Recognition and management of medical and specific associated neurological complications in stroke rehabilitation

John Schmidt, MD  
Cornell University Medical College at The Burke Rehabilitation Center  
White Plains, New York

Mike Reding, MD  
Cornell University Medical College at The Burke Rehabilitation Center  
White Plains, New York

Up to 38% of patients receiving inpatient rehabilitation following stroke will have emergent medical problems requiring transfer back to an acute care ward. A much larger number require treatment intervention for less emergent medical problems that interfere with rehabilitation efforts.

This review will describe the medical and neurologic problems that have come to be accepted as part of the rehabilitation process following stroke. A number of these medical complications can be anticipated and are either part of the comorbid medical problems that are prevalent in the stroke population or are complications due to stroke-related neurologic impairment. References are provided whenever available, otherwise the reader should assume that the observations and/or recommendations made are based on the authors' past experience or treatment preferences.

CARDIOVASCULAR COMPLICATIONS

Myocardial infarction, angina pectoris, cardiac arrhythmias, congestive heart failure
(CHF), hypertension, and peripheral vascular disease are all part of the atherosclerotic process of which stroke is but one manifestation.\textsuperscript{2-7} We expect the stroke patient admitted to our inpatient rehabilitation unit to have at least one or more of these comorbid medical problems. Stroke rehabilitation is a process of progressive mobilization and exercise. One must follow the patient’s responses to exercise with appropriate medication adjustments to ensure optimal care. Review of the patient’s prestroke cardiovascular history and exercise tolerance helps predict problems.\textsuperscript{5}

Two levels of risk are considered. A low-risk group consists of patients with a past history of myocardial infarction, angina pectoris, or congestive heart failure with only minimal residual limitation of exercise tolerance. A high-risk group is identified by an antecedent history of ischemic heart disease or congestive failure meeting criteria for class 3 or 4 functional disability using the New York Heart Association 0 to 4 scale.\textsuperscript{5} This usually precludes the possibility of an uncomplicated stroke rehabilitation program.

Patients having stroke following acute myocardial infarction are still considered for admission to the stroke rehabilitation unit because they would be expected to return to routine daily self-care and ambulation activities in the community within one to three weeks post acute myocardial infarction. The same is true for patients with stroke following coronary artery bypass grafting or valvular heart surgery. Assignment of low vs high risk is based on the presence of residual symptomatic congestive heart failure, exercise induced cardiac arrhythmias, or the presence of angina pectoris with minimal exertion.

A number of patients carry the diagnosis of congestive heart failure based on chest x-ray film evidence of cardiomegaly. Cardiomegaly is by itself a poor guide in predicting stroke rehabilitation complications. A past history of pulmonary edema or recent chest x-ray film evidence of venous congestion are more sensitive indicators of patients at high risk of CHF complications during stroke rehabilitation.

Patients with a past history of myocardial infarction, angina pectoris, congestive heart failure, or cardiac arrhythmias requiring medical management all are listed as requiring "cardiac precautions" during their therapy programs. This is simply a means of alerting the therapy team to pay special attention to exercise-related changes in blood pressure, pulse, and respiration. If the patient’s vital signs do not return to baseline levels within three to five minutes of a specific exercise task, the task has been too stressful.\textsuperscript{6} Subsequent tasks are made less demanding. Body weight is recorded once or twice each week. Patients at risk for orthostatic hypotension are checked for both initial and delayed blood pressure responses to change in position. Patients with a history of cardiac arrhythmia or evidence of abnormal pulse rate response to exercise have a 24-hour Holter monitor study. This is done to document the safety of the patient’s exercise program.\textsuperscript{5} It is also used to ensure that concurrent cardiac medications are effectively controlling any rate or rhythm abnormalities.

With the above-mentioned screening criteria and patient surveillance measures in place, the following cardiovascular complications arise in an inpatient stroke rehabilitation population. These figures represent an estimate as seen on a 30 bed inpatient stroke rehabilitation unit caring for approximately 150 patients with stroke each year.

Symptomatic orthostatic hypotension occurs in about 20% of patients. This is seen most often within the first two weeks of the rehabilitation program. As long as the patient is in bed,
the blood pressure seems well controlled. With increased time spent sitting, standing, and walking, there may be progressive venous pooling of blood in the lower extremities with resultant hypotension. Treatment options are: reduce any antihypertensive medications, ensure adequate hydration, reduce then progressively increase the patient's length of time out of bed.

The second most common cardiovascular management problem is hypertension and the need for increased antihypertensive medications.\(^{10}\) This problem is usually seen later in the rehabilitation process when the patient's self-care and ambulation function have shown maximal recovery. This is thought to represent a secondary effect of exercise and mobilization on blood pressure—an effect opposite that of bedrest.

Approximately 5% of patients require an upward adjustment in their antianginal medications. This is usually seen when patients are moving from the mat and pre-gait level of their rehabilitation program to ambulation and stair climbing activities. Hemiplegic patients propelling their wheelchairs with their unaffected extremities expend approximately one third less energy compared to normal subjects walking at the same speed.\(^{11}\) Patients walking with a quad cane and ankle brace move considerably slower than normal individuals but expend approximately the same amount of energy per unit time. They may expend up to twice as much energy to cover the same distance.\(^{12,13}\) The energy expenditure per unit time is the same for hemiplegic patients climbing stairs as for normal individuals.\(^{14}\) The energy expenditure per step is, however, approximately one-third greater than that required for normal individuals.\(^{14,15}\) It is the energy expenditure per unit of time that is most relevant. As long as patients are allowed to move at their self-selected speed, the energy demands for hemiplegic ambulation and stair climbing activities do not appear to exceed those of the normal population. Comparative energy expenditure data for patients involved in daily activities with speed and time constraints such as crossing at a light or accommodating the pace of a crowd in a hallway are not currently available.

Approximately 3% of patients experience increased congestive heart failure. This is usually managed with further restricting dietary salt intake and increasing oral diuretics. Congestive heart failure with pulmonary edema is seen rarely (less than 1%) reflecting the selection criteria and monitoring techniques described above. The same prevalence is also noted (less than 1%) for myocardial infarction.

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Cardiac arrhythmias requiring medical intervention are seen in approximately 10% of the patient population. The most frequent arrhythmia seen is atrial fibrillation. During the acute stroke phase, when patients are primarily bedbound, their ventricular response rate may appear well controlled even without digoxin. When mobilization is attempted the ventricular response rate may accelerate unacceptably. Such exercise-related ventricular response rates are best documented with a 24-hour Holter monitor recording. This allows calculation of the average ventricular rate as well as the slowest and fastest rates seen during a 24-hour period. Based on this, the dosage of digoxin is raised or lowered. The use of ancillary negative chronotropic agents such as propranolol or verapamil is based on an assessment of the
Holter monitor study as well as blood digoxin levels. We advance the patient's digoxin dosage until the blood level reaches 2.0 ng/ml or the patient has unacceptable resting bradycardia. If the patient's ventricular response rate is not adequately controlled with this dosage of digoxin, then propranolol or verapamil are added. The patient must be observed for worsening of any underlying congestive heart failure with the addition of either of these two medications. Premature ventricular contractions, "R on T" couplet beats, and unsustained ventricular tachycardia are seen in approximately 10% of the Holter monitor studies obtained for the above-mentioned indications. When "R on T" couplet beats or unsustained ventricular tachycardia are observed, cardiac consultation is requested for an expert opinion concerning the need for antiarrhythmic agents. Medical intervention is usually not recommended unless the patient has rate-related symptoms. If treatment intervention is undertaken, then follow-up Holter monitor studies are obtained to document the efficacy of the medication given.

Peripheral vascular disease is rarely seen as a problem impeding the stroke rehabilitation process. As mentioned above, the ambulation speed of the stroke patient is considerably reduced from that of the normal individual. If the patient was ambulatory for several blocks without claudication prior to stroke, then peripheral vascular disease is not likely to interfere with hemiplegic ambulation poststroke. Patients with cardiogenic thromboembolic strokes will occasionally have ischemic leg pain at rest but this is usually associated with obvious ischemic changes in the skin over the foot and toes. Peripheral vessel disease can also be a factor contributing to poor wound healing of pressure sores on the lateral malleolus or heel of the paretic leg. Medical management with pentoxifylline or vascular surgical intervention are considered based on the urgency of the clinical situation.

DEEP VEIN THROMBOSIS AND PULMONARY EMBOLISM

The prevalence of deep venous thrombosis (DVT) varies widely from one study to the next based on the screening criteria used to select patients for study and on the diagnostic technique used to confirm the presence of venous thrombosis. The question of DVT as a complication following stroke is further confounded by the fact that calf vein DVT rarely leads to pulmonary embolization.16,17 There is general agreement that thigh vein DVT is clinically relevant and may lead to life-threatening pulmonary embolization. The highest prevalence of venous thrombosis is seen with studies using 125I labeled fibrinogen as a marker for clot formation.18,19 McCarthy et al., using this technique, found a prevalence of 75% for either calf or thigh vein DVT in patients following stroke.18 Cope et al., using venography, found evidence of acute DVT in 19% of patients three months poststroke.17 They reported a prevalence of pulmonary embolization of 2.7% in the same population. Serial plethysmography is probably the most cost-efficient way of monitoring for DVT in the stroke population.20 Unlike venography, it is noninvasive. Unlike the 125I fibrinogen technique, it does not require costly nuclear medicine radiopharmaceuticals or nuclear medicine imaging systems. Plethysmography requires a minimum of equipment and can be performed by a skilled technician. It can be performed at the bedside and appears to have an accuracy in excess of 95% for detecting thigh vein thrombosis.

A number of clinical features have been thought to be associated with an increased risk
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of DVT: limb edema, tobacco use, obesity, muscle flaccidity, and length of bedrest. These clinical factors for DVT are so insensitive as to be clinically useless.

In spite of the above controversies, it is universally accepted that the hemiplegic patient is at significant risk of both DVT and pulmonary embolization (PE). The American Medical Association has published recommendations that all patients at risk for either DVT or pulmonary embolization receive graded compression stockings. They recommend thigh length stockings. These are difficult to put on, extend to the diaper area and are easily soiled, and are frequently rejected as uncomfortable by patients. Knee length thromboembolic hose may be an acceptable compromise.

In an effort to develop some rational criteria for how long to continue anti-thromboembolic therapy following stroke, the authors studied the prevalence of these complications in patients at different levels of ambulation recovery. The risk of DVT or PE decreased twofold for patients walking with quadcane and ankle brace compared to patients who were still at mat or pregait levels of ambulation recovery. This decreased risk was present irrespective of the distance walked or the amount of therapist assistance required. Patients who were ambulatory but still required support of a rigid hemibar were at a 4.5 times lesser risk of DVT or PE compared to patients who were still at mat or pregait activity levels. These data indicate that patients should be assisted to ambulate as soon as possible following stroke with whatever bracing, assist device, or therapist assistance is needed. Patients who are ambulatory with a quad cane and ankle brace may no longer require DVT prophylaxis. Patients who are still at mat or pregait activity levels are at greatest risk for DVT or PE. These patients are considered for low-dose warfarin therapy or mini-dose subcutaneous heparin. Critical prospective studies looking at the efficacy of low-dose warfarin or mini-dose subcutaneous heparin for DVT and PE prophylaxis in the stroke population have not been performed.

DIABETIC MANAGEMENT

One third of the patients on our stroke rehabilitation unit are diabetic. Approximately half of these will be taking insulin for management of adult onset diabetes mellitus. Patients and their families will have been instructed in split dose insulin regimens or sliding scale regular insulin administration. Following stroke, however, the patient’s insight into diabetic management may be altered. The patient’s ability to recognize impending hypoglycemia is often compromised. If expressive language deficits are present, the patient will have difficulty communicating needs to therapy staff. Hemianopsia, dyspraxia, and stroke-related cognitive impairment frequently preclude patients from drawing-up and administering their own insulin dosage. Dysphagia can complicate attempts at correcting impending hypoglycemia with oral glucose loading. There is a need to manage the patient’s blood glucose as tightly as is safe and practical. This is usually achieved with a fixed split-dose morning and evening insulin administration schedule. The goal is to have fasting, noon, 5 PM, and 10 PM blood sugars between 100 mg and 200 mg percent.
With this regimen less than 1% of patients will develop symptomatic hypoglycemia that cannot be corrected with oral glucose loading. Ketoadicosis and nonketotic hyperosmolar state have not occurred with this diabetic regime in place.

UROLOGIC MANAGEMENT

Incontinence is a common problem following stroke. Brocklehurst et al. concluded that incontinence was due to the patients' decreased mobility, not being able to transfer on and off the toilet without waiting for others to come and assist them. They report that after one year the prevalence of incontinence among patients with stroke was similar to that of the general elderly population. This appears to be an overly simplistic view of incontinence following stroke.

Studies by Reding et al. indicate that incontinence poststroke is probably due to more than just difficulty with performing motor functions. Less than 10% of patients with pure motor hemiparesis were found to have urinary incontinence. More than 70% of patients with hemiparesis plus somatic sensory deficits plus homonymous hemianopsia were incontinent. Reding et al propose that parietal association area stroke involvement is probably the most essential neurologic impairment associated with the presence of urinary incontinence. Urologiccystometrogram/electromyogram (CMG/EMG) studies in patients with poststroke incontinence show impaired sensation of filling and increased detrusor irritability. These findings are most compatible with what Blavis calls a "failure to store" syndrome. Patients with brain stem involvement particularly at the medullary level may show CMG/EMG abnormalities compatible with detrusor-sphincter dyssynergy. This latter syndrome is rather unusual in the stroke population and is more classic for patients with spinal neurologic problems. In a prospective study of patients with unilateral supratentorial ischemic stroke, Reding et al found that 3 of 45 patients one month poststroke had anatomic large volume bladder capacity characterized by "failure to empty." These patients did not show detrusorsphincter dyssynergy. These patients were started on intermittent catheterization to decompress the bladder. By three months poststroke, all three had developed a more classic small volume bladder capacity with detrusor irritability no longer requiring catheterization.

The cause of urinary incontinence following stroke can usually be documented with a postvoid bladder catheterization study to measure the volume of urine retained after a maximum effort to void. This specimen is sent for urine analysis and culture to identify any associated urinary tract infection. If urinary retention is observed, one should check with the nursing staff to make sure that the patient was indeed given a maximum attempt to void on the toilet. Use of a bedpan is a nonphysiologic situation and is frequently associated with artificially elevated residual urine volume determinations. Occasionally, patients will have such poor sitting balance that they are not able to sit on a toilet with armrests. Such patients may benefit from a fully padded commode chair with extended reclining backrest, armrests, and elevating leg rests. In such a commode chair the patient has a sense of security and can void in a more physiologic manner than when perched atop a bed pan in a hospital bed. If urinary retention is documented with retained volumes in excess of 100-150 cc, consideration is given to an intermittent catheterization schedule. It is rare for patients with hemispheric stroke to require intermittent
catheterization beyond one month poststroke. Those that do usually have some confounding problem such as diabetic autonomic neuropathy or significant prostatic hypertrophy which complicate control of bladder function. An indwelling Foley catheter is reserved for patients who have actual or impending decubitus ulcer formation. The time course for recovery of bladder control following stroke is favorable. Those still having difficulty with urinary urge incontinence can be managed symptomatically with timed-prompted voiding regimens, evening fluid restriction, and incontinence pads. External condom catheters for men and several recently marketed custom molded cup systems for females can also be recommended.

PRESSURE SORES

Patients with impaired mobility are at constant risk of pressure sore formation. This risk is significantly heightened by associated urinary incontinence, sensory impairment, cognitive impairment, and the presence of contractures or other musculoskeletal constraints on patient positioning. Textbooks will frequently make the comment that pressure sores are preventable. This is theoretically true, but, given the constraints of practical patient care, one must acknowledge that they will occur and that their prevalence is related to the severity of the patient's neurologic impairment, the chronicity of the neurologic disorder, the patient's underlying nutritional status, and the intensity of nursing support available. Pressure sore formation has been estimated to occur in 3% to 45% of chronically institutionalized patients with immobility due to neurologic impairment.

The incidence of pressure sores is not surprising given the results of animal studies. For example, a constant compressive force of 70 mm Hg for two hours is sufficient to cause microscopic cellular damage in rat muscle tissue. Compressive forces are most problematic over bony prominences such as the lateral malleolus, heel, greater trochanter, and ischial tuberosities.

