Management of Medical Complications

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ABSTRACT
Medical comorbidities and complications are expected following stroke, traumatic brain injury, and spinal cord injury. The neurorehabilitation physician's role is to manage these comorbidities, prevent complications, and serve as a medical and neurologic resource for the patient, family, and neurorehabilitation team. The most common comorbidities are similar to those found in the general population, namely hypertension, dyslipidemia, diabetes mellitus, and ischemic heart disease. Frequent complications encountered in the neurorehabilitation unit relate to medication side effects, medical comorbidities, and the direct effect of the neurologic injury. They include orthostatic hypotension; syncope or presyncope; cardiac arrhythmia; bowel and bladder dysfunction; seizures; pressure sores; dysphagia-related pneumonia, dehydration, and malnutrition; venous thromboembolism; falls; and sexual dysfunction. This article discusses strategies for managing comorbidities and avoiding complications.

THE PHYSICIAN'S ROLE IN NEUROREHABILITATION
The neurorehabilitation physician's primary responsibility is to optimize the patient's medical, neurologic, and behavioral status. This is best accomplished by (1) treating and managing comorbidities that preceded the onset of neurologic impairment, (2) preventing common complications of neurologic injury, (3) providing ongoing assessments of the patient's progress, and (4) modifying treatments based on patient response. The physician may also serve as a neurorehabilitation researcher, team leader, and medical and neurologic educational resource for the patient, family, and neurorehabilitation team members.

Physician rounds on the rehabilitation nursing unit are an integral part of assessing the patient's response to treatment. Nursing reports highlight patient-specific problems that have arisen. This information plus direct observation of the patient's level of alertness, behavior, and vital signs are important indicators of the patient's progress or of intercurrent problems. Having physical therapy, occupational therapy, and speech therapy treatment areas on the nursing unit rather than on another floor or at a remote location provides the most efficient means of staff communication. Nursing staff are available to help therapists with patient care if needed. Nursing and physician staff can directly observe patient performance in therapy activities. This direct and frequent interaction among therapy, nursing, and physician staff allows for a true interdisciplinary approach to neurorehabilitation.

During daily rounds, the physician pays close attention to the patient's medical issues and assesses the adequacy of his or her rehabilitative equipment (eg, Is the wheelchair properly adapted to the patient's needs? Are splints and paretic shoulder support systems in place? Is the patient's spasticity adequately managed?).
Formal weekly conferences with all members of the neurorehabilitation team are an essential component of patient care. These team conferences allow team members to communicate patient-specific goals and progress or impediments to reaching these goals. Each impediment (eg, depression, pain, somnolence, fatigue, spasticity, extrapyramidal manifestations, cardiovascular or respiratory instability) is a target for physician intervention.

Anticipation of expected complications is based on the type and extent of the neurologic injury and should prompt evaluation and intervention to prevent their development. Examples of complications include dysphagia, pneumonia, dehydration, malnutrition, pressure sores, bowel and bladder dysfunction, spasticity, contractures, and depression. Ongoing surveillance of these efforts will document their efficacy or signal the need to change treatment.

Comorbidities are often inherent in the etiology of the neurologic impairment. Stroke is a vascular disorder and hence carries with it a high frequency of cardiac, renal, and diabetic comorbidities. Stroke is also a geriatric disorder with associated neurodegenerative, pulmonary, gastrointestinal, and musculoskeletal comorbidities. Traumatic brain injury (TBI) and spinal cord injury (SCI) are more common in younger, physically active individuals who are more likely to demonstrate risk-seeking behavior and recreational drug use and to have psychosocial comorbidities.

Because medical, neurologic, and behavioral complications are often predictable and comorbidities are common, it is important for the neurorehabilitation physician to develop the skill set and confidence necessary for their management. Use of consultants can help inform and support the neurorehabilitation physician; however, consultant opinions and advice need to be weighed by the neurorehabilitation physician to assure they are appropriate and effective in optimizing the patient's ability to participate in and benefit from the neurorehabilitation programs. The neurorehabilitation physician must continue to assume primary responsibility for patient care. The neurorehabilitation physician, working with the neurorehabilitation team, observes patient performance on the nursing unit and in therapy sessions and is therefore ideally situated to make adjustments in medications to regulate heart rate, orthostatic hypotension (OH), pulmonary function, blood glucose, somnolence, behavior, and pain.

The frequency and severity of medical complications are related to the severity of neurologic impairment, the interval following the onset of neurologic impairment, and the number and severity of medical and neurologic comorbidities. Physician practices and care settings with patients with severe impairments who have many medical comorbidities will encounter the most medical complications.

**MANAGEMENT OF COMORBID CONDITIONS**

**Hypertension and Hyperlipidemia**

Hypertension and hyperlipidemia are expected in geriatric neurorehabilitation patients, and their effective management is an essential part of American Heart Association recommendations for secondary stroke prophylaxis. In patients on the neurorehabilitation unit, labile blood pressure fluctuations are common because of exertion, pain, or anxiety. Adjusting the intensity of exercise, giving pain medication prior to exercise, or giving anxiolytic medications are all appropriate interventions for such patients.

**Diabetes**

Diabetic management should target normal or near-normal blood glucose levels. Numerous studies have shown

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**KEY POINTS**

- The neurorehabilitation physician's role is to manage comorbidities and prevent complications.
- Daily physician rounds optimize assessment of the patient's response to neurorehabilitation programs.
- Weekly team conferences identify problems interfering with functional improvement.
- Comorbidities are related to the etiology of neurologic impairment.
- The neurorehabilitation physician must accept responsibility for the patient's overall medical management. Consultants can be used to optimize patient care and to help inform and support the neurorehabilitation physician.
- The frequency and severity of medical complications are related to the severity of neurologic impairment, the interval following the onset of neurologic impairment, and the number and severity of medical and neurologic comorbidities.
that the better the blood glucose regulation the better the functional outcome. However, a number of special issues interfere with diabetic management following neurologic injury: inability to express hypoglycemic symptoms because of aphasia, altered awareness of impending hypoglycemia, dysphagia-related variability in food intake, and meals and exercise that are regulated by the patient’s schedule and not by the patient. Blood glucose is frequently difficult to control when the patient is on tube feedings, and the timing of monitoring and insulin administration should be adjusted. The goal is to balance the need for near-normal blood glucose levels with avoidance of symptomatic hypoglycemia. Because glycosylated hemoglobin levels change slowly, we have not found them to be a useful guide for diabetic management during the several-week inpatient neurorehabilitation stay. Fingertip blood glucose determinations before meals and at bedtime remain the most valid assessment of diabetic management.

