Chapter 6

TRAUMATIC BRAIN INJURY

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INTRODUCTION

The area of traumatic brain injury (TBI) rehabilitation is fast becoming one of the most highly technical, medically complex areas of physiatry. Physiatrists are physicians specializing in Physical Medicine and Rehabilitation (PMR). Given the broad scope and complexity of the field of brain injury rehabilitation, this chapter will provide only an overview of clinically germane topics in TBI rehabilitation rather than a review of all topics pertinent to military personnel following TBI. Important philosophical, clinical, and programmatic aspects have been emphasized regarding the clinical care of this special patient population.

Brain injury rehabilitation, unlike any other subspecialty area in PMR, has undergone a rapid maturation over the last 10 to 15 years. Additionally, the neuromedical multidisciplinary expertise required to care for this special patient population is not matched by any other area of rehabilitation. with which these authors are familiar. Much of the literature pertinent to the clinical care of individuals with TBI is scattered among various medical disciplines, including neurosurgery, physiatry, general surgery, neurology, psychiatry, ophthalmology, otolaryngology, orthopedic surgery, and gastroenterology. There is, however, a growing literature base in areas outside of medicine that is pertinent to rehabilitative care of military personnel with TBI. This literature encompasses areas such as neuropsychology, neurophysiology, neuroanatomy, physical therapy, occupational therapy, speech language pathology, therapeutic recreation, rehabilitation nursing, and rehabilitation social work.

Rehabilitationists have the opportunity to not only support ongoing neurological recovery, but to also affect facilitation of this recovery and allow for better functional adaptation to residual neurologic impairments, as well as physical disabilities. This chapter will consolidate important information regarding rehabilitative care for military personnel with TBI; care which will maximize not only the neurologic and functional outcomes, but also the rehabilitation cost efficiency from "coma to community."

NOMENCLATURE

Although there is general consensus regarding the importance of a common nomenclature within this field of brain injury rehabilitation,[1] one limiting factor has been the lack of consistency in neuromedical and "technical" rehabilitative terminology. Without a standardized nomenclature, clinicians cannot know if they are comparing "apples to apples" or "apples to oranges." One of the most frequently misused or misunderstood areas of nomenclature is that pertaining to low level neurologic patients following brain injury. Terminology such as coma, vegetative state, persistent vegetative state, akinetic mutism, and locked-in syndrome are frequently used interchangeably when, in fact, the neurobehavioral status is variable in each of these diagnoses, which affects the care as well as prognosis.[2] Another area of frequent miscommunication deals with the term "head injury" as opposed to "brain injury." Although cranial trauma (head injury), can be seen concomitantly with TBI, often the two diagnoses are not synonymous. Many of the sequelae that result from presumptive TBI may be due to other diagnostic entities such as cranial trauma, cranial adnexal trauma, or cervical injury secondary to hyperextension-hyperflexion injury ("whiplash"). Labeling an individual as having a head injury when he actually has a traumatic brain injury not only exacerbates patient denial regarding the diagnosis, but is pathophysiologically and diagnostically inaccurate.[3]

Another area of nomenclature misuse is description of muscle tone. Many times clinicians incorrectly interchange the terms "spasticity" and "hypertonicity." Spasticity is an increase in tonic stretch reflexes that are velocity dependent and associated with increased tendon reflexes; this is one component of upper motor neuron syndrome. Hypertonicity, on the other hand, describes an increase in tone of a muscle during movement when the subject attempts to relax.[4] Both of these terms should be differentiated from rigidity, which is not associated with hyperreflexia, has no clonus or clasp-knife response, and is not velocity dependent.

Clinicians should also accurately define brain injury etiology; specifically, open or penetrating TBI should be differentiated from closed or nonpenetrating TBI. Unlike civilian populations, military personnel may incur much higher percentages of penetrating injuries. Most occur as a result of missile wounds such as bullets or assaults with blunt objects. An open injury always occurs when the dura, a membrane immediately covering the brain,

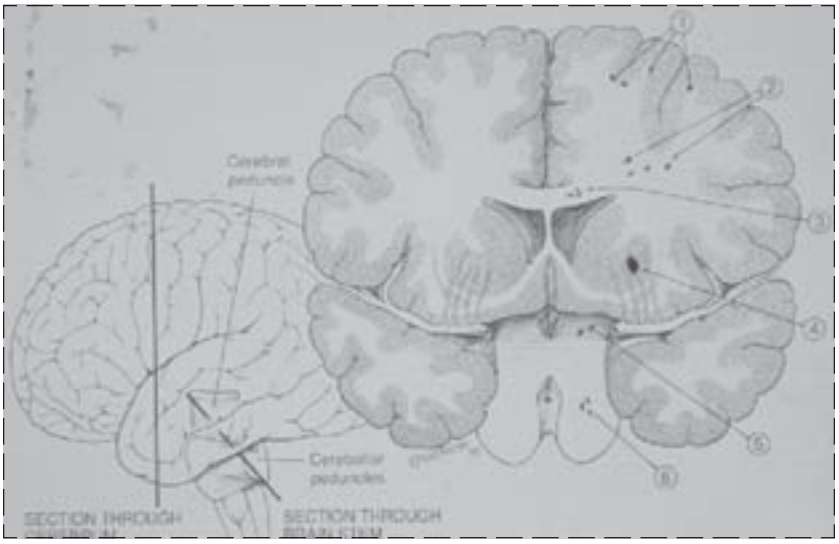

Fig. 6-1. Diagrammatic representation of neuropathologic changes associated with diffuse axonal shear injury relative to areas of higher predilection for this type of insult. (1) axonal shear at gray/white matter junction; (2) axonal shear injury in corona radiata; (3) axonal shear injury in corpus callosum; (4) axonal shear injury of lenticul-striate vessels leading to basal ganglia infarction/hemorrhage; (5) axonal shear injury of cerebral peduncles; (6) axonal shear injury of cerebellar peduncles. Reprinted with permission. Copyright Robert Shepherd. Shepherd Visual Communications: Richmond, VA;1993.

is penetrated. Nonpenetrating (closed) TBI more commonly results from acceleration-deceleration type injuries and falls. Confusion continues regarding descriptive terms that delineate the neuropathology of TBI. Often, punctate hemorrhages are documented as focal contusions even though the two are not similar in appearance or pathoanatomical origin. Classically, punctate hemorrhages are a result of diffuse axonal shear injury, which may occur, to varying degrees, in all brain injuries and affects not only axonal integrity, but also neurovascular structures (Figure 6-1). Contusions, on the other hand, occur as the result of direct impact to the involved area, resulting in either bland (nonhemorrhagic) or hemorrhagic lesions (Figure 6-2). Diffuse axonal injury and focal cortical contusion, comprise the "primary" etiologies of brain injury. Proper use of such neuroanatomic and neuropathologic nomenclature has significant implications for diagnosis, treatment, and prognosis.[5,6]

Although the above examples comprise only a fraction of the commonly misunderstood or misused nomenclature, they represent a global clinical issue that must be addressed through appropriate education and professional demands for nomenclature consistency.

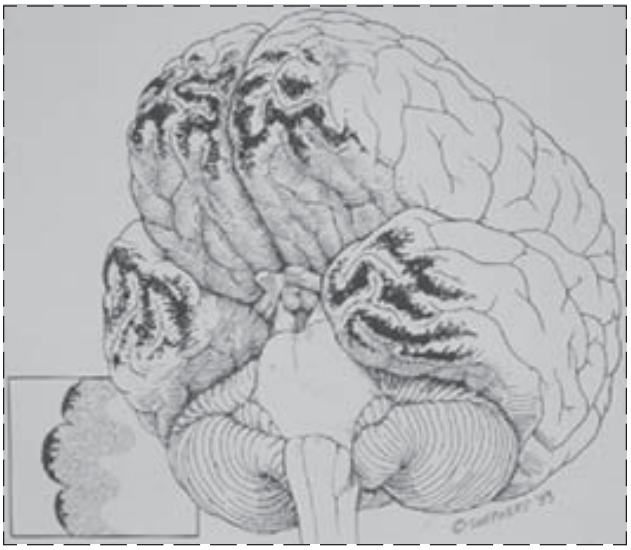

Fig. 6-2. Diagrammatic representation of neuropathologic changes and areas of predilection for focal cortical contusion involving the medial frontal lobes and anterolateral temporal lobes. Reprinted with permission. Copyright Robert Shepherd. Shepherd Visual Communications: Richmond, VA;1993.

EPIDEMIOLOGY

Discussion of TBI epidemiology, either civilian or military, is often complicated by the lack of consistency regarding (*a*) how brain injury is defined (nonpentrating vs penetrating brain injury), (*b*) the differences in screening methodologies for TBI (TBI plus multitrauma vs TBI alone), and (*c*) the differences in populations studied (inclusion or exclusion of subpopulations, such as patients with a prior history of substance abuse or TBI, and so forth). It might seem surprising that during the last decade, more deaths have resulted from nonwartime TBI than all the U.S. wars fought since the founding of the Republic. In the civilian population, accidental death is the fourth most frequent cause of death in

the United States, exceeded only by coronary artery disease, cancer, and cerebral vascular disease. The primary cause of death to both sexes ages 15 to 24 years is accidental injury.[7] Conservative civilian statistics suggest that more than two million individuals per year incur traumatic brain injury and approximately one quarter of these require hospitalization.[8] Most deaths associated with TBI occur at the time of injury or within the first several hours thereafter. Of those individuals who survive, 50,000 to 70,000 will be left with permanent neurologic impairments that will prevent their return to a preinjury lifestyle.

In civilian populations, motor vehicle accidents account for about 50% of all TBIs. Falls are the second most common cause of injury at approximately a 20% incidence; violence, including gunshot wounds and assaults, account for 12%; followed by recreational injuries at about 10%.[9] In civilian populations, research demonstrates that males typically are injured two to three times more frequently than females. Males between the ages of 15 to 24 years have the highest rate of injury. The frequency distribution of TBI relative to age is trimodal, with the young adult group being of highest frequency. The two other peaks occur during infancy and in the geriatric population.

Some studies[10] have attempted to examine racial correlates of TBI in civilian populations and have found that suburban whites seem to have about a 50% lower incidence of TBI than suburban and inner city blacks. Most TBI in inner city blacks apparently correlates with assault, whereas the single most common cause in both suburban blacks and whites is automobile accidents.[10] Unfortunately, child abuse contributes to over 50% of infant TBI. Falls account for most brain injuries in the geriatric population.

Military studies examining incidence and type of TBI include the extensive data collected and published by the Vietnam Head Injury Study group. Interested readers can find a discussion of this detailed follow-up study at the end of this chapter, in a section entitled The Vietnam Head Injury Study: Overview of Results to Date. The data suggest that penetrating TBI accounts for approximately 50% of all military-related injuries.[11] Penetrating injuries, high velocity bullets, and fragments from explosive devices seem to predominate as the mechanisms of injury.[12] During nonwartime, U.S. Army epidemiological studies have shown a fairly equal incidence of concussion across sexes; although, in general, males predominated with regard to intracranial injury and TBI.[13] McCarroll and Gunderson[14] published data from a 5-year survey that indicated that skull fractures and intracranial diagnoses were found almost exclusively in males; whites had a higher incidence of injury than blacks, and only 10% of the injuries were alcohol related (97% of which occurred in males).

PROGNOSTICATION ISSUES

Rehabilitation physicians are often asked to make prognostic statements about patients who have suffered TBI. The main factors that can aid the postinjury prognostication of this patient population include preinjury, injury, and postinjury parameters. A significant amount of literature has been published dealing with acute injury and the prognostic implications of clinical and laboratory parameters.[15] Few sound data are available that do justice to preinjury and postinjury factors relative to their role in prognosis of either neurologic or functional outcome.

Preinjury Parameters

The preinjury parameters that must be considered when analyzing a patient's prognosis include (*a*) psychosocial background; (*b*) history of learning disability; (*c*) prior psychological and psychiatric problems, or both; (*d*) prior history of substance abuse; (*e*) prior brain injury (regardless of etiology); and (*f*) developmental history.[16,17] Available data support the conclusion that people with higher preinjury levels of function have a better functional outcome after TBI than individuals with suboptimal preinjury psychosocial or intellectual status, or both. Although it is poorly quantified, factors such as the amount of neural reserve, play at least some part in determining how well an individual will recover from TBI. Individuals with any significant prior injury may find an otherwise trivial neurologic insult much more devastating than if they not had such a history. Research literature also supports the general contention that younger patients typically have a better outcome than older patients, from both neurological and functional standpoints regardless of injury severity, although this relationship of age does not appear to be linear.[18]

Injury Parameters

In the acute care setting, a significant number of clinical parameters can be analyzed separately or

together to assess both neurologic and functional outcome prognosis. Probably the best known of these is the Glasgow Coma Scale (GCS)[19] score. The scale provides clinicians with a brief, standardized neurologic assessment tool for use in the acute care setting. The GCS score has been shown to be highly correlated with acute morbidity and mortality, as well as, although not as strongly as, long term functional outcome. The three clinical parameters that make up the total GCS score (which ranges from 3 to 15) are (1) best motor response, (2)verbal response, and (3) visual response. The scores are differentially weighted with eye opening ranges from 1 to 4, best verbal response from 1 to 5, and best motor response from 1 to 6. GCS scores between 3 and 8 define severe neurologic insult; scores between 9 and 12, moderate; and scores between 13 and 15, mild. Although each of the three variables on the GCS holds prognostic utility in and of itself, the motor score has proved to be the most sensitive relative to long-term outcome. The predictive validity, specificity, and sensitivity increase significantly when all three parameters are conjointly assessed.

Variations of the GCS scores such as the Glasgow-Liege Scale allow for more sensitive prediction of outcome by taking into consideration clinical evidence of brain stem dysfunction.[20] Clinical findings that are useful in early prognostication include oculocephalic and oculovestibular reflex abnormalities, which may indicate significant brain stem damage. Longer durations of coma and posttraumatic amnesia have also been associated with poorer neurologic and functional outcomes. Advances in neuroimaging have enabled correlations to be made between early static imaging, as well as functional imaging and outcome. Electrophysiologic assessment by means of multimodal evoked potentials (including visual evoked responses, auditory brain stem responses, and somatosensory evoked responses) and a variety of electroencephalographic (EEG) modalities, such as compressed spectral analysis and quantitative EEG, have also been correlated either singularly or in multifactorial analysis of outcome prognosis. A variety of cerebrospinal fluid markers, including creatine kinase and lactate dehydrogenase, have also been studied relative to their correlation with the severity of central neurologic damage. Cerebrospinal fluid neurotransmitter and neurotransmitter metabolite levels have also been studied relative to their relation with acute neurologic morbidity and mortality; levels tend to have a direct correlation with a higher degree of neurologic morbidity. Acute medical variables that have been associated with a worse outcome include mass lesions on imaging, protracted elevated intracranial pressure beyond 40 mm Hg, and cardiopulmonary complications.[21]

Concurrent hypoxic ischemic injury, whether internal or external, must also be considered as a comorbidity that is associated with poorer short- and long-term neurological and functional prognosis. Hypoxic ischemic insult may be focal or diffuse. The focal variety is generally the result of infarction of a vascular territory quite commonly in the distribution of the posterior cerebral artery as a result of transtentorial temporal lobe herniation. Diffuse hypoxic insult, similar to diffuse axonal injury as a result of trauma, has a predilection for damaging certain areas more than others, including the medial temporal lobe structures (hippocampi), basal ganglia, and purkinje fibers of the cerebellum. When severe, hypoxic insult may result in diffuse cortical neuronal loss. Ischemic insult may also result in so-called "watershed" infarctions in parenchymal areas between major arterial vascular territories.[22]

Postinjury Parameters

Numerous studies have been conducted in an attempt to provide clinicians with information to aid them in prognosticating outcome and morbidity in the postinjury phase. Typically, once a patient has reached rehabilitation, many clinicians are not as attuned as they should be to the prognosticatory significance of specific clinical findings or functional impediments, or both. The longer the duration of the vegetative state, particularly when over 3 months, the greater the likelihood that the individual will remain vegetative, all other things being equal. In minimally responsive patients, some researchers have correlated the presence of communicating hydrocephalus and central dysautonomia with poorer neurologic and functional outcomes.[23] Agitation during the recovery phase has been theorized to correlate with better than worse functional outcomes in comparison to withdrawn, psychomotorically retarded behavior. Anosognosia, or denial of deficit, can be a troublesome neurobehavioral sequela of TBI due to safety implications and the inability to appreciate the need for further rehabilitative interventions. Lower extremity flexion synergy patterns are typically considered to be a poor prognostic sign for functional ambulation. Significant behavioral problems tend to indicate a poorer prognosis for successful independent community reentry. Further research is obviously necessary to clarify which specific impairments are poor prognostic indicators for specific functional goals and abilities.

MECHANISMS OF NEURAL RECOVERY

Multiple mechanisms have been proposed in an attempt to explain recovery of function following central nervous system (CNS) insult after TBI. However, there is little empirical support for a causal link between the theorized phenomena and the resultant functional recovery. Resolution of transient neurophysiologic phenomena, including elevated intracranial pressure, edema, and hypoxia, may be responsible for some of the reversible impairments noted in the earlier stages postinjury. Modification of synaptic function has been suggested as a possible explanation for the phenomenon of diaschisis (reversible depression of parenchymal function associated with focal insult to adjacent areas of brain tissue). Alterations in neural connections through axonal regeneration (not necessarily functional) and collateral sprouting have also been suggested as neural mechanisms mediating recovery of function.

Other theories posited include functional substitution, vicarious functioning, and redundancy. Functional substitution entails the overt or covert use of alternative strategies to achieve the desired functional outcome. In other words, following CNS insult, the organism learns compensatory strategies to cope with its functional disabilities. Vicarious functioning implies that neural structures alter their function in some indeterminate manner to allow for subserving the direction of new functional tasks. Redundancy, on the other hand, implies that following neural insult, there are "dormant" neural circuits that have the capability of directing particular functions, but only do so "when called upon."[24]

The concepts of neuronal sparing and neuronal reorganization broadly define the two major putative neural mechanisms involved in enhancing the potential for functional reorganization and recovery of function following brain injury. Multiple experimental treatment regimens for inhibiting or blocking the "neurotoxic cascade" following TBI are presently being researched and are expected to have clinical applicability in the near future. Research utilizing agents that interfere with cholinergic and glutaminergic tertiary nerve cell death, and that impede calcium induced cellular damage and vasoconstriction, remains furtive. Free radical research continues at a very active pace. Experimental studies[25] have demonstrated that oxygen free radicals may be important mediators of brain injury and brain edema. Researchers have found a multitude of sources for oxygen radicals after neuronal injury, including xanthine oxidase, peroxidases, catecholamines, and amine oxidases. Agents being examined to impede oxygen-free-radical damage include superoxide dismutase, catalase, vitamin E, and dimethyl sulfoxide on lipid peroxidation inhibitors such as the 21-aminosteroids ("Lazaroids" being one example of this class of drugs).[25]

The release of excessive excitatory amino acids, including glutamate and aspartate, were initially hypothesized by Olney[26] in 1969 to be associated with neuronal death due either to acute osmotic lysis or delayed excess intracellular free calcium. Current thought among bench neural science researchers is that excitotoxic phenomena may render neurons dysfunctional without necessarily killing them. It is well known that TBI results in the widespread depolarization and nonspecific release of a multitude of neurotransmitters and neuromodulators, both excitatory (glutatmate, aspartate, and acetylcholine) and inhibitory (γ-amino butyric acid [GABA] and opioids). The resultant "sublethal" toxicity is theorized to be mediated by elevations in intracellular calcium levels.[25] The glutatmate receptor has three subtypes, named according to their selective agonists: (1) *N*-methyl D-aspartate (NMDA),(2) quisqualate, and (3) kainate. Laboratory studies[27] have demonstrated that NMDA receptor antagonists may protect against brain injury secondary to cerebral ischemia and trauma. Issues of dosing and toxicity need further investigation prior to undertaking human trials. Recent work[28] suggests a role for anticholinergic and GABAergic agents in suppressing some of the adverse side effects from this class of presumptive neuroprotective agents.

A variety of other neurotransmitter systems are presently being studied relative to their contribution to acute brain injury. Cholinergic systems seem to play variable roles in mediation of brain injury and neural recovery relative to the time postinjury. Evidence[29] suggests that acute anticholinergic drug administration after TBI tends to decrease the period of unconsciousness either through decreasing the extent of active inhibition of systems responsible for regulation of consciousness, or lessening the extent of neural injury. Researchers[25] have hypothesized that early anticholinergic therapy benefits on long term motor deficits may be more related to its effect at blocking release of excitotoxins.

Catecholamines, particularly norepinephrines, are actively being investigated relative to their role in recovery from TBI related behavioral deficits.

Early work by Hovda and Feeney[30] demonstrated that α-noradrenergic agonists and perhaps dopaminergic agents actually accelerated motor recovery following sensorimotor cortex injury; whereas their antagonists retarded recovery. The exact location at which noradrenergic fibers emanating from the locus ceruleus need to be "stimulated" to mediate accelerated motor recovery is a much debated area of current research. Boyeson[25] believes that the critical area is not related to diaschisis-like effects in the sensorimotor cortex itself, but rather to alterations in noradrenergic function in the cerebellum contralateral to the site of sensorimotor cortex injury. The present evidence[31] is strong for an acute role for noradrenergic treatment if given at appropriate times and under specific conditions. Conversely, in certain circumstances, noradrenergic antagonists may actually be detrimental relative to their potential to reinstate motor behavior deficits following acute brain injury.

Stimulation of GABAergic systems have been associated with ischemic events following both experimental and clinical brain injury. Studies have demonstrated that early administration of GABAergic agents (such as diazepam) may be deleterious relative to slowing of neural recovery and potential reinstitution of neural deficits. Evidence also suggests that increased central GABA levels may enhance glutamate neurotoxicity by mechanisms not yet clearly defined. Such evidence would suggest a more conservative approach to the use of GABAergic agonists, including benzodiazepines and valproic acid, in the very acute setting after TBI.[25]

One area of active clinical research that may turn out to be the "magic bullet" is that of hypothermia. Researchers are presently investigating the role of acute hypothermia in animal and human subjects to determine the effects on a variety of neurochemical cascades, as well as the morbidity and mortality associated with acute brain injury. Clifton and associates[32] have evaluated the effects of moderate hypothermia (30°C to 36°C) on mortality after experimental fluid percussion TBI in a rat model and found significant protection from hypothermia at the lower range of this temperature scale. Theoretically, hypothermia, among other explanatory mechanisms, may actually slow down the neurotoxicity associated with the multitude of events occurring immediately following TBI.

No neurotransmitter system acts in a totally isolated manner. Therefore, treatment with a specific neurotransmitter agonist or antagonist may directly or indirectly modify functioning in other neurotransmitter systems within the CNS. Clinically, "therapeutic cocktails" may be necessary, either with or without hypothermia treatment. The focus of these polypharmaceutical "potions" will become better as our understanding of neurotransmitter system interrelationships improves. Once the science of acute neurochemical alterations following TBI is better understood, the potential exists for acute treatment in the field by emergency medical technician personnel or in hospital emergency rooms with specific drug or hypothermia protocols, or both. As we "go where no man has gone before," the hope for more successful treatments for acute brain injury grows brighter by the day.[25,27,32]

Denervation supersensitivity and reactive synaptogenesis (also called axon collateral sprouting) have also been theorized[33] to play a potential role in neural reorganization; however, it remains unclear whether such reorganization is always adaptive as opposed to maladaptive.

Some clinicians[24] believe that functional recovery is a consequence of an inherent, albeit poorly defined and understood, ability on the part of the CNS to adapt to injury. It is critical to understand the realities of age-related, genetically driven, central processes that drive functional recovery. Specifically, neuronal sparing mechanisms in early development are distinct in comparison with those processes occurring in more mature organisms. In the real world, most of the evolutionary consequences of brain injury for nonhumans are functionally and physiologically maladaptive because they result in the organism being more prone to predation in the wild. Parallels can be drawn to what happens to humans after severe brain injury. In the best of all worlds, based on sound scientific rationale, rehabilitationists can intervene in the recovery process to beneficially (or even negatively) affect rehabilitation outcome.

Research[34] suggests that the rate of neurologic recovery is more amenable to interventional manipulation than the ultimate level of neurologic recovery. It should also be realized that faster is not necessarily better. There are inherent risks associated with rushing recovery. Specifically, maladaptive pathoanatomic mechanisms and functional behaviors may be triggered or reinforced, or both. The development of a more comprehensive understanding at a basic-science level of what really happens as patients recover following brain injury is critical if rehabilitationists are to intercede optimally into this complex process and maximize neurologic and functional recovery.[33]

ADVANCES IN CLINICAL TREATMENT: POSTACUTE PHARMACOTHERAPY

The basic tenant of positively affecting neurological outcome and functional status after brain injury through the use of pharmacologic agents is by no means new.[35,36] Nonetheless, most rehabilitation professionals have relied almost exclusively on nonpharmacologic modalities to address sequelae following traumatic and nontraumatic brain injury. Physiatrists, as of late, have become more comfortable at managing both the pharmacologic and the more traditional nonpharmacologic rehabilitative aspects of care of individuals following brain injury.

Until recently, there was little if any evidence that medications could make a difference in either the rate or plateau of neurologic and functional recovery following brain injury. Now, good evidence indicates that many acute, subacute, and chronic neurologic and functional sequelae resulting from brain injury can be lessened and potentially abated through the thoughtful and appropriate use of pharmacologic agents.[37] Many pharmacologic agents may have potential utility in altering function following brain injury. Much of what is known about pharmacologic TBI rehabilitation is based on theories derived from work done at the basic-science level with animal models, or from individual clinical experience. The peer reviewed scientific literature, as it presently stands, does not provide much well controlled, methodologically sound, prospective research data regarding this topic. Nonetheless, clinicians should be aware of the major pharmocologic agents in each neurotransmitter class in order to better grasp how they *may* have an affect, positive or otherwise, on neurological recovery and functional capabilities following brain injury. In addition, rehabilitation professionals should be familiar with the major side effects of these drugs. Although drug interactions, precautions, and contraindications must also be considered, these topics are beyond the scope of this chapter. The reader is referred to other sources for this information, but should remember that these reference texts provide guidelines and information only on Food and Drug Administration (FDA) approved drug uses and dosage ranges. It should be noted that many of the medications prescribed for post-TBI sequelae are not FDA approved for the particular application in which they are being utilized. Physicians should be aware of potential medicolegal issues inherent in utilizing non-FDA approved medications, medications in applications that are not FDA approved, or medications in higher doses than FDA approved. The following paragraphs will outline the major drug groups presently in use by physiatrists versed at neuropsychopharmacology and TBI rehabilitation.

The major catecholaminergic drugs are levodopa (L-dopa), amantadine, bromocriptine, pergolide, lisuride, and some of the more classic stimulant drugs, such as dextroamphetamine, methylphenidate, and pemoline. The classic dopamine agonist has historically been L-dopa. A combination formulation of L-dopa and Carbidopa is also available. The use of the combination drug minimizes peripheral (non-CNS) side effects and increases the amount available for CNS incorporation. L-dopa has its action presynaptically and is agonistic at both the D1 and D2 receptor sites.[38] Side effects are numerous, but the more frequent ones include dyskinesias, various bradykinetic episodes (ie, "on–off" phenomena), psychiatric disturbances, gastrointestinal disturbances (nausea, vomiting, anorexia, and slowing of gastric motility), as well as orthostatic hypotension. Carbidopa-L-dopa is available in ratios of 1:10 (100 mg L-dopa to 10 mg carbidopa) and 1:4 (100 mg L-dopa to 25 mg carbidopa). Most patients with clear clinical evidence of dopaminergic deficiency will respond to a 1:10 ratio provided the daily dosage of carbidopa is 70 mg or more. When the 1:4 ratio is used, the usual starting dose is 1 tablet three times a day, increasing by one tablet every 2 days up to a maximum dosage of 6 tablets daily. If the 1:10 ratio is used, the usual starting dose is 1 tablet three to four times a day, increasing by one tablet every 2 days, to a maximum of 8 tablets daily.[39] In addition to carbidopa, the enzyme inhibitors benserazide and L-deprenyl (a monoamine oxidase Type B inhibitor) have been used in conjunction with L-dopa in an attempt to increase therapeutic efficacy.

Amantadine hydrochloride has been utilized clinically as an antiviral agent, as well as an anti-Parkinsonian agent. Its exact mechanism of action is still not fully understood; however, it has been theorized to have a presynaptic action, as well as a possible postsynaptic action.[40] Some researchers have speculated that amantadine may also increase central cholinergic and GABAergic activity.[41] Therapy can be initiated at between 50 to 100 mg/d and increased to a maximum of 400 mg daily. Since the drug is not metabolized and is excreted unchanged in the urine, dosage adjustments must be made when there is concurrent decreased renal func-

tion, such as in the elderly or in patients with renal disease. Peripheral side effects include, but are not limited to, peripheral edema, lightheadedness, orthostatic hypotension, hot and dry skin, rash, and livedo reticularis. Livedo reticularis is a discoloration of the skin that occurs in a reddish-blue to purple blotchy pattern. The reaction tends to occur after at least 1 month of treatment and it may occur more commonly at higher doses. Livedo reticularis is totally benign and the medication does not need to be discontinued unless the cosmetic aspects outweigh the therapeutic benefits.[39] Central side effects, which are more commonly seen in the geriatric population, include confusion and hallucinations.

Due to the L-dopa "indirect" mechanism of action, researchers have pursued and developed several direct dopamine-receptor stimulating agents, all of which fall in the ergotalkaloid class. These direct agents include bromocriptine, lisuride, and pergo-lide. Both bromocriptine and lisuride are antagonistic at the D1 receptor and agonistic at the D2 receptor. Pergolide, on the other hand, is agonistic at both the D1 and D2 receptor sites. Bromocriptine mesylate tends to produce fewer problems with dyskinesias, but more problems with mental side effects, orthostasis, and nausea than L-dopa.[38] Clinical results have demonstrated a triphasic response to bromocriptine with dopamine agonism, occurring only in the midrange doses. Dosing should start with a test dose of 1.25 mg and, if tolerated, the patient can then receive 2.5 mg daily, increasing fairly quickly to a three to four times a day dose. Once at 10 mg/d, the dose can be increased every 4 days by 2.5 mg. Typically, clinical experience has dictated that doses higher than 60 mg/d are unnecessary in patients with acquired brain injury. The manufacturer has not established safety limits for dosages greater than 100 mg daily.

Pergolide and lisuride are relatively new agents in this country and there is little if any literature on their utility in the pharmacologic rehabilitation of individuals with brain injury. It should be noted that pergolide is an extremely potent dopamine agonist and only very small doses are required. In this author's limited experience with pergolide, most patients with brain injury are unable to tolerate the drug secondary to sedation. Lisuride is also extremely potent and therapeutic effects are typically seen with daily doses ranging from 4 to 10 mg daily.[38] Most of the ergot alkaloids also have concomitant central serotonergic receptor agonism, which might explain the high incidence of changes in mental status with this class of dopamine agonists.

The classic "psychostimulant" drugs include dextroamphetamine, methylphenidate, pemoline, and to a lesser extent, activating tricyclic antidepressants. These agents have typically been theorized to have mixed dopaminergic and noradrenergic agonist activity. Dextroamphetamine has been theorized to produce noradrenergic agonism by blocking the reuptake mechanism for norepinephrine. In higher doses, it is also dopaminergic by a similar mechanism of dopamine reuptake blockade.[42] Dosing of dextroamphetamine should be initiated at 5 mg once to twice daily. The maximum recommended dose of dextroamphetamine is 60 mg/d; however, there are little if any data addressing dosing limits in individuals following brain injury. To avoid problems with insomnia, the last dose of medication should be given at least 6 hours before the patient's bedtime. There is evidence that "pulsed" dosing of noradrenergic agonists by standard formulations rather than extended release dosing may be preferential with regard to the resultant psychostimulant effects. Generally, adults are fairly sensitive to psychostimulant therapy, particularly after brain injury. Relative or absolute "toxicity" may be manifested by anxiety; dysphoria; increased irritability; cardiovascular symptoms; headache; palilalia (pathological use of words and phrases); stereotypical thoughts; cognitive impairment; hallucinations; insomnia; and motor disorders including dyskinesias, tics, and worsening of spasticity.[43]

Methylphenidate hydrochloride is a mixed dopaminergic, noradrenergic agonist whose pharmacologic action is similar to amphetamines. The main sites of action appear to be the cerebral cortex and subcortical structures such as the thalamus. Dosing typically should be initiated at 5 mg twice a day and titrated up to a maximum dose of 60 mg daily. An extended release formulation is also available. The adverse effects of this drug are analogous to those of dextroamphetamine.

