Traumatic Brain Injury, Polytrauma, and Pain: Challenges and Treatment Strategies for the Polytrauma Rehabilitation

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Problem: Chronic pain conditions are common sequelae of traumatic brain injury (TBI). Unfortunately, the incidence of TBI among personnel deployed for Operations Enduring Freedom and Iraqi Freedom (OEF/OIF) is significant, and there is growing evidence that ongoing pain, particularly headaches, will be a primary concern for these individuals. Objective: This article synthesizes empirical data from civilian and veteran populations and clinical experience with OEF/OIF personnel with polytrauma to provide recommendations for the assessment and treatment of chronic pain among those with TBI. Conclusions: The available data signal the need for the incorporation of early and aggressive pain management strategies into existing treatment models. Challenges to providing effective pain management for OEF/OIF veterans are numerous and include comorbid cognitive, medical, and emotional impairments that complicate readjustment to civilian life. It is likely that the problem of polytrauma pain and associated comorbid conditions such as posttraumatic stress disorder and postconcussive syndrome will require the development of integrated approaches to clinical care which bridge traditional subspecialty divisions. A proposed model of treatment is presented.

Keywords: pain, headache, polytrauma, traumatic brain injury, postconcussive syndrome, posttraumatic stress disorder

Due to improvements in battlefield protection, medical care, and casualty evacuation, survival rates following combat injuries in Afghanistan and Iraq exceed 90% (Clark, Bair, Buckenmaier, Gironda, & Walker, 2007). As a result, many service members with once lethal multiorgan injuries now are in need of intensive and prolonged medical and rehabilitative care to facilitate tissue healing and maximize long-term functioning. Traumatic brain injuries (TBIs) are one of the most common components of these polytraumatic injuries (i.e., injuries that affect two or more body systems or organs), and their frequency appears to be increasing. Whereas the prevalence of TBIs among those undergoing treatment for combat injuries during Desert Storm was about 20% (Carey, 1996; Leadham, Blood, & Newland, 1993), initial injury data from Operations Enduring Freedom and Iraqi Freedom (OEF/
OIF) suggests a 30% prevalence rate (Military TBI Task Force, 2007). Furthermore, this rate may not capture the 10–20% of OEF/OIF service members who meet criteria for less severe TBI following routine postdeployment medical screenings (Zoroya, 2006).

As might be expected, TBI prevalence is greatest among the most seriously injured service members. A recent record review found that TBI was present in 77% of 190 consecutive inpatient admissions to one of the four active Department of Veterans Affairs (VA) Polytrauma Rehabilitation Centers (PRCs), which provide medical and rehabilitative services to the most seriously injured (Walker, 2008). In this sample, TBI was more prevalent among those injured in combat (81%) than among those with noncombat injuries (70%). In part, the higher prevalence of TBI in war-injured service members likely reflects the high rates of exposure to blasts, which account for approximately 65% of OEF/OIF injuries. It has been reported that up to 60% of service members exposed to blasts suffer concurrent head injuries (Veterans Health Administration, 2006). Blast exposure may be associated with increased severity of the TBI and associated physical injuries. While comparative data from earlier wars are not available, Warden et al. (2005) reported that 56% of TBIs experienced by a sample of service members evacuated following blast injury were classified as moderate to severe.

It is widely recognized that the experience of pain is a common sequela of TBI. Although the empirical evidence is sparse and the estimation of reliable prevalence rates is hampered by methodological inconsistencies in the existing literature, it is expected that 22% to 95% of individuals who suffer a TBI will experience a chronic pain condition (Uomoto & Esselman, 1993). Compounding this risk among OEF/OIF service members is the fact that persistent pain is one of the most frequent health problems identified by noninjured military personnel returning from Afghanistan and Iraq with prevalence rates ranging between 42% and 81% (Clark, 2004; Gironda, Clark, Massengale, & Walker, 2006; Kalra et al., 2003; Lew et al., in press; Ruff, Ruff, & Wang, 2008). Among the more severely wounded OEF/OIF service members with polytrauma, pain is an almost universal experience. Pain was an identified problem in 89% of the 190 polytrauma inpatients in one PRC sample (Walker, 2008). Another recent record review of service members who sustained polytraumatic injuries while participating in OEF/OIF revealed that pain was reported by 88% of those who had been injured in a blast (96% of whom had a TBI) and by 65% of those whose injuries were not blast related (99% of whom had a TBI). Importantly, pain was the second most frequently reported impairment after cognitive deficits for both groups (Sayer et al., 2008). Lew et al. (2007) found that 97% of a sample of 62 outpatients with polytrauma identified pain as a presenting problem.

For most individuals who suffer a TBI, headaches are the primary pain condition (Nicholson & Martelli, 2004), and they are typically accompanied by the other common symptoms of persistent postconcussive syndrome (i.e., fatigue, sensory disturbances, photophobia, phonophobia, sensitivity, memory and executive functioning deficits, attention/concentration deficits, and emotional distress). However, pain conditions attributable to musculoskeletal, neuropathic, and central mechanisms are known to be frequent as well (Clark, Scholten, Walker, & Gironda, 2009; Ofek & Defrin, 2007; Sherman, Goldberg, & Bell, 2006) and may occur independently, often as a consequence of other injuries suffered during the traumatic event, or as a component of a primary persistent postconcussive syndrome. In civilian populations there is strong evidence of an inverse relationship between the prevalence of pain, particularly posttraumatic headache, and the severity of the TBI (Couch & Bearss, 2001). Uomoto and Esselman (1993) reported that 95% percent of individuals with mild TBI reported pain while only 22% of those with moderate to severe TBI did so. These data mirror headache specific estimates, most likely as an artifact of the high percentage of headache diagnoses counted and classified globally as pain conditions in the TBI pain epidemiological literature. Rates of headache have been reported to range between 30% and 90% among those with a mild TBI, in contrast to the approximately 33% prevalence found among those with moderate to severe injuries (Couch & Bearss, 2001; Evans, 2004; Lahz & Bryant, 1996; Uomoto & Esselman, 1993). It may be that this wide variation in headache prevalence among individuals with mild TBI is due to difficulties in accurately diagnosing mild TBIs. For example, while Ruff et al. (2008) reported that 63% of a sample of 126 OEF/OIF veterans with historical evidence of mild TBIs experienced headaches, when only those with objective neurological or neuropsychological findings were considered headache prevalence increased to 92%. It is also possible that headache frequency or intensity may vary as a function of the mechanism of TBI injury. For example, Andrews, Walker, and Clark (2009) found that headaches were significantly more likely in veterans with polytrauma who sustained penetrating head injuries when compared to those with closed head injuries or those without TBIs. Clarification of the nature of relationships among TBI severity, injury mechanism, and headache characteristics awaits future research.

Another factor known to affect TBI headache prevalence that may account for some of the variation in these data is time since the injury. At 1 month following injury, rates of headaches range from 31.3% (Keidel & Diener, 1997) to 90% (Rutherford, Merret, & McDonald, 1979). However, it appears that many of posttraumatic headache conditions do resolve in the year following the injury. At 3 months, which corresponds to the threshold criterion for diagnosis of chronic posttraumatic headache (Headache Classification Subcommittee of the International Headache Society, 2004), the rates have been found to be as high as 78%. Six-month prevalence has been reported to range up to 44% (De Benedittis & De Santis, 1983), and it is likely that a substantial proportion of individuals who continue to have problems at this time point will do so indefinitely. When assessed 4 years following injury, approximately 20% to 24% of persons with TBI report headaches (Edna, 1987; Keidel & Diener, 1997). Unfortunately, there are few available data on the prevalence and course of other specific pain diagnoses in TBI populations. Although the existing data, which are drawn primarily from civilian populations, may provide some indication of the course and clinical features of the pain conditions likely to be observed among those returning from OEF/OIF with TBIs, it remains to be seen if these conditions will conform to the patterns reported in the literature.

There are several critical differences between the civilian populations represented in the bulk of the current literature and service members who sustained polytraumatic injuries during OEF/OIF. It is probable that some of these differences will prove to be moderating factors in the relationship between pain and TBI.
most salient are the nature and extent of the injuries sustained by OEF/OIF service members. Often the consequence of exposure to high-energy blasts, polytrauma may include pressure-wave, penetration, burn, traumatic amputation, high temperature gas inhalation, and physical displacement injuries that produce multiple physical insults. Among service members injured in combat by blasts, TBIs are almost always present, tissue damage often is extensive, and sensory loss is very common (Sayer et al., 2008). Rates of ear injuries, spinal cord damage, compartment syndromes, and burns far exceed those in service members with noncombat polytrauma. Pain associated with these severe and multiple wounds often is pervasive and disabling. Concurrent nociceptive, neuropathic, and headache pain conditions are common (Clark, Scholten, et al., 2009), and it can be hypothesized that these injuries have a synergistic effect that has implications for the experience of pain and its amenability to treatment. For example, Clark, Walker, Gironda, and Scholten (2009) found that OEF/OIF soldiers injured by combat-related blasts experienced significantly less improvement in pain intensity when compared to soldiers with other types of combat injuries or those injured in noncombat situations despite equivalence in TBI frequency, severity, and improvement. Additionally, even for those combat troops who do not experience moderate to severe physical trauma as a consequence of blast exposure, it is likely that they will be subject to repeated blasts throughout their tour of duty, possibly suffering mild, unidentified TBIs as a result. It may be that this recurring exposure produces diffuse neurological changes that directly or indirectly contribute to the subsequent development of headache and other pain conditions. In support, there is recent animal study evidence that brain tissue may be more susceptible to pressure wave damage than other tissue, and that neural cell dysfunction may occur at pressures that are not sufficient to cause identifiable structural damage to brain cells (Kato et al., 2007).

Another set of factors that may affect all OEF/OIF service members pertains to the psychosocial context in which these injuries occur. Certainly for those who sustained injuries during combat operations, the risk of comorbid psychiatric conditions, particularly posttraumatic stress disorder (PTSD), is high. However, it is likely that all OEF/OIF service members are at elevated risk for emotional disorders given the variety of stressors associated with repeated and/or prolonged deployment, which may include significant periods of time in active combat zones. In addition, it is well known that there are few safe zones in Iraq as soldiers are always vulnerable to mortar attacks, hidden improvised explosive devices, and snipers even on base. Furthermore, given the nature of insurgent warfare, OEF/OIF service members must always be “on guard” as it may be impossible to distinguish “friendlies” from enemy combatants. All of these factors coalesce to produce a heightened and sustained level of hypervigilance that may increase the probability of subsequent stress reactions. Additionally, for those with polytraumatic injuries, the already difficult challenge of reintegration into familial, social, and occupational roles will be complicated by adjustment to the physical and cognitive impairments resulting from the injuries. The reciprocal relationship between chronic pain and emotional disorders is well established, and those with psychiatric conditions are known to experience higher levels of pain-related disability and to have poorer treatment outcomes (Asmundson, Coons, Taylor, & Katz, 2002; Breslau, Lipton, Stewart, Schultz, & Welch, 2003). In addition, the overlap among the signs and symptoms of TBI, PTSD, and pain (particularly headaches) is significant and presents a challenge to accurate diagnosis and effective treatment that may require innovative and integrative approaches to clinical service delivery. As such, it is not unrealistic to expect that the pain conditions associated with deployment-related polytrauma may be more severe, disabling, and resistant to treatment. However, this remains an unanswered empirical question, and it is quite possible that there are protective factors in this cohort that contribute to an increased resiliency.

In summary, war-injured service members with polytrauma and pain often suffer from extensive, multisite tissue wounds complicated by significant TBIs, frequent surgical revisions or extensions, lengthy tissue healing periods, altered physical appearance, sensory loss, limb amputation, emotional trauma, and the activation of multiple pain systems. Given these factors, it appears likely that risk for the development of chronic pain and chronic pain syndromes may be greater for these individuals than for civilians who have sustained a TBI. The extended tissue healing time associated with polytraumatic injuries, which can exceed 2 years, may represent a key transition period in the continuum from acute to chronic pain, which Clark et al. (2007) termed post-acute pain. Aggressive and effective pain treatment during this prolonged period may reduce the risk for chronic pain development or moderate the severity and life impact of the persistent pain sensations. Unfortunately, the cognitive and physical limitations associated with the high prevalence of TBIs in this group of war-injured service members often impede pain assessment and treatment efforts. The intent of this article is to synthesize the existing empirical data from civilian and OEF/OIF populations and clinical experience with persons with polytrauma to provide recommendations for the assessment and treatment of chronic pain among those with polytrauma.

Polytrauma Nonheadache Pain Treatment

The hypothesized increased risk for the development of complex, disabling chronic pain syndromes among OEF/OIF service members with polytrauma highlights the need for proactive intervention for all pain complaints in this population. Because there is some divergence in evidence-based treatment approaches to headache and nonheadache pain conditions, these broad classes of pain disorders will be treated separately in the remainder of this article. It should be noted that the information contained in the Nonheadache Pain Treatment section represents a summary of a more comprehensive treatment of the subject (Clark, Walker, et al., 2009).

Pharmacologic Management of Nonheadache Pain

Pharmacologic management of pain in this population is complex due to the potential for multiple injuries and pain generators, as well as the high risk for polypharmacy. The most common pain pathways in persons with TBI, nociceptive (associated with stimulation of receptors sensitive to noxious stimulation) and neuropathic (associated with a primary lesion or dysfunction of the nervous system), require differing pharmacologic approaches. Medications used to treat nociceptive pain include nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, topical agents, antispas-
ticity medication, and opioids. Topical agents, such as capsaicin and other muscle creams, can be effective pain relieving agents and should be considered in this population due to diminished drug-drug interactions. Opioid pain medications are most effective in treating acute nociceptive pain, but difficult side effects are common. If opioids are used, careful monitoring for constipation, worsening cognitive dysfunction, medication abuse, and respiratory dysfunction is necessary. Due to these side effects, it is our recommendation that the opioid dose should be monitored closely with the goal being to taper as soon as clinically feasible.

Medications used to treat neuropathic pain in persons with TBI most commonly include topical agents, opioids, tramadol, anticonvulsants, and antidepressant medications. Of the antidepressant medications, tricyclic antidepressants are useful in neuropathic pain management but may cause significant sedation and possible adverse effects on balance among those with TBI. Despite some of the potential side effects, anticonvulsant medications are now more widely used to manage neuropathic pain. Opioid pain medications are frequently used to treat more severe neuropathic pain conditions in persons with TBI. However, nonopioid medications are preferred for chronic pain management in persons with TBI given the likelihood of increased cognitive impairments with higher opioid doses and the lack of research evaluating the effectiveness of opioids in this population. Interventional techniques, such as trigger point injections, nerve blocks, and epidural steroids, also may be used for pain management in persons with TBI, in conjunction with therapies and medication management.

When treating complex pain syndromes, medication selection should include a consideration of comorbid conditions. Attempts should be made to choose medications that have side effects with the potential to beneficially affect other symptoms. For instance, choosing a more sedating tricyclic antidepressant may help the individual with pain control, emotional distress, and sleep disturbance. Attempts should be made to minimize poly-pharmacy, but at times using medications in different classes may be necessary in order to target the suspected mechanisms of the pain. Medications should be chosen based on their mechanism of action and then combined in a rational manner to target pain and associated impairment. This approach to prescriptive practice has been labeled rational polypharmacy (Gallagher, Drance, & Higginbotham, 2006).

Rehabilitation Modalities for Nonheadache Pain

Rehabilitation interventions most likely to be effective in this population are those known to be effective in other therapeutic settings. However, it is possible that these modalities may require a more intensive multidisciplinary care setting than is normally utilized for traditional rehabilitation populations (Clark et al., 2007). Useful modalities may include the application of ice or heat, range of motion and stretching exercises, ultrasound therapy, and transcutaneous electrical nerve stimulation (Clark, Walker, et al., 2009). Prescribed exercise is an underutilized, but effective, approach to pain management (Koltyn & Arbogast, 1998) that also may help to improve the common comorbid problems of sleep and mood disturbances in persons with TBI. All therapeutic interventions should be introduced to individuals in a stepwise fashion based on careful assessment of cognitive and physical capacities, as well as treatment readiness. Passive modalities are the first to be offered, and active treatments may be added as the individual demonstrates the necessary mastery and initiative. This graduated approach can facilitate the development of the individual's sense of pain control, and this paradigm is generally consistent with overall cognitive and physical rehabilitation efforts.

Cognitive-Behavioral Interventions for Nonheadache Pain

Standard cognitive-behavioral interventions for pain may be tailored to the polytrauma population in order to facilitate engagement in rehabilitation. The primary goals of treatment are to enhance pain coping skills, reduce pain-related disability and distress, and decrease pain intensity and frequency. It may be particularly important in this population to address pain-related fear and activity avoidance, fears and misconceptions regarding opioid analgesics, the potential for medication addiction or abuse, and the distinction between pain as a sensation and as a sign of tissue damage. Family engagement in rehabilitation goal setting and education, which includes behavioral pain management, reinforces the multidisciplinary efforts of the team, enhances compliance, and reduces family distress (Sherer et al., 2007).

As noted above, polytrauma pain also is complicated by the high frequency of accompanying emotional disorders or symptoms, the most common of which is PTSD. Given the bidirectional interactions between pain and several emotional states or symptom clusters (Asmundson et al., 2002; Breslau et al., 2003), symptoms of one condition may exacerbate the other which can interfere with the rehabilitation process. Additionally, the overlap with symptoms of TBI is likely to complicate diagnostic and intervention efforts. Cognitive-behavioral interventions targeting pain likely will need to address these emotional and cognitive issues concurrently with particular attention being given to those conditions that may aggravate pain (e.g., PTSD; depression) or impede rehabilitation (e.g., cognitive deficits, substance abuse) (Otis, Keane, & Kems, 2003). While pain, emotional disorders, and cognitive impairments may be treated independently, all need to be addressed during rehabilitation in order to achieve optimal long term functional outcomes (Clark et al., 2007). When PTSD is present or suspected, it is particularly important to identify pain sensations that trigger recall and re-experiencing of the traumatic event and traumatic memories that exacerbate pain during rehabilitation efforts. Occasionally, specific pain treatments that temporally increase pain (e.g., exercising an extremity injured in a blast) unexpectedly trigger strong emotional reactions due to the pain-trauma association. If these interactive associations are not identified and addressed, avoidance of rehabilitation activities that stimulate pain or traumatic memories are likely. It is possible that innovative integrated treatment approaches will be needed to effectively address this unique constellation of symptomatology. One such program is described later in this article.

Posttraumatic Headache

Anecdotal evidence suggests that the headache conditions presented by those with blast-related polytrauma may exhibit unique clinical features that are not common to typical posttraumatic headache conditions. One common clinical report is the perceived resistance to standard treatment approaches, but these clinical observations have not been validated empirically. It is quite pos-
sible that exposure to the positive and negative pressure waves of high-energy blasts produces unique diffuse neurological insults that manifest as a distinct type of posttraumatic headache. However, epidemiologic, imaging, and animal model studies (e.g., Kato et al., 2007) are required to examine this hypothesis, and current treatment recommendations follow primarily from existing evidence-based practice for posttraumatic headache.

**Posttraumatic Headache Diagnosis**

The International Headache Society classification system includes a distinct category for posttraumatic headaches (Headache Classification Subcommittee of the International Headache Society, 2004), which is classified as a subset of the headaches associated with head and neck trauma. Posttraumatic headaches usually develop within 7 days of head trauma or return of consciousness, although headache onset may occur with a delay of weeks to months in occasional cases. The category was established because the most frequent forms of civilian head trauma also cause injury to the cervical spinal cord and neck musculature. Individuals who have sustained head and neck injury can have headaches in which the pain originates from both the head and the neck.

Although posttraumatic headaches represent a unique category of headache, they often share features of other types of headaches. The three most common patterns of posttraumatic headaches resemble tension-type, migraine, and mixed migraine and tension-type headaches. Table 1 provides a scheme for categorizing posttraumatic headaches as tension-like, migraine-like, or cervicogenic.

<table>
<thead>
<tr>
<th>Headache feature</th>
<th>Tension-like</th>
<th>Cervicogenic</th>
<th>Migraine-like</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain intensity</td>
<td>Usually mild to moderate</td>
<td>Usually mild to moderate</td>
<td>Often severe or debilitating</td>
</tr>
<tr>
<td>Pain character</td>
<td>Dull, aching, or pressure; sharp pain may be present but is not predominant</td>
<td>Dull, aching, or pressure; sharp pain may be present but is not predominant</td>
<td>Throbbing or pulsatile, can also be sharp/stabbing or electric-like</td>
</tr>
<tr>
<td>Duration</td>
<td>Usually less than 4 hr</td>
<td>Usually less than 4 hr</td>
<td>Can last longer than 4 hr</td>
</tr>
<tr>
<td>Phonophobia or photophobia</td>
<td>One but not both may be present Usually</td>
<td>Usually neither is present Usually</td>
<td>One or both usually present Usually not</td>
</tr>
<tr>
<td>Able to carry out routine activities or work</td>
<td>Usually</td>
<td>Usually</td>
<td>Usually</td>
</tr>
<tr>
<td>Location</td>
<td>Bilateral frontal, retro-orbital, temporal, or holocephalic</td>
<td>Cervical and occipital</td>
<td>Usually unilateral and may vary in location among episodes</td>
</tr>
<tr>
<td>Nausea or malaise</td>
<td>Not present Pericranial muscles, including temporalis, masseter, pterygoid, sternocleidomastoid, splenius, or trapezius</td>
<td>Not present Posterior neck muscles</td>
<td>Usually present</td>
</tr>
<tr>
<td>Pulpable muscle tenderness</td>
<td>Not present</td>
<td>Localized muscle tenderness is not typical, muscle tenderness may be present with long-duration headaches</td>
<td></td>
</tr>
</tbody>
</table>

Table 1: Criteria for Characterizing Posttraumatic Headaches as Tension-Like, Cervicogenic, or Migraine-Like

Cervicogenic pain is focused in the neck and occipital region of the head. It is often associated with palpable contraction of posterior cervical muscles and can be partially alleviated by cervical massage or heat applied to the neck. Topical treatments that increase blood flow to skin and underlying muscle applied to the posterior neck may also reduce pain. Cervicogenic pain can be increased by neck movement, particularly extreme forward flexion or rotation. In contrast, tension headaches have a pattern of pain that is band-like, temporal, or perceived as behind the eyes. Tension headache pain can be perceived as a holocephalic pressure or as if a belt or cap is tightening about the head. The character of the pain for both cervicogenic and tension headaches is usually a dull, aching type of pain. Due to the coexistence of cervicogenic pain and tension headaches, persons with head and neck trauma may have features of both cervicogenic pain and tension headaches.

Most cases of posttraumatic migraine headaches resemble migraine without aura (common migraine). The criteria for migraine are recurrent episodes of pain lasting for 4 to 72 hr with at least two of the following four characteristics: (1) unilateral pain, (2) pulsating quality, (3) moderate or severe intensity, or (4) exacerbation by or causing avoidance of routine physical activity. In addition, migraines have at least one of the following two characteristics: (1) nausea, vomiting, or both or (2) phonophobia or photophobia. Among persons who experienced migraine headaches prior to head trauma, posttraumatic migraine headaches are associated with a definite increase in headache frequency or intensity or a change in the pain pattern following head trauma. In civilian populations, many headaches resolve within 3 months. Mixed migraine tension headaches have features of both headache types. For example, a mixed headache may involve severe bilateral nonthrobbling pain...
associated with nausea or accompanied with both photo- and phonophobia.