Congestive Heart Failure
American Heart Association standards for managing congestive heart failure (CHF) advocate use of three classes of medication: a diuretic, a beta-blocker, and an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker. Use of these standard medications may have to be modified during neurorehabilitation because of dysphagia-related dehydration, diabetics or vascular renal insufficiency with hyperkalemia, and bradycardia or OH. Daily rounds by the physician are important for adjusting dosages of CHF medications. Patients with dysphagia may not be able to tolerate the use of diuretic medications. They may report that they do not like the taste of thickened liquids and hence cannot maintain adequate oral hydration if given a diuretic. Frequent small amounts of thickenened liquid several times per day will help supplement hydration. It is difficult to maintain adequate hydration for those on honey-thick liquids. It is important to note that diuretics are used to enhance renal sodium excretion. Extracellular water volume decreases as a result of enhanced renal sodium clearance. Coupling a diuretic with adequate hydration is necessary to ensure adequate renal function. On daily rounds the physician can observe the patient’s resting respiratory rate while the patient is in a comfortable position in bed or in a wheelchair. Auscultation of the lungs with detection of basilar rales, dullness to percussion over the lung bases, presence or absence of jugular venous distension (more than 12 cm above the sternomanubrial joint is abnormal in either supine or sitting position), and presence of either bilateral pedal or presacral edema are all indicators of CHF. Daily observation for the presence of these signs of CHF should trigger further evaluations such as serum β-natriuretic peptide (BNP) assessment. BNP is a reliable marker of atrial stretch response to intravascular volume expansion unless significant renal dysfunction is present. In the presence of chronic kidney disease with serum creatinine greater than 2.0 mg/dL, the BNP may not accurately reflect CHF status. A follow-up chest x-ray can confirm the clinical assessment. Follow-up echocardiograms are not usually available on the neurorehabilitation unit. With daily physician assessments and appropriately timed serum BNP, blood urea nitrogen (BUN), serum creatinine, and serum electrolyte assessments it is usually possible to titrate the patient’s CHF medications to optimize functional status and avoid dehydration.

PREVENTION OF COMMON COMPLICATIONS
Cardiovascular Complications
Orthostatic Hypotension. OH is defined by the American Academy of
Neurology and the Consensus Committee of the American Autonomic Society as a drop in systolic blood pressure of 20 mm Hg or more or a drop in diastolic blood pressure of 10 mm Hg or more. OH is common on the neurorehabilitation unit. It is often attributed to overaggressive antihypertensive medication management during the acute hospital phase of patient care. Blood pressures seem well managed when patients are supine in bed, but OH becomes apparent when they begin spending more time sitting in a wheelchair, standing, and walking. OH can be a manifestation of deconditioning, dehydration, or another comorbidity such as diabetic autonomic neuropathy. Dehydration is a particularly common cause of OH following neurologic injury with dysphagia. While diuretics are frequently used as one of the primary medications for hypertension management, dysphagia requiring use of nectar- or honey-consistency liquids is an indication of severely compromised ability to maintain oral hydration. Diuretics should be avoided in patients with dysphagia requiring thickened liquids unless they are needed for management of advanced CHF.

SCI is commonly associated with OH in the acute phase postinjury. OH following SCI may persist several weeks into the chronic phase and is frequently asymptomatic. OH is particularly severe in patients with cervical SCI. These patients may require use of a tilt table and abdominal binder to gradually accommodate to the upright posture.

Management of OH includes decreasing or stopping antihypertensive medications, ensuring adequate hydration with oral fluids if possible or with IV fluids if needed, applying elastic compression wraps to the lower extremities, and using an abdominal binder to help increase peripheral and visceral venous return. Such treatment is outlined in Case 6-1. Patients should be kept out of bed as much as possible and encouraged to sit up or stand as they are able with appropriate monitoring of blood pressure. Patients who cannot tolerate being upright even with the above interventions may need to practice incremental habitation to upright posture using a tilt table or a specialized wheelchair that tilts to a recumbent position with elevating leg rests and a fully reclining backrest (Figure 6-1). Medical management may include IV volume expansion and adding vasoconstrictors such as midodrine. Observational studies have supported the use of fludrocortisone.

**KEY POINT**

- Orthostatic hypotension following stroke or traumatic brain injury is most commonly due to antihypertensive medication or dehydration with prerenal azotemia and is particularly severe in patients with cervical spinal cord injury.
Case 6-1
A 74-year-old man with a history of left basal ganglia infarct resulting in right hemiparesis and dysarthria was admitted to the neurorehabilitation unit. Review of his hospital chart revealed that the patient had a history of uncontrolled hypertension, coronary artery disease, diabetes mellitus, and hyperlipidemia. During his acute hospitalization he had frequent blood pressure spikes with systolic blood pressure recordings of up to 200. He was being treated with hydralazine, metoprolol, and lisinopril and was on a pureed diet and nectar-thick liquids because of dysphagia. On his second day of admission, the patient began physical therapy. He was ambulating 10 feet with moderate assistance at the hemibar when he suddenly became diaphoretic and weak and reported dizziness. He was immediately placed in a reclining wheelchair and brought back to his room. His heart rate was 90 beats/min, his blood pressure was 90/68 mm Hg, his oxygen saturation was 96% breathing room air, and his blood sugar was 114. His morning laboratory results showed a serum sodium of 147, a blood urea nitrogen of 35, and a creatinine of 1.0. The patient appeared lethargic. He was placed in Trendelenburg position, and over the next 20 minutes his mental status slowly returned to baseline. His medications were reviewed and a decision was made to stop his hydralazine. Because of his history of diabetes and coronary artery disease, the lisinopril and metoprolol were reduced in dose but not stopped. Orders were given to obtain blood pressure measurements with the patient sitting up to avoid falsely high readings. Orders were also written to give 3 ounces of nectar-thick fluids with each therapy session to improve hydration. Elastic wraps and an abdominal binder were applied to further provide circulatory support. The patient was allowed to return to his therapy sessions after his symptoms subsided.

Comment. This case illustrates that even patients with baseline hypertension and high blood pressure readings when supine may suffer from orthostatic hypotension when standing or sitting up in a chair. The doses of blood pressure medications need to be decreased, with an emphasis on those that do not benefit other medical conditions the patient has. Blood pressure readings for this patient should be taken in a sitting position. Because he has dysphagia and is taking nectar-thick liquids (which he does not like), he is having trouble maintaining adequate hydration.

Another stimulus to the sacral, lumbar, or thoracic spinal cord often triggers an AD episode. Placing the patient in a seated rather than supine position may be of immediate benefit. Using lidocaine gel to ease noxious stimuli associated with bladder catheterization or digital disimpaction may terminate the episode. The Consortium for Spinal Cord Medicine clinical practice guidelines acknowledge that no randomized controlled trials to identify optimal medical management of AD have been reported.15 Reasonable alternatives are oral rilnidipine, hydralazine, prazosin, captropril, and transdermal nitrate. In severe persistent episodes, IV nitroprusside in an emergency department or intensive care unit setting may be required for patient stabilization. Use of sildenafil for erectile dysfunction within 24 hours is a relative contraindication for use of nitrates.

Syncope or Presyncope. If a clear orthostatic association with syncope or presyncope exists, the etiology of the
event is clearly related to hypotension. Time in the wheelchair may also be an important variable. Vasovagal events associated with toileting are also common. Constipation with or without fecal impaction or functional vesicourethral outflow obstruction may be precipitating events and should be treated appropriately. Every syncopal or presyncopal event should be considered as possibly related to some other etiology unless the situation and recovery are sufficiently clear. Fingerstick blood glucose, pulse oximetry, EKG, and observation for absence seizure, cryptic seizure, or another etiology should be considered.

Patients taking multiple antihypertensive medications and those with underlying dehydration or vascular volume contraction may not show a quick return of blood pressure to baseline level when placed supine or in Trendelenburg position, and supplemental IV hydration may be needed. Likewise, recovery of baseline level of alertness may be prolonged for minutes to an hour. Focal neurologic findings that had improved since an initial stroke may worsen and reflect hypoperfusion of marginally functioning ischemic stroke penumbra. Modification of antihypertensive medication may be needed to allow a slightly higher blood pressure to maintain perfusion. If symptoms persist despite measures to increase cerebral blood flow, follow-up imaging would be indicated to evaluate stroke progression versus secondary hemorrhage into an ischemic stroke.

**Transient Arrhythmias.** Asymptomatic bradycardia or minimally symptomatic bradycardia with fatigue, poor endurance, or exertional dyspnea is often observed in patients participating in physical therapy and occupational therapy programs. If this is attributable to the use of beta-blockers and the EKG is unchanged from baseline (except for heart rate), a reduction in beta-blocker dosage is indicated. Patients with chronic stable atrial fibrillation may show occasional asymptomatic 2- to 3-second pauses between QRS complexes. This is most common while resting in a wheelchair or in bed and resolves with minimal activity. Reduction in beta-blocker, digoxin, or calcium channel blocker dosage may be sufficient to correct the problem. The frequency of such events is easily documented on the neurorehabilitation unit with Holter monitoring and can help guide medical management. A system for urgent cardiology consults should be available. Mobitz type II heart block, complete heart block, or bradycardia associated with signs or symptoms of CHF or angina pectoris are treated according to advanced cardiac life support protocol, and patients with these arrhythmias should be emergently transferred to an acute care hospital setting.

Tachycardia is also commonly noted by nursing or therapy staff. EKG confirmation of sinus tachycardia may indicate hypoglycemia, deconditioning, dehydration, anemia, withdrawal from beta-blocker, hyperthyroidism, CHF, or pulmonary embolism. Each should be considered and judged according to physical examination, laboratory results, chest x-ray, and blood gas findings. Tachycardia due to deconditioning is a diagnosis of exclusion. New-onset atrial fibrillation, often with a heart rate above 150, requires further cardiovascular evaluation to exclude acute myocardial ischemia, heart failure, hyperthyroidism, or another etiology. The severity of the tachycardia, the need for further cardiac evaluation, the need for repeat neuroimaging, and the decision to initiate warfarin treatment usually warrant transfer back to an acute care hospital setting.

**Seizures**
The incidence of seizures within the first 2 weeks of ischemic stroke has been estimated to be approximately 2% to 23% depending on study design. The
incidence of clinical seizures following intracerebral hemorrhage (ICH) has been reported to be 14%, with most occurring at or shortly after the acute event.\textsuperscript{14} Electroencephalographic evidence of seizure activity may be as high as 28% to 31% in patients with ICH having continuous EEG monitoring. No clear evidence exists for prophylactic use of anticonvulsants to improve functional outcome or survival. Patients with ischemic stroke or ICH who have clinically apparent seizures and patients with electroencephalographic evidence of seizures with accompanying change in mental status should be given anticonvulsants. Evidence from animal studies suggests that phenobarbital, topiramate, lamotrigine, and phenytoin may impair motor recovery\textsuperscript{15}; however, clinical evidence is still lacking.

**Pressure Sores**

Pressure sores are defined as wounds resulting from pressure or friction on any part of the body. Immobilized neurorehabilitation patients in hospital and nursing home settings following stroke or SCI are at high risk of developing pressure sores. Pressure sores can cause pain, increase disability, and lead to systemic infections and death.\textsuperscript{16} The incidence of pressure sores is estimated to be 25% to 30% in patients with SCI\textsuperscript{17,18} and 20% in patients with stroke.\textsuperscript{19,20}

Pressure ulcers develop when the pressure on the tissue is persistently greater than the capillary pressure, which has been traditionally quoted to be 32 mm Hg, for more than 2 hours.\textsuperscript{21} However, newer studies have shown that with increasing external pressure the arteriolar pressure increases through autoregulation unless the external pressure exceeds the diastolic pressure. At this point, a significant decrease in tissue oxygen partial pressure ($P_{O_2}$) occurs with resulting tissue hypoxia and necrosis.

Risk factors include compressive forces and shearing forces.\textsuperscript{21} Compressive forces are directed perpendicular to the skin surface, whereas shearing forces are exerted parallel to the skin surface. Shearing forces depend on the position of the patient. The shear exists between the different layers of the fascia. The influence of body weight on shearing and compressive forces is such that in patients with cachexia higher peak pressures exist than in larger persons, in whom the pressure is dispersed throughout a larger surface area.\textsuperscript{22} The duration of pressure has been found to be an independent risk factor, and the frequency of turning the patient has been linked to the incidence of pressure ulcers.\textsuperscript{23} The risk of developing pressure sores is further increased in patients with decreased sensitivity to pain from the pressure and those who have decreased ability to shift their weight in order to reduce the pressure.

Poor nutrition is an independent risk factor for pressure sores. Low albumin levels have been associated with a higher risk of developing these lesions. Individuals who are malnourished have a twofold to threefold higher risk of developing ulcers than patients with normal nutritional status, and providing individuals with nutritional supplements has been shown to accelerate healing.\textsuperscript{24} Patients with albumin levels of less than 3.5 have been shown to have a greater risk of developing ulcers and slower ulcer healing.\textsuperscript{25}

Pressure ulcers develop over bony protrusions. A study involving patients with SCI found that the ischial tuberosities, sacrum, greater trochanter, lateral malleoli, and heels are commonly affected.\textsuperscript{26} Shear forces and incontinence with skin maceration further traumatize the skin and facilitate bacterial and fungal infections.

The Braden scale and the Norton scale are used to identify patients at
risk for developing pressure ulcers. The Norton scale rates the patient's physical condition, mental status, activity, mobility, and continence. The Braden scale measures friction, shearing forces, sensory perception, skin moisture, nutritional intake, and physical activity. Both scales have been shown to have limited interrater reliability and tend to underestimate the risk of developing pressure sores.27

Preventive measures suggested by the Consortium for Spinal Cord Injury include avoidance of prolonged immobilization; daily inspection of skin with particular attention to coccyx, ischium, trochanters, and heels because these areas are at greatest risk for development of pressure ulcers; avoidance of shearing forces; preventing moisture accumulation; and providing pressure relief support for areas at risk.

Computerized bed mattress systems are available to attenuate pressure points and change the patient's position from one side to the other (Figure 6-2). Gel or graduated foam cushions may alleviate pressure when the patient is sitting up in a wheelchair (Figure 6-3). Frequent weight shifting is also important for these patients, as well as avoidance of any pressure points on the paretic limb by leg rests or foot rests. Pressure points over the heels can be reduced by pressure relief ankle-foot orthoses. Adequate intake of calories, protein, micronutrients, and fluid should be provided.

Treatment is based on pressure sore staging (Table 6-1); an example of appropriate pressure sore treatment is given in Case 6-2. The ulcer should be measured with every dressing change, and good hand hygiene should be ensured before and after dressing changes. Cleaning of the ulcer should be performed with normal saline; solutions containing alcohol and rubbing of the wound should be avoided. Stage I ulcers can be treated with a hydrocolloid dressing. These nonbreathable dressings are biodegradable and adhere directly to the skin, making additional taping unnecessary. They have to be changed every 3 days or more frequently when excess drainage from the wound occurs.

For stage II and III ulcers, dressings made from sodium carboxymethylcellulose can be used. These dressings form a gel that absorbs wound exudates and traps bacteria. They may also be impregnated with silver. Silver ions are released in the wound and exert an antimicrobial effect against a wide range of bacterial organisms, including vancomycin-resistant Enterococcus, methicillin-resistant Staphylococcus aureus, and Pseudomonas.28 Collagenase-containing gels can be used for minor debridement of the ulcer bed if needed. Surgical debridement is indicated for removal of significant necrotic tissue. More advanced pressure sores, such as stage III and stage IV, require more intense treatment. The use of vacuum-assisted closure (VAC) therapy has been shown to be effective in these wounds. VAC is safe and easy to apply and is thought to work by increasing local blood flow to the wound, increasing cell proliferation, and increasing formation of granulation tissue. These
mechanisms promote wound healing and increase speed of wound closure. Surgical debridement may be required prior to using a VAC system whenever significant necrotic tissue is present. Surgical closure of the wound by creating skin flaps is the most aggressive treatment option, but it depends on the patient's surgical risk and the prognosis of the underlying neurologic or medical disorder.

**Dysphagia-Related Complications**

Dysphagia and its management are discussed in detail in the article "Treatment of Language, Motor Speech Impairments, and Dysphagia." The most important medical complications of dysphagia are pneumonia, dehydration, malnutrition, unreliable medication intake, and upper airway obstruction.

A bedside evaluation of swallowing should be conducted prior to initiation of oral intake of food or liquids following stroke or TBI. Based on the bedside dysphagia evaluation, patients at risk for aspiration can be given nectar- or honey-consistency liquids. Patients deemed unsafe for oral feeding require nasogastric feeding tube placement. Nasogastric tubes are poorly tolerated by patients and proper positioning must be checked by air bolus auscultation over the abdomen prior to each feeding. If a nasogastric tube is required for more than 1 or 2 weeks a percutaneous endoscopic gastrostomy (PEG) tube should be placed.

Patients with dysphagia significant enough to require thickened liquids are at risk for recurrent aspiration of saliva and other thin liquids between meals and at night. Using an oral swab to cleanse the teeth, tongue, and buccal folds after meals can help decrease the amount of bacterial and particulate material available for aspiration. Use of incentive spirometry, nebulized bronchodilator treatments, and
### TABLE 6-1 Criteria for Staging of Pressure Ulcers

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<tr>
<th>Stage</th>
<th>Criteria</th>
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<tbody>
<tr>
<td>I</td>
<td>Nonblanchable erythema of intact epidermal skin, usually over bony prominence. This is an indication of individuals at risk for developing skin ulceration. The affected skin can be painful, soft or firm, and may be warmer or cooler than the surrounding tissue.</td>
</tr>
<tr>
<td>II</td>
<td>Shiny or dry, shallow superficial ulcer penetrating into the dermal skin layer, often with red or pink wound bed.</td>
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<tr>
<td>III</td>
<td>More extensive tissue damage with full-thickness skin loss penetrating into the subcutaneous fat tissue layer but not into layers of fascia, tendon, muscle, or bone. Can present as a deep crater in areas with increased adipose tissue but may be shallow in areas without significant subcutaneous fat.</td>
</tr>
<tr>
<td>IV</td>
<td>Extensive damage with full-thickness skin loss and involvement of underlying fascia, tendons, muscle, or bone. Eschar formation may be present in the wound bed. Ulcers may be shallow in areas without underlying adipose tissue but can otherwise be very deep.</td>
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*For pressure-related injury not included in this staging system is deep tissue injury, which is an injury to the subcutaneous tissue layers caused by pressure that frequently gives the appearance of a deep bruise. Such lesions can be recorded as unstaged.*

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chest percussion and postural drainage by physical therapists, respiratory therapists, and nursing staff can also help mobilize increased tracheobronchial mucus formed in response to chronic, low-grade aspiration. Chest auscultation, pulse oximetry, and vital signs can signal significant change in respiratory status prompting an x-ray to evaluate for pneumonia. Guidelines outlining the antibiotic treatment of adults with hospital-acquired pneumonia and health care–acquired pneumonia have been published by the American Thoracic Society and the Infectious Diseases Society of America.

Dysphagia significant enough to require use of thickened liquids places patients at significant risk of dehydration. Patients will report that they do not like thickened liquids and will often refuse them. Allowing patients access to thickened liquids may improve hydration and nutrition.

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**Case 6-2**

A 36-year-old man was admitted to the spinal cord unit 2 weeks after a motorcycle accident. He had sustained multiple bone fractures, liver and spleen lacerations, and a T8 spinal cord transection. On examination, the nurse noticed a 4 cm diameter erythematous area over his sacrum that did not blanch when touched. In the center, a 2 cm diameter shallow ulcer was present. The base of the ulcer was pink and clean with hair follicles visible. The wound was classified as a stage II pressure ulcer. It was gently cleaned with normal saline, and a silver impregnated sodium carboxymethylcellulose dressing was applied. The patient was frequently turned, and pressure on his sacral area was minimized using an alternating pressure air mattress. While sitting in a wheelchair, he was supported with a gel-based wheelchair cushion. His wheelchair was placed in a reclining position at regular intervals during the day to further alleviate any pressure points. The patient’s albumin level was 2.8, so he was started on a multivitamin, vitamin C, and a protein-enriched diet. With the above measures, his ulcer slowly improved.

**Comment.** This case illustrates the importance of aggressive management of pressure ulcers. Management includes wound care, patient positioning, alleviation of pressure points, and nutritional support.
to water, such as with the Frazier Rehab Institute Free Water Protocol (permitting small sips of water between meals with nursing staff providing oral and dental hygiene following each meal), may be appropriate. This allows patients to safely consume small volumes of thin liquids and improves hydration. Diuretics should be stopped unless needed for management of active CHF. To ensure adequate hydration, it is reasonable to request that patients receive at least 3 ounces of thickened liquid with each therapy session. Renal and electrolyte checks can be used to follow the patient's response. If necessary, a nursing order can be written to give the patient 250 mL of thickened liquid as a medication every 4 to 6 hours while awake. Supplemental IV hydration may also be needed. It is common to monitor the BUN/creatinine ratio, with values greater than 20 indicating prerenal azotemia due to dehydration. Patients with dysphagia may be sufficiently malnourished so as to show only mildly elevated BUN/creatinine ratios because of dietary protein depletion. Such patients may have elevated serum sodium values in the 150 range, serving as a reliable secondary marker for significant dehydration.

Malnutrition due to dysphagia and inadequate calorie intake can be followed using body weight, but values fluctuate and change slowly. Urinary ketones may be detectable after a 24-hour fast. Use of urinary ketone levels as a marker for inadequate calorie intake is valid even in patients with diabetes as long as no glycosuria is present. Serum prealbumin levels fall much earlier than serum albumin or total serum protein levels and are the best marker for inadequate protein intake. All patients on an altered-consistency diet should be assessed for the need for dietary supplements and multivitamins.

If the above measures are not effective in maintaining hydration, nutrition, and medication administration, a PEG tube should be considered. Having nursing staff estimate the amount of food on the diet tray that is consumed provides a reasonable approximation of total calorie consumption. Patients and families may reject discussions of PEG placement because they perceive it as artificially prolonging life. However, case-matched controlled studies have shown that patients given PEG tube feedings do as well as equally severely impaired patients who do not need PEG feeding. They have similar Functional Independence Measure efficiency values and are equally likely to return to their home. PEG feedings allow patients who are effectively starving to show significant functional recovery. Most will not need their PEG tube for more than 4 to 10 weeks, and most PEG tubes can be easily removed by traction or balloon deflation on the neurorehabilitation unit or in the office. To prevent spilling of gastric content to the peritoneum, PEG tubes should remain in place for at least 2 weeks prior to removal, with some recommending up to 6 weeks.

**Deep Vein Thrombosis**

In patients admitted to a rehabilitation hospital and in patients within 21 days of stroke onset, the prevalence of deep vein thrombosis (DVT) has been reported to be 34% to 40.2% and even as high as 50%. The prevalence of DVT in patients with TBI has been reported to be 11%. Most of these patients were asymptomatic. Higher incidences seem to be related to greater severity of weakness. DVTs usually occur in the paretic leg.

It is important to be vigilant for the development of DVT in the neurorehabilitation population because clinical signs may not be present in these patients owing to sensory loss and the inability to report symptoms because of cognitive or language impairment. Screening tools should be cost-efficient,
noninvasive, easily obtainable, and highly sensitive and specific. The criterion standard for diagnosing DVT is contrast venography; however, this procedure is invasive and not without risks such as allergic reaction, nephrotoxicity in patients with preexisting kidney disease, and postvenography thrombosis. Serum D-dimer levels, which are elevated in the presence of venous clot formation, have been evaluated in several studies. In patients with low clinical suspicion of DVT, a normal D-dimer serum value can be used to safely exclude the presence of DVT. Another accepted diagnostic modality is Doppler ultrasonography, which is cost-efficient and safe and has a high sensitivity and specificity (96% and 98%, respectively). Compressibility of veins under probe pressure is also a very accurate test. The sensitivity is best for proximal DVTs and less optimal for isolated calf DVTs.

Prevention of DVT and venous thromboembolism (VTE) is important for patients with immobilization due to TBI, SCI, or stroke. Prophylactic use of knee- or thigh-length graded compression stockings (GCSs) has been studied, but their use has not shown a significant decrease in the incidence of VTE in patients with stroke. One study using thigh-length GCSs showed no significant decrease in the incidence of DVT after stroke but found that their use was associated with a significant increase in superficial skin lesions. However, in studies examining the incidence of DVT in patients hospitalized for any reason, the use of GCSs has been shown to be beneficial. Because of the possibility that they may have some benefit and the benign nature of their use, GCSs are used as part of routine DVT/VTE prophylaxis in most neurorehabilitation units.

Successful pharmacologic prophylaxis with unfractionated heparin (UFH) has been demonstrated in surgical patients. In patients with ischemic stroke, prophylaxis with UFH has shown an almost 80% reduction in the incidence of DVT. Low-molecular-weight heparin (LMWH) given once daily has been found to be at least as effective in preventing DVT as UFH given 3 times a day and to be superior to twice daily injections. The risk of major adverse events such as bleeding has been reported as similar or slightly reduced following the use of LMWH versus UFH in these trials. LMWH is less likely than UFH to produce heparin-induced thrombocytopenia.

Recently published guidelines for the management of ICH state that "after documentation of cessation of bleeding, low-dose subcutaneous low-molecular-weight heparin or unfractionated heparin may be considered for prevention of VTE in patients with lack of mobility after 1 to 4 days from [ICH] onset." This recommendation is listed as Class IIIb, indicating that the benefit may be greater than the risk based on limited patient populations studied. The Consortium for Spinal Cord Medicine guidelines recommend the use of LMWH within 72 hours of SCI provided no active bleeding is present. They recommend enoxaparin 30 mg subcutaneous every 12 hours, dalteparin 5000 units subcutaneous daily, or warfarin to a therapeutic international normalized ratio of 2 to 3. DVT prophylaxis for patients with SCI is recommended for 8 weeks for patients with complete motor deficits and for 12 weeks for such patients with other risk factors for VTE complications. DVT prophylaxis for patients with stroke and TBI is usually continued until the patient can ambulate distances of 150 feet with or without therapist assistance or until the patient is sent home or to a custodial care setting.

Venous compression pump systems are mostly reserved for patients unable to take LMWH or UFH because of the
KEY POINT

Chronic constipation is expected in immobilized patients and can be prevented.

presence of active bleeding, heparin-induced thrombocytopenia, or other disorders affecting blood clotting. Foot, calf, and sequential calf-thigh compression pump systems are available. Sequential calf-thigh systems provide optimal prophylaxis but are poorly tolerated because of discomfort and interruption of sleep. Foot-pump systems are tolerated the best but are not as effective as either calf or sequential calf-thigh applications. Rapid compression of the venous plexus in the instep of the foot is able to produce a 100 mm Hg plethysmographically detectable pulse wave in calf veins. The pulse wave is thought to provide venous pulse pressures similar to stepping and weight bearing during the gait cycle; however, these devices are not frequently used or not used correctly.

Bowel and Bladder Dysfunction

Colonic stasis is expected as a result of immobility of any cause. A high-fiber diet containing 35 grams of fiber plus a stool softener such as docusate sodium 100 mg 3 times daily is an initial prophylactic option. Use of bisacodyl suppositories daily or every other day if no bowel movement occurs is reasonable. Manual insertion of the suppository initiates the anal-colon defecation reflex. Bisacodyl also has a direct effect on the smooth muscle of the colon, stimulating colonic emptying. Additional strategies such as enemas or lactulose may be needed. Lactulose can produce loose stools in any individual if given in higher doses. Lactulose is not absorbed and is without systemic toxicity. Polyethylene glycol compounds (eg, Miralax) may also be helpful. However, if a patient cannot move his or her bowels for several days, the possibility of an obstruction must be ruled out.

