Neurologic Examination in Rehabilitation

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ABSTRACT

The focus of the neurologic examination of the patient with rehabilitation needs is on determining deficits that impair functional recovery and identifying a patient's ability to carry out certain tasks. This article discusses the ''team concept'' of rehabilitation and the roles of each of the team members, including the rehabilitation physician. The article also reviews the aspects of the neurologic examination as they specifically relate to the patient undergoing rehabilitation.

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THE REHABILITATION TEAM

Rehabilitation may be defined as the coordinated use of medical, social, educational, technologic, and vocational measures to bring a person to the highest functional level possible. Development of an active rehabilitation program depends on an understanding of the principles of learning theory (ie, what methods achieve optimal learning in specific conditions) and on the recognition of the influence of rehabilitation on intrinsic and adaptive recovery (ie, changes in performance produced by specific training). Such a program combines active and substitutive techniques while incorporating assistive devices and technology into an individualized rehabilitation scheme. A coordinated interdisciplinary team is essential to the success of such programs.

The physician who specializes in rehabilitation combines the medical management of a disease or injury, including comorbidities, with the management of the functional effects of resulting impairments on the life of the person and family. Most physicians have been trained extensively in the medical model and focus on addressing the signs and symptoms of a disease. In contrast, the rehabilitation physician receives training and experience in the functional model and is thus prepared to address the effects that impairments have on the person's ability to perform specific activities and to fulfill roles in society.

Although the medical model may be appropriate for acute episodic illness, it does not effectively deal with the chronic and complex problems resulting from neurologic disease or injury. For example, the physician may focus on treating spasticity simply because it is present, while the patient is concerned with being able to walk. As a member or leader of the interdisciplinary rehabilitation team, the physician must acknowledge the skills and competencies of the other rehabilitation professionals while remaining able to assess the effects of their proposed treatments on the patient's overall level of function. To do so requires that the physician have broad-based knowledge of the expertise and roles of each discipline and the techniques that they use. Facilitating the team process requires excellent communication, interpersonal, and organizational skills.

KEY POINTS

- Rehabilitation combines active and substitutive techniques and incorporates assistive devices into an individualized program.
- A coordinated interdisciplinary team is essential to a successful rehabilitation program.
- The rehabilitation physician moves beyond the medical model to coordinate a team approach to care.

The rehabilitation team generally follows a series of steps to ensure the accomplishment of rehabilitation goals (**Table 2-1**). Each team member actively participates in the goal-setting process, which should combine the recommendations of the team with the interests, values, and lifestyle of the patient and family. Goals are prioritized based on their importance to the patient and family, feasibility, degree of health risk, and the initial steps needed to reach larger overall goals. Rehabilitation goals should be realistic, achievable, and measurable to allow for accurate documentation of progress and outcome.

TABLE 2-1 Rehabilitation Team Process

▶ **Evaluate**
Impairments
Comorbidities
Functional abilities
Premorbid, baseline, and treatment response
▶ **Identify**
Resources
Interests
Values
Lifestyle
Support system
▶ **Set Goals**
Prioritize
Reset
Adjust
▶ **Treat**
Facilitate recovery
Adapt
Compensate
Problem solve
Train
Educate
Motivate
▶ **Follow up**
Monitor patient status
Problem solve
Consult
Coordinate care

Regardless of specialty, all team members work on broad interdisciplinary goals such as orientation, communication, mobility, and behavior. Response to treatment and progress toward goals are reviewed, updated, and modified by the team as the patient's medical and functional status change. These goals are also regularly reviewed with the patient and family, and their input is solicited, with all conversations clearly documented in the patient record.

Rehabilitation programs operate in a variety of settings, including inpatient facilities, outpatient clinics, homes, schools, and the workplace. Each of these programs must develop written admission criteria that define the characteristics of the patients to be accepted. Policies and procedures must also be developed that define the program's mission and organizational structure, govern specific treatments, provide for a safe environment, review credentials of providers, and establish a pattern of review of program quality through outcome evaluation and follow-up.

The rehabilitation of patients with neurologic disorders, including stroke, traumatic brain injury (TBI), and spinal cord injury, involves a coordinated team effort involving a number of disciplines. This team often includes a rehabilitation physician; other physician specialists; physical, occupational, speech, and therapeutic recreation therapists; a neuropsychologist; a prosthetist/orthotist;

and a social worker and/or case manager. Other services that may be offered include pastoral care, vocational rehabilitation, and substance abuse counseling (**Table 2-2**).

The neurologist is often involved in caring for patients in a rehabilitation setting, either as a consultant or as the primary physician, depending on his or her training and the availability of physiatry services in the community. For inpatient acute rehabilitation programs, current Medicare guidelines require the admitting physician to be trained in rehabilitation and to be the coordinator of care on the rehabilitation unit. The rehabilitation physician is responsible for directing medical care,

TABLE 2-2 Members of the Inpatient Rehabilitation Team and Their Roles

Team Member	Role
Rehabilitation physician	Provides consultation to other providers regarding rehabilitation treatments
	Assesses patients for rehabilitation needs
	Directs and coordinates the rehabilitation program and medical care with appropriate goal setting and documentation of progress toward goals
Other physician specialists	Provide specialty care to patients requiring these services (eg, internal medicine, urology, psychiatry)
Rehabilitation nurses	Provide primary nursing care focusing on bowel, bladder, and skin issues
	Further rehabilitation goals by working on bathing, dressing, toileting, eating, transfers, and ambulation
Physical therapist	Addresses recovery of the lower extremity and mobility skills, including bed mobility, transfers, wheelchair propulsion, and ambulation
	May apply serial casting modalities, taping, and other conservative approaches to reduce tone and contractures
	Provides assessment for wheelchair and orthotic prescription
Occupational therapist	Addresses recovery of the upper extremity and activities of daily living, including bathing, dressing, feeding, grooming, and homemaking skills; constraint-induced therapy
	Provides assessment for orthotic prescription
Speech-language pathologist	Addresses and treats swallowing, speech and language, and cognitive dysfunction
Recreational therapist	Works with patients on leisure activities and community reentry skills
Neuropsychologist	Assesses cognitive abilities and establishes behavioral modification programs
	Assesses for and assists in treatment of depression and other psychological disorders
	Works with patients and families on coping with acute and chronic disability
Orthotist	Designs and fabricates orthotic devices to improve patient positioning, comfort, and function
Social worker/case manager	Addresses discharge planning, ordering of equipment, and disability issues
	Deals with third-party payers regarding coverage for rehabilitation services
	Sets up ongoing therapy and home care and coordinates services

determining goals for the rehabilitation stay, and documenting progress toward the stated goals. The rehabilitation physician is required to participate in team and family meetings. Other physicians, including internists or hospitalists, urologists, pulmonologists, cardiologists, orthopedists, and psychiatrists, often provide consultant care during patients' rehabilitation stays.

Physical therapists primarily address lower extremity strength and range of motion as well as balance and apraxia. They seek to improve function including mobility skills, transfers, wheelchair propulsion, and ambulation, if appropriate. Physical therapists may use specific therapy approaches based on the amount of motor recovery and motor learning. Wheelchair seating and design are critical to facilitating optimal mobility and preventing poor posture, injury, or skin breakdown. Physical therapists are also knowledgeable about the impact that visual-perceptual disorders, neglect, and apraxia have on mobility skills and will work with other members of the team to develop a regimen aimed at either correcting or circumventing problems. They will often work with the orthotist to prescribe appropriate splinting/bracing of the knee and/or ankle.

Occupational therapists address upper extremity strength, range of motion, and pain as well as activities of daily living (ADLs) such as bathing, dressing, feeding, and grooming. Practical cognitive skills (eg, making a reservation or grocery list, paying bills) and domestic activities, including cooking and cleaning, may also be addressed for appropriate individuals. The ability of patients to perform these activities safely is key, and the occupational therapist must take into account language, cognitive, and visual-perceptual issues.

Speech-language pathologists treat problems with speech and language, cognition, and swallowing. They often work in concert with dietitians to provide a diet that is nutritionally optimal and of a consistency that minimizes the risk of aspiration pneumonia, one of the more common medical complications in a rehabilitation unit. Speech-language pathologists work closely with neuropsychologists in cognitive rehabilitation. They may also assess a patient's ability to use augmentative communication devices as well as design a simple device and train the patient.

Neuropsychologists assess cognition and develop cognitive retraining strategies that other team members can implement. Identifying what strategies might facilitate new learning is critical to individual program design. For patients with brain injuries, neuropsychologists often develop an appropriate behavioral modification program. They also counsel patients and families in coping and adjusting to functional impairments. In the outpatient setting, detailed neuropsychological testing is used to give prognosis and to guide medical management.

Therapeutic recreation specialists serve a critical role in guiding patients in leisure and community activities that are safe and appropriate for them given their neurologic deficits. They design activities that further patients' functional goals. At many institutions, recreation specialists host group activities in the evening. After discharge they can help patients access activities in the community and provide information on adaptations that allow participation in a wide range of activities (eg, bowling, skiing, fishing, and hunting).

A social worker and/or case manager works with the rehabilitation physician during the preadmission phase, assembling information, gathering records, touring the facility with family, and obtaining insurance approval. Social workers and/or case managers also work

with patients and families on discharge planning, order and arrange delivery of adaptive equipment for home, address work disability issues, and coordinate postdischarge therapies and home care, if needed.

THE NEUROLOGIC EXAMINATION WITH A FOCUS ON FUNCTION

The focus of the neurologic examination is different for a patient needing rehabilitation compared to a typical patient seen on an inpatient neurology service or in an office setting. For the latter, the primary purpose of the examination is to localize the site of the lesion and help guide the appropriate work-up to determine the diagnosis and most appropriate treatment for that condition. In contrast, the focus of the examination for the patient needing rehabilitation is on determining the physical, neurologic, cognitive, and behavioral deficits that will impair a patient's functional recovery, as well as identifying his or her ability to carry out specific tasks. The rehabilitation physician must go beyond neurologic impairment and assess the impact that other diseases such as arthritis, chronic obstructive pulmonary disease, and cardiovascular disease may have on the patient's function. For example, muscle weakness may affect a person's ability to transfer, dress, or walk independently. Spasticity might impede nursing care by causing difficulty in catheterization or by causing problems with positioning in a wheelchair or splint. Identifying these deficits and determining their effect on function allows the rehabilitation team to set appropriate goals and develop specific treatment strategies to address a patient's needs. This also allows the team to plan for a patient's return home and reentry into the community. Serial examinations in a rehabilitation setting may also provide useful information regarding prognosis for functional recovery.[1]

The initial evaluation of a patient in the rehabilitation setting should include a detailed history, including history of psychiatric disorders or substance abuse. Because many patients with brain injuries have cognitive and language impairments, obtaining the history from family and medical records may be necessary. Additional information about the inciting event should be sought. In patients with TBI, the presence and duration of posttraumatic amnesia is important. Concurrent medical problems, such as brain hemorrhage, hypoxia, hypotension, and seizures; systemic injuries, including skeletal fractures and peripheral nerve injuries; and the presence of intoxicant drugs and alcohol may help in establishing a rehabilitation prognosis.[2] Knowledge of premorbid cognitive and functional status is important. An education and employment history is essential, and for younger patients school records are helpful.

A physical examination should be performed on all patients. This examination should include assessment of level of consciousness, posture in bed, and presence of catheters and tubes (eg, tracheostomy, Foley catheter, gastrostomy tube). The skin should be examined for evidence of skin breakdown (decubitus ulcers). A thorough musculoskeletal examination should be performed, focusing on joint range of motion, skeletal deformities, and abnormal postures of limbs. Finally, a detailed neurologic examination should be performed including an assessment of mental status, cranial nerves, motor and sensory systems, reflexes, coordination, and gait.

Mental Status

Cognitive, behavioral, and language deficits are common in patients with brain

KEY POINTS

- The focus of the neurorehabilitation history and examination is on identifying functional abilities.
- The details of the history and examination are often obtained over time and in collaboration with other members of the rehabilitation team.

disorders. Manifestations include problems with attention and concentration, memory, language, perception, and executive functions. Although a detailed evaluation of these areas is often performed by the speech-language pathologist and neuropsychologist, a basic assessment can be obtained by simple bedside testing.

Level of consciousness. An alteration in level of consciousness may occur in patients with TBI and stroke if there is injury to both cerebral hemispheres or to the reticular activating system in the brainstem. Other contributing factors may include systemic infections and toxic/metabolic abnormalities, including adverse effects of medication. These factors should always be considered, especially when an abrupt change in mental status occurs while participating in a rehabilitation program. An altered level of consciousness is a common cause for denial of a patient's acceptance to an inpatient rehabilitation program or for a transfer back to a medical ward, as this adversely affects an individual's ability to actively participate in the therapy program.[1]

Orientation. Confusion and disorientation are common following stroke and TBI, often because of diffuse cerebral injury, especially involving limbic structures.

Attention and concentration. Attention and concentration are often impaired in patients with frontal lobe insults.[3] This often manifests in therapy as easy distractibility and difficulty remaining focused for the duration of a treatment session. Patients who are inattentive pose a heightened safety risk and have a higher risk of falls and other injuries.