Shear forces must also be considered in the etiology of pressure sore formation. Clinically relevant shear forces are generated when superficial fascia slides over deeper fascia that is firmly attached to bone. These shear forces stretch tissues and vessels resulting in local ischemia. Shear forces are most relevant for pressure sore formation over the sacrum in patients positioned in a semirecumbent position in their beds.

Stroke patients who are unable to roll from side to side in bed are placed on a waterbed mattress. Nursing staff routinely reposition the bedridden patient on a two-hour schedule. Attention is given to daily inspection of the sacrum, ischial regions, skin over the greater trochanter, lateral malleolus, and heel. Heel protectors and sandbag positioning to prevent external rotation of the plegic lower extremity are part of routine nursing care of bedridden patients.

Photographs are taken of all areas of pressure sore formation. Reference marks are made on the skin to help with quantification of wound healing using serial photographs. Wound tracings have also been advocated to objectify wound healing. Treatment of pressure sores focuses attention on the patient's general nutritional status ensuring adequate protein intake and proper hydration. Proper positioning of the patient to avoid pressure over the wound is stressed. Recent controlled studies have shown "hydrocolloid dressings" to be more effective than wet-to-dry dressings. Hydrocolloid dressings appear to promote faster wound healing and require less nursing time as these
dressings are applied for three to four days at a time.\textsuperscript{43,44}

If the above measures are not associated with documented wound healing, then the patient is placed on an air-fluidized bed. Allman et al\textsuperscript{45} have shown a fivefold improvement in pressure sore healing for patients in air-fluidized beds compared to those receiving conventional therapy. Conventional therapy was defined as every two-hour repositioning, heel and elbow protectors, and a vinyl alternating air mattress covered by a 19 mm thick pad over a standard hospital bed mattress. They found that the air-fluidized beds were mildly bacteriostatic and were associated with significantly less shear and compressive forces over bony pressure points. The expense of the air-fluidized beds, the need for staff training in their maintenance, and their disinfection are the major limitations to their use. They are reserved for patients not responding to more cost-efficient measures. Using the above techniques, the authors have not had patients develop pressure sores that have required surgical debridement or surgical closure.

**FALLS**

Falls are a consequence of attempts to mobilize patients with neurologic impairment. Falls cannot be eliminated without sedating the patients and restraining them. Restraints alone often increase a patient’s agitation and struggle to be free. Most rehabilitation units acknowledge that attempts at mobilizing neurologically impaired patients will be associated with patient falls. Risk factors that have been identified for falls are obvious: cognitive impairment, perceptual deficit such as hemianopsia or hemihypesthesia, muscle weakness, coordination problems, and dyspraxia.\textsuperscript{47-49} Virtually all patients on a neurological rehabilitation unit by definition are at risk of falling. A judgment based on observation of the patient’s ability to transfer from bed to wheelchair, transfer from wheelchair to toilet and back, and walk about within the hospital room provide an initial assessment of a patient’s likelihood of falling. During the initial phase of the rehabilitation program, there is probably a bias to error on the side of being overly protective. For patients considered to be at risk of falling, orders are written to have them up with assistance only, to have wheelchair lap restraints in place, and bedrails up when patients are in their beds. None of these measures prevent falls. They simply represent easily tolerated constraints on the patient’s activity. The wheelchair lap restraint, for example, can help to remind patients of their need for assistance when they attempt to transfer from their wheelchair to other surfaces. Bedrails help the patient who awakens at night and who would otherwise attempt to get out of bed and walk to the bathroom without assistance. Most of the falls observed on our unit occur when patients do not understand or appreciate the need for their restraints. These patients will remove their wheelchair lap restraints or crawl over the bedrails. Sedative medications that render patients more passive probably only add to the patient’s confusion and further impair safety awareness. Asking a family member to function as a private duty sitter for such a confused patient may serve as a short-term way of managing patient safety awareness problems. The cost of private duty

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sitters as routine management of such problems on a rehabilitation ward makes this an unrealistic long-term treatment option.

Rehabilitation units without falls are probably overly restrictive in encouraging independent patient mobility. Rehabilitation units with excessive falls are probably lax in this respect. There is no agreement concerning the minimal prevalence of falls compatible with meaningfully aggressive patient mobilization following stroke. Fear of litigation fosters an overly restrictive approach to encouraging patient independence.