Pemoline is an oxazolidinone derivative stimulant with pharmacologic actions qualitatively similar to dextroamphetamine and methylphenidate. Evidence suggests that pemoline may have its stimulatory effect through dopaminergic mechanisms. The drug is typically dosed initially at 37.5 mg daily as a morning dose with increases of 18.75 mg made weekly, as appropriate. The effective dose typically ranges from 56.25 to 75 mg daily. The most frequently encountered adverse effects include insomnia and anorexia, both being dose related.

The major serotonergic drugs are trazodone hydrochloride, fluoxetine, buspirone, sertraline, par-

oxetine, and L-tryptophan. Trazodone hydrochloride is a triazolopyridine derivative that selectively inhibits serotonin uptake. Initial dosing should begin at low doses (50–150 mg), typically at bedtime with food. The dose should be on the lower end of the dosing range in geriatric patients secondary to more common side effects such as sedation and orthostatic hypotension. The dose may be increased by 50 mg/d every 3 to 4 days to a maximum of 400 mg daily. If closely monitored, as in an inpatient setting, the maximum dose may be as high as 5 mg/kg daily. Fluoxetine is also a serotonin reuptake inhibitor, but it tends to be more activating than other serotonergic drugs like trazodone. Initial dosing should be 20 mg/d as a morning dose. Doses above 20 mg/d should be given on a twice a day schedule with a maximum daily dose of no more than 80 mg. The major reported side effects include headache, nausea, nervousness, and insomnia.[44] Buspirone is a novel benzodiazepine anxiolytic which is theorized to work through its serotonergic agonist activity at the 5-HT1 receptor. It should also be noted that this medication is presynaptically antagonistic at the D2 dopaminergic receptor.[45] The medication should be initiated at a dose of 10 to 15 mg twice a day and increased over 4 to 6 weeks to a maximum of 60 mg/d, based on patient response and tolerance.[46] The main side effects with buspirone are dizziness, headache, nervousness, and lightheadedness.

Newer selective serotonin reuptake inhibitors (SSRIs), such as paroxetine and sertraline, also show promise in the treatment of affective disorders. Generally, paroxetine is dosed from 10 mg to 50 mg/d and may be taken as a single dose. The most common side effects are nausea, headache, dry mouth, and an altered sleep–wake cycle, as well as—albeit less commonly—ejaculatory dysfunction.[47] Sertraline, also an SSRI, is dosed from 50 mg to 200 mg daily. Commonly reported side effects are gastrointestinal (nausea and diarrhea), headache, tremor, dizziness, dry mouth, altered sleep–wake cycle, and male sexual dysfunction.[48] L-tryptophan is a serotonergic precursor that has recently received quite a bit of attention secondary to the incidence of eosinophilia-myalgia syndrome. This syndrome has been purportedly traced to a bad batch of this pharmacologic agent produced in Japan.[49] For now, its use remains barred by the FDA. Newer serotonergic uptake inhibiting drugs that are still under clinical investigation and may prove useful from a neuropharmacologic rehabilitative standpoint include citalopram and fluvoxamine.

The two most commonly used opioid antagonists are naloxone and naltrexone, the latter being preferred secondary to its oral route of administration and prolonged mode of action. Dosing typically starts low, with 12.5 mg to 25 mg daily with titration up to 150 mg/d with an average daily dose of 50 mg. Exact dosing schedules and upper limits for TBI have not been established. The major side effects relate to gastrointestinal complaints and hepatocellular injury.

Agents with GABAergic activity are commonly used in the general rehabilitation setting. It should be noted, however, that only a few of these can be recommended for use in a patient with concomitant brain injury, particularly in the early phase of neural recovery. Classic antispasticity agents such as valium and baclofen are GABAergic agents, GABA A and GABA B, respectively. Many of the presently available anticonvulsant agents are also GABAergic, specifically, valproate, barbiturates, and benzodiazepines. Other commonly utilized anti-convulsants, such as phenytoin and carbamazepine, are felt to mediate anticonvulsant effect through other neurochemical systems.[50] From a clinical standpoint, many GABAergic agents tend to be overly sedative with concomitant suppression of cognitive processes. The use of these agents in the subacute and chronic phases following brain injury should be examined carefully given their potential side effects.[37]

Valproic acid is typically dosed at 15 mg/kg/d. Dosages may be increased by 5 mg/kg/d to 10 mg/kg/d at weekly intervals until clinical efficacy is achieved or adverse side effects prevent further increases. Due to potential adverse gastrointestinal side effects, it is recommended that the drug be administered in two or more divided dosages. The maximum daily recommended dose is 60 mg/kg. Side effects are generally dose dependent.

Although various agents fall under the category of cholinergic substances, most of them have fairly limited utility secondary to their lack of CNS specificity, poor ability to penetrate the CNS, short half-life, and side-effect profile. Various drugs, including direct agonists, acetylcholine precursors, and acetylcholinesterase inhibitors have been utilized in an attempt to provide "cholinergic stimulation" following brain injury. Newer drugs such as tetrahydro-9-aminoacridine (THA), also commercially known as Tacrine, may hold better promise than more standard drugs, such as physostigmine.

The following discussion reviews some of the potential pharmacologic approaches to dealing with

posttraumatic impairments. The specific residua have been listed alphabetically.

Appetite Dysregulation

Alterations in appetite are common in patients with brain injury. The hyperphagic patient, or "bulimic type," must be contrasted with the hypophagic, or "anorectic type." Presumptive central neurochemical and neurophysiologic mechanisms responsible for alterations in appetite regulation form the basis of drug treatment for these functional sequelae.[51] The present consensus, based on animal as well as human studies,[52,53] suggests that serotonergic agonists (sertraline, fluoxetine, and fenfluramine), opioid antagonists (naltrexone), and possibly corticotropin releasing hormone may all inhibit feeding behavior. Interestingly, there is recent evidence that questions whether the anorexigenic effects of sertraline and fluoxetine are mediated by 5-hydroxytrytophan.[54] Central serotonergic antagonists such as cyproheptadine can be utilized when there are problems with anorexia or hypophagia.[52]

Ataxia

Various forms of brain injury can result in cerebellar ataxia, including trauma, stroke, tumor, degenerative conditions, and inherited ataxias such as Friedreich's ataxia. Several authorities have reported that the serotonergic precursor L-tryptophan can significantly improve cerebellar ataxia due to a variety of primary etiologies.[55–57] Oral thyrotropin-releasing hormone also appears to be a promising agent.[38,39] Other agents that have been utilized with some success include propranolol, gamma-vinyl GABA, acetazolamide, and phthalazinol.[57] Peterson and associates have reported good success with amantadine for Friedreich's ataxia, presumably through either a dopaminergic or more likely a GABAergic mechanism.[41]

Autonomic Dysregulation

One of the most challenging clinical conditions to treat following severe CNS injury is that of autonomic dysregulation with associated symptoms of hyperthermia,[58] diaphoresis, tachycardia, and tachypnea. Numerous neurochemical systems have been theorized to be involved with central control of temperature regulation, but relatively speaking, hypothalamic dopaminergic systems seem to play a very significant role.[59,60] Hyperpyrexia following brain injury has been successfully treated at a central level with dopaminergic agonists,[61] morphine, β-blockers, and neuroleptics. Dantrolene sodium has also been utilized to help decrease peripheral systemic effects such as rigidity commonly associated with this condition.

Cognitive Behavioral Dysfunction

Before any pharmacologic agent is administered in an attempt to improve cognitive function, one must first establish whether the individual's internal and external environments have been maximized and stabilized.

The examination of the internal environment should be composed of assessing the individual's present neuromedical condition. Neuromedical issues that could present as alterations in cognitive function include (*a*) basic metabolic aberrations; (*b*) nutritional depletion; (*c*) occult infection; (*d*) neuroendocrine dysfunction; (*e*) suboptimal cerebral blood flow or oxygenation, or both; (*f*) posttraumatic hydrocephalus; (*g*) late extra-axial collections; and (*h*) unrecognized posttraumatic seizure disorders. Other critical internal factors include the neuronatomic correlates of injury made evident by either static or dynamic imaging technologies; postinjury medical history (ie, significant hypoxic-ischemic injury, elevated intracranial pressures, etc.); and preinjury factors, such as prior brain injury, substance abuse, learning disability, or psychiatric illness.

The examination of the external environment must take into consideration the extent of stimulation and the cognitive-behavioral status of the individual at that particular time. It must be recognized that, structured or not, the extent and complexity of environmental stimulation must be gauged by the individual's cognitive-behavioral profile. A patient who is highly volatile, easily irritated, or hyperaroused will do better with less stimulation than with more. On the other hand, the individual who tends to be at the lower end of the functional scale, or who becomes confused in unfamiliar surroundings, will perform better, cognitively speaking, when provided with more structured stimulation in a familiar environment. Many times, individuals with brain injuries who have severe physical disabilities are assumed to be also disabled from a cognitive standpoint; this is not always the case. In these individuals, cognitive performance may actually suffer secondary to inadequate environmental stimulation, sometimes nearing the point of environmental sensory deprivation.

It is also critical to consider issues of aging (including the inherent decline in learning ability and retention of new information that is known to occur with aging) on the potential response of an individual to medication following TBI.[62] Additionally, certain situational variables appear to influence the performance of geriatric subjects; specifically, older individuals perform more poorly when task difficulty is high, or when complex encoding strategies or mnemonics are required.

A variety of neurochemicals have been hypothesized as being involved with the mediation of cognitive processes. It is rather a limited viewpoint that attempts to explain cognitive function by Occam's razor, that is, hypothesizing the simplest explanation possible to rationalize a particular process. Cognition is most likely a set of processes mediated through the interaction of a variety of neurochemical systems. Some of the neurochemical substrates that have been proposed to be involved in mediation of cognitive processes (both facilitory and inhibitory) include cholinergic, catecholaminergic, neuropeptidergic (vasopressin, thyrotropin releasing hormone [TRH], endogenous opioids, neuropeptide γ, and adrenocorticotropic hormones [ACTH]), GABAergic, and hormonal systems. Other substances (vitamin cofactors and trace metals) have also been theorized to play important roles in allowing normal neurophysiologic reactions to proceed unabated.

Many drugs have been advocated to improve memory, learning, and general cognitive function. Disappointingly, there is as yet no magic bullet, possibly as a result of the reflection of the nature of the basic neurophysiological and neurochemical processes in question rather than a lack of adequate understanding on the part of researchers regarding cognitive processes. Of the agents that have been studied, the response rates have been quite variable, or the sample populations or experimental methodologies (or both) have been suboptimal. Ultimately, research may find a "cognitive enhancement cocktail" that combines various agents in an attempt to normalize and even maximize the neurochemical environment deemed to be most conducive to enhancement of cognitive processes. At this time, there does not appear to be one single cognitive enhancing drug (CED) that works *all* the time for *every* individual who suffers from posttraumatic cognitive deficits.

In recent years, there has been a fairly extensive body of literature examining the potential influence of hypothalamic and pituitary neuropeptides on learning and memory.[63] Vasopressin analogues and ACTH have been reported to improve memory and learning in numerous test situations in humans and several species of animals. One hypothesis is that ACTH/MSH (melanocyte stimulating hormone) affects attentional and motivational processes, whereas vasopressin is more directly involved in memory processes. On the other hand, opioids specifically, beta-endorphin and met-enkephalin) seem to have amnestic qualities that can be reversed through administration of opioid antagonists, such as naloxone or naltrexone.[64] The only published double-blinded, placebo controlled studies that specifically examined the utility of vasopressin in persons with TBI found no clinically significant benefit.[65,66] Regardless of ultimate efficacy, the electrolytic effects of vasopressin on sodium homeostasis may be the limiting factor in clinical application of this CED. More recent research on the utility of TRH as well as vasopressin, has been conducted at the University of Washington in the Department of Rehabilitation Medicine as part of their Rehabilitation Research and Training Center in Traumatic Brain Injury.[67] Preliminary data seem encouraging regarding a potential role of these agents in memory enhancement mediated by cholinergic systems.

Of all neurotransmitter systems proposed to play a role in memory function, the cholinergic system has without question received the most attention. Most of the work in this area emanates from research in senile dementia, Alzheimer's type. Although the scientific literature is mixed regarding the role of cholinergic pathways in memory function, an increasing number of drug studies[67] in humans and animals suggest that pretreatment with anticholinergic drugs disrupts memory storage, whereas cholinergic agonists may actually produce dose-dependent facilitation or disruption. Some research[67] also suggests that a neurochemical dissociation of cholinergic memory systems exists, such that cholinergic neurotransmission is required for declarative, but not procedural, memory. Interestingly, there may actually be a "therapeutic window" for cholinergic agents so that beneficial effects are present only at middle range doses and are absent at low range doses; whereas high doses lead to impaired cognitive function.[68]

The approach to treatment of cognitive deficits referable to cholinergic system augmentation may take one of three main routes: (1) precursor agents such as choline or lecithin, (2) anticholinesterases such as physostigmine or THA, or (3) direct cholinergic agonists such as bethanecol or oxotremorine.[69] Only a few scattered studies, with rather mixed results, have specifically addressed the utility of

cholinergic agents in individuals with TBI.[70–72] Tetrahydro-9-aminoacridine, a potent anticholinesterase, may be a cholinergic "drug of the future" secondary to the fact that it can be administered orally, has a relatively long half-life, and has a reasonable side effect profile.[73] Most recently, it has achieved attention relative to a potential role in the treatment of cognitive deficits associated with senile dementia. The utility of this specific drug is yet to be clarified in individuals with TBI. CDP-choline (cytidene 5′-diphosphocholine), an essential precursor in the synthesis of brain glycerophospholipids, has been studied in patients with TBI to enhance neurorecovery, including cognitive performance. Results to date, including several placebo controlled or double blind studies, or both, look promising.[74,75] For now, the pharmacological side effects and suboptimal modes of administration of many of the cholinergic agents limit their clinical usefulness.

A large body of evidence indicates that catecholamines may be involved in the modulation of learning and memory. A number of drug studies have suggested that drugs that disrupt catecholamine systems disrupt memory storage, while catecholamine agonists produce dose-dependent facilitation or disruption. As an example, amphetamine has been shown to have no effect at low doses, improvement at restricted dosage ranges, and impairment at higher doses.[76] The major catecholaminergic neurotransmitters are norepinephrine and dopamine. It is possible to affect the net balance of neurotransmitter effects, as well as turnover, through the administration of agents that ultimately affect the net activity at the postsynaptic receptor site. Drugs may exert their effect by increasing release from presynaptic stores (methylphenidate), increasing production and release from the presynaptic vesicles (L-dopa/carbidopa), decreasing reuptake into presynaptic vesicles (nortriptyline, desipramine), or acting directly at the postsynaptic receptor site (bromocriptine). Some agents, such as amantadine, may effectuate their ultimate agonistic effect at both the presynaptic and postsynaptic receptors through a variety of mechanisms.[77] The use of psychostimulants, such as amphetamine or Ritalin, typically results in improved concentration and performance, a suppression of fatigue, and an elevated mood. These noradrenergic drugs may also produce the adverse side effects of anorexia, hypertension, tachycardia, and aberrant behavioral changes (ie, euphoria or dysphoria). Although multiple studies have utilized catecholaminergic agonists after brain injury, very few have specifically assessed their utility for remediation of cognitive dysfunction.[78,79] One case study[80] involving the assessment of clonidine (a central α-2 noradrenergic agonist) found no benefit to this particular pharmacologic intervention.

Nootropics are a relatively new class of CNS-active drugs that have a direct functional impact on the higher integrative mechanisms of the brain. A few of the nootropic-like drugs that have been advocated to improve cognitive function include piracetam, etiracetam, aniracetam, pramiracetam, vincamine, dihydro-ergotamine, and centrophenoxine. Their chemical structures are quite different and their specific mechanisms of action are still unknown. Some of the proposed mechanisms of nootropic action include facilitation of dopamine release, increase of acetylcholine turnover, and inhibition of α-adrenoreceptors. One study[81] in TBI did demonstrate some beneficial effects of pramiracetam. Unfortunately, most of the more promising nootropic agents are still unavailable for clinical use in this country. The beneficial effects of Hydergine, a dihydrogenated ergot alkaloid, were recently reviewed by McDonald,[82] and he concluded that it produced some global improvement in memory. However, a recent well controlled study[83] using ergoloid mesylates for Alzheimer's disease failed to show any significant memory benefit. No studies have specifically assessed the utility of this drug for treatment of cognitive dysfunction in individuals after TBI.

Numerous drugs aside from Hydergine have been utilized to improve cognitive function secondary to their presumed beneficial effects on cerebrovascular blood flow. These drugs include papaverine hydrochloride, cyclanedelate, naftidrofuryl, and pentoxifylline. Although some literature suggests a beneficial effect of these agents in geriatric populations with concomitant "dementia," there has been no substantial exploration of the benefits of these agents in persons with cognitive dysfunction following TBI.

Given the neurochemical complexity of cognitive processes, it should not be surprising that pharmacologic agents may have the potential to actually impair cognitive processes in both noninjured and injured brains. It is critical to remain aware of the *relative* risks of certain pharmacologic agents in terms of their *potential* to impede cognitive processing.

Three main classes of drugs are felt to have the potential to interfere with cognitive functioning by way of their basic neurochemical mechanisms of action: (1) catecholaminergic antagonists, (2) GABAergic agonists, and (3) cholinergic antagonists. Agents that block catecholaminergic receptor

sites have been linked with deficits in attention, concentration, and memory. The main drug categories in this group are neuroleptics and antihypertensives. Neuroleptics such as haldol, thiothexene, and mellaril are primarily used for treatment of behavioral disturbances and act by dopaminergic blockade. Ideally, they should only be used for treatment of acute agitation and are rarely needed for long term behavioral management. Antihypertensives such as methyldopa, propranolol, and prazosin act by noradrenergic blockade and, therefore, may impair cognitve function in the individual with brain injury. All possible attempts should be made to avoid these agents given the availability of many other antihypertensives that act peripherally and are just as clinically effective (ie, ACE [angiotensin converting enzyme] inhibitors, calcium channel blockers, etc.).[84]

The multiple potential clinical uses of GABAergic agonists include seizure management, spasticity treatment, control of aggression, and as sedative hypnotics. The adverse cognitive effects that have been reported with this class of drugs include state-dependent learning, paradoxical agitation, and transient global amnesia. One must, therefore, realize that medications such as valium, baclofen, clonazepam, lorazepam, and temazepam, are not innocuous agents with regard to their potential cognitive side effects.

The association of anticholinergic use and cognitive impairment is by no means foreign to most practicing physicians. The fact that antidepressants are so commonly prescribed following TBI bespeaks judicial use of this class of medications, including an awareness of the relative anticholinergic potencies of specific agents.[84] Given the fact that newer and less anticholinergic agents are now available (eg, fluoxetine, trazodone, sertraline, and paroxetine), it would seem reasonable to assess the efficacy of these agents in individuals with TBI to ascertain the true potential of these agents in the treatment of organic affective disorders.[85]

Hemiinattention and Neglect

Ascending dopaminergic pathways have been experimentally implicated in mediation of attentional processes, including hemispatia neglect. Two small studies have demonstrated a potential utility of dopamine agonists, specifically bromocriptine, in the treatment of neglect secondary to cerebrovascular accident[86] and TBI.[87] Both studies utilized an A–B–A paradigm and demonstrated significant differences in testing performance, as well as functional capabilities, while patients were receiving dopamine agonist pharmacotherapy. Further studies are obviously warranted based on the encouraging results of these two.

Movement Disorders

A variety of movement disorders have been treated with some success following brain injury. These include dystonia, tremors, Parkinsonism, tics, akathisia, myoclonus, and dyskinesias (such as chorea, ballismus, and athetosis).

Dystonia, whether focal, segmental, or generalized, has been treated with a variety of agents, but with mixed results. Dopaminergic agonists and antagonists, anticholinergics, baclofen, benzodiazepines, and carbamazepine have all been utilized in the treatment of this class of movement disorders.[88,89]

Following TBI, tremors are typically of the postural or kinetic type (or both), whereas resting tremors are typically seen with nontraumatic degenerative cerebral disorders, which result in dopaminergic deficiency. Pharmacologic treatment tends to work better for nontraumatically induced tremor than for tremor resulting from trauma. A variety of drugs have been utilized, including β–adrenergic blocking agents, benzodiazepines, dopaminergic agents, valproic acid, and anticholinergics.[90–92] Drug induced tremor must always be considered a result of iatrogenic prescription or patient use of nicotine, or both.[93]

Parkinsonism, when a result of trauma, can be treated fairly well with pharmacologic intervention. Following diffuse brain injury, numerous authorities have reported patients with Parkinsonian-like symptoms, such as bradykinesia, dysarthria, decreased facial expression, and rigidity.[79,94] Drugs that have been shown to be effective for " posttraumatic Parkinsonism include dopaminergic agonists and, to a lesser extent, anticholinergics.

Tics are a rare consequence of acquired brain injury.[88,89] The drugs used to treat tics include GABAergic agonists, dopamine antagonists, and to a lesser extent, noradrenergic drugs, such as clonidine.[47]

Akathisia has been reported following brain injury and in animal models is thought to be associated with a relative dopaminergic deficiency in the prefrontal area. Successful treatment of akathisia using bromocriptine has been reported.[95] Other drugs that have been utilized, but with fairly limited success, include benzodiazepines and β-adrenergic blockers.[96] Myoclonus is a common sequela of severe hypoxic ischemic brain injury, but can also

be seen after nonhypoxemic brain injury. Cortical myoclonus must be differentiated from epilepsy partialis continua.[97] A variety of drugs, including benzodiazepines (clonazepam), serotonergic agonists (such as trazodone and L-tryptophan), valproic acid, primidone, and piracetam, have all been reported effective.[98,99]

Dyskinesias can occur in a variety of conditions and be manifested as ballismus, chorea, or athetosis. As a result of trauma, these types of movement disorders can result from thalamic or striatal injury, or both. Typically, the drugs that have shown some utility for TBI associated dyskinesias include dopaminergic antagonists and a variety of anticonvulsants, including carbamazepine, phenobarbital, valproic acid, and phenytoin.[88,100,101] It should be noted that certain dyskinesias may actually be atypical presentations of posttraumatic epilepsy.

Neurogenic Heterotopic Ossification

The only pharmacologic therapies presently available to minimize the extent of morbidity associated with neurogenic heterotopic ossification following brain injury involves the use of etidronate disodium[102] and nonsteroidal antiinflammatory agents (NSAIDs). Didronel presumably works by interfering with biological calcification; specifically, impairing the calcification of osteoid. When there is still an acute phase to the condition, NSAIDs have been advocated to decrease the suspected inflammatory component of this pathologic process.

Didronel therapy is typically initiated at 20 mg/kg/d and the dose is subsequently lowered after several weeks to months to 10 mg/kg/d. There are no well controlled, reliable trials examining the use of this agent in homogeneous brain injury populations; therefore, many, if not all, of the recommendations are based on spinal cord injury literature. The main side effect of the medication involves gastrointestinal complaints in the form of diarrhea and nausea.

Posttraumatic Seizures

At the present time, most neurosurgeons in this country use either phenytoin or phenobarbital for early management of seizures or seizure prophylaxis (or both) due to the fact that these medications can be administered parenterally (by intravenous route in the acute care setting). It is still unclear as to the exact utility of anticonvulsant agents in the prevention versus suppression of posttraumatic seizures.[103–105]

Recent literature by Temkin and associates[103] is highly supportive of the conclusion that phenytoin treatment for prophylaxis (treating with anticonvulsant medication to suppress potential seizures even though none have occurred) is efficacious only during the first week postinjury. Ongoing research also reveals that prophylaxis with other agents (such as carbamazepine) is also ineffective. Studies are presently underway to examine the prophylactic use of other agents, such as valproic acid. Except in very high risk cases, such as a penetrating brain injury, the common practice in most progressive rehabilitation centers is to discontinue anticonvulsant treatment if it has been prescribed prophylactically. Even in high risk cases it may be prudent to wean patients off medications and see how they do, treating only if they declare themselves. Whether or not to treat after one late (after the first week) seizure or to wait for the second late seizure remains controversial.

A current trend within the field of brain injury rehabilitation is to advocate the utilization of specific anticonvulsants in the postacute setting following brain injury (traumatic or nontraumatic), specifically, carbamazepine and valproic acid.[105,106] In general, carbamazepine should be a first line agent for treatment of partial seizures, whether simple or complex. On the other hand, valproic acid should be the agent of choice for multifocal epilepsy and generalized tonic-clonic seizures. This is not to say that select patients may not achieve better seizure control, potentially with fewer side effects, on agents such as phenytoin and phenobarbital. Clinicians should also be aware that valproic acid has been associated with encephalopathy and alterations in consciousness, most likely secondary to hyperammonemia.[107] On the other hand, recent experimental evidence suggests that valproate may be the most efficient agent relative to suppression of kindling phenomena. Another advantage that psychotropic anticonvulsants have over other seizure medications are their potentially positive effects on behavior. This is not to say that some patients may not do better on other agents, such as phenytoin for seizure control; however, the clinician must examine all aspects of a particular agent prior to instituting treatment. Obviously, drug interactions, side effects, cost, and compliance issues of the various anticonvulsant medications must be taken into consideration on a case-by-case basis.

Various studies[108–110] have demonstrated significant negative effects on cognitive function secondary to phenytoin and phenobarbital. A recent study[111] questioned these general findings, bringing

to light the need for further research in this area. A variety of newer agents, such as oxcarbazepine, felbamate, vigabatrin, flunarizine, lamotrigine, and others, are presently being studied in an attempt to develop more effective drugs with fewer cognitive and systemic side effects.[112]

Sexual Dysfunction

Following brain injury, it is common for individuals to have problems in the area of sexuality. One of the most regular complaints is alteration in libido.[113] Hyposexuality can be treated with a number of different pharmacologic agents, including activating antidepressants, yohimbine (a noradrenergic agonist), dopamine agonists, and hormonal supplementation.[114] Hypersexuality, on the other hand, is a relatively rare clinical condition that is more difficult to broach from a pharmacotherapeutic standpoint. Hormonal agents, specifically, medroxyprogesterone acetate, have been utilized to "chemically castrate" individuals with severe hypersexuality problems.[115] For patients who have bitemporal involvement and associated hypersexuality as seen in Kluver-Bucy syndrome, carbamazepine is generally considered the treatment of choice.[116] Other agents that may hold potential utility for treatment of the hypersexual patient following brain injury include serotonergic GABAergic and opioid agonists.[115] There are obviously significant ethical and medicolegal ramifications in the utilization of agents affecting sexual drive in this population.

Speech and Language Disorders

A number of different medications have successfully been used for a variety of speech and language disorders in patients with brain dysfunction. Bromocriptine has been reported to improve speech dysfunction in patients with diffuse TBI with dosages ranging from 20 to 40 mg daily.[117] Another series of studies demonstrated the efficacy of bromocriptine in the treatment of dysphasia, specifically, the transcortical motor variant.[118,119] Animal studies have yielded some support for the role of dopaminergic pathways in both spontaneous and reflex swallowing,[120,121] leading to human studies that support the potential efficacy of dopamine agonist therapy for dysphagia following brain injury utilizing L-dopa/carbidopa.[122] Parkinsonian hypokinetic dysarthria has been treated with low dose clonazepam (0.25–0.5 mg/d); the probable mechanism for its efficacy being striatal GABAergic agonism.[123] Lastly, a case of posttraumatic adult onset stuttering responsive to anticonvulsant treatment has been reported, suggesting that ictal speech disorders should always be considered in this patient population.[124]

NEUROMEDICAL ISSUES

There are numerous neuromedical issues that the physiatrist must take into consideration when treating the survivor of TBI. Early identification of cranial nerve injuries not only has potential prognostic significance, but is also critical relative to institution of appropriate treatment regimens.[125] The clinician must be familiar with the diagnosis and management of central hyperthermia and autonomic dysregulation syndrome,[61,126] neurogenic heterotopic ossification,[127] hypertonicity,[128] fractures,[129] nutritional issues,[130] pulmonary and tracheostomy problems,[131] peripheral nerve injuries including neuropathies and plexopathies,[132] and concurrent spinal cord injury.[133] It is also critical for the physiatrist to have specific knowledge of subpopulations of TBI patients, including pediatric and geriatric.[134–136] A number of neurologic conditions may not manifest themselves until sometime after the initial neurologic insult. Posttraumatic communicating hydrocephalus may occur in up to 8% of survivors of severe TBI and typically present with evidence of neurologic deterioration or plateau in the face of ongoing ventriculomegaly without associated sulcal enlargement.[137] Sophisticated procedures, such as cerebrospinal fluid infusion studies, may assist the clinician in making a more definitive diagnosis and proceeding with shunting, the definitive treatment. Subdural hematomas and hygromas may develop and progress in the postacute period, thereby providing some rationale for the practice of follow-up imaging.[138] Posttraumatic seizures and epilepsy are relatively common clinical conditions, particularly after severe TBI, and physiatrists should have a thorough understanding of the potential clinical presentations, workup, and treatment of this class of posttraumatic disorders.[139] Although rare, other late neurological disorders include posttraumatic movement disorders, neuropsychiatric complications, functional deterioration associated with aging, and certain neuroendocrine disturbances.

Cognitive and behavioral problems are frequent following all severities of TBI. Cognitive dysfunction may take many forms. Clinicians must ad-

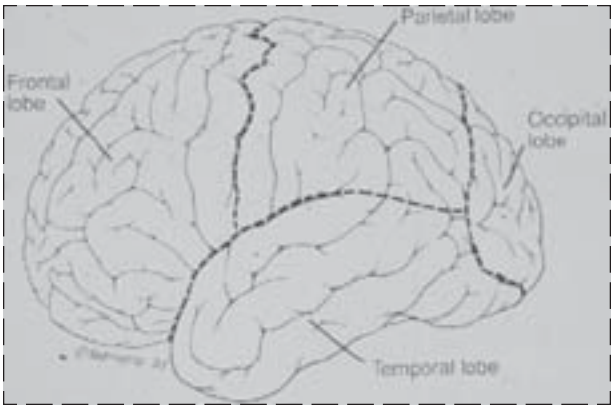

Fig. 6-3. Basic surface anatomy of the brain demonstrating the relative demarcations for the four main lobes: frontal, parietal, temporal, and occipital. Reprinted with permission. Copyright Robert Shepherd. Shepherd Visual Communications: Richmond, VA; 1993.

equately delineate the type of dysfunction present to determine if it is at all remediable. Many concurrent issues may exacerbate cognitive problems, including inappropriate medication prescription, vestibular problems, sleep–wake cycle dysfunction, chronic pain due to posttraumatic headache or myofascial dysfunction, and visuovestibular dysfunction. Although, as previously mentioned, there are no magic bullets for cognitive dysfunction, the professional may want to consider more traditional cognitive remediation interventions, including computer based cognitive retraining. There is generally a trend toward functionally based remediation of cognitive dysfunction, preferably in the environment in which the behavior occurs, whether at home or work.[140] As previously discussed, pharmacotherapeutic interventions may also augment the potential benefit gained from more traditional physiatric interventions.[141] Patients with other factors exacerbating their cognitive dysfunction must have these factors addressed concurrent with other interventions.