Memory. Both long- and short-term memory may be affected in patients with brain injuries, especially in those with insults to the mesial temporal lobes and thalamus.[4] Anoxic or hypotensive brain injuries often result in selective injury to the hippocampi, which can lead to severe chronic memory deficits. Both old (retrograde) memory and the ability to learn new material (anterograde memory) may be affected. For patients with TBI, the extent and duration of posttraumatic amnesia is an important prognostic factor for recovery.[5] Memory impairments often affect a patient's rehabilitation progress, especially if he or she has difficulty learning new information or has poor carryover of information learned in therapy sessions.

Calculations. Stroke or other injuries to the dominant parietal lobe can affect a person's mathematical abilities. Problems in this area can affect functional tasks, such as balancing a checkbook, paying bills, or shopping.

Speech and language. Language skills are commonly impaired in patients with stroke and brain injuries affecting the dominant hemisphere. Approximately 38% of stroke patients will have some degree of language impairment.[6] Patients may have difficulty with spoken and written language or with language processing. In patients with TBI, anomia is common and is characterized by naming problems, word finding difficulties, and paraphasic errors. Language of confusion (comments not pertinent to the topic) is also frequently seen in TBI. Wernicke (fluent or receptive) aphasia is caused by stroke or focal injury to the dominant temporal lobe and is characterized by fluent paraphasic speech with impaired comprehension (difficulty following verbal commands) and repetition. Broca aphasia is caused by injury to the dominant frontal lobe and is characterized by nonfluent speech with disturbed prosody and perseveration. Injuries to the medial frontal cortex and basal ganglia, often seen in

children with TBI and in recovery from coma, can result in apathy, abulia, and akinetic mutism.[7] Higher-level language skill impairments may become apparent as patients recover from aphasia; these include problems with complex auditory processing, sentence construction, spelling, and abstract tasks, such as picture description.

Dysarthria, or impairment of articulation, is another common sequela of stroke and TBI.[8] Deficits range from mild inarticulation to unintelligible speech. Bilateral involvement of corticobulbar pathways—seen in patients with TBI, bilateral stroke, cerebral palsy, and ALS—results in ''pseudobulbar'' speech, characterized by labored speech, often with a harsh or ''strained'' quality. Cerebellar lesions may result in dysrhythmic speech with irregularity of pitch and loudness. Injury to the basal ganglia may result in jerky, dysrhythmic speech or slow, slurred speech that lacks inflection and modulation. Lesions of the lower brainstem or hypoglossal nerve cause unilateral tongue weakness and difficulty articulating lingual consonants (*t*, *d*, *l*, *r*, *n*). Patients with facial weakness often have difficulty with labial consonants (*p*, *b*, *m*, *w*, *f*, *v*).

Spatial orientation and perception. Patients with stroke or other brain injuries affecting the parietal lobes will often have difficulty with spatial orientation and perceptual tasks. In one series, 82% of right-hemispheric strokes and 65% of left-hemispheric strokes caused visual neglect in the contralateral hemifield.[9] Other manifestations of parietal lobe insults may include neglect of the contralateral side of the body, difficulty with copying geometric designs (constructional apraxia), or, most severely, anosognosia, the inability to recognize one's own deficits. If present, these issues hinder progress with the rehabilitation program. Patients tend to wander or get lost, unable to find their way to their room. They become a safety risk because they may ignore their deficits, dangle a paretic arm in the wheelchair spokes, or bump into objects on their affected side.

Patients may miss objects in the impaired hemispace (eg, fail to eat the food located on the far side of the meal tray).

Affect, mood, and behavior. Depression is a fairly common sequela of stroke, with one-third of stroke survivors developing depression at some point in their recovery.[10] Disturbances in affect and mood are also common in patients with TBI, especially when recovering from coma. Patients with acute neurologic conditions may also demonstrate delirium, which is manifested by disorientation and confusion, distractibility, restlessness, irritability, hallucinations, and delusions, and is generally caused by injuries to the temporal or frontal lobes. Adverse effects of medication, metabolic disorders, and infections may also contribute to behavioral abnormalities. Later during recovery from brain injury, patients may demonstrate alterations in mood, affect, and behavior. Patients with frontal lobe injuries may become irritable and aggressive or demonstrate childlike behavior. They may develop disinhibition, emotional lability and loss of temper control, or, conversely, emotional blunting, diminished drive and initiative, and mental rigidity. Patients with injury to the basomedial frontal cortices demonstrate impaired social judgment and lack of sexual control. Injuries to the dorsolateral frontal cortices often cause problems with executive function, defined as the inability to plan and execute a complex task (eg, planning a meal for guests).[3]

Behavior and affect can be observed during a patient interview. More formal assessment is typically performed by the rehabilitation neuropsychologist.

Cranial Nerves

Cranial nerve I. Although uncommon following stroke, anosmia (olfactory dysfunction) occurs in 13% to 50% of patients with TBI, most commonly because of shearing injury to olfactory pathways in the region of the cribriform plate.[11] Anosmia also develops in 18% of patients following ruptured cerebral aneurysms, correlating with the presence of intraventricular hemorrhage.[12] Anosmia can cause decreased life satisfaction and can lead to safety concerns, for example, the inability to smell smoke, gas, or spoiled food. Standardized, commercially available "scratch-and-sniff" tests may be used for formal testing.

Cranial nerve II. The optic nerve and anterior visual pathways are affected in 5% of patients with brain injuries, resulting in impaired visual acuity, visual field defects, or blindness.[13] Loss of vision occurs most commonly with trauma that causes orbital bone fractures.[14] Stroke can affect the visual pathways anywhere along their course, with monocular blindness resulting from retinal or optic nerve injury, bitemporal hemianopsia from ischemia to the optic chiasm, homonymous hemianopsia from injury to the optic radiations, and cortical blindness from an insult to the calcarine cortex in the occipital lobes.

Visual acuity may be affected by direct injury to the optic nerve or by diffuse occipital lobe injury. Loss of vision can significantly impair function by affecting the ability to read, navigate safely, and perform ADLs.

Cranial nerves III, IV, and VI. Injury to the oculomotor, trochlear, or abducens nerves can occur following a brainstem stroke or contusion, orbital wall fracture, or basilar skull fracture resulting in cavernous sinus injury.[15] Patients may complain of double vision and dizziness, and findings on examination may include eye deviation, dysconjugate gaze, abnormal head postures, and problems with balance and coordination. Alternate eye patching may be beneficial, especially during therapy sessions.

Cranial nerve V. Trigeminal nerve injuries occur in 4% of patients with head injuries, most commonly because of facial bone fractures.[16] These injuries can also occur following brainstem stroke or contusion. Complete trigeminal nerve injury causes hemianesthesia of the face, whereas partial injuries generally result in facial pain. Motor branch involvement can lead to chewing problems, and loss of sensation inside the mouth may cause pocketing of food and increase the risk of aspiration.

Cranial nerve VII. Facial muscle weakness is common in patients who have experienced a stroke or TBI and can affect articulation and swallowing. Injury to the upper motor (corticobulbar) pathways in the frontal lobe, internal capsule, and upper brainstem causes contralateral facial weakness, usually sparing the forehead. Lower motor neuron injury in the pons (brainstem stroke or trauma) results in ipsilateral facial weakness, including the forehead.

Cranial nerve VIII. Hearing loss occurs in 18% to 56% of patients with TBI because of trauma to the middle and inner ear and related structures.[17] Longitudinal fractures of the temporal bone can result in conductive hearing loss from dislocation and disruption of the ossicles, while transverse fractures cause sensorineural hearing loss, vertigo, and disequilibrium from injury to the acoustic nerve, cochlea, and/or labyrinths. Brainstem contusion or stroke, damaging the acoustic or cochlear nuclei, can result in similar symptoms.

Vestibular dysfunction can lead to problems with balance and coordination. The presence of direction-fixed

horizontal nystagmus is suggestive of unilateral vestibular nerve injury. Vertical nystagmus may be seen following brainstem or cerebellar injuries. Certain medications, including anticonvulsants, can also cause nystagmus.

Cranial nerves IX and X. The glossopharyngeal and vagus nerves are often affected in patients with medullary strokes and may be affected following basilar skull fractures extending into the foramen magnum. Injury results in impaired phonation and swallowing. The gag reflex is diminished or absent on the side of nerve injury. The palate and uvula may also be deviated to the opposite side. The gag reflex may be hyperactive in patients with injuries to the corticobulbar tracts bilaterally, usually a consequence of bilateral strokes or injuries to the deep white matter. This is often accompanied by spastic quadriparesis and emotional lability.

Cranial nerve XI. The spinal accessory nerve, innervating the ipsilateral sternocleidomastoid and trapezius muscles, is only rarely injured following basilar skull fractures. Spinal accessory nerve injuries can cause limited neck rotation and shoulder abduction affecting the ability to do activities above the head, such as reach for objects in a high cabinet.

Cranial nerve XII. The hypoglossal nerve, which provides motor function to the ipsilateral tongue, is rarely affected as a consequence of a basilar skull fracture or medullary stroke. Swallowing difficulties can arise because patients may have difficulty manipulating a food bolus in the mouth.

