Rehabilitation of Patients with Neurologic Tumors and Cancer-Related Central Nervous System Disabilities

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Neurologic tumors may involve the brain or spinal cord and are either primary or metastatic. Patients may become increasingly less independent as a result of direct injury of neural structures responsible for motor, sensory, cognitive, and speech functions. The indirect effects of chemotherapy and radiation therapy (RT) add to the functional deficits patients experience. The number of patients involved is quite large. More than 15,000 new cases of primary brain tumor and 4000 new spinal tumors are diagnosed every year (American Cancer Society, 1990). Approximately 2% of all cancer deaths are caused by brain tumors, which account for roughly 11,000 deaths per year (American Cancer Society, 1990). Metastatic lesions from various sites account for 20% to 40% of brain tumors (American Cancer Society, 1990), occur in approximately 15% of cancer patients (Black, 1991), and produce neurologic symptoms in approximately 85,000 patients each year. Most spinal tumors are extradural and are predominantly metastatic carcinomas, lymphomas, or sarcomas (Posner and Chernik, 1978).

Patients may undergo surgical resection, chemotherapy, and/or radiation to combat their tumors. It is important to note that significant functional deficits can exist even before treatments. The following signs were noted in 162 patients with cerebral metastases: impaired cognition (77%), hemiparesis (66%), unilateral sensory loss (27%), ataxia (24%), and aphasias (19%) (Caraceni and Martini, 1999a). Spinal cord compression can occur in 5% to 10% of cancer cases (Barron et al., 1959). Immediate functional consequences can include pain, sensory deficits, motor deficits, neurogenic bowel and bladder, and sexual dysfunction.

Rehabilitation management of impairments and disabilities is approached in the same manner as in noncancerous neurologic diseases. However, the pathology of the tumor and the anticipated course of disease progression must be considered carefully when developing rehabilitation goals as well as the time frame required to achieve these goals for an individual patient. The purpose of rehabilitation for cancer patients is similar to that for patients with other diseases; emphasis is placed on restoring or maximizing independence with activities of daily living (ADL), mobility, cognition, and communication. Rehabilitation interventions can be applied in all stages of the disease, although rehabilitation goals change as the stage of illness advances. Preventive rehabilitation maintains maximum functional independence in patients who undergo treatment and who have potential loss of function. When tumor progression causes a decline in functional skills, or the disease causes fluctuating abilities, rehabilitation assumes a supportive role, with goals adjusted to accommodate persistent anatomic and physiologic limitations. During terminal stages of illness, palliative rehabilitation can improve and maintain comfort and quality of life until the end of life. An optimal re-
habilitation team consists of a physiatrist, primary physician, nurse, physical therapist, occupational therapist, speech-language therapist, recreational therapist, social worker, case manager, dietitian, and chaplain (Garden and Gillis, 1996). Benefits of rehabilitation are noted in Table 22–1.

REHABILITATION OF BRAIN TUMOR PATIENTS

It is important to understand that even a small low-grade malignant tumor may cause significant residual functional deficits if it resides in a critical location. Lesions located near the brain stem can be particularly damaging to motor functions, sensory functions, coordination, and cranial nerves. Primary malignant tumors in adults are mostly gliomas, which account for more than 90% of lesions (Bondy and Wrensch, 1993). Of these, glioblastoma multiforme has the worst prognosis and low-grade astrocytoma, the best (Black, 1991). The location of these tumors may or may not permit resection. Pituitary tumors may result in headaches, bilateral visual loss (due to their central location), and hormonal abnormalities (Black, 1991). With acoustic neuromas, hearing loss and/or vertigo may occur due to their proximity to the cranial nerve. Other symptoms associated with these tumors include facial palsy and numbness, dysphagia, and hydrocephalus. Visual loss and sexual dysfunction can be present with craniopharyngiomas in adults, and growth failure may occur in children with these tumors. Changes in behavior, appetite, memory, and endocrine function may be seen following radiation treatment (Black, 1991).

The lung and breast are the most frequent primary sources of metastatic CNS tumors. Other common primary sources are carcinomas from the colon/rectum, kidney, and pancreas, as well as malignant melanomas. These tumors tend to be highly invasive and destructive. Edema is often present and may extend for some distance beyond the tumor. Leptomeningeal metastases with multiple cranial nerve and spinal root involvement may also occur.

General Considerations

It is essential to consider the fluctuant nature of disease progression for many of these patients and that the overall prognosis may not be very good when these tumors are present. Rehabilitation interventions should be guided by the evidence regarding the nature and behavior (aggressive or indolent) of each patient’s tumor, the ongoing clinical course, and the patient’s neurologic status. If the prognosis is very limited, or severe cognitive injury impedes patient learning and retention of new information, caregiver education and adaptation of the patient’s environment become dominant components of the rehabilitation plan. In cases of expected survival of less than 2 months, primary goals usually shift to injury prevention, safety for patient and caregivers, and ease in performing tasks of hygiene and transfers into and out of bed. Common complications influencing the rehabilitation program for these patients are listed in Table 22–2.

With temporal lobe tumors, dysnomia, disturbance of comprehension, and defective hearing and mem-

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Table 22–1. Benefits of Rehabilitation

| Training to maximize functional independence |
| Facilitation of psychosocial coping and adaptation by patient and family |
| Improved quality of life through community reintegration: includes resumption of prior home, family, recreational, and vocational activities |
| Recognition, management, and prevention of co-morbid illnesses that limit or impede function |

Table 22–2. Common Complications of Brain Tumors and Their Treatments

| Weakness |
| Sensory loss |
| Visuospatial deficits |
| Hemi-neglect or bilateral visual deficits |
| Ataxia |
| Cognitive deficits: thought processes, memory changes, apraxia, etc. |
| Speech difficulties |
| Dysphagia |
| Bowel and bladder dysfunction |
| Psychological issues |
| Behavioral abnormalities |
| Endocrine issues |
| Skin issues |
| Fatigue |

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ory may occur (Nelson et al., 1993). Loss of vision, spatial disorientation, memory loss, dressing apraxia, and proprioceptive agnosia may occur with parietal lobe tumors. Behavioral abnormalities can occur with frontal lobe tumors, and these may include personality or libido changes, with impulsive behavior, labile emotions, and excessive jocularity. Hyponatremia, as seen in the syndrome of inappropriate diuretic hormone (SIADH), may lead to mental status changes (Nelson et al., 1993). Fatigue may become an issue with radiation treatment. Steroid psychosis occasionally complicates the rehabilitation course.

With prolonged immobility in bed, supportive care is important. Measures should be taken to prevent pressure ulcers and deep venous thrombosis. Range of motion of all joints should be maintained with daily exercises or passive stretch if paralysis or altered mental status is present. Sensory stimulation should be provided, along with socialization.

Corticosteroids, which are commonly used to combat peritumoral edema, tend to improve diffuse neurologic dysfunction rather than focal deficits. Myopathy with proximal muscle weakness often ensues and is very difficult to reverse until steroid doses have been tapered or discontinued. Unfortunately, immobility and myopathy frequently create cumulative deficits in strength and endurance. Patients should receive strengthening therapies and exercise programs when steroid therapy is initiated.

Patients may have uninhibited bladder due to lack of cortical influence and may require frequent prompting. Behavioral training may be helpful in patients with unimpaired cognition. This involves progressively increasing the time between voiding, often by 10 to 15 minutes every 2 to 5 days until a reasonable interval between voiding is obtained. Drugs to inhibit bladder evacuations, such as anticholinergic and antispasmodic agents, should be judiciously used. External (condom) catheters may be an option for some male patients. If a diaper is used, it should be changed within 2 to 4 hours to avoid skin breakdown. Immobile or sedentary patients become constipated easily and may require a bowel program with higher fluid intake, stool softeners, and digital stimulation, along with suppositories, laxatives, and enemas.

Orthotic devices that support a limb or joint and assistive devices such as walkers and canes may be issued. Use of a wheelchair may be necessary for those with significant weakness and balance impairment. Occupational and physical therapists should be consulted early for evaluation and teaching of ADL, ambulation, and strengthening and stretching exercises. Speech therapists can assist with the assessment of cognition, linguistic, and communication deficits. They can also determine the presence of swallowing difficulties and recommend therapeutic exercises, compensatory maneuvers, and modified-consistency diets.

Seizures and hydrocephalus are complications of brain tumors that often negatively impact the course of rehabilitation through declining functional performance. Todd’s paralysis and subclinical seizures may mimic other etiologies for declining neurologic status and prevent participation in a rehabilitation program. Hydrocephalus may also have a presentation suggestive of other diagoses, may be acute or chronic in nature, and usually leads to a decline in functional status. It is classically described as a triad of subcortical dementia, incontinence, and gait disorder. Hydrocephalus should be suspected when changes in mentation occur, when a patient fails to make expected functional gains, or when spasticity, seizures, and emotional problems are present. Workup may include computed tomography (CT) scan, lumbar puncture, CT cisternography, and radionuclide cisternography. Neurosurgical consultation for shunt placement should be obtained.