A diagnosis of posttraumatic headache requires a standard comprehensive clinical interview which, at a minimum, addresses the diagnostic criteria outlined by the International Headache Society (Headache Classification Subcommittee of the International Headache Society, 2004). Additionally, because headaches are episodic phenomena, it is important to have individuals keep a headache diary in order to record the frequency, intensity, duration, prodromal signs, and triggers of headaches. Diaries can assist in establishing the characteristics of the headaches and their response to interventions. This may be particularly important in the cases of blast-related headaches so that unique features, should they exist, can be identified and addressed by the clinician. Persons with cognitive deficits may need to be assisted in completing the headache diary, and family members should be enlisted if possible. It may be necessary to reduce the extent of information recorded in the diary if an individual is unable to comply.

**Pathophysiology of Posttraumatic Headaches**

Despite relatively well-defined diagnostic distinctions between headache types, the physiological basis of many headache conditions is not well understood. However, there are some aspects of head trauma that may potentiate factors that are believed to contribute to headache. Cervicogenic head and neck pain is associated with injuries to posterior cervical paraspinal muscles and cervical vertebrae (Kaniecki, 2003), which are common sequelae of head trauma particularly if there is rapid cervical flexion and extension, compression, or traction. Posttraumatic headaches that resemble tension-type headaches may be associated with the existence of hypersensitivity of central pain processing networks. Persons with tension headaches experience more intense pain in response to noxious stimulation to the face, and they have lower thresholds for inducing pain (Ashina, 2004), and central sensitization is implicated at the level of the upper cervical spinal dorsal horns and trigeminal nuclei (Bendtsen, 2000). It is possible that head trauma may precipitate tension headaches due to traumatic injury of the central pain modulation pathways that disrupts the normal mechanisms for suppressing pain.

The impact of nervous system trauma on the genesis of pain has been most extensively studied in spinal cord injury, but the findings for spinal cord injury may provide insight into the pain production after head trauma. There are several classes of neuronal Na⁺ channels that differ in their voltage gated properties. An interesting, but unfortunate, consequence of central nervous system injury is enhanced expression of Na⁺,1.3 channels (Black, Liu, Tanaka, Cummins, & Waxman, 2004; Hains, Saab, & Waxman, 2005). The gating properties of the Na⁺,1.3 channels are such that increasing their expression causes neuronal hyperexcitability (Waxman & Hains, 2006). Trauma leads to upregulation of Na⁺,1.3 channels in thalamic neurons that participate in the pain transmission and pain modulation pathways (Hains et al., 2005). The maintenance of pain after trauma and the continued upregulation of Na⁺,1.3 channels is related to persistent activation of microglia that produce prostaglandin (PG) E₂ (Hains & Waxman, 2006; Zhao, Waxman, & Hains, 2007). Decreasing PG E₂ production will reduce the expression of Na⁺,1.3 channels and reduce pain (Zhao et al., 2007).

Another hypothesis is that head trauma may trigger or rekindle migraine due to either damage to meningeal blood vessels or injury-induced neuronal hyperexcitability. Migraine is now seen as a neurovascular disorder with the primary dysfunction arising within the central nervous system. Altered cerebral activity changes the activation of pain-producing intracranial meningeal structures that give rise to headache pain (Hargreaves & Shepheard, 1999). Episodes of migraine are associated with trigeminal nerve mediated activation of pain-sensitive meningeal structures (Bolay et al., 2002). For the subset of individuals with a clear genetic linkage to migraine, most of the recognized genetic alterations lead to mutations of calcium channels in cerebral vascular smooth muscle or are mitochondrial mutations that alter the function of cerebral blood vessels (Montagna, 2000). It is possible that head trauma injures the meninges and alters the trigeminal communication with the meninges. By means of mechanisms similar to those discussed above for tension headaches, injury to excitable elements of the meninges and its innervating nerve fibers may produce a hyperexcitable state leading to the genesis of migraine. There is also increased thalamocortical traffic associated with attacks of migraine (Coppola et al., 2007). The thalamocortical activity is associated with pathological trigeminal stimulation of the meninges. The trigeminal sensory fibers not only transmit sensory information, they also release substance P, which is a small neuropeptide that acts as both a neurotransmitter and a pro-inflammatory agent. Meningeal inflammation contributes to migraine pain (Bolay et al., 2002).

In addition to increased trigeminal activation of meningeal structures, migraine is associated with cortical hyperexcitability (Aurora & Wilkinson, 2007; Coppola, Pierelli, & Schoenen, 2007; Sandor, 2007; Stanekwitz & May, 2007). Persons with migraine have exaggerated cortical responses to visual and auditory stimuli. The phenomenon of cortical spreading depression entails a wave of cortical excitation that moves over the cortex that is followed by reduced cortical excitability (Mulleners, Chronic, Palmer, Koehler, & Vredeveeld, 2001). Spreading depression is most prominent in the occipital cortex. The occipital poles are particularly vulnerable to injury due to head trauma, and, perhaps, injury-induced hyperexcitability may lead to conditions of cortical excitability that permit spreading depression to develop.

**Posttraumatic Headache Treatment**

**Episodic tension-like headache.** Episodic tension headaches usually respond to nonsteroidal anti-inflammatory medications (NSAIDs) that can be obtained over-the-counter. Pain treatment is more likely to be successful if the medication is taken at the onset of a headache rather than waiting for the headache pain to escalate. On occasion, opioid medication may be needed to treat a severe tension headache; however, repeated use of opioid medication can exacerbate the cognitive and behavioral manifestations of TBI and lead to problems associated with dependence. Aspirin and acetaminophen are often combined with caffeine or a sedative drug in a single medication. Combination drugs may be more effective than NSAIDs or acetaminophen alone, but persistent usage may lead to rebound headaches. Analgesic-sedative combinations can be obtained only by prescription because they may produce dependency or, in vulnerable individuals, trigger addiction. They may also lead to chronic daily headache. Combinations of acet-
aminophen or aspirin and an opioid should be used with caution. These drugs should not be used more than 2 days a week due to concern for side effects and the potential for dependency.