Behavioral issues are relatively commonplace following TBI. Comprehensive assessment should include elicitation of an adequate history, use of symptom rating scales and behavioral assessment measures, evaluation of brain structure and function by way of imaging and electrophysiological assessment, and neuropsychological evaluation. Several behavioral assessment tools are available, including the Structured Clinical Interview,[142] Neurobehavioral Rating Scale,[143] Hamilton Rating Scale for Depression,[144] and the Overt Aggression Scale.[145] A variety of neurobehavioral alterations may be seen following brain insult. Personality changes, intellectual deterioration, and affective disorders may manifest themselves as a direct result of TBI. Other conditions, not directly related to TBI, must also be taken into consideration, including posttraumatic stress disorder, reactive depression, and sleep–wake cycle disturbances related to pain from myofascial dysfunction or orthopedic injuries, or both. Organically based affective disorders may present as depression, mania, delirium, and psychosis. Anxiety, aggression, irritability, mood lability, and sleep–wake cycle disturbances are also quite common in this patient population.[146] Clinicians should be aware of both behavioral and pharmacological approaches to the management of behavioral issues.[147] Some of the other issues that may aggravate behavioral problems following TBI include chronic pain, vestibular dysfunction, organically based fatigue, medication side effects, substance abuse, preinjury psychologic and psychiatric dysfunction, organic affective disorders, recurrent head injury, learned maladaptive behaviors, and situational specific conflicts.[146] Clinicians should also be familiar with the array of neurobehavioral conditions that may be seen with brain injury, both focal and diffuse. Knowledge of regional brain anatomy (Figure 6-3) and neurobehavioral correlates is critical for the practicing clinician who works with TBI survivors (Table 6-1).

TABLE 6-1

NEUROBEHAVIORAL PROBLEMS ASSOCIATED WITH DAMAGE TO SPECIFIC LOBES OF THE BRAIN

Lobe	Problems
Frontal	Orbitofrontal and dorsolateral neurobehavioral syndromes
Parietal	
Dominant	Gerstmann's syndrome (finger agnosia, dysgraphia, dyscalculia, and left-right disorientation)
Nondominant	Topographical disorientation, dysprosodia, and neglect
Temporal	Episodic dysphoria, alterations in libido, episodic dyscontrol, temperolimbic epilepsy (TLE)
Occipital	Anton's syndrome, cortical blindness

ADVANCES IN FUNCTIONAL ASSESSMENT

As brain injury rehabilitation has matured, clinicians have realized the critical need for valid and reliable assessment and outcome evaluation measures. The development of such measures has resulted from a lack of applicability of overall health and functional measures from other areas of medical rehabilitation, and general and mental health. Physiatrists working with TBI survivors must delineate the cornucopia of impairments that each patient presents, and must also identify how these specific impairments result in disability and handicap.

Clinicians have tried to develop outcome and functional assessment measures that correlate with real world function. These measures may also be used to track functional progress, response to a specific treatment or nontreatment, and to assess the efficacy of specific interventions or programs, by utilizing review and case management for possible outcome prognostication. Prior to utilizing any measure for any purpose, clinicians need to be fully apprised of the specificity and sensitivity of these scales, as well as their validity and reliability.[148]

Many general status measures have been developed for TBI,[149] including the Disability Rating Scale, Rancho Los Amigos Scale, Glasgow Outcome Score, and Glasgow Assessment Scale. Other measures have been designed specifically to use with patients at low levels of neurological function, including the Coma Recovery Scale, Coma/Near Coma Scale, Sensory Stimulation Assessment Measure, and Western Neurosensory Stimulation Profile. Several scales address multidimensional functional status evaluation, including the Functional Inventory Measure, Functional Assessment Measure and Patient Evaluation Conference System, and others.

There are a number of more focused clinical measures for delineation of communication, cognitive, behavioral and psychosocial function. Measures addressing community and vocational integration, and life satisfaction and stress have been less well developed for this specific patient population. Refer to Exhibit 6-1 for a review of specific measures for individuals with TBI.

It is critical for clinicians to realize that there are inherent limitations in functional assessment and outcome measures. For example, measures, per se, do not give evidence of intervention effectiveness. Use of functional measures to "prove" effectiveness is at best an inference based either on research design or preexisting causal knowledge garnered from controlled research.

There are also a number of domains of function that are not adequately addressed in TBI functional assessment and outcome measures. Some of these parameters that are particularly critical include psychosocial functioning, avocational activities, neurobehavioral status, higher level physical and cognitive deficits, limitations secondary to lack of insight and judgement, and job maintenance capability.

Given the aforementioned, it is extremely important for clinicians to understand why they are utilizing a specific measure, and the strengths and limitations of each instrument. When applied appropriately, functional outcome and assessment measures can serve the clinician, third party payers, patient, and family in a positive manner. When used indiscriminately, these measures can do more harm than good.

EXHIBIT 6-1

BRIEF REVIEW OF SOME OF THE MORE FREQUENTLY UTILIZED FUNCTIONAL ASSESSMENT MEASURES IN TBI REHABILITATION

General Scales

- Disability Rating Scale
- Glasgow Outcome Scale
- Modified Barthel Index
- Functional Independence Measure (FIM)
- Functional Assessment Measure (FAM)
- Rancho Los Amigos Scale
- Adapted PULSES
- Glasgow Assessment Schedule

Scales for Persons with Severe Alterations in Consciousness

- Coma Recovery Scale
- Western Neurosensory Stimulation Profile
- Sensory Stimulation Assessment Measure
- Coma/Near Coma Scale

MODEL SYSTEMS CONTINUUM OF CARE FOR TRAUMATIC BRAIN INJURY

The need for a comprehensive continuum of neuromedical and rehabilitative care for individuals with TBI, regardless of severity, cannot be overemphasized.[150] Coordination of services, across medical disciplines and among healthcare specialists will increase the ultimate quality of patient care. Timely and appropriate intervention also decreases both short- and long-term morbidity and mortality associated with TBI.[151] The VA medical system has recently designated a select number of its hospitals to serve as brain injury treatment centers and provide comprehensive services to soldiers with TBI. In the military there are frequently no physiatrists assigned to evacuation or corps-level hospitals.

Leadership Issues

It has become apparent from the Model Systems[152] experience that the success of the interdisciplinary team, and the system itself, is dependent on a strong team leader. For a team to fully integrate across interdisciplinary as well as multidisciplinary "barriers," there must be firm guidance and strong leadership from a senior clinician. The central leadership role is, in essence, the glue that holds the system together. In the field of brain injury rehabilitation, many challenges face physicians relative to both clinical and nonclinical training. These include brain injury experience during residency, specialization controversies, continued medical education, and an ability to serve in administrative capacities as leader of an interdisciplinary or transdisciplinary team.[152,153]

General TBI Education

Ignorance of TBI sequelae is quite common in the community at large and among health-care professionals. Regional TBI education by neuromedical and rehabilitative professionals who specialize in TBI care, both acute and chronic, should be included in a comprehensive rehab-ilitation program. This would serve not only to increase awareness of the problems faced by individuals with TBI, but would improve the quality of care given to such individuals.[152]

Emergency Medical Services

Appropriate emergency medical services are an essential feature of comprehensive TBI neuromedical care continuum. The ultimate goal of a well trained and efficient casualty evacuation service is rapid, early intervention; patient stabilization; and subsequent transport to a *predesignated* medical facility that can provide the equivalent of level 1 trauma center care for patients with severe head injuries (as defined by a GCS score of 3 to 8). Designated military medical facilities treating moderate and severe brain injury should have neuroimaging facilities available; specifically, computed tomography (CT) scanning and magnetic resonance imaging (MRI). Based on ongoing research, other imaging modalities, including single photon emission computed tomography (SPECT) may soon become standard. Appropriate ground and air transportation services should be available to transport more severely injured patients to level 1 trauma centers or their functional equivalent. Expeditious evacuation from the accident or battle scene to acute neuromedical facilities will allow for more accurate neurosurgical diagnosis and treatment, thereby minimizing secondary brain injury and its associated morbidity and mortality. Any patient seen in emergency room facilities who is subsequently discharged to duty should have appropriate supervision and adequate monitoring, as well as referral for follow-up if needed. If appropriate supervision is unavailable, the patient should be admitted to the hospital for observation.[152]

Professionals evaluating more subtle head injuries and TBI should be aware of criteria that justify hospital admission, such as GSC scores of less than 15, focal neurologic deficits, altered mental status, abnormal CT scans, and so forth.[3] Mild brain injured patients evaluated in emergency room facilities should be given general information sheets and provided with appropriate information regarding postconcussional disorders. Line commanders should be educated regarding postconcussive symptoms and the specifics of monitoring behavioral and performance abilities of their soldiers. If the soldier exhibits any lasting postconcussive problems, more definitive assessment is warranted through referral to a larger military hospital.

Acute Neurosurgical Care

Patients admitted for treatment or observation, or both, regardless of severity of injury, should be screened, preferably by a rehabilitation medicine

consultation service. Proper communication across disciplines, that is, neurosurgery and rehabilitation medicine, has proven to be a critical factor in the development of a continuum of quality care for TBI patients. Additionally, it is not uncommon that problems that lead to potential morbidity issues and higher level cognitive linguistic deficits are sometimes overlooked by acute care physicians, thereby necessitating the involvement of a physiatric consultant. If possible, interdepartmental rounds should occur to review patient condition, individual team recommendations, and disposition issues. A timely and efficient hierarchy of communication should exist in order to inform consulting rehabilitative services of patient admissions to the neurosurgical service. Routine interservice communications ultimately allow for smoother transitions of care if and when the patient is transferred to the care of the rehabilitation medicine physician.

Neuromedical and rehabilitative issues that can be addressed by the physiatrist for patients with mild TBI include diagnosis and treatment of postconcussive symptoms, such as posttraumatic headaches, audiovestibular disorders, balance disorders, visual changes, olfactory and gustatory dysfunction, and cognitive behavioral deficits.[3]

For more severely injured patients, the major issues of neuromedical management to be addressed by the physiatrist include skin care, bowel and bladder management, behavioral management, tone control, maximization of nutritional status, maintenance of joint range-of-motion, and optimization of the patient's potential for maximum neurologic and functional recovery through both pharmacologic and nonpharmacologic modalities. During the acute period of treatment, particularly in cases of severe injury, it is essential to have a multidisciplinary neuromedical staff available.[152] The staff must be familiar with complications associated with TBI including, but not limited to, neurogenic heterotopic ossification, traumatic myositis ossificans, posttraumatic seizures, spasticity management, neuroendocrine disorders, neuroophthalmologic problems, olfactory dysfunction, audiovestibular deficits, orthopedic injuries, dysphagia, tracheostomy management, and posttraumatic psychologic, as well as psychiatric disturbances.[154]

Services that should be readily available for consultative purposes as part of the acute continuum of TBI care include neuroophthalmology, ophthalmology, gastroenterology, general and orthopedic surgery, neurology, dental medicine, oral surgery, plastic surgery, urology, substance abuse, dermatology, and ear, nose and throat.

In both acute neurosurgical and acute brain injury rehabilitation care, prevention of morbidity is a critical contribution of the physiatrist and rehabilitation team. Specifically, aggressive efforts should be made to counteract the adverse effects of immobility. Early care that can decrease the complications associated with protracted immobilization are (*a*) passive and passive assisted range-of-motion exercises to decrease muscle atrophy, (*b*) mobilization efforts (ie, getting the soldier out of bed once intracranial pressures [ICPs] are controlled), (*c*) contracture prevention through positioning, (*d*) ranging and splinting, (*e*) turning protocols to prevent skin breakdown, and (*f*) deep vein thrombosis prophylaxis. Spasticity treatment, including potential use of neurolytic agents for motor point or nerve blocks (or both), should also be a focus of early rehabilitative care.

Acute Brain Injury Rehabilitation

Once patients are medically stable, and at the discretion of the consulting physiatrist, certain patients with brain injury may meet criteria for admission to acute inpatient brain injury rehabilitation programs. Ideally, these units should be dedicated to the severe TBI patient population, in both the space allocated for the unit and the treatment team. In the modern military, this may mean certain designated continental medical centers or specialized VA hospital facilities. Use of a transdisciplinary team approach, and training the staff to be sensitive to medical and psychosocial issues commonly encountered after TBI helps to maximize treatment efficacy. Specific admission criteria should be developed for all units.

As appropriate, some programs should attempt to designate separate units or a small portion of bed space to "early recovery management programs"[2] for patients who exhibit slow neurologic recovery. Ideally, patients should be admitted within the first 3 months postinjury; however, patients who are further along than 3 months postinjury should also be considered as candidates. It should be understood that their potential for recovery is much smaller than the potential of those patients who are within the first 3 months postinjury.

Slow to recover patients should generally be given a 2 to 3 month trial of inpatient care with the goal set at maximization of their recovery potential and minimization of neurologic and functional morbidity. If there are no significant improvements, other dispositions, such as long-term placement, need to be considered. Long-term care facilities,

typically based in skilled nursing homes, should be staffed by healthcare professionals adept at dealing with the multitude of functional and neuromedical issues relevant to TBI survivors. Some opportunity for rehabilitative follow-up is critical in order to assess neurologic or functional change, whether positive or negative, and the need for appropriate modification of existing treatment plans.[154]

Ideally, a transdisciplinary team approach should be implemented when working with survivors of TBI. The team works with the patient and the family to maximize recovery from both a neurologic and functional standpoint. The team consists of a variety of disciplines beyond the medical ones. These disciplines include rehabilitation nursing, physical therapy, occupational therapy, speech-language pathology, cognitive therapy, neuropsychology, rehabilitation nursing, rehabilitation social work, dietetics, pharmacy, and spirituality/religion. Team rounds and conferences should be held regularly, and, when possible, all treating team members should be in attendance. This process increases the team's ability to assess and treat the variety of issues that may be new or ongoing with regard to the individual's recovery and rehabilitation. Preferentially, didactic lectures, journal club, and weekly administrative meetings should be held to promote the cohesiveness of the team.[153]

All patients admitted to an inpatient brain injury rehabilitation unit should receive a complete neuromedical workup to fully assess any factors that may be compromising their neurological or functional recovery process. The standard neuromedical workup should include a thorough history and physical; including full neurologic and functional assessment; EEG; static brain imaging; neuroendocrine assessment as indicated; as well as full nutritional and metabolic evaluation, and comprehensive evaluation to rule out concurrent infection. To rule out inappropriate or excessive medications, a thorough review and assessment of medications must be conducted, preferably in conjunction with a pharmacist. As indicated, patients should be seen by a psychiatric consultant for evaluation and treatment of postinjury neurobehavioral sequelae. Preinjury substance abuse issues, which may effect short- and long-term potential, should be addressed as early as possible with the assistance of substance abuse consultants.[6,155]

A critical part of any brain injury rehabilitation program, regardless of the neurologic or functional level of the patient, should involve family training. Early and ongoing family involvement as active members of the rehabilitation process, including the time after discharge, improves patient outcome and should be encouraged. Institutional as well as community resources should be developed to allow families to cope with the changes that have incurred in their own lives, as well as the life of their loved one. Such resources include rehabilitation service support groups and community support groups through such organizations as local and state chapters of the National Head Injury Foundation (NHIF). (The main office is in Washington, DC and can be contacted at 202-296-6443 or 1-800-444-6443.)

Outpatient Clinical Services

Once patients are discharged from an inpatient brain injury rehabilitation unit they will require ongoing therapeutic services. Patients who are referred to the outpatient clinic, regardless of the severity of injury, should be evaluated by a transdisciplinary outpatient team. If such a team is not available, there should be an assessment performed by the releasing rehabilitation medicine physician in conjunction with the new unit's available therapy staff. All relevant areas should be addressed, including issues relating to mobility status, activities of daily living (ADLs), communication, bowel and bladder status, cognitive and behavioral status, sexuality, and vocational and avocational status. Both patient and family should be included in these discussions.

Appropriate professional resources should be available to work with patients in the outpatient clinic setting. These professional resources include psychologic, neuropsychologic, rehabilitation nursing, and rehabilitation social work. Outpatient clinic staff, including the physician, should be familiar with resources within the community and the surrounding regions so they can optimize the quality of long term care services for the patient and his family. Such services include driver evaluation designed to assess driving skills after TBI, vocational services, behavioral management programs, transitional and independent living programs, and long term care facilities. Appropriate coordination of outpatient rehabilitation services, including day rehabilitation and outpatient therapies, is critical to providing a smooth transition from inpatient status. Families should be referred to a variety of community resources, including their local chapter of the Brain Injury Association (BIA). Community mental health services and recreational programs should be recommended as needed. Literature on TBI from various sources, including BIA, should be distributed to patients and families to increase their understanding and awareness of TBI deficits.

Special diagnostic and therapeutic programs should be implemented in all facilities caring for TBI patients, and for those individuals who have suffered so-called mild traumatic brain injury with associated postconcussive symptoms or cognitive-behavioral dysfunction, or both. Implementation of such programs requires that the treating physician have a thorough knowledge of the etiology, diagnosis, and management of these problems. Additionally, adequate resources, either within the institution of origin or in the surrounding community, are necessary for accurate diagnosis and management of specific postconcussive disorders, including audiovestibular dysfunction, cognitive and behavioral disturbance, and posttraumatic headache. An integral part of such a program should include compensatory strategies for attention deficits, memory problems, and impaired mental flexibility; rapid processing is imperative. For the patient with mild TBI, the neuropsychological staff must have specialized training and expertise in TBI evaluation, interpretation, and treatment. Counseling services for patients and families should be available to help their adjustment to associated sequelae. Higher level cognitive and behavioral changes, which may interfere with vocational reintegration, need to be addressed by a qualified vocational specialist.[156]

Neurobehavioral Programs

Neurobehavioral programming is one of the least met needs of survivors of TBI relative to the continuum of clinical care. Typically, neurobehavioral programs are rendered in skilled nursing facilities or in community based environments. Only a few select programs exist in acute care hospital settings, and those are for the most severe patients who also might require aggressive and significant neuromedical workups. This problem stems from a lack of a generally agreed-on programmatic content for this aspect of treatment. Additionally, the level of expertise in nonpharmacologic and pharmacologic management varies greatly across programs. Given that psychosocial and behavioral issues make a major subset of posttraumatic sequelae and compromise individual survivor capacities for significant community reentry, it is surprising that so few resources have been allocated to this clinical service.[157]

Home Based Services

In recent years, one of the major advances in service provision has been the development of home based, neurologic rehabilitation care. This service has allowed patients to make quicker transitions from institutionalized care to their home environments. It also negates the significant concerns regarding the general applicability of information learned in environments foreign to the home setting (ie, instructions). Many community based rehabilitation programs go beyond administrative home care and extend their services to vocational and avocational activities. The successful programs to date have utilized a model of physician directed transdisciplinary care, with a sensitivity to the general preponderance of the individual's nonneuromedical needs. General healthcare trends suggest that such programs, from both a clinical and cost efficacy standpoint, may usurp many of the present modalities of providing rehabilitation care.[158]

Vocational Rehabilitation

An ultimate measure of how successful rehabilitative efforts have been is how well survivors of TBI are able to reintegrate into the work place. Numerous methodologies are being utilized to facilitate work reentry, including vocational retraining, supported employment, sheltered workshops, and work hardening.[159] The combination of early intervention and follow-up tends to maximize results of vocational reentry efforts; however, it does not guarantee success. Vocational reentry efforts must focus on the functional strengths of the person with TBI, and must also be keenly sensitive to areas of functional deficit. It is not yet known which methodology works best for patient population subtypes relative to preinjury job characteristics and education, postinjury sequelae, or sociocultural background, but to achieve a maximal level of survivor interdependency, both in and outside the work place, skills that are applicable in the "real world" must be taught.[160]

Trends in Service Provision: Case Management

Case management is probably one of the strongest movements in the field of brain injury rehabilitation. In the best of worlds, case management can provide TBI patients a safety net in several ways: (*a*) the case manager can assist in securing needed services while at the same time assuring that every dollar spent is used judiciously; (*b*) case management can provide a "common-thread" that spans the network of services each survivor must negotiate; and (*c*) when properly applied, timely institution of case management services, for patient and

family, produces better outcomes of neurologic and functional morbidity and lowers overall costs.[161]

Community Based Living Alternatives

For survivors of TBI who have significant cognitive and behavioral, or physical limitations, the options for community based living are typically quite limited. Just as a continuum exists for general service provision, so does one exist for community based living. The most restrictive settings are usually institutional and involve 24-hour supervised living environments and structured daily therapeutic intervention. As the environment becomes less restrictive, based on client ability to function more safely and independently, the level of supervision, as well as the intensity of that supervision, typically decreases. Additionally, clients tend to spend less time institutionalized and more time "in community" with concomitant increases in personal freedoms and free choices.[162] One area in the service continuum for community based living that needs more attention is that of accessible and affordable housing for cognitively and physically challenged TBI survivors.[163] As comprehension of how to maximize client interdependency and simultaneously protect civil rights and other constitutional freedoms improve, the continuum of community based services will ultimately develop and grow.

For a continuum of neuromedical and rehabilitation care to be truly effective, there must be a multifaceted approach that involves preventative education, as well as rehabilitation; and that develops institutional and community based TBI services, maximizes communication across medical disciplines, and promotes better integration of rehabilitation professionals in the long-term management of individuals with TBI. To broaden service access, there is also a need to network community providers who are inside and outside the immediate field of rehabilitation. Lastly, healthcare providers and clinicians must be willing and committed to examining the efficacy of rehabilitation interventions. This is the only way to fully maximize the neurologic and functional outcome for TBI patients, and to also optimize their reintegration into society.[164,165]

CURRENT ISSUES IN THE REHABILITATION OF THE SOLDIER WITH TRAUMATIC BRAIN INJURY

Role of the Physiatrist

A physiatrist is a practitioner of PMR, a recognized medical specialty established in 1947. It is concerned with (*a*) the optimal functional restoration of patients with disabilities; (*b*) physical treatment of neuromuscular impairments; and (*c*) the use of electrodiagnostic studies, including electromyography, and evoked potentials. The more familiar medical specialties, such as internal medicine, orthopedic surgery, and neurology, address the diagnosis and treatment of specific diseases or conditions that result in disability. By comparison, a physiatrist focuses on the diagnostic, therapeutic, and management procedures that will potentially enhance an individual's residual functional capabilities. As opposed to other medical specialties, the emphasis, both in terms of residency training and eventual clinical practice, is on the maximal physical functional capacity and psychosocial adjustment of the physically challenged individual.

Physiatrists approach the patient from a holistic view and address not only the rehabilitative and neuromedical issues, but also lead the interdisciplinary therapy team in the long-term care of the disabled individual. Physiatry, clinically rooted in basic sciences that include anatomy, kinesiology, exercise and muscle physiology, nerve physiology, and biomechanics, also has a clinical suprastructure that combines elements of internal medicine, neurology, neurosurgery, cardiology, rheumatology, orthopedics, pediatrics, geriatrics, and the behavioral sciences. This broad foundation of scientific and clinical knowledge particularly qualifies the physiatrist to evaluate and treat the complicated problems of individuals with disability, and to manage interdisciplinary and transdisciplinary teams.[153]

Mild Traumatic Brain Injury

Mild TBI, although quite prevalent, is still poorly understood by most clinicians, and thereby promulgates many of the fallacies and foibles common to this diagnostic label. This discussion is designed to educate professionals and acknowledge the need to diagnose and treat individuals with mild TBI appropriately from as early postinjury as possible.

Mild TBI accounts for approximately 80% of all traumatic brain injuries.[166,167] It typically results from motor vehicle accidents and involves young males 15 to 24 years of age.[166,168] The role of alcohol use in injuries resulting in mild TBI seems to be significant and that fact should not be ignored in the development of accident prevention programs.[168,169] Many individuals who incur mild TBI do not seek medical attention in acute hospital settings, if at all,

thereby causing an underestimation of the true incidence of this phenomenon.

Given the incidence of this condition, it is crucial for practitioners to be familiar with the diagnosis and treatment of problems that individuals with mild TBI may present. Postconcussive sequelae may have an adverse impact on an individual's ability to function well in a number of different spheres, including physical, emotional, social, marital, vocational, and avocational.[170] The professional healthcare provider must also be aware of resources within the immediate community that provide neuromedical and nonmedical services for this special population of patients. The BIA is an excellent source of information on TBI, mild and otherwise, for professionals, "survivors," and families alike. Typically, each state has its own affiliate association of the BIA.

Mild TBI is defined as a traumatically induced physiological disruption of cerebral function as manifested by at least one of the following: (*a*) loss of consciousness of no longer than 20 minutes; (*b*) any loss of memory, either retrograde (memory loss for events prior to concussive injury) or anterograde (memory loss occurring after the injury and reflecting a time between injury and the point at which continuous memory is regained); (*c*) any alteration in mental status at the time of the accident, even in the absence of loss of consciousness or amnesia; (*d*) physical symptoms that are potentially brain related (eg, nausea, headache, dizziness, tinnitus, visual aberrations, olfactory deficits, or extended periods of fatigue); and (*e*) development of posttraumatic cognitive deficits that cannot be completely accounted for by emotional factors. Given these factors, TBI severity must not exceed the following in order to qualify as mild: (*a*) GCS score of 13 to 15 without subsequent worsening; (*b*) posttraumatic amnesia of 24 hours or less; and (*c*) loss of consciousness lasting no longer than 30 minutes.[171] Recently, several investigators have questioned the inclusion of individuals with intracranial lesions under the mild diagnosis, even if they meet the diagnostic criteria. This call for reconsideration of the present classification is due to the apparent higher incidence of significant neurobehavioral sequelae and resultant functional disability that occur in this subpopulation.[172]

It is critical to recognize that loss of consciousness is not essential for a diagnosis of TBI. Concussive injuries can occur without loss of consciousness, a so-called mild concussion. Recent evidence[172] also suggests that the severity of associated neuropsychological deficits is independent of the neurological status immediately following the injury. It should be noted here that even seemingly sound scientific studies, which have attempted to address such issues, have weaknesses and faults that open them up to further criticism.[173]

Concussive injuries occur along a continuum from mild to classic, the latter involving loss of consciousness. Direct impact to the skull is not necessary to incur TBI. The pathophysiologic hallmark of concussive brain injuries is strain, which can occur secondary to acceleration forces on the head, a stretched cervical spine, and skull distortion due to pressure gradients. Strain disrupts axonal function along a range from physiological disruption, due to transient alterations in membrane function, to actual pathological changes secondary to direct axonal injury.[174] Although the magnitude of strain may vary relative to anatomic variations, the force vectors are normally directed centripetally from the brain's center of gravity, which is approximately in the area of the pineal gland. Due to the centripetal nature of the force vectors, the cerebral cortex and lower brain stem are affected first, followed by the upper brain stem. Many of the transient physiologic responses seen in experimental models of brain injury can help explain the clinical picture of transient coma, pupillary and corneal areflexia, and decerebration seen with upper brain stem dysfunction.[174]

Rehabilitative evaluation of TBI patients should include a thorough history and a physical examination. Historical information should include accident circumstances, alteration in consciousness (dazed vs true loss of consciousness), presence and duration of retrograde (preevent) and anterograde (postevent) amnesia, blood alcohol level and drug screen (if available), as well as initial GSC score. Other significant information pertaining to the initial evaluation includes neurological status and any diagnostic data such as brain CT or MRI, cervical spine or skull films, or both. Pertinent preinjury information should be elicited, including (*a*) prior psychologic or psychiatric problems, or both; (*b*) history of learning disability; (*c*) prior substance abuse; (*d*) criminal record; and (*e*) any history of prior loss of consciousness or TBI. All these factors may adversely effect neurologic and psychologic recovery.[175] Due to the array of injuries and symptoms that many postconcussive patients may have incurred, it is imperative to consider central neurologic dysfunction as well as the peripheral injuries related to cervical hyperextension-hyperflexion injury (whiplash) and cranial and cranial adnexal trauma. Common postconcussive disorder symptoms are

- visual dysfunction: blurry and double vision;
- audiologic dysfunction: tinnitus, high frequency hearing loss;
- vestibular dysfunction: dizziness secondary to peripheral vestibular dysfunction;
- olfactory dysfunction;
- balance dysfunction;
- cognitive-behavioral alterations;
- sleep–wake cycle dysfunction; and
- fatigue.

The physical examination should include a thorough neurological evaluation, including a test of higher level cognitive and linguistic function. It is critical for the physician to be familiar with the associated musculoskeletal and peripheral neurologic (both somatic and autonomic) sequelae of head trauma, as well as cervical spine flexion and extension injuries. Adequate and timely recognition of myofascial pain disorders related to traumatic injury of the neck or cranium is critical due to the array of symptoms (both somatic and autonomic) that can be related to referred pain from trigger points in the facial, cranial, cervical, and upper back musculature.[176]

For proper diagnosis and treatment of injury related problems, it is critical to differentiate head trauma sequelae from true brain injury sequelae. Many postconcussive symptoms that are purported to be secondary to brain injury may actually be head injury sequelae. Head injury sequelae that may be mistaken for TBI-related problems include certain visual disturbances, olfactory and gustatory deficits, audiovestibular deficits, headaches, and peripheral nerve dysfunction (both somatic and sympathetic).[175,177] Postconcussive symptoms due to brain injury may include various types of visual sequelae; audiovestibular, olfactory, and gustatory deficits; headaches; imbalance; excessive daytime somnolence and fatigue; and sexual dysfunction.[178] Maximization of functional potential and expeditious recovery necessitates the appropriate use of pharmacologic agents to treat postconcussive problems of organic affective disorders, sleep–wake cycle disturbances, fatigue, and decreased libido.[170]

As necessary, patients should also be referred for therapy services. Physical therapy referrals should mainly focus on myofascial concurrents of injury and balance retraining, as well as vestibular habituation training. Occupational therapy services are appropriate if the patient presents with issues germane to higher level organizational difficulties that affect ADL, driving problems, decreased performance at work or school, perceptual difficulties, and functional memory and problem solving difficulties. Speech therapy referrals are appropriate when the patient presents with reading problems, auditory difficulties, verbal and written expression impairments, and pragmatic language deficits. Therapy should preferably be administered in an interdisciplinary, function oriented fashion.[175]

Neuropsychological evaluation is a critical component in the diagnosis and ongoing treatment of persons with mild TBI. This evaluation provides objective evidence of higher level cognitive and behavioral, linguistic, and motor dysfunction, not typically evident on bedside evaluation, and it also provides a basis providing compensatory strategies to both patient and family.[179] Neuropsychological testing also provides potentially critical medicolegal information that can be followed to demonstrate initial and subsequent profiles consistent with those seen after similar injuries. Neurodiagnostic tests may provide useful clinical and medicolegal information in this patient population. These tests utilize static, as well as functional imaging (SPECT and positron emission tomography); electrographic monitoring by EEG (with or without compressed spectral analysis); evoked potentials including, but not limited to, BAER (brain stem auditory evoked response) and cognitive evoked potentials (so-called P-300s); quantitative EEG; electronystagmography with calorics; posturographic evaluation; polysomnography; and olfactory and gustatory evaluation, among others.[3]

Patients and family members should be given educational materials and the clinician should explain the condition, history, and prognosis. A frequent and tragic occurrence in clinical practice is the patient with true postconcussive deficits who has gone from doctor to doctor only to be told there is nothing wrong, to the point where the patient actually thinks he is "losing his mind." The patient and family *must* understand what has happened to him, why he feels and behaves the way he does, what the prognosis is, and what can be done about the condition.[3]

In the early period after mild TBI, the patient should follow the directions of the emergency room physician. For the first few days after a more significant injury, the patient should be told to rest, avoid alcohol and caffeinated beverages, keep a regular schedule, avoid recreational drugs, not overdo, avoid distractions, and return to a daily routine gradually. If symptoms continue for more than a week, the patient should consider consulta-

tion with a brain injury physician who is experienced with postconcussive patients.

Given the variety of postconcussive symptoms that may exist following mild TBI, it is unlikely that a true postconcussive syndrome exists. Given the potential complexity of such injuries, it is not uncommon for one set of symptoms to go totally undiagnosed or be misdiagnosed. The astute clinician will assess all the aforementioned potential diagnostic factors before developing an integrated holistic treatment plan. Some of the more frequently reported postconcussive symptoms include headache, dizziness, memory problems, weakness and fatigue, nausea, and tinnitus. Based on sound research and the experience of innumerable clinicians, most individuals who incur mild TBI do not have long-term sequelae. A definite subpopulation of patients with mild TBI, however poorly defined, do have persistent, and sometimes, disabling long term somatic and neuropsychological sequelae. It is critical to take into consideration both organic and nonorganic factors that might give cause to protracted periods of disability after otherwise innocuous insults to the brain.

