve regeneration.

Systemic diseases frequently require more complex treatments. Strict control of blood glucose levels has been shown to reduce neuropathic symptoms and help people with diabetic neuropathy avoid further nerve damage.

Inflammatory and autoimmune conditions leading to neuropathy can be controlled in several ways. Immunosuppressive drugs such as prednisone, cyclosporine, or azathioprine may be beneficial. Plasmapheresis — a procedure in which blood is removed, cleansed of immune system cells and antibodies, and then returned to the body — can help reduce inflammation or suppress immune system activity. Large intravenously administered

doses of immunoglobulins (antibodies that alter the immune system, and agents such as rituximab that target specific inflammatory cells) also can suppress abnormal immune system activity.

Symptom Management

Neuropathic pain, or pain caused by the injury to a nerve or nerves, is often difficult to control. Mild pain may sometimes be alleviated by over-the-counter analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs). More chronic and discomforting pain may need to be addressed through the care of a physician. Medications that are used for chronic neuropathic pain fall under several classes of drugs: antidepressants, anticonvulsant medications, antiarrhythmic medications, and narcotic agents. The antidepressant and anticonvulsant medications modulate pain through their mechanism of action on the peripheral nerves, spinal cord, or brain and tend to be the most effective types of medications to control neuropathic pain. Antidepressant medications include tricyclic antidepressants such as amitriptyline or newer serotonin-norepinephrine reuptake inhibitors such as duloxetine hydrochloride or venlafaxine. Anticonvulsant medications that are frequently used include gabapentin, pregabalin, topiramate, and carbamazepine, although other medications used for treating epilepsy may also be useful. Mexiletine is an antiarrhythmic medication that may be used for treatment of chronic painful neuropathies.

For pain that does not respond to the previously described medications, the addition of narcotic agents may be considered. Because the use of prescription-obtained pain relievers that contain opioids can lead to dependence and addiction, their use is recommended only after other means of controlling the pain have failed. One of the newest narcotic medications approved for the treatment of diabetic neuropathy is tapentadol, a drug with both opioid activity and norepinephrine-reuptake inhibition activity of an antidepressant.

Topically administered medications are another option for neuropathic pain. Two agents are topical lidocaine, an anesthetic agent, and capsaicin, a substance found in hot peppers that modifies peripheral pain receptors. Topical agents are generally most appropriate for localized chronic pain such as herpes zoster neuralgia (shingles) pain. Their usefulness for treating diffuse chronic diabetic neuropathy is more limited.

Transcutaneous electrical nerve stimulation (TENS) is a non-invasive intervention used for pain relief in a range of conditions, and a number of studies have described its use for neuropathic pain. The therapy involves attaching electrodes to the skin at the site of pain or near associated nerves and then administering a gentle electrical current. Although data from controlled clinical trials are not available to broadly establish its efficacy for peripheral neuropathies, TENS has been shown in some studies to improve peripheral neuropathy symptoms associated with diabetes.

Other complementary approaches may provide additional support and pain relief. For example, mechanical aids such as hand or foot braces can help reduce pain and physical disability by compensating for muscle weakness or alleviating nerve compression. Orthopedic shoes can improve gait disturbances and help prevent foot injuries in people with a loss of pain sensation. Acupuncture, massage, and herbal medications also are considered in the treatment of neuropathic pain.

Surgical intervention can be considered for some types of neuropathies. Injuries to a single nerve caused by focal compression such as at the carpal tunnel of the wrist, or other entrapment neuropathies, may respond well to surgery that releases the nerve from the tissues compressing it. Some surgical procedures reduce pain by destroying the nerve; this approach is appropriate only for pain caused by a single nerve and when other forms of treatment have failed to provide relief. Peripheral neuropathies that involve more diffuse nerve damage, such as diabetic neuropathy, are not amenable to surgical intervention.

What research is being done?

The mission of the National Institute of Neurological Disorders and Stroke (NINDS) is to seek fundamental knowledge about the brain and nervous system and to use that knowledge to reduce the burden of neurological disease. The NINDS is a component of the National Institutes of Health (NIH), the leading supporter of biomedical research in the world.

NINDS-funded research on neuropathy ranges from clinical studies of the genetics and natural history of hereditary neuropathies to basic science investigations of the biological mechanisms responsible for chronic neuropathic pain. Other efforts are focused on understanding how immune system dysfunction contributes to peripheral nerve damage. Together, these diverse research areas will advance the development of new therapeutic and preventive strategies for peripheral neuropathies.

Specific genetic mutations have been identified for some of the known hereditary neuropathies. NINDS therefore supports studies to identify other genetic defects that may play roles in causing or modifying the course of disease. The Inherited Neuropathies Consortium, focused on Charcot-Marie-Tooth neuropathies, seeks to better characterize the natural history of several different forms and to identify genes that modify clinical features in these disorders. Better knowledge of genetic causes may help identify people who are at high risk for developing peripheral neuropathy before symptoms appear. Understanding the role of genetic mutations may also lead to the development of gene therapies that prevent or reduce cumulative nerve damage. In addition, advances from genetics research inform studies to understand disease mechanisms. For example, scientists are using animal models to study how inflammation and nerve damage result from mutations in the Autoimmune Regulator (AIRE) gene, the cause of chronic inflammatory demyelinating polyneuropathy (CIDP) in some people.

Several NINDS-funded studies aim to determine why nerve axons degenerate in different types of peripheral neuropathies. Rapid communication between the peripheral nervous system and the central nervous system depends on myelination, a process through which special cells called Schwann cells create an insulating sheath around axons. Research has shown that Schwann cells play a critical role in the regeneration of nerve cell axons in the peripheral nervous system. By better understanding myelination and Schwann cell function, researchers hope to find targets for new therapies to treat or prevent nerve damage associated with neuropathy.

One promising area of research focuses on a class of molecules called neurotrophic factors. These substances, produced naturally by the body, protect neurons from injury and enhance their survival. Neurotrophic factors also help maintain normal function in mature nerve cells, and some stimulate axon regeneration. Several NINDS-supported studies seek to learn more about the effects of these powerful chemicals on the peripheral nervous system.

Another area of research aims to better understand inflammatory peripheral neuropathies, such as Guillain-Barre syndrome (GBS), in which the body's immune system attacks peripheral nerves, damaging myelin and impairing signal conduction along affected nerves. NINDS-funded researchers are investigating the mechanisms by which the body's immune system stops recognizing peripheral nerves as "self" and starts

attacking them. GBS is usually preceded by a microbial infection, some as common as food poisoning or the flu, and researchers hypothesize that antibodies generated by the immune system to fight bacteria also attack nervous system proteins. Studies to test this hypothesis may lead to treatments that prevent these antibodies from damaging nerves. As a different strategy, researchers are studying the blood-nerve barrier in inflammatory nervous system disorders and developing ways to reduce the movement of immune cells from the bloodstream into nerve tissue, which may reduce inflammation, demyelination and nerve injury.

Transcranial magnetic stimulation (TMS), which uses a coil either held above or placed on the scalp that delivers electromagnetic pulses to activate electrical currents in general or specific parts of the brain, has shown some analgesic effect in treating various pain conditions. Current studies are examining the effectiveness of TMS in treating peripheral and chronic neuropathies.

In addition to efforts to treat or prevent underlying nerve damage, other NINDS-supported studies are informing new strategies for relieving neuropathic pain. Researchers are investigating the pathways that carry pain signals to the brain and are working to identify substances that will block this signaling.

Where can I get more information?

For more information on neurological disorders or research programs funded by the National Institute of Neurological Disorders and Stroke, contact the Institute's Brain Resources and Information Network (BRAIN) at:

BRAIN

P.O. Box 5801

Bethesda, MD 20824

800-352-9424

<http://www.ninds.nih.gov>

Information also is available from the following organizations:

American Chronic Pain Association (ACPA)

P.O. Box 850

Rocklin, CA 95677-0850

916-632-0922

800-533-3231

<http://www.theacpa.org>

The Neuropathy Association, Inc.

110 W. 40th Street, Suite 1804

New York, NY 10018

888-PN-FACTS (888-763-2287)

<http://www.neuropathy.org>

National Kidney & Urologic Diseases Information Clearinghouse (NKUDIC)

3 Information Way

Bethesda, MD 20892-3580

301-654-4415

800-891-5390

<http://www.niddk.nih.gov>

Charcot-Marie-Tooth Association (CMTA)

2700 Chestnut Parkway

Chester, PA 19013-4867

610-499-9264

800-606-CMTA (2682)

<http://www.cmtausa.org>

Muscular Dystrophy Association

3300 East Sunrise Drive

Tucson, AZ 85718-3208

520-529-2000

800-572-1717

<http://www.mda.org>

American Diabetes Association

1701 North Beauregard Street

Alexandria, VA 22311

800-DIABETES (342-2383)

703-549-1500

<http://www.diabetes.org>

National Diabetes Information

Clearinghouse (NDIC)

1 Information Way

Bethesda, MD 20892-3560

301-654-3327

800-860-8747

<http://www.diabetes.niddk.nih.gov>

Foundation for Peripheral Neuropathy

485 Half Day Road, Suite 200

Buffalo Grove, IL 60089

877-883-9942

<http://www.foundationforpn.org>





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