Urinary incontinence (UI) or urinary retention affects a large percentage of patients with acquired brain or spinal cord injury. The prevalence of UI after stroke has been reported as almost 50% (including complete and incomplete UI). However, this prevalence is linked to the severity of neurologic injury. Only 5% of patients with pure motor strokes are expected to have UI, while approximately 75% of patients with motor, sensory, and hemianopic neurologic impairments are expected to experience UI during the first month following stroke. Case 6-3

Case 6-3

A 78-year-old woman with recent infarct involving the left temporal-parietal cortex had resulting global aphasia and right hemiparesis. On the second day of her admission to the inpatient rehabilitation unit, the physical therapist noticed that she seemed less alert than on the day of admission and that she appeared uncomfortable. She was afebrile. Discussion with nursing staff revealed that the patient had not voided over the past 8 hours. On physical examination, dullness to percussion over the suprapubic region was present. Bladder scan showed a postvoid residual volume (PVR) of 700 mL. Intermittent catheterization was initiated, and urine analysis and urine culture were obtained. PVR assessments were ordered every 6 hours with instructions to perform intermittent catheterization for a PVR volume greater than 250 mL. Additionally, she was started on tamsulosin for internal urethral sphincter relaxation.

Ciprofloxacin was started subsequently when her urine analysis showed significant pyuria.

Comment: This case illustrates that it is necessary to be vigilant for stroke complications such as urinary retention because patients often cannot report specific symptoms because of their neurologic dysfunction. Careful assessment of the patient's voiding status and frequency of bowel movements must be made to avoid obstructive uropathy and fecal impactions. Urinary retention is treated with intermittent catheterization rather than an indwelling catheter.
gives an example of a patient with
global aphasia with urinary retention
who is unable to verbalize her con-
cerns and has only nonspecific symp-
toms, highlighting the importance of
routinely evaluating patients for the
presence of urinary retention. Patients
with poststroke UI have higher mor-
tality rates than those without UI (60% 
versus 20% in one study), and dis-
ability is worse at 3 months in patients 
with UI. The natural progression of 
poststroke UI is such that most patients
improve. Age younger than 75 years 
and lacunar infarcts are associated with
significant probability of recovery. UI
following neurologic injury can be due
to either impairment of the bladder’s
ability to store (urge incontinence) or
failure to empty (urinary retention with
overflow incontinence). Stress inconti-
ience is usually caused by lax pu-
dendal musculature due to childbirth
or uterine, vesicle, or rectovesical pro-
lapse and is not a primary sequela of
neurologic injury.

Urinary retention is common (75% 
of patients during the first month)
following middle cerebral artery dis-
tribution stroke with hemiparesis and 
hemisomatosenory and hemianopic
visual impairments. Bladder emptying
is normally under voluntary control
regulated by the medial frontal cortex
which controls the pontomesencephal-
ic reticular formation micturition 
center. The pontomesencephalic micturition center coordinates parasympa-
thetic fibers from S2 through S4 that
cause detrusor muscle contraction
and sympathetic fibers from T11 to
L1 that cause relaxation of the internal
urethral sphincter. Overall, activa-
tion of the sympathetic nervous system
leads to urinary continence, while ac-
tivation of the parasympathetic ner-
vous system facilitates voiding. Aware-
ness of bladder volume is mediated by
spinal cord dorsal column propriocep-
tive afferents projecting to the som-
asensory cortex of the paracentral
lobe on the medial aspect of the
interhemispheric fissure. During cy-
tometric instillation of carbon dioxide,
the first sensation of bladder fullness
starts at a filling volume of about
125 mL. Voluntary inhibition of voiding
is controlled by the motor cortex in
the paracentral lobe giving rise to
descending efferents in the lateral col-
umns which initiate contraction of the
external urethral sphincter and the
urogenital diaphragm. Voluntary con-
traction of the external sphincter and
urogenital diaphragm results in reflex
inhibition of the detrusor muscle and
inhibition of the urge to void.

Neurologic injuries can impair urin-
ary continence in several ways. Large
middle cerebral artery distribution hemi-
spheric injury may initially cause de-
trusor atony that may last for several
weeks, causing urinary retention requir-
ing intermittent urinary catheterization.
With time such lesions then give rise
to disinhibition of the pontomesence-
phalic micturition center with detrusor
hyperreflexia and urge incontinence.
Several studies have shown a correla-
tion between frontal lobe lesions and
urinary urge incontinence. SCI above
the conus medullaris as well as brain
stem lesions affecting the pontomesence-
phalic reticular formation can cause
dysynergia between detrusor muscle
contraction and internal sphincter re-
lexation leading to urinary retention.
With time such SCI lesions may also
give rise to a spastic, hyperreflexic urge-
inentcontinent bladder.

Urinary tract infections (UTIs) are
common following neurologic injury.
Patients with urinary urgency, frequency,
dysuria, or incontinence should have
a urinalysis with urine culture if indi-
cated. It is important to realize that a
clean voided midstream urine speci-
men is often unobtainable in patients
KEY POINTS

- Intermittent bladder catheterization is preferable to indwelling catheter for management of urinary retention.
- Time-prompted voiding schedules can ameliorate urge incontinence.

with neurologic impairments who are unable to participate in the procedure. Such patients may need external condom specimens (males) or catheterized urine specimens (females). Contamination of the urine specimen by vaginal or perineal mucus frequently gives rise to artifact in laboratory analysis of pyuria and bacteruria. Early recognition and treatment of UTI may prevent development of symptomatic infections or urosepsis which can impede stroke recovery and prolong the hospital stay.51

Urinary retention is an independent risk factor for UTI,52 and if it is severe enough it may lead to renal insufficiency and renal failure. Indwelling Foley catheters are one means of treating urinary retention. They have, however, been shown to significantly increase the risk of developing a UTI.53 Intermittent catheterization based on bladder ultrasound determinations every 6 hours is associated with a significant decrease in UTIs. The risk of inducing infection versus the need to prevent bladder distension is probably optimized by requesting that the patient be catheterized when postvoid residual volume by Doppler determinations is in excess of 350 mL. This is based on the observation that the normal upper limit of bladder volume is 500 mL for women and 700 mL for men. Indwelling Foley catheters should be reserved for patients for whom intermittent catheterization is difficult because of their size, presence of urethral trauma, infected sacral sores made worse by incontinence, or behavioral issues. Antiseptic Foley catheters as well as catheters coated with antibiotics suitable for chronic use have been examined; however, no good evidence exists regarding their efficacy in preventing UTIs. Studies examining prophylactic treatment with antibiotics in patients with indwelling Foley catheters have shown mixed results. The prophylactic use of antibiotics carries the risk of selecting for resistant organisms and is not recommended. Urinary tract antiseptics such as metenamine mandelate and metenamine hippurate, which are concentrated in the urine and metabolized to formaldehyde in an acidic urine, are bactericidal for all organisms and may have a limited role in the prevention of recurrent UTIs in selected patients requiring indwelling Foley or suprapubic catheters.

