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Physiatry and Acquired Brain Injury

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Introduction

Physiatrists are specialists who focus not only on the disease process but also on the secondary effects that may occur as a result of the disease process. We utilize a biopsychosocial model that is unlike conventional medicine, which tends to focus on the diagnosis and treatment specifically geared toward the disease process (biomedical model) (Stiens, 2002). The underlying principle is based on treating each patient as a "whole." Rehabilitation medicine takes physical, emotional, and social needs into account when formulating a treatment plan. The physiatrist utilizes therapeutic exercises and physical agents in addition to medications to treat patients. The role of the physiatrist is to restore a patient's overall quality of life. Our emphasis is on maximizing a patient's functional capabilities.

Historical Perspective

Rehabilitation medicine dates back as far as World War I. It was during this era that physicians began to incorporate physical agents to help rehabilitate injured and disabled soldiers in what was known as "reconstruction hospitals." In 1926, Dr. John Stanley Coulter made it possible for physical medicine to be a part of formal education in medical school. Dr. Coulter joined the faculty of Northwestern University Medical School as the first academic physician in physical medicine and was the first to initiate a training program for physicians (Association of Academic Physiatrists, 1999).

In 1936, Dr. Frank Krusen established the first 3-year residency training program in physical medicine at Mayo Clinic. Dr. Krusen was the first to coin the word "physiatrist" to describe the physicians who applied physical medicine to treat various neurological and musculoskeletal ailments.

In 1942, Dr. Howard A. Rusk, as Chief of Army Air Forces Convalescent Training Program, demonstrated how rehabilitation allowed injured soldiers to be reinstated to duty and disabled soldiers to return back home at a functional level. He believed in an aggressive approach to rehabilitation medicine and advocated early ambulation, physical therapy, and activities of progressive intensity supplemented with psychological support (Association of Academic Physiatrists, 1999). After World War II, the daunting task of helping the thousands of disabled soldiers return to their previous way of life led to a realization and the eventual recognition of the importance of rehabilitation medicine. This led to a surge in the development of this field of medicine.

In 1947, the Advisory Board of Medical Specialties declared Physical Medicine and Rehabilitation (PM&R) as a specialty of medicine. Since then it has been a rapidly growing specialty. PM&R residency entails one year of internship (medicine or surgery) and three years of training in the field of PM&R. Today there are up to 80 ACGME accredited residency training programs in the field of rehabilitation medicine.

The Role of the Physiatrist

It is the physiatrist's role to identify a patient's physical deficits as well as the functional impact of these deficits in order to better highlight a patient's impairment, disability and handicap. The World Health Organization defines impairment as "any loss or abnormality of psychological, physiological or anatomical structure or function"; disability as "any restriction or lack of ability, resulting from an impairment, to perform an activity in the manner or within the range considered normal"; and handicap as "a disadvantage for a given individual, resulting from an impairment or a disability, that limits or prevents the fulfillment of a role that is normal for that individual in the community." By identifying these three components of functional assessment, the physiatrist can construct a treatment plan that can help to minimize the impact of the impairments in day-to-day life.

This holistic way of treating patients requires an interdisciplinary approach. The multifaceted nature of the clinical consequences of acquired brain injuries make the interdisciplinary team approach the most appropriate strategy for treatment (Roth & Harvey, 2000). The rehabilitation team typically consists of the physiatrist, physical therapist, occupational therapist, recreational therapist, a rehabilitation nurse, speech therapist, neuropsychologist, dietitian, and social worker. In an interdisciplinary team approach, the patient's progress with each discipline is communicated through team meetings which are led by the physiatrist. Team meetings are scheduled on a regular basis. These meetings allow the members of the rehabilitation team to establish goals in order to provide patients with a unified, coordinated treatment plan. The team will determine the most appropriate disposition (home versus an alternate living facility), establish a discharge date and identify services the patient may need upon discharge. Good communication skills among the members of the team are required to construct a rehabilitation program that is individualized for each patient. The overall goal of comprehensive rehabilitation is to optimize a patient's quality of life and achieve maximal independent functioning.

The Physiatrist's Role in the Acute Care Hospital

A study done by Wagner et al. (2003) showed that "early physical medicine and rehabilitation consultation positively impacts upon functional status and length of stay for patients with traumatic brain injury during acute hospitalization". Rehabilitation should begin as early as possible to provide stimulation and prevent further complications.

Physiatrists will often be consulted during the patient's acute hospitalization. Early intervention is crucial. Clinical trials have shown that early initiation of therapy results in a more favorable outcome (Brandstater, 1998). Early rehabilitation consultation provides an opportunity to educate other members of the health care team about rehabilitation issues.

Mr. Smith, a 39-year-old with no significant past medical history, was an unrestrained driver involved in a motor vehicle accident. He sustained loss of consciousness for about 30 minutes. Upon regaining consciousness he had a Glasgow coma score of 11 on the field. On admission to the hospital, imaging was positive for a left subdural hematoma. He was placed on phenytoin (Dilantin) for seizure prophylaxis and haloperidol (Haldol) for agitation by the medical team. Three days later, physiatry was consulted. The evaluation involved assessing the severity of the brain injury and prognosis as well as the patient's impairments and functional status. Mr. Smith was found to have both motor and cognitive deficits, but he was able to follow commands. As per the initial GCS score, Mr. Smith's injury was classified as a moderate traumatic brain injury. Based on the evaluation, he was deemed to be a candidate for acute inpatient rehabilitation in the brain injury unit.

The initial consultation provides an opportunity for the physiatrist to structure the rehabilitation program while the patient is on the acute medical floor. Therapists must be apprised of any precautions the patient may have. This can include cardiac, pulmonary, fall, and sensory precautions. The consultation should include a thorough physiatric history and physical exam including a thorough functional assessment. Recommendations are made by the physiatrist, which focus on minimizing the patient's impairment or disability (Stiens, 2002). As a consultant, the physiatrist helps to identify, treat, and prevent problems such as contractures, pressure sores/ulcers, bowel and bladder dysfunction, heterotopic ossification, and spasticity.

Some concern about massed practice in the first few days after stroke has been raised after the experimental finding in rats that the size of the infarct increased or perhaps more neurons were damaged secondary to early overuse of an affected, paretic limb (Dobkin, 2004). However, the author noted that "the level of exercise of a rat running on a rotating wheel is much greater than that a patient could possibly experience" (Dobkin, 2004). Generally, exercise by rats that was initiated several days after an induced stroke had positive effects on mechanisms of plasticity, such as production of brain-derived neurotrophic factor (BDNF) (Dobkin, 2004).

The preventative aspect of the rehabilitation consult should emphasize issues such as lifestyle changes and medical treatment for secondary stroke prophylaxis as well as prophylaxis for deep venous thrombosis. Encouraging early range of motion while the patient is on the medical floor helps to prevent contractures. Nurses should be made aware of the importance of weight shifting and frequent position changing to prevent pressure sores. Staff should also be educated on aspiration precautions in the presence of dysphagia and techniques that should be used when assisting the patient with meals.

Rehabilitation of the Patient with Acquired Brain Injury

Acquired brain injury (ABI) is a diagnostic category that includes traumatic brain injury, anoxia, stroke, infection, toxic-metabolic injury, and brain tumors. Many patients share a similar clinical course regardless of the etiology of the brain injury. The clinical course, which begins with global impairment of brain function, goes through a period of functional recovery and ends in a stable level of functioning with no further deterioration, is the basis for treating patients with different etiologies of injury in the same rehabilitation program.

ABI affects all age groups and poses a unique challenge to the rehabilitation professional. The effective management of ABI patients requires an understanding of the physical and cognitive impairments that may be seen. ABI can be divided into primary injuries, occurring at the moment of impact, and secondary injuries, which begin after the trauma and continue indefinitely. Secondary injuries occur as a result of the injuring event (Elovic et al., 2004). The injury may be further classified as focal (for example, contusions) or diffuse (diffuse axonal injury or DAI). Issues specifically related to stroke (CVA) and the rehabilitation of stroke survivors will be covered later in this chapter.

Common ABI-Related Impairments

The patient with ABI may be left with a combination of physical and neurobehavioral impairments. These interact to produce a broad array of handicaps and disabilities that may persist long after the injury. The pattern of deficits seen in ABI varies greatly from person to person, based on the severity of injury, location and nature of the brain injury, and medical complications (Whyte et al., 1998). However, deficits in cognition are nearly ubiquitous after moderate or severe ABI (Whyte et al., 1998). Changes in behavior, mood, and personality are frequently seen. Other common impairments that must be addressed after ABI include cranial nerve injuries; sensory deficits; increased muscle tone and contractures; motor disturbances; vestibular dysfunction; visual impairments, including oculomotor and accommodative dysfunction as well as visual field loss; dysphagia; dysarthria; aphasia; apraxia; and bladder and bowel dysfunction.

Upon admission to the brain injury unit, Mr. Smith was noted to have impaired speech, moderate to severe cognitive deficits, dysphagia, and dense right-sided weakness. He was *at a cognitive functioning level of IV on the Ranchos Los Amigos Scale. His functional assessment on admission revealed that he had limited active range of motion on the right side of his body. He required maximum assistance with transfers and he was unable to ambulate. Mr. Smith was started on a comprehensive rehabilitation program that included rehabilitation nursing, dietary evaluation, physical therapy, occupational therapy, speech therapy, recreational therapy, and neuropsychological therapy.*

During the initial team conference, an appropriate rehabilitation program was constructed in an interdisciplinary manner by all the members of the team under the guidance of the physiatrist to address Mr. Smith's impairments. Initial functional independent measure scores were recorded by each discipline. Based on the initial evaluation, goals were set by each discipline.

A bedside swallowing evaluation performed by the physiatrist on admission revealed signs of dysphagia. A full evaluation was completed by the speech pathologist. The modified barium swallow test revealed penetration of thin liquids. As a result, Mr. Smith was placed on a soft diet with thickened liquids.

While on the medical floor, Mr. Smith had developed a Stage I decubitus ulcer in the buttock region and right heel. On the brain injury unit, nurses were instructed regarding weight shifting and placement of pressure relief ankle foot orthoses while in bed. A gel-filled cushion for Mr. Smith's wheelchair was ordered by the occupational therapist to ensure pressure relief as well.

Predictors of Outcome Post ABI

Brain injuries are classified as mild, moderate, or severe based on the Glasgow Coma Scale score (see Chapter 2, Table 2.1). The GCS score is one of the best indicators of the severity of brain injury and it is a good predictor of outcome post-ABI. The duration of post-traumatic amnesia (PTA) has also been used as an index of injury severity and predictor of outcome (Russell, 1932). PTA is described as the time when patients are out of coma but are disoriented and amnesic for day-to-day events. There is little to no carryover from one day to the next while a patient is said to be in PTA, which typically lasts four times the length of coma. The duration of PTA is measured from the onset of ABI to the resumption of ongoing memory, so the duration of coma is included. The Galveston Orientation and Amnesia Test (GOAT), developed by Harvey Levin and colleagues, is a standard technique for assessing PTA. It is both reliable and objective. GOAT scores range from 0 to 100, with a score greater than 75 defined as normal, a score of 66 to 75 defined as borderline, and a score less than 65 defined as impaired. The period of PTA has ended when a patient achieves a score greater than 75 for 2 consecutive days.

Other early predictors of severity and outcome include age, pupillary and motor response (both are components of GCS), and the presence or absence of intracerebral lesions (Watanabe et al., 2003). Generally, these are better at predicting survival than eventual outcome. Patients that experienced a compromise in hemodynamic stability, oxygenation, or maintenance of adequate cerebral perfusion pressure, typically have a poorer functional outcome.

Stages of Recovery Following ABI

The stages of neurobehavioral recovery from anoxic brain injury, anterior communicating artery aneurysm rupture, and many other nontraumatic brain injuries are similar to those of ABI (Boake et al., 2000). The Levels of Cognitive Functioning scale was developed at Rancho Los Amigos Medical Center (Malkmus et al., 1980) to describe the sequence of neurobehavioral recovery from ABI and to provide a rationale for cognitive rehabilitation at each recovery stage. In reality, recovery from ABI is much more variable, but the scale is still useful for distinguishing major stages of recovery and determining appropriate rehabilitation strategies.

Neuroplasticity and the Rehabilitation Process

The main goal of neuro-rehabilitation is to promote recovery in patients with acquired brain injury through various therapeutic interventions involving retraining and facilitating neuroplasticity. Patients improve after brain injury by several different means. Adaptation and training may help patients compensate for their deficits and reduce disability even in the absence of neurological recovery. Pharmacotherapeutic agents that affect certain central neurotransmitters may modulate recovery. Natural spontaneous neurological recovery may lead to a decrease in the extent of neurological impairment. This may be explained by resolution of local edema, resorption of local toxins, improved local circulation, and recovery of partially damaged ischemic neurons (Roth & Harvey, 2000).

Neuroplasticity is a concept that refers to the potential ability of the CNS to modify its structural and functional organization. It is another explanation for recovery after brain injury. According to Flanagan et al. (2003), "a growing body of evidence has emerged demonstrating that the brain remains highly dynamic throughout adulthood, and remains capable of changing in response to experience and injury."

Neural plasticity is influenced by the environment and stimulation, repetition of tasks, and motivation. It occurs through neuronal regeneration or collateral sprouting, and the unmasking of previously latent functional pathways. Following cerebral injury, surviving neurons retain the ability to form new synapses. Research shows that "animals housed as adults in complex environments with access to various toys and activities develop more dendritic branching and more synapses per neuron and have higher gene expression for trophic factors than animals housed individually or in small groups in standard cages" (Johansson, 2000).

According to Cotman and Berchtold (2002), "exercise induces the expression of genes associated with plasticity, such as that encoding brain-derived neurotrophic factor (BDNF), and in addition promotes brain vascularization, neurogenesis, functional changes in neuronal structure and neuronal resistance to injury." Physical activity initiates a cascade of changes in gene expression in the hippocampus, a brain region critical for learning and memory (Cotman & Engesser-Cesar, 2002). This suggests that exercise initiates modifications in molecular mechanisms supporting the health and enhancing the plasticity of the brain (Cotman & Engesser-Cesar, 2002).

Pharmacological Augmentation of Recovery

Even with optimized neuro-rehabilitation, pharmacological manipulation may be necessary during the process of brain injury recovery. Many of the medications that a physiatrist may prescribe are reviewed by Scicutella (Chapter 6). The current emphasis will be on particular examples of pharmacology as related to patient care from the physiatrist's perspective.

Physicians must be aware of potential negative effects of several commonly prescribed medications and substitute agents that may be more appropriate for patients with acquired brain injury. One example of this is metoclopramide, which is a commonly prescribed antiemetic, which pharmacologically is a neuroleptic. There is some evidence that neuroleptic medications may impact negatively on cognition, and thus replacing metoclopramide with erythromycin, an antimicrobial that increases gastrointestinal motility without the sedating and negative effects of the neuroleptics, may be an option. In addition, if a patient is being prescribed a neuroleptic for a behavioral problem, it is best to minimize the number of agents from the same class to avoid the possibility of additive side effects. The antiepileptic medications are another example of this principle, some of which can have more cognitive and mood side effects than others. Phenytoin, which may cause confusion and drowsiness, may be substituted with carbamazepine (Tegretol), valproic acid (Depakote), gabapentin (Neurontin), or lamotrigine (Lamictal).

In contrast, other medications can help to enhance recovery by augmenting certain neurotransmitter systems which are known to be damaged in brain injury. For example, it has been demonstrated by Meythaler et al. (2002) that in the first few hours after ABI, catecholamine levels are increased in the cerebrospinal fluid (CSF), whereas later, catecholamine production is decreased and CSF levels drop (Bakay et al., 1986). Animal studies have shown that amphetamine increased the rate of motor recovery in experimentally injured rats as compared to controls and norepinephrine has a beneficial effect on recovery, while depletion completely blocks improvement of functional skills (Flanagan et al., 2003; Hovda & Feeney, 1984; Kline et al., 1994; Schmanke & Barth, 1997; Kikuchi et al., 2000; Hovda et al., 1989). Clinically this has had application for those with acquired brain injuries through the use of psychostimulants which cause the release of catecholamines such as dopamine and norepinephrine from presynaptic neurons. A recent study by Walker-Batson et al. (2001) suggested that dextroamphetamine administration resulted in a significant improvement in language skills in a group of patients with stroke-induced aphasia when paired with speech-language therapy as compared to controls.

Medications that enhance the dopaminergic pathway have been successful in improving levels of awareness and alertness in patients with moderate to severe ABI (Meythaler et al., 2002). Dopaminergic fibers are involved in stimulating the reticular activating system, which modulates arousal. Dopaminergic receptors are found in areas of the brain linked to movement, learning, and memory (McElligott et al., 2003). Amantadine facilitates the release of dopamine and delays its reuptake by neural cells. Amantadine may have NMDA receptor antagonist effects as well, and this action may contribute to its neuroprotective effects early after injury (Zafonte et al., 2001). In addition, dopamine agonists have also been used with some success for the treatment of neglect. Mukand et al. (2001) administered levodopa and carbidopa (Sinemet) to four patients with left neglect. In this small case series, three of the four patients had substantial improvements on a modified version of the Behavioral Inattention Test and their functional status on the Functional Independence Measure (FIM) assessment.

Upon review of his medications, phenytoin was discontinued since Mr. Smith had been seizure free for an entire week on the medical floor. In addition, the haloperidol was immediately discontinued because of its negative impact on recovery. Future episodes of agitation were managed successfully with nonpharmacologic interventions, which included behavioral and environmental strategies. On the third day following admission to the brain injury unit, staff expressed their concerns regarding Mr. Smith's inability to concentrate and follow through with commands. Mr. Smith's neurobehavioral deficits involving attention, cognitive efficiency, memory, reasoning, and judgment proved to be very challenging due to their effects on all aspects of rehabilitation. He was started on a trial of methylphenidate (Ritalin) and donepezil (Aricept), which proved to have a remarkable effect on his attention and memory as well motor recovery.

Spasticity

Spasticity is defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome" (Katz et al., 2000). It is the increase in resistance that is felt while passively moving the patient's limb at a particular joint.

Spasticity is commonly found in patients with acquired brain injury. At the bedside, spasticity is graded using the modified Ashworth Scale. Complications secondary to spasticity include contractures, heterotopic ossification, pressure ulcers, and respiratory infections.

When considering treatment for spasticity, it is important to determine if the spasticity is causing functional impairment. This should be done by observing the patient's functional activities, including gait and the synergy patterns. Functional goals of treatment are to improve hygiene, decrease pain, decrease deformity, improve orthotic fit, improve gait, decrease energy expenditure of gait, and facilitate motor control (Boake et al., 2000). In some patients, spasticity may be required to carry out certain functional activities. For example, extensor spasticity in the lower extremities may help some patients to stand or ambulate.

The first step in treating spasticity is to avoid exposure to noxious stimuli (i.e., pressure ulcers, urinary tract infection, constipation, ingrown toe nails). Proper positioning, stretching, and range of motion are essential components of the treatment process. This can be accompanied by modalities, serial casting, and/or dynamic splinting.

Cryotherapy has been shown to decrease muscle stretch reflex excitability and increase range of motion (Katz et al., 2000). Other modalities that can be used are biofeedback and electrical stimulation. Electrical stimulation has been shown to decrease tone in antagonistic muscle groups in hemiplegic and quadriplegic patients (Katz et al., 2000). Serial casting involves placing casts on the spastic limb to increase joint range of motion progressively. Skin should be monitored when placing and removing the casts. Skin breakdown can be a noxious stimulus and worsen the spasticity. Similarly, splinting is another option to help stretch the spastic limb.

Oral medications can also be used in conjunction with these methods. There is a long list of anti-spasticity medications. Unfortunately many of them, such as diazepam, cause cognitive impairments as previously described. In the setting of acquired brain injury, the recommended medication is dantrolene sodium. Dantrolene is the only antispasticity medication that acts peripherally, thereby sparing any central side effects. The mechanism of action involved is depolarization-induced calcium efflux into the sarcoplasmic reticulum (Boake et al., 2000). The side effects of this medication include lethargy and generalized weakness. Due to its hepatotoxic effects, liver function tests should be performed periodically for patients on this medication. Patients usually start at 25 mg/day.

Other treatment options include phenol or botulinum toxin injections for local treatment and intrathecal baclofen pump placement for generalized spasticity management. The mechanism of action for phenol injections involves chemical neurolysis. The effects of this type of injection may last close to a year although more typically it lasts approximately 3 months. A common side effect is dysesthesias occurring as a result of blocking sensory nerves. Botulinum toxin prevents acetylcholine vesicles from binding with proteins needed for fusion to surface membranes. This decreases the number of presynaptic transmitter vesicles preventing neuromuscular transmission. In essence, this produces a weakening of the muscle. The effects of the botulinum toxin injections typically last up to 3 months.

The intrathecal baclofen pump allows baclofen to be administered directly into the intrathecal space. It has been shown to be more efficacious for lower extremity spasticity. A patient who is considering the baclofen pump can undergo a trial in which the baclofen is injected intrathecally and the patient's response is monitored. Surgical procedures for spasticity are less frequently used. These include tendon lengthening and transfers and rhizotomies.

Mr. Smith exhibited spasticity in the right upper and lower extremity muscles. The extensor tone in the right lower extremity enabled him to stand up and walk. However, in occupational therapy, it was noted that functional use of the right upper extremity was limited because of pain secondary to increased tone. The physiatrist performed a selective botulinum toxin injection in the biceps brachii muscle to relieve flexor tone. Stretching was also performed by the therapists to work in conjunction with the effects of the botulinum toxin injection.

Fall Prevention

Most patients on the rehabilitation unit are at a high risk for falling. Therefore, fall prevention is a major initiative on most rehabilitation units. Urinary incontinence, neglect, and visuospatial deficits, as well as the use of sedatives increase the risk for falls after stroke. Additional risk factors for falls include impulsivity, bilateral strokes, confusion, male gender, and poor activities of daily living performance. Right hemispheric strokes confer a greater risk due to the association with neglect, visuospatial deficits, and impulsivity. Preventive measures must be in place to minimize falls. These may include adequate staff supervision of patients, fall prevention education, balance training, bed and chair alarms, timed voiding, minimizing the use of sedatives and diuretics, and restraints when absolutely necessary. One option includes issuing “ambulation orders” based on the patient’s evaluation and performance, without which patients are not permitted to ambulate independently on the rehabilitation unit. These orders are brought to the attention of the patient and the entire rehabilitation team, including aides, as well as the patient’s family and visitors. Ambulation orders are frequently updated as the patient progresses through rehabilitation.

Medical Complications Commonly Encountered on the Neuro-Rehabilitation Unit

All members of the rehabilitation team should be vigilant in monitoring for signs of complications. Up to 75% of patients admitted to a rehabilitation unit may experience medical complications following a stroke (Moroz et al., 2004).

Deep Venous Thrombosis (DVT)

Venous thromboembolic disease (VTE), including DVT and pulmonary embolus (PE), are among the most significant complications seen in both stroke and ABI survivors. VTE is related to increased mortality in the rehabilitation setting. The incidence of DVT in neurorehabilitation admissions ranges from 10% to 18% (Elovic et al., 2004), and in the stroke population occurs in 20% to 75% of survivors. Stroke patients remain at high risk for PE for up to 3 months post-stroke. Clinicians must have a high index of suspicion for DVT among these patients with frequent motor weakness and ambulation difficulties. DVT occurs most frequently in the lower limbs and is classically associated with venous stasis, endothelial vessel damage, and hypercoagulable states. It is also associated with prolonged immobility, paresis, fractures, soft tissue injuries, and age over 40 (Elovic et al., 2004). Most patients admitted to the rehabilitation unit are on a prophylactic regimen for DVT prevention. Commonly used agents for anticoagulation include low-dose unfractionated heparin (5,000 units q8 to 12 hours), low molecular weight heparin, and warfarin. An inferior vena cava (IVC) filter can be placed in patients who have contraindications to anticoagulation use, for example acute cerebral hemorrhage, although a filter should not be used as the sole method for prophylaxis. Intermittent pneumatic compression devices are also used in conjunction with another method of prophylaxis.

The diagnosis of DVT requires a high index of suspicion. Noninvasive techniques include ultrasonography, impedance plethysmography, contrast venography, and D-dimer serum assays. Contrast venography remains the gold standard for the diagnosis of clinically suspected DVT. Pulmonary angiography remains the gold standard for diagnosing PE. Treatment of DVT usually involves anticoagulation for 3–6 months.

Dopplers of the lower extremities on admission were negative for DVT. Given the history of SDH, anticoagulation was contraindicated for deep venous thrombosis (DVT) prophylaxis. As a result, Mr. Smith was placed on intermittent pneumatic compression devices. Range of motion was performed by the therapists on a daily basis to prevent contractures and to minimize the risk of formation of heterotopic ossification.

Heterotopic Ossification (HO)

HO is the formation of bone in abnormal, ectopic locations such as soft tissue. HO occurs primarily in the proximal joints of the upper and lower extremities in 11% to 76% of severely injured patients. Risk factors for HO in brain-injured patients include prolonged coma (greater than 2 weeks), spasticity, long-bone fractures, and decreased range of motion. Joints most frequently affected in brain-injured patients are hips, elbows, shoulders, knees. HO may present with pain, warmth, swelling, and contracture formation, but may be occult as well. The earliest method for detecting HO is the triple phase bone scan. HO may be seen within the first 2–4 weeks in Phase I and Phase II (blood-flow and blood-pool phases) of a triple phase bone scan. Phase III (static phase) will detect HO after 4–8 weeks. HO may not be evident on plain X-rays for 3 weeks to 2 months. Serum alkaline phosphatase levels are increased but this finding is nonspecific.

Prophylaxis for HO includes ROM exercises, control of muscle tone, nonsteroidal anti-inflammatory drugs (NSAIDS) such as indomethacin, and radiation, although use of radiation in younger patients is controversial. The treatment for HO is diphosphonates (etidronate) and NSAIDS (indomethacin). Salicylates may also be used. Etidronate will decrease the ongoing formation of HO. ROM exercises play an important role in both the prophylaxis and treatment of HO. ROM exercises are crucial in preventing ankylosis. Surgical resection of HO may only be performed after bony maturation, typically 12–18 months after the onset of HO. At the point of maturation, serum alkaline phosphatase levels return to normal.

Post-Traumatic Hydrocephalus (PTH)

Ventriculomegaly, due to cerebral atrophy and focal infarction of brain tissue, or hydrocephalus ex vacuo, is commonly seen in patients after ABI. The reported incidence is 40% to 72% of severe ABI patients (Elovic et al., 2004). True hydrocephalus is not as common, with the incidence being 3.9% to 8%. Hydrocephalus in ABI patients is most commonly the communicating type, specifically, normal pressure hydrocephalus. Unfortunately, the classic trial of incontinence, ataxia, and dementia is not always seen and is of little help in severely disabled patients. Initial manifestations of hydrocephalus can include headache, vomiting, confusion, and drowsiness. The physician must have a high index of suspicion and team members must report subtle behavioral or functional deteriorations. Failure to improve or deterioration of cognitive or behavioral function should prompt assessment with a CT scan (Whyte et al., 1998). The "tap test," the withdrawal of cerebrospinal fluid via lumbar puncture, may be both diagnostic and therapeutic. Patients may require surgical shunt placement.

Patients that already have ventricular shunts may experience shunt failure. Therefore, CT scanning and shunt flow study or pressure measurements should be performed if clinical deterioration is noted.

Autonomic Dysfunction and Post-Traumatic Hypertension

ABI patients may suffer from central dysautonomia. This may present with alterations in blood pressure and pulse and impaired temperature regulation. Hypertension and tachycardia may be secondary to a hyperadrenergic state. It may be caused by injuries to the hypothalamus and is usually self-limited. HTN may also be iatrogenic (methylphenidate).

Hyperthermia or "central fever" may be due to a lesion in the hypothalamus or can be an indication of decerebration. Temperatures may reach higher than 104° F. Central fever is always a diagnosis of exclusion and other causes for the fever must be ruled out. Treatment involves cooling the patient with indomethacin or dopamine agonists and dantrolene. Hypothermia may be secondary to a lesion in the posterior hypothalamus.

Post-traumatic hypertension is associated with intracerebral hemorrhage and DAI. Hypertension, tachycardia, and increased cardiac output in the acute post-injury period may result from the increased levels of circulating catecholamines (Whyte et al., 1998). Beta blockers may be useful for treatment. The use of highly polar beta blockers which only minimally cross the blood-brain barrier is suggested. These include atenolol and nadolol. Sustained hypertension is infrequent in ABI, unless it was a premorbid medical condition. When long-term therapy is needed, angiotensin-converting enzyme (ACE) inhibitors, calcium channel blockers, and some diuretics are least likely to cause cognitive impairment.

Endocrine Complications

Individuals who have had a brain injury may be subject to associated neuroendocrine problems due to injury to the hypothalamus or pituitary gland. During stressful episodes such as trauma, release of antidiuretic hormone (ADH) from the hypothalamus is increased. Elevated ICP may further contribute to release of this hormone. The most commonly seen syndromes are cerebral salt-wasting (CSW) syndrome and the syndrome of inappropriate antidiuretic hormone secretion (SIADH). CSW and SIADH both result in hyponatremia; however, patients with CSW syndrome are in fact volume depleted, whereas in SIADH, patients are euvolemic. CSW is presumed to occur because of direct neural effect on renal tubular function.

The treatment for CSW is hydration/fluid replacement and serum electrolyte correction. Remember these patients are volume-depleted. In SIADH, the treatment usually begins with fluid restriction to $\sim$1 L/day with or without use of a loop diuretic. Serum sodium levels should be checked daily and weight changes should be closely monitored. Hypertonic saline is administered in patients with symptoms such as confusion, convulsions, or coma. Profound hyponatremia can be fatal but serum sodium must be corrected gradually to avoid central pontine myelinolysis.

Diabetes insipidus (DI) is a less common disorder of the pituitary gland and is often associated with basal skull fractures. A fracture in or near the sella turcica may tear the stalk of the pituitary gland. This can result in DI due to disruption of ADH secretion from the posterior lobe of the pituitary. It is characterized by polyuria, polydipsia, and hypernatremia. The hallmark is excessive excretion of dilute urine (urine osm $<$290 mmol/kg, SG 1.010). The decrease in intravascular volume can lead to hypotension and decreased cerebral perfusion pressure.

Treatment for DI is hormone replacement with desmopressin acetate, an analog of antidiuretic hormone. The medication chlorpropamide potentiates the effects of ADH on the renal tubules and is used in patients with partial ADH deficiency. If the patient is experiencing significant mental status changes, intravenous hypotonic fluid replacement must be administered.

Other endocrine problems may occur as well. Menstrual irregularities are common following severe head trauma and it may take several months to resume a normal menstrual cycle. Gynecomastia and galactorrhea may occur from elevations in prolactin levels. Sexual dysfunction is commonly attributed to psychosocial issues but may be partially accounted for by endocrine dysfunction. If a patient appears to show a premature plateau or decline in function and recovery, a complete endocrine profile should be assessed including thyroid and adrenal gland function.

Common Stroke-Related Impairments

Disability in stroke is a result of CNS injury by which physical, cognitive, and psychological functioning become impaired. Specific impairments appear when focal regions and neural systems within the brain are damaged by vascular compromise (Roth & Harvey, 2000). Common impairments caused by stroke include: motor weakness (90%), deficits in higher mental function, ataxia (20%), hemianopsia (25%), visuoperceptual deficits (30%), aphasia (35%), cranial nerve impairments, dysarthria (50%), apraxia, neglect syndrome, dysphagia, spasticity, sensory impairment, balance, coordination and posture impairments, and bladder and bowel dysfunction.

Rehabilitation of Stroke Survivors

A stroke is a nontraumatic, acquired brain injury caused by the occlusion or rupture of cerebral blood vessels resulting in the sudden development of a persisting neurological deficit (Roth & Harvey, 2000). The focal brain lesions encountered in patients with stroke produce a wide array of neurologic deficits. Roth (1992) listed five major functions of stroke rehabilitation:

1. Prevention, recognition, management, and minimizing the impact of preexisting medical conditions, ongoing general health functions, and secondary medical complications.
2. Training for maximal functional independence.
3. Facilitating optimal psychosocial adaptation and coping by both the patient and family.
4. Promoting community reintegration, resumption of prior life roles, and the return to home, family, recreational, and vocational activities.
5. Enhancing quality of life.

Predictors of Outcome Post Stroke

The most reliable and consistent predictor of functional outcome is the person's functional ability at admission. An admission FIM score of 60 or greater is associated with a higher likelihood of functional improvement. Other noted predictors of functional outcome include age, previous stroke, urinary incontinence, consciousness at onset, severity of paralysis, sitting balance, visuospatial deficits, unilateral hemineglect, and level of social support. Unilateral visual neglect may adversely affect functional outcome and is associated with poor safety awareness, disrupted daily activities, and decreased likelihood of returning to work or driving (Mukand et al., 2001). Wade and associates (1983) studied 83 stroke patients and found the best predictors of function after 6 months were sitting balance, age, hemianopsia, urinary incontinence, and motor deficit in the arm. The mortality from all strokes ranges from 17% to 34% in the first 30 days (O'Neill et al., 2004). The rate in hemorrhagic strokes may be as high as 48%, but this may be related to stroke severity, and not necessarily the type of stroke. Patients with intracerebral hemorrhage are more likely to have higher stroke severity and poorer outcome (Jorgensen et al., 1995).

Stages of Motor Recovery after Stroke

Up to 88% of acute stroke patients have hemiparesis (Zorowitz et al., 2004). Hemiparesis and motor recovery have been the most studied of all stroke impairments. Motor recovery following stroke has been described by Twitchell (1951) and Brunnstrom (1970). Twitchell described the pattern of motor recovery following a stroke. The pattern he described is most consistent with recovery in patients with a CVA in the middle cerebral artery (MCA) distribution. Total loss of voluntary movement and loss or decrease of tendon reflexes occurs at the onset of hemiplegia, typically involving the arm more than the leg. Tone and "tight coupling of movement at adjacent joints" (later termed synergy by Brunnstrom) developed before isolated voluntary movement returned. As spasticity increased, clonus appeared (Zorowitz et al., 2004). He also noted that motor function typically returned proximally before distally and lower extremity function recovered earlier and more completely than upper extremity function. The majority of recovery occurred in the first 12 weeks, with only minor additional recovery after 6 months.

Brunnstrom divided the recovery process into seven different stages. Initial loss of voluntary motion is accompanied by limb flaccidity. This stage is followed by an increase in reflexes, spasticity and weak basic synergy patterns. Stage 3 is delineated by prominent spasticity with patient regaining some voluntary control over synergy patterns. This is followed by some voluntary, selective activation of muscles outside of synergy patterns with some reduction in spasticity. Stage 5 is marked by further decrease in spasticity with most limb movement independent of synergy. Stage 6 is resolution of spasticity with near normal coordination and isolated movements. Normal function is restored in the seventh and final stage.

Major Theories of Rehabilitation Training Post-Stroke

Traditional approaches for improving motor control and coordination emphasize the need for repetition of specific movements for learning, the importance of sensation for the control of movement, and the need to develop basic movements and postures. Several neurophysiological theories of rehabilitation for motor deficits have been developed. No single approach has been proven to be more effective and therapists typically incorporate aspects from several theories when formulating a treatment plan.

Proprioceptive neuromuscular facilitation (PNF)-based rehabilitation uses diagonal and spiral pattern techniques to facilitate movement patterns. It uses mechanisms such as maximum resistance, quick stretch, and spiral diagonal patterns to facilitate normal movement by "irradiation" of impulses to other parts of the body associated with the primary movement.

The Bobath/neurodevelopmental treatment (NDT) approach emphasizes suppressing abnormal muscle patterns and avoiding mass synergies, because these may reinforce spasticity and increased reflexes. The goal of NDT is to normalize tone, inhibit primitive patterns of movement, and to facilitate automatic, voluntary reactions and subsequent normal movement patterns (Zorowitz et al., 2004).

In contrast to NDT, the Brunnstrom approach (Choi et al., 2003) utilizes primitive postural reactions and synergies to facilitate motor function. Patients are encouraged to learn to control and use the synergistic motor patterns available at each particular phase of recovery.

The Rood or sensorimotor approach employs cutaneous sensorimotor stimulation such as brushing, stroking, tendon tapping, vibration, icing, and quick stretch to activate motor function and inhibit spastic antagonists.

Carr and Shepherd's motor relearning program (Roth, 2002) is based on a theory of cognitive motor relearning and emphasizes functional training, practice, and repetition in the performance of specific tasks, and carryover of those skills into functional activities.

Electrical neuromuscular stimulation may also elicit motor and functional gains. According to Dobkin (2004), "functional electrical stimulation is also used to activate paretic muscles timed to a movement, such as contraction of the tibialis anterior muscle to clear the foot during the swing phase of walking."

Body weight-supported (BWS) treadmill training is geared specifically to the recovery of walking ability. BWS has been shown to restore gait faster in nonambulatory patients when compared with nonambulatory patients who received conventional therapy (Wernig & Muller, 1992). Gait retraining in this approach consists of walking a patient on a treadmill at his/her maximum comfortable speed, whereas a percentage of his/her body weight is supported centrally at the trunk by an overhead harness. The amount of body weight that is supported typically ranges from 0% to 40% (Bogey et al., 2004). BWS treadmill walking is typically well tolerated after stroke. In a trial with acute-stroke patients, Malouin et al. (1992) demonstrated good compliance. Patients were able to withstand up to 45 minutes of treadmill walking. However, no trials have been done studying the efficacy of BWS walking therapy in the acute stroke rehabilitation setting. The optimal timing for intervention with BWS walking has not been determined and there is some evidence supporting this type of training even 2 years post-stroke (Malouin et al., 1992). An approach such as BWS treadmill training is not meant to be an exclusive method of training and must be complemented by other treatment approaches.

In the past, functional gains were incorrectly said to plateau by 3–6 months post injury. However, many patients retain latent sensorimotor potential that may be nurtured and released any time after injury with a course of goal-directed therapy.

Constraint-Induced Movement Therapy

Constraint induced movement therapy (CIMT) is based on the observation that some of the disability in stroke patients resulted from lack of use of the affected limb. Learned nonuse of the affected arm is common because of pain, slow and increased effort, and energy requirements to use the affected limb, and ease of use of the unaffected limb. In this "use it or lose it" forced-use intervention, the unaffected limb is restrained in an attempt to force use of the affected limb. Research has shown that CIMT is both feasible and tolerated, and associated with less short-term arm impairment than traditional therapy (Dromerick et al., 2000). According to Dobkin (2004), "traditionally CIMT is performed for approximately 6 hours per day for 2 weeks, but less intensity may work as well, and may be effective even when initiated 2 years post stroke." The patient must have a minimum of 20 degrees of voluntary wrist extension and 10 degrees of extension in two fingers at the metacarpophalangeal or interphalangeal joints of the paretic hand to qualify for enrollment into a CIMT protocol (Levy et al., 2002).

Post-Stroke Complications

Up to 75 percent of patients admitted to a rehabilitation unit may experience medical complications after a stroke (Moroz et al., 2004). Death within the first week of a stroke is attributed to the stroke itself in 90% of patients (Moroz et al., 2004). This may be from cerebral edema, mass effect, or herniation. The most common cause of death within the first 2 to 4 weeks post-stroke is pulmonary embolism. Pneumonia is the most common cause of death during the second and third months post stroke, with cardiac disease responsible thereafter. Patients remain at high risk for pulmonary embolism for up to 3 months after stroke (Moroz et al., 2004).

Secondary Prevention

Secondary prevention of stroke is an important aspect of rehabilitation management (O'Neill et al., 2004). The physiatrist must make recommendations to the patient and family members for prevention of subsequent strokes. Approximately 7% of all patients with a history of transient ischemic attack (TIA) or stroke will have a recurrent event each year. Risk factors for stroke include modifiable and unmodifiable factors. Modifiable risk factors include hypertension, diabetes, heart disease, TIA or prior stroke, hypercholesterolemia, obesity, sedentary lifestyle, cigarette smoking, alcohol abuse, and cocaine use. Nonmodifiable risk factors include age, gender, race, and family history. Furthermore, the use of antiplatelet agents, anticoagulation and carotid endarterectomy may be utilized for secondary stroke prevention in some patients. The careful identification and reduction of risk factors may significantly reduce the risk of recurrent stroke and patient/family education must be addressed as part of the rehabilitation program.

Conclusion

Many individuals are left with a combination of physical, cognitive, and psychosocial impairments following an acquired brain injury. The challenge for physiatrists specializing in neuro-rehabilitation is in applying the knowledge gathered from group studies to the management of each patient's unique pattern of deficits, which produce a broad array of disabilities and handicaps that may persist long after the initial injury (Whyte et al., 1998). The pattern of impairments varies greatly from patient to patient. Handicaps may be affected by differences in the social and physical environments to which the brain injury survivor returns providing further treatment challenges for the physiatrist specializing in neuro-rehabilitation (Whyte et al., 1998). Therefore, an interdisciplinary treatment approach that considers the complex and unique pattern of deficits seen in an individual patient is the best approach to treatment. Physiatrists must be able to communicate effectively with patients, families and staff and provide assessments of prognosis based on literature, prognostic parameters, and clinical experience. At the same time we must never take away hope (Zasler, 1999).

Much progress has been made in the area of brain-injury rehabilitation. Rehabilitation and community integration focus on helping the person achieve a new sense of "self." While we are often unable to "cure" those who have suffered a devastating acquired brain injury, we are capable of ameliorating the impact of physical and cognitive impairments on the individual's functional status. Physiatrists and the rest of the rehabilitation team provide individuals with the tools they require to adapt and adjust to their new circumstances.

Four weeks after beginning acute inpatient rehabilitation, Mr. Smith had made a considerable amount of gains in all the disciplines. He was on a regular diet. He was ambulating with a quad cane and he was able to transfer independently. He continued to have some cognitive deficits for which he required supervision during all his activities. However, his comprehension and awareness of his environment and people around him had improved drastically. He was able to recognize his wife and son, which was something he could not do initially. Mr. Smith was discharged home with a referral to comprehensive outpatient neuro-rehabilitation program for continued therapy, and he is continuing to make gains.

"Hospital discharge should be looked on as the beginning of a new life in which the patient faces the challenge of adopting different roles and relationships and search for new meaning in life" (Brandstater, 1998). Brain-injury survivors face many challenges to community living every day of their lives, which often include a combination of chronic physical, cognitive and psychosocial impairments. According to Gordon et al. (1999), these individuals experience the unique challenge of "walking the sometimes conflicting paths of who they were, who they are, and who they want to be." Helping to meet the complex, ongoing needs of this population is one of the key challenges for all involved in neuro-rehabilitation, including clinicians, survivors, as well as families and friends.

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