**Cranial Nerve Deficits**

Cranial nerve function should be routinely assessed in patients with brain tumors, as appropriate intervention may greatly improve functional status. Visual and hearing deficits are frequently seen in meningiomas, acoustic neuromas, and pituitary adenomas. Ophthalmoplegia and facial pain may also present as symptoms of central nervous system (CNS) tumor (Rowland, 1995).

Suprasellar lesions can cause bitemporal hemianopsia, but can also cause diminished visual acuity, scotomata, quadradric deficits, and blindness of one or both eyes. When treating patients with visual deficits, rehabilitation management should include an ophthalmology consultation to quantify the extent of the visual field loss. Training the patient to utilize compensatory techniques such as scanning will improve visual spatial awareness. Driving recommendations should be given before discharge, with plans
for further evaluations as vision improves. Vision impairment typically leads to adverse effects on independent living and must be considered in discharge planning. Patients with double vision can be treated with alternating-eye patching.

Facial pain can be very debilitating and may be treated with tricyclic antidepressant, antiepileptic, or analgesic medications, alone or in combination or in combination with mild narcotic medications. Facial and eyelid paralysis may necessitate plastic surgery interventions for corneal protection or cosmesis. Hearing deficits may have a central or peripheral etiology. Audiology evaluations will differentiate sensorineural from conductive hearing loss. Speech pathology consultation is necessary to establish appropriate routes for communication.

**Balance Abnormalities**

The neurologic components of human balance are the visual, vestibular, and somatosensory systems. The brain stem and cerebellum process and integrate information about balance from various peripheral receptors, which is then sent onward through corticospinal and brain stem pathways. Balance abnormalities may include dizziness, unsteadiness, vertigo, muscle weakness, and proprioceptive sensory loss. Injuries of the posterior columns of the spinal cord or of the parasagittal or sensory cortex of the brain can lead to these difficulties. Many cancer-related problems and treatments contribute to imbalance, including poor nutrition, anemia, anxiety, postural hypotension, and dehydration. Medications such as antiemetics, tranquilizers, opiates, vestibulotoxic antibiotics (e.g., aminoglycosides), and diuretics may also cause loss of balance. Radiation therapy that includes the temporal bone and/or the posterior fossa can lead to intermittent vertigo. While brain tumors at many different locations may lead to a sense of vertigo, nystagmus occurs with vestibular or posterior cranial fossa tumors. Patients frequently compensate by tilting their head to decrease the nystagmus. Acoustic neuromas can present as Ménière’s disease, where vertigo is associated with hearing loss and tinnitus. Tumors affecting the cerebellum may lead to ataxia and dysdiadochokinesia.

Vestibular disturbance can be treated by habituation, which leads to decreased sensitivity of the vestibular response. The goals of rehabilitation are to resolve reversible deficits and to learn compensatory and adaptive techniques for irreversible deficits, thereby improving safety and increasing independence. Rehabilitation may include training patients to effectively use other sensory input and habituation to control symptoms provoked by activity. Spontaneous resolution can occur and is often related to the severity of the initial insult as well as the possible plasticity of the CNS.

The Balance Master System is a medical device (NeuroCom International, Clackamas, Oregon) used to quantify and treat balance abnormalities. It utilizes a partially enclosed environment with a monitor screen that changes visual orientation input. It has platforms on which a patient stands (both outside and inside the environment) to measure movements and/or provide tilts or weight shifts. Harnesses are available for safety. Parameters measured include (1) amount of weight bearing on either foot, (2) sway with upper body movement, (3) rhythmic weight shift with body movement in all planes, (4) limits of stability whereby patients are provided a mechanical force toward which they try to shift their weight to compensate to maintain balance, and (5) weight shifts during movements such as transfers from sitting to standing and walking. Visual feedback is then given regarding the patient’s position while the therapist can give verbal cues. The results are stored for quantitative and graphic analysis. Vestibular and visual components can be isolated by the device’s environment. A custom exercise program can then be developed based on the determined deficits. Proprioceptive responses may be improved via controlled mobility, improved anterior-posterior weight shifts, increasing trunk strength and range of motion, as well as increasing midline symmetry and transitional movements.

Pharmacologic treatments include meclizine and dimenhydrinate, which may cause sedation. Transdermal scopolamine patches can also be used and are believed to cause less sedation.

**Cognition/Speech Deficits**

Deficits in cognition and speech occur; they vary in type and severity by the location and type of tumor, anticancer treatment, pre-morbid cognitive baseline, and co-morbid medical conditions. Cognitive deficits arise from tissue injury caused by the tumor itself, surgical resection, and the acute effects of radiation and chemotherapy (Silberfarb, 1983). The neu-
ropsychiatric side effects of both steroid and anti-
convulsant therapy should also be considered in cog-
nitive assessment (Lewis and Smith, 1983). Emotional
sequelae such as depression and anxiety are com-
mon, may worsen cognitive functions, or are over-
looked in the presence of cognitive deficits. Coexist-
ing medical conditions such as hypothyroidism are
treatable and should be considered in the differential
diagnosis of cognitive impairment.

Cognitive deficits are most often seen in areas in-
volving memory, attention, initiation, and psychomo-
tor retardation. Primary interventions for memory im-
pairment include memory aids and the use of visual
imagery. Cognitive remediation programs teach pa-
tients adaptive strategies and compensatory tech-
niques. Psychostimulants have been reported to be
useful in treating psychomotor retardation, depres-
sion, and opioid-induced drowsiness (Bruera et al.,
1989; Weitzner et al., 1996). Dopamine agonists and
stimulating antidepressants improve attentional dys-
function, particularly distractibility and difficulty fo-
cusing, in higher level patients (Gualtieri et al., 1989).
Bromocriptine can be effective for motor aphasias and
neglect in some patients (Grujic et al., 1998). Carba-
mazepine, tricyclic antidepressants, trazodone, aman-
tadine, and β-blockers have been prescribed to man-
age agitation in patients with traumatic brain injury
(Brooke et al., 1992; Mysiw et al., 1988; Whyte, 1988).

Aphasia is a language disorder, whereas dysarthria
is an articulation disorder. In contrast to aphasia,
naming, fluency, repetition, and comprehension are
normal in dysarthric conditions, and dysarthric pa-
tients can read and write without errors. The sever-
ity of aphasia correlates significantly with communi-
cation difficulty. Other disorders such as apraxia,
visual constructive difficulties, and neglect need to be
considered in the differential diagnosis of communi-
cation dysfunction.

REHABILITATION OF PATIENTS WITH
SPINAL CORD TUMORS AND
TREATMENT-INDUCED INJURY

Mylopathy with Tumors

Mylopathy may occur due to tumor involvement, ir-
radiation, and intrathecal chemotherapy. Metastatic
tumors may involve the spine or spinal cord. Back
pain is a frequent symptom and in 10% of cases may
be due to spinal instability (Gilbert et al., 1977;
Portenoy et al., 1987; Rodichok et al., 1981). Any tu-
mor can metastasize to the spine and cause sufficient
destruction to produce spinal instability. The thoracic
spine is the segment most commonly involved, fol-
lowed by lumbosacral and then cervical vertebral lev-
els (Casciato and Lowitz, 1983; Schlicht and Smelz,
1994).

Spinal cord compression eventually occurs in ap-
proximately 5% of patients with cancer (Casciato and
Lowitz, 1983). Abrupt neurologic deterioration from
spinal cord involvement may occur from rapidly
growing lesions in the extradural space. Infarction of
the vertebral blood supply can cause cord injury.
Radiculopathy at any level is also possible (Gilbert et
al., 1977; Rodichok et al., 1981). By the time treat-
ment is pursued, as many as 50% of patients may not
be able to ambulate, and 10% to 30% may be para-
plegic (Shapiro and Posner, 1983; Shaw et al., 1980).

Symptoms include weakness, incoordination, gait
abnormality, spinal or radicular pain, paresthesias,
sensory disturbances, autonomic disturbances, as
well as bowel or bladder problems. Pinprick and
deep pain sensation is often retained until later in
the course of the disease. Motor involvement typi-
cally occurs before sensory involvement with epid
dural extension (Galasko, 1999). With radiation
treatment alone, ambulation is maintained in 79% of
patients if they were ambulatory before treatment and
in 42% with paraparesis. In 20% to 25% of patients,
significant neurologic deterioration was noted dur-
ing the course of treatment with radiation alone
(Findlay, 1984).

