Women with Traumatic Brain Injury

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> *"Every year, it is estimated that 1.9 million Americans sustain a traumatic brain injury (TBI) with approximately 475,000-570,000 (25-30%) of those injuries occurring to women (Forkosch et al, 1996; Ommaya et al, 1996; Rothweiler et al, 1998; Sandercock, 1989). Between 50-75% of these injuries are mild in severity and complications tend to resolve within a few months (Annegers et al, 1980; Klauber et al, 1981; Kraus et al, 1984; Jagger et al, 1984; Rimel, 1981; MacKenzie et al, 1990; Whitman et al, 1984); however, 10-25% of these injuries are severe enough to necessitate a hospital stay and produce some form of long term disability. The direct medical costs per year incurred by people with TBI has been estimated to be at least $4 billion (Forkosch et al, 1996; Kalsbeek et al, 1980). Total annual costs have been estimated to be $25 billion, prompting statements that TBI is "the most important public health issue facing America today."*
>
> *(Genarelli, 1988).*

Estimates of incidence traumatic brain injury range from 125 to 800 per 100,000 individuals (Annegers et al, 1980; Cooper et al, 1983; Forkosch et al, 1996; Fife et al, 1986; Jagger et al, 1984; Klauber et al, 1978; Krause et al, 1984; MacKenzie et al, 1990; Rimel, 1981; Whitman et al, 1984) while prevalence estimates vary from 27 to 100 per 100,000 (Bryden, 1988; Furrie, 1987; Willer et al, 1990). Incidence rates in particular are influenced by factors such as age, ethnicity, and gender. There is a general consensus that incidence rates peak among teenagers and young adults, decline through the adult years, and exhibit a second rise after age 60 (Cooper et al, 1983; Jagger et al, 1984; Klauber et al, 1981; Krause et al, 1984; MacKenzie et al, 1990). Incidence rates among different ethnic groups show individualized patterns. For example, in urban Chicago, African-Americans had an almost 2-fold higher incidence rate of TBI than Caucasians (Whitman et al, 1984) and in rural Virginia, minorities had a 1.5-fold higher TBI incidence rate than Caucasians (Jagger et al, 1984).

INCIDENCE AND PREVALENCE

The primary causes of TBI have remained relatively stable over time. Approximately 30-50% of all injuries involve a motor vehicle, as driver, passenger or pedestrian (Annegers et al, 1980; Forkosch et al, 1996; Klauber et al, 1981; Kraus et al, 1984; Jagger et al, 1984; Whitman et al, 1984). The second most common cause of injury is falls, followed by recreational activities. However, the incidence of TBI due to violence and firearms appears to be on the increase, particularly in metropolitan areas (Cooper et al, 1983; Forkosch et al, 1996; Whitman et al, 1984; TBI Model Systems National Data Center, 1999).

EPIDEMIOLOGY OF TBI IN WOMEN

Studies that have analyzed gender differences in the epidemiology of TBI find significant differences between males and females. Incidence rates for females range from a low of 126/100,000 to a high of 425/100,000 compared with rates for males which have been estimated to range from 180 to 680/100,000 (Kalsbeek et al, 1980; Klauber et al, 1981; MacKenzie et al, 1990; Ommaya et al, 1996; Sorenson and Kraus, 1991). The male: female incidence ratio ranges from 1.8 to 2.9. (Ommaya et al, 1996; Rothweiler et al, 1998; Sandercock, 1989). Prevalence rates also follow the same pattern with approximately twice as many males with TBI in the population than women (Kraus, 1980; Kraus, 1991; Moscato et al, 1994).

Age also has an effect on the incidence and prevalence rates for women. In one study in Canada, women were slightly older on average, 42 +19 years, than men, 37 +16 years (Wong et al, 1993). Most studies that examine the effect of age on TBI incidence and prevalence and that also separate male and female rates, report that the same major peak for TBI is observed between 15 to 24 years of age (Katz and Alexander, 1994; Sorenson and Kraus, 1991; Wong et al, 1993). In fact, when examining the age distribution of TBI by gender, females tend to show a similar peak-and-valley pattern to males but with the magnitude of the rates for females approximately half that of the males (Sorenson and Kraus, 1991). However, there have been multiple reports indicating that the incidence of TBI rises almost two-fold for females after the age of 60 (Englander and Cifu, 1999; Katz and Alexander, 1994; Wong et al, 1993).

Etiology of injury also shows gender differences. While the most common causes of TBI - motor vehicle crashes, falls, assaults, and sports – are the same in males and females, the relative proportion of individuals injured by each cause is different. Males are far more likely to be injured while fighting or engaging in sporting activities while females are more likely to be injured in motor vehicle crashes or falls (Ommaya et al, 1996; Wong et al, 1993; Klauber et al, 1981).

The severity of injury also differs between males and females as indicated by several measures. One study looked at the number of fatalities per 100,000 cases and found that males had a significantly higher case fatality rate, 31.8, than females at 12.1 (Klauber et al, 1981). Two indicators of severity of injury, duration of coma and length of rehabilitation stay, were found to be significantly longer in men than women (Spettell et al, 1991). Complicating factors that could contribute to the apparent greater severity of injury for males are the increased association of the injury with alcohol and drug use – three-fold higher for alcohol and two-fold higher for drug - as well as a history of previous TBI (Wong et al, 1993).

CLASSIFICATION OF TBI

Classification of TBI usually refers to the acute injury and secondary complications. The Glasgow Coma Score (GCS) is the most widely accepted measurement of initial injury severity by emergency medical systems (EMS) and departments (Jennett and Bond, 1975). It combines the best response obtainable in the evaluation of verbal, eye and motor exams (Table 1). GCS scores of 3-8 are indicative of "severe" injury; GCS 9-12 of "moderate" injury; and GCS 13-15 of "mild or minor" injury. However, this is an overly simplified classification system as individuals may have a GCS of 14-15 with a large focal injury from a gunshot wound, intracerebral, subdural or epidural hematoma, requiring emergency evacuation. Conversely, GCS may be artificially depressed secondary to drug or alcohol intoxication. Therefore it is crucial to ascertain other characteristics of injury, including concomitant arterial hypotension, hypoxemia, hypercapnea, anemia, hyperpyrexia, hypoglycemia, hyponatremia, raised intracranial pressure, intracranial lesions, seizures, and vasospasm (Pentland and Wittle, 1999).

Given the anatomy of the inner skull tables, there is a predilection for focal injury to occur to the inferior and dorsolateral surfaces of the frontal lobes and the medial surfaces of the temporal lobes, regardless of the location of external injury. This occurs when the cortical surfaces impact upon bony ridges inside the skull. In addition long axons are vulnerable to shearing or diffuse axonal injury (DAI) from sudden deceleration of the body/head during vehicle crashes, falls, and pedestrian or cycle collisions.

Quantification of the impact of the amount and type of brain injury is crucial to understanding the effects of the cumulative pathology on the individual. Given the fact that brain injuries have the potential to affect personality, behavior, cognition, motor and sensory functions, it is very important to obtain accurate information about the individual's pre-injury level of functioning in all of the above areas. This often requires inquiry of spouses, children, friends, and co-workers, as the person with the TBI may not be aware of "changes" in capabilities because of their injury.

Table 1. Glasgow Coma Scale (Jennett and Bond, 1975)

Category	Response	Score
Eye Opening	Spontaneous	4
	To Voice	3
	To Pain	2
	None	1
Best Verbal Response	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Best Motor Response	Obeys command	6
	Localizes pain	5
	Withdraws (pain)	4
	Flexion (pain)	3
	Extension (pain)	2
	None	1

LANDMARKS IN RECOVERY FROM TBI: PROGRESSION THROUGH LEVELS OF CARE

The circumstances of the injury help to put into perspective the nature of the person's lifestyle, propensity towards risk-taking behavior and the attitude of the person and their support system as it relates to the injury itself. The first step in assessing the potential for recovery is to determine the injury severity from the patient, observers at the scene and acute medical data.

The initial GCS as measured by EMS and emergency department personnel as well as the need for resuscitation of breathing and circulation are the most important and easily obtainable, objective measurements of injury severity. Concomitant injuries to the spinal column, internal organs, limbs, and sensory systems should also be delineated.

Injury pathology may also appear on CT or MRI scans. CT scans are most helpful in the first several weeks post-TBI to ascertain mass lesions that require neurosurgical intervention and bony fractures of the skull and auditory canals. MRI exams are more sensitive for punctate hemorrhages, suggestive of DAI, such as cortical and brainstem contusions adjacent to bony structures that may be inadequately imaged by CT due to artifact. However, both CT and MRI scans may show minimal or no anatomical lesion if the injury is caused by microscopic DAI, without gross hemorrhage, or hypoxia, even if the injury is severe. Later imaging, more than 6-8 weeks post-injury, may or may not show atrophy reflective of DAI or hypoxia. Either imaging method is effective in detecting hydrocephalus (see below). Newer imaging methods, such as single photon emission computed tomography (SPECT) imaging and positron emission tomography (PET) scans, are more physiologic measures of brain functioning. They are the most sensitive tests but also the least studied with regard to quantification of injury (Katz and Black, 1999).

Early focal neurologic signs such as unilateral dilated pupil or absent pupillary reflexes in a non-intoxicated patient, decorticate or decerebrate posturing, hemiparesis, gaze preference, or seizures are helpful localizing signs which are all indicative of more severe injury (Jennett and Teasdale, 1981; Pentland and Black, 1999; Plum and Posner, 1980).

Other landmarks of recovery include the following:

- Length of coma is usually measured by the time to follow commands (GCS motor = 6), time to initiate willful movements (GCS motor=5), or attempt to verbalize (GCS verbal = 3). Length of coma correlates with survival, length of posttramatic amnesia (PTA), and many functional outcomes. See Table 1.
- Spontaneous eye opening may indicate emergence from coma to a vegetative state and will occur within 3-4 weeks of injury. Unless eye signals can be reliably correlated with yes/no responses, they are difficult to correlate with meaningful interaction with the environment. Those who do not emerge from coma by "following commands" may remain in a vegetative state for variable periods of time.
- Length of posttraumatic confusion or posttraumatic amnesia (PTA) is the next important recovery point. Prospectively, it can be measured when the person is consistently oriented or by the Galveston Orientation and Amnesia Test (GOAT, see insert), with two consecutive scores greater than 75/100 within 24 hours.

Retrospectively, PTA can be estimated by the patient's independent recall of specific events post-injury and demonstration of continuous memory from that point forward. Length of PTA is strongly correlated with an individual's ability to return to previous vocational and household responsibilities (Levin et al, 1979). See insert.

- Recovery rate, particularly over the first 3-6 months post-TBI is probably the most helpful predictor of further recovery. Individuals with DAI have a typical sequence of recovery as described in the Rancho Los Amigos Levels of Cognitive Functioning (Table 2; Hagen et al, 1972). The length of time an individual spends in each phase is variable, and some individuals' recovery may "get stuck" in one stage for weeks-months. However, most individuals experience ongoing improvement for up to 2 years post injury.

Rehabilitation from moderate to severe TBI starts within the first few days post-TBI, even in the ICU. In this setting the focus is on positioning to prevent contractures, improving alertness, establishing a reliable method of communication, evaluating swallowing capabilities so that nutritional delivery can be planned, starting bowel and bladder routines, and helping manage behavioral issues such as agitation or decreased initiation. With mild TBI, education is helpful, particularly with written brochures (Brain Injury Association, NHIF) so that patients and caregivers can have reasonable expectations for their course of recovery.

Once the acute medical and surgical problems can be managed in another level of care, patients are usually discharged from the trauma center (see Table 2). Medication interventions should be targeted at specific symptoms such as sleep disturbances, agitation, arousal, and emotional liability. Discontinuation or tapering of medications used during acute care that have cognitive and behavioral side effects, e.g., steroids, benzodiazepines, anti-psychotics, sedating analgesics, is usually more effective than adding new medications (Englander and Cifu, 1999; Zafonte et al, 1999).

Those who are not confused but with remaining cognitive or physical impairments (Rancho 7-8) may be managed in acute inpatient rehabilitation, day or residential treatment, or even home settings with "home and community" programs. Noting the type, intensity and success of rehabilitation interventions during the immediate and post-acute care periods will be helpful in determining ongoing rehabilitation benefit.

Table 2. Rancho Los Amigos Levels of Cognitive Functioning Scale (Hagen et al, 1972).

Level	Response	Description of Behavior	Rehabilitation Setting
I	None	-unresponsive to all stimuli	Subacute
II	Generalized	-inconsistent, nonpurposeful, non-specific reaction to stimuli -responds to pain, but may be delayed	Subacute
III	Localized	-inconsistent reaction related to stimuli -responds to some commands -may respond to discomfort	Subacute/Acute Rehabilitation
IV	Confused, agitated	-disoriented, unaware of present events -frequent bizarre and inappropriate behavior -short attention span, impaired ability to process information	Acute Rehabilitation
V	Confused, inappropriate, non-agitated	-nonpurposeful, random or fragmented responses when task exceeds abilities -appears alert and responds to simple commands -performs previously learned tasks, cannot learn new ones	Acute Rehabilitation
VI	Confused, appropriate	-goal-directed behavior -appropriate responses to situation -incorrect responses due to memory difficulties	Acute Rehabilitation
VII	Automatic, appropriate	-correct routine responses which are robotlike -appears oriented to setting -insight, judgment, problem-solving are poor	Acute Rehabilitation/ Transitional Living/ Outpatient or Day Treatment
VIII	Purposeful, appropriate	-purposeful, correct responding -carryover of new learning -no supervision required -poor tolerance for stress -some abstract reasoning difficulties	Transitional Living/Outpatient or Day Treatment

GOAT Insert

Galveston Orientation & Amnesia Test (GOAT)

Error Points

1) What is your name? (2)________________ Where were you born? (4)___________ [__]
Where do you live? (4)____________________

2) Where are you now? (5) city_______________ (5) hospital____________________ [__]
(unnecessary to state name of hospital)

3) On what date were you admitted to this hospital? (5)________________________ [__]
How did you get here? (5)____

4) What is the first event you can remember after the injury? (5) ________________ [__]
Can you describe in detail (e.g., date, time, companions) the first event you can recall after injury? (5)
____

49. Can you describe the last event you recall before the accident? (5)_____________ [__]
Can you describe in detail (e.g., date, time, companions) the first event you can recall before the injury? (5)
____

6) What time is it now?______________(1 for each 1/2 hour removed from correct time to maximum of 5) [__]

7) What day of the week is it?_____________________(1 for each day removed from correct one) [__]

8) What day of the month is it?____________(1 for each day removed from correct date to maximum of 5) [__]

9) What is the month?________________(5 for each month removed from correct one to maximum of 15) [__]

10) What is the year?___________________(10 for each year removed from correct one to maximum of 30) [__]

Total Error Points [________]

Total Goat Score (100 total error points)[________]

EXAMINATION

In conducting routine medical exams, the practitioner needs to be aware that recovery from TBI can be a protracted process with gains continuing to be noted over a period of years. Thus the routine medical exam should continue to assess mental and physical status in the context of the TBI in addition to the standard primary health considerations for all patients.

Mental Status

The mental status examination (MSE) is the most important portion of the general medical and neurologic exams of the individual with TBI. Alertness, attention, orientation, language, memory, perceptual skills, and executive functioning are the core areas to explore. Items from the initial examination that are challenging to the individual with TBI should be repeated at subsequent examinations to ascertain progress. If the individual still experiences post-traumatic amnesia, the GOAT or modified MSE can be used (Folstein et al, 1975; Levin et al, 1979). When PTA has resolved, other tests are more appropriate. Some of these tests are quick and easy to perform: the amount of time to perform serial subtractions (100-7 or 31-3 or reciting the months backwards); giving a ten word list and recording the order and number of words correct at each of 3-4 trials, followed in 5 minutes by delayed recall; and the number of animals or foods named in 60 seconds. Other examples of the MSE are available in the literature (Katz and Black, 1999; Strub and Black, 1993).

Cranial Nerves

Cranial nerve deficits are common and can occur to peripheral or central components. Cranial nerve I is not routinely tested by most physicians, yet it may be the most common nerve affected in TBI. Some practitioners prefer to use commonly available substances like toothpaste or coffee; others prefer "scratch and sniff" methods (Sensonics Ltd.). Deficits have implications for personal hygiene and safety around smoke or chemicals; 50-80% of these deficits are still present 3 months post-TBI. Cranial nerve II can be injured with orbital fractures, occipital contusion or infarction and should be evaluated with pupillary response, visual field and acuity testing. Cranial nerves III, IV, and VI present with difficulty reading, diplopia, unilateral squinting, or tilting of the head. They often resolve spontaneously over the first year post-TBI. Neuroophthalmalogical consultation is helpful in quantifying the extent of visual pathology and in planning interventions. Patching of one eye to decrease symptoms of diplopia is safe and results in no loss of vision if the individual is more than 6 years old (Leigh and Zee, 1991).

Cranial nerve V is most commonly injured peripherally with facial fractures or carotid-cavernous fistulas and usually recovers. Injury of cranial nerve VII can result in incomplete eye closure, hyperacusis, loss of taste, poor oral control of secretions, and facial asymmetry. Damage to cranial nerve VIII can result in tinnitus, hearing loss, and/or vertigo with or without nystagmus. Individuals are often more symptomatic with partial injury to the vestibular system than with very severe, complete injury.

Damage to cranial nerves IX, X, and/or XII may result in dysphagia with aspiration of secretions or food, dysarthria, or dysphonia. The gag reflex is not a sufficient indicator of one's ability to swallow safely; swallowing usually requires evaluation by a speech and language, occupational, or physical therapist trained to perform these maneuvers. Videofluoroscopy of swallowing allows trials of various food consistencies with visualization of the swallowing mechanisms to ascertain deficits in different swallowing stages and aspiration potential. Injury to cranial nerve XI is uncommon in TBI.

Motor Deficits

Motor examination in mild TBI is often "normal" until the individual is challenged to stand on one leg or asked to perform tandem gait backwards. Those individuals with more severe injuries can have alterations in power, tone, coordination, isolated movement, and movement disorders. The impact of the motor disorder on function will determine the extent of medical therapeutic and physical/occupational therapy interventions that are necessary.

Rigidity and bradykinesia may respond to antiparkinsonian medications. Tremors and myoclonus can interfere with upper extremity motor activities and gait. They may respond to beta-blockers, valproic acid, carbamazepine, and/or clonazepam. Maximizing the tolerated dose of one medication should occur before adding a second or third one, but these conditions often require polypharmacy.

Spasticity in local muscle groups is best managed by nerve or motor point blocks in conjunction with serial casting. Phenol can be safely used in nerves with minimal sensory components, for example, the musculocutaneous nerve for elbow flexor spasticity or obturator nerve for adductor spasticity. The effectiveness is generally known within 1-2 weeks and repeated injections may be done safely within that time frame. Botulinum toxin is more useful for smaller muscles innervated by mixed nerves (wrist and finger flexor muscles, median nerve); up to 400 units can be used every 12 weeks (Mayer et al, 1999; Yablon, 1995). Generalized spasticity may respond to systemic medication such as dantrolene, tizanidine, baclofen, or diazepam; these agents all have side effects that may be undesirable in individuals with systemic weakness (dantrolene) or cognitive deficits (tizanidine, baclofen, diazepam).

Some Useful Medications for Motor Deficits after Brain Injury

Medication	Utility	Dose & Monitoring
Sinemet	rigidity, bradykinesia	25/100 with meals and gradually increase
Propranolol	action tremor	20 mg tid and gradually increase
Valproic acid	action tremor	10 mg/kg/day in divided dose
Carbamazepine	action tremor	100-200 mg bid-tid, gradual increase
Clonazepam	action tremor	0.5 mg bid, gradual increase
Phenol	spasticity	local injection guided by nerve stimulator
Botulinum toxin A	spasticity	local injection, up to 400 units/12 weeks
Dantrolene	spasticity	25 mg tid, gradual increase to 400 mg/day Monitor LFTs
Tizanidine	spasticity	2 mg qhs, gradual increase to 36 mg/day Monitor LFTs
Baclofen	spasticity	5 mg tid, gradual increase to 80 mg/day
Diazepam	spasticity	2 mg bid, gradual increase, addictive

Peripheral Nerve Injury

Brachial plexopathies, usually incomplete and of the upper trunk are associated with clavicular and upper extremity fractures. Lumbosacral plexopathies are associated with pelvic fractures. Individual mononeuropathies, most commonly peroneal and ulnar nerves, are associated with long bone fractures or with prolonged bedrest with inadvertent pressure. Quantification of severity, specific localization, and localization of such injuries is determined by electromyographic and nerve conduction studies.

MEDICAL ISSUES

Women have a constellation of health concerns that are unique from those of men. Physical problems may arise directly from the brain injury itself or secondary to the changes that occur in the body as a result of the TBI. Women with TBI more frequently report weight changes, changes in the texture/growth of hair or skin, body temperature changes, headaches and frequent colds (Hibbard et al, 1998). Reports of thyroid conditions, sleep difficulties, loss of urinary control, and arthritis are also reported more frequently by women, although these conditions are linked to increasing age as well (Hibbard et al, 1998). Management of these conditions is often more difficult because of inaccurate history and subsequent poor compliance with recommended interventions. Because of changes in cognition, medication use on a scheduled program is likely to be followed more accurately than "prn" basis. The following sections provide a more detailed description of specific health issues for individuals with TBI.

Post Concussion Syndrome

Management of the individual with post concussion symptoms can be a literal headache for the individual with TBI, their family and friends, and for medical practitioners. Besides headaches, common symptoms are changes in mood, behavior, sleep, fatigue, and dizziness. It is crucial to ascertain the actual instance of a brain injury versus a mere head collision. Head collisions can result in headaches and dizziness with injury to the labyrinth without alteration in consciousness or awareness that would occur with a concussion. Such a symptom complex, when recognized, will require a prolonged appointment for complete evaluation. If the individual is scheduled for a short visit, use that time to obtain an initial history with delineation of where additional information and records must be secured and have the patient start a quantitative record of the most bothersome symptoms, e.g., a headache chart or sleep chart on a calendar, so both the patient and practitioner will have some quantification of symptoms with which to measure future interventions. As with many chronic conditions that have multiple somatic complaints, there is a frequent association with previous traumas that were incompletely resolved, e.g., prior head injury, earlier or ongoing physical or sexual abuse, rape, time as prisoner of war/torture, even witnessing a traumatic event, etc.

Once the diagnosis is confirmed, treatment must focus on the "resolvable" symptoms. Targeting medications to these symptoms, especially chronic sleep disturbances with trazodone or irritability with a mood stabilizer (valproic acid, carbamazepine, or selective serotonin reuptake inhibitor) can be helpful. Dizziness, if true vertigo, is usually positional in nature and can be brought out by asking the patient to move their head or eyes rapidly from side to side. Quick sitting from supine or sidelying positions may also elicit symptoms. Medications are usually not helpful for this condition. Physical therapy programs for dizziness habituation can be helpful if the individual is willing to tolerate some vertiginous symptoms in the process (Brant and Daroff, 1980; Shumway-Cook and Horak, 1990). Tinnitus is a bothersome symptom which may indicate hearing deficits. If the hearing deficit is not correctable, distraction with more pleasant sounds (music) is sometimes helpful. Gabapentin, carbamazepine, and low dose tricyclic antidepressants have been reported to help some individuals.

Seizures

Post traumatic seizures (PTS) are classified as early (0-7 days post-TBI) or late (> 7 days post-TBI). Early seizures occur in 5-10% of individuals with TBI. They are usually benign and associated with late PTS 25-40% of the time. Late PTS occurs in 9% of women and 19% of men post-TBI. They are most commonly associated with multiple or bilateral intracerebral hematomas (33%), a shift of > 5mm on early CT scans (40%), and subdural hematomas requiring surgical evacuation (39%). Any kind of seizure may occur after TBI. Generalized and focal seizures are more easily diagnosed than complex partial seizures and are therefore reported to be more prevalent in individuals with TBI (Annegers et al, 1995; Englander et al, submitted for publication).

Individuals with TBI and their caregivers should be educated on risk factors that can lower the seizure threshold:

1. substance use, especially cannabis and alcohol withdrawal, stimulant use;
2. high fever;
3. some medications, including tricyclic antidepressants, buproprion, amantadine;
4. pregnancy (Morell, 1995).

They should also learn first aid for a seizure event.

After the first PTS, a medical evaluation is warranted. If a recurrent intracranial lesion is suspected (because the individual fell and re-injured her head) or if the individual does not regain consciousness within 3-4 minutes, emergency department evaluation is probably the most efficient. Otherwise, the patient should be seen in the office that day to assess for aggravating factors. A drug screen and metabolic work-up will probably yield more than an imaging study, unless a recurrent bleed is suspected.

Once the diagnosis of a late PTS is established, there is an 85% likelihood of future seizures (Haltiner et al, 1997). However, status epilepticus is rare as a PTS event, so it is worth making an accurate diagnosis. Electroencephalograpy (EEG) is not particularly helpful in confirming the presence of seizures since studies in individuals with TBI usually show some abnormalities, e.g. focal slowing, maybe even periodic lateralizing epileptiform discharges (PLEDs). Twenty-four hour EEG monitoring may be useful in differentiating pseudoseizures from true seizures and should be done in consultation with an epileptologist. Unless the clinical history is convincing, one should be cautious about initiating treatment that may last years if not a lifetime (Yablon, 1993).

Seizure Prophylaxis

Guidelines from the American Academy of Neurological Surgeons and the American Academy of Physical Medicine and Rehabilitation recommend that phenytoin, phenobarbital, or carbamazepine may be helpful during the first week post-TBI to prevent early PTS; anti-epileptic drug use beyond the first week has not been shown to prevent late PTS. (Brain Injury Special Interest Group, AAPM&R, 1998; Guidelines for the Management of Severe Head Injury, 1995).

Anti-epileptic Drugs (AEDs) — In treating late PTS, an individual therapeutic decision weighing the benefits and risks of anticonvulsant treatment is highly recommended, due to the need for good compliance with medication use. Given the low incidence of status epilepticus with PTS in the absence of other metabolic abnormalities, medications usually can be started slowly to build up therapeutic levels and avoid side effects.

Medication	Dose	Monitoring	Side Effects
P-450 enzyme			
phenyto CYP1A2in CYP3A4/5	15-20 mg/kg load LFTs, 300-400 mg/day	CBC q 3-6 mo level 10-20	facial hair, gingival hyperplasia slowed cognition, multiple interactions
carbamazepine	100-200 mg tid	CBC q mo x 3	ataxia if too quickly increased,
CYP3A4/5	increase q 2-3 days	then q 3-6 mo level for compliance	helps with behavioral dyscontrol and dysesthetic pain
valproic acid (VPA)	10 mg/kg/day load up to 20 mg/kg/day	CBC, LFTs q mo x 3 then q 3-6 mo level 50-110	helps with behavioral dyscontrol, mood stabilizer, weight gain, some interactions
phenobarbital CYP3A4/5 CYP2CC9/10 CYP1A2	2-4 mg/kg/day	level 15-40	lethargy, cognitive deficits, multiple interactions
gabapentin	300 mg/day up to 3600 mg/day	none	lethargy, ataxia, good additive drug (no interactions)
lamotrigine	25 mg/day with VPA up to 150 mg/day 50 mg/day with others up to 700 mg/day	none	headache, tremor, ataxia, weight gain additive drug
topiramate	50 mg/day up to 800 mg/day	none	lethargy, ataxia, anorexia, nephrolitiasis, min interactions

Any of the above AEDs can cause anticonvulsant hypersensitivity syndrome, a severe skin desquammation with pyrexia, and often bone marrow and other organ failure. There is some controversy about the likelihood that other AEDs may cause cross reactivity in an individual with anticonvulsant hypersensitivity syndrome (Morkunas and Miller, 1997).

Gabapentin and topiramate can be useful as additive drugs for difficult to control seizures because they are metabolized by the kidney and do not alter the primary drug's level.

Anti-epileptic Drugs and Pregnancy — Fetal exposure to anticonvulsants results in a 4-6% incidence of major congenital malformations compared to 2-4% without such exposure. Midline facial abnormalities, cardiac defects and digital anomalies are the most common. Valproic acid (1-2%) and carbamazepine (0.5-1%) are also associated with a higher incidence of neural tube defects than the general population. Management of the pregnant women with known PTS is best done with monotherapy at the lowest dose that will prevent seizures. Benzodiazepines may be the safest to use during pregnancy but they can have significant addictive and mood altering effects (Morell, 1995).

Hydrocephalus

Hydrocephalus occurs in less than 10% of individuals with severe TBI and is caused by disruption in the flow or absorption of cerebrospinal fluid (CSF). Acute hydrocephalus can occur within the first weeks after TBI when the ventricular system is obstructed with blood or by brain swelling in the posterior fossa. Later hydrocephalus is associated with early subarachnoid or intraventricular hemorrhage, leptomeningeal infection, or subdural hematoma where the flow or uptake of cerebral spinal fluid may be disrupted. Symptoms of classic hydrocephalus are a stiff, shuffling, wide-based gait, declining mentation (especially alertness), and urinary incontinence. Unfortunately these are common symptoms of TBI and thus difficult to attribute solely to hydrocephalus. If an individual has been making a steady recovery over several weeks which then stops or declines, hydrocephalus should be investigated by obtaining a CT or MRI scan. Typical findings are normal or absent cortical sulci and distention of the entire ventricular system with periventricular extravasation of CSF into the white matter. This differentiates between ventriculomegaly from brain atrophy, where cortical sulci are quite prominent (Hammond and McDeavitt, 1999).

Limb Swelling: Deep Venous Thrombosis or Heterotopic Ossification

For unknown reasons, individuals with severe TBI are at risk for development of heterotopic ossification (HO), particularly at hips, knees, elbows, and shoulders. This is a condition of bone formation in areas where bone should not be. Incidence of HO is highest in fractures requiring open reduction and internal fixation but it can occur without fractures (Garland, 1991). Therapists, nurses, or family members are often the first to notice that range of motion (ROM) has a "hard end feel" at one or more of these joints. These signs will pre-date standard x-ray findings. Swelling and warmth in a limb is also an early sign and may be suggestive of a deep venous thrombosis. That condition is more seriously life-threatening and must be ruled out first with an ultrasound/doppler or venogram.

A triple phase bone scan is the most sensitive radiologic test if the plain x-rays are negative. Once diagnosed, indomethicin for adults or aspirin for children should be started, if necessary with an H2 blocker or misoprostol. Gentle but persistent ROM is crucial to maintain available ROM. Tone management with nerve or motor point blocks or antispasticity medications may help facilitate ROM. If indomethicin is contraindicated or poorly tolerated, or if multiple joints are involved and the HO appears to be spreading faster than the ROM regimen, the individual should be evaluated for low dose radiation. NSAIDs should be continued for 6 months (Djergaian, 1996; Hammond and McDeavitt, 1999).

The role of etidronate disodium is more controversial as only one prospective study has shown it to be efficacious (Spielman et al, 1983). The dose is 20 mg/kg given between meals to avoid binding to dietary calcium for 2 weeks, followed by 10 mg/kg for 3-6 months.

Surgical resection of HO can be done within 12-18 months following trauma; some surgeons are opting for earlier resection followed by low dose radiation and ROM as soon as the drains are removed (Varghese, 1992).

ENDOCRINE ISSUES

Endocrine abnormalities such as the syndrome of inappropriate antidiuretic hormone secretion (SIADH), cerebral salt wasting syndrome, diabetes insipidus, and anterior pituitary insufficiency occur in approximately 25% of individuals with moderate to severe TBI (Stewart and Cifu, 1997). The most frequently seen is SIADH, which can usually be treated with water restriction alone and with the addition of demeclocycline, if necessary (Doczi et al, 1982). Diabetes insipidus is less common and must be differentiated from compulsive water drinking, if necessary by a water deprivation test. Treatment requires fluid replacement and administration of vasopressin subcutaneously or via nasal spray. Most of these conditions are temporary, although DI can persist as a chronic condition.

Anterior pituitary dysfunction is much less common and is found in less than 5% of those with moderate or severe TBI (Kalinsky et al, 1985). Elevated prolactin levels are the most common finding and can result in gynecomastia, galactorrhea, and anovulation. Hyperprolactinemia can also be a side effect of neuroleptic use. Primary or secondary adrenal insufficiency is rare from organ failure; it is more commonly iatrogenic, secondary to therapeutic use of steroids.