Unfortunately, tension-like headaches associated with TBI may be resistant to medication alone. Some persons may achieve better pain relief if medication treatment is coupled with other treatment modalities. Cognitive-behavioral intervention, such as relaxation training and biofeedback, can help the individual to learn to identify tension and relax the muscle groups whose contraction may be contributing to the tension headache. Patients also should be encouraged to engage in physical therapy to exercise neck muscles and maintain appropriate range of motion. Increased physical activity also may help to reduce the frequency and intensity of tension headaches. In addition, if headache onset is associated with flaring of PTSD, such that the episodes are triggered by flashbacks or panic, successful headache treatment requires the development of control over stress reactions.

Cognitive-behavioral interventions for headache conditions are known to produce headache relief comparable to that obtained with medication alone (Holroyd & Penzien, 1990; Penzien, Rains, & Andrasik, 2002). In addition to the aforementioned relaxation training or biofeedback, cognitive-behavioral interventions may include trigger identification, stress-management, and the development of a headache management plan. Education regarding headaches and wellness may help to correct pain-related cognitive distortions, encourage proactive self-management, and promote an increased sense of control over the headache conditions. Finally, because those with recent onset posttraumatic headache are at risk for transformation to treatment-resistant chronic daily headache conditions, persons with polytrauma should be educated regarding the issue of analgesic overuse.

Spinal manipulation can be used to treat cervicogenic pain provided that the neck is mechanically stable. The effectiveness of spinal manipulation may be comparable to that of pharmaceutical treatment of tension headaches of cervicogenic origin. In one study, persons with tension headaches receiving either spinal manipulation or an antidepressant medication experienced comparable improvement (Boline, Kassak, Bronfort, Nelson, & Anderson, 1995). A review of nine studies that tested spinal manipulative therapy for tension headaches concluded that spinal manipulation is comparable to medications used for the prevention of tension headaches (Bove & Nilsson, 1998). Persons who experience more than three tension headaches per week may benefit from treatments that are designed to prevent tension headaches. Pharmaceutical agents that may be useful in reducing the likelihood of an individual developing a tension headache include (1) tricyclic antidepressant agents, (2) regular dosing of NSAID medication or acetaminophen, and (3) anxiolytic medication if the headaches are associated with anxiety as might occur as part of a stress reaction (Mathew & Ward, 2004; Ruff et al., 2008; Ruoff & Urban, 2004). Healthy sleep patterns facilitate synaptic homeostasis (Tononi & Cirelli, 2003) and other processes that contribute to cerebral plasticity (Frank, Issa, & Stryker, 2001; Jha et al., 2005), which will enhance the ability of the brain to adapt after traumatic injury.

In the experience of one of the authors (R.L.R.), beta-blockers and topiramate are the best tolerated prophylactic agents for OIF/OEF veterans with TBI who have migraine or mixed headache conditions (Ramadan, 2007). These agents are the preferred prophylactic treatment of migraine, and they have been demonstrated to improve the quality of life of migraineurs (Garcia-Monco, Fonceca, Bilbao, Ruiz, & Gomez-Beldarrain, 2007). Beta-blockers are particularly useful when headaches occur in association with episodes of anxiety. Another advantage of the use of beta-blocker treatment with persons with polytrauma is that these agents typically do not impair cognition. There is anecdotal concern that beta-blockers may trigger or worsen depression; however, careful analysis suggests that beta-blockers in fact do not cause depression (Huffman & Stern, 2007; Stoudemire et al., 1984). Beta-blockers may worsen sleep disorders and may intensify nightmares. Therefore, sleep should be carefully monitored if beta-blockers are used. If beta-blockers are effective in reducing the frequency and intensity of headaches, but are associated with nightmares, the nightmares can be addressed using prazosin (Daly, Doyle, Radkind, Raskind, & Daniels, 2005; Dierks, Jordan, & Sheehan, 2007; Raskind et al., 2007). Topiramate may be the first choice for veterans with impaired sleep. The dose of topiramate needs to be increased slowly to minimize daytime somnolence and impaired cognition. A potential advantage of topiramate is that it may potentiate neural repair after injury (Follett et al., 2004). Weight loss, a potential side effect of topiramate, may be of benefit for some individuals.

Effective abortive treatment requirements that the veterans recognize their own warning signs that a headache is developing. These may include a classic aura or prodromal symptoms such as changes in mood, onset of fatigue, sensitivity to sensory input, and difficulty with attention and concentration. Abortive treatment is more likely to succeed if treatment is initiated as soon as the veteran recognizes a headache is beginning. Prompt initiation of acute treatment will also reduce the need for opioid medications to “rescue” the effective intervention typically requires the use of a combination of acute and preventative medications and nonpharmacologic prevention strategies including lifestyle regulation, stimulant reduction, and trigger avoidance. Cognitive-behavioral migraine intervention, which utilizes many of the components used to treat tension headaches, should include a focus on identification and avoidance of migraine triggers. Common headache risk factors and triggers include sleep disruption, increased time between meals, stress, and specific foods, beverages, and odors. Other nonpharmacologic treatments commonly employed are extracranial pressure and cold compresses. Regular exercise, maintaining regular sleep and meal schedules, and practicing overall good health strategies are also an important part of the treatment regimen. However, these lifestyle changes are viewed primarily as prophylactic measures.

Obtaining deep, restful sleep may be particularly important for those with posttraumatic headache. It is well known that impaired sleep will worsen both tension and migraine headaches as well as other pain conditions (Kaniecki & Lucas, 2004; Landy & Smith, 2004; Mathew & Ward, 2004; Ruff et al., 2008; Ruoff & Urban, 2004). Healthy sleep patterns facilitate synaptic homeostasis (Tononi & Cirelli, 2003) and other processes that contribute to cerebral plasticity (Frank, Issa, & Stryker, 2001; Jha et al., 2005), which will enhance the ability of the brain to adapt after traumatic injury.