Significant neurologic deterioration and progres-
sive spinal instability require a neurosurgical con-
sultation. High-dose steroids are used in the acute
phase to control neurogenic damage. Tetraplegia,
whether complete or incomplete, occurs with spinal
cord involvement at T1 or above. Below this level,
injuries more commonly result in paraplegia, conus
medullaris syndrome, or cauda equina syndrome.
Local pain is typically described as constant and
aching, whereas radicular pain is classically sharp
and shooting. Referred pain can be either aching or
sharp and at a location distant to the involved site.
Pain can also occur with epidural involvement; this
pain worsens with Valsalva maneuvers, coughing,
and neck and back flexion (Gilbert et al., 1978; Ger-
ber and Vargo, 1998). Functional prognostic factors
are listed in (Tables 22–3 and 22–4).
With use of radiation treatment and posterior laminectomy, the overall results were that complete paraplegia patients rarely recovered, but those who were ambulatory remained ambulatory and approximately one-half of patients with incomplete paraplegia regained ambulation (Posner, 1995). Recovery tends to occur first in the area of sensory disturbance, followed by motor abnormalities (Casciato and Lowitz, 1983; Schlicht and Smelz, 1994).

**Radiation-Related Myelopathies**

The detrimental effects of radiation are multifactorial and cannot be entirely attributed to dosage, site, or technique. Such myelopathy may be transient or delayed (Dropcho, 1991). With transient myelopathy, peak onset is at 4 to 6 months (Dropcho, 1991). Clinical onset may involve symmetric paresthesia or shock-like sensations in a nondermatomal pattern from spine to extremities (Leibel et al., 1991). Radiologic studies are usually normal. Symptoms typically resolve in 1 to 9 months (Dropcho, 1991). Conversely, delayed myelopathy is irreversible, has a latency period of 9 to 18 months, and generally occurs within 30 months (Dropcho, 1991). The incidence is reported at 1% to 12%. The latency period is decreased with increased radiation dose and in children (Leibel et al., 1991). The onset of symptoms begins with lower extremity paresthesias and is followed by sphincter disturbance. Partial Brown-Séquard syndrome (motor weakness on one side and some sensory changes on the contralateral side) may occur below the level of injury. Central pain syndrome may also occur, which is typically characterized by mid-back pain and dysesthetic pain in the lower extremities. Such pain is usually treated with steroids, anticonvulsants, and tricyclic antidepressants. Rehabilitation concerns are listed in Table 22–5.

**Rehabilitation Considerations**

In cases of myelopathy or significant radiculopathy, physical and occupational therapists should be consulted early to address ADLs and ambulation and to provide adaptive equipment and strengthening exercises. When spinal metastasis has occurred, other bony areas may also be affected, particularly the pelvis, femur, ribs, and skull. When there is skull involvement, compromise of adjacent neurologic structures can occur. Use of orthoses to limit spinal mobility, such as sternal-occipital-mandibular immobilization (SOMI), may be required for spinal stability for patients with tumors affecting the spine. Rigid thoracic-lumbar-sacral orthoses with a "clamshell" design can provide good external support but may not be tolerated by patients with painful rib or iliac crest bony involvement or by those with fragile skin due to steroids or chemotherapy (Garden and Gillis, 1996). The rehabilitation team must consider metastatic disease as a possible etiology for new pain or weaknesses that arise during the course of therapy. Adequate pain control is essential and enables patients to participate in therapy.

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**Table 22–3. Findings Associated with Better Prognosis for Functional Recovery Following Cord Compression**

- Diagnosis of myeloma, lymphoma, or breast cancer
- Slow evolution of symptoms or early neurologic signs
- Ambulatory status at time of diagnosis of spinal cord involvement

**Table 22–4. Findings Associated with a Poorer Prognosis for Functional Recovery Following Cord Compression**

- Sphincter incontinence
- Complete paraplegia
- Rapid evolution of symptoms (<72 hours)

**Table 22–5. Rehabilitation Issues in Cancer and Treatment-Related Myelopathy**

- Pain
- Motor loss and difficulty with ambulation and transfers
- Sensory loss
- Autonomic dysreflexia (T6* or above)
- Orthostatic hypotension
- Neurogenic bowel and bladder
- Spasticity
- Pressure ulcers at sacrum, heel and trochanters
- Spinal instability (with spinal column destruction)
- Altered weight-bearing, limited lower extremity range of motion

*T6, The sixth thoracic spinal cord level.
Pain Management

Pain (see later in this chapter and Chapter 23) may be both acute and chronic. Pharmacologic options include opiates, nonsteroidal anti-inflammatory agents, tricyclic antidepressants, various antiepileptics, steroids, and other medications such as β-blockers and α-adrenergic agonists. Use of modalities such as heat, cold, and transcutaneous electrical nerve stimulation (TENS) should be considered. Nontraditional interventions such as acupuncture have also been used with success. In patients with spinal hardware, worsening pain could indicate malfunction or loosening of hardware or infection in the surrounding tissues.

Bladder Management

Patients with myelopathy can develop detrusor-sphincter-dyssynergia (DSD), a condition in which the bladder contracts but the bladder outlet (sphincters) fails to relax in a timely manner, leading to impaired emptying and increased bladder pressure. Patients may attempt to void on their own; however, postvoid residual volumes must be checked on multiple occasions to confirm complete emptying. If incomplete emptying occurs (i.e., residual volume >100 to 150 cc), intermittent catheterization should be performed every 4 hours. The goal is to have no more than 350 to 400 cc of urine in the bladder at any time to avoid overdistension, detrusor muscle injury, and retropropulsion of urine into the ureters. With chronically increased bladder volumes, bladder flaccidity may occur secondary to detrusor muscle injury.

The amount of urine produced is affected by the volume of fluid intake, medications, and hormonal abnormalities, such as SIADH, which may be seen particularly with lung malignancies and pituitary adenomas. Certain types of foods and drinks may also act as diuretics. Fluid intake should initially be restricted to 2 L per day if other medical concerns permit. The frequency of bladder catheterization may at first be kept at every 4 to 6 hours and can be adjusted so that bladder volumes do not exceed 400 cc. Patients should not restrict fluid intake simply to avoid catheterization. The intermittent catheterization program (ICP) should be taught to patients and caretakers. Patients with a cord injury at C7 or below can usually learn to independently perform such a program. Condom catheters may be used by men with hyperactive bladder (without dyssynergia) or those with normal bladder function but with incontinence due to impaired cognition or mobility. Indwelling catheters may be needed in women who cannot perform ICP and in men who cannot wear condom catheters or who have contraindications to ICP.

Bowel Management

A bowel program (more details follow in a later section) with fiber, stool softeners, and digital stimulation, along with judicious use of suppositories, laxatives, and enemas should be started. Warm fluids may be given after meals to supplement the gastrocolic reflex. Patients should be allowed to sit on a commode at regular times to facilitate bowel movements. Establishment of a set pattern (daily or every other day) for evacuation will minimize constipation and incontinence.

Management of Autonomic Dysreflexia

Autonomic dysreflexia is a medical emergency that occurs when a patient manifests a massive sympathetic discharge in response to a noxious stimulus. The clinical presentation is that of an anxious patient with paroxysmal hypertension, nasal congestion, sweating above the level of lesion, facial flushing, piloerection, and reflex bradycardia. Autonomic dysreflexia typically occurs with a spinal cord injury at the level of T6 or above. Most commonly, the noxious stimulus is a distended bladder or bowel. Other causes include enemas, tight clothing, infection, deep venous thrombosis, ingrown toenails, bladder catheterization, and pressure ulcers. Treatment focuses on eliminating the underlying noxious stimulus, such as emptying of the distended bladder or bowel. Such measures usually resolve the episode quickly, however, if a cause cannot be found promptly, treatment with antihypertensives must be initiated to prevent complications of rising blood pressure.

REHABILITATION OF PATIENTS WITH PLEXOPATHIES

Aplexopathy may result from direct invasion by neoplasm or from radiation injury (see Chapter 18).
Chemotherapy can enhance the radiation-induced injury in irradiated tissue and decrease the latency period for development of plexopathy. Predominant symptoms are pain and paresthesias. Clinical signs include sensory loss, decreased or absent reflexes, and weakness.

### Brachial Plexopathy

Plexus lesions can result from compression or infiltration by tumor lying in contiguous tissues or may be seen after RT for breast and lung cancers. Characteristic differences between neoplastic and post-radiation brachial plexopathies are listed in Table 22–6.

Pain typically occurs in the shoulder, elbows, hand, and fourth/fifth digits, whereas sensory disturbance occurs in the axilla with C8, T1, and T2 involvement. Breast cancer in particular may affect the upper brachial plexus, where pain referral is to the paraspinal region, shoulders, biceps, elbow, and hand. Burning dysesthesias in the index finger or thumb are common. A hallmark of this syndrome is the neuropathic character of the pain, with numbness, paresthesias, allodynia, and hyperesthesia complaints. All patients with brachial plexopathy should have a scan of the contiguous paravertebral region before RT because extension of disease is common in this area. Epidural invasion can occur in some patients with brachial plexopathy. Imaging of the epidural space is essential when a patient develops Horner’s syndrome, panplexopathy, or vertebral body erosion or if a paraspinal mass is detected on CT scan. With radiation dosages exceeding 60 Gy, or large fractions of >190 cGy/day, fibrosis of the plexus can occur.

### Lumbosacral Plexopathy

Pelvic malignancies, including bladder, uterus, prostate, and/or lung cancer or melanoma can lead to lumbosacral plexopathy. Retroperitoneal tumors, including sarcomas and metastatic nodal tumors, may affect the lumbosacral plexus or its roots more proximally (Table 22–7). The presenting symptom is usually pain in the buttocks or legs, and it often precedes other symptoms by weeks or months. Other symptoms include numbness, weakness, and later edema. Pain is usually of an aching or pressure-like quality, rarely burning (Caraceni and Martini, 1999b).

<p>| Table 22–6. Characteristic Differences Between Neoplastic and Post-radiation Brachial Plexopathies |</p>
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Neoplastic</th>
<th>Post-radiation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>10 times more common</td>
<td>Dose related</td>
</tr>
<tr>
<td>Initial symptom</td>
<td>Progressive pain 90%</td>
<td>Numbness, paresthesias, pain in &lt;20%</td>
</tr>
<tr>
<td></td>
<td>(preceding other symptoms by weeks/months)</td>
<td>Pain stabilizes with onset of weakness</td>
</tr>
<tr>
<td>Signs</td>
<td>Lower trunk, Horner’s syndrome</td>
<td>Upper trunk</td>
</tr>
<tr>
<td>Progression rate</td>
<td>Slow</td>
<td>Insidious, self-limiting</td>
</tr>
<tr>
<td>EMG</td>
<td>Denervation, no myokymia</td>
<td>Myokymia</td>
</tr>
</tbody>
</table>

<p>| Table 22–7. Characteristic Differences Between Neoplastic and Post-radiation Lumbosacral Plexopathies |</p>
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Neoplastic</th>
<th>Post-radiation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial symptoms</td>
<td>Pain in 93%, paresthesia</td>
<td>Weakness in 50%</td>
</tr>
<tr>
<td>Signs</td>
<td>Bilateral in 10%–25%</td>
<td>Bilateral in 80%</td>
</tr>
<tr>
<td>Latency</td>
<td>Variable</td>
<td>Median 5 yr (1–31 yr)</td>
</tr>
<tr>
<td>Tumor progression</td>
<td>Focal CT/MRI changes</td>
<td>No focal abnormality</td>
</tr>
<tr>
<td>EMG</td>
<td>Denervation</td>
<td>Myokymia</td>
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Lesions most commonly occur in the lower plexus (L4–S1) with an incidence of 50%, followed by upper lumbar plexus (L1–L4) at 33%. Pampxopathy involving two sites is less common at 20% (Jaeckle et al., 1985).

Pain management may require tricyclic antidepressants, antiepileptic agents such as gabapentin and carbamazepine, along with epidural catheters and neurosurgical approaches in more resistant cases. Neuropathic pain may also respond to radiation treatment. After acute inflammation and pain subside, low-resistance weight exercises and functional activities should be encouraged. Neck exercises and range of motion programs should be included in the treatment of brachial plexopathy, especially with injury to the upper portion of the plexus, to avoid functionally devastating contractures in that area. A sling should be given to prevent glenohumeral subluxation. With lower extremity involvement, assistive devices for ambulation, such as a cane, may be required by those patients with weakness and proprioceptive feedback loss. Orthoses and splints may be required for joint or limb support or to enable function and prevent injury.

**REHABILITATION OF PATIENTS WITH NEUROPATHIES**

Peripheral polyneuropathy may occur as a result of direct tumor invasion, as part of a paraneoplastic syndrome, or with chemotherapy (see Chapter 17). Paraneoplastic neuropathy may be related to an autoimmune process and may be sensorimotor in nature (see Chapter 19). Chemotherapy-associated peripheral neuropathies are generally distal and symmetric. Most frequently these neuropathies occur with Vinca alkaloid, taxane, or platinum-based therapies (Amato and Collins, 1998). Symptoms include numbness, paresthesias, and occasionally severe neuropathic pain.

Tricyclic antidepressants and antiepileptics should be given to manage pain. Adaptive strategies such as energy conservation, orthotics, and assistive and adaptive devices are prescribed. With sensory loss in the lower extremity, preventive measures such as those used for management of diabetic neuropathy and neuropathic ulcers should be instituted. Education, nonconstricting footwear, and daily inspection of feet are important in such cases. Patients with acute inflammatory demyelinating polyneuropathy benefit from a resistive exercise program.

**REHABILITATION OF PATIENTS WITH LEPTOMENINGEAL DISEASE**

Leptomeningeal disease is also known as carcinomatous meningitis or meningeal carcinomatosis (see Chapter 16). It is caused by dissemination of cancerous cells throughout the subarachnoid space. Life expectancy is usually very short, often only 3 to 6 months with treatment (Sause et al., 1988; Siegal et al., 1994). Both central and peripheral nervous system involvement can occur, along with cerebrospinal fluid flow obstruction leading to hydrocephalus. Symptoms can include mental status changes, polyradiculopathy with radicular pain, and cauda equina syndrome. Rehabilitation management is similar to that outlined earlier, based on the sites involved and the deficits encountered. The rehabilitation goals should include supportive and safety concerns and reflect the generally poor prognosis.

**REHABILITATION OF PATIENTS WITH MUSCULOSKELETAL PROBLEMS**

Primary and metastatic nervous system cancers and their treatments can cause multiple musculoskeletal problems. These problems can significantly compromise normal function. They require an accurate diagnosis, assessment of functional impairments, and implementation of appropriate rehabilitation interventions.

**Corticosteroid-Induced Myopathy**

Myopathies are a group of muscle diseases whose common principal symptom is weakness, usually in the proximal muscles of the shoulder and hip joints. Steroid myopathies most commonly occur in patients who undergo high-dose, long-term corticosteroid therapy. These patients generally show recovery after decreasing or discontinuing medication. Myopathy patients usually present with difficulty climbing stairs, rising from chairs, and performing transfers. Inpatient rehabilitation is sometimes necessary to regain strength, learn to perform ADLs safely, and use ambulatory aids. In the most severe cases, patients may
require a wheelchair for all mobility and ADLs, but can achieve independence in those functions despite the use of a wheelchair. In such cases, adaptive equipment (e.g., transfer board, raised toilet seat, bathtub bench) is necessary for them to perform ADLs safely. As patient strength improves, gait training under the supervision of a physiatrist and physical therapist can continue on an outpatient basis.

**Avascular Necrosis and Osteoporosis**

Avascular necrosis and osteoporosis frequently occur in cancer patients. These problems are diagnosed radiographically and may be asymptomatic until the involved bone is subject to fracture or infection. Most avascular necrosis is attributable to the direct effects of RT or to the systemic effects of corticosteroids, although these effects appear to be dose dependent (Zizic, 1991). In addition to RT and steroids, avascular necrosis in cancer patients has been anecdotally associated with single-agent cyclophosphamide or methotrexate, as well as cyclophosphamide in combination with methotrexate and 5-fluorouracil (Pizzo and Poplack, 1997).

Like avascular necrosis, osteoporosis has been related to steroids and to RT (Duthie and Katz, 1998; Tefft et al., 1976). Glucocorticosteroids inhibit new bone formation and calcium absorption and increase bone resorption and renal calcium excretion. Steroid-induced hypogonadism contributes to the problem in both men and women. More than 50% of patients taking long-term steroids develop some degree of osteoporosis (Goroll et al., 1995). The risk of developing steroid-induced osteoporosis can be reduced by using a short-acting preparation at the lowest possible dose in an alternate-day regimen, by maintaining physical activity, and by ensuring adequate daily intake of calcium and vitamin D. Treatments for osteoporosis include:

1. Therapies used to slow down bone involution and prevent contracture formation and postural deviations: weight-bearing exercises, upper and lower extremity muscle strengthening, balance training, back extension and chest expansion exercises, pectoralis muscle stretching, posture correction, and proper lifting techniques
2. Cessation or tapering of glucocorticoid intake
3. Hormone replacement therapy for men and women who do not have contraindications
4. Thiazide therapy for hypercalcemia
5. Vitamin D and calcium supplementation when appropriate
6. Bisphosphonate therapy
7. Environmental modification: proper footwear, adjustment of medications that may contribute to falling; assistive devices
8. Education of patients regarding risk factors such as smoking

Compression fractures may ensue with only minor trauma once sufficient structural integrity is lost. Pain may be managed with analgesic and anti-inflammatory medications and the use of spinal orthoses. Surgical fixation is sometimes necessary to attain stability. Early weight-bearing and limited immobilization should be encouraged to minimize continued bone loss.

**Contracture of Joints**

A limitation of passive joint range of motion, contracture commonly results from a restriction in connective tissue, tendons, ligaments, muscles, and joint elements. Contractures are most often related to spasticity, bed rest, localized heterotopic ossification, bleeding, infection, trauma, and edema. Prevention is achieved by minimizing the duration of bed rest and encouraging daily range of motion exercises.

