Cerebral palsy

Spastic cerebral palsy is characterized by hyperactive deep tendon reflexes, increased stretch reflexes, rapid alternating muscle contraction and relaxation, muscle weakness, underdevelopment of affected limbs, muscle contraction in response to manipulation, and a tendency to contractures. Typically, a child with spastic CP walks on his toes with a scissors gait, crossing one foot in front of the other. In athetoid cerebral palsy, involuntary movements—grimacing, wormlike writhing, dystonia, and sharp jerks—impair voluntary movement. Usually, these involuntary movements affect the arms more severely than the legs; involuntary facial movements may make speech difficult. These athetoid movements become more severe during stress, decrease with relaxation, and disappear entirely during sleep. Ataxic cerebral palsy is characterized by disturbed balance, incoordination (especially of the arms), hypoactive reflexes, nystagmus, muscle weakness, tremor, lack of leg movement during infancy, and a widebased gait as the child begins to walk. Ataxia makes sudden or fine movements almost impossible. Some children with CP display a combination of these clinical features. In most, impaired motor function makes eating (especially swallowing) difficult and retards growth and development. Up to 40% of these children are mentally retarded, about 25% have seizure disorders, and about 80% have impaired speech. Many also have dental abnormalities, vision and hearing defects, and reading disabilities.

Hydrocephalus

In infants, the unmistakable sign of hydrocephalus is rapidly increasing head circumference, clearly disproportionate to the infant's growth. Other characteristic changes include widening and bulging of the fontanels; distended scalp veins; thin, shiny, and fragile-looking scalp skin; and underdeveloped neck muscles. (See Signs of hydrocephalus.) In severe hydrocephalus, the roof of the orbit is depressed, the eyes are displaced downward, and the sclerae are prominent. Sclera seen above the iris is called the “setting-sun sign.” A high-pitched, shrill cry, abnormal muscle tone of the legs, irritability, anorexia, and projectile vomiting commonly occur. In adults and older children, indicators of hydrocephalus include decreased level of consciousness (LOC), ataxia, incontinence, loss of coordination, and impaired intellect.

Cerebral aneurysm

Occasionally, rupture of a cerebral aneurysm causes premonitory symptoms that last several days, such as headache, nuchal rigidity, stiff back and legs, and intermittent nausea. Usually, however, onset is abrupt and without warning, causing a sudden severe headache, nausea, vomiting and, depending on the severity and location of bleeding, altered consciousness (including deep coma). Bleeding causes meningeal irritation, resulting in nuchal rigidity, back and leg pain, fever, restlessness, irritability, occasional seizures, and blurred vision. Bleeding into the brain tissues causes hemiparesis, hemisensory defects, dysphagia, and visual defects. If the aneurysm is near the internal carotid artery, it compresses the oculomotor nerve and causes diplopia, ptosis, dilated pupil, and inability to rotate the eye. The severity of symptoms varies considerably from patient to patient, depending on the site and amount of bleeding. To better describe their conditions, patients with ruptured cerebral aneurysms are grouped as follows:

Grade I (minimal bleed): Patient is alert with no neurologic deficit; he may have a slight headache and

nuchal rigidity. Grade II (mild bleed): Patient is alert, with a mild to severe headache, nuchal rigidity and, possibly, third-nerve palsy. Grade III (moderate bleed): Patient is confused or drowsy, with nuchal rigidity and, possibly, a mild focal deficit. Grade IV (severe bleed): Patient is stuporous, with nuchal rigidity and, possibly, mild to severe hemiparesis. Grade V (moribund; commonly fatal): If nonfatal, patient is in deep coma or decerebrate. Generally, cerebral aneurysm poses three major threats: Death from increased ICP: Increased ICP may push the brain downward, impair brain stem function, and cut off blood supply to the part of the brain that supports vital functions. Rebleed: Generally, after the initial bleeding episode, a clot forms and seals the rupture, which reinforces the wall of the aneurysm for 7 to 10 days. However, after the 7th day, fibrinolysis begins to dissolve the clot and increases the risk of rebleeding. Signs and symptoms are similar to those accompanying the initial hemorrhage. Rebleeds during the first 24 hours after initial hemorrhage aren't uncommon, and they contribute to cerebral aneurysm's high mortality. Vasospasm: Why this occurs isn't clearly understood. Usually, vasospasm occurs in blood vessels adjacent to the cerebral aneurysm, but it may extend to major vessels of the brain, causing ischemia and altered brain function. Other complications of cerebral aneurysm include pulmonary embolism (a possible adverse effect of deep vein thrombosis or aneurysm treatment) and acute hydrocephalus, occurring as CSF accumulates in the cranial cavity because of blockage by blood or adhesions.

Arteriovenous malformations

An AVM may be asymptomatic until complications occur; these may include rupture and a resulting sudden bleed in the brain, known as a hemorrhagic stroke. Arteriovenous malformations vary in size and location within the brain. Systolic bruit may be auscultated over the carotid artery, mastoid process, or orbit on examination. Symptoms that occur prior to an AVM rupture are related to smaller and slower bleeding from the abnormal vessels, which are usually fragile because their structure is abnormal.

In more than half of patients with AVM, hemorrhage from the malformation is the first symptom. Depending on the location and the severity of the bleed, the hemorrhage can be profoundly disabling or fatal. The risk of bleeding from an AVM is approximately 2% to 4% per year. The first symptoms often include headache, seizure, or other sudden neurological problems, such as vision problems, weakness, inability to move a limb or a side of the body, lack of sensation in part of the body, or abnormal sensations, such as ringing and numbness. Symptoms are the same as for stroke. The individual with an AVM may complain of chronic mild headache, a sudden and severe headache, or a localized or general headache. The headache may resemble migraine and vomiting may occur. Seizures may result from focal neurologic deficits (depending on the location of the AVM) resulting from compression and diminished perfusion. Symptoms of intracranial (intracerebral, subarachnoid, or subdural) hemorrhage result. Muscle weakness and decreased sensation can occur in any part of the body. Mental status change can occur where the individual appears sleepy, stuporous, lethargic, confused, disoriented, or irritable. Additional symptoms may include stiff neck, speech or sense of smell impairment, dysfunctional movement, fainting, facial paralysis, eyelid drooping, tinnitus, dizziness, and decreased level of consciousness (LOC). If an AVM bleeds once, the risk is greater that it will bleed again in the future. Intracerebral or subarachnoid hemorrhages are the most common first symptoms of cerebral arteriovenous malformation. In some cases, symptoms may also occur due to lack of blood flow to an area of the brain (ischemia), compression or distortion of brain tissue by large AVMs, or abnormal brain

development in the area of the malformation. Progressive loss of nerve cells in the brain may occur, caused by mechanical (pressure) and ischemic (lack of blood supply) factors.

Headache

Initially, migraine headaches usually produce unilateral, pulsating pain, which later becomes more generalized. They're commonly preceded by a scintillating scotoma, hemianopsia, unilateral paresthesia, or speech disorders. The patient may experience irritability, anorexia, nausea, vomiting, and photophobia. (See Clinical features of migraine headaches.) Both muscle contraction and tractioninflammatory vascular headaches produce a dull, persistent ache, tender spots on the head and neck, and a feeling of tightness around the head, with a characteristic “hatband” distribution. The pain is usually severe and unrelenting. If caused by intracranial bleeding, these headaches may result in neurologic deficits, such as paresthesia and muscle weakness; narcotics may fail to relieve pain in these cases. If caused by a tumor, pain is most severe when the patient awakens.

Seizure disorder

The hallmarks of seizure disorder are recurring seizures, which can be classified as partial or generalized (some patients may be affected by more than one type). Partial seizures arise from a localized area of the brain, causing specific symptoms. In some patients, partial seizure activity may spread to the entire brain, causing a generalized seizure. Partial seizures include simple partial (jacksonian) and complex partial seizures (psychomotor or temporal lobe). A simple partial motor-type seizure begins as a localized motor seizure characterized by a spread of abnormal activity to adjacent areas of the brain. It typically produces stiffening or jerking in one extremity, accompanied by a tingling sensation in the same area. For example, it may start in the thumb and spread to the entire hand and arm. The

patient seldom loses consciousness, although the seizure may progress to a generalized seizure. A simple partial sensory-type seizure involves perceptual distortion, which can include hallucinations. The symptoms of a complex partial seizure vary but usually include purposeless behavior. The patient experiences an aura immediately before the seizure. An aura represents the beginning of abnormal electrical discharges within a focal area of the brain and may include a pungent smell, GI distress (nausea or indigestion), a rising or sinking feeling in the stomach, a dreamy feeling, an unusual taste, or a visual disturbance. Overt signs of a complex partial seizure include a glassy stare, picking at one's clothes, aimless wandering, lipsmacking or chewing motions, and unintelligible speech; these signs may last for just a few seconds or as long as 20 minutes. Mental confusion may last several minutes after the seizure; as a result, an observer may mistakenly suspect intoxication with alcohol or drugs or psychosis. Generalized seizures, as the term suggests, cause a generalized electrical abnormality within the brain and include several distinct types: Absence (petit mal) seizures occur most commonly in children, although they may affect adults as well. They usually begin with a brief change in level of consciousness, indicated by blinking or rolling of the eyes, a blank stare, and slight mouth movements. There's little or no tonic-clonic movement. The patient retains his posture and continues preseizure activity without difficulty. Typically, each seizure lasts from 1 to 10 seconds. If not properly treated, seizures can recur as often as 100 times per day. An absence seizure may progress to generalized tonic-clonic seizures. A myoclonic (bilateral massive epileptic myoclonus) seizure is characterized by brief, involuntary muscular

jerks of the body or extremities, which may occur in a rhythmic fashion and may precede generalized tonic-clonic seizures by months or years. A generalized tonic-clonic (grand mal) seizure typically begins with a loud cry, precipitated by air rushing from the lungs through the vocal cords. The patient then falls to the ground, losing consciousness. The body stiffens (tonic phase) and then alternates between episodes of muscular spasm and relaxation (clonic phase). Tongue-biting, incontinence, labored breathing, apnea, and subsequent cyanosis may also occur. The seizure stops in 2 to 5 minutes, when abnormal electrical conduction of the neurons is completed. The patient then regains consciousness but is somewhat confused and may have difficulty talking. If he can talk, he may complain of drowsiness, fatigue, headache, muscle soreness, and arm or leg weakness. He may fall into deep sleep after the seizure. These seizures may start as facial seizures and spread to become generalized. An akinetic seizure is characterized by a general loss of postural tone (the patient falls in a flaccid state) and a temporary loss of consciousness. It occurs in young children and is sometimes called a “drop attack” because it causes the child to fall. Status epilepticus is a continuous seizure state that can occur in all seizure types. The most life-threatening example is generalized tonicclonic status epilepticus, a continuous generalized tonic-clonic seizure without intervening return of consciousness. Status epilepticus is accompanied by respiratory distress. It can result from abrupt withdrawal of anticonvulsant medications, hypoxic encephalopathy, acute head trauma, metabolic encephalopathy, or septicemia secondary to encephalitis or meningitis.

Stroke

Clinical features of stroke vary with the artery affected (and, consequently, the portion of the brain it supplies), the severity of damage, and the extent of collateral circulation that develops to help the brain compensate for decreased blood supply. If the stroke occurs in the left hemisphere, it produces symptoms on the right side; if in the right hemisphere, symptoms are on the left side. However, a stroke that causes cranial nerve damage produces signs of cranial nerve dysfunction on the same side as the hemorrhage. Symptoms are usually classified according to the artery affected: Middle cerebral artery: aphasia, dysphasia, visual field cuts, and hemiparesis on affected side (more severe in the face and arm than in the leg) Carotid artery: weakness, paralysis, numbness, sensory changes, and visual disturbances on affected side; altered level of consciousness (LOC), bruits, headaches, aphasia, and ptosis Vertebrobasilar artery: weakness on affected side, numbness around lips and mouth, visual field cuts, diplopia, poor coordination, dysphagia, slurred speech, dizziness, amnesia, and ataxia Anterior cerebral artery: confusion, weakness, and numbness (especially in the leg) on affected side, incontinence, loss of coordination, impaired motor and sensory functions, and personality changes Posterior cerebral arteries: visual field cuts, sensory impairment, dyslexia, coma, and cortical blindness; typically, paralysis is absent.

Symptoms can also be classified as premonitory, generalized, and focal. Premonitory symptoms, such as drowsiness, dizziness, headache, and mental confusion, are rare. Generalized symptoms, such as headache, vomiting, mental impairment, seizures, coma, nuchal rigidity, fever, and disorientation, are typical. Focal symptoms, such as sensory and reflex changes, reflect the site of hemorrhage or infarct and may worsen.

