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Posttraumatic disorders following injury: an empirical and methodological review

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Abstract

Although there has been a marked increase in research on psychological disorders following physical injury in recent years, there are many discrepancies between the reported findings. This paper reviews the prevalence outcomes of recent studies of the mental health sequelae of physical injury with a focus on posttraumatic stress disorder (PTSD), acute stress disorder (ASD), and depression. The review critically outlines some of the methodological factors that may have contributed to these discrepancies. The phenomenological overlap between organic and psychogenic symptoms, the use of narcotic analgesia, the role of brain injury, the timing and content of assessments, and litigation are discussed in terms of their potential to confound findings with this population. Recommendations are proposed to clarify methodological approaches in this area. It is suggested that a clearer understanding

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of the psychological effects of physical injury will require the widespread adoption of more rigorous, standardized and transparent methodological procedures.

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1. Introduction

In recent years, increasing attention has been devoted to psychological morbidity that can develop following traumatic physical injury. This attention is understandable considering that physical injury is a very frequent phenomenon. For example, in 2000, 11% of the U.S. population (approximately 30 million people) were treated in emergency departments following nonfatal injuries ([National Centre for Injury Prevention and Control, 2000](#)). The high prevalence of physical injury partly explains why the psychological effects of physical injuries, such as those resulting from motor vehicle accidents (MVAs), industrial accidents, and related events, represent one of the major causes of psychological injury ([Norris, 1992](#)). Considerable research has attempted to investigate the nature and prevalence of psychiatric sequelae either by directly examining physically injured populations or by examining other traumatized populations where injury may be a component of the experience. Despite increasing data on this topic, there is marked variability in the findings concerning the prevalence of psychological disorders associated with traumatic injury.

The purpose of this paper is to provide an overview of the psychiatric prevalence data for this population and to highlight the methodological problems inherent in much of this research. We suggest that many of the inconsistent findings in this field may be attributed to variability in methodological approaches. An underlying premise is that future developments in our understanding of psychological effects of traumatic injury depend on these methodological issues being resolved. While we recognize that factors other than methodology may also contribute to differences in prevalence rates between studies (e.g., cross-national differences in populations), addressing these issues is beyond the scope of this paper. This article critically reviews some of the significant procedural issues underpinning much of the prevalence research on this topic and, where possible, suggests recommendations regarding a consistent approach to assessing severely injured survivors. We commence with a summary of the current evidence concerning psychological morbidity following physical injury, before discussing the major methodological problems that confront researchers in this area. The paper concludes with a series of recommendations for future research methodologies utilizing injured populations. The key focus of this paper is on prevalence of psychopathology following injury, with particular reference to post-traumatic stress disorder (PTSD), acute stress disorder (ASD), and depression, although most of the methodological issues have relevance also for other research designs utilizing this population. While methodological issues have been noted by previous authors (e.g., [Blaszczyński et al., 1998](#)), little attempt has been made to provide a comprehensive and

integrated review of the key methodological dilemmas confronting researchers in this area. This paper is not intended to be overly prescriptive, nor does it claim to identify and solve all methodological problems. Rather, it is hoped that by raising some of the key issues, an increase in methodological transparency and improved reporting of research methodology details with this population will occur.

2. Posttraumatic psychopathology

2.1. Posttraumatic stress disorder

The diagnosis of PTSD was introduced to the diagnostic nomenclature in 1980 with the advent of DSM-III (American Psychiatric Association, 1980). Although the diagnostic criteria have been modified a little over the years, the disorder is now thought to comprise three primary symptom clusters: re-experiencing of the traumatic memories, active and passive avoidance of reminders, and persistent hyperarousal. Symptoms from each of these clusters are required for a diagnosis of PTSD, but they must persist for at least 1 month and be accompanied by clinically significant distress and impairment.

There has been marked variability in the reported prevalence of PTSD in injured populations. Rates of PTSD assessed 1–6 months postinjury range from 17.5% to 42%. For example, Ehlers, Mayou, and Bryant (1998) found a 3-month PTSD rate of 23.1% in a population of consecutive attendees to an emergency department following an MVA. Bryant and Harvey (1998) found a 6-month PTSD rate of 24% following mild traumatic brain injury (TBI). Shalev et al. (1998) assessed a population of consecutive attendees to an emergency department and found a PTSD rate of 30% at 1 month that declined to 17% at 4 months. Michaels, Michaels, Moon, et al. (1999) found a PTSD rate of 42% at 6 months postadmission to hospital following accidental injury.

Studies reporting PTSD prevalence at 12 months postinjury show even greater diversity, with PTSD rates between 2% and 36%. Schnyder, Moergeli, Klaghofer, and Buddeberg (2001) found a rate of only 2% in consecutively admitted Intensive Care Unit (ICU) patients 12 months following severe injury. Ehlers et al. (1998) found a rate of 16% when they followed up their cohort of injured MVA survivors at 12 months. A number of studies have found a 12-month PTSD rate in the 30–36% range (Blanchard, Hickling, Taylor, & Loos, 1995; Koren, Arnon, & Klein, 1999; Zatzick, Kang, et al., 2002). While rates of PTSD vary across studies and across time periods, longitudinal studies generally point to decreased prevalence over time.

Although each of the above studies has its strengths and has contributed to the literature in the area, they all contain methodological issues that may affect their prevalence rates and thus contribute to the disparity between results. For example, few studies clearly identify any attempt to differentiate between symptoms attributable to organic (e.g., injury related) causes and those which have a psychogenic basis, an issue highly relevant for injury survivors. In particular, studies using self-report scales for diagnosing PTSD (e.g., Ehlers et al., 1998; Mayou & Bryant, 2001; Michaels, Michaels, Zimmerman, et al., 1999; Zatzick et

al., 2001) are at particular risk for confounding results associated with this issue. Most studies fail to clarify whether, at the time of assessment, participants were using medications (especially analgesics) that may influence reported symptomatology. A few studies state that they have measured current pain levels; however, they fail to report any relationship between pain level and symptom severity (e.g., Koren et al., 1999). Few studies have evaluated, in addition to PTSD symptoms, the presence of concurrent distress and disability (DSM IV PTSD criterion F). Finally, only a minority of studies (e.g., Zatzick, Kang, et al., 2002) state whether their sample is representative of the larger population from which they drew their sample (e.g., trauma service population, admissions to emergency department). Others clearly fail to utilize a representative sample (e.g., Blanchard, Hickling, Barton, & Taylor, 1996).

2.2. *Acute stress disorder*

The diagnosis of ASD was introduced into the diagnostic nomenclature in DSM-IV in 1994. This diagnosis describes psychological reactions in the initial 4 weeks posttrauma that purportedly predict subsequent development of longer term PTSD. The diagnosis has been widely criticized on the grounds that little supporting evidence was available when it was introduced (Bryant & Harvey, 1997), it placed prominent emphasis on dissociative responses (Marshall, Spitzer, & Liebowitz, 1999), and the primary function of the diagnosis was to predict another diagnosis, namely, PTSD (Harvey & Bryant, 2002).

Of the few studies of ASD that exist, most have focused on physically injured populations. In terms of reported incidence of ASD, prevalence rates of 14% have been reported for MVA survivors with minor TBI (Bryant & Harvey, 1998), 16% of non-TBI MVA survivors (Harvey & Bryant, 1999b), and 12% of industrial accident and burns survivors (Harvey & Bryant, 1999a). Similar findings are reported by Mellman, David, Bustamante, Fins, and Esposito (2001), who found a 16% rate of ASD in their study of hospitalized injury survivors. In contrast, other studies have reported much lower rates of ASD, such as 6% (Fuglsang, Moergeli, Hepp-Beg, & Schnyder, 2002).

Methodological issues are critical for assessment of acute injury survivors. Of the above studies, only Mellman et al. (2001) make direct reference to screening out symptoms which were directly attributable to injury or environmental factors and thus were deemed not to be psychological symptoms. Some of the studies address analgesia issues by screening out those currently using analgesia (e.g., Bryant & Harvey, 1998; Harvey & Bryant, 1999a, 1999b) or by recording analgesia usage (Fuglsang, 2000; Mellman et al., 2001). However, these latter studies fail to report any relationship between ASD symptom levels and analgesia usage. Current pain levels are assessed by few studies despite the finding that pain significantly predicts ASD severity (Fuglsang, 2000). Importantly, Fuglsang et al. (2002) also discuss issues regarding the timing of the assessment and how this may have influenced the results. The majority of the above studies show methodological strength by utilizing structured clinical interviews to assess ASD, although it is difficult to gauge how the use of the different interviews impacts on prevalence rates. The tools utilized to diagnose ASD include the Acute Stress Disorder Interview (Bryant,

Moulds, & Guthrie, 2000) (e.g., Bryant & Harvey, 1998; Harvey & Bryant, 1999a, 1999b), or a combination of the CAPS and the Peritraumatic Dissociative Experiences Questionnaire (PDEQ: Marmar, Weiss, & Metzler, 1997) (e.g., Fuglsang et al., 2002; Mellman et al., 2001). Although Fuglsang et al. and Mellman et al. utilize the same tools to diagnose ASD, it may be the case that the use of the self-report PDEQ by the Mellman study (rather than the interview version chosen by the Fuglsang study) contributed to the disparate findings.

2.3. *The range of psychopathology*

It is now well established that PTSD is not the only psychiatric condition that may develop following traumatic exposure and that, when it does occur, comorbidity in PTSD is the norm rather than the exception. In large community samples, for example, between 80% and 85% of individuals with a diagnosis of PTSD also meet criteria for at least one other psychiatric condition (Brady, Killeen, Brewerton, & Lucerini, 2000; Creamer, Burgess, & McFarlane, 2001). A recent meta-analysis of psychopathology following civilian trauma suggested that increased rates of generalized anxiety disorder, substance abuse, phobias, and major depressive disorder occur following exposure to traumatic events, while panic disorder and dysthymic disorder do not significantly increase (Brown, Fulton, Wilkeson, & Petty, 2000). The available literature on the prevalence of these other psychiatric conditions following physical injury is very limited. Few studies report prevalence and, of those that do, many do not clarify whether the disorder is the sole condition or comorbid with PTSD or another diagnosis. Exceptions to this are the findings reported by Blanchard et al. (1995), who comprehensively reports PTSD comorbidity with a range of anxiety disorders, mood disorders, and substance use disorders. However, the potential selection bias associated with this study's sample renders the prevalence rates unclear.

In terms of mood disorder, Schnyder, Moergeli, Trentz, Klaghofer, and Buddeberg, (2001) reported a 9% depression rate at 12 months. Shalev et al. (1998) reported a rate of major depressive episode of 14% at 4 months posttrauma. Holbrook et al. (1998) reported depression rates at discharge from hospital of 60%, dropping to 31% by 6 months after the trauma. Depression is also one of the most common comorbid conditions in PTSD generally (Creamer et al., 2001). The few studies of injured populations that have reported comorbid depression have pointed to rates as high as 53% in those with a PTSD diagnosis (Blanchard et al., 1995; Koren et al., 1999; Shalev et al., 1998). A small number of studies have identified increased rates of anxiety disorders and substance use disorders other than PTSD (Blanchard et al., 1995; Mayou, Bryant, & Ehlers, 2001).