**Heterotopic Ossification**

Heterotopic ossification is the formation of mature, lamellar bone in soft tissues. The variable incidence of heterotopic ossification has been reported in spinal cord injury patients (20% to 25%) and in head injury patients (10% to 20%) (An et al., 1987; Brooker et al., 1973; Evans, 1991; Garland, 1988; Ishikawa et al., 1982; Jensen et al., 1988; Stoikovic et al., 1955; Storey and Tegner, 1955; Stover et al., 1975). This condition has been observed in patients who require a prolonged ICU stay and is rarely seen in the cancer population. The chief symptoms of heterotopic ossification are joint and muscle pain and compromised range of motion. A triple-phase bone scan is able to detect heterotopic ossification at an early stage. Therapies include bisphosphonates, nonsteroidal anti-inflammatory drugs, RT, and physical therapy. Passive joint mobilization helps to maintain or increase joint mobility without promoting heterotopic ossifi-
cation. Matured heterotopic ossification can be surgically excised.

**Shoulder Pain**

This problem often occurs in neurologically impaired populations. Shoulder pain may originate from rotator cuff tears, bicipital tendinitis, adhesive capsulitis, and subdeltoid bursitis. Other causes of shoulder pain in the hemiplegic population include excessive shoulder capsule stretch secondary to paresis of shoulder musculature, sympathetically maintained pain (reflex sympathetic-dystrophy, shoulder-hand syndrome), and thalamic syndrome. Management is diagnosis dependent.

**GENERAL REHABILITATION PATIENT CARE CONCERNS**

**Physiologic Deconditioning**

Immobilization syndrome may occur during recovery from operative or other treatments, be caused by medical complications, or result from the neurologic sequelae of cancer. It negatively impacts multiple body systems and often causes a decline in the patient’s functional status. Immobilization can contribute to intellectual, emotional, and behavioral disturbances, decreased muscle strength and endurance, poor coordination, and contracture of joints. Cardiovascular and pulmonary deconditioning may present with orthostatic hypotension, deep vein thrombosis, decreased vital capacity, and impairment of the cough mechanism. Anorexia, constipation, electrolyte disturbances, and pressure ulcers are also manifestations of immobilization (Hoffman et al., 1998). The best management of this syndrome is through prevention. Physical therapy should begin early, emphasizing progressive mobilization, starting with passive range of motion if necessary, progressing to assisted active range of motion; then to active range of motion. When postural hypotension is pronounced or when patients have been or are expected to be bed bound for more than one week, tilt-table use should begin as soon as the patient is stable. This device is beneficial for cardiovascular and respiratory reconditioning and can also help prevent osteoporosis. Once the patient tolerates a 70-degree angle for 30 minutes, standing and ambulation should begin. Signs and symptoms of hypercalcemia, pressure ulcer, urinary tract infection, and pneumonia should be watched for vigilantly.

**Venous Thromboembolism**

Deep venous thrombosis (DVT) has a high incidence in stroke patients (30% to 50%) (Gibberd et al., 1976), in spinal cord injury patients (Merli et al., 1993; Weinmann and Salzman, 1994), after hip arthroplasty (Imperiale and Speroff, 1994), in patients with cancer (Marik et al., 1997), and in immobilized patients (Giuntini et al., 1995). Elastic hosiery and sequential compression pumping of the calves should be continued until mobilization is underway. In spinal cord-injured and hemiplegic patients, administration of subcutaneous low-molecular-weight heparin is recommended. Exceptions are made for patients following intracranial surgery to avoid devastating hemorrhage. Patients with thrombocytopenia, especially those with hematologic malignancies and hemorrhagic tumors, require individualized assessment, and their anticoagulation risks should be addressed with the primary oncology team. A consensus on the optimal duration of prophylactic anticoagulation has not yet been reached.

A high index of suspicion for thrombosis should be maintained for patients with recent surgery or anesthesia, smoking history, current or recent prolonged bed rest, prior history of DVT, cardiac disease, obesity, extremity trauma, neoplasm, and in the elderly. If DVT is suspected, Doppler ultrasound and venography can be used to confirm the diagnosis. Once the clinical diagnosis of DVT is seriously considered or confirmed, intravenous heparin can be started, followed by oral anticoagulation therapy for a 3 month period. When a pulmonary embolism has occurred, 6 months of treatment is usually suggested (Bone et al., 1998).

**Spasticity**

Spasticity is a motor disorder characterized by a velocity-dependent resistance to movement associated with exaggerated phasic stretch reflexes (tendon jerks), representing one component of the upper motor neuron syndrome. Tone is the sensation of resistance felt by the examiner as passive range of motion is tested. Spasticity can be caused by a wide variety of disorders that damage descending motor tracts at the cortical, subcortical, brain stem, or spinal cord.
levels. Examples include primary CNS tumors, metastatic CNS tumors, and radiation injury of the CNS.

Spasticity can be quantified by using the Modified Ashworth Scale (Table 22–8). Only those patients whose spasticity interferes with present function or potential future function, or whose condition is painful, should be treated. Spasticity treatment should begin with the least invasive techniques and advance as needed. Basic treatment includes a daily stretching program, use of proper positioning, and avoidance of noxious stimuli. Topical cold may provide short-term benefit. Casting and splinting techniques can improve the range of motion in hypertonic joint contractures. Oral medications (Table 22–9) to treat spasticity should be combined with basic treatment and are only effective in mild to moderate cases. Chemical neurolysis, such as phenol block, injections, epidural infusion of medications, botulinum toxin via an implantable pump, and surgery are options for severe spasticity management.

Table 22–8. Clinical Scale for Spastic Hypertonia (Modified Ashworth Scale)

<table>
<thead>
<tr>
<th>Scale</th>
<th>Physical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No increase in tone</td>
</tr>
<tr>
<td>1</td>
<td>Slight increase in muscle tone manifested by a catch and release or by a minimal resistance at the end of the ROM when the affected part(s) are moved in flexion or extension</td>
</tr>
<tr>
<td>1+</td>
<td>Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than one-half) of the ROM</td>
</tr>
<tr>
<td>2</td>
<td>More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved</td>
</tr>
<tr>
<td>3</td>
<td>Considerable increase in muscle tone and passive movement difficult</td>
</tr>
<tr>
<td>4</td>
<td>Affected part(s) rigid in flexion or extension</td>
</tr>
</tbody>
</table>

ROM, Range of motion of a joint.

Skin and Wound Care

After Radiation Therapy

Radiation may impair wound healing and cause skin tightening. Persistent wound drainage with impaired wound healing, cutaneous fistulas, electrolyte imbalances, decreased protein reserves, and infections may also develop. Prior radiation and ongoing chemotherapy can disrupt normal wound healing, thus increasing the likelihood of postoperative wound infection and dehiscence (Alekhteyar et al., 1996; Springfield, 1993). Changes in skin integrity with radiation encompass local skin reactions, which may include epilation (loss of hair), erythema, and dry and wet desquamation. With a short course of cranial irradiation, mild scalp erythema may occur, especially around the external pinna. Complete alopecia is a more common problem with longer courses of cranial treatment; hair regrowth may take as long as 2 to 3 months. With larger dosages of radiation

Table 22–9. Oral Spasmolytic Medications

<table>
<thead>
<tr>
<th>Agent</th>
<th>Daily Dosage</th>
<th>Half-Life (Hours)</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baclofen</td>
<td>10 to &gt;80 mg</td>
<td>3.5</td>
<td>Presynaptic inhibitor by activation of GABA B receptor</td>
</tr>
<tr>
<td>Diazepam</td>
<td>4 to &gt;60 mg</td>
<td>27–37</td>
<td>Facilitates postsynaptic effects of GABA, resulting in increased presynaptic inhibition</td>
</tr>
<tr>
<td>Dantrolene</td>
<td>25 to &gt;400 mg</td>
<td>8.7</td>
<td>Reduces calcium release, interfering with excitation-contraction coupling in skeletal muscles</td>
</tr>
<tr>
<td>Clonidine</td>
<td>0.1–0.4 mg (oral); 0.1–0.3 mg (patch)</td>
<td>12–16 (oral)</td>
<td>α2-adrenergic agonist</td>
</tr>
<tr>
<td>Tizandine</td>
<td>4–36 mg</td>
<td>4</td>
<td>α2-adrenergic agonist</td>
</tr>
</tbody>
</table>

GABA, Gamma-aminobutyric acid.
(>40 Gy) to the scalp, hair loss is usually permanent. Combination of irradiation with cytotoxic agents such as doxorubicin or actinomycin D may significantly increase skin toxicity (D’Angio et al., 1976).

For radiation-induced changes, skin should be kept dry and clean without use of lotions. Exposure to sunlight and temperature extremes must be avoided. Alterations in sweat glands may lead to drying, so petrolatum may be useful. Cornstarch may alleviate pruritus.

### Pressure Ulcers

Pressure and shear forces are the two most important factors in ulcer formation. Risks are persistent pressure to the skin located above a bony prominence, shear forces, friction, and sensory deficits. Poor nutritional status and contact with moisture (such as urine, feces, or wound drainage) compound the problem. In bed-bound patients, the most common site for pressure ulcer formation is the sacrum, followed by the heels, ischium, scapula, and occiput. Prolonged pressure across a bony prominence initially causes damage to the overlying muscle. Prevention entails frequent turning (every 2 hours), daily skin checks, avoidance of friction and excessive moisture or dryness, and the use of specialized mattresses in high-risk situations. Understanding universal criteria like those listed in Table 22–10 can assist in treatment.

When ulcers develop, treatment requires complete pressure relief for healing to occur. Patients who cannot maintain positions of pressure relief and develop stage II ulcers should use low air-loss mattresses, whereas with stage III and IV ulcers, air-fluidized beds may be beneficial. Higher stage ulcers require plastic surgery consultation. Orthotic devices that elevate and disperse pressure over the heels will usually prevent pressure ulceration. Conditions that potentially aggravate wounds such as diabetes, hypoproteinemia, and infection, should be treated. Supplementation with zinc and vitamin C may be helpful.

### Bowel and Bladder Management

Constipation may result from prolonged immobilization or develop secondary to changes in metabolic demand, endocrine function, or decreased gastric and intestinal motility. Constipation in cancer patients is frequently related to narcotic medication. Some patients may present with diarrhea due to impaction rather than lack of bowel movements.

For patients with neurogenic bowel, establishing a consistent bowel program early in the course of treatment is extremely important. The management of a typical reflexic neurogenic bowel consists of a diet high in fiber to improve transit time, stool softeners, digital stimulation with or without suppositories, judicious use of laxatives, enemas in case of impaction and at the inception of the program, and performance of the bowel program 30 to 60 minutes after a meal to utilize the gastrocolic reflex to assist with peristalsis. This management can also be applied to the patient with constipation caused by prolonged bed rest and narcotic medication, omitting the digital stimulation component. Patients with thrombocytopenia (<10,000) or severe neutropenia should not be given suppositories or utilize digital stimulation.

Patients with lower motor neuron injuries, such as conus or cauda equina injuries or pudendal nerve injuries, have an areflexic bowel and a hypotonic external sphincter and are often more difficult to successfully manage. Excessive stool softeners may increase bowel accidents, and digital stimulation and cathartic suppositories are of limited use. Manual removal, straining, and enemas are often the only means of emptying the lower colon in this patient group.

Assuming an upright posture as frequently as possible, increasing ambulation, and maintaining an adequate fluid intake will help minimize difficulties initiating a urinary stream. An intermittent catheterization program (ICP) can be used in cases of urinary retention. Timed voiding is the management of choice for patients with an intracranial lesion and hyperreflexic bladder. Patients with spinal cord lesions may present with either a failure to store urine or a failure to empty the bladder.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Characteristic</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Nonblanchable erythema</td>
</tr>
<tr>
<td>II</td>
<td>Partial skin loss (epidermis to dermis)</td>
</tr>
<tr>
<td>III</td>
<td>Full thickness</td>
</tr>
<tr>
<td>IV</td>
<td>Damage through level of deep fascia, muscle or bone</td>
</tr>
</tbody>
</table>
The goals for neurogenic bladder management are to promote preservation of the upper urinary tract, maintain low storage and evacuation pressures in the bladder, and ensure patient compliance by choosing a technique appropriate for his or her lifestyle. A basic evaluation should include a clear history for difficulty or inability to void and a neurologic examination. The examination should include perianal sensation (touch and pinprick), anal tone and voluntary contraction of the anal sphincter, and bulbocavernous reflex. Evaluating the prostate size during rectal examination is important for assessing obstruction.

Urodynamic studies should be performed for patients with spinal cord lesions whose survival is expected to exceed 1 year. Postvoid residual (PVR) measurements should be routinely done for all patients with known or suspected spinal cord injuries. A PVR > 100 ml or more than 20% of the total voided urine is considered abnormal, and catherization with a straight catheter (14 French) should be continued every 4 hours with a bladder volume goal of no more than 500 cc. These patients should later be objectively evaluated by urodynamic study and treated accordingly.

Failure of the bladder to store urine is treated with anticholinergic medications, such as oxybutynin chloride (5 mg orally two to three times/day), or propantheline bromide (5 to 30 mg orally three to four times/day), or dicyclomine hydrochloride (10 to 20 mg orally four times/day). Failure of the bladder to empty secondary to a hyperreflexic sphincter in male patients can be treated with a combination of external sphincterotomy and use of an external collecting device.

Nutrition
Proper nutrition is an important aspect of rehabilitation. Cancer and its associated treatments can adversely affect nutritional status. Weight loss may be due to an increase in energy requirements and/or decrease in oral intake, directly or indirectly related to the cancer. Some of the direct nutritional effects include the physical location of a tumor leading to obstruction of the alimentary canal and the type of surgical treatment rendered. Indirect effects occur with decreases in appetite related to the release of cytokines and with the nausea and vomiting associated with chemotherapy.

Rapidly reproducing cells of the gastrointestinal tract are vulnerable to the effects of chemotherapy. Acutely, nausea, vomiting, and anorexia are the most common gastrointestinal side effects. Delayed side effects may include stomatitis, mucosal ulceration, pharyngitis, gastroenteritis, glossitis, and malabsorption. Nutritional deficiencies may also occur with chemotherapy.

Surgical cancer interventions may interfere with the ability to eat. Radiation treatment of the head and neck region may lead to alterations in taste and saliva production. Changes to the oral mucosa cause distortion of temperature and texture sensations. Other post-radiation changes adversely affecting nutrition include nausea, vomiting, anorexia, and esophagitis.

Medical treatment should be given as necessary to prevent or reduce nausea, vomiting, hyposalivation, and decreased appetite. Antiemetics include phenothiazines such as prochlorperazine, promethazine, and chlorpromazine. Selective 5-HT3 receptor antagonists for treatment of nausea and vomiting include ondansetron, dolasetron, and granisetron. Cannabinoid medications (dronabinol) and trimethobenzamide may also be used. To prevent dry mouth and hyposalivation, anticholinergic medications should be avoided and lubricating mouth products should be used as necessary. Agents for appetite stimulation are dronabinol, megestrol, and cyproheptadine.

Patients often reject specific foods or certain flavors during the course of cancer treatment. Such behavior may be associated with side effects following consumption of certain foods, such as meats, vegetables, and caffeineated beverages (Mattes et al., 1987). To avoid this aversion to familiar food items, consumption should occur 24 hours before nausea-producing therapy (Gerber and Vargo, 1998). Intake of other high protein sources should be encouraged, such as dairy products, eggs, and liquid nutritional supplements. Cold foods and foods with little odor and less seasoning may be better tolerated.

In order to speed recovery after anticancer treatments and for general improvement in functional status, optimal nutritional status should be maintained. Caloric intake should range from 115% to 130% of the patient’s resting metabolic rate. Protein requirements range from 1.5 to 2.5 g/kg per day (Burgess, 1989; Maillet, 1987). Vitamin and mineral supplementation should be given as needed. Fluid and electrolyte balance should be verified in the presence of malabsorption, diarrhea, and large wounds with...
associated fluid losses. Enteral or parenteral feeding supplementation should be considered without delay in nutritionally compromised patients who are not eating well. Nutritional status may be followed with albumin, pre-albumin, serial weights, lymphocyte count, and calorie count.

**Pain**
Distinguishing whether pain (see Chapter 23) is acute or chronic in nature can assist in selecting appropriate management. Pain tends to be less prominent in patients with brain tumors, but may be significant with spinal column and cord involvement. In patients with spinal stabilization using hardware, increasing pain should prompt an evaluation of the construct’s integrity. Headaches occur in 48% to 71% of patients with brain tumors. These are usually mild to moderate and can resemble tension headaches but may increase with changes in position (Forsyth and Posner, 1993; Suwanwela et al., 1994; Caraceni and Martini, 1999b). Increasing severity or accompanying nausea and vomiting may signify increasing intracranial pressure, which often responds to steroids (Caraceni and Martini, 1999b).

Medical management typically includes non-steroidal anti-inflammatory agents (NSAIDS) and non-narcotic and narcotic analgesics. Neuropathic pain, which may be seen with spinal cord involvement, can be managed with tricyclic antidepressants, anticonvulsants, steroids, and occasionally opiates. Tricyclic agents may potentiate opiate analgesia. Antihistamine agents such as hydroxyzine may help with analgesia and provide antiemetic effects, but these usually occur only with relatively high parenteral dosages (Beaver and Feise, 1976). Benzodiazepines may be helpful in managing anxiety or muscle spasms but are not useful for analgesia (Beaver et al., 1966). Short-term administration of high-dose corticosteroids can provide significant pain relief in patients with bony or neural structure involvement. Dosage of steroids should be tapered as alternative means are implemented (Ettinger and Portenoy, 1988; Brueera et al., 1985). Bisphosphonates should be considered for patients with refractory bone pain (Payne, 1989). Anticholinergic drugs like scopolamine should be considered for refractory pain from bowel obstruction. Neurostimulants such as methylphenidate and dextroamphetamine can be analgesic in low doses (Brueera et al., 1987).

Physical medicine modalities for pain control can serve as an adjunct to cancer pain management (U.S. Department of Health and Human Services, 1994). Cold modalities are generally safe. Heat modalities can be superficial or deep (usually ultrasound) and may increase circulation to the involved area. However, this method may increase the potential for metastatic spread, and application of ultrasound over malignant tissues is generally contraindicated. Transcutaneous electrical stimulation (TENS) is particularly helpful in the management of radiculopathy or incisional pain. Conventional high-frequency settings are usually effective, but expertise in electrode placement may be required to attain pain relief. Trigger point injections can help myofascial pain. Nerve blocks, epidural injections, and ablative surgical procedures may also be useful for treating acute pain.

Appropriate use of orthotics can be invaluable. Examples include shoulder support with a sling in patients with malignant brachial plexopathy or glenohumeral subluxation with brain tumor; or use of cervical, thoracic, and lumbosacral orthoses for patients with metastatic spinal involvement. Psychological approaches including hypnosis, relaxation training, and distraction techniques may be considered. Acupuncture has also been useful in acute pain management.

**Delirium and Dementia**
Alterations in consciousness may occur during the rehabilitation evaluation or treatment course and require accurate diagnoses and intervention to maximize functional outcome. Delirium (see Chapter 27) is a confusional state with an acute onset, manifesting as a global impairment of mental function. It occurs frequently in elderly cancer patients (Breitbart and Cohen, 1998). The causes of delirium include a variety of drugs, primary intracranial diseases, systemic diseases secondarily affecting the brain, withdrawal from alcohol or sedative-hypnotic medications, metabolic disorders such as hyponatremia and hypoglycemia, infections, and seizures. Determination of the causative agent or factor and removal or correction of the cause is the primary treatment.

Detecting dementia is important for rehabilitation decision-making. Rehabilitation is based on a patient’s ability to learn and retain information. Moderately or severely demented patients have limited rehabilitation potential due to their difficulty retaining
new information. A brief trial of rehabilitation may still be justified in such situations to train caregivers and to clarify learning abilities. Too often, mental status observations in the acute hospital setting underestimate the patient’s cognitive function in a more supportive and stimulating environment and their function following resolution of acute illness. Discharge planning for patients with dementia needs to include caregiver education to ensure awareness of the individual’s cognitive strengths and weaknesses and instructions for how to handle potential behavioral problems. Community resources and educational materials can be very helpful to caregivers. The incidence of dementia is higher in the cancer patient population for the following reasons:

1. Occurrence of leukoencephalopathy secondary to chemotherapy such as intrathecal chemotherapy, especially the combination of irradiation and methotrexate (Abrey et al., 1998)
2. Slowly progressing viral infections (Manuelidis et al., 1988)
3. Radiation-related dementia characterized either by dementia alone or by dementia with gait abnormalities and incontinence. A small number of patients will also have hydrocephalus and benefit by ventricular-abdominal shunting (Asai et al., 1989)

Parkinsonism

The major clinical features of Parkinson’s disease are recognized as a symptom complex manifested by any combination of six cardinal features: tremor at rest, rigidity, bradykinesia-hypokinesia, flexed posture, loss of postural reflexes, and the freezing phenomenon. At least two of these features, with at least one being either tremor at rest or bradykinesia, must be present for a diagnosis of Parkinson’s disease. The biochemical pathology in this disorder is decreased dopaminergic neurotransmission in the basal ganglia. Parkinsonism can occur in cancer patients for the following reasons:

1. Use of dopamine antagonists and depleting agents
2. Radiation injury, including radiation necrosis
3. Hydrocephalus/normal pressure hydrocephalus
4. CNS hypoxia
5. Following encephalitis
6. Parathyroid dysfunction
7. Tumor
8. Multi-infarct state
9. Idiopathic Parkinson’s disease co-existing in the cancer population

The clinical features of tremor, rigidity, and flexed posture are referred to as positive phenomena; bradykinesia, loss of postural reflexes, and freezing are negative phenomena. In general, the negative phenomena are more disabling. Bradykinesia results in difficulty with speech, swallowing, ADL, and mobility. Walking, transferring, and even bed mobility can be affected. Severe bradykinesia prevents these patients from driving due to slowed foot movement between the accelerator and the brake pedal. Loss of postural reflexes leads to increased risk of falls and a high incidence of hip fractures in parkinsonian patients. Affected patients also have cognitive and behavioral signs such as decreased attention span, visuospatial impairment, and personality changes. They are often more fearful, indecisive, and passive, as well as depressed, than is normal (Dropcho, 1991). Autonomic disturbances are also encountered. Patients may experience constipation, urinary retention, hypotension, and/or erectile dysfunction.

Treatment is aimed at controlling symptoms through use of standard antiparkinsonian medications and rehabilitation interventions. Physical and occupational therapies play an important role in maintaining ADL and muscle strength and slowing development of contractures and the accompanying characteristically stooped posture. Functional deficits often worsen disproportionately with periods of immobility; thus mobility should be preserved as much as possible despite intercurrent illnesses. Rehabilitation also involves treatment of dysphagia, management of bowel and bladder problems, and assistance with psychosocial difficulties caused by declining cognition.

Psychological Issues

Psychological symptoms can include reactive anxiety and depression, major depression, and organic brain disorder. The incidence of these disorders is generally greater with higher levels of disability and advanced illness (Breitbart et al., 1998). Symptoms of depression may include anorexia, insomnia, fatigue, weight loss, dysphoric mood, hopelessness, worth-
lessness, excessive guilt, and suicidal ideation (Massie and Holland, 1990). Symptoms are initially likely to be reactive to the diagnosis of a malignancy and then depressive as the functional deficits caused by neurologic impairments are manifested. Endicott (1984) suggested substitution criteria for making the diagnosis of depression, as somatic symptoms of depression might be unreliable and nonspecific in cancer patients.

Anxiety is frequently encountered during the course of rehabilitation. Recognition of anxiety can be challenging in the face of neurologic disease, use of corticosteroids, and other medications. Common signs and symptoms include restlessness, jitteriness, vigilance, insomnia, distractibility, dyspnea, numbness, apprehension, autonomic hyperactivity, and worry. Physical symptoms may be more prevalent than psychological or cognitive ones.

**Sexual Dysfunction**

Sexual dysfunction may be due to a malignancy or its related treatments. It can be affected by changes in nervous, vascular, endocrine, as well as psychological function. Along with depression, patients may feel less sexually attractive. Frontal lobe brain tumors can cause libido changes. Endocrine changes may occur with pituitary involvement and with hormonal treatments for prostate cancer. Hormonal treatments reduce sexual desire and function in most cases. Chemotherapy can cause changes in testosterone production, spermatogenesis, and premature menopause with associated symptoms. Problems include low sexual drive, dry orgasm, vaginal mucosal changes leading to dyspareunia, erectile dysfunction, and decreased pleasure with orgasm (Schover et al., 1993; Gerber and Vargo, 1998). Neuropathies can amplify all of these problems.

Because psychological adjustment is an important determinant of sexual function, counseling should be provided. Patients should be encouraged to pursue intimacy and physical closeness, focusing on various aspects of an intimate relationship. Hormonal replacement therapy should be given for premature menopause when no contraindications are present, along with water-based lubricants. Regular douching should be encouraged to avoid odor. A peer-support system can also be of benefit (Gerber and Vargo, 1998; Garden and Gillis, 1996).

**Family Interaction**

Lack of an adequate support system can be a barrier to successful rehabilitation. Family interventions include counseling, education, and identifying additional support frameworks for the caregiver. Both education and counseling interventions significantly improve caregiver knowledge. Specific techniques for care should be taught, including:

1. Performing physical functions such as transfers, mobility, and other ADL
2. Encouraging patients to perform any activity that he or she is capable of doing
3. Coping and compensatory strategies to deal with cognitive deficits
4. Preventing complications

Common teaching points can include maintaining bowel and bladder function, administering medications, swallow training with appropriate dietary modifications, maintenance of nutrition and hydration, safety training, and a home exercise program.

**Equipment/Orthosis Needs**

Patient equipment needs are usually assessed when they approach discharge or experience a sudden decline in function. Equipment available for in-home medical management includes ventilators, suctioning devices, supplemental oxygen, and tube feeding devices. Mobility equipment includes wheelchairs, walkers, crutches, and canes. Rehabilitation professionals choose devices according to the patient’s functional level. Assistive devices help to achieve an improved level of independence in ADLs and include transfer boards, tub/shower chairs, raised toilet seats, long-handled reachers, sock-aids, elastic shoelaces, dressing sticks, modified eating utensils, and bath and grooming aids.

Orthoses are prescribed for support, alignment, and protection. Four important functions of the upper limb—reaching, carrying, prehension, and release—should be taken into account when considering an orthosis for the upper extremity. Lower extremity orthoses are primarily to assist with safety in weight bearing.

**Discharge Planning/Family Training**

The following factors are considered when planning discharge: architectural barriers, available assistance,
availability of home therapy, and quality of life. A suitable caregiver, either a family member or a hired provider, needs to be adequately trained before discharge. If a patient is not safe cognitively or physically and there is no assistance at home, a nursing home or assisted living facility must be considered. If a patient’s prognosis is poor, hospice care can provide tremendous support for the family and patient and may improve the patient’s quality of life.

**PATIENT OUTCOME GOALS**

**Functional/Social Outcomes**

The most widely used scale for clinical and research outcome measurement in the oncologic literature is the Karnofsky Performance Scale (KPS) (Table 22–11). In the absence of any medical treatment, the KPS was found to be the best determinant of ultimate

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### Table 22–11. Karnofsky Performance Status Scale

<table>
<thead>
<tr>
<th>Activity Level</th>
<th>Scale %</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Able to carry on normal activity and to work; no special care needed</td>
<td>100</td>
<td>Normal; no complaints; no evidence of disease</td>
</tr>
<tr>
<td></td>
<td>90</td>
<td>Able to carry on normal activity; minor signs or symptoms of disease</td>
</tr>
<tr>
<td></td>
<td>80</td>
<td>Normal activity with effort; some signs or symptoms of disease</td>
</tr>
<tr>
<td>Unable to work; able to live at home and care for most personal needs; varying amount of assistance needed</td>
<td>70</td>
<td>Able to care for self; unable to carry on normal activity or to do active work</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>Requires occasional assistance, but is able to care for most of own needs</td>
</tr>
<tr>
<td></td>
<td>50</td>
<td>Requires considerable assistance and frequent medical care</td>
</tr>
<tr>
<td>Unable to care for self; requires equivalent of institutional or hospital care; disease may be progressing rapidly</td>
<td>40</td>
<td>Disabled; requires special care and assistance</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>Severely disabled; hospitalization indicated; although death is not imminent</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>Very sick; hospitalization is necessary; active supportive treatment is necessary</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>Moribund; fatal process progressing rapidly</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>Dead</td>
</tr>
</tbody>
</table>

### Table 22–12. Eastern Cooperative Oncology Group (ECOG) Scale Performance Status

<table>
<thead>
<tr>
<th>Grade</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Fully active, able to carry on all pre-disease performance without restriction</td>
</tr>
<tr>
<td>1</td>
<td>Restricted in physically strenuous activity; ambulatory; able to perform light or sedentary work</td>
</tr>
<tr>
<td>2</td>
<td>Capable of all self-care; ambulatory; unable to perform work activities; up and about more than 50% of waking hours</td>
</tr>
<tr>
<td>3</td>
<td>Only capable of limited self-care; confined to bed more than 50% of waking hours</td>
</tr>
<tr>
<td>4</td>
<td>Completely disabled; cannot carry out any self-care activity; totally confined to bed or chair</td>
</tr>
</tbody>
</table>
patient survival in a national hospice study. With a score of 40, patients lived on the average less than 50 days; with a score of 20, they lived only 10 to 20 days (Reuben et al., 1988).

The KPS does not address cognitive function or quality of life. Additional limitations of this scale, which hinder its use as a functional outcomes measure, are its inability to objectively quantify the amount of assistance needed and its linkage of physical function with medical status. These criteria fail to accommodate severely disabled but otherwise healthy patients. Its linkage of medical status with

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### FIM™ Instrument

<table>
<thead>
<tr>
<th>LEVELS</th>
<th>NO HELPER</th>
<th>HELPER</th>
</tr>
</thead>
<tbody>
<tr>
<td>7 Complete Independence (Timely, Safely)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Modified Independence (Device)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Modified Dependence</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Supervision (Subject = 100%+)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Minimal Assist (Subject = 75%+)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Moderate Assist (Subject = 50%+)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Complete Dependence</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Maximal Assist (Subject = 25%+)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Total Assist (Subject = less than 25%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Self-Care
- A. Eating
- B. Grooming
- C. Bathing
- D. Dressing - Upper Body
- E. Dressing - Lower Body
- F. Toileting

#### Sphincter Control
- G. Bladder Management
- H. Bowel Management

#### Transfers
- I. Bed, Chair, Wheelchair
- J. Toilet
- K. Tub, Shower

#### Locomotion
- L. Walk/Wheelchair
- M. Stairs

### Motor Subtotal Score

#### Communication
- N. Comprehension
- O. Expression

#### Social Cognition
- P. Social Interaction
- Q. Problem Solving
- R. Memory

### Cognitive Subtotal Score

### TOTAL FIM Score

---

**NOTE:** Leave no blanks. Enter 1 if patient not testable due to risk

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**Figure 22–1. Function Independence Measure (FIM). Copyright 1990 by the Research Foundation of the State University of New York.**
work status is often heavily influenced by completely nonmedical factors (insurance, family support, type of work done, and so forth). These criticisms also hold true for the ECOG and similar performance status scales (Table 22–12).

Functional status at presentation was the most important outcome predictor in a study on mortality and functional decline in a group of 92 patients with malignant glioma receiving radiotherapy (Davies et al., 1996). In this study, 13% of predominantly bed-bound patients had functional improvement, whereas 80% of patients presenting with no disability continued to have no deficits at 6 months (Davies et al., 1996). In patients with spinal cord tumor involvement, research suggests a better 1-year survival rate among those who remained ambulatory (66% vs. 10%) (Hill et al., 1993). Bell et al. (1998) found that a population of tumor patients admitted to inpatient rehabilitation units had a generally poorer functional prognosis than did noncancer patients.

Among rehabilitation professionals, one of the most commonly used scales has been the Functional Independence Measure (FIM) (Fig. 22–1). This may also be inadequate to assess brain tumor patients, who have fewer persisting motor and communication disorders than for patients with other neurologic disorders such as stroke (Meyers, 1994).

Quality of Life

General quality of life (see Chapter 26) questionnaires for patients with cancer are listed in Table 22–13. Quality of life status may be more strongly predictive of survival than performance status (Goates et al., 1992; Kaasa et al., 1989; Osoba and MacDon-ald, 1999; Ruckdeschel and Piantadosi, 1991). Factors associated with a better quality of life include absence of depression, good social involvement, greater energy, and fewer symptoms. Age has not been demonstrated to be a significant factor (Bell et al., 1998; Giovagnoli et al., 1996; Mackworth et al., 1992; Weitzner et al., 1996).

Employment

Work is identified in many studies as a significant factor in quality of life. There is little literature regarding employment among patients with neurologic tumors specifically, although in one study of cancer patients only 56% were working (Bell et al., 1998; Rothstein et al., 1995). Cognitive impairment, which would prevent return to work, is more likely among patients with brain tumors compared with other tumors. Kleinberg and associates reported in a study that of 30 primary glioma patients who underwent resection and irradiation, 68% returned to work after treatment, 62% remained at work 1 year later, and 58% were still working 2 to 4 years later (Bell et al., 1998; Kleinberg et al., 1993).

CONCLUSION

Successful rehabilitation of patients with neurologic tumors requires understanding the behavior of tumor pathology, flexibility in determining functional goals and timelines for achievement of goals, and awareness of the complications of cancer and its treatments, which negatively impact patient function. The ultimate goals of rehabilitation interventions are to maximize

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### Table 22–13. General Quality of Life Questionnaires for Patients with Cancer

<table>
<thead>
<tr>
<th>Name</th>
<th>Acronym</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer Rehabilitation Evaluation System Short Form</td>
<td>CARES-SF</td>
</tr>
<tr>
<td>European Organization for Research and Treatment of Cancer Core Quality of Life Questionnaire</td>
<td>EORTC QLQ-C-30</td>
</tr>
<tr>
<td>Functional Assessment of Cancer Therapy</td>
<td>FACT</td>
</tr>
<tr>
<td>Functional Living Index for Cancer</td>
<td>FLIC</td>
</tr>
<tr>
<td>Linear Analog Self-Assessment Scale</td>
<td>LASA</td>
</tr>
<tr>
<td>Medical Outcomes Study Short Form</td>
<td>MOS SF-36</td>
</tr>
<tr>
<td>McGill Quality of Life Questionnaire</td>
<td>MQOL</td>
</tr>
<tr>
<td>Quality of Life Index</td>
<td>QLI</td>
</tr>
<tr>
<td>Rotterdam Symptom Checklist</td>
<td>RSCL</td>
</tr>
</tbody>
</table>
function, promote adaptive and compensatory strategies when full function cannot be restored, and enhance quality of life for cancer patients.

REFERENCES


Rothstein MA, Kennedy K, Ritchie KJ, Pyle K. 1995. Are can-
cer patients subject to employment discrimination? Oncology (Huntingt) 9:1303–1306.