Meningitis

The cardinal signs of meningitis are infection (fever, chills, and malaise) and increased intracranial pressure (ICP; headache, vomiting and, rarely, papilledema). Signs of positive Brudzinski's and Kernig's signs, exaggerated and symmetrical deep tendon reflexes, and opisthotonos (a spasm in which the back and extremities arch backward so that the body rests on the head and heels). (See Two signs of meningitis, pages 198 and 199.) Other manifestations of meningitis are irritability; sinus arrhythmias; photophobia, diplopia, and other visual problems; and delirium, deep stupor, and coma. An infant may not show clinical signs of infection but may be fretful and refuse to eat. Such an infant may vomit often, leading to dehydration; this prevents a bulging fontanel and thus masks this important sign of increased ICP. As the illness progresses, twitching, seizures (in 30% of infants), or coma may develop. Most older children have the same symptoms as adults. In subacute meningitis, the onset may be insidious.

Encephalitis

All viral forms of encephalitis have similar clinical features, although certain differences do occur. Usually, the acute illness begins with sudden onset of fever, headache, and vomiting and progresses to include signs and symptoms of meningeal irritation (stiff neck and back) and neuronal damage (drowsiness, coma, paralysis, seizures, ataxia, tremors, nausea, vomiting, and organic psychoses). After the acute phase of the illness, coma may persist for days or weeks. The severity of arbovirus encephalitis may range from subclinical to rapidly fatal necrotizing disease. Herpes encephalitis also produces signs and symptoms that vary from subclinical to acute and commonly fatal fulminating disease. Associated effects include disturbances of taste or smell.

Brain abscess

Onset varies according to cause, but generally brain abscess produces clinical effects similar to those of a brain tumor. Early symptoms result from increased intracranial pressure (ICP) and include constant intractable headache, worsened by straining; nausea; vomiting; and focal or generalized seizures. Typical later symptoms include ocular disturbances, such as nystagmus, decreased vision, and unequal pupil size. Other features differ with the site of the abscess: temporal lobe abscess: auditory-receptive dysphasia, central facial weakness, and hemiparesis cerebellar abscess: dizziness, coarse nystagmus, gaze weakness on lesion side, tremor, and ataxia frontal lobe abscess: expressive dysphasia, hemiparesis with unilateral motor seizure, drowsiness, inattention, and mental function impairment. Signs of infection, such as fever, pallor, and bradycardia, are absent until late stages unless they result from the predisposing condition. If the abscess is encapsulated, they may never appear. Depending on abscess size and location, level of consciousness (LOC) varies from drowsiness to deep stupor.

Huntington's disease

Onset is insidious. The patient eventually becomes totally dependent— emotionally and physically—through loss of musculoskeletal control, and he develops progressively severe choreic movements. Such movements are rapid, usually violent, and purposeless. Initially, they're unilateral and more prominent in the face and arms than in the legs, progressing from mild fidgeting to grimacing, tongue smacking, dysarthria (indistinct speech), athetoid movements (especially of the hands) related to emotional state, and torticollis. Ultimately, the patient with Huntington's disease develops progressive

dementia, although the dementia doesn't always progress at the same rate as the chorea. Dementia can be mild at first, but eventually causes severe disruption of the personality. Personality changes include obstinacy, carelessness, untidiness, moodiness, apathy, inappropriate behavior, loss of memory and concentration and, occasionally, paranoia.

Parkinson's disease

The cardinal symptoms of Parkinson's disease are muscle rigidity and akinesia and an insidious resting tremor that begins in the fingers (unilateral pill-roll tremor), increases during stress or anxiety, and decreases with purposeful movement and sleep. Muscle rigidity results in resistance to passive muscle stretching, which may be uniform (leadpipe rigidity) or jerky (cogwheel rigidity). Akinesia causes the patient to walk with difficulty (gait lacks normal parallel motion and may be retropulsive or propulsive) and produces a high-pitched, monotone voice; drooling; a masklike facial expression; loss of posture control (the patient walks with body bent forward); and dysarthria, dysphagia, or both. Occasionally, akinesia may also cause oculogyric crises (eyes are fixed upward, with involuntary tonic movements) or blepharospasm (eyelids are completely closed). Parkinson's disease itself doesn't impair the intellect, but a coexisting disorder, such as arteriosclerosis, may do so.

Myelitis and acute transverse myelitis

In acute transverse myelitis, onset is rapid, with motor and sensory dysfunctions below the level of spinal cord damage appearing in 1 to 2 days. Patients with acute transverse myelitis develop flaccid paralysis of the legs (sometimes beginning in just one leg) with loss of sensory and sphincter functions. Such sensory loss may follow pain in the legs or trunk. Reflexes disappear in the early stages but may reappear later.

The extent of damage depends on the level of the spinal cord affected; transverse myelitis seldom involves the arms. If spinal cord damage is severe, it may cause shock (hypotension and hypothermia).

Alzheimer's disease

Onset is insidious. Initially, the patient undergoes almost imperceptible changes, such as forgetfulness, recent memory loss, difficulty learning and remembering new information, deterioration in personal hygiene and appearance, and an inability to concentrate. Gradually, tasks that require abstract thinking and activities that require judgment become more difficult. Progressive difficulty in communication and severe deterioration in memory, language, and motor function result in a loss of coordination and an inability to write or speak. Personality changes (restlessness, irritability) and nocturnal awakenings are common. Patients also exhibit loss of eye contact, a fearful look, wringing of the hands, and other signs of anxiety. When a patient with Alzheimer's disease is overwhelmed with anxiety, he becomes dysfunctional, acutely confused, agitated, compulsive, or fearful. Eventually, the patient becomes disoriented, and emotional lability and physical and intellectual disability progress. The patient becomes susceptible to infection and accidents. Usually, death results from infection.

Creutzfeldt-Jakob disease

Early signs and symptoms of mental impairment may include slowness in thinking, difficulty

concentrating, impaired judgment, and memory loss. Dementia is progressive and occurs early. Involuntary movements, such as muscle twitching, trembling, and peculiar body movements, and visual disturbances, appear with disease progression and advancing mental deterioration. Hallucinations are also common. Duration of the typical illness is 4 months.

Reye's syndrome

The severity of the child's signs and symptoms varies with the degree of encephalopathy and cerebral edema. In any case, Reye's syndrome develops in five stages. After the initial viral infection, a brief recovery period follows when the child doesn't seem seriously ill.

A few days later, he develops intractable vomiting; lethargy; rapidly changing mental status (mild to severe agitation, confusion, irritability, and delirium); rising blood pressure, respiratory rate, and pulse rate; and hyperactive reflexes. Reye's syndrome commonly progresses to coma. As coma deepens, seizures develop, followed by decreased tendon reflexes and, usually, respiratory failure. Increased ICP, a serious complication, is now considered the result of an increased cerebral blood volume causing intracranial hypertension. Such swelling may develop as a result of acidosis, increased cerebral metabolic rate, and an impaired autoregulatory mechanism.

Guillain-Barré syndrome

About 50% of patients with Guillain-Barré syndrome have a history of minor febrile illness (10 to 14 days before onset), usually an upper respiratory tract infection or, less commonly, gastroenteritis with Camphylobacter jejuni. When infection precedes onset of Guillain-Barré syndrome, signs of infection subside before neurologic features appear. Other possible precipitating factors include surgery, rabies or swine influenza vaccination, viral illness like Epstein-Barr virus, cytomegalovirus, hepatitis, and HIV, Hodgkin's or other malignant disease, and lupus erythematosus. Symmetrical muscle weakness, the major neurologic sign, usually appears in the legs first (ascending type) and then extends to the arms and facial nerves in 24 to 72 hours. Sometimes, muscle weakness develops in the arms first (descending type) or in the arms and legs simultaneously. (See Testing for thoracic sensation.) In milder forms of this disease, muscle weakness may affect only the cranial nerves or may not occur at all. Another common neurologic sign is paresthesia, which sometimes precedes muscle weakness but tends to vanish quickly. However, some patients with this disorder never develop this symptom. Other clinical features may include facial diplegia (possibly with ophthalmoplegia), dysphagia or dysarthria and, less commonly, weakness of the muscles supplied by cranial nerve XI. Muscle weakness develops so quickly that muscle atrophy doesn't occur, but hypotonia and areflexia do. Stiffness and pain in the form of a severe “charley horse” commonly occur.

Myasthenia gravis

The dominant symptoms of myasthenia gravis are skeletal muscle weakness and fatigability. In the early stages, easy fatigability of certain muscles may appear with no other findings. Later, it may be severe enough to cause paralysis. Typically, myasthenic muscles are strongest in the morning but weaken throughout the day, especially after exercise. Short rest periods temporarily restore muscle function.

Muscle weakness is progressive; more and more muscles become weak, and eventually some muscles may lose function entirely. Resulting symptoms depend on the muscle group affected; they become more intense during menses and after emotional stress, prolonged exposure to sunlight or cold, or infections.

Amyotrophic lateral sclerosis

Progressive loss of muscle strength and coordination eventually interfere with everyday activities. Patients with ALS develop fasciculations, accompanied by atrophy and weakness, especially in the muscles of the feet and the hands. Other signs include impaired speech; difficulty chewing, swallowing, and breathing and, occasionally, choking and excessive drooling. Mental deterioration doesn't occur, but patients may become depressed as a reaction to the disease.

Multiple sclerosis

Clinical findings in MS depend on the extent and site of myelin destruction, the extent of remyelination, and the adequacy of subsequent restored synaptic transmission. Signs and symptoms in MS may be transient, or they may last for hours or weeks. They may wax and wane with no predictable pattern, vary from day to day, and be bizarre and difficult for the patient to describe. In most patients, visual problems and sensory impairment, such as numbness and tingling sensations (paresthesia), are the first signs that something may be wrong. Other characteristic changes include: ocular disturbances—optic neuritis, diplopia, ophthalmoplegia, blurred vision, and nystagmus muscle dysfunction—weakness, paralysis ranging from monoplegia to quadriplegia, spasticity, hyperreflexia, intention tremor, and gait ataxia urinary disturbances—incontinence, frequency, urgency, and frequent infections emotional lability—characteristic mood swings, irritability, euphoria, and depression. Associated signs and symptoms include poorly articulated or scanning speech and dysphagia. Clinical effects may be so mild that the patient is unaware of them or so bizarre that he appears hysterical.

Trigeminal neuralgia

Typically, the patient reports a searing or burning pain that occurs in lightninglike jabs and lasts from 1 to 15 minutes (usually 1 to 2 minutes) in an area innervated by one of the divisions of the trigeminal nerve, primarily the superior mandibular or maxillary division. The pain rarely affects more than one division and seldom the first division (ophthalmic) or both sides of the face. It affects the second (maxillary) and third (mandibular) divisions of the trigeminal nerve equally.

Bell's palsy

Bell's palsy usually produces unilateral facial weakness, occasionally with aching pain around the angle of the jaw or behind the ear. On the weak side, the mouth droops (causing the patient to drool saliva from the corner of his mouth), and taste perception is distorted over the affected anterior portion of the tongue. The forehead appears smooth, and the patient's ability to close his eye on the weak side is markedly impaired. When he tries to close this eye, it rolls upward (Bell's phenomenon) and shows excessive tearing. Although Bell's phenomenon occurs in normal people, it isn't apparent because the

eyelids close completely and cover this eye motion. In Bell's palsy, incomplete eye closure makes this upward motion obvious. Other symptoms may include loss of taste and ringing in the ear.

Peripheral neuritis

The clinical effects of peripheral neuritis develop slowly, and the disease usually affects the motor and sensory nerve fibers. Symptoms vary according to which type of nerve is affected (sensory, motor, or autonomic). Neuropathy can affect any one or be a combination of all three types. Sensory changes: Damage to sensory fibers results in changes in sensation, ranging from abnormal sensations, such as burning, nerve pain, or tingling, to numbness or an inability to determine joint position in the area. Sensation changes often begin in the feet and progress toward the center of the body with involvement of other areas as the condition worsens. Motor changes: Damage to the motor fibers interferes with muscle control and can cause weakness, loss of muscle bulk, and loss of dexterity. Muscle cramping may be a sign of motor nerve involvement. Other muscle-related symptoms include lack of muscle control, difficulty or inability to move a part of the body (paralysis), muscle atrophy, muscle twitching (fasciculation) or cramping, difficulty breathing or swallowing, falling (from legs buckling or tripping over toes), or lack of dexterity (such as the inability to button a shirt). Autonomic changes: The autonomic nerves control involuntary or semivoluntary functions, such as control of internal organs and blood pressure. Damage to autonomic nerves can cause blurred vision, decreased ability to sweat (anhidrosis), dizziness that occurs when standing up or fainting associated with a fall in blood pressure, heat intolerance with exertion (decreased ability to regulate body temperature), nausea or vomiting after meals, abdominal bloating (swelling), feeling full after eating a small amount (early satiety), diarrhea, constipation, unintentional weight loss (more than 5% of body weight), urinary incontinence, feeling of incomplete bladder emptying, difficulty beginning to urinate (urinary hesitancy), and male impotence.

Complex regional pain syndrome

Patients usually report severe and constant pain; severe pain is common with CRPS2 in particular. The affected area may have altered blood flow, feeling either warm or cool to the touch, with discoloration, sweating, or swelling. In time, skin, hair, and nail changes may occur along with impaired mobility and muscle wasting, especially if adequate treatment is delayed.