Accessibilty to advanced neurodiagnostic facilities and to subspecialists, such as neurootologists, neuroophthalmologists, and neuropsychiatrists, may be critical in patient diagnosis, treatment, and medicolegal settlement. Preferably, the primary physician should have subspecialty knowledge and sufficient clinical experience in dealing with this patient population to truly optimize patient care. Ideally, a continuum of clinical services, both neuromedical and rehabilitative, should be available for this patient population, including full physical examinations (including a thorough cognitive-behavioral assessment), neurodiagnostic workups as indicated, and functional assessment. Sophisticated clinicians can assist the primary physician with further diagnoses (Table 6-2) and treatment interventions aimed at maximizing functional status and community reentry. Additionally, such tests can provide additional corroboration for relevant medicolegal purposes. Appropriate use of pharmacologic interventions can also contribute significantly in the treatment of this patient population (Table 6-3). Generally, the practice is to not make final neuromedical or functional prognoses

TABLE 6-2

NEURODIAGNOSTIC EVALUATION OF POSTCONCUSSIONAL DISORDERS

Posttraumatic Sequela	Neurodiagnostic Procedure
Vestibular dysfunction—peripheral and central	Electronystagmography with calorics
Eye movement disorders	Electrooculography
Olfactory and gustatory deficits	Chemosensory evaluation
Perilymphatic fistula	Posturography ?
Sensorineural and conductive hearing loss	Audiologic evaluation
Neuralgic scalp pain	Diagnostic block with lidocaine
Structural parenchymal abnormalities	Computed tomography and/or magnetic resonance imaging
Cerebral blood perfusion abnormalities	Single photon emission computed tomography*
Cerebral metabolic abnormalities	Positron emission tomography*
Electroencephalographic abnormalities	Sleep deprived electroencephalogram or variant thereof, BEAM (brain electrical activity mapping)* ?
Attentional deficits	Cognitive evoked potential (P-300)* ?
Balance dysfunction	Posturographic evaluation
Sleep disturbance	Polysomnography
Erectile dysfunction	Nocturnal penile tumescence (NPT) monitoring
Comprehension deficits	Central auditory processing

*Still in research phase
?Questionable efficacy

TABLE 6-3

PHARMACOLOGIC INTERVENTIONS FOR COMMON POSTTRAUMATIC SEQUELAE

COMMON POSTTRAUMATIC SEQUELA	PHARMACOLOGIC INTERVENTION
Anxiety	Serotonergic agonist Buspirone, sertraline, trazodone, and fluoxetine ?
Basilar artery migraine (BAM)	Antimigraine regimens Psychotropic anticonvulsants
Depression	Tricyclic antidepressants (TCAs) Newer generation serotonergics Monoamine oxidase inhibitors (MAOIs) Lithium carbonate Carbamazepine
Emotional lability and/or irritability	Serotonergic agonists Psychotropic anticonvulsants TCAs Lithium carbonate
Libidinal alteration	
Decreased	Noradrenergic agonists, hormone replacement if low to borderline low
Increased	Serotonergic agonist, hormonal treatment—cyproterone or medroxy-progesterone acetate
Myofascial pain/dysfunction	Nonsteroidal antiinflammatory agents (NSAIDs) TCAs and other antidepressant type medications Mild muscle relaxants
Neuralgic pain	Capsaicin TCAs and related compounds Carbamazepine and other anticonvulsants NSAIDs Local anesthetic blockade
Posttraumatic stress disorder	Antidepressant medications Psychotropic anticonvulsants Propranolol Clonidine MAOIs Lithium Benzodiazepines
Sleep initiation problems	Serotonergic agonists—trazodone
Sleep maintenance problems	Catecholaminergic agonists—nortriptyline
Tinnitus	Gingko biloba ? Tocainide ?
Vascular headache	Antimigraine regimens used in the following protocols Symptomatic Abortive Prophylactic
Fatigue	Catecholaminergic agonists Methylphenidate Caffeine Amantadine
Cognitive dysfunction	Nootropes Catecholaminergic agonists Cholinergic agonists and/or precursors Neuropeptides ? Vasoactive agents ?

?: Questionable efficacy

until at least 18 to 24 months postinjury, due to the anticipated neurologic recovery curve. During the recovery period from postconcussive disorders, judgments should be made by the treating physician and rehabilitation team to determine if the soldier should be placed in a supervised setting or one that does not require full work capacity skills. Work reentry should occur in a structured, monitored fashion to minimize risks of frustration and failure, and to optimize long-term reentry success (see the section on Vocational Reentry for further discussion).

Healthcare professionals should remain aware that secondary gain, malingering, chronic pain, and preinjury psychologic and psychiatric disorders may have an impact on subjective symptoms or the recovery course. If there is opportunity for financial or other conscious secondary gains, the issue of malingering should always be kept in mind.[180] The majority of symptomatic patients, however, are *not* malingerers and do have some sort of legitimate injury, although it may not necessarily relate to TBI. Prior to labeling anyone with a diagnosis that may have a negative impact on functional status and community reintegration, careful scrutiny must be made of all cases to determine any contributing factors, conscious or otherwise, as well as the methodical neuromedical procedures.

Early identification of patients with mild TBI is critical in assuring timely and adequate identification of treatable sequelae. The evaluation and treatment of this patient population must be holistic in nature, ideally utilizing an interdisciplinary rehabilitative model and, as appropriate, a multidisciplinary neuromedical model. Rehabilitative efforts should focus on neuropsychological assessment; physical therapy for vestibular habituation, balance retraining, and myofascial dysfunction; occupational therapy for provision of compensatory strategies for functional cognitive deficits; and speech language pathology for cognitive-linguistic and pragmatic deficits. Appropriate neuromedical diagnosis and treatment, reassurance, education, support, counseling, and regular monitoring are also essential components that will optimize the expediency and quality of overall functional recovery.[170,178]

Low Level Neurologic States: Terminology

One of the most confusing issues for families as well as many professionals is the language used to describe the condition of an individual after TBI. The word *coma* simply conveys that the patient is neither alert nor aware. The comatose patient remains unconscious and "asleep." Typically, there is absence of vegetative functions (such as sleep–wake cycles) during coma. From a neurologic standpoint, coma can be fleeting or prolonged, but generally lasts no longer than 3 to 4 weeks.[181] Most patients with very short durations of coma (ie, seconds) will generally not suffer any significant degree of long-term disability or impairment; however, a small percentage do have temporary and sometimes even permanent problems. Generally, the longer the period of coma the more extensive the associated brain damage. Typically, longer periods of coma are correlated with more extensive diffuse axonal injury. Once comatose, patients will take one of three possible courses: (1) they will regain some level of consciousness; (2) they will die; or (3) they will emerge into a vegetative state. More than 50% of individuals with severe brain injury will die, regardless of the quality of care rendered during the acute period. The majority of those who survive will eventually emerge to some level of consciousness. Patients who emerge from coma into vegetative state may remain vegetative for a very short period of time or may remain in this state permanently, the so-called "permanent vegetative state."[2]

The term *vegetative state* is a confusing one and commonly misunderstood. It is not meant to imply that the person has become a "vegetable," instead it refers to neurologic changes, such as a return of sleep–wake cycles and progression from a state of nonarousal to arousal. Very few patients with severe brain injury (approximately 1%–3%) will remain permanently in a vegetative state.[182] Permanent vegetative state is a prognostic term, not a diagnostic one, and should, therefore, only be used when it is quite clear that the patient will permanently remain in this state.[183] The length of time someone has been in a vegetative state should always be specifically qualified. When there has been significant hypoxic brain injury (a secondary form of brain injury), the prognosis may be more clear cut earlier on in the patient's course, but even in these cases the permanent vegetative state label should not be given until at least 3 months postinjury.[182,183]

Recent guideline development has resulted in further evolution of nomenclature and practice guidelines for management of persons with severe alterations in consciousness. One example of this has been the recommendation to dispense with the use of the phrase "persistent vegetative state" because it adds nothing to the diagnostic accuracy of the vegetative state. Additionally, the assignation, at 1 month postinjury, of the term "persistent" is totally arbitrary. The word "permanent" should be

assigned only after 12 months have passed in a vegetative state following trauma or 3 months following hypoxic ischemic brain injury. It must be realized nonetheless, that even in such cases, the statistical odds of emergence from vegetative state do not reach 100%, although they certainly are nearing this level. The Aspen Workgroup, which has met over the last few years, has also identified a further subgroup of patients who are at the very impaired end of the severe disability category, and although not vegetative, demonstrate intermittent signs of awareness. The suggested terminology for describing this previously not–well-studied or identified subgroup of patients is the "minimally conscious state."[182]

Social Concerns

Significant ethical and legal issues surround the care and potential withdrawal of care from patients in the persistent vegetative state.[184,185] Decisions regarding withdrawal of medical support, whether artificial breathing machines, nutrition, or medications, should probably not be made until at least 2 years postinjury. In actuality, if someone is truly vegetative at 1 year postinjury, that patient is *extremely* unlikely to regain consciousness, although such cases have been reported. Clinicians who deal with this patient population must familiarize themselves with the position papers that have been published on this topic.[182,183,186,187] Clinical care and ethical issues are distinctly different for individuals with profound and irreversible paralysis who have retained consciousness and cognition, such as in locked-in syndrome. Ethically, legally, and medically if these patients are medically judged to have the capacity to make such a decision, they have the inalienable right to forego life-sustaining therapy.[187]

Stimulation Programs

In this era of high technology and aggressive medical care, coma stimulation programs seem to have taken a foothold as an integral part of most continuums of rehabilitative care for patients with severe brain injury, regardless of the specific etiology. It is disconcerting, therefore, to learn that the content of coma situation programs is quite variable. Additionally, little, if any, methodologically sound literature supports the efficacy of such programs in terms of altering the course of neurologic recovery either with regard to the maximization of final neurologic outcome or an increased rate of recovery.[2]

There are major issues regarding how coma stimulation may have a beneficial effect on a brain that is "unaware" of its environment. Pathologic[181] studies of patients who have died while in a coma or vegetative state show that there is no consistent pathology associated with these conditions. In the early descriptions of the persistent vegetative state, it was theorized that significant brain stem involvement was a requisite neuropathologic finding; however, subsequent studies[188] have shown a variety of neuropathologic findings that vary from extremely diffuse gliosis to relatively grossly normal appearing brains. More importantly, experimental data on cerebral glucose metabolism in patients in the vegetative state might lead to questions of how, if at all, any external stimulation can effect any positive change in this patient population.[189,190] Care should be taken when applying these results to the TBI population at large because most of the patients studied had suffered significant hypoxic brain injury; however, data still *suggest* that patients who are truly vegetative from a neurobehavioral standpoint do *not* have the dynamic physiologic cerebral function to support *any* type of cognitive processes.

A most important issue to address in assessing the efficacy of coma stimulation is the standardization of terminology across all disciplines. The lack of a common neurologic and neurobehavioral terminology, such as coma, vegetative state, and persistent vegetative state, is a major obstacle presently facing all professionals who treat individuals with severe brain injury. Additionally, there is a great need for standardization of assessment tools for low-level patients, whether they are comatose, vegetative, or severely disabled. At the present time, the ongoing development of assessment tools by many institutions does not appear to be a constructive process because of the nonuniformity of data that are subsequently generated, and the inability to compare treatment design and efficacy data across centers.

Most coma stimulation is not *coma* stimulation at all, but rather vegetative or low-level stimulation. Therefore, it is probably appropriate, both from a standpoint of proper terminology and, ultimately, from a standpoint of reimbursement, to dispense with the phrase *coma stimulation*. Additionally, labeling a program as offering coma stimulation suggests that this is the sole or major component of such a program; such an implication is a disservice to the third party payers, the families, and most important, the patients. Appropriate management of the low-level patient, whether comatose or vegetative, should be comprehensive in nature with sen-

sory stimulation (rather than coma stimulation) merely being *one* component of such a program.

It is important to differentiate between environmental and structured sensory stimulation from both clinical and research perspectives. Environmental stimulation simply implies that the patient is subjected to ongoing environmental stimuli of various types, including sights, sounds, textures, and so forth. Due to the fact that these are environmental stimuli, they are not presented in any type of structured or systematic fashion, such as music in the room, a picture mobile, or the smells of food or perfume on a loved one. Structured sensory stimulation implies that stimuli are presented in a labor intensive, systematic fashion, whereby the patient is subjected to multisensory stimulation, which typically includes tactile, visual, vestibular, auditory, olfactory, and gustatory sensations. It may be surprising to some proponents of environmental and sensory stimulation programs that there are presently no well-controlled, prospective, statistically significant, population based studies to indicate that such programs actually alter the recovery course or final neurologic outcome of patients who are comatose or vegetative. Most of the studies that are used by proponents of stimulation programs are flawed by multiple factors, including small sample size, retrospective nature, lack of control groups, lack of correlation to functional status and recovery, poor descriptions of possible neuromedical factors (ie, hypoxic ischemic brain injury), lack of scientific peer review, or inadequate information regarding control group selection criteria, or all of the above.[2,191]

There are two recent experimental studies[192,193] that are much more methodologically sound, although they still lack a significant sample population size or adequate control groups, or both. Neither of these studies showed any significant beneficial effect from sensory stimulation. It should be noted that there are animal studies, particularly rodent models, that do indicate a beneficial effect of enriched environments relative to sensory deprivation on recovery processes after experimentally induced brain injury. However, none of the animal protocols utilized vegetative or comatose subjects. Additionally, the relationship of lower animal nervous system recovery to human recovery in persistent vegetative state requires a quantum leap in phylogenic physiologic assumptions.[194]

Given what is presently known about individuals in coma and vegetative states, from both a neuromedical and rehabilitative standpoint, it would seem that present knowledge and literature, when taken as a whole, do not strongly support the utility of structured sensory stimulation as a means of altering the course of neurologic recovery after severe brain injury. Nonetheless, various professionals have *theorized* that sensory stimulation, although not empirically validated, might help structure the patient's interaction with the environment, monitor the patient, and lastly, increase the input into the reticular activating system.[195]

Program descriptions, such as coma stimulation and sensory stimulation, should be abolished and more appropriate phraseology such as early recovery management programs (ERMPs) or "medically complex care." Appropriate interdisciplinary management of this patient population involves the issues of potential morbidity prevention and provides appropriate neuromedical and rehabilitative interventions to maximize neurologic and functional outcome. An ERMP program should focus on several main goals, including maintenance of range of motion, prevention of complications associated with immobility, prescription of appropriate orthotics for preventive and corrective positioning, prescription of seating systems for transport and mobilization, and treatment of neuromedical conditions that could potentially compromise ongoing neurologic recovery and are germane to low level neurologic states. For those who wish to read further on this topic, the management of this patient population has been delineated in several publications.[194–196]

Coma stimulation programs, per se, should not be the sole means by which patients undergo frequent and close reevaluation. A rhetorical argument could be that the rationale of coma stimulation, (ie, stimulating patients to "wake up" faster than they would without stimulatory intervention) may actually be detrimental to the organism. Specifically, what if coma and the vegetative state are normal evolutionary responses to severe brain injury? Would not the more severely injured brain need more time to recover than the less severely injured one? In this sense, trying to rush the process might actually be detrimental to the long-term viability and recovery of the injured organism. Given that there is presently little or no solid evidence that structured sensory stimulation is harmful, there is still great controversy and debate over whether it is even efficacious, or if this aspect of clinical service should be maintained in any ERMP. Theories aside, the current evidence strongly suggests that rehabilitation professionals need to more closely scrutinize the role of any stimulation program in the greater context of the rehabilitative care of these patients. If sensory stimulation is offered, it should

be done in a cost efficient, ethical, and responsible fashion and not as the major component of the total program. For those patients who remain vegetative beyond 1 year and become permanently vegetative, it is best to take a step back and ask the question, "when is enough, enough?" Based on present knowledge about recovery from prolonged vegetative state, it would seem reasonable to withdraw *intensive* rehabilitation efforts, including coma stimulation, after 1 year. A full neuromedical workup must be performed prior to labeling any patient as persistently vegetative. All patients should receive ongoing monitoring for *any* change in neurologic or functional status before *and* after the 1 year mark. If and when changes occur, decisions should be made regarding whether or not to proceed with more intensive rehabilitation efforts.

The controversies surrounding coma stimulation will continue until more definitive studies are available to answer the questions at hand. Until that time, rehabilitation professionals must strive to maintain better uniformity with regards to nomenclature, treatment strategies, and assessment tools. By doing so, clinicians will be better able to assess the efficacy of their interventions, and will be able to provide families with much needed information, which will ultimately lead to a higher quality of patient care, both ethically and qualitatively.

NEUROBEHAVIORAL OUTCOME, ASSESSMENT, AND INTERVENTION

Following TBI, individuals of all injury severity levels are often confronted with a range of neurobehavioral problems. Cognitive deficits, such as impaired memory and concentration abilities, are commonly noted. Interpersonal difficulties, depression, and diminished self-awareness are also typical after TBI. Comprehensive assessment of neurobehavioral functioning is essential to develop effective rehabilitation plans for military personnel after brain injury. The following section outlines a protocol for conducting neurobehavioral evaluations on military personnel with brain injury. An overview of the evaluation process addresses the development of referral questions, interviewing strategies, test selection and administration, and development of treatment recommendations. These guidelines are preceded by a review of the neurobehavioral outcome literature. Research on cognitive outcome is presented first. Next, an overview of psychosocial sequelae highlights emotional, behavioral, and substance abuse problems after TBI. Finally, a rationale for utilizing a neurobehavioral methodology is presented as a preface to the evaluation protocol and a section on intervention.

Cognitive Outcome Literature

An undisputed consequence of TBI is its effects on cognitive functioning. Residual deficits in memory, processing speed, and other cognitive factors often compromise a patient's ability to resume preinjury activities, including work and self-care.[197,198] Unfortunately, research that delineates the recovery of cognitive functioning after TBI is scarce.[199] Of the existing investigations, few have reported on recovery beyond the first year postinjury and even fewer have explored the course of recovery following an open head injury (eg, penetrating missile wound).

Most of the existing research on recovery comes from Glasgow, Scotland, where Jennett and colleagues studied cognitive outcomes after severe brain injuries.[200,201] The Glasgow researchers found that recovery occurs with, and is characterized by, rapid early improvement, which slows down and levels off in most patients by 6 months. Notably, these studies relied on a global outcome measure, the Glasgow Outcome Scale (GOS), to assess change over time. The GOS is an ordinal scale that assigns patients to one of five broad outcome categories: (1) dead, (2) persistent vegetative state, (3) severe disability, (4) moderate disability, and (5) good outcome.[202] Because the GOS is a global measure, it may not be sensitive to the more subtle cognitive changes beyond the first 6 months.

More recent studies[203] challenge the assumption that cognitive recovery halts at 6 months postinjury. Using a matched control design, Dikmen and colleagues administered an expanded version of the Halstead-Reitan Neuropsychological Test Battery to moderately and severely injured patients. Patients and control subjects were tested longitudinally at 1, 12, and 24 months postinjury. The investigators concluded that significant improvements occurred beyond the first year. However, recovery in the second year tended to be more specific, depending on the severity of the injury and type of function. In contrast, the first year of recovery was marked by improvements in all functional areas. Dikmen's group also noted that despite signs of recovery, "marked impairment" across a broad spectrum of neuropsychological functions was still present at 2 years postinjury.

An earlier investigation by this same research group[199] proposed a deficit-proportional model to account for differences in recovery curves. Using this statistical model, they concluded that the amount of cognitive recovery is proportional to the initial severity of the deficit. In general, brain injured patients are expected to recover a portion of their initial losses over time. Accordingly, individuals with substantial losses (eg, moderate to severe TBI) show greater amounts of improvement, but also have more residual deficits. In contrast, those with less initial impairment (eg, mild to moderate TBI) show smaller amounts of improvement and fewer residual deficits. According to these authors, the deficit-proportional model affords better prediction of cognitive outcome than do other models that assume a constant change over time, regardless of injury severity. In turn, the deficit-proportional model highlights the predictive power of initial severity indexes, such as duration of coma, in forecasting cognitive recovery.

Psychosocial Outcome Literature

Emotional and Behavioral Sequelae

Unfortunately, the effects of TBI are not typically confined to cognitive functioning.[204] Many patients, including those with minor injuries, undergo undesirable emotional and behavioral changes.[205,206] These changes may persist for many years after the injury and include such problems as increased anger, frustration, and depression.[204,207] Underscoring the significance of these problems is the testimony of relatives who report that personality and behavioral changes cause more problems than cognitive and physical deficits.[205,207,208] Moreover, research suggests that emotional problems often impede a brain injured person's ability to return to work.[97,209] The path of emotional and behavioral sequelae following TBI has been charted through a series of outcome studies typically focused on severe closed head injury.[204,205,207,208,210] One of the earliest investigations took place in Denmark, where Thomsen followed a group of 50 severely injured patients and their families for up to 5 years postinjury.[211] Patients were examined by a neurologist, a neuropsychologist, and a speech pathologist to assess physical and cognitive functioning. Relatives were interviewed in regards to a number of areas, including the patient's emotional and psychological status. During their interviews, 84% of the relatives complained of changes in the patient's character, including irritability, hot temper, loss of spontaneity, emotional lability, and emotional regression. Reportedly, these changes in personality created the greatest problems in daily living. In contrast, relatives reported that motor dysfunction and other physical problems were not problematic.

Ten years after the initial investigation, Thomsen published a follow-up study[207] on 40 patients from the original sample. The follow-up included an expanded version of the original relatives' questionnaire, which was administered up to 15 years after the patient's injury. Thomsen found that the majority of patients remained unchanged since her initial investigation 10 years prior; moreover, the relatives again reported that the most salient changes were psychosocial in nature. Summarizing her findings, Thomsen[207] wrote that "though physical impairment, dysarthria, and defects of memory remained severe in many cases, the psychosocial sequelae presented the most serious problems." Thomsen further noted that two thirds of the patients in her study underwent "permanent changes in personality and emotion." Unfortunately, most patients lacked awareness of the changes in their character, making it difficult for them to modify their behavior.

Thomsen's conclusions are corroborated by a number of other studies on psychosocial outcome after TBI.[204,205,208,210,212] In an early outcome study from Scotland, McKinlay and colleagues[212] assessed a group of severely brain injured patients at 3, 6, and 12 months postinjury. Using relatives' reports, the researchers charted the course of emotional, cognitive, and physical outcome during the first year after injury. At 3 months, the most commonly endorsed problem was related to cognitive functioning; specifically, 86% of relatives stated that the patient had processing speed deficits. In comparison, emotional problems, such as mood changes and social withdrawal, were infrequently endorsed. At 6 months, slow processing speed was again the most commonly endorsed problem (69%), followed by "bad temper" (56%). At 1 year postinjury, the most frequently endorsed problems were bad temper and cognitive slowness, both at 67%. In summary, problems related to cognitive and physical functioning tended to decrease, whereas emotional and behavioral problems tended to become more frequent during the first year after injury.

This same group of Scottish researchers continued to explore the course of psychosocial recovery from severe TBI in a series of outcome studies. In 1987, they reported on 134 patients who were within 7 years postinjury.[205] Patients and some close relative were questioned about an assortment of areas,

including physical, communication, emotional, behavioral, and cognitive problems. Patients were reported to have altered personalities, evidenced by increased irritability, anger, depression, and mood swings. A disturbing number of patients threatened violence (47%) or acted violently (26%) toward a relative. Other commonly reported problems included disturbances in memory and concentration. Patients and relatives agreed that physical complications caused relatively few problems.

In a comparable study,[204] a group of British researchers reported on psychosocial adjustment 7 years after severe traumatic brain injury. Their findings were remarkably consistent with those from the Danish and Scottish investigations. Relatives reported that behavioral, personality, and memory difficulties interfered the most with long-term adjustment after brain injury. Oddy's[204] group also found a pervasive pattern of social isolation among patients and their families. Only half of those surveyed had even "limited contact" with friends. Other studies[208,210] confirm that social isolation is a major barrier following TBI. Socialization difficulties are often attributed to negative changes in the patient's personality.[208]

A clear pattern emerges from these international outcome studies. First, research indicates that the course of recovery and adjustment following brain injury varies over time. In the period immediately following the injury, the patient's physical and cognitive status are of primary concern. Just as these problems begin to subside after the first year, personality and behavioral problems become more noticeable. Many patients reportedly exhibit aggressiveness, increased irritability, mood swings, and depression. Unfortunately, lack of awareness about their personality changes contributes to interpersonal difficulties, and ultimately, social isolation.

The issue of emotional adjustment following mild TBI has thus far been understudied. However, preliminary research[206] suggests that persons with mild brain injury report more emotional problems following their injury than do severely injured persons. Leininger and colleagues[206] administered the Minnesota Multiphasic Personality Inventory[213] (MMPI) as a measure of emotional and personality function to groups of mildly and severely injured outpatients. The authors found that the emotional profile of individuals with mild TBI was indicative of greater subjective distress than was the profile of severely injured persons. Specifically, individuals with mild TBI showed significantly higher elevations on clinical numbers scale 1, 2, 3, 7, and 8. Individuals with similar profiles were highly distressed and reported feelings of depression, social isolation, and somatic concerns (Table 6-4). Because these results were based on responses within the first year postinjury, more research is needed to delineate the long term emotional sequelae of persons with mild TBI.

TABLE 6-4

EMOTIONAL PROFILES OF PERSONS WITH MILD VS SEVERE TRAUMATIC BRAIN INJURY

Level of severity	Symptoms
Mild	Somatic concerns, clinical depression, anxiety, feelings of social inadequacy, interpersonal alienation, difficulty concentrating and/or confusion
Severe	Clinical depression, interpersonal alienation, difficulty concentrating, confusion

Substance Abuse

The widespread use and abuse of alcohol in American society is a well documented phenomenon. Analysis of national survey data indicates that more than two thirds of all Americans have at least an occasional drink.[214] Highest drinking rates are seen among young adult males, 19% of whom meet the criteria for classification as heavy drinkers. The rate of alcohol abuse among Americans is staggering. A report[215] published by the National Academy of Sciences revealed that 10 to 13 million American adults either abuse or are dependent on alcohol. Other investigations[216,217] have concluded that as many as 12% of American drinkers consume excessive and potentially lethal amounts of alcohol. While these statistics reflect the population at large, it is reasonable to assume that problems with alcohol abuse generalize to a significant portion of military personnel.

From the clinician's point of view, the many issues regarding the use and misuse of alcohol by individuals after TBI tend to be extremely problematic. Some of the alcohol-related issues that professionals must deal with include fears about potential lowering of seizure threshold, alcohol induced cognitive and behavioral changes, neuro-physical side effects, possible suppression of concomitant

neural recovery processes, and interactions with various medications. Probably the most important, yet not necessarily most obvious, factor that can be levied against alcohol consumption after TBI is the greater potential for recurrent brain injury. Of additional interest, although not well studied, is that after significant brain injury a person's CNS becomes more sensitive to the effects of ethanol. Many of these issues are poorly understood by most professionals dealing with this patient population, yet there are reasonable solutions, some possibly surprising, to most of these concerns.

Many clinicians advise their TBI patients not to drink even though they have no real rationale for doing so. It is known that ethanol has multiple effects on CNS neurotransmitter systems, the strongest of which appears to be GABAergic mediated. Recent literature[218] suggests that GABAergic agents may actually impede neurologic recovery following brain injury; this thereby, provides at least one piece of evidence in support of this practice. More research is obviously needed to clarify the potential detriment caused by ethanol during the early neural recovery processes.

Probably one of the most controversial alcohol-related issues facing clinicians is the concern about the lowering of the seizure threshold. It is commonly perceived that individuals with documented seizure disorders will experience problems with seizure control if they use alcohol; however, this is not confirmed by the few experimental studies that have tested this hypothesis. The rare situations where alcohol can act as a convulsant drug, and the mechanisms whereby long term neurotoxic effects of alcohol may lead to chronic epilepsy, have been poorly studied and need further clarification.[219]

The effects of acute alcohol intoxication on the CNS can have a significantly more profound effect on someone who already has preexisting neurologic dysfunction secondary to TBI. Although no neuropathologic changes, per se, have been associated with acute intoxication, alterations in the neuronal membranes, because of the incorporation of alcohol and central neurotransmitter aberrations, have been clearly demonstrated. The reason why individuals with TBI are seemingly more susceptible to the effects of alcohol may be due to posttraumatic alterations in CNS membrane permeability, neurochemical alterations, or some other, unidentified factor.[219]

Acute alcohol intoxication may worsen already existing postinjury behavioral sequelae, including akathisia, aggressiveness, irritability, and disinhibition. From a cognitive perspective, a variety of posttraumatic neuropsychological problems may be further compromised, including staying on task, mental processing speed and flexibility, learning, problem solving, attention, concentration, judgment, and reasoning. From a neurophysical standpoint, acute intoxication adversely affects all motor behaviors, from the simplest to the most complex, including ambulation, speech, eye movements, and postural control. Many TBI patients, who do not drink any alcohol at all can relate at least one incident where they have been stopped by police for suspicion of alcohol intoxication.[219]

Chronic effects of alcohol on the CNS have been well documented. Given the neuropathology associated with TBI (specifically, primary injury due to focal cortical contusion with greatest propensity for frontal and temporal parenchyma, or diffuse axonal injury, or both, as well as frequent cases with some degree of secondary brain injury), it would seem unwise for anyone with a brain injury to consume alcohol on a chronic basis. This word of caution carries more weight given the occurrence, in alcoholics, of significant neuropathologic changes with chronic alcohol consumption, including brain shrinkage, loss of frontal cerebral cortical neurons, and cerebellar degeneration.[219]

Alcohol ingestion may actually be contraindicated in conjunction with various medications, including antidepressants, anxiolytics, benzodiazepines, neuroleptics, anticonvulsants, lithium carbonate, and many others. Induction of hepatic enzymes with chronic alcohol ingestion can alter drug levels of agents hepatically metabolized. Acute alcohol intoxication may cause serum levels of certain drugs to rise secondary to competition for binding sites, and due to its diuretic effect, alcohol can potentially cause lithium toxicity. The added sedative effects of alcohol with many pharmacologic agents must also be taken into consideration because it can adversely affect safety in driving, operation of equipment, and other activities.

Lastly, it is well documented that much of the morbidity and mortality associated with TBI is linked to alcohol consumption. Given this fact, it becomes obvious that the individual who drinks after sustaining a TBI is at higher risk for recurrent injury than one who does not. Accidents with alcohol are typically related to vehicular mishaps, falls, or fights. The aforementioned facts should be sufficient justification for more conservative recommendations, rather than more liberal ones, regarding alcohol use following traumatic brain injury.[219]

Several published investigations[218,220] have revealed troublesome data regarding the relationship

between brain injury and alcohol use. The investigators examined admission blood alcohol levels among persons with TBI and found that many TBI patients had been drinking at the time of injury.[218,220] Galbraith and colleagues[221] completed a prospective study on 918 consecutive civilian brain injury admissions and found that 62% of male and 27% of female patients had positive blood alcohol levels on admission. Rimel and colleagues[218] investigated blood alcohol levels using 199 moderately injured patients (GCS = 9–12) and 538 patients with minor brain injury (GCS = 13–15) from the University of Virginia Hospitals. In screening blood alcohol levels, positive findings emerged for 78% of moderately injured patients and 53% of patients with minor injuries. Of the moderately injured group, 57% had blood alcohol levels greater than or equal to the 100 mg/dL, the maximum legal limit for driving in Virginia and many other states. Furthermore, 28% of those tested had blood alcohol levels in excess of twice the legal limit. Among minor-injury patients who tested positive, 43% exceeded the legal limit for intoxication while 23% had blood alcohol levels equal to or greater than twice the legal limit. These figures, in part, reflect the fact that more than a third of moderately injured and 10% of minor brain injury patients reportedly abused alcohol prior to their injury.

While alcohol clearly contributes to the incidence of traumatic injuries, its use also complicates the rehabilitation process. The use of alcohol can increase the expense of rehabilitation by interfering with physical recovery.[220] This situation is further complicated when alcohol is combined with prescription medications, causing adverse interactions.[222] The likelihood of alcohol interfering with the rehabilitation process is compounded when patients use intoxication as a coping mechanism. Research suggests that patients who suffer physical disabilities related to brain injury are likely to consume alcohol in their effort to subdue emotional distress.[223,224] In spite of these concerns, few investigations have examined patterns of preinjury and postinjury alcohol use among head injured persons.