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individual from severe headache pain. If acute treatment of a migraine is not effective, rescue treatments may be needed to break the migraine. If rescue therapy is required on a regular basis (e.g., more than once a month), then the veteran should receive prophylactic treatment as well as abortive treatment.

It is important that acute migraine treatment be used prudently to avoid inducing headaches due to medication overuse or rebound. Headaches associated with medication overuse are typically tension-like in character. Persons with frequent disabling headaches may overuse their medication, leading to chronic daily headache and habituation to the medication. There is growing evidence that overuse of opioid medication may contribute to the transformation from episodic migraine to chronic migraine conditions (Bigal & Lipton, 2009; Bigal et al., 2008). Treatment of medication overuse headaches requires stopping daily use of acute treatment, which often produces withdrawal symptoms that include rebound headaches. In many cases, individuals fall into a pattern of continued medication overuse in an effort to avoid rebound headaches. When an individual is caught in a pattern of medication overuse, the headaches are usually refractory to preventive medications. In most cases, headaches improve after an analgesic washout period. It is important to educate individuals that acute migraine medication should be limited to no more than three treatments per week on a regular basis. A headache diary that documents headache frequency and medication usage patterns may be useful in detecting overuse.

Barriers to Effective Pain Management in Polytrauma Populations

Given the complex and heterogeneous nature of polytrauma conditions, selection and delivery of effective pain management interventions is complicated by a range of person-specific factors. In this final section, we focus on two barriers in particular: pain assessment in the person with communication impairment, and treatment of those with multiple somatic and cognitive symptoms of mixed or undifferentiated etiology.

Pain Assessment in the Communication Impaired

The assessment of pain in persons with communication impairment, such as those with moderate to severe TBI, represents one of the most significant challenges in the field of pain management (Weiner & Herr, 2002), and this barrier is compounded by the complexities of polytraumatic injuries. Persons with impairment in cognition and communication as a result of TBI may not be able to convey pain complaints, and the interaction between pain and cognitive symptoms may exacerbate functional impairments and impede response to treatment (Ivanhoe & Hartman, 2004). For instance, unmanaged pain may be associated with increased cognitive dysfunctions in areas of attention, processing speed, memory, and executive function (Ivanhoe & Hartman, 2004). Furthermore, the diagnosis of pain conditions and the evaluation of treatment response can be difficult due to the cognitive, linguistic, and behavioral deficits commonly found in moderate to severe brain injury (Ivanhoe & Hartman, 2004; Sherman et al., 2006; Young, 2007). Quite simply, health care providers have difficulty knowing when these individuals are in pain and when they are experiencing pain relief, which places the individual at risk for both undertreatment and overtreatment.

Although the experience of pain is common in cases of TBI, the assessment of pain in persons with brain injury has received little research attention to date (Sherman et al., 2006). Given the current state of the art, providers must adapt assessment techniques from other patient groups. Strategies based on research with elderly persons with cognitive impairment and communication deficits resulting from dementia provide the most empirically sound basis for assessment of pain in the case of moderate to severe brain injury. Guidelines for the assessment of pain in persons with impaired cognition and communication have been developed by several groups (e.g. AGS Panel on Persistent Pain in Older Persons, 2002; Royal College of Physicians, British Geriatrics Society, & British Pain Society, 2007). The consensus of these evidence-based guidelines is that pain assessment in the cognitively impaired often requires the use of multiple sources of data including self-report, family or caregiver report, behavioral observation, physiological markers (which are most useful in acute pain states and less reliable in chronic pain), and empirical trials of analgesics.

Because pain is a subjective experience, the individual’s self-report is regarded as the best source of information and clinicians should avoid reliance on their own judgment to evaluate the individual’s experience. Initial effort should be directed toward determining if an individual can use some form of self-report, which may include vocal or nonvocal channels, to communicate the existence of pain and its intensity. Neuropsychological and speech/language testing can be used to assess an individual’s abilities to provide self-report, to identify specific deficits to consider in the assessment, and to recommend best strategies for obtaining valid information. Several studies have demonstrated that persons with mild to moderate cognitive impairment are capable of responding reliably to standard validated measures of pain intensity (Chibnall & Tait, 2001; Ferrell, Ferrell, & Rivera, 1995; Weiner, Pieper, McConnell, Martinez, & Keefe, 1996). In addition, the reliability of self-report can be enhanced by using the same scale consistently with an individual. Among those with minimally requisite levels of intact cognitive functioning, the ability to use self-report scales has been demonstrated to improve with practice (Ferrell, 2000). Finally, the clinician should specifically question the person about headaches as OEF/OIF service members typically will not report having them when asked only about their experience of “pain.”

Behavioral observation can be used to validate self-report and to assess pain when it is determined that the person is unable to reliably provide self-report information. Family members or consistent caregivers who are familiar with the person’s usual behavior can provide information about behavioral changes that might indicate the presence of pain (Weiner et al., 2002). Patterns of behaviors that are common in the presence of pain include facial expressions, verbalizations, or vocalizations, body movements, changes in interpersonal interactions, changes in activity patterns or routines, and mental status changes (AGS Panel on Persistent Pain in Older Persons, 2002; Herr & Garand, 2001; Morello, Jean, Alik, Sellin-Peres, & Feminian, 2007). However, these pain behaviors have not been systematically evaluated in younger persons with cognitive impairments, and it is not always clear that these signs of distress are in fact related to pain rather than unrelated mental and physical states such as agitation, perceptual distur-
bances, and other types of physical discomfort. Observations of behavior should include patient behaviors during movement as well as at rest (Feldt, 2000; Hadjistavropoulos, LaChapelle, MacLeod, Snider, & Craig, 2000; Morello et al., 2007).