Falls appear to be equally prevalent in patients with left hemisphere strokes as well as right hemisphere strokes. Fractures are more prevalent in older stroke victims. When hip fractures do occur they are seen almost exclusively in the paretic leg. Women are more prone to hip fractures than men. This reflects underlying osteoporosis associated with the postmenopausal state.

SPECIFIC ASSOCIATED NEUROLOGICAL COMPLICATIONS

Isolated Shoulder Pain

Isolated shoulder pain may be seen in 72% of patients with unilateral hemispheric stroke still requiring inpatient rehabilitation two months following stroke. Questioning the patient or family may indicate that pain is due to exacerbation of problems antedating the stroke such as trauma, bursitis, tendinitis, or arthritis. An x-ray film of the shoulder compatible with calcific tendinitis or arthritis can be of diagnostic benefit. Preservation of reflexes, spasticity, and absence of muscle atrophy can be used to exclude pain due to traction plexopathy. Isolated shoulder pain not due to the aforementioned etiologies is usually ascribed to capsulitis, tendinitis, or dystonic muscle pain. Interobserver diagnostic variability has not been studied. Prophylaxis-treatment strategies are directed at specific etiologies when present, and/or to shoulder protection, support, range of motion, thermal and electrical modalities, and use of nonsteroidal antiinflammatory agents.

Shoulder-Hand Pain Syndrome

Shoulder-hand pain syndrome (SHS) is a form of reflex sympathetic dystrophy with pain in the paretic shoulder, wrist, and hand. The pain is associated with edema and evidence of autonomic dysfunction. Cross-sectional studies have shown that shoulder-hand pain syndrome occurs in about 41% of patients with unilateral hemispheric stroke still requiring inpatient stroke rehabilitation two months following stroke. The prevalence increases to 80% in patients who have associated hemiparesis, hemihypesthesia, and hemianopsia.

Steinbrocker and Argyros published the first clear description of SHS and described three stages in its evolution. Stage 1 is characterized by hand edema, pain, and evidence of autonomic dysfunction such as altered sweating or skin color changes due to altered capillary flow. Stage 1 usually does not develop until a month or more poststroke and may last three to six months. Stage 2 is associated with the above but is characterized by the onset of atrophic skin changes and Sudeck's atrophy of bone seen on x-ray film. Stage 2 follows Stage 1 and may also last three to six months. Stage 3 is the pain resolution phase with all of the above findings but with end-stage contractures. No longitudinal studies have been reported so it is not known how many patients with Stage 1 manifestations will improve and how many will progress to Stage 2. All studies of the relationship between glenohumeral subluxation
and development of SHS have been cross-sectional studies.  
Caillet has proposed that subluxation with chronic traction on the shoulder ligaments and capsule causes soft tissue ischemic-inflammatory changes which later give rise to pain. If Caillet’s hypothesis is true, then one would not expect cross-sectional studies to show a correlation between subluxation and pain. Studies done during the first month, when flaccidity is most prominent before Stage 1 develops, would show maximum subluxation and no pain. Studies done during Stage 2 would show much pain but little subluxation due to advancing shoulder spasticity. Until data are available from longitudinal studies it is prudent to continue directing one’s efforts to adequate shoulder-hand support and range of motion.

Methods of preventing or treating shoulder-hand syndrome are empiric and no systematic approach to study the effectiveness of therapeutic intervention has been conducted. Several pharmacologic treatments have been recommended based on anecdotal patient studies. Nonsteroidal antiinflammatory drugs such as ibuprofen are prescribed for symptomatic management of associated musculoskeletal pain. Oral prednisone from 40 to 60 mg per day tapered over two to three weeks has been recommended for patients not responding to more conservative management. Stellate ganglion blocks have been suggested for patients with severe residual pain who have not responded to oral prednisone administration. Shoulder subluxation, finger, hand, and wrist edema have been managed with either wheelchair-based or patient-based support systems. The two most common wheelchair-based support systems are either the lap-board or arm-trough. Foam wedges can be used to elevate the hand to approximately the level of the right atrium when the patient is seated in his wheel-