Motor Function

Muscle bulk. Generalized muscle atrophy can occur in the rehabilitation patient because of prolonged immobility. Damage to the lower motor neuron causes focal muscle atrophy. This can occur as a result of direct trauma to the peripheral nerve, plexus, nerve root, or anterior horn cells in the spinal cord. Examples include traumatic spinal cord injury with segmental muscle atrophy or a brachial plexopathy from being thrown from a motorcycle onto the neck and shoulder. Focal nerve injuries can also occur because of limb ischemia following trauma or from improper positioning or casting (eg, peroneal neuropathy with a footdrop from an excessively tight leg cast).

Muscle tone. Spasticity is the most common abnormality of tone seen in patients with stroke, TBI, and spinal cord injury. In patients with brain injuries, spasticity predominantly affects the flexor muscles of the arms and extensor muscles of the legs, while in spinal cord injuries it predominates in the flexor muscles of both the arms and legs. Tone may also be increased in trunk muscles. Spasticity is caused by injury to the corticospinal tracts and is often accompanied by muscle weakness, hyperreflexia, and an extensor plantar reflex response.[18]

Hypotonia may be seen in association with cerebellar lesions and also often occurs early following stroke and spinal cord injuries (spinal shock). In the latter, spasticity may develop later, after a period of days to weeks. A long period of hypotonia in this setting usually suggests a poorer likelihood of functional motor recovery.

Rigidity generally results from injury to the basal ganglia. Although most common in Parkinson disease, rigidity also occurs in patients who have had subcortical strokes, trauma involving the basal ganglia, and anoxic brain injury. Paratonia is a consequence of bilateral frontal lobe injury or dementia.

Spasticity may be painful, can be accompanied by muscle spasms, and may affect rehabilitation progress by interfering with positioning, bracing, transfer skills, nursing care (such as perineal

KEY POINT

■ In traumatic brain injury, any deviation from the expected pattern should trigger a search for undiagnosed injury.

hygiene), and ADLs. Neck and head control can be affected, hampering feeding and grooming. Spasticity of laryngeal and pharyngeal muscles can affect breathing, articulation, phonation, and swallowing. Truncal spasticity can affect wheelchair positioning, standing, and ambulation. If spasticity is severe and prolonged, fixed joint contractures can develop, further impeding the rehabilitation progress.

Muscle strength. Following TBI, the most common patterns of weakness are hemiparesis or tetraparesis because of injury to the corticospinal tracts in the cerebral hemispheres or brainstem. Strokes typically result in hemiparesis, with the arm affected to a greater extent than the leg in middle cerebral artery distribution infarcts affecting cortical structures. In patients with anterior cerebral artery distribution infarcts, the leg is predominantly affected. Subcortical strokes generally affect the arm and leg equally. Any deviation from an expected pattern should trigger a search for additional spinal cord or peripheral nerve injuries (see **Case 2-1**).

With spinal cord injuries, following the period of spinal shock, muscle tone generally returns and patients may regain some sensory and motor function. Cervical spinal cord injuries often result in tetraparesis, while thoracic and lumbar spine injuries lead to paraparesis. The level of spinal cord injury is defined as the most rostral cord level innervating muscles with at least grade 3 strength. Spinal cord injuries are often graded on the American Spinal Injury Association Impairment Scale (**Table 2-3**).

Abnormal movements. Abnormal motor movements or postures may result from brain injuries (**Table 2-4**). Dystonia can occur because of basal ganglia injury (trauma or stroke) or may be seen as an adverse effect of neuroleptic medications and metoclopramide. Dyskinesias of the limbs or orofacial muscles and choreoathetosis may also result from basal ganglia injury or adverse effects of anticonvulsants, oral contraceptives, or antipsychotic medications. Ballismus may occur as a result of trauma or hemorrhage involving the subthalamic region. Tremor of the head or limbs may also result from brain injuries.[19]

Myoclonus can be focal, segmental, or generalized and can occur as a direct consequence of brain injury, including anoxic encephalopathy. Myoclonus is also a common sequela of metabolic

Case 2-1

A 21-year-old man was admitted to rehabilitation 3 weeks after experiencing a severe traumatic brain injury from a motorcycle accident. He was confused and generally noncooperative during the examination. He moved his left side quite well, his right leg less, and his right arm not at all. Reflexes in his right arm were absent. He appeared to have poor sensation in his right arm. After a week, his right arm had not improved, while his right leg was at 80% strength. He was sedated for a cervical MRI which revealed C5 through C8 root avulsion on the right. Several weeks later, nerve conductions revealed normal F waves in the setting of absent sensation and absent motor function.

Comment. The differential diagnosis in this case was cervical root avulsion or brachial plexus injury. Root avulsions carry a very poor prognosis for recovery, and patients frequently develop severe neuropathic pain.

TABLE 2-3 American Spinal Injury Association Impairment Scale

- A = Complete: No motor or sensory function below the level of injury, including sacral segments S4–S5
- B = Incomplete: No motor but sensory preservation below the level of injury, including sacral segments S4–S5
- C = Incomplete: Motor function is preserved below the level of injury but over half of the key muscles have strength grades less than 3
- D = Incomplete: Motor function is preserved below the level of injury and over half of the key muscles have strength grades greater than 3
- E = Normal: Motor and sensory function are normal

abnormalities, including hepatic and renal failure. Asterixis most commonly manifests as a wrist flap when holding the arms outstretched. This can occur in patients with injury to the thalamus, internal capsule, parietal cortex, and midbrain but is often associated with liver failure. Posttraumatic parkinsonism can result from TBI or anoxic brain injury.[20]

Abnormal movements or postures interfere with normal coordinated movements, hampering a patient's ability to perform ADLs, such as feeding and grooming, or to carry out mobility skills, including wheelchair positioning, sitting balance, standing, or ambulation.

TABLE 2-4 Movement Disorders Associated With Brain Injury

- Asterixis
- Ballismus
- Choreoathetosis
- Dyskinesia
- Dystonia
- Myoclonus
- Parkinsonism
- Tremor

Sensory Perception

Sensory perception is commonly affected in patients with brain and spinal cord injuries, although sensory deficits are generally overshadowed by motor and cognitive deficits. Thalamic injuries result in loss of sensation on the contralateral side of the body. Parietal lobe injuries cause loss of ability to localize the site of sensory stimulation, with impaired joint position sense, stereognosis, and graphesthesia. Sensory neglect, including visual neglect, hemiinattention, tactile extinction, and anosognosia, may also be present and is more common following nondominant parietal lobe involvement. Spinal cord injuries result in impaired sensation below the level of the injury, and even in the absence of weakness bilateral lower extremity proprioceptive loss can significantly impair gait.

Sensory deficits can lead to functional impairments. The inability of a patient to detect or localize pain or the presence of sensory neglect can result in injury, as patients may be unable to protect their affected limbs. The inability to control limb position in space because of impaired proprioception can cause problems with feeding and grooming. Lack of feeling in the hands can lead to difficulty with fine motor tasks such as buttoning or fastening

KEY POINT

- Even in the absence of weakness, bilateral lower extremity sensory loss can significantly impair gait.

snaps or zippers. See **Video Segments 8** through **10** for an example of the effects of sensory loss on hand function. Lower extremity sensory deficits can lead to problems with transfers and walking because of impairment in foot placement and balance. Patients with impaired sensation of the buttocks and lower extremities are at increased risk of developing decubitus ulcers, especially if spasticity, impaired mobility, and bowel and/or bladder incontinence are present.

Coordination

Coordination is modulated by a number of peripheral and central nervous system structures and can be affected by brain and spinal cord injuries. Injury to the corticospinal tracts results in muscle weakness with slowing of gross and fine motor tasks. Basal ganglia insults result in slowed initiation of movements. Cerebellar injuries can lead to truncal and limb ataxia, dysmetria, dysdiadochokinesia, dyssynergia, and intention tremor. Sensory ataxia can result from impaired proprioception due either to peripheral neuropathy or spinal cord injury involving the posterior columns. Truncal ataxia can affect sitting and standing balance, impairing the ability to sit upright in a wheelchair or to walk. Limb ataxia can make ADLs difficult.

Reflexes

Evaluation of muscle stretch reflexes helps localize the site of neurologic injury. Hyperreflexia suggests injury to corticospinal tracts either in the brain or spinal cord and is often associated with spasticity and muscle weakness. Hyporeflexia is associated with lower motor neuron injuries and also occurs in the period of acute spinal shock below the level of injury. Hyporeflexia may also be seen in association with peripheral neuropathies and, at times, with cerebellar disease. The bulbocavernosus reflex is useful in the assessment of spinal cord injuries. This is a polysynaptic reflex mediated by spinal segments S2 through S4. It is elicited by squeezing the glans penis or clitoris or tugging on a Foley catheter; the response is contraction of the external anal sphincter. This reflex is generally the first to recover after the period of spinal shock in rostral spinal cord injuries. The absence of this reflex in lower cord injuries suggests injury to the conus medullaris or sacral nerve roots.

Posture and Gait

Brain and spinal cord injuries often affect posture and gait because of injury to the sensory and motor pathways that affect ambulation. Patients with spastic hemiparesis due to stroke or other brain injuries often have weakness and spasticity of the chest and abdominal musculature leading to trunk instability and difficulty with weight shifting. Gait deviation may be observed. Weakness of hip flexors and ankle dorsiflexors results in an impaired swing-through of the limb and inadequate toe clearance during the swing phase of gait, resulting in hiking of the hip and circumduction of the leg. Decreased arm swing on the paretic side may also occur. Spasticity may limit range of motion of the hip, knee, and ankle. Patients with basal ganglia disorders often have a shuffling-type gait. Cerebellar disorders may result in gait ataxia. Patients with proprioceptive deficits may have problems with foot placement and balance. Spinal cord injuries typically result in spastic paraparesis or tetraparesis with difficulty walking as a result. Patients with cervical spinal cord injuries may have weakness of chest and abdominal muscles affecting their ability to sit upright and transfer without support, as well as compromising respiratory reserve.

REFERENCES

1. Gelber DA, Callahan CD. Neurologic examination of the patient with traumatic brain injury. In: Ashley MJ, editor. Traumatic brain injury: rehabilitation, treatment, and case management. 3rd ed. Boca Raton, FL: CRC Press; 2010:3–27.
2. Tate PS, Freed DM, Bombardier S, et al. Traumatic brain injury: influence of blood alcohol level on post-acute cognitive function. Brain Inj 1999;13(10):767–784.
3. Brooks N. Behavioral abnormalities in head injured patients. Scand J Rehabil Med Suppl 1988;17:41–46.
4. McAllister TW. Neuropsychiatric sequelae of head injuries. Psychiatr Clin North Am 1992;15(2):395–413.
5. McFarland K, Jackson L, Geffe G. Post-traumatic amnesia: consistency-of-recovery and duration-to-recovery following traumatic brain impairment. Clin Neuropsychol 2001;15(1):59–68.
6. Pedersen PM, Jørgensen HS, Nakayama H, et al. Aphasia in acute stroke: incidence, determinants, and recovery. Ann Neurol 1995;38(4):659–666.
7. Marin RS, Fogel BS, Hawkins J, et al. Apathy: a treatable syndrome. J Neuropsychiatry Clin Neurosci 1995;7(1):23–30.
8. Sarno MT, Buonaguro A, Levita E. Characteristics of verbal impairment in closed head injured patients. Arch Phys Med Rehabil 1986;67(6):400–405.
9. Stone SP, Halligan PW, Greenwood RJ. The incidence of neglect phenomena and related disorders in patients with an acute right or left hemispheric stroke. Age Ageing 1993;22(1):46–52.
10. Hackett ML, Yapa C, Parag V, Anderson CS. Frequency of depression after stroke: a systematic review of observational studies. Stroke 2005;36(6):1130–1140.
11. Callahan CD, Hinkebein J. Neuropsychological significance of anosmia following traumatic brain injury. J Head Trauma Rehabil 1999;14(6):581–587.
12. Bor AS, Niemansburg SL, Werner MJ, Rinkel GJ. Anosmia after coiling of ruptured aneurysms: prevalence, prognosis and risk factors. Stroke 2009;40(6):2226–2228.
13. Gjerris F. Traumatic lesions of the visual pathways. In: Vincken PJ, Bruyn GW, eds. Handbook of clinical neurology, vol 24. Amsterdam: North Holland Publishing; 1976:27–57.
14. Hoyt CS. Brain injury and the eye. Eye (Lond.) 2007;21(10):1285–1289.
15. Dhaliwal A, West AL, Trobe JD, Musch DS. Third, fourth, and sixth cranial nerve palsies following closed head injury. J Neuroophthalmol 2006;26(1):4–10.
16. Yadav YR, Khosla VK. Isolated 5th to 10th cranial nerve palsy in closed head trauma. Clin Neurol Neurosurg 1991;93(1):61–63.
17. Kochhar LK, Deka RC, Kacker SK, Raman EV. Hearing loss after head trauma. Ear Nose Throat J 1990;69(8):537–542.
18. Gelber DA, Jozefczyk PB. Therapeutics in the management of spasticity. Neurorehabil Neural Repair 1999;13(1):5–14.
19. Flanagan SR, Kwasnica C, Brown AW, et al. Congenital and acquired brain injury. 2. Medical rehabilitation in acute and subacute settings. Arch Phys Med Rehabil 2008;89(3 suppl 1):S9–S14.
20. Bhatt M, Desai J, Mankodi A, et al. Posttraumatic akinetic-rigid syndrome resembling Parkinson's disease: a report on three patients. Mov Disord 2000;15(2):313–317.