Empirical trials of analgesic medication can also be used as a tool in pain assessment among the noncommunicative persons with polytrauma. These trials should be done in conjunction with other methods of assessment to evaluate the hypothesis that the behaviors are indicative of significant pain (Baker, Bowring, Brignell, & Kafford, 1996; Gallagher, Drance, & Higginbotham, 2006; Kovach, Weissman, Griffie, Matson, & Muchka, 1999). Unfortunately, there are no validated protocols for this assessment practice. It is very important to consider other potential causes of distress behaviors or agitation, such as infections, constipation, bladder problems, and primary mood disorders that could be masked or aggravated by analgesics. Some analgesics can negatively impact cognitive status, and this risk should be evaluated throughout the course of a trial to minimize effects on overall rehabilitation progress. It is important to systematically assess changes in function, activity, and pain behaviors during the analgesic trial.

A number of instruments has been developed to assess pain in cognitively-impaired elderly adults based on observation of nonverbal pain behaviors. In comprehensive reviews of these tools, Herr, Bjoro, and Decker (2006) and Stolee et al. (2005) concluded that there is no standardized measure of nonverbal pain indicators that can be recommended for broad adoption in clinical practice. These tools, which were developed for use with the elderly persons with dementia, vary widely in conceptualization, psychometric properties, administration, and characteristics of their normative samples. Although several show promise for clinical application, none have been validated for use with persons with TBI. As such, the current evidence base only supports recommendations for general assessment approaches among those with polytraumatic injuries as outlined above, and specific tools must be used with caution informed by awareness of their psychometric limitations. Despite these barriers, it is crucial for any pain intervention to be preceded by some form of systematic assessment that utilizes all available sources of data to provide a basis for the selection of the treatment approach and a baseline for outcomes evaluation.

Pain Management Among Those With Multiple Somatic and Cognitive Symptoms of Mixed or Undifferentiated Etiology

Emerging anecdotal, clinical, and empirical evidence suggests that the most frequently reported problems among returning OEF/OIF military personnel is a cluster of overlapping symptoms labeled by Walker et al. (2009) as postdeployment multisymptom disorder (PMD). This heterogeneous syndrome is characterized primarily by diffuse and wide-ranging somatic, cognitive, and emotional complaints with pain, attentional deficits, and trauma-related emotional distress and dysregulation being the core features. The constellation of mild TBI, chronic pain, and PTSD symptoms has been observed among varied cohorts of returning service members and veterans (e.g., Hoge et al., 2008; Lew et al., 2007; Ruff et al., 2008; Sayer et al., 2008); and as Clark (2009) reported, preliminary evidence suggests that PMD may pose special challenges to treatment. In fact, in one recent study of OEF/OIF VA outpatients the PMD triad was the most common presentation observed when compared to pain, TBI, or PTSD alone or in any combination (Lew et al., in press). If true, innovative integrated treatment programs will be required to address this complex clinical presentation. Despite having differing treatment foci and target behaviors, current empirically supported treatment models for postconcussive syndrome, chronic pain, and PTSD do share common principles, such as the role of cognitive distortions, anxiety sensitivity, and fear-avoidance behaviors in symptom maintenance and exacerbation. Similarly, many of their treatment components, such as psychoeducation and coping skills training, may have a common format (Asmundson et al., 2002; Breslau et al., 2003; Foa & Meadows, 1998; Miller & Mittenberg, 1998). However, current systems of care dictate that these interventions are delivered through separate specialized programs, and in many cases OEF/OIF returnees with diffuse but not severe symptomatology or comorbidities may not meet the criteria for treatment in specialty clinics. Additionally, those who do meet criteria for specialty care may not receive full benefit from the interventions due to barriers associated with the comorbid symptomatology.

An integrated, intensive outpatient treatment program may be the most efficient and effective approach for those with PMD. Such a program would be staffed by medical staff, rehabilitation therapists, and behavioral health providers with specialty training in postconcussive syndrome, pain, and PTSD who would deliver both integrated and/or discrete interventions designed to reduce symptomatology, improve overall functioning, and prevent development of long-term disability. A stepped care model would allow matching of treatment intensity and components based on the constellation of presenting symptomatology and severity of functional impairment. Specific components of an integrated treatment program would be individual and group-based psychoeducation, coping skills training, and cognitive behavioral therapy with an emphasis on instilling proactive self-management techniques. Specific psychoeducational topics would include sleep hygiene, anger management, physical conditioning, relaxation training, substance use/abuse moderation or prevention, vocational rehabilitation, analgesic overuse, and cognitive restructuring. It is expected that some patients might require additional intensive specialty treatment (i.e., PTSD inpatient program) in coordination with PMD treatment, and that ongoing case management would be necessary to coordinate patient care and ensure that routine follow up care is provided. This integrated provider model would help minimize the stigma sometimes associated with mental health programs among current or former service members.

Conclusions

The prevalence of polytrauma with TBI among service members returning from OEF/OIF is significant. Preliminary data suggest that a substantial percentage of these individuals will develop complex pain conditions that will impede their ability to engage in and benefit from cognitive and physical rehabilitation. Additionally, there are several unique features of this population of OEF/OIF service members that may moderate the relationship between TBI and pain, operating as risk factors for the development of disabling chronic pain syndromes. Compounding these barriers to successful rehabilitation is the lack of well-validated measures of the pain experience among those with TBI. Unfortunately, almost
nothing is known about the long-term course of pain conditions associated with polytrauma, and the current state of the art requires reliance on evidence-based practice guidelines developed for general pain populations. While it is likely that many of these practice approaches will prove to be useful, a systematic and comprehensive program of research is needed to determine the most effective timing, dosage, and combination of interventions for the commonly observed patterns of physical, cognitive, and emotional impairment in the polytrauma population. In addition, epidemiological, imaging, animal, and randomized controlled studies are needed to explore the phenomenon of blast-related headaches, which may or may not represent a distinct type of posttraumatic headache. It is likely that more severe cases of polytrauma pain will require the development of integrated approaches to clinical care which bridge traditional subspecialty divisions. Ultimately, the complexity of chronic pain conditions experienced by those with polytrauma will require a truly interdisciplinary approach to clinical practice and scientific investigation.

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