The potential of alcohol to impede the rehabilitation of head injured patients is alarming and indisputable. Unfortunately, there is a dearth of information regarding alcohol use and disability, and policies for substance abuse among this population are simply absent. Rohe and DePomolo[225] surveyed 52 rehabilitation medicine departments, only a fraction of which screened patients for substance abuse as routine policy and, furthermore, staff members were found to be generally ambivalent regarding the issue. For example, the majority of respondents (90%) supported prohibition of alcohol consumption for inpatients; however, half the sample felt that alcohol should be prescribed to outpatients for desirable reasons, including appetite stimulation and relaxation. The survey further revealed that patient and staff education programs concerning substance abuse were notably deficient. While over half of the programs expressed support for substance abuse education, only 29% actually provided such programs to patients and even fewer were available to the clinical staff. In reviewing these findings, Rohe and DePompolo concluded that the issue of substance abuse has been virtually ignored or unrecognized by rehabilitation professionals in the brain injury field.

Theoretical Rationale for Neurobehavioral Assessment

Historically, researchers have incorporated two assessment methodologies to describe the neurobehavioral consequences of TBI: (1) self-report measures of emotional and behavioral functioning, and (2) neuropsychological assessment. Researchers[218,226] have used questionnaires and interviews to elicit family members' and patients' perceptions regarding the behavioral and emotional effects of injury. Partly because of concerns related to diminished self-awareness arising from injury, most investigators have relied primarily on family members' rather than patients' reports. More recently, rehabilitation professionals have begun to use traditional psychodiagnostic measures, such as the MMPI, to assess the patient's personality functioning.[227]

Neuropsychological assessment procedures have also been used extensively to describe patients' diverse cognitive, intellectual, sensory, motor, linguistic, and perceptual skills.[228] Neuropsychological testing serves a variety of functions and offers an objective description of diverse abilities. Postinjury performance on neuropsychological measures may be compared with estimates of preinjury ability to help determine the consequences of the injury. Comparisons can also be made with normative data available for uninjured persons. Additionally, follow-up evaluations related to recovery, aging, pharmacologic intervention, and cognitive rehabilitation, can help delineate changes over time.[229]

Each assessment methodology is subject to bias and uncertain validity. Psychological denial may cause relatives and patients to minimize difficulties on self-report measures. Patient perceptions of disability may be affected by memory deficits and

impairment of self-awareness that arises from pathophysiological changes. Psychological distress may contribute to exaggerated reports by relatives. Neuropsychological tests are clearly more objective than self-report measures and they also avoid the issue of biased reporting. Unfortunately, several factors contribute to uncertainties regarding the value of neuropsychological assessment. A clear relationship between neuropsychological test scores, ability to perform daily living activities, and patients' and relatives' perceptions of sequelae has not been demonstrated.[230] Furthermore, neuropsychological assessment procedures represent an unusual and highly structured situation. As such, test results may not provide a good representation of characteristic behavior evident in daily living activities.

Efforts to enhance the validity of conclusions regarding neurobehavioral outcome and the complexity of interrelationships among outcome variables have contributed to an increased reliance on a combination of assessment methodologies. Neurobehavioral assessment offers this comprehensive methodology by combining neuropsychological assessment techniques with measures of behavioral and emotional functioning. Consolidation of these methodologies assures that a comprehensive picture of the patient's emotional, behavioral, and cognitive problems is presented.

A Protocol for Neurobehavioral Assessment

This section presents an overview of issues pertaining to neurobehavioral assessment of military personnel following a TBI. The suggested protocol highlights four key elements of the assessment process: (1) developing the referral question, (2) interview and behavioral observations, (3) test selection and administration, and (4) interpretation and reporting. While the information presented herein is intended to benefit any interested reader, administering and interpreting the evaluation requires the supervision of a well-trained professional, typically a clinical neuropsychologist.

Developing the Referral Question

Military personnel with probable TBI will typically be referred for neurobehavioral evaluation by their physicians. Primary objectives of the evaluation include description of the patient's neuropsychological strengths and deficits (eg, intellectual, cognitive, sensory, and psychomotor skills), description of the patient's emotional and behavioral status, and development of a treatment plan. In addition to these broad objectives, explicit referral questions can enhance the value of the examination by guiding the assessment process. Consultation with referral sources is essential to ensure that the lengthy examination process meets the unique needs of each patient and answers the questions that the treating professionals deem most important. Typical referral questions among military personnel will include those that assess the extent of the patient's disability and the probability that he may return to active duty. Examples of other referral questions are presented in Exhibit 6-2.

Interview and Behavior Observations

An initial interview with the patient provides useful data regarding any neurobehavioral problems. The patient's reactions to the injury are of particular concern because depression and other forms of psychological distress often accompany TBI.[231] Clinicians should assess the patient's perception of emotional changes, present coping ability, and level of pessimism. Patients should also be screened for the presence of depressive symptoms, including suicidal ideation or intent. Symptoms of depression after TBI include

- difficulty enjoying activities;
- feelings of worthlessness;
- feelings of hopelessness;
- lack of initiative;
- lack of self-confidence;
- feelings of sadness, being "blue";
- diminished appetite;
- changes in sleep patterns;
- changes in weight; and
- suicidal ideation or intent.

A medical examination can help distinguish between pathophysiological changes that have arisen from injury, and psychological changes. An interview with family members provides another important perspective on the patient's emotional status.

During the initial interview and throughout the evaluation, qualitative information may be gained by observing the patient's behaviors and reactions to the testing process. Examiners may, for example, observe whether the patient is able to recognize errors and, furthermore, if he demonstrates initiative to correct these errors. If present, socially inappropriate behaviors should be noted in the report because such behaviors can negatively impact the

EXHIBIT 6-2

SAMPLE REFERRAL QUESTIONS FOR A NEUROBEHAVIORAL EVALUATION OF MILITARY PERSONNEL

- Describe the patient's cognitive, intellectual, and psychomotor abilities.
  Which areas would you consider relative strengths and which areas would you consider to be weaknesses?
- To what extent are the patient's deficits attributable to his injury?
  What preinjury factors (eg, premorbid educational history, prior brain injury, substance abuse) might be contributing to the patient's impairment?
- What is the patient's present emotional status?
  Is there evidence of depression, feelings of hopelessness, or diminished self-esteem?
  Is the patient a suicide risk?
- Will the patient be able to return to his previous position in the military?
  If so, when might a return to active duty be possible?
  If not, are there other jobs within the military that would be better suited to this individual's abilities?
- Does the patient's injury require him to assume permanent disability status with the military?
- What is the patient's competency to drive, manage finances, operate machinery, and take medications?
  Will the patient require supervision for his personal safety or the safety of others?
- Does the patient evidence any behavioral problems (eg, aggressiveness, socially inappropriate, low frustration tolerance)?
- To what extent will the patient benefit from rehabilitation services, including pharmaceutical, psychotherapeutic, and behavioral management strategies?
- When will the patient require additional evaluation? For what purpose?

vocational and community reintegration process. Information should also be obtained about executive skills, including planning and organization, as well as problem-solving strategies. Rate of information processing and mental flexibility can also be assessed formally and informally during the examination process. This information can be used by vocational rehabilitation professionals to develop structured work environments, and by therapists who provide direct feedback to the patient.

Test Selection and Administration

The neurobehavioral test battery should include a variety of neuropsychological tests that measure cognitive functioning (eg, attention, processing speed, verbal, and auditory memory) and psychological tests that assess emotional and behavioral status. Qualitative and quantitative criteria should be considered when selecting tests for the assessment battery. Quantitative criteria reflect a test's empirical integrity as indicated by indexes of reliability and validity. Qualitative standards refer to a test's ability to address the referral question. For example, if the patient is being evaluated for suspected mild TBI, the test battery should include measures that are sensitive to mild brain injury.[232,233]

When selecting neuropsychological measures for the test battery, it is important to include diverse tests that are indicative of a wide variety of abilities. There are many neuropsychological tests available and clinicians occasionally have difficulty selecting the most appropriate test. As noted earlier, tests must meet reasonable standards of reliability and validity.[234] Recently, the validity of neuropsychological tests has become a central issue among researchers and clinicians in the field. Professionals are particularly interested in the predictive validity of neuropsychological tests. Predictive validity refers to the ability of test scores to predict performance on some criteria behavior, such as return to work. Some neuropsychological tests (such as the Paced Auditory Serial Addition Test and the Wechsler Memory Scale - Logical Memory subtest) have

demonstrated their ability to predict return to work after TBI.[197] Such tests could assist the clinician in addressing questions related to vocational reentry.

Other criteria to consider in selecting neuropsychological tests include the availability of normative data for persons varying in age, education, race, and sex. Normative scores from an appropriate comparison group help to establish the patient's level of functioning relative to his peers. Preference should also be given to neuropsychological tests that have been used extensively with the brain injury population, as evident in the research literature. Clinical neuropsychologists are also encouraged to use tests that have the greatest relevance to real-life functioning. For this reason, standardized measures of spelling, reading accuracy, reading comprehension, and arithmetic reasoning are useful. As a guide for the reader, other sources are available that specify commonly used neuropsychological tests and the corresponding skill areas they assess.[228,235] Assessment of the patient's emotional and behavioral status includes formal and informal methods. Informal methods consist of observing the patient's behavior during the testing process and interviewing the family. Formal methods of assessment incorporate psychological measures of personality and emotional status. Such measures should be used with caution, because most tests have been standardized on psychiatric as opposed to brain injured populations. Nonetheless, some tests can yield useful insights when cautiously interpreted.

Information from psychological tests should be compared with the patient's self-report, report of family members, and clinician's knowledge about the common effects of injury. For example, the Beck Depression Inventory[236,237] can provide useful information regarding self-esteem, levels of pessimism, appetite disturbance, and libido. However, this instrument should be interpreted carefully as it is highly susceptible to a social desirability response style. The MMPI[213] provides some indication of test taking attitudes and social desirability through examination of this test's validity scales. Other valuable information about depressive symptoms, family disturbance, somatic discomforts, and social isolation can be obtained through use of this instrument.[238,239]

Interpretation and Reporting

Clinical neuropsychologists are typically asked to make four important judgments for each cognitive skill area tested. A checklist has been developed to summarize and cogently present the results of these judgments (Exhibit 6-3[240]).

Clinicians also have the responsibility of commenting on the patient's ability to engage in certain activities that may have associated risks. Patients and family members frequently have questions about the patient's ability to drive, operate mechanical and electrical equipment, manage finances, and take medication as prescribed. Interview information about the patient's current re-

EXHIBIT 6-3

NEUROPSYCHOLOGICAL PROFILE: PERFORMANCE AND IMPAIRMENT LEVELS

1. The patient's ability in a given domain is determined by comparing his performance to the normal population. This comparison allows the clinician to estimate how the patient performed relative to persons of similar age, education, and sex.
2. A judgment is made of how the patient's performance has been affected by the injury. Reported sequelae, review of academic and vocational records, estimation of preinjury level of ability, and knowledge of common consequences of injury can help the clinician determine which skills are impaired relative to preinjury status.
3. Clinical neuropsychologists should estimate whether a patient's functioning in each area has improved, remained the same, or declined relative to previous testing. Direct comparisons with prior test scores can help establish whether therapy, subsequent injury or disease, recovery, or medication has affected patient status over time.
4. The neuropsychologist should review a profile of the patient's test scores to determine areas that are relative strengths as well as identify those areas that are clearly weakest in terms of the individual's overall performance.

sponsibilities is integrated with test information to estimate levels of competence in these skill areas. When possible, the opinions of other professionals should be solicited before reaching final determinations. For example, based on neuropsychological testing that reveals visuoperceptual problems, the clinical neuropsychologist may suggest that the patient submit to a formal driving evaluation before resumption of driving.

As noted earlier, interpretation of psychological tests should be done with particular caution as the majority of these tests were developed for use with psychiatric populations. Clinicians using the MMPI should avoid standard clinical interpretation of scale elevations because certain scales are commonly elevated over a T-score of 70 for persons with brain injury (numbers scale 1, 2, 3, 7, 8).[206,241] In these cases, standard interpretive labels, such as *schizophrenia*, would be inappropriate for patients with thought disturbances attributable to a brain injury rather than psychopathology. Clinicians who use the MMPI may derive the most valid conclusions about the emotional and personality functioning of brain injured persons by examination of responses to individual test items, including those designated as "critical items".

Practical recommendations tailored to the individual patient's needs should be included in the neurobehavioral report. In response to the problem areas identified through assessment, clinical neuropsychologists should suggest compensatory strategies and general health guidelines. All patients should, for example, be encouraged to abstain from the use of alcohol or illicit substances. Specific deficits, such as memory impairment, can be managed through the use of compensatory strategies. Numerous TBI patients report memory problems, yet they learn to cope with this handicap by maintaining a personal journal in which they record any information they need to remember. Patients who are depressed or having other adjustment problems should be encouraged to seek counseling or be evaluated for pharmacological intervention, or both. Ultimately, the value of the neurobehavioral evaluation will be seen in the clinician's ability to consolidate test scores, behavioral observations, and interviews into a set of realistic recommendations that will promote the patient's successful reintegration into the community.

Intervention

As detailed earlier, TBI often causes a diversity of emotional, behavioral, and cognitive problems, even among patients with mild injuries. A comprehensive neurobehavioral evaluation is invaluable for the diagnosis of problematic areas, and subsequent development of a treatment agenda. A variety of treatment modalities are appropriate for working with TBI individuals. Selecting the treatment of choice will depend on the needs and goals of the patient. The following section provides a brief overview of several popular treatment modalities: cognitive remediation, psychotherapy, and substance abuse counseling. For a more comprehensive review of this topic, interested readers should consult other resources.[235,242,243]

Cognitive Remediation

Individuals who sustain TBI typically suffer from a number of cognitive deficits, including impaired memory and concentration skills. The negative repercussions of cognitive deficits can be seen in all areas of function, ranging from vocational ability to social skills. For example, many patients are unable to recall news stories minutes after hearing them on television. These patients are then unable to discuss these events with others, leading them to feel socially inadequate.

Over the past two decades, cognitive remediation programs have been developed to treat problems with cognition.[243] Cognitive rehabilitation involves teaching patients strategies that will allow them to compensate for their cognitive deficits. Compensatory strategies range from simple behavioral techniques, such as keeping a written or taped memory log, to the use of mnemonic devices or computers. The efficacy of cognitive rehabilitation programs depends on a number of essential characteristics. These include multimodal training, integration of cognitive and skills training, intervention rooted in theory, and adaptation of training experiences to the real-world setting.[243]

Psychotherapy

Depression, low self-esteem, poor interpersonal relationships, and other emotional disturbances are often seen among persons with brain injury. Interestingly, patients with mild injuries report higher levels of depression than do persons with severe TBI.[206] This phenomena is attributable to the mildly injured patient's potential for greater insight and awareness of their deficits. Thus, awareness of deficits and loss caused by a brain injury may place individuals at greater risk for psychological maladaptation.

Psychotherapy is a well-established treatment modality that is intended to reduce an individual's emotional distress. The application of psychotherapy to the TBI population has only recently been demonstrated scientifically. Patients with brain injury may use the psychotherapeutic process as an opportunity to grieve the loss of their former, uninjured selves. Therapy may also teach brain injured clients effective methods for coping with depression and low self-esteem. Clients may also receive interpersonal training and feedback from their therapists, which may focus on ways to become more effective in social situations. In the context of the therapeutic relationship, the therapist is a source of empathy and support, and provides the soldier with objective guidance, facilitating the adjustment process.

Substance Abuse Counseling

Given the high incidence of preinjury alcohol abuse and the compromising effects alcohol and illicit drugs have on the rehabilitation process, professionals working in the field should closely monitor potential substance abuse problems among individuals with TBI. Interviews with the patient and family may directly address questions regarding the patient's usage patterns pre- and postinjury. If a family history of drug or alcohol problems, or both, is discovered, the patient should automatically be considered at risk. Other at-risk factors to be considered include the patient's sex (male) and age (under 25), job performance (inadequate productivity or excessive absenteeism), and blood alcohol levels at the time of injury.

A variety of measures may be undertaken to increase the likelihood of patient abstinence. Patients and families should certainly be educated about the potential dangers of substance abuse. Physicians and other rehabilitation professionals should communicate to their patients consistent guidelines about abstinence. Family, friends, and clinicians should promote participation in social and recreational activities that do not involve the use of alcohol. Finally, contracts with patients can be used to establish clear expectations, guidelines, and behavioral contingencies. Reward systems may be built into such contracts to reinforce compliant patients.

Because there are few treatment programs specifically designed to treat drug and alcohol problems among disabled individuals, professionals are encouraged to network with existing substance abuse agencies in their community in order to provide services for those in need. Rehabilitation professionals can serve to educate these agencies about specific issues related to TBI. In turn, substance abuse counselors can instruct rehabilitation professionals about substance abuse and treatment while also meeting the intensive treatment needs of disabled patients. Given the dangers of alcohol and illicit drug use and their compromising effects on recovery, substance abuse treatment must be aggressively confronted among TBI patients to protect the integrity of rehabilitation.

VOCATIONAL REENTRY

Over the past decade, data on vocational outcome following TBI have begun to accumulate and the findings are not encouraging.[209,210,244] Admittedly, these outcome studies vary on a number of parameters, including (*a*) different levels of injury severity studied, (*b*) lack of a consistent definition of employment outcome (eg, return to work vs employability), (*c*) inconsistent verification of work status, and (*d*) lack of reliable follow-up over time.[209] Despite such differences, the data inevitably lead to the same conclusion—that TBI often compromises an individual's ability to return to preinjury employment levels. Vocational reentry following TBI is a crucial issue for the military. The following section provides an overview of this issue, beginning with a review of the employment outcome literature. Vocational rehabilitation models are then presented, with an emphasis on supported employment. Criteria for decision making regarding return to active duty are also considered, particularly with regard to mild brain injury.

Employment Outcome Literature

Vocational outcome research consistently indicates that unemployment is a common sequela of brain injury. In an exemplary study, Brooks and colleagues[205] followed 134 severely brain injured persons for up to 7 years and reported that fewer than 30% of the sample were employed at follow-up. When compared to an 86% preinjury employment rate, this finding indicates the prolonged adverse effects of brain injury. Another study[54] on severe TBI reported similarly disconcerting findings. Of 60 patients seen 3.5 years after their injuries, only 13% had returned to preinjury employment levels. The majority of the sample was either unemployed (52%) or employed in positions that were less de-

manding, such as sheltered workshops (35%).

Studies on work outcome for mixed severity groups are equally alarming. Lezak[228] studied persons with mild, moderate, and severe TBI at 3 years postinjury and found that only 15% were employed in situations consonant with their preinjury educational or vocational status. Similarly, McMordie and colleagues followed a mixed severity group for up to 27 years postinjury (mean = 7 years) and found that only 19% were competitively employed.[244] Even studies limited to mild brain injury (ie, no loss of consciousness) conclude that a return to previous levels of employment can be a difficult if not impossible task.[245]

In response to these staggering findings, researchers[205,244,246,247] have begun to identify the primary risk factors associated with employment difficulties after brain injury. Despite variability between studies (eg, differing levels of severity, variable time postinjury), a number of factors were consistently related to poor work outcome. These included the following: (*a*) cognitive difficulties, including memory and processing speed[205,244]; (*b*) longer duration of coma[244,247]; (*c*) age (ie, older patients had poorer outcomes)[244,246,247]; and (*d*) family attitudes.[246] Additionally, many investigators argued that personality and behavioral disturbances (such as impaired interpersonal skills, depression, and limited self-awareness) were the greatest impediments to successful return to work.[205,209]

In a 15-year follow-up, Dresser and associates[248] looked at prognostic factors related to work outcome among 864 U.S. veterans of the Korean War.[248] The sample of veterans had a mean age of 22.7 years at the time of injury. Three quarters of the sample sustained penetrating missile wounds, while the remainder suffered closed head injuries. Although Dresser's diagnostic criteria are unclear, the results indicate that the majority of the soldiers (70%) sustained mild to moderate injuries. In their conclusions, the researchers argued that the longer lengths of posttraumatic amnesia predicted poor vocational outcome. Other predictive factors included aphasia, seizures, motor impairment, and preinjury intellectual status.

Unfortunately, few studies chronicle the long-term effects of brain injury on return to work, making it difficult to validate Dresser's predictions. Nonetheless, the existing studies[205,207] confirm that employment difficulties persist for many years after brain injury. In one of the latest outcome studies, Thomsen[207] reported on 40 severely injured patients up to 15 years postinjury. While the vast majority of patients were employed prior to their injuries, fewer than 8% had resumed full-time employment despite many years of recovery.

In summary, the work outcome literature does not paint an encouraging picture. Findings suggest that regardless of injury severity, persons with brain injury are at a substantial risk for employment difficulties. A number of factors, including cognitive deficits and behavioral and personality disturbances, appear to increase the probability of unemployment. Unfortunately, these problems do not appear to wane with the passage of time, leaving patients and their families to face the financial and emotional burdens of chronic employment problems.

Vocational Rehabilitation: The Supported Employment Model

Military personnel who have sustained a brain injury must be directed down one of three vocational paths: (1) return to the position which they held prior to their injury, (2) placement and training for a different position that better matches their postinjury abilities, or (3) pursuit of permanent disability status. The last option is clearly the most expensive to the military and demotivating to the disabled soldier. The severest of cases, however, will require permanent disability in order to safeguard the injured person and others against potentially dangerous situations.

The ideal choice of returning the soldier to his prior position is appropriate when the injury has not compromised his ability to perform his duties safely and accurately. Making this decision requires a comprehensive assessment of the individual as well as the job. For example, an individual with a mild brain injury would have more difficulty returning to an executive position than to a manual job. Safety should also be a primary consideration as even a mild injury can reduce an individual's ability to make fast decisions or react quickly to a physical threat. Other considerations include the injured person's attitude; many people have difficulty accepting that it takes longer to do things than it did before their injury. Soldiers who do return to prior positions following a brain injury should be encouraged to work slowly and carefully until they again feel comfortable in their positions. This topic is further addressed in the section, Return to Active Duty Following Mild TBI: Decision-Making Criteria.

A third option for the soldier with brain injury is to place and train him in a new position that better matches his abilities and limitations. This approach

to vocational rehabilitation, known as supported employment, is designed specifically for individuals who need support to maintain employment.[249] Supported employment offers direct on-site job training and intervention to address problems associated with physically or mentally challenged employees. While supported employment was initially developed as a community intervention method, its basic components are applicable to the task of vocational reentry among military personnel. The following sections describe the components involved in implementing a supported employment approach, including client assessment, job assessment, job placement, job site training and enabling, and follow-along services.

Client Assessment

The function of client assessment is to identify the individual's interests as well as his strengths and weaknesses so that the most appropriate placement can be made. Individualized assessment may be best accomplished by synthesizing information from a variety of sources including the individuals themselves, family members, neuropsychologists, physiatrists, neurobehavioral assessments, and especially a functional assessment of the individual's skills. Neuropsychological tests can be used to identify specific cognitive deficits—not to limit employment options, but rather to specify areas that may require compensatory strategies. A thorough medical history, including history of substance abuse and treatment for psychiatric disorders, should also be reviewed. Interviewing family members can be particularly helpful since the families know how the individual performs in real-world settings. For instance, family members may have developed compensatory strategies for memory problems or found effective techniques for dealing with behavioral concerns. Other considerations include the presence of physical limitations or other medical conditions that might interfere with successful return to duty. The individual's work history and work interest should also be considered when developing a placement site.

In the supported employment model, a "job coach" is assigned to act as a liaison between the client and the placement site. Ideally, the job coach would have training in vocational rehabilitation, but an employee from the target placement site could also be trained for this position. Most importantly, the job coach should be the client's advocate. The development of rapport between the job coach and employment candidate is a primary factor in effectively implementing the supported employment model. If good rapport is developed in the initial stages of service delivery, it will improve the job coach's ability to solve employment problems that may develop. Failure to develop rapport may result in persistent resentment or power struggles within this all too critical partnership.

Job Assessment and Placement

Prior to job placement, the job assessment phase involves a formal analysis of the skills and responsibilities necessary to perform the task. Each job must be broken down according to functional areas. For example, the job of "combat soldier" would involve a number of functions ranging from "ability to operate an assault rifle" to "possess physical stamina necessary to survive under harsh physical conditions." A careful analysis of the job requirements is necessary to determine which position is most compatible with the individual's strengths and limitations.[250,251] At the point that an individual is determined to be a good candidate for a particular job, the job placement phase is initiated. This phase includes facilitating communication between the individual and his prospective coworkers, immediate supervisors and commander, as well as family members. Conditions of employment, including work schedule and job requirements, are clarified for all concerned so that there is less chance of confusion and errors in the initial employment period. If necessary, the job coach helps make transportation arrangements and provides transportation training to the individual.

For individuals with TBI, the component of job placement should include the broader concept of environment placement. For people who are easily distracted, the seemingly "perfect" job in a stressful or chaotic environment may result in job failure. Having job candidates observe a potential employment site allows them to observe the job first hand and evaluate if they like the job or if they feel comfortable trying the job. After being given skill training or physical adaptions, or both, the job coach and the employment candidate can decide together if the work is acceptable, or if it is more beneficial to consider a different position. The social aspects of dealing with coworkers, the particular employment supervisor, and the amount of socialization required in an employment setting are also key factors in making a successful placement with long-term retention.

Job Site Training and Enabling

Following the clarification of placement arrangements, the primary activity for supported employment is job-site training (enabling). Beginning the first day of employment, the job coach accompanies the new worker to the job site and provides one-to-one training to the degree of intensity needed for the individual. The degree of training intensity expands and contracts based on the individual's progress in assuming the work responsibilities. Training strategies include the use of behavioral and systematic instructional techniques, with heavy reliance on task analytic procedures, production training strategies, and data collection procedures as on-going processes. Other sources[249,252] detail the technology of job-site training within a supported employment approach.

For individuals recovering from TBI, persons with physical disabilities, or long-term mental illness, there are going to be other major activities in addition to training during the job-site enabling component.[251] Individuals with physical impairments in addition to cognitive deficits, will need to make adaptations or modifications to the work setting and equipment (or both) to enhance the individual's ability to perform the work activities. In some cases the job coach may have the expertise to make the needed adaptations. In other instances, the coach will need to act in the role of coordinator to access other resources that can provide the service. An example of this would be to arrange for an occupational therapist to visit the employment setting to confer on the physical positioning of the worker so that fatigue is avoided and maximum efficiency of movement can be achieved. Rehabilitation technologists or engineers are also resources that may need to be accessed to help the individual achieve the ultimate requirements of the job. Another example of service coordination would be accessing psychological counseling services to help the individual work on personal or interpersonal issues that may be interfering with his ability to work independently. Generally speaking, the job coach should provide direct support or access indirect support services as needed to help the individual succeed in the employment situation.

Finally, with regard to the job-site enabling component, the job coach plays a major role as advocate for the new worker with the employer, coworkers, and family members. The job coach will need to negotiate on behalf of the individual worker on any number of issues that may come up as a result of the new work situation. For example, the job coach may negotiate a trade with the individual's coworkers which would involve swapping job duties to match each individual's interests and skills. Advocacy is of major importance in the successful implementation of supported employment, and is an ongoing activity throughout the time an individual is employed.

Job-site training and enabling intervention need to be flexible, according to the level of independence demonstrated by the employee. At times, the level of independence may fluctuate due to disrupted sleep patterns, anxiety, certain medical changes, or cognitive deficits. Specific cognitive deficits can include problems with storage, retrieval, and processing of information. Feedback from the job coach to the employee regarding job concerns should be specific. Written instructions, daily journals, directive feedback, and role playing are strategies that can be individualized and used on job sites when appropriate.

As the soldier learns the work activities and begins to demonstrate competence in independently performing job duties, the job coach's level of involvement gradually diminishes. Ideally, the supported employment model is designed so that the job-site intervention can increase or decrease based on the needs of the individual worker. When the intervention requirements of the individual have stabilized at a low level (eg, less than 10% to 20% of the required work hours per week for a period of 30 to 60 days) the job coach will initiate an ongoing schedule of follow-along support. This model of supervised return to duty would be valuable for recuperating injured soldiers on a temporary disability retirement list. Although these soldiers are not on active duty, if they were to have a trial of active duty during this time, an objective assessment of fitness could be made. The "on-site" experience gained would then help make a determination of "fitness for duty" based on more objective data about duty performance capabilities.

Follow-Along Services

Follow-along services within a supported employment approach means that the job coach maintains regular contact with the employee, his supervisor, and family members throughout the duration of the employment situation. Contacts are scheduled as needed by the employee, including off-job site contacts with family members or the individual worker, or others involved with providing employ-

ment related support services. Follow-along intervention levels may need to increase in response to changes in the work environment, such as the introduction of new work equipment, which requires the employee to be retrained. Other occasions for increased follow-along services include the occurrence of interpersonal problems between the worker and his supervisor or another employee.

With TBI clients, follow-along needs to be as proactive as possible. Regular communication with the employee in a work setting can be an effective strategy for proactive troubleshooting. Compensatory strategies or physical adaptations may need to be modified or discontinued according to the employee's level of independence. When appropriate, supervisors, coworkers, and family members can provide the job coach with information regarding work-related issues. Most important, the job coach should be available to counsel the employee and to support his efforts to be a productive and satisfied member of the armed services.

Return to Active Duty Following Mild TBI: Decision-Making Criteria

Decisions regarding vocational reentry for military personnel are particularly complex when mild brain injury is involved. Soldiers who have sustained moderate to severe injuries will typically require job retraining in their previous position, placement in a new position, or disability status. In contrast, the subtler effects of mild brain injury often complicate the decision-making process regarding return to active duty. Making this decision requires a neurobehavioral assessment of the individual, including interviews with family members, peers, and supervisors. A number of critical questions should be answered, including (*a*) what the individual's emotional status is, (*b*) what his attitude about returning to his prior position is, and (*c*) to what extent might cognitive or behavioral deficits impair judgment of safety and decision-making skills. Safety should be a primary consideration as even a mild injury can reduce reaction time and impair an individual's ability to make quick and accurate decisions. The physical evaluation board (PEB) makes the official determination of fitness for duty.

In addition to assessment of the brain-injured soldier, the decision regarding return to active duty requires a comprehensive assessment of the target job. Identical to the procedure described in the context of supported employment, the task assessment phase involves analyzing the skills and responsibilities necessary to perform the job. Each job must be broken down into its functional elements, such as "operate a fork lift" and "supervise manufacturing employees for quality control." Descriptions should be as detailed as possible to help identify those skills that are necessary to perform the task. The job environment should also be assessed and rated for safety risks. Finally, coworkers should be interviewed regarding their expectations of the returning employee's duties, responsibilities, and limitations. This process is necessary to determine if coworkers are more likely to support or disrupt the reentry process.

Ultimately, the decision regarding return to active duty following mild brain injury hinges on three issues: (1) will reinstating active duty status compromise the individual's safety or that of his coworkers; (2) will emotional, behavioral, or cognitive deficits negate the individual's ability to successfully perform his job duties; and (3) will the individual have difficulty tolerating changes in his job performance that are caused by his brain injury (eg, decreased processing speed and frustration tolerance)? If these three questions can be answered with a "no," a decision to return the soldier to active duty would be warranted. Otherwise, the alternatives of retraining, placement in a different position (eg, the supported employment model), or disability should be considered.

FAMILY OUTCOME

There is little doubt that patients are not the only victims when TBI occurs. Many clinicians believe that immediate family members frequently suffer as much, and in some cases more, than patients themselves. The negative impact on family members is not surprising given the many common adverse behavioral, emotional, and intellectual sequelae, the dramatic shift in responsibilities and roles, and the difficulties inherent in finding rehabilitation services throughout the continuum of care. The research of Kozloff[253] and Jacobs[254] has indicated that extended family and friends are rarely available to provide long-term assistance. Consequently, the burden of care falls upon immediate family members.

Clearly, family members will play a central role in the rehabilitation of soldiers with brain injury. Because of their importance, family members should be included as an intrinsic part of the rehabilitation team. Important components of an effec-

tive rehabilitation program include the family's education regarding the effects of the injury, the treatment planning, and coping strategies. Clinicians have long been aware that families can make or break the rehabilitation process. To encourage relatives' positive involvement in the patient's recovery, professionals must appreciate the nature and course of family reactions to brain injury. The following section provides an overview of family reactions to TBI. A review of the literature is presented first, followed by guidelines for family assessment and intervention.