Again, these studies are subjected to similar methodological problems as described in the PTSD and ASD sections. For example, in the above studies utilizing injury populations, few if any describe any attempt to differentiate psychogenic from organic symptoms, state population representation, or control for current medication. It may be speculated that the high reported levels of depression upon discharge in the Holbrook et al. (1998) study may be associated with the use of a self-report tool to diagnose depression and thus failure to differentiate organic from psychogenic symptoms.

3. Methodological issues

In the above literature review, we have briefly highlighted some potential methodological difficulties in many studies utilizing an injured population. The following section elaborates on these and other methodological issues which may potentially be associated with the variability in reported prevalence rates of posttraumatic conditions and impact on prediction studies. In this section, methodological issues pertaining to physical injury, TBI, subsequent traumatization, use of narcotic analgesia, timing of the assessment, sample selection, and the role of litigation will be covered.

3.1. *The role of physical injury*

Injured patients may report a range of symptoms that can be associated with either organic or functional pathology. The difficulty of differentiating between physical and psychological causes of specific symptoms may result in misdiagnosis and may contribute to the observed variability in reported prevalence rates. An assumption that a given symptom is part of a psychiatric condition, with no attempt to elucidate other potential etiology, will result in artificially inflated rates of psychopathology.

Hyperarousal is a core feature of posttraumatic anxiety, including PTSD. Sleep disturbance, concentration difficulties, and irritability are all features of both PTSD and depression. In acutely hospitalized patients, however, these problems may be secondary to pain (Raymond, Nielsen, Lavigne, Manzini, & Choiniere, 2001), noise in the ward environment (Aaron et al., 1996), or the injury (Haboubi, Long, Koshy, & Ward, 2001). These explanations should be ruled out before these symptoms are used to contribute to a psychiatric diagnosis.

Whereas behavioral avoidance is central to PTSD and most other anxiety disorders, it can be difficult to accurately assess in patients who have not yet left hospital and have not been exposed to many anxiety-provoking situations. It is possible that different rates of avoidance symptoms may be reported depending on the inpatient or outpatient status of the patient. Several studies have found low levels of avoidance in inpatient injury survivors (e.g., Fuglsang, 2000; Shalev, Peri, Canetti, & Schreiber, 1996). However, to date there is little empirical evidence to suggest why this is the case. Even after discharge from hospital it may be difficult to reliably differentiate psychologically based avoidance from that caused by medical factors. Behaviors that resemble avoidance may be more a function of physical limitations than psychological reasons. For example, “avoiding” driving because of the potential for seizures or having a leg in plaster should not be interpreted as avoidance. In making a diagnosis of PTSD, it is critical to confirm that the avoidance is a function of attempts to prevent exposure to distressing reminders of the trauma.

Also important in making a PTSD diagnosis is the differentiation between intrusive memories of the traumatic event (which invade consciousness) and more voluntary rumination about the incident and its potential sequelae. Several cognitive theorists have touched on these different phenomena in the context of PTSD (e.g., Brewin, Dalgleish, & Joseph, 1996; Creamer, Burgess, & Pattison, 1992; Ehlers & Clark, 2000). Not surprisingly, many patients

report a preoccupation with the injury-causing event and the potential impact upon their life. This represents intentional thinking about the incident and its ramifications elaborated within a broader autobiographical contextual framework. This should be differentiated from the ego-dystonic, intrusive re-experiencing of the traumatic event itself, usually in an unmodified and unelaborated form, that is central to the phenomenology of ASD and PTSD.

A final point to consider is the interface between body injury parameters (e.g., surface of burns) and psychological responses. A reasonably robust finding is that injury severity (as measured by Injury Severity Score; Baker, O'Neil, Haddon, & Long, 1974) is a poor predictor of later psychopathology (Koren et al., 1999; Mayou et al., 2001; Schnyder, Moergeli, et al., 2001), although it may have indirect effects (Michaels, Michaels, Zimmerman, et al., 1999). However, there is emerging evidence that other dimensions of injury such as injury mechanism (Holbrook, Hoyt, Stein, & Sieber, 2001), the specific body areas affected (e.g., face) (Fukunishi, 1999), and/or permanent bodily changes such as disfigurement (Madianos, Papaghelis, Ioannovich, & Dafni, 2001) are associated with poor psychological adjustment.

3.2. *Traumatic brain injury*

Presence of a TBI presents considerable difficulty when assessing psychological responses to injury. The effects of TBI share several common symptoms with psychiatric disorders. There is considerable overlap between the dissociative symptoms described in ASD and symptoms that result from TBI, including derealization, reduced awareness of one's surroundings, depersonalization, and amnesia (Alexander, 1995; Goronwall & Wrightson, 1980). Numerous hyperarousal symptoms can also be part of the postconcussive syndrome following TBI, including concentration deficits, irritability, sleep disturbance, and agitation (Bohnen & Jolles, 1992). It is also possible that survivors of TBI can experience spontaneous intrusive imagery as a result of frontal lobe pathology (Kopelman, 1987).

The issue of amnesia is particularly important in cases of TBI. Both ASD and PTSD include psychogenic amnesia as part of the diagnostic criteria. In patients with TBI, the risk of false-positive identification of dissociative amnesia is increased because of the difficulty in differentiating between organic and psychogenic amnesia (Sivec & Lynn, 1995). Previous research has attempted to deal with this problem in different ways. Some studies that have diagnosed PTSD in participants who have experienced a TBI fail to specify how they dealt with this problem (e.g., Bryant & Harvey, 1999; Mayou, Black, & Bryant, 2000; McMillan, 1996). In other studies, an a priori decision was made to exclude dissociative amnesia as a possible symptom of ASD and PTSD in individuals who have potentially experienced a TBI (e.g., Bryant, Marosszeky, Crooks, Baguley, & Gurka, 2001; Bryant, Marosszeky, Crooks, & Gurka, 2000; Harvey & Bryant, 2000) or where it is difficult to determine if reported amnesia is a function of dissociative response or organic injury (Harvey & Bryant, 1998, 1999a; Schnyder, Moergeli, Klaghofer, et al., 2001; Schnyder, Moergeli, Trentz, et al., 2001). The PTSD module of the CIDI (Peters et al., 1996), one of the most widely used instruments in large-scale epidemiological research, includes amnesia for those without head injury and

excludes amnesia for those with TBI. Regrettably, there is no reliable means of differentiating between functional and organic amnesia following TBI (Bryant, 2001).

Amnesia for the event secondary to TBI also raises questions about the nature of subsequent re-experiencing symptoms. There is increasing evidence that people who are amnesic of the event can display distress or physiological reactivity in response to trauma reminders, even if they have no verbal or visual memories of the event (Bryant, Marosszky, et al., 2000). Alternately, patients following TBI may report reconstructions of the event that are not historically accurate but reflect attributions that are made in the vacuum of amnesia caused by the brain injury (Harvey & Bryant, 2001). Although these manifestations of PTSD may appear atypical, they need to be recognized as valid indications of PTSD following TBI.

Assessment of psychiatric morbidity following TBI is often variable because of different approaches employed in this assessment. There is convergent evidence that patients with TBI tend to underestimate their symptoms. Further, TBI may mask psychiatric symptoms. For example, damage to the frontal lobe may produce expressive aprosody that may reduce the expression of dysphoria (van Reekum, Cohen, & Wong, 2000). Similarly, cognitive impairment may contribute to inaccurate completion of self-report scales of psychological functioning (McMillan, 2001).

Another possible source of variability is the extent to which researchers agree on the appropriateness of Criterion A2 in the diagnosis of ASD/PTSD for trauma survivors with TBI. Criterion A2 stipulates that, “the person’s response involved intense fear, helplessness, or horror.” It may be argued that individuals who are amnesic of the event that caused their injury are, by definition, unable to meet this criterion because they do not respond to the event itself because of their lack of awareness. There is currently little research to inform the operational definition of Criterion A2 for individuals who are amnesic of the event. It may be argued that a subsequent reaction of intense fear, helplessness, or horror should satisfy the DSM-IV requirement.

3.3. Subsequent and prior traumatization

The majority of research in psychological response to physical injury has focused on the injury-producing incident as the main cause psychological difficulty. However, it is important to recognize the potential role of subsequent traumatization related to factors that occur in the posttrauma period. For example, trauma may be experienced secondary to medical factors, including uncontrollable pain (Schreiber & Galai Gat, 1993) or pain related to treatment (as in burns victims; Yu & Dimsdale, 1999), ongoing medical problems (Ehlers et al., 1998), or physical disability (Michaels et al., 2000). Little research has examined the influence of these potentially stressful experiences on reported posttraumatic disorders after injury. While beyond the scope of this review, it is recognized also that pretrauma variables specific to this population—particularly prior traumatization—may increase risk for subsequent psychopathology. Zatzick, Kang, et al. (2002), for example, noted that hospitalized injury survivors were significantly more likely to have a history of prior traumatic events than the general population.

3.4. Narcotic analgesia

A significant proportion of physically injured patients will be administered analgesic medication. The side effects of narcotic analgesia may include sweating, confusion, mood changes, restlessness as well as sedation, drowsiness, disorientation, depersonalisation, derealization, and nausea. These symptoms overlap with a range of dissociative and anxiety reactions. For example, the derealization, depersonalisation, reduced awareness of one's surroundings, irritability, and concentration deficits described in the ASD criteria may be attributed to the effects of opioids. Narcotic analgesia may also serve to mask a variety of symptoms in the acute phase. The sedative effects of analgesics may limit awareness of information about symptoms and patients may have limited capacity for accurate symptom reporting.

3.5. Timing of assessment

The timing of the assessment after the injury is critical. There is convergent evidence that different rates of psychological morbidity will be observed depending on the temporal proximity of the assessment to the traumatic injury. Whereas there is evidence that many PTSD symptoms abate in the initial weeks after trauma (Foa & Riggs, 1993; Solomon, Mikulincer, & Benbenishty, 1989), there is also preliminary evidence that certain symptoms may emerge after the initial weeks (Shalev et al., 2000). Despite the importance of timing of assessments, many studies report only the range of the time when the assessments were conducted. Few studies actually use the timing of the assessment as a variable that may influence observed incidence rates of morbidity.

3.6. Sample selection

Many studies fail to take a representative sample of the injured population. This issue is particularly relevant for gender distributions. For example, 67–75% of hospitalized physically injured patients in North America are male (National Centre for Injury Prevention and Control, 2000). Accordingly, a study that has a male/female ratio of 1:2 is not representative of the general (North American) injured population. An overrepresentation of females in an injured sample has the possibility of skewing the data because women are more likely than males to develop psychopathology following trauma (Breslau, Chilcoat, Kessler, Peterson, & Lucia, 1999; Breslau, Davis, Andreski, Peterson, & Schultz, 1997). Further, recent evidence points to the greater incidence of PTSD symptomatology in females following MVAs (Fullerton et al., 2001), although not all studies to date agree that female survivors are more likely to develop PTSD at the aftermath of accidental injury (e.g., Kessler, Sonnega, Hughes, & Nelson, 1995) or road traffic accidents (Freedman et al., 2002). This factor is often a problem when the research population is self-selected, such as those presenting for treatment or volunteers responding to a request for research participation.

Related to the issue of sample selection is the issue of sample size. Many studies utilize samples of less than 200. This pattern is problematic because it increases the probability of outliers influencing the incidence rates. Prospective studies that attempt to index the

longitudinal course of response to traumatic injury are particularly flawed by small sample sizes because predictive analyses that attempt to identify the hallmarks of acute reactions that lead to chronic disorders are limited by small participant numbers. Small sample sizes also limit our ability to understand less frequent presentations of trauma response. For example, although delayed-onset PTSD is often discussed, it is poorly understood. Most prospective studies that have indexed delayed-onset PTSD after traumatic injury have been limited to less than 10 cases because only approximately 5% of cases develop this condition (Bryant & Harvey, 2002; Buckley, Blanchard, & Hickling, 1996; Ehlers et al., 1998). Although many specific questions pertaining to posttraumatic stress can be addressed with modest sample sizes, the outstanding issues concerning the capacity of acute reactions to predict longer-term functioning require very large cohorts. Ideally, studies that address the predictive nature of acute trauma reactions should comprise samples of several thousands of participants in order to have sufficient power to reliably index the predictive power of acute reactions. Moreover, samples of this magnitude will allow more rigorous investigation of the common forms of trauma response. This highlights the benefits of collaborative, multisite trials in investigating this population and/or the use of standardized methodologies so that data sets can be subsequently combined across studies.

3.7. Role of litigation

In many jurisdictions there is the potential for compensation following traumatic injury. Since an early influential study by Miller (1961) found that 41 out of 45 claimants resolved their “psychoneuroses” after their claims were settled, the issue of psychological disorder following injury has been clouded by the possibility that reported impairment is inflated by motivation to receive compensation. There is conflicting evidence concerning the relationship between involvement in litigation and the reported incidence of posttraumatic disorders. Some studies indicate that compensation issues do not significantly influence PTSD status (Kelly & Smith, 1981). Several longitudinal studies of MVA victims have found no difference in PTSD over time between those who did and did not settle their compensation claims (Blanchard et al., 1998). There is also evidence, however, that symptom exaggeration is particularly prevalent in compensation-seeking individuals (Frueh, Hammer, Cahill, Gold, & Hamlin, 2000; Frueh, Smith, & Baker, *in press*) and that PTSD symptoms are higher in those involved in litigation (Bryant & Harvey, 1995). The studies that are available are from different jurisdictions with very distinct compensation systems. There is a need for future research to index the nature and role of litigation involvement in the incidence of posttraumatic disorders because this will provide a more accurate index of psychological injury and will inform us about the role of litigation in reported symptoms after injury.

4. Recommendations for future research

We now turn to a number of recommendations for future research into psychological disorders following traumatic injury. The following recommendations are made with the aim

of providing a guide that will permit some uniformity in approach and comparability between studies. We emphasize that while we suggest operational definitions where possible, we recognize in many cases operational definitions are not available and as a result decisions are made on clinical judgement. At a minimum, we suggest that authors describe how they have dealt with some of the key methodological issues.

4.1. Injury severity

Future studies should report injury severity of all participants. Indexing the level of injury in the sample and measuring the role of injury in psychological response requires standardised measures of severity of physical injury. The two most widely accepted injury severity scales available at this time are the Abbreviated Injury Scale ([American Association for Automotive Medicine, 1980](#)) and the Injury Severity Score ([Baker et al., 1974](#)). Both scales provide an understandable index of injury severity that can allow research studies to explicitly report the nature of physical injury. Objective injury severity should also, where possible, be supplemented by measures of subjective responses at the time of the trauma (e.g., fear and other negative appraisals) which have been shown to predict subsequent PTSD ([Ehlers et al., 1998](#); [Schnyder, Moergeli, Klaghofer, et al., 2001](#); [Schnyder, Moergeli, Trentz, et al., 2001](#)).

4.2. Traumatic brain injury

Research that includes TBI populations often fails to use internationally recognized indices of the level of brain injury. This ambiguity in a range of studies has contributed to the mixed findings concerning the relationship between TBI and psychiatric disorder. The Glasgow Coma Scale (GCS; [Teasdale & Jennett, 1974](#)) is widely used by paramedics around the world to indicate levels of consciousness and is often used as an indicator of TBI. The American Congress of Rehabilitation Medicine ([American Congress of Rehabilitation Medicine, 1993](#)) defines mild TBI as requiring at least one of the following: a loss of consciousness of approximately 30 min or less, a GCS score of 13–15 after 30 min, and posttraumatic amnesia (PTA) not greater than 24 h. Moderate and severe TBI are less well defined, but are generally dependent on GCS score and length of PTA.