Amenorrhea and Dysmenorrhea

Transient amenorrhea is common in women with TBI (Cytowic and Smith, 1986). It may spontaneously self-correct within about six months after trauma, so that extensive work-up is probably not warranted early on in the absence of other clinical findings. Screening pregnancy tests are indicated in all women of childbearing age regardless of their report of last menstrual period or sexual activity. If it has been greater than 6 months from the initial trauma and pregnancy has been ruled out, it is reasonable to begin the work-up by obtaining a prolactin level. High levels are most likely secondary to neuroleptic use or, rarely, a coincidental prolactinoma. If prolactin is low or normal, administration of medroxyprogesterone, 10 mg/day for 5-10 days, may result in withdrawal bleeding and resumption of regular menses. If there is no response, further gynecologic or endocrine work-up is indicated to differentiate ovarian failure from hypothalamic or pituitary dysfunction (Lalenta and De Feo, 1996).

Menstrual or menopausal symptoms may be exaggerated in women with TBI and may lead to episodic behavioral dyscontrol. Episodes of behavioral dyscontrol can be recorded by women themselves or by family members by using a chart similar to a headache chart, in order to determine whether there is a correlation between those episodes and menstruation. If such correlation is evident, non-steroidal anti-inflammatory medication may be given a few days prior to beginning of menstruation.

Contraception

Contraception choices are rarely limited because of medical considerations in women post-TBI, although memory, attention, and initiation deficits may lead to poor compliance with oral contraceptives and barrier methods. Long-acting injectable contraception with leuprolide or every 3 month use of progestin lovonorgestrel will likely have lower failure rates by minimizing the daily compliance issues. Those individuals who smoke or have decreased lower extremity mobility are more risky candidates for hormonal contraception because of increased risk for deep venous thrombosis. Furthermore, individuals on phenytoin or carbamazepine have higher pregnancy rates. Intrauterine devices or tubal ligation are alternative choices for those who are not candidates for hormonal treatment or who cannot reliably use barrier methods (Sandel, 1996). Women with TBI who have poor judgment may put themselves at risk for contracting sexually transmitted diseases from unprotected sexual activity and need to be counseled on and monitored for such.

Osteoporosis

Osteoporosis has not been studied extensively in individuals with TBI. Those with prolonged immobility due to severe motor deficits can develop disuse osteopenia (Hangartner, 1995). Women with TBI and impaired balance or strength are at higher risk of fractures because of more frequent falls. Calcium supplementation and hormone replacement therapy should be considered in light of general health maintenance.

COMMUNITY LIVING CHALLENGES

A number of challenges are experienced by individuals with TBI who return to the community as they begin the process of reintegrating into their social, work, and family environments. The main areas of difficulty, for both men and women, include returning to work or other productive activity, continuing health issues, social integration, and resumption of interpersonal relationships.

Behavioral and Cognitive Issues

Behaviors and cognitive deficits that may be tolerated in the rehabilitation or home setting are often problematic during social interactions. Verbal or physical outbursts, disinhibition, decreased initiation, poor judgment, inability to process simultaneous tasks or tolerate interruptions, changes in memory and organizational skills are all common after TBI. Individuals with TBI may have difficulty translating behavioral responses and skills learned in clinical settings to real-life settings because of memory and/or executive functioning problems. Major depression has been diagnosed in similar percentages of men and women; however, more women were found to have anxiety disorders (Hibbard et al, 1998).

The quantification of intellectual capabilities is best accomplished with formal neuropsychological, language and perceptual testing by psychologists, speech and language pathologists, and occupational therapists (Putnam and Fichtenberg, 1999). However difficult testing may be, implementation of cognitive and behavioral strategies in community settings is the real challenge of individuals with TBI, caregivers, family members, and post acute care and acute rehabilitation professionals.

Treatment in community settings such as the home, neighborhood, school, and at the work site with a therapist, tutor or job coach are often necessary to achieve the desired outcome in those settings (Mateer and Raskin, 1999; McNeny, 1999; Wehman et al, 1999). Compensation techniques for behavioral and cognitive challenges range from gradual withdrawal of supervision, use of memory notebooks, pagers or electronic devices, simplification of tasks, holding crucial conversations in non-distracting environments, altered work schedules, etc. Successful therapeutic interventions often require more than one discipline, e.g., occupational therapist (OT) and/or speech pathologist and psychologist, OT and job coach. Case managers can help facilitate these team efforts and interface with third party payers, which is usually necessary.

Adjustment to individual's, friend's, family member's or co-worker's altered capabilities after TBI is also challenging. Individual or group counseling, support groups, individual peer support can be helpful (Delmonico et al, 1998). Acknowledgement of these altered capabilities is crucial to learning how to accommodate to them for all of those involved with the life of the individual with TBI. Accessing appropriate resources can be facilitated by local, state, and national Brain Injury Associations or National Institute of Disability and Rehabilitation Research (NIDRR) TBI Model Systems (Brain Injury Association, 105 N Alfred Street, Alexandria, VA, 22314, (703) 236-6000; TBI Model Systems Website: www.tbims.org).

Driving

Driving after TBI must take into account the individual's new capabilities. Some jurisdictions require physicians and other health care practitioners to report to public health authorities or the department of motor vehicles (DMV) physical and cognitive conditions that may affect an individual's ability to drive safely. Uncontrolled seizures and visual disturbances are obvious problems that can affect driving safety. California's DMV revokes licenses from individuals with moderate or severe cognitive impairments reported by health care practitioners (Reuben and St. George, 1996). Pre-driving perceptual and simulated driving assessments, usually performed by occupational therapists, can identify obvious safety issues that would make driving unrealistic (Galski et al, 1993). More subtle cognitive and judgment problems are impossible to evaluate in an office or even simulated setting. Ideally, such individuals should be evaluated by a health care practitioner who is also certified as a driving instructor (McNeny, 1999). Identified problem areas can then be practiced by the individual with a standard driving instructor. If these resources are not available or too costly, individuals should minimally be re-tested on the rules of the road and on the road by the DMV, even for purposes of self-protection from future injury to themselves or others.

Return to Work/School

The primary factors that influence return to work/school are age, level of education, pre-injury work history and severity of injury. More specifically, older individuals, persons with less education or no work history, and individuals with more severe injuries generally have much lower success rates in returning to work/school even to settings that are at a lower level than their pre-injury activity (Wehman et al, 1999).

The majority of studies that examine return to work/school rates for men and women with TBI report no significant differences between the genders (Greenspan et al, 1996; Ip et al, 1995; O'Neill et al, 1998; Mills et al, 1992). Corroborative evidence comes from the TBI National Database, where 53.6% of all individuals were competitively employed at the time of injury (48% for women and 55.4% for men); however, at one year post-injury only 22.6% of women and 21.8% of men had returned to work. Similar percentages are reported at two years post-injury. In contrast, one study reported that in a group of similarly injured individuals, 51% of the women returned to some form of work compared to only 40% of the men (McMordie et al, 1990). Similarly, Grosswasser et al (1998) reported that, at discharge from acute rehabilitation, a higher percentage of women with TBI were deemed capable of returning to work than men with similar injuries.

Social Integration

Social integration, defined as an individual's ability to participate in and maintain customary social relationships (WHO, 1980), is a critical component of a satisfactory quality of life for most individuals. Many key elements influence the degree to which an individual is socially integrated, and no single measure assesses social integration to everyone's satisfaction. Regardless, all studies to date have shown that women with TBI are more socially integrated than men (O'Neill et al, 1998; Dawson and Chipman, 1995; Gutman and Napier-Klemic, 1996). While one study reported no differences in the number and kind of social interactions (O'Neill et al, 1998), another reported that women were not only more likely to maintain post-TBI participation in pre-injury activities but also to undertake activities that would forge social connections (Gutman and Napier-Klemic, 1996).

Women with TBI were also found to have a higher degree of handicap in completion of activities of daily living than men; however, this may be because the types of ADLs probed, such as doing finances and grocery shopping, may have been ones that women typically did pre-injury, with women thus more likely to report difficulties with these activities post-injury (Dawson and Chipman, 1995).

Interpersonal Relationships and Sexuality

After a TBI, individuals can experience dramatic changes to their sense of being feminine or masculine (gender identification), self-image, emotional make-up, and ability to communicate and interact with others. All of these changes impact on the individual's ability to achieve and maintain interpersonal relationships.

Gender identification, and satisfaction with that role, is important for an individual in order to maintain a healthy self-esteem, to interact with others, and to feel satisfied with the attained quality of life. Following a TBI, internal gender identification appears to be less impaired in women than in men (Gutman and Napier-Klemic, 1996). This was attributed to the fact that women did not appear to rely on the ability to engage in typically feminine activities, such as cooking and needleworking, to define and express their own sense of gender (Gutman and Napier-Klemic, 1996). Instead, women described participating in a variety of activities that cross gender lines, including playing sports, attending baseball games, and learning computers.

Men and women do present with similar difficulties in the maintenance of intimate relationships (Aloni and Katz, 1999; Kreuter et al, 1998). The most commonly reported problem is reduced libido (Aloni and Katz, 1999). In one study, almost half of those questioned stated that the frequency of sexual intercourse significantly decreased or ceased completely (Kreuter et al, 1998). There is debate as to whether the occurrence of changed libido can be attributed to a locus of injury in the brain. Hyposexuality has been linked to lesions of either the medial temporal lobe (Garden, 1991), frontal temporal region (Zasler, 1998), basal hypothalamus and temporal lobe (Lundberg, 1992), or diffuse frontal injury (Griffith et al, 1990), or to no particular locus of injury (Rosenbaum and Hoggs, 1989). However, it has been reported that, although primary factors may be present, the far more important influence on libido is the secondary factors such as depression, decreased self-esteem, and performance anxiety that may accompany brain injury (Aloni and Katz, 1999).

Reports of hypersexuality in women with TBI are rare (Britton, 1998; Zencius et al, 1990). Injuries in the right cerebral hemisphere have been correlated with higher self-reported sexual arousal and sexual experiences (Sandel et al, 1996). Damages to the frontal lobes can result in dysinhibition and inappropriate sexual behavior (Elliott and Biever, 1996). Behavioral feedback techniques in which verbal feedback is provided for inappropriate behavior have been used for this condition (Zencius, 1990).

Men and women also appear to experience the same frequency of difficulties in arousal (Aloni and Katz, 1999). In the majority of cases, these difficulties are probably secondary to communication problems, poor self-image, and increases in anxiety, stress, and distractibility that are associated with a TBI (Aloni and Katz, 1999). There are conflicting reports about the incidence of anorgasmia in women following TBI (Aloni and Katz, 1999; Kreuter et al, 1998); however, it may be as high as 40% (Kreuter et al, 1998). A number of factors need to be considered when problems with sexual intercourse are encountered; the potential causes could be related to the primary brain injury, hormonal abnormalities that may occur secondary to the TBI, or from medication use.

CONCLUSIONS

Women comprise 25-30% of individuals who experience TBI. In young women brain injury occurs primarily as a result of motor vehicle crashes and to older women as a result of falls. Injury pathology helps to determine the acute complications that occur. Women with TBI encounter similar challenges to men in terms of recovery of their own physical, cognitive and emotional capacities. The natural course of recovery may take up to 2 years post injury. Women seem to have better outcomes with regard to community reintegration and lower incidence of post-traumatic seizures. The reasons for these different outcomes deserve further study.

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