Family Outcome Literature

The long-term burden of caring for a person with brain injury is typically assumed by some member of the patient's family.[253,254] Family members frequently identify themselves as the primary providers of care and rehabilitative therapies (eg, physical therapy) for many years following the person's injury. Unfortunately, the vast majority of these individuals receive no formal training to help them assume this caregiving role.[254] Dependent on their own resources and abilities, families members must learn to cope with someone whose character and intellect have been permanently altered. Successful adaptation, although critical, is not common.[226] More often, family members report that the burden of caring for the patient forces them into social isolation[255] and depression.[256] These negative responses have far reaching consequences, such as effecting the duration and level of the patient's ultimate recovery.[212,257]

Family outcome following TBI, although a critical factor, has been an understudied topic.[258] Moreover, the existing literature is plagued by methodological shortcomings, such as inadequate description of samples and use of instruments with questionable reliability and validity.[259] Several exceptional studies do exist[205,212,226]; however, these studies typically focus on severe TBI or the acute stages of injury, or both, thereby limiting the scope of their findings.

Despite our limited knowledge of family outcome after brain injury, the existing literature has yielded a number of interesting findings. For example, families report that physical complications cause fewer problems than do cognitive and character changes,[211] which are more disruptive to family functioning.[260] Alterations in the patient's character result in numerous stresses for the family, including (*a*) feelings that the injured person is a "stranger"[256]; (*b*) changes in family roles[261]; and (*c*) difficulties managing the patient's behavior.[257] Family members often assume the brunt of the patient's frustration and are subjected to verbal abuse and threats of physical harm.[256,260] Lacking the formal training necessary to handle these stressors,[254] many relatives utilize maladaptive coping behaviors, such as taking tranquilizers and sleeping pills.[262]

Several investigators[256,257,263] have found that the burden of caring for someone with a brain injury places relatives at an increased risk for depression, anxiety, and physical illness. In a longitudinal study during the first year after severe TBI, Oddy and colleagues[257] reported on the responses of parents and spouses to the Wakefield Depression Scale[264] at 1, 6, and 12 months postinjury. Based on the Wakefield's clinical cutoff score, 39% of the relatives were considered depressed at 1 month postinjury in comparison to 25% at the 6 and 12 month intervals. The researchers concluded that the incidence of depression among relatives declines over the first year after injury. Oddy and associates[257] also investigated the prevalence of physical illness among relatives and found that approximately 25% had suffered at least one major illness since the injury.

In a comparable study,[263] researchers from Glasgow administered psychosocial measures to relatives of severely injured patients at 3, 6, and 12 months postinjury. Family members completed a number of surveys, including the Leeds anxiety and depression scales.[265] At 3 months, 34% of the relatives reported significant anxiety and 20% were depressed. These percentages rose at 6 months to 37% on the anxiety scale and 23% on the depression scale. At 12 months, the percentage of anxious relatives remained constant at 37% while depression increased to 26%. In contrast to Oddy's group,[257] the Glasgow researchers concluded that distress among relatives does not decline during the first year after injury.

Based on extensive clinical experience, Lezak[266] developed a stage model of family reactions following a relative's brain injury. Lezak proposed that families initially (ie, first 3 months following hospitalization) experience anxiety, confusion, and denial. During this period, the family believes that the patient will return to premorbid functioning within a year. As the family attempts to help the patient return to premorbid activities (eg, work and social events), the members begin to acknowledge the patient's cognitive and behavioral deficits. This marks the second stage which, according to Lezak's model, occurs 3 to 12 months following hospitalization. During this period, caregivers' initial bewilderment evolves into depression and anxiety as

their hopes for the patient's full recovery begin to fade. In the latter stages of Lezak's model (15 months and beyond), families must work through and accept the patient's permanent deficits. Consequently, the family structure must be reorganized to accommodate role changes. In some cases, this process requires emotional or physical detachment (or both) from the patient.

The existing literature suggests that family responses to TBI are moderated by a number of factors, including family, patient, and injury characteristics (Exhibit 6-4). Family characteristics, including demographic, dynamic, and economic factors, have been insufficiently studied and their impact on family outcome is ambiguous. The only exception is the characteristic of family relationships. Preliminary research suggests that spouses have more difficulty adjusting to the injured person than do parents.[207,211,256,262] Panting and Merry[262] were among the first researchers to compare the reactions of spouses and parents to a relative's brain injury. Family members of 30 British patients with severe TBI received questionnaires and structured interviews. Based on this information, Panting and Merry concluded that injury was a "great strain" on all family members. They also observed that spouses had more difficulty adjusting to the injury than did parents, although this conclusion was not supported by any data. Unfortunately, a number of other shortcomings, such as using instruments of questionable reliability and validity, limit the effectiveness of their findings.

In a later study, Mauss-Clum and Ryan[256] compared the responses from wives and mothers of neurologically impaired patients. Of the 30 patients included in the study, the majority had sustained TBI (57%), while the remainder had suffered vascular accidents (30%), or neurological degenerative disease. Overall, family members reported feeling frustrated, irritable, and annoyed with their situations. However, a greater proportion of wives were reportedly experiencing more negative emotional reactions and life changes than were mothers. Unfortunately, a number of factors confound this finding, including the patient's age and the type of injury incurred, making it uncertain if the differences are attributable solely to the variable of relationship.

Although methodological limitations dilute any conclusions, clinical evidence supports the legitimacy of a spouse–parent distinction. Experts in the field of brain injury rehabilitation identify familial role changes as an inevitable consequence of TBI. Typically, patients become more dependent on others and, in some cases, their behavior becomes more childlike. Accepting theses changes would presumably be less difficult for parents who consider dependency a natural part of a parent–child relationship,[207,211] while childish dependence is likely to be foreign to a marital relationship and, in turn, more stressful to the uninjured spouse. Panting and Merry[262] also suggest that marital relationships are especially burdened when children are involved. In such cases, uninjured spouses lose their partners' assistance in caring for their children and are essentially forced into single parenthood.

Certain characteristics of the patient have also been found to predict family outcome following brain injury. Current research[205,212] suggests that the severity of the patients' cognitive deficits is related to family outcome. However, this is based on two indirect lines of evidence and the findings should be considered preliminary. The first line of evidence comes from research on other disability groups, including rheumatoid arthritis, spinal cord injury, and multiple sclerosis. Bishop and Miller[267] conducted a discriminant validity study on a measure of family functioning, the McMaster Family Assessment Devise (FAD).[268] The FAD was designed to assess different dimensions of family functioning, including communication, problem solving, and affective responsiveness. Bishop and Miller[267] administered the FAD to family members of persons with physical disabilities (lupus erythematosus, rheumatoid arthritis, spinal cord injury, stroke, and multiple sclerosis) and to a "nonclinical" group (no family member with a chronic physical illness). They found that the "nonclinical" families were relatively high functioning. Furthermore, families from the lupus, arthritis, and spinal cord injury groups were functioning at levels comparable to the nonclinical fami-

EXHIBIT 6-4

FACTORS THAT MODERATE FAMILY REACTIONS TO TRAUMATIC BRAIN INJURY

- Relatives' relationship to the patient
- Patient's behavioral and emotional status
- Degree of patient's cognitive impairment
- Severity of injury
- Time postinjury

lies. In contrast, the stroke and multiple sclerosis (MS) groups were reporting significant disturbances in family functioning. Bishop and Miller[267] noted that the presence of cognitive disturbances among individuals with MS and stroke differentiated these patients from the other disability groups. In turn, Bishop and Miller concluded that "cognitive and mental disturbances have the most deleterious effect on family functioning."

The second line of evidence comes from the reports of relatives living with a person with brain injury. Researchers from Glasgow, Scotland, developed two rating scales for family members of persons with brain injury.[115,212] The first was a 7-point "subjective burden" scale on which the relative chose a number between 1 (I feel no strain as a result of the changes in my family member) and 7 (I feel severe strain as a result of the changes in my family member). On the second measure (objective burden), relatives rated the patient's functioning in different categories, including memory, emotion, language, physical, and disturbed behavior. A comparison of these rating scales revealed that relatives who reported greater memory impairment were likely to endorse higher levels of stress.

In addition to cognitive impairments, many survivors of TBI undergo emotional and behavioral changes. These changes include depression, apathy, emotional lability, pathological laughter, and irritability.[204,205,207,208,210] In a number of studies,[205,207,211] family members identified psychosocial changes as the greatest impediment to daily living. In a recent investigation, parents of adult brain injury survivors responded to a number of psychosocial outcome measures, including the Symptom Checklist-90 (SCL-90)[269] and the Hassles Scale.[270] Parents also rated their children on severity of psychosocial and physical impairment using the Sickness Impact Profile.[271] Tarter[272] reported that ratings of psychosocial dysfunction in the patients were correlated with severity of stress in the parents. Furthermore, parents who endorsed higher stress levels were more likely to be psychologically maladjusted. Tarter concluded that the patient's emotional and behavioral problems not only correlated with increased parental stress, but also with psychological distress among parents.

A final category of moderating variables pertains to characteristics of the injury, such as injury severity and time postinjury. Theories of coping with crisis suggest that coping responses are mediated by the severity of a stress as well as temporal factors.[273] The brain injury literature is consistent with this hypothesis. Many studies report that family reactions vary according to the severity of the patient's injury and the time postinjury.[212,257] Historically, studies relating injury severity to family outcome have been limited by methodological oversights, inconsistencies in classification, and restricted samples. In spite of these restrictions, research on injury severity and family outcome has yielded a number of noteworthy findings.

In several investigations, researchers found that marital instability is most common among severely injured persons and their spouses.[274,275] Using a battery of standardized measures, Peters and colleagues[274] compared all injury severity groups (ie, mild, moderate, severe) on dimensions of marital adjustment. Findings indicated that greater injury severity was associated with greater marital dysfunction, particularly in the areas of affectual expression and dual consensus. In an earlier study on long-term outcome, Walker[275] found that nearly 3 years postinjury, individuals with less severe injuries were more likely to be married than were severely injured individuals. This contrasts preinjury marital status, where the proportion of married individuals was equivalent across severity groups.

Preliminary findings indicate that injury severity interacts with time postinjury to affect the relative's perception of stress[205,212] and the psychological adaptation.[257] In an early outcome study, Livingston and colleagues[276] compared the relatives of individuals with mild and severe TBI at 3 months postinjury. Family members completed the Leeds depression and anxiety scale[265] along with a number of other measures. Relatives of severely injured patients were found to be significantly more anxious than were family members of individuals with mild TBI. Levels of depression were comparable for both groups of relatives.

In a longitudinal study described earlier, researchers in Glasgow[212] examined subjective burden among relatives of severely injured patients at 3, 6, and 12 months postinjury. Length of posttraumatic amnesia (PTA), which assesses the patient's orientation to person, place, and time, was used as an index of injury severity. Reportedly, longer durations of PTA were associated with higher levels of burden among relatives at 3 and 6 months postinjury. However, PTA and subjective burden were not significantly correlated at 12 months, suggesting that the relationship between injury severity and stress weakens over time.

In a long term outcome study, researchers[226] explored changes in familial stress levels after several years postinjury. Family members of 42 severely injured patients reported levels of subjective bur-

den at 1 and 5 years postinjury using a 7-point rating scale. Scores were collapsed into three categories of subjective burden: (1) low (1–2), (2) medium (3–4), and (3) high (5–7). At 1 year postinjury, 57% of relatives were reporting medium or high levels of subjective burden. In contrast, 89% of relatives reported medium or high burden at 5 years postinjury. Brooks and colleagues[226] concluded that relatives experience higher levels of stress as time postinjury increases.

Overall, these studies suggest an interesting relationship between injury characteristics and family outcome. Temporal factors appear to influence family reactions, as relatives report more stress over time.[226] This trend is likely related to the scarce availability of professional assistance beyond the acute stages of injury.[207,211] Initially, the patient and family receive a great deal of attention from medical professionals who are working to stabilize the patient. Once the patient is released from the hospital, the availability of resources declines and family members are left to deal with the patient on their own. Over time, the family must adjust to the patient's character and cognitive changes as well as increased financial pressures and social isolation. Relatives' reports that stress levels increase with time are, therefore, understandable.

The amount of time postinjury also appears to moderate the effects of injury severity on family outcome. Research suggests that injury severity is related to stress levels and anxiety among relatives within the first 6 months of injury.[212,276] However, initial severity indexes are not correlated with relative's depression or stress at 12 months.[212,257] These preliminary findings suggest that initial severity indexes are not predictive of relatives' psychosocial outcome beyond the acute stages of recovery. The absence of a correlation between injury severity and family outcome over time is reasonable because initial severity measures are only moderately related to patient outcome.[203,232,277] Individual differences account for wide variability in outcome among patients, even among the most severely injured.[203,277]

Assessment and Intervention

Any intervention strategy must be preceded by an assessment of the family's strengths, weaknesses, and needs. Because of their specialized training and experience, social workers and psychologists often play an important role in this evaluation process. Information gathered by them is often supplemented by other professionals in medicine and healthcare. Assessment of family systems can be accomplished through interview and clinical observation. Recently, several standardized assessment instruments have been developed to provide more objective means of analyzing family status. For example, the FAD[268] is a 60-item questionnaire divided into seven subscales. Descriptive information pertaining to communication, emotional involvement, roles, problem-solving skills, and rules for behavior can be derived through examination of scale score patterns. Scores can be used to establish goals for intervention and priorities can be developed by examining scale scores and responses to individual questionnaire items. Furthermore, global information regarding overall family "health" can be derived from the seventh subscale: the General Functioning Scale. Other well-developed measures of postinjury family functioning should also be considered, including the Family Adaptability and Cohesion Scales[278] and the Family Environment Scale.[279]

Clinicians may choose to use a measure developed specifically for use with families after brain injury, such as the Family Needs Questionnaire (FNQ).[280] Measures like the FNQ provide a wealth of information about how families react to and cope with the unique stresses associated with TBI. Using the FNQ, family members rate different needs on two dimensions: (1) perceived importance (not important, somewhat important, important, and very important); and (2) the extent to which each need is met (not met, partly met, met). Each dimension provides qualitatively different information about family needs after TBI. The importance of family needs is seen in its theoretical relationship to family coping and levels of stress. Presumably, families with fewer unmet needs adapt better and experience less stress than do families with more unmet needs. Additional information about quantitative family outcome measures can be found in the review by Bishop and Miller.[267]

The amount and type of intervention must complement the strengths, weaknesses, and needs of the family that were revealed in the evaluation process. Rosenthal and Young[281] identified six modes of family intervention that may be useful following TBI:

1. *Family education* provides family members with general information about traumatic brain injury and specific information about the patient.
2. *Marital and sexual counseling* directs couples toward restructuring marital roles and redeveloping a positive sexual relationship.

3. *Family counseling* helps members of the immediate (and extended) family cope with the changes in family roles, as well as issues of loss.
4. *Family support groups* provide emotional support through members sharing their experiences, problems, and information about community resources.
5. *Family networking* develops an extended family and friendship system to share the burden of care for clients and provide mutual support.
6. *Family advocacy* helps families learn to fully utilize existing community resources, enhance existing resources, and develop new resources.

Family members should be encouraged to develop realistic expectations for patients and help them to accomplish reasonable goals. Unreasonable or overly optimistic expectations enhance the likelihood of failure and ultimately contribute to depression, anxiety, and reduced self-esteem. Conversely, being overprotective and setting overly simplistic goals does not enable soldiers to fully use existing skills. Lifelong dependence accompanied by feelings of inadequacy and resentment can easily result from chronic underestimation the client's abilities. Treatment team members should carefully work with family members to develop appropriate expectations. Regular discussions of rehabilitation program goals and progress are helpful.

Although good interpersonal communication is hard to establish and maintain, frequent contact between rehabilitation professionals, clients, and families is an essential feature of effective rehabilitation systems. Professionals can develop positive relationships with family members through sharing information about community resources, focusing on positive client aspects, speaking in practical terms, and avoiding jargon. The use of jargon will not only create interpersonal distance between professionals and families, but may cause families to feel ignorant and overwhelmed.

Rehabilitation teams are encouraged to build family education systems that routinely provide information about the patient's problems, professional treatment strategies, and means by which the family may facilitate improvement. Ongoing education is necessary given the many different challenges faced at each point of the recovery process and significant changes in family members' abilities to accommodate information. Although a single team member may be designated to provide education, team members often share the responsibility. Information may be provided in the form of written materials, lectures, workshops, or individual meetings. Rehabilitation professionals are also encouraged to learn about community support and rehabilitation programs, and to routinely share their knowledge with clients and families.

Licensed professionals such as psychologists, counselors, and social workers are most qualified to provide marital counseling, family counseling, and family networking services. Credentialed mental health workers who are employed by the military would certainly be able to provide this treatment. However, mental health workers who have not been trained in TBI rehabilitation should collaborate with a physiatrist or clinical neuropsychologist. In the greater community, family support groups are usually available through such agencies as the local NHIF.

PREVENTION

One of the biggest issues in addressing the prevalent problem of TBI is the education of laypersons about ways to avoid it. Educational programs aimed at high school and college students, community service agencies, and the mass media have impacted greatly on compliance issues regarding seatbelts, drinking and driving, and helmet use with bicycles and motorcycles. No long term comprehensive TBI program hoping to have an impact on the devastation associated with this disability would be complete without such an educational component. TBI professionals and unit commanders should be involved in an organized military effort to address this "epidemic" through education of troops and officers.

CONCLUSION

There is no question that the field of brain injury rehabilitation is still in its infancy. We have learned quite a bit regarding what interventions can decrease neurologic and medical morbidity, increase functional capabilities, and potentially expedite both neurologic and functional recovery. We still require a better basic science foundation for our methodologies and clinical theories, although we have

come a long way in addressing this issue in the last decade. By encouraging continued examinations, the rationale behind our rehabilitative treatment and its overall efficacy we will only improve the quality of care being rendered to our patients and their families. In the interim, much of rehabilitation remains an integration of art and science; the key is to understand how the two interact.

REFERENCES

1. Dixon T. Systems of care for the head-injured. *Phys Med Rehabil*. 1989;3(1):169–181.
2. Zasler ND, Kreuzter JS, Taylor D. Coma stimulation and coma recovery: A critical review. *NeuroRehabilitation: An Interdisciplinary Journal*. 1991;1(3):33–40.
3. Zasler ND. Neuromedical diagnosis and management of post-concussive disorders. In: Horn LJ, Zasler ND eds. *Phys Med Rehabil*. 1992;6(1):33–67.
4. Katz RT. Mechanisms, measurement, and management of spastic hypertonia after head injury. *Phys Med Rehabil Clin North Am*. 1992;3(2):319–336.
5. Gennarelli TA. Mechanisms of brain injury. *J Emerg Med*. 1993;11(Suppl 1):5–11.
6. Kreutzer JS, Zasler ND, Devany CW. Neuromedical and psychosocial aspects of rehabilitation after traumatic brain injury. In: Fletcher G, Jann B, Wolf S, Banja J, eds. *Rehabilitation Medicine: State of the Art*. New York: Lea and Febiger; 1992: 63–102.
7. Chicago National Safety Council Statistics Department. *Accident Facts*. Chicago: Chicago National Safety Council; 1989.
8. Frankowski RF, Annegers JF, Whitman S. The descriptive epidemiology of head injury in the United States. In: Becker DP, Povlishock JT, eds. *Central Nervous System Trauma Research Status Report*. Bethesda, Md: National Institute of Neurological and Communicative Disorders and Stroke, NIH; 1985:33–44.
9. Annegers JF, Grabow MD, Kurland LT, Laws ER. The incidence, causes, and secular trends of head trauma in Olmsted County, Minnesota: 1935–1974. *Neurology*. 1980;30:912–919.
10. Whitman S, Coonley-Hoganson R, Desai BT. Comparative head trauma experiences in two socio-economically different Chicago-area communities: A population study. *Am J Epidemiol*. 1984;119:570–580.
11. Rautio J, Paavolainen P. Afghan war wounded: Experience with 200 cases. *J Trauma*. 1988;28(4):523–525.
12. Levi L, Borovich B, Guiburd JN, Grushkiewicz I, Lemberger A, Linn S, Schachter I, Zaaroor M, Braun J. Wartime neurosurgical experience in Lebanon, 1982–1985. I: Penetrating craniocerebral injuries. *Isr J Med Sci*. 1990;26(10):548–554.
13. McCarroll JE, Zych KA. Descriptive epidemiologic survey of head injury in the army: 1983 hospitalized cases. *Neuroepidemiology*. 1989;8:48–52.
14. McCarroll JE, Gunderson C. 5-year study of incidence rates of hospitalized cases of head injuries in the US army. *Neuroepidemiology*. 1990;9:296–305.
15. Jennett B, Teasdale G, Murray G, Murray L. Head injury. In: Evans RW, Baskin DS, Yatsu FM, eds. *Prognosis of Neurological Disorders*. New York: Oxford University Press; 1992:85–96.
16. Bond MR. Assessment of psychosocial outcome of severe head injury. *Acta Neurochir*. 1976;34:57–70.
17. Heiskanen O, Martilla I, et al. Prognosis of depressed skull fractures. *Acta Chir Scand*. 1973;139:605–608.
18. Choi SS, et al. Chart for outcome prediction in severe head injury. *J Neurosurg*. 1983;59:294–297.

19. Jennett B, Teasdale G. Assessment of coma and impaired consciousness: A practical scale. *Lancet.* 1974;2:74.

20. Born JD. The Glasgow-Liege Scale. Prognostic value and evolution of motor response and brainstem reflexes after severe head injury. *Acta Neurochir*. 1988;9:1–11.

21. Horn LJ, Cope DN. Traumatic brain injury. *Phys Med Rehabil.* 1989;3(1).

22. Katz DI. Neuropathology and neurobehavioral recovery from closed head injury. *J Head Trauma Rehabil*. 1992;7(2):1–15.

23. Groswasser Z, Sazbon L. Outcome in 134 patients with prolonged posttraumatic unawareness. *J Neurosurg*. 1990;72:81–84.

24. Whyte J. Mechanisms of recovery of function following CNS damage. In: Rosentahl M, Griffith ER, Bond MR, Miller JD, eds. *Rehabilitation of the Adult and Child with Traumatic Brain Injury*. Philadelphia, Pa; FA Davis. 1990:79–88.

25. Boyeson MG. Neurochemical alterations after brain injury: Clinical implications for pharmacologic rehabilitation. *NeuroRehabilitation: An Interdisciplinary Journal*. 1991;1(1):33–43.

26. Olney JW, Sharpe LG. Brain lesions in an infant rhesus monkey treated with monosodium glutamate. *Science.* 1969;166:386–388.

27. Lyeth BG, Jenkins LW, Hayes RL. The NMDA receptor: Theoretical and clinical implications in the acute treatment of traumatic brain injury. *NeuroRehabilitation: An Interdisciplinary Journal*. 1991;1(1):50–57.

28. McDonough JH Jr, Shih TM. A study of the N-methyl d-aspartate antagonistic properties of anticholinergic drugs. *Pharmacol Biochem Behav.* 1995;51(2–3):249–253.

29. Hayes RL, Pechura CM, Katayama Y, et al. Activation of pontine cholinergic sites implicated in unconsciousness following cerebral concussion in the cat. *Science.* 1984;233:301–303.

30. Hovda DA, Feeny DM. Amphetamine with experience promotes recovery of locomotor function after unilateral frontal cortex injury in the cat. *Brain Res.* 1984;298:358–361.

31. Boyeson MG, Feeny DM. The role of epinephrine in recovery from brain injury. *Soc Neurosci Abstr*. 1984;10:68.

32. Clifton GL, Jiang JY, Lyeth BG, Jenkins LW, Hamm RJ, Hayes RL. Marked protection by moderate hypothermia after experimental traumatic brain injury. *J Cereb Blood Flow Metab.* 1991;11(1):114–121.

33. Boyeson MG, Bach-y-Rita P. Determinants of brain plasticity. *J Neurol Rehab*. 1989;3:35–37.

34. Boyeson MG, Jones JL. In: Horn LJ, Zasler ND, eds. *Theoretical Mechanisms of Brain Plasticity in Therapeutic Implications in Medical Rehabilitation of Traumatic Brain Injury*. Philadelphia, Pa: Hanley & Belfus; 1996: 77–102.

35. Chavany JA. Rôle des causes occasionelles dans le déterminisme du ramolissement cérébral (réfléxions thérapeutiques à propos de). *Pratique medicale francaise*. 1928;7:285–295.

36. Sciclounoff F. L'acetylcholine dans le traitement de l'ictus hémiplegique. *Presse medicale*. 1934;42:1140.

37. Feeney DM, Sutton RL. Pharmacotherapy for recovery of function after brain injury. *CRC Crit Rev Neurobiology*. 1987;3:135–197.

38. Cedarbaum JM. Clinical pharmacokinetics of anti-Parkinsonian drugs. *Clin Pharmacokinetics*. 1987;13:141–178.

39. Berg MJ, Ebert B, Willis DK, et al. Parkinsonism: Drug treatment. *Drug Intelligence & Clinical Pharmacy*. 1987;21(1):10–21.

40. Gualtieri CT, Chandler M, Coons TB, et al. Amantadine: A new clinical profile for traumatic brain injury. *Clin Neuropharm*. 1989;12:258–270.

41. Peterson PL, Saad J, Nigro MA. The treatment of Friedreich's Ataxia with amantadine hydrochloride. *Neurology*. 1988;38:1478–1480.

42. Cooper JR, Bloom FE, Roth RH. *The Biochemical Basis of Neuropharmacology*. New York: Oxford University Press; 1986.

43. Gualtieri CT. Pharmacotherapy and the neurobehavioral sequelae of traumatic brain injury. *Brain Injury*. 1988;2:101–129.

44. Sommi RW, Crisomn ML, Bowden CL. Fluoxetine: A serotonin-specific, second generation antidepressant. *Pharmacotherapy*. 1987;7:1–15.

45. Kastenholz KV, Crismon ML. Buspirone, a novel non-benzodiazepine anxiolytic. *Clin Pharmacy*. 1984;3:600–607.

46. Eison AS, Temple DL. Buspirone: Review of its pharmacology and current perspectives on its mechanism of action. *Am J Med*. 1986;80:1–9.

47. Cardoni AA. Focus on paroxetine: A potent, selective serotonin uptake inhibitor for use in major depression. *Hosp Formul*. 1992;27:445–461.

48. Guthrie Sk. Sertraline: A new specific Serotonin reuptake blocker. *DICP*. 1991;25:952–961.

49. Slutsker L, Hoesly FC, Miller L, et al. Eosinophilia-myalgia syndrome associated with exposure to tryptophan from a single manufacturer. *JAMA*. 1990;264;213–217.

50. Bleck TP, Klawans HL. Covulsive disorders: Mechanisms of epilepsy and anticonvulsant action. *Clin Neuropharm*. 1990;13:121–128.

51. Rolls ET. Psychopharmacology and food. In: Sandler M, Silverstone, eds. *The Neurophysiology of Feeding*. New York, NY: British Association for Psychopharmacology Monograph. 1985:1–16.

52. Morley JE. An approach to the development of drugs for appetite disorders. *Neuropsychobiology*. 1989;21:22–30.

53. Childs A. Naltrexone in organic bulimia: A preliminary report. *Brain Injury*. 1987;1:49–55.

54. Garattini S, Bizzi A, Caccia S, Mennini T. Progress report on the anorectic effects of dexfenfluramine, fluoxetine, and sertraline. *Int J Obes.* 1992;16(3):43–50.

55. Sandyk R, Lacona RP. Post-traumatic cerebellar syndrome: Response to L-tryptophan. *Int J Neuroscience*. 1989;47:301–302.

56. Trouillas P, Brudon F, Adeleine P. Improvement of cerebellar ataxia with levorotatory form of 5-hydroxytryptophan: A double-blind study with quantified data processing. *Arch Neurology*. 1988;45:1217–1222.

57. Manyam BV. Recent advances in the treatment of cerebellar ataxia. *Clin Neuropharmacology*. 1986;9:508–516.

58. Rudy TA. Pathogenesis of fever associated with cerebral trauma and intracranial hemorrhage: Thermoregulatory mechanisms and their therapeutic implication. In: *4th International Symposium on the Pharmacology of Thermoregulation*. Oxford, UK: Karger; 1979: 75–81.

59. Lipton JM, Clark WG. Neurotransmitters in temperature control. *Ann Rev Phsyiology*. 1986;48:613–632.

60. Yamawaki S, Lai H, Horita A. Dopaminergic and serotonergic mechanisms of thermoregulation: Mediation of thermal effects of apomorphine and dopamine. *J Pharmacol Exp Ther*. 1983;227:383–388.

61. Rossitch E, Bullard DE. The autonomic dysfunction syndrome: Etiology and treatment. *Br J Neurosurg*. 1988;2:471–478.

62. Gordon M, Rreiksaitis HG. Drugs and the aging brain. *Geriatrics*. 1988;43:69–78.

63. Greidanus TJ, Van Wimersma B, Jolles J, De Wied D. Hypothalamic neuropeptides and memory. *Acta Neurochir*. 1985;75:99–105.

64. Serby M, Resnick R, Jordan B, Adler J, Corwin J, Rotrosen JP. Naltrexone and Alzheimer's disease. *Prog Neuropsychopharmacol Biol Psychiatry.* 1986;10:587–590.

65. Reichert WH, Blass JP. A placebo controlled trial shows no effect of vasopressin on recovery from closed head injury. *Ann Neurol.* 1982;12:390–392.

66. Bohnen NI, Twijnstra A, Jolles J. A controlled trial with vasopressin analogue (DGAVP) on cognitive recovery immediately after head trauma. *Neurology*. 1993;43:103–106.

67. Zasler ND. Pharmacologic Aspects of Cognitive Function Following Traumatic Brain Injury. In: Kreutzer JS, ed. *Cognitive Rehabilitation for Persons with Traumatic Brain Injury: A Functional Approach*. Baltimore, Md: Paul H Brooks; 1991: 87–93.

68. Davis KL, Mohs RC, Tinkleberg JR, Pfefferbaum A, Hollister LE, Kopell BS. Physostigmine: Improvement of long term memory processes in normal humans. *Science*. 1978;201:272–274.

69. Hock FJ. Drug influences on learning and memory in aged animals and humans. *Neuropsychobiology*. 1987;17:145–160.

70. Weinberg RM, Auerbach SH, Moore S. Pharmacologic treatment of cognitive deficits: A case study. *Brain Injury*. 1987;1(1):57–59.

71. Levin HS, Peters BH, Kalisky Z, High WM, Laufen AV, Disenberg HM, Morrison DP, Gary HE. Effects of oral physostigmine and lecithin on memory and attention in closed head injured patients. *Central Nervous System Trauma*. 1986;3(4):333–342.

72. Goldberg E, Gerstman JL, Mattis S, Hughes JE, Bilder TM, Sirio CA. Effects of cholinergic treatment on post-traumatic anterograde amnesia. *Neurology*. 1982;39:581.

73. Summers WK, Majovsky LV, Marsh GM, Packiki K, Kling A. Oral tetrahydroaminoacridine in long term treatment of senile dementia. *N Engl J Med.* 1986;315(20):1241–1287.

74. Levin HS. Treatment of post-concussional symptoms with CDP-choline. *J Neurol Sci.* 1991;103:39–42.

75. Maldonado VC, Perez JBC, Escario JA. Effects of CDP-choline on the recovery of patients with head injury. *J Neurol Sci.* 1991;103:15–18.

76. Zornetzer SR. Neurotransmitter modulation and memory: A new neuropharmacological phrenology? In: Lipton MA, DiMascio A, Killian KF, eds. *Psychopharmacology—A Generation of Progress*. New York: Raven Press; 1978: 637–649.

77. Gualtieri T, Chandler M, Coons TB, Brown LT. Amantadine: A new clinical profile for traumatic brain injury. *Clin Neuropharmacol*. 1989;12:258–270.

78. Gualtieri T, Evans R. Pharmacotherapy and the neurobehavioral sequelae of traumatic brain injury. *Brain Injury*. 1988;2(2):101–129.

79. Lal S, Merbitz C, Grip J. Modification of function in head-injured patients with Sinement. *Brain Injury*. 1988;2(3):225–234.

80. McIntyre FL, Gasquoine P. Effects of clonidine on post-traumatic memory deficits. *Brain Injury*. 1989;4(2):209–211.

81. McLean A, Stanton K, Cardenas D, Bergerud D. Memory training combined with the use of oral physostigmine. *Brain Injury*. 1987;1:145–159.

82. McDonald RJ. Hydergine. A review of 26 clinical studies. In: Guski J, ed. *Determining the Effects of Aging on the Central Nervous System*. Berlin: Schering; 1980; 121–138.

83. Jenike MA, Albert MS, Heller H, Locastro S, Gunther J. Combination therapy with lecithin and ergoloid mesylates for Alzheimer's disease. *J Clin Psychiatry*. 1986;47:249–251.

84. O'Shanick GJ, Zasler ND. Neuropsychopharmacological approaches to traumatic brain injury. In: Kreutzer JS, Wehman P, eds. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990: 15–27.

85. Glenn MB, Wroblewski BW. Update on pharmacology: The choice of antidepressants in depressed survivors of traumatic brain injury. *J Head Trauma Rehabil.* 1989;4(3):85–88.

86. Fleet WS, Valenstein E, Watson RT, et al. Dopamine agonist therapy for neglect in humans. *Neurology*. 1987;37:1765–1770.

87. Zasler ND, McNeny R. Neuropharmacologic rehabilitation following traumatic brain injury via dopamine agonists. *Arch Phys Med Rehabil.* 1989;70:12–13.

88. Koller WC, Wong GF, Lang A. Post-traumatic movement disorders: Review. *Movement Disorders*. 1989;4:20–36.

89. Katz DI. Movement disorders following traumatic head injury. *J Head Trauma Rehabil.* 1990;5(1):86–90.

90. Biary N, Cleeves L, Findley L, et al. Post-traumatic tremor. *Neurology*. 1989;1:103–106.

91. Ellison PH. Propranolol for severe post-head injury action tremor. *Neurology*. 1978;28:197–199.

92. Samie MR, Selhorst JB, Koller WC. Post-traumatic midbrain tremor. *Neurology*. 1990;40:62–66.

93. Zdonczk D, Royse V, Koller WC. Nicotine and tremor. *Clin Neuropharmacol*. 1988;11:282–286.

94. Eames P. The use of Sinemet and bromocriptine. *Brain Injury*. 1989;3:319–320.

95. Stewart JT. Akathisia following traumatic brain injury: Treatment with bromocriptine. *J Neurol Neurosurg Psychiatry.* 1989;52:1200–1203.

96. Adler LA, Reiter S, Corwin J, et al. Neuroleptic-induced akathisia: Propranolol versus benztropine. *Biol Psychiatry*. 1988;23:211–213.

97. Watanabe K, Kuroiwa Y, Toyokura Y. Epilepsia partialis continua: Epileptogenic focus in motor cortex and its participation in transcortical reflexes. *Arch Neurol.* 1984;41:1040–1044.

98. Obeso JA, Artieda J, Rothwell JC, et al. The treatment of severe action myoclonus. *Brain.* 1989;112:765–777.

99. Jankovic J, Pardo R. Segmental myoclonus: Clinical and pharmacologic study. *Arch Neurol.* 1986;43:1025–1031.

100. Robin JJ. Paroxysmal choreoathetosis following head injury. *Ann Neurol.* 1977;2:447–448.

101. Richardson JC, Howes JL, Celniski MJ, et al. Kinesigenic choreoathetosis due to brain injury. *Can J Neurol Sci.* 1987;14:626–628.

102. Bontke CF. Medical complications related to traumatic brain injury. In: Horn LJ, Cope DN, eds. *State of the Art Reviews: Physical Medicine and Rehabilitation*. Philadelphia, Pa: Hanley & Belfus; 1989: 43–59.

103. Temkin NR, Dikmen SS, Wilensky AJ, et al. A randomized, double blind study of phenytoin for the prevention of post-traumatic seizures. *N Engl J Med*. 1990;323:497–502.

104. Kuhl DA, Boucher BA, Muhlbauer MS. Prophylaxis of post-traumatic seizures. *DICP Ann Pharmacotherapy.* 1990;24:277–285.

105. Dalmady-Israel C, Zasler ND. Post-traumatic seizures: A critical review. *Brain Injury*. 1993;7(3):263–273.

106. Glenn MB, Wroblewski B. Anticonvulsants for prophylaxis of post-traumatic seizures. *J Head Trauma Rehabil.* 1986;1:73–74.

107. Jones GL, Matsuo F, Baringer JR, et al. Valproic acid-associated encephalopathy. *West J Med.* 1990;153:199–202.

108. Collaborative Group for Epidemiology of Epilepsy. Adverse reactions to anti-epileptic drugs: A multicenter survey of clinical practice. *Epilepsia*. 1986;27:323–330.

109. Thompson PJ, Trimble MR. Comparative effects of anticonvulsant drugs on cognitive functioning. *Br J Clin Pract*. 1982;18:154–156.

110. Trimble MR. Anticonvulsant drugs and cognitive function: A review of the literature. *Epilepsia*. 1987;28:Suppl 37–45.

111. Meador KJ, Loring DW, Huh K, et al. Comparative cognitive effects of the anticonvulsants. *Neurology*. 1990;40:391–394.

112. Levy RH, ed. *Antiepileptic Drugs*. New York: Raven Press; 1989.

113. Kreutzer J, Zasler ND. Psychosexual consequences of traumatic brain injury: Methodology and preliminary findings. *Brain Injury*. 1989;3:177–186.

114. Zasler ND, Horn LJ. Rehabilitative management of sexual dysfunction. *J Head Trauma Rehabil.* 1990;5:14–24.

115. Zasler ND. Sexual dysfunction and traumatic brain injury. In: Silver JM, Yudofsky SC, Hales RE, eds. *Psychiatric Aspects of Traumatic Brain Injury*. Washington, DC: American Psychiatric Press; 1994: 443-470.

116. Stewart JT. Carbamazepine treatment of a patient with Kluver-Bucy Syndrome. *J Clin Psychiatry.* 1985;46:496–497.

117. Crismon ML, Childs A, Wilcox RE, et al. The effect of bromocriptine on speech dysfunction in patients with diffuse brain injury (akinetic mutism). *Clin Neuropharmacol*. 1988;11:462–466.

118. Bachman DL, Morgan A. The role of pharmacotherapy in the treatment of aphasia: Preliminary result. *Aphasiology*. 1988;2:225–228.

119. Albert ML, Bachman DL, Morgan A, et al. Pharmacotherapy for aphasia. *Neurology*. 1988;38:877–879.

120. Bieger D, Giles SA, Hockman CH. Dopaminergic influences on swallowing. *Neuropharmacology*. 1977;16:245–252.

121. Bieger D, Weerasuriya A, Hockman CH. The emetic action of L-dopa and its effect on the swallowing reflex in the cat. *J Neural Transm*. 1978;42:82–87.

122. Rucker KS, Zasler ND, Narasimhachari N, et al. Resolution of swallowing disorder with L-dopa and evaluation of CSF neurotransmitters in brain injury. *Arch Phys Med Rehabil.* 1988;69:734.

123. Biary N, Pimental PA, Langenberg PW. A double-blind trial of clonazepam in the treatment of parkinsonian dysarthria. *Neurology*. 1988;38:255–258.

124. Baratz R, Mesulam MM. Adult-onset stuttering treated with anticonvulsants. *Arch Neurol.* 1981;38:132.

125. Berrol S. Cranial nerve dysfunction. In: Horn LJ, Cope DN, eds. *State of the Art Reviews: Physical Medicine and Rehabilitation*. Philadelphia: Hanley & Belfus; 1989: 85–94.

126. Hilten Van JJ, Roos RAC. Posttraumatic hyperthermia: A possible result of frontodiencephalic dysfunction. *Clin Neurol Neurosurg*. 1991;93(3):223–225.

127. Paul SM, Barlow JR. Neurogenic heterotopic ossification. *NeuroRehabilitation*. 1993;3(3):66–78.

128. Katz RT. Mechanisms, measurement, and management of spastic hypertonia after head injury. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):319–336.

129. Guanche C, Keenan MAE. Principles of orthopedic rehabilitation. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):417–426.

130. Young B, Ott L, Phillips R, McClain C. Metabolic management of the patient with head injury. In: Eisenberg HM, Aldrich EF, eds. *Neurosurg Clin N Am.* 1991;2(2):301–320.

131. Pacult A, Gudeman SK. Medical management of head injuries. In: Becker DP, Gudeman SK, eds. *Textbook of Head Injury*. Philadelphia, Pa: WB Saunders; 1989: 192–220.

132. Bachman DL. The diagnosis and management of common neurologic sequelae of closed head injury. *J Head Trauma Rehabil.* 1992;7(2):50–59.

133. Narayan RK. Head injury. In: Grossman RG, ed. *Principles of Neurosurgery*. New York: Raven Press; 1991: 235–291.

134. Nelson VS. Pediatric head injury. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):461–474.

135. Jaffe KM, Brink JD, Hays RM, Chorazy AJL. Specific problems associated with pediatric head injury. In: Rosenthal M, Griffith ER, Bond MR, Miller JD, eds. *Rehabilitation of the Adult and Child with Traumatic Brain Injury*. Philadelphia, Pa: FA Davis; 1990: 539–557.

136. Goldman H, Englander J. Traumatic brain injury in elderly individuals. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):441–460.

137. Doherty D. Post-traumatic hydrocephalus. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):389–405.

138. Cope DN, Date ES, Mar EY. Serial computerized tomographic evaluations in traumatic head injury. *Arch Phys Med Rehab*. 1988;69:483–486.

139. Temkin NR, Dikmen SS, Winn HR. Post-traumatic seizures. In: Eisenberg HM, Aldrich EF, eds. *Neurosurg Clin North Am.* 1991;2(2):425–436.

140. Kneipp S. Cognitive remediation within the context of a community reentry program. In: Kreutzer JS, Wehman PH, eds. *Cognitive Rehabilitation for Persons with Traumatic Brain Injury: A Functional Approach*. Baltimore, Md: Paul H Brooks; 1991: 239–250.

141. McLean A, Cardenas DD, Hasekorn JK, Peters M. Cognitive psychopharmacology. *NeuroRehabilitation*. 1993;3(2):1–14.

142. Spitzer R, Williams JB, Gibbon M. *Structured Clinical Interview for DSM-III-R*. New York: Biometrics Research Department at New York State Psychiatric Institute; 1986.

143. Levin HS, High WM, Goethe KE, et al. The Neurobehavioral Rating Scale: Assessment of the behavioral sequelae of head injury by the clinician. *J Neurol Neurosurg Psychiatry*. 1987;50:183.

144. Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatry*. 1960;23:56–62.

145. Yudofsky SC, Silver JM, Jackson W, et al. The Overt Aggression Scale for the objective rating of verbal and physical aggression. *Am J Psychiatry*. 1986;143:35–39.

146. Silver JM, Hales RE, Yudofsky SC. Neuropsychiatric aspects of traumatic brain injury. In: Yudofsky SC, Hales RE, eds. *The American Psychiatric Press Textbook of Neuropsychiatry*. Washington, DC: American Psychiatric Press; 1992: 363–395.

147. Wood RLI. *Neurobehavioral Sequelae of Traumatic Brain Injury*. New York: Taylor & Francis; 1990.

148. Hall KM, Hamilton BB, Gordon WA, Zasler ND. Characteristics and comparisons of functional assessment indices: Disability Rating Scale, Functional Independence Measure, and Functional Assessment Measure. *J Head Trauma Rehabil.* 1993;8(2):60–74.

149. Hall KM. Overview of functional assessment scales in brain injury rehabilitation. *NeuroRehabilitation*. 1992;2(4):98–113.

150. Uomoto J, McLean A. Care continuum in traumatic brain injury. *Rehabil Psych.* 1989;34:71–80.

151. Mackay LE, Bernstein BA, Chapman PE, et al. Early intervention in severe head injury: Long-term benefits of a formalized program. *Arch Phys Med Rehabil*. 1992;73:635–641.

152. Ragnaarson KT, Thomas JP, Zasler ND. Model systems of care for individuals with traumatic brain injury. *J Head Trauma Rehabil.* 1993;8(2):1–11.

153. Zasler ND. A medical perspective on physician training and brain injury rehabilitation. In: Durgin CJ, Schmidt ND, Fryer LJ, eds. *Staff Development and Clinical Intervention in Brain Injury Rehabilitation*. Baltimore, Md: Aspen Publishers; 1993:257–269.

154. Bontke CF, Baize CM, Boake C. Coma Management and Sensory Stimulation. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):259–272.

155. Sandel ME. Rehabilitation management in the acute care setting. In: Horn LJ, Cope DN, eds. *State of the Art Reviews: Physical Medicine and Rehabilitation*. Philadelphia, Pa: Hanley & Belfus; 1989: 27–42.

156. Deaton AV, Holland A, Stonnington H, Veach S, Wilkins SL. Postacute rehabilitation: The day rehabilitation model. *NeuroRehabilitation*. 1991;1(3):62–69.

157. Blackerby WF, Gualtieri T. Recent advances in neurobehavioral rehabilitation. *NeuroRehabilitation*. 1991;1(3):53–61.

158. Cope DN, Cole JR, Hall KM, Barkan H. Brain injury: Analysis of outcomes in a post-acute rehabilitation system: General analysis. *Brain Injury*. 1991;5(2 part 1):111–125.

159. Fraser RT, Clemmons DC, McMahon BT. Vocational rehabilitation counseling. In: Kreutzer JS, Wehman P, eds. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990: 169–184.

160. McMahon BT, Shaw LR, eds. *Work Worth Doing: Advances in Brain Injury Rehabilitation*. Orlando, Fla: Paul M Deutsch; 1991.

161. Cervelli L. Re-entry into the community and systems of post-hospital care. In: Rosenthal M, Griffith ER, Bond MR, Miller JD, eds. *Rehabilitation of the Adult and Child with Traumatic Brain Injury*. Philadelphia, Pa; FA Davis; 1990: 463–475.

162. Cervelli L, Berrol S. Description of a model care system. In: *Head Injury Rehabilitation Project Final Report*. Grant #13-P-59156/9. San Jose, Calif: Santa Clara Valley Medical Center; 1982:4.

163. Van Der Schaaf S. *Personal communication*. 1992.

164. Dixon TP. Systems of care for the head-injured. In: Horn LJ, Cope DN, eds. *State of the Art Reviews: Physical Medicine and Rehabilitation*. Philadelphia, PA: Hanley & Belfus; 1989: 169–181.

165. Horn LJ. Systems of care for the person with traumatic brain injury. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):475–492.

166. Anderson DW, McLaurin RL. The national head and spinal cord injury survey. *J Neurosurgery.* 1980;53:Suppl 1.

167. Miller JD, Jones PA. The work of a regional head injury service. *Lancet*. 1985;1:1141.

168. Rimel RW, Giordani B, Barth JT, et al: Clinical and scientific communications: Disability caused by minor head injury. *Neurosurgery*. 1981;9:221–228.

169. Rimel RW, Jane JA. Minor head injury: Management and outcome. In: Williams RH, Rengachery SS, eds. *Neurosurgery*. New York: McGraw-Hill; 1985.

170. Horn LJ, Zasler ND. Rehabilitation of post-concussive Disorders. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus; 1992;6(1).

171. Lehmkuhl LD. *Brain injury glossary*. Houston, Tex: Traumatic Brain Injury Model System Research Program: The Institute for Rehabilitation and Research; 1990.

172. Leininger BE, Gramling SE, Farrell AD, et al. Neuropsychological deficits in symptomatic minor head injury patients after concussion and mildconcussion. *J Neurol Neurosurg Psychiatry*. 1990;53:293–296.

173. Wilkinson HA, Binder LM. Commentary. *J Neurol Neurosurg Psychiatry.* 1991;54:846–847.

174. Gennarelli TA. Mechansims and pathophysiology of cerebral consussion. *J Head Trauma Rehabil.* 1986;1:23–29.

175. Zasler ND. Mild traumatic brain injury: Medical assessment and intervention. *J Head Trauma Rehabil.* 1993.

176. Travell JG, Simons OG. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. Baltimore, Md: Williams & Wilkins; 1983.

177. Vernon H, ed. *Upper Cervical Syndrome: Chiropractic Diagnosis and Treatment*. Baltimore, Md: Williams & Wilkins; 1988.

178. Gronwall D. Rehabilitation programs for patients with mild head injury: Components, problems and evaluation. *J Head Trauma Rehabil.* 1986;1:53–62.

179. Kay T. Neuropsychological diagnosis: Disentangling the multiple determinants of functional disability after mild traumatic brain injury. In: Horn LJ, Zasler ND, eds. Rehabilitation of Post-Concussive Disorders. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus: 1992;6(1):109–127.

180. Binder LM. Malingering following minor head trauma. *Clin Neuropsychologist.* 1990;4(1):25–36.

181. Plumb F, Posner JB. *The Diagnosis of Stupor and Coma*. Philadelphia, Pa: FA Davis; 1980.

182. Giacino JT, Zasler ND, Katz D, Kelly JP, Rosenberg J, Filley C. Practice guidelines for assessment and management of the vegetative and minimally conscious states: Proceedings of the Aspen Neurobehavioral Consensus Conference. *J Head Trauma Rehabil.* In press.

183. American Academy of Neurology. Multisociety task force on PVS. Medical aspects of the persistent vegetative state: Statement of a multi-society task force. *N Engl J Med.* 1994;330(21):1499–1508.

184. Weiner JD. Legal issues regarding patients in coma or in persistent vegetative state. In: Sandel ME, Ellis DW, eds. The Coma Emerging Patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus: 1990;4(3):569–578.

185. Banja JD. Ethical aspects of treatment for coma and the persistent vegetative state. In: Sandel ME, Ellis DW, eds. The Coma Emerging Patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus: 1990;4(3):579–592.

186. Council on Scientific Affairs and Council on Ethical and Judicial Affairs. Persistent vegetative state and the decision to withdraw or withhold life support. *JAMA*. 1990;263:426–430.

187. Ethics and Humanities Sub-committe of the American Academy of Neurology. Position Statement: Certain aspects of the care and management of profoundly and irreversibly paralyzed patients with retained consciousness and cognition. *Neurology*. 1993;43:222–223.

188. McLellan DR. The structural basis of coma and recovery: Insights from brain injury in humans and experimental animals. In: Sandel ME, Ellis DW, eds. The Coma Emerging Patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus; 1990;4(3):389–408.

189. DeVolder AG, Goffinet AM, Bol A, et al. Brain glucose metabolism in postanoxic syndrome: positron emission tomographic study. *Arch Neurol.* 1990;47:197–204.

190. Levy DE, Sidtis JJ, Rottenberg DA, et al. Differences in cerebral blood flow and glucose utilization in vegetative versus locked-in patients. *Ann Neurol.* 1987;22:673–682.

191. Ellis DW, Rader MA. Structured sensory stimulation. In: Sandel ME, Ellis DW, eds. The Coma Emerging Patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus; 1990;4(3):465–478.

192. Pierce JP, Lyle DM, Quine S, et al. The effectiveness of coma arousal intervention. *Brain Injury*. 1990;4:191–197.

193. Rader MA, Alston JB, Ellis DW. Sensory stimulation of severely brain injured patients. *Brain Injury*. 1989;3:141–147.

194. Giacino JT, Zasler ND. Outcome following severe brain injury: The comatose, vegetative and minimally responsive patient. *J Head Trauma Rehabil.* 1995;10(1);40–56.

195. Whyte J, Glenn MB. The care and rehabilitation of the patient in a persistent vegetative state. *J Head Trauma Rehabil.* 1986;1:39–53.

196. Sandel ME, Ellis DW, eds. The coma emerging patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus; 1990.

197. Brooks N, McKinlay W, Symington C, Beattie A, Campsie L. Return to work within the first seven years of severe head injury. *Brain Injury*. 1987;1:5–19.

198. Heaton RK, Pendleton MG. Use of neuropsychological tests to predict adult patients' everyday functioning. *J Consulting Clin Psychology.* 1981;49:807–821.

199. Dikmen S, Reitan RM, Temkin NR. Neuropsychological recovery in head injury. *Arch Neurol.* 1983;40:333–338.

200. Jennett B, Snoek J, Bond MR, Brooks N. Disability after severe head injury: Observations on the use of the Glasgow Outcome Scale. *J Neurol Neurosurg Psychiatry*. 1981;50:1613–1618.

201. Jennett B, Teasdale G. *Management of Head Injuries*. Philadelphia, Pa: FA Davis; 1981.

202. Jennett B, Bond M. Assessment of outcome after severe brain damage: A practical scale. *Lancet*. 1975;1:480–487.

203. Dikmen S, Machamer J, Temkin N, McLean A. Neuropsychological recovery in patients with moderate to severe head injury: 2 year follow-up. *J Clin Experimental Neuropsychology.* 1990;12:507–519.

204. Oddy M, Coughlan T, Tyerman A, Jenkins D. Social adjustment after closed head injury: A further follow-up 7 years after injury. *J Neurol Neurosurg Psychiatry*. 1985;48:564–568.

205. Brooks N, Campsie L, Symington C, Beattie A, McKinlay W. The effects of severe head injury on patient and relative within seven years of injury. *J Head Trauma Rehabil.* 1987;2:1–13.

206. Leininger BL, Kreutzer JS, Hill MR. Comparison of minor and severe head injury emotional sequelae using the MMPI. *Brain Injury*. 1991;5:199–205.

207. Thomsen IV. Late outcome of severe blunt head trauma: A 10–15 year follow-up. *J Neurol Neurosurg Psychiatry.* 1984;47:260–268.

208. Lezak MD. Relationships between personality disorders, social disturbances, and physical disability following traumatic brain injury. *J Head Trauma Rehabil.* 1987;2:57–69.

209. Ben-Yishay Y, Silver S, Piasetsky E, Rattok J. Relationship between employability and vocational outcome after intensive holistic cognitive rehabilitation. *J Head Trauma Rehabil.* 1987;2:35–48.

210. Rappaport M, Herrero-Backe C, Rappaport ML, Winterfield KM. Head injury outcome up to ten years later. *Arch Phys Med Rehabil.* 1989;70:885–892.

211. Thomsen IV. The patient with severe head injury and his family. *Scand J Rehabil Med.* 1974;6:180–183.

212. McKinlay WW, Brooks DN, Bond MR, Martinage DP, Marshall MM. The short-term outcome of severe blunt head injury as reported by relatives of injured persons. *J Neurol Neurosurg Psychiatry*. 1981;44:527–533.

213. Hathaway SR, McKinley JC. *Manual for the Minnesota Multiphasic Personality Inventory*. Minneapolis: University of Minnesota Press; 1982.

214. Clark W, Midanik L. *Alcohol use and alcohol problems among U.S. adults: Results of the 1979 national survey*. In: US Department of Health and Human Services, Alcohol and Health Monograph. No. 1: Alcohol consumption and related problems. Washington, DC: US Government Printing Office; 1982. DHHS Pub No ADM 82-1190.

215. Institute of Medicine, National Academy of Sciences. *Alcoholism, Alcohol Abuse, and Related Problems: Opportunities for Research.* Washington, DC: National Academy Press; 1980.

216. Greer BG, Roberts R, May G, Jenkins WM. Identification of substance abuse in a vocational evaluation setting. *J Rehabil.* 1988;54:42–45.

217. Johnson P, Armor D, Polich S, Stambol H. *U.S. adult drinking practices: Time, trends, social correlates, and sex roles*. Draft report prepared for the National Insitute on Alcohol Abuse and Alcoholism under Contract No ADM 281-76-0200. Santa Monica, Calif: Rand Corporation; 1977.

218. Rimel R, Giordani B, Barth J, Jane J. Moderate head injury: Completing the clinical spectrum of brain trauma. *Neurosurgery*. 1982;11(3):344–351.

219. Zasler ND. Neuromedical aspects of alcohol use following traumatic brain injury. *J Head Trauma Rehabil.* 1991;6(4):78–80.

220. Sparadeo FR, Gill D. Effects of prior alcohol use on head injury recovery. *J Head Trauma Rehabil.* 1989;4:75–82.

221. Galbraith S, Murray W, Patel A, Knill-Jones R. The relationship between blood alcohol and head injury and its effect on the consciousness level. *Br J Surg.* 1976;63:128–130.

222. Murray PK. Clinical pharmacology in rehabilitation. In: Caplan B, ed. *Rehabilitation Psychology Desk Reference*. Rockville, MD: Aspen Publishers; 1987.

223. Greer BG. Substance abuse among people with disabilites: A problem of too much accessibility. *J Disability.* 1986;14:34–38.

224. Hackler E, Tobis JS. Re-integration into the community. In: Rosenthal M, Griffith E, Bond M, Miller J, eds. *Rehabilitation of the Head Injured Adult*. Philadelphia, Pa: FA Davis; 1983.

225. Rohe D, DePompolo R. Substance abuse policies in rehabilitation medicine departments. *Arch Phys Med Rehabil.* 1985;66:619–624.

226. Brooks N, Campsie L, Symington C, Beattie A, McKinlay W. The five year outcome of severe blunt head injury: A relative's view. *J Neurol Neurosurg Psychiatry.* 1986;49:764–770.

227. Devany CW, Kreutzer JS, Halberstadt LJ, West DD. Referrals for supported employment after brain injury: Neuropsychological, behavioral, and emotional characteristics. *J Head Trauma Rehabil.* 1991;6(3):59–70.

228. Lezak MD. *Neuropsychological Assessment*. 2nd ed. New York: Oxford University Press; 1983.

229. Ruff RM, Baser CA, Johnston JW, et al. Neuropsychological rehabilitation: An experimental study with head-injured patients. *J Head Trauma Rehabil.* 1989;4(3):20–36.

230. Hart T, Hayden ME. The ecological validity of neuropsychological assessment and remediation. In: Uzzell BP, Gross Y, eds. *Clinical Neuropsychology of Intervention*. Boston: Martinus Nijhoff; 1986: 21–50.

231. Kreutzer JS, Leininger BE, Sherron PD, Groah CH. Managing psychosocial dysfunction. In: Wehman P, Kreutzer JS, eds. *Vocational Rehabilitation for Persons with Traumatic Brain Injury*. Rockville, MD: Aspen Publishers; 1990.

232. Barth JT, Macciocchi SN, Giordani B, Rimel R, Jane JA, Boll TJ. Neuropsychological sequelae of minor head injury. *Neurosurgery*. 1983;13:529–533.

233. Leininger BE, JS Kreutzer. Neuropsychological outcome of adults with mild traumatic brain injury: Implications for clinical practice and research. In: Horn LJ, Zasler ND, eds. Rehabilitation of Post-concussive Disorders. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus. 1992;6(1):169–182.

234. Anastasi A. *Psychological Testing*. 6th ed. New York: Macmillan; 1988.

235. Kreutzer JS, Devany CW, Myers SL, Marwitz JH. Neurobehavioral outcome following traumatic brain injury: Review, methodology, and implications for cognitive rehabilitation. In: Kreutzer J, Wehman P, eds. *Cognitive Rehabilitation for Persons with Traumatic Brain Injury: A Functional Approach*. Baltimore, Md: Paul H Brooks; 1991: 55–71.

236. Beck AT. *Depression: Clinical, Experimental, and Theoretical Aspects*. New York: Harper & Row; 1967.

237. Beck AT, Ward C, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry.* 1961;4:561–571.

238. Dahlstrom WG, Welsh GS, Dahlstrom LE. *An MMPI Handbook, Volume 1: Clinical Interpretation*. Minneapolis: University of Minnesota Press; 1972.

239. Graham JR. *The MMPI: A Practical Guide*. 2nd ed. New York: Oxford University Press; 1987.

240. Kreutzer JS, Leininger BE, Harris JA. The evolving role of neuropsychology in community integration. In: Kreutzer J, Wehman P, eds. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990.

241. Novack TA, Daniel MS, Long CJ. Factors related to emotional adjustment following head injury. *Int J Clin Neuropsychology*. 1984;6:139–142.

242. Kreutzer JS, Wehman P. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990.

243. Gordon WA, Hibbard MR. The theory and practice of cognitive remediation. In: Kreutzer J, Wehman P, eds. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990: 13–22.

244. McMordie WR, Barker SL, Paolo TM. Return to work after head injury. *Brain Injury*. 1990;4:57–69.

245. Wrightson P, Gronwall D. Time off work and symptoms after minor head injury. *Brain Injury*. 1981;12:445–454.

246. Humphrey M, Oddy M. Return to work after head injury: A review of post-war studies. *Brain Injury*. 1980;12: 107–114.

247. Rao N, Rosenthal M, Cronin-Stubbs D, Lambert R, Barnes P, Swanson B. Return to work after rehabilitation following traumatic brain injury. *Brain Injury*. 1990;4:49–56.

248. Dresser A, Meirowsky A, Weiss G, McNeel M, Simon G, Caveness W. Gainful employment following head injury: Prognostic factors. *Arch Neurol.* 1973;29:111–116.

249. Kreutzer JS, Wehman P, Morton MV, Stonnington HH. Supported employment and compensatory strategies for enhancing vocational outcome following traumatic brain injury. *Brain Injury*. 1988;2:205–223.

250. Kreutzer JS, Morton MV. Supported employment for persons with head injury. In: Wehman PS, Moon MS, eds. *Vocational Rehabilitation and Supported Employment*. Baltimore, Md: Paul H Brooks; 1988.

251. Wood W. Supported employment for persons with physical disabilities. In: Wehman PS, Moon MS, eds. *Vocational Rehabilitation and Supported Employment*. Baltimore, Md: Paul H Brooks; 1988.

252. Wehman P, Moon MS. *Vocational Rehabilitation and Supported Employment*. Baltimore, Md: Paul H Brooks; 1988.

253. Kozloff R. Networks of social support and the outcome from severe head injury. *J Head Trauma Rehabil.* 1987;2:14–23.

254. Jacobs HE. The Los Angeles Head Injury Survey: Procedures and initial findings. *Arch Phys Med Rehabil.* 1988;69:425–431.

255. Lezak MD. Living with the characterologically altered brain injured patient. *J Clin Psychiatry.* 1978;39:111–123.

256. Mauss-Clum N, Ryan M. Brain injury and the family. *J Neurosci Nurs.* 1981;13:165–169.

257. Oddy M, Humphrey M, Uttley D. Stresses upon the relatives of head-injured patients. *Br J Psychiatry.* 1978;133:507–513.

258. Florian V, Katz S, Lahav V. Impact of traumatic brain damage on family dynamics and functioning: A review. *Brain Injury*. 1989;3:219–233.

259. Kreutzer JS, Marwitz JH, Kepler K. Traumatic brain injury: Family response and outcome. *Arch Phys Med Rehabil.* 1992;73(8):771–778.

260. Lezak M. Brain damage is a family affair. *J Clin Exp Neuropsychol.* 1988;10:111–123.

261. Rosenbaum M, Najenson T. Changes in life patterns and symptoms of low mood reported by wives of severely brain-injured soldiers. *J Consulting Clin Psychology.* 1976;44:881–888.

262. Panting A, Merry PH. The long term rehabilitation of severe head injuries with particular reference to the need for social and medical support for the patient's family. *Rehabilitation*. 1972;38:33–37.

263. Livingston MG, Brooks DN, Bond MR. Three months after severe head injury: Psychiatric and social impact on relatives. *J Neurol Neurosurg Psychiatry.* 1985;48:870–875.

264. Snaith RP, Ahmed SN, Mehta S, Hamilton M. Assessment of the severity of primary depressive illness. *Psychol Med.* 1971;1:143–149.

265. Snaith RP, Bridge GW, Hamilton M. The Leeds Scales for the self-assessment of anxiety and depression. *Br J Psychiatry.* 1976;128:156–165.

266. Lezak MD. Psychological implications of traumatic brain damage for the patient's family. *Rehabil Psych.* 1986;31:241–250.

267. Bishop DS, Miller IW. Traumatic brain injury: Empirical family assessment techniques. *J Head Trauma Rehabil.* 1988;3:16–30.

268. Epstein NB, Baldwin LM, Bishop DA. The McMaster family assessment devise. *J Marital Fam Ther*. 1983;9:171–180.

269. Derogatis LR. The *SCL-90 Manual: Scoring, Administration, and Procedures for the SCL-90.* Baltimore, Md: Johns Hopkins University School of Medicine; 1977.

270. Kanner A, Coyne J, Schaefer D, Lazarus R. Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. *J Behav Med.* 1981;4:1–39.