4.3. Timing of the assessment

The timing of the psychiatric assessment should be guided by the research question. If the purpose is to identify acute stress reactions and index the course of posttraumatic adjustment, then it is appropriate to assess reactions in the initial days and weeks after the injury. If the aim is to index more stable psychological disorder, however, it is important to delay the assessment until transient stress reactions have abated (indicated, for example, by stability of symptoms over a period of a few weeks) and the individuals are in a setting that reflects their normal environment (ideally back in their own home). A more reliable assessment is likely if the individual has been exposed to a range of potential trauma reminders, as well as to the normal demands of everyday life, rather than being in a protected environment such as a

hospital. It is imperative that researchers specify when assessments are conducted in relation to the precipitating stressor, as well as the setting in which assessments occur.

4.4. Assessment of psychological symptoms

Researchers should adopt a common operational definition for each posttraumatic symptom. For example, the parameters for distinguishing between functional and organic bases of a reported symptom should be standardized within the study and reported. This practice would allow the role of amnesia, concentration deficits, or derealization to be clearly understood between studies. Attempts should also be made to establish that symptoms such as altered sleep patterns, appetite changes, poor concentration, and decreased interest in activities are not simply a function of the physical recovery process or the hospital environment. As a general rule, if a symptom is better accounted for by an alternative explanation, it should not be categorized as a psychological symptom. Although every attempt should be made to operationalize and standardize these decisions within the study, it is recognized that, in many cases, this differentiation will come down to clinical judgement. At the very least, authors should report the manner in which the key differential diagnostic decisions have been handled.

4.5. Medication status

Psychological assessments should be conducted at least 24 hours after cessation of narcotic analgesic medication. The confound of dissociation and altered awareness secondary to narcotics would be minimized by this practice. At the very least, the time spent on narcotic analgesia (both intravenous and per oral), the current dosage, and time since last dose should be reported as well as any association between narcotic intake and symptom levels. Other potentially confounding effects of medication (e.g., steroids may affect mood or cause psychomotor agitation) should similarly be noted.

4.6. Sample representativeness

In order to obtain representative samples, consecutive admissions should ideally be used or, at the very least, random allocation to the research should be conducted. To demonstrate representativeness, samples should be compared to the larger population from which they came. For example, a sample of injured emergency department admissions could be compared to all injured emergency department admissions for that year. Further, future studies should attempt to study larger samples, preferably with participant numbers in excess of 1000.

4.7. Measurement of outcome

As research advances, it is important to consider a broader array of outcomes. Quality of life, subjective perception of recovery, and return to work are generally not systematically

indexed by researchers. The validity of findings based solely on psychopathology is questionable in that they fail to address other major parameters relevant to this population. Seminal research in this area has been conducted by various research groups (e.g., Holbrook, Anderson, Sieber, Browner, & Hoyt, 1998; Michaels et al., 1998; Zatzick, Jurkovich, Gentilello, Wisner, & Rivara, 2002).

5. Conclusion

The evidence pertaining to psychological consequences of physical injury has expanded markedly in recent years. The extent to which the increasing evidence accurately develops our understanding of postinjury psychological functioning depends, however, on consistent and empirically defensible methodologies. This review has highlighted several issues that researchers need to consider if the current discrepancies between research outcomes are to be clarified. At the very least, recognizing and reporting on these core factors of postinjury response will facilitate understanding of the reported differences between studies. Considering the prevalence and impact of posttraumatic disorders after injury, it is critical that future research refines its methodologies in order to enhance our understanding and management of injured people.

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