chair. If such support and elevation is not enough to reduce hand edema then Isotoner or Jobst gloves can be added. Newer alternatives, such as continuous passive movement systems, have produced up to a 30% reduction in hand edema in controlled prospective studies. Enthusiasm for patient-based shoulder-hand support systems has varied with time. The two most popular shoulder slings have been the Bobath and the Hemicuff slings. The Bobath sling consists of a small soft foam rubber roll under the axilla with cloth straps over the shoulder and around the chest to elevate the humeral head into a normal position in the glenoid fossa. The Hemicuff sling consists of a cloth “cuff” applied to the upper arm, held by cloth straps over the shoulder and around the chest to elevate the shoulder and bring the humerus into a more natural position. Wheelchair-based systems are more appropriate for patients largely confined to wheelchairs. The patient-based supports are most appropriate for those who are independent with ambulation.

Hemianopsia and Visual-Spatial Neglect

Unilateral visual neglect and homonymous hemianopsia, which will be addressed in greater detail elsewhere in this issue, are common in the stroke population and can inhibit the patient’s rehabilitation progress. These visual perceptual deficits are not only an impediment to function but also present safety hazards. Treatment approaches to hemianopsia and visual-spatial neglect have stressed techniques

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that force the patient to search into the affected visual field. Optical aids such as wide angle lenses, Fresnel prisms, and mirror attachments worn on the patient’s glasses have all been advocated.\textsuperscript{68-72} Fresnel prisms have been subjected to a controlled trial and appear beneficial for management of these visual perception deficits interfering with table-top activities.\textsuperscript{71} Pharmacotherapy with bromocriptine, a dopamine agonist, has been used to treat visual-spatial neglect but is still experimental.\textsuperscript{73}

**Seizures**

Poststroke seizures are relatively rare on the inpatient stroke rehabilitation unit. Seizures usually occur during the first several days following stroke or from three months to two years poststroke.\textsuperscript{74-77} Delayed onset of seizures may reflect neural changes associated with maturation of the cortical infarct.\textsuperscript{78} Overall, approximately 10% of patients with stroke will have subsequent seizures.\textsuperscript{79} Up to 40% of patients with cortical infarction may have seizures.\textsuperscript{80} Less than 5% of patients with isolated subcortical infarction will have seizures.\textsuperscript{80} Seizures are usually focal motor in type with secondary generalization or are complex partial in type. The drug of choice is either phenytoin or carbamazepine. An oral loading dose of phenytoin with approximately 900 mg given over the first 24 hours in divided dosages is usually sufficient to initiate anticonvulsant therapy. Intravenous diazepam is not given unless the patient has recurrent seizure activity without recovery of consciousness.

**Depression-Neuropsychiatric Disorders**

Depression is seen in 50% of patients following major hemispheric stroke.\textsuperscript{81} It can significantly impede rehabilitation progress.\textsuperscript{82} Some studies view poststroke depression as a form of grief reaction secondary to catastrophic illness.\textsuperscript{83-88} Human autopsy studies and animal models of stroke have shown depletion of noradrenaline and serotonin monoamine transmitters in both hemispheres ipsilateral and contralateral to the stroke.\textsuperscript{89,90} If one accepts the prevalent monoamine deficit hypothesis for depression, then depression following stroke is clearly more than a simple grief reaction. The signs and symptoms of depression seen in patients following stroke are as follows: emotional lability, frustration, anger, anorexia, constipation, insomnia, poor motivation, apathy, withdrawal, and pessimism.

Pharmacotherapy has been shown to be effective in improving depressive symptomatology following stroke.\textsuperscript{91} Antidepressant therapy has also been shown to improve measures of functional outcome following stroke rehabilitation. Two double-blind controlled studies have been conducted, one using nortriptyline and the other using trazadone for management of poststroke depression.\textsuperscript{92,93} Both appear to be beneficial and well tolerated. Trazadone may have theoretical advantages, having less anticholinergic activity and less effect on cardiac conduction pathways.\textsuperscript{93}

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There are a number of medical neurologic complications that are associated with the stroke rehabilitation process. They are important to recognize because they can inhibit progress in stroke rehabilitation and limit overall functional outcome.
REFERENCES