271. Gilson B, Bergner M, Bobbitt R, Carter W. *The Sickness Impact Profile: Final Development and Testing.* Seattle, Wash: Center for Health Services, University of Washington; 1979.

272. Tarter SB. Factors affecting adjustment of parents of head trauma victims. *Arch Clin Neuropsychology.* 1990;5:15–22.

273. Auerbach SM. Assumptions of crisis theory and a temporal model of crisis intervention. In: Auerbach SM, Stolberg A, eds. *Crisis Intervention with Children and Families*. Washington: Harper & Row; 1986.

274. Peters LC, Stambrook M, Moore AD, Esses L. Psychosocial sequelae of closed head injury: Effects on the marital relationship. *Brain Injury*. 1990;4:39–47.

275. Walker AE. Long term evaluation of the social and family adjustment to head injuries. *Scand J Rehabil Med*. 1972;4:5–8.

276. Livingston MG, Brooks DN, Bond MR. Patient outcome in the year following severe head injury and relatives' psychiatric and social functioning. *J Neurol Neurosurg Psychiatry.* 1985;48:876–881.

277. Brooks N, McKinlay W. Personality and behavioural change after severe blunt head injury a relative's view. *J Neurol Neurosurg Psychiatry.* 1983;46:336–344.

278. Olson DH, Portner J, Lavee Y. *FACES-III: Family Adaptability and Cohesion Scales*. St. Paul, Minn: Family Social Science, University of Minnesota; 1985.

279. Moos R, Moos B. *Family Environment Scale: Manual*. Palo Alto, Calif: Consulting Psychologists Press; 1974.

280. Kreutzer JS. *Family Needs Questionnaire*. Richmond, VA: Rehabilitation Research and Training Center on Severe Traumatic Brain Injury, Medical College of Virginia; 1988.

281. Rosenthal M, Young T. Effective family intervention after traumatic brain injury: Theory and practice. *J Head Trauma Rehabil.* 1988;3:42–51.

THE VIETNAM HEAD INJURY STUDY: OVERVIEW OF RESULTS TO DATE

Excerpted from Andres M. Salazar, M.D., Colonel, Medical Corps, U.S. Army (Ret); H. M. Brown; Karen Schwab, Ph.D.; Jordan Grafman, Ph.D. Unpublished report, 1997. VHIS Collaborative Group, Vietnam Head Injury Study, Building 1, Walter Reed Army Medical Center, Washington, DC 20307-5001.

INTRODUCTION

The Vietnam Head Injury Study (VHIS), a collaborative project of the Department of Defense (DOD), the Veterans Administrations (VA, now the Department of Veterans Affairs [DVA]), National Institutes of Health (NIH), and the American Red Cross (ARC), involves a detailed follow-up evaluation of 1,221 veterans who survived brain wounds during the Vietnam War. The VHIS was conceived and developed by William F. Caveness at NIH in Bethesda, Maryland, who had also been responsible for similar studies of Korean War veterans. At the beginning of the Vietnam conflict he developed a one-page head and spinal injury registry form and secured the support for disseminating it throughout the Vietnam theater. Registry forms were completed by neurosurgeons in field hospitals throughout Vietnam on their head-injured patients who had survived the first week postinjury. Completed forms were returned to Dr. Caveness at NIH on patients injured from 1967 to 1970, when the initial registry was closed at 2,000 cases.

The primary intent was to register a large number of wounded men for a prospective follow-up study, and not necessarily to obtain a random sample of all head wounds incurred in combat. Nevertheless, patients were entered from army, navy, marine, and air force units throughout the theater of operations and represented a wide spectrum of injury types and severity; the sample amounts to about 7% of all casualties with head injuries in the Vietnam War. The registry forms included demographic data, as well as information from the field on the circumstances of the casualty's injury, wound location, initial status, early treatment, and initial hospital course. Complete military medical and VA follow-up records were subsequently obtained on 1,221 registry cases; of these, 77% had suffered missile fragments wounds, 15% had gunshot wounds, and only 8% had a closed head injury.

The VHIS registry consists of a population with unique advantages for the study of head injury outcomes. Prior to their injuries, these men were healthy, young, and employed. In addition, preinjury intelligence test data were available for these casualties, having been collected as part of their evaluation on entering the military.

EVALUATION AND ANALYSIS OF DATA

The Vietnam Head Injury Study is a rich source of knowledge about the nature of militarily relevant head injuries. This brief review of the study to date cites some of the important findings of the VHIS group, with special emphasis placed on findings and conclusions that are relevant to the rehabilitation of military personnel with head injuries. Analysis of the VHIS data was carried out in two phases, the second of which is still ongoing:

- Phase I of the VHIS consisted of a detailed, standardized review and computerization of all medical records by a small group of experienced neurosurgeons and neurologists.
- In Phase II, an unbiased sample of 520 of these casualties, some 15 years postinjury, along with 86 uninjured Vietnam veterans as controls, underwent an extensive, standardized, 1-week-long, multidisciplinary, inpatient evaluation at Walter Reed Army Medical Center (WRAMC), Washington, DC. By the end of this evaluation, over 22,000 datapoints had been gathered and computerized on each Phase II participant.

Phase I: Records Review

Phase I of the VHIS began in 1975, some 5 years after the end of data collection. Medical records of all military and VA hospitalizations and all outpatient care visits were collected. After excluding patients with associated spinal cord injury and those for whom adequate medical records could not be located, the formal registry was reduced to 1,221 men (1,133 with penetrating head injury and 88 with closed head injury). A detailed review and codification of these records was undertaken by a small group of experienced neurologists and neurosurgeons under the direction of W. F. Caveness. About 2,000 datapoints were recorded on each patient and organized into various domains for analysis. Several reports have been published based on this data: on posttraumatic epilepsy,[1] retained bone fragments and abscess,[2,3] motor recovery,[4] and other neurosurgical issues.[5]

Phase II: Follow-up Evaluation

Phase II of the VHIS, organized in 1981 by Dr. Caveness, was made possible by a unique collaboration of the three military services, the DVA, and the ARC. The ARC contacted patients and conducted the initial in-home interviews;

the US Air Force provided transportation; WRAMC provided the hospital beds and some personnel; the National Naval Medical Center, Bethesda, Maryland, provided the computed tomography (CT) scans; and the DVA provided operational funds for the project.

Registry patients were located by the ARC and were invited to participate in the follow-up study. Trained ARC workers conducted a social work and family history interview, and volunteers who were able then came to WRAMC for a week-long hospital evaluation. Of the 1,125 men still alive at the time, more than 750 initially agreed to participate and underwent the ARC interview, although only 520 were actually able to come to WRAMC over the next 3 years (1981–1984).

Control subjects were uninjured Vietnam veterans selected from DVA beneficiary files. Of those invited to participate, 86 came to WRAMC and received the full VHIS evaluation battery (except for the CT scan).

The extensive multidisciplinary evaluation included the following:

- neurology history and examination (2 h),
- neuropsychological evaluation (16 h),
- speech and language evaluation (6 h),
- rehabilitation/motor function evaluation (4 h),
- audiology examination (2 h),
- CT scan, and
- electroencephalography (EEG) and visual and auditory evoked potentials.

CT scans were performed on a General Electric 8800 Scanner in standard cuts. Involvement of specific brain areas was coded for computer entry by using templates prepared for each slice, assigning code numbers to specific brain structures or areas. Structures were coded as normal, partly involved, fully involved, or unreadable due to metal artifact.

RESULTS: PHASE II FOLLOW-UP STUDY

Perhaps the most encouraging finding to date has been the amazing ability of many of these young men to compensate for their injuries despite the large size of many of their brain wounds. CT scanning showed that 80% of the registry patients had injuries involving multiple lobes of the brain, and 33% had bilateral injury (the injuries were thus much larger than had previously been estimated from surgical reports and X-ray examinations of the skull alone, the only means of assessing brain wound size in the Vietnam era). To the casual observer, almost two thirds of these patients might appear to be functioning normally. Nevertheless, careful examination almost invariably revealed some neurological or neurobehavioral functional deficit. Unrecognized cognitive and especially memory deficits often resulted in a failure to seek medical help or veteran's benefits; many patients with large brain wounds had been returned to duty after cranioplasty and had eventually received nonmedical discharges from the military services. Thirty-eight percent of our brain-injured patients (vs 28% of controls) received a recommendation for psychological intervention or therapy, although many had previously undergone such therapy before participation in the VHIS. Overall, recommendations for neurological or psychological follow-up were made in 72% of the brain-injured patients and 52% of the controls; the brain-injured group received more recommendations per person than the controls. Much of the analysis made possible the formulation of guidelines, which may be useful in (1) predicting the eventual outcome in brain-injured patients, (2) providing such patients and their families some insight into difficulties that they may expect, and (3) targeting specific therapies for them early in their convalescence.[6]

Mortality

A 15-year mortality study on men in the registry showed 90 deaths (8%). Most of the deaths occurred early in the first year after trauma and were secondary to the direct effects of brain injury or the sequelae of coma. Complications, particularly infections, were significant mortality factors. Coma was the best prognostic predictor of early death. Posttraumatic epilepsy was not related to mortality except for the risks accompanying each seizure. The population began to approach the actuarial mortality norm of their peers within about 3 years of injury.[5]

Neurology and Neurosurgery

Thanks in large part to helicopter evacuation and the deployment of neurosurgeons close to the battlefront, a wounded soldier in Vietnam usually received prompter and better medical care for such wounds than was available anywhere else in the world at that time. Most men had definitive neurosurgery within 6 hours of injury, but a preliminary analysis of complication rates by delay in provision of initial surgery suggests that mortality and morbidity begin to rise significantly only with delays of longer than 24 to 48 hours. Combined with data on early hospital mortality, this type of information may be important for establishing military medical logistical and evacuation policy

in future conflicts (in which battlefield conditions and distance from hospitals, for example, might make such prompt care impossible to provide).

Retained Bone Fragments

One important and controversial neurosurgical issue addressed has been the significance of retained bone within the intracerebral wound tract. The experience of previous wars had suggested that retained bone fragments increased morbidity and mortality. It thus became standard operating procedure in Vietnam to remove such fragments surgically, even if this called for repeated brain operations in otherwise healthy, convalescing patients. Over 10% of our patients thus underwent repeat surgery for this purpose, some of them multiple times. Retrospective analysis of CT scans now shows that more than 20% of the VHIS population still have retained bone fragments, including almost 75% of those who had had secondary surgery for removal of such fragments. Moreover, a detailed review of the medical records of each of these men shows that in this population, retained bone, per se, has no significant effect on mortality, morbidity (including infection rate), or sequelae of brain injury. This strongly suggests that repeat operations for retained bone, in the absence of complications, are not warranted and may be detrimental.[7]

Other neurosurgical questions that are currently being addressed in the data include the relation of ventricular enlargement to intraventricular wounds, clinical and cognitive deficits, and eventual community adjustment; and the relation of surgical complications such as infection to wound type, fragment type and size, surgical procedure, spinal fluid leaks, and eventual outcome.

Posttraumatic Epilepsy

The incidence of posttraumatic epilepsy (PTE) some 15 years after injury in the VHIS was 51%. This overall incidence appears to be somewhat higher than figures reported for previous wars (World War I, 38%; World War II, 34%, 43%; Korea, 36%), the Iran–Iraq and Lebanese conflicts, and even for these same patients at an average 5-year follow-up (34%).[1] Explanations of this apparent discrepancy include the longer follow-up, the fact that detailed histories were available in person from the patient and family, and inclusion in the VHIS cohort of patients with injuries so severe that they would not have survived in previous wars. In 57% of the Vietnam group with seizures, attacks began within 1 year of injury; in 18%, 5 to 10 years after injury; and in 7%, 10 or more years after injury. When compared with a normal age-matched population, the relative risk of epilepsy in the Vietnam cohort was 520 in the first year after injury, 90 in years 2 to 5, and 36 in years 5 to 10. At years 10 to 15 postinjury, the relative risk of developing PTE was still 25 times higher than normal.

A number of clinical and injury factors were found to be associated with PTE. As expected from prior studies, total brain volume loss on CT was significantly associated with PTE ($P = .0001$), as was the presence of hematoma ($P = .01$) or retained metal fragments ($P = .02$). However, tangential high-velocity gunshot wounds, retained bone fragments, use of a dural graft for closure, cranioplasty, and brain abscess showed no relationship to PTE. Similarly, preexisting factors such as family history of epilepsy or preinjury intelligence as measured by the Armed Forces Qualification Test had no impact on incidence of PTE. Among neurological outcomes, hemiparesis ($P = .03$), aphasia ($P = .009$), organic mental disorder (DSM III) ($P = .01$), visual field loss ($P = .01$), or headache ($P = .001$) were all associated with seizures, but traumatic loss of consciousness, either immediate or at first neurological examination, was not. Neither subsequent head injury, other encephalopathy, nor alcohol abuse played important roles in occurrence, particularly in late-onset cases.[8]

Motor Function

Forty-seven percent of our patients were recorded as having a paralysis early after injury, and about half of those have now recovered. Analysis of the clinical and anatomical correlates of recovery from hemiparesis has resulted in a simple initial model that may allow us to predict which patients will recover. Clinical findings significantly ($P \leq .05$) associated with nonrecovery were sensory loss, organic mental disorder, abnormal EEG, partial simple seizures, and an initial extensor plantar response. Anatomical correlates included large, total brain volume loss and involvement of the following anatomical structures on CT: sensory-motor cortex, supplementary motor area, posterior temporal cortex, temporal white matter, and the posterior limb of the internal capsule. Clinical and anatomical factors were then allowed to interact in a stepwise logistical regression model comparing unrecovered patients to those with delayed recovery (> 1 mo postinjury). Items significantly ($P < .05$) predicting recovery in this model were involvement, seen on CT scan, of (1) vertex or medial sensory motor cortex, (2) central corona radiata and caudate body, (3) extensor plantar response, and (4) sensory loss, in that order. Probability of recovery was .05 for patients with all items present and .97 when all were absent. This model was 82% accurate.[9] Most patients who are going to recover

motor functions will do so within the first 6 months after injury (15% recover within 1 mo), but a small percentage may not do so for several years.

Considerable ipsilateral as well as contralateral deficits in complex hand motor functions can be found in patients with lesions in the frontal and parieto-occipital lobes, even in the absence of an overt hemiparesis. This is most pronounced in patients with right hemisphere brain injuries and in right-handed individuals.[10] Preliminary analysis of our data also shows that the relation of persistent hemiparesis to eventual successful community adjustment is not direct and that other factors, primarily cognitive status, may play a more important role than paralysis per se. Ongoing follow-up studies will clarify this relationship. Further analyses of the pattern of motor recovery, the relation of paresis to language function, and the relation of spasticity to lesion location are also planned.

Traumatic Unconsciousness and Amnesia

Analysis of traumatic unconsciousness and amnesia in the VHIS casualty database showed that only 15% of the patients had prolonged unconsciousness and 53% had no or only momentary unconsciousness after injury, emphasizing the focal nature of these wounds. There was a clear dominance of the left (or language-dominant) hemisphere for the "wakefulness" or vigilance component of consciousness. The areas of the posterior limb of the left internal capsule, the left basal forebrain, midbrain, and hypothalamus were most associated with unconsciousness. Left dominance is not seen for posttraumatic amnesia after elimination of the "wakefulness" variable, suggesting that the latter may be linked to the well-recognized role of the left hemisphere in certain verbal memory processes.[11] This particular analysis illustrates another example of the functional asymmetry of the two halves of the brain and has also helped to sharpen the distinction between the two major aspects of the arousal component of consciousness: "wakefulness" (left hemisphere) and attention (right hemisphere).

Basal Forebrain Lesions and Cognition

The neurological and cognitive performance of 15 young veterans who suffered unilateral penetrating missile wounds of the basal forebrain was compared with that of patients without basal forebrain lesions and uninjured controls.[12] They did somewhat more poorly on tests of episodic memory, reasoning, and arithmetic, and had more prolonged unconsciousness than patients with lesions elsewhere in the brain. However, their performance on tests of intelligence, attention, and language was not consistent with that of demented patients. These results suggest that the basal forebrain may be a component of limbic-hippocampal memory processing systems, but it is not responsible for the maintenance of cognitive processing in general.

Electrophysiological Studies

The relationship between EEG findings and clinical and radiological features was studied in the first 300 VHIS subjects. EEGs were performed on 16- and 18-channel Grass equipment using the international 10–20 system. Fifty age-matched Vietnam veterans were used as controls. The EEG was abnormal in 48% of the patients. Epileptiform findings (EF; spikes or spikes wave) were found in 15% of the records and focal slowing (FS) in 38%. Of the patients with EF, 80% had one or more seizures after head injury, compared with 64% of patients with FS and 41% of patients with normal EEGs. Epileptiform findings were seen in 16% of patients who had had their initial seizures during the first year following head injury but in only 7% of those with onset after 5 years. EEG was normal in 31% of the former group and 71% of the latter. No correlation was found between EF and family history of epilepsy, seizure frequency in first year after injury, or seizure persistence. Both EF and FS correlated significantly with hemiparesis ($P = .0001$), aphasia ($P = .00074$), and CT scan revealed evidence of deep cerebral injury ($P = .0004$).[13]

Another analysis studied the relationship between visual evoked potentials (VEPs), perimetry, clinical, and CT findings of the first 150 patients in our study. Full field (FF) and half field (HF) responses were obtained via a television screen that delivered checkerboard-pattern reversal stimuli at the rate of 2.1 per second. Responses were recorded on four medial and lateral occipital electrodes simultaneously, placed 5 cm and 10 cm from the midline. Visual fields were obtained by Goldmann perimetry and CT scans by a General Electric 8800 scanner. Fifty age-matched Vietnam veterans served as controls. Fourteen patients (9%) showed a mono-ocular delay of VEP on the side of head injury. Seven of these patients had no visual complaints, suggesting that VEP detected a subclinical traumatic macular or optic nerve dysfunction. HF stimulation and perimetry produced concordant data in 88% of the patients. When abnormal, both tests correlated highly with a parieto-occipital site of injury. In six patients, abnormality of HF-VEP pointed correctly to the side of head injury but perimetry was normal; while in a few patients, perimetry showed small hemianopic field defects and HF-VEP missed them. These data indicate that HF-VEP is a sensitive measure of optic radiation dysfunction in penetrating head injury. Information derived from HF-VEP and perimetry complement each other in retrochiasmatic brain lesions.[14]

Audiology

Analysis of central auditory testing on 250 VHIS subjects was done, including correlations with CT evidence of damage to eight different regions of the temporal lobe. The location and degree of temporal lobe injury was compared with dichotic speech test results in an effort to establish auditory correlates of physical damage. Results indicate that speech test scores are significantly affected by injury site. In addition, three dichotic speech tests were administered to 300 individuals with brain injury in various locations. The sensitivity of each test was studied relative to the percentage of normal/abnormal scores for specific injury groups. A high rate of false-negative and false-positive results was present for all measures. The three dichotic tests did not vary substantially in their ability to detect damage to the right or left temporal lobes.

Separate analyses have also studied the relationship of lesion site to loss of perception of time-compressed speech, which has been reported to be a useful test in the identification and differentiation of central auditory deficits.[15]

Speech and Language

Initial analyses of the speech and language data have included studies of recovery from Broca's aphasia,[16] speech discrimination deficits,[17] and acquired stuttering.[18] The first was designed to determine which language faculties are retained in the chronic form of expressive aphasia, and what characteristics of brain lesions differentiated between patients who recovered and those who did not recover from expressive aphasia within 15 years following penetrating head injury. Two groups of men who sustained penetrating head injuries and had an expressive aphasia during the first 6 months following injury were examined 15 years later. One group had a chronic expressive nonfluent aphasia; the other had recovered and was without symptoms of aphasia. On a comprehensive battery of speech and language tests, the patients with chronic expressive aphasia demonstrated specific deficits in syntactic processing in all language modalities, while they were within the normal range in other language faculties. The recovered group demonstrated syntactic deficits only in written expressive syntax. The CT lesions of the two groups differed in the extent of left hemisphere lesion volume and the degree of posterior and deep lesion extension. Broca's area was equally involved in both groups but was not involved in all patients in either group. All the nonrecovered group had posterior extension of the lesion to involve Wernicke's area, with some involvement of the underlying white matter and basal ganglia in the left hemisphere.[16]

Speech discrimination and identification tasks assessing voicing and place distinctions were given to 16 unilaterally brain-injured subjects free of aphasic or dysarthric symptoms 12 to 15 years postinjury. Seven subjects did not demonstrate any difficulty with these speech tasks, while five subjects who had been injured on the left side of the brain and four who had been injured on the right showed moderate difficulties. These difficulties were more pronounced on the discrimination than on the identification tasks. Analysis of CT scans demonstrated that the lesion locations most clearly associated with the speech discrimination deficits were upper levels of the white matter subjacent to cortical regions in either hemisphere.[17] Other analyses of the VHIS database now underway will study recovery from Wernicke's aphasia and patients with dysprosody.

Neuropsychology

The Neuropsychology Section of the VHIS was developed to broadly address certain critical issues regarding brain–behavior relationships, the conceptual validity of specific cognitive theories, and the persistence of cognitive deficits and their effect on the clinical course of a patient. One unique characteristic of the VHIS population is the availability of preinjury intelligence testing in the Armed Forces Qualification Test (AFQT). The same test was then administered at follow-up to all patients. We assessed the impact of education, preinjury intelligence, brain volume loss, and lesion location on postinjury intelligence level.[19] We found that, in general, the most important determinant of postinjury intelligence was preinjury performance on the AFQT. One exception was seen in patients with severe left hemisphere lesions, which was not surprising given the linguistic processing demands of the AFQT. In addition, we discovered that the more global a cognitive process, the greater the effect of brain loss volume; that is, specific cognitive processes were affected relatively more by lesion location.[20] This methodological approach will continue to guide our research effort: distinguishing between effects on global versus specific cognitive processes and mood presentations, and their interactions, by considering both anatomical and behavioral variables.

Exploitation of the VHIS data has been proceeding in several separate areas: cognition, mood, injury characteristics, and functional and clinical outcome utilizing lesion location, brain loss volume, and preinjury intelligence as covariates. We believe that the initial studies in each area not only will contribute to the scientific and clinical literature but will also provide the basis for continuing analysis in the future. This continuing analysis is necessary to refine the models of brain–behavior relationships we have only barely begun to construct.

Cognitive Process

One example of a specific cognitive process is the ability to discriminate and recognize faces. Our analysis indicates that *both* hemispheres of the brain contribute to this process, with the left hemisphere storing face knowledge information and the right hemisphere storing procedures that allow for rapid face discrimination and form memory. Face recognition that requires transformation of features (eg, the person has to rely on specific facial features for recognition) seems to require the integrity of the frontal lobe.[21]

A second example of a specific cognitive process involves the semantic encoding of recently presented verbal information. We have tested an individual who presented with a restricted deficit in this process in contrast to superior skills in all other cognitive areas. We argue that of his brain lesions, the critical one for this cognitive process is the one in the columns of the fornix.[22]

Mood Presentation

We have taken a parallel course in examining the mood presentation of our patients. A particularly interesting area of investigation is the effects of frontal lobe lesions on the maintenance of control of anxiety, fear, and hostility. We have demonstrated the rather profound and persistent effects of orbitofrontal lesions on the modulation of feelings of anxiety, dorsofrontal lesions on feelings of sluggishness, and the acute effects of frontal lobe lesions in general on control of anger and hostility.[23] Patients with left dorsofrontal and right orbitofrontal lesions were most disinhibited, edgy, angry, and depressed. Ongoing studies are investigating single cases with limited orbitofrontal lesions, Beck Depression Inventory group profiles, Minnesota Multiphasic Personality Inventory group profiles, and factor analysis of the Bear-Fedio Trait Scales. Our purpose is to develop a rudimentary model of mood state representation and to discover how mood state interacts with cognitive processes.

Although for many years anecdotal reports have linked violent and aggressive behavior to frontal lobe injury, the VHIS gives us an opportunity to examine this issue in a large cohort of well-characterized survivors with injuries to the frontal lobe and other parts of the brain. Using factor analysis and other statistical techniques, VHIS psychosocial and other data were used to generate two indices of violence or aggression in a cohort of 336 subjects and controls. These were then correlated with anatomical lesion location. Results indicate that it is ventromedial frontal lesions that correlate with violence and aggression, which, in this cohort, was usually manifested verbally in family situations.[24]

Psychosocial Outcome

A final analysis scheme addresses the impact of penetrating brain wounds on ultimate functional psychosocial outcome, the major attribute, for purposes of this study, being the subject's ability to return to work. Earlier analyses in Korean War veterans with head injuries indicated that a considerable proportion of such men were able to return to gainful employment. We have investigated the effects of residual impairments resulting from head injury on the work status of the VHIS population as well. Extensive standardized testing of neurological and neuropsychological and social functioning was done some 15 years postinjury on the VHIS subjects (N = 520), as well as on the uninjured controls (N = 85). Fifty-six percent of the head-injured subjects were working at follow-up, compared with 82% of the uninjured controls. Up to 80% of the subjects with head injuries reported having worked at some time postinjury. Furthermore, the occupational distribution of our working, head-injured veterans does not differ significantly from that of the uninjured controls or the age-matched population of the United States.[25] After excluding several patients with severe triplegia or global aphasia, none of whom worked, we investigated the relationship of selected neurological, neuropsychological, and social impairments to the ability to return to work, using factor analysis and multiple logistic regression.

In the final model, we identified seven systematically defined impairments that were significantly related to the ability to return to work. These were posttraumatic epilepsy, hemiparesis, visual field loss, verbal memory loss, visual memory loss, psychological problems (anxiety and depression), and violent behavior. These disabilities had a cumulative and nearly equipotent effect on the likelihood of returning to work. A simple summed score of the number of these seven disabilities can yield a residual "disability score," which may prove to be a practical tool for assessing the likelihood that patients in the VHIS population, and perhaps in other brain-injured populations, will return to work. Patients with up to any three of these impairments had a 65% or better likelihood of returning to work. However, the presence of five or more impairments was associated with only a 20% likelihood of returning to work. These findings may also help to focus rehabilitation efforts on those disabilities most likely to affect the ability to return to work.[26]

SUMMARY

The VHIS contains a rich database on a well-defined group of casualties with penetrating head injuries. This ongoing study will continue to provide material for analysis for years. While many of the questions posed in the

original planning for the study have already been answered, new questions have arisen and will continue to arise as investigators explore the data. The VHIS evaluation has also helped us identify subsets of patients with specific types of wounds or deficits who can be invited to return for more detailed experimental testing that concentrates on their specific disabilities, or on hypothesized functions of the brain areas involved in their injuries. Many of the questions have immediate practical implications for prediction of outcome, therapy, and determination of disability status. However, perhaps the most valuable aspect of the study will be the long-term benefits resulting from a better scientific understanding of brain function and its localization.

REFERENCES

1. Caveness WF, Meirowsky AM, Rish BL, et al. The nature of posttraumatic epilepsy. *J Neurosurg*. 1979;50:545–553.

2. Rish BL, Caveness WF, Dillon JD, Kistler JP, Mohr JP, Weiss GH. Analysis of brain abscess after penetrating craniocerebral injuries in Vietnam. *Neurosurgery*. 1981;9(5):535–541.

3. Rish BL, Dillon JD, Meirowsky AM, et al. Cranioplasty: A review of 1,030 cases of penetrating head injury. *Neurosurgery*. 1979;4(5):381–385.

4. Mohr JP, Weiss GH, Caveness WF, et al. Language and motor disorders after penetrating head injury in Vietnam. *Neurology*. 1980;30(12):1273–1279.

5. Rish BL, Dillon JD, Weiss GH. Mortality following penetrating craniocerebral injuries: An analysis of the deaths in the Vietnam Head Injury Registry population. *J Neurosurg*. 1983;59:775–780.

6. Sweeny JK, Smutok MA. Preliminary analysis of the functional and anatomical sequelae of penetrating head trauma. *Phys Ther*. 1983;63(12):2018–2025.

7. Myers PW, Brophy J, Salazar AM, Jonas B. Retained bone fragments after penetrating brain wounds: Long term follow-up in Vietnam veterans. *J Neurosurg.* 1989;70:319A. Abstract.

8. Salazar AM, Jabbari B, Vance SC, Grafman J, Amin D, Dillon JD. Epilepsy after penetrating head injury, I: Clinical correlates: A report of the Vietnam Head Injury Study. *Neurology*. 1985;35(10):1406–1414.

9. Smutok MA, Vance SC, Salazar AM, Foulkes M, Grafman J. Neurologic and anatomic correlates of recovery from hemiparesis following penetrating head injury. *Neurology.* 1985;35(1):83.

10. Smutok MA, Grafman J, Salazar AM, Sweeney JK, DiRocco PJ. The effects of unilateral brain damage on contralateral and ipsilateral upper extremity function in hemiplegia. *Phys Ther*. 1989;69:195–203.

11. Salazar AM, Grafman J, Vance SC, Weingartner H, Dillon JD, Ludlow CL. Consciousness and amnesia after penetrating head injury: Neurology and anatomy. *Neurology*. 1986;36:178–187.

12. Salazar AM, Grafman J, Schlesselman S, et al. Penetrating war injuries of the basal forebrain: Neurology and cognition. *Neurology*. 1986;36(4):459–465.

13. Jabbari B, Vengrow MI, Salazar AM, Harper MG, Smutok MA, Amin D. Clinical and radiological correlates of EEG in the late phase of head injury: A study of 515 Vietnam veterans. *Electroencephalogr Clin Neurophysiol.* 1986;64:285–293.

14. Jabbari B, Vance SC, Harper MG, Salazar AM, Smutok AM, Amin D. Clinical and radiological correlates of somatosensory evoked potentials in the late phase of head injury: A study of 500 Vietnam veterans. *The EEG Journal*. 1987;67:289–297.

15. Mueller HG, Sedge RK, Salazar AM. Central auditory nervous system dysfunction: Preliminary findings of the Vietnam Head Injury Study. In: Miner ME, Wagner KA, eds. *Neurotrauma: Treatment, Rehabilitation and Related Issues*. Boston, Mass: Butterworth; 1985: 155–188.

16. Ludlow C, Rosenberg J, Fair C, Buck D, Schlesselman S, Salazar AM. Brain lesions associated with nonfluent aphasia 15 years following penetrating head injury. *Brain*. 1986;109:55–80.

17. Yeni-Komshian GH, Ludlow CL, Rosenberg J, Fair C, Salazar AM. Lesion locations associated with speech perception deficits following penetrating head injury. *Neuropsychologia*. 1986;24:631–647.

18. Ludlow CL, Rosenberg J, Salazar A, Grafman J, Smutok M. Site of penetrating brain lesions causing chronic acquired stuttering. *Ann Neurol*. 1987;22:60–66.

19. Grafman J, Jonas BS, Martin A, et al. Intellectual function following penetrating head injuries in Vietnam veterans. *Brain*. 1988;111:169–184.

20. Grafman J, Salazar AM, Weingartner H, Vance SC, Amin D. The relationship of brain-tissue loss volume and lesion location to cognitive deficit. *J Neurosci*. 1986;6:301–307.

21. Grafman J, Salazar AM, Weingartner H, Amin D. Face memory and discrimination: An analysis of the persistent effects of penetrating brain wounds. *Int J Neurosci*. 1986;29:125–139.

22. Grafman J, Salazar AM, Weingartner H, Vance SC, Ludlow CL. Isolated impairment of memory following a penetrating lesion of the fornix cerebri. *Arch Neurol*. 1985;42:1162–1168.

23. Grafman J, Vance SC, Weingartner H, Salazar AM, Amin D. The effects of lateralized frontal lesions upon mood regulation. *Brain*. 1986;109:1127–1148.

24. Grafman J, Schwab K, Warden D, Pridgen A, Brown HR, Salazar AM. Frontal lobe injuries, violence, and aggression: A report of the Vietnam Head Injury Study. *Neurology*. 1996;46:1231–1238.

25. Kraft JF, Schwab KA, Salazar AM, Brown HR. Occupational and educational achievements of head injured Vietnam veterans at 15-year follow-up. *Arch Phys Med Rehabil*. 1993;74:596–601.

26. Schwab K, Grafman J, Salazar AM, Kraft J. Residual impairments and work status 15 years after penetrating head injury: Report from the Vietnam head injury study. *Neurology*. 1993;43:95–103.