Time-prompted voiding schedules 30 minutes after meals and every 2 hours between meals while awake can decrease urge incontinence. Kegel exercises are appropriate for women with comorbid stress incontinence but have limited use in patients with neurologic injury. Condom catheters are useful for symptomatic management of urge incontinence in men without urinary retention.

Pharmacologic treatment of urinary retention due to internal urethral sphincter disinhbition can be initiated with $\alpha_1$ receptor antagonists, which exert their action specifically on $\alpha_{1A}$ receptors in the prostatic urethra and the vesicourethral junction. Such medications have also shown benefit in women with urinary retention due to stroke, TBI, or SCI.54 They are also useful in men with coexisting prostatic hypertrophy. Urecholine may be helpful in stimulating detrusor contractility but is relatively contraindicated in patients with cardiac disease.

Drugs with anticholinergic and antimuscarinic effects, such as oxybutynin and solifenacin, are often used to treat urge incontinence. They have limited ability to cross the intact blood-brain barrier; however, they should be used with caution in patients with strokes or other CNS disorders that can interfere with the blood-brain barrier because they may potentially interfere
with cognition, memory, and overall stroke recovery.

**Sexual Dysfunction**

Stroke may lead to complex issues concerning a patient’s sexuality. Altered self-esteem due to physical dependency on spousal assistance for self-care functions, depression with loss of libido, hemiparesis with inability to accommodate accustomed sexual positions, and medication side effects are all relevant contributing factors. Loss of libido may be due to depression, which is readily treatable. Impotence may be due to side effects of medications such as antihypertensive agents or due to antidepressants themselves. Altered self-esteem can be addressed by psychological counseling. It is important for the physician to broach the subject of sexuality with an open-ended question inviting the patient’s response, which can be done when probing for poststroke depression symptoms.

Sexual dysfunction following TBI is also common and has been widely reported. TBI is a disorder affecting younger, more physically active individuals. Bifrontal involvement may give rise to inappropriate sexual expression, pseudobulbar emotional lability that can interfere with sexual expression, apathy, and depression. Psychological counseling for the patient and his or her partner may help implement behavior modification techniques. Use of fluoxetine or sertraline may help with management of pseudobulbar affect. Antidepressants that are unlikely to affect sexual function, such as mirtazapine and bupropion, can be recommended.

Surveys of both men and women with SCI show that sexual function is lower in priority than concern for mobility, self-care independence, and bowel and bladder continence. Reflex erections in response to manual stimulation can be achieved by men with complete SCI but may require oral sildenafil, tadalafil, or vardenafil to allow penetration. Intraurethral pellet or intracavernous injection of alprostadil can also be used. Such methods are not likely to produce ejaculation. Semen collection for artificial insemination can be obtained in a fertility clinic with an anal electrode used to stimulate ejaculation. Women with preservation of sensitivity in the T11 to L2 dermatomes may have adequate psychogenic stimulation to provide vaginal lubrication. A water soluble lubrication jelly can also be recommended. Obstetrical counseling concerning the risks of pregnancy following SCI is appropriate to allow an informed decision prior to unprotected intercourse. Women with SCI above T6 are at risk for AD during labor and delivery.

**Falls and Fractures**

Every patient requiring neurorehabilitation is at risk for falls. Use of standardized falls risk scales such as the Berg Balance Scale are useful to quantify this risk and help prioritize safety intervention strategies and equipment. The Berg Balance Scale assesses balance in 14 tasks relevant to daily living such as sitting and standing unsupported, transferring between sitting and standing, picking up items from the floor, turning around to look over shoulders, and reaching forward with an outstretched arm. Each task is graded on a 4-point scale.

The neurorehabilitation team should allow as much independence as possible without the patient incurring a significant risk of falling. Patients with cognitive impairment, impulsivity, dyspraxia, or mixed expressive-receptive aphasia may not be able or willing to cooperate with safety precautions. Bed or wheelchair alarms, direct nursing or therapist observation of the patient...
when he or she is out of bed, wheelchair lap belts, and enclosed bed systems offer graded interventions to reduce falls. These safety interventions may not be tolerated by some patients and actually increase their risk of falling if they cause agitation or the patient struggles against them. Moving the patient to a private room and having a family member stay with the patient is sometimes feasible. Providing individual patient supervision is costly but should be provided if other measures fail. Psychiatric consultation may be requested when pharmacologic intervention is required to ensure patient safety. Many neurorehabilitation physicians are experienced in the use of psychoactive medications and manage patients who are agitated or confused without psychiatric consultation.

Special undergarments with impact-absorbing pads over the greater trochanters may reduce the risk of fractures.57 Patient compliance with their use is poor because of their bulky appearance and the mechanics of taking them on and off while toileting.

Evidence supports the use of bisphosphonates in patients following stroke to prevent fractures.58 Bisphosphonates are also commonly prescribed following SCI and in patients with severe TBI and limited mobility. Use of a calcium supplement with vitamin D is also reasonable for such patients.59

REFERENCES


10. Grommes TE, Huang CT. Orthostatic hypotension after spinal cord injury:


