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What is This?

Emotional Risk Factors and Postconcussional Disorder

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Emotional risk factors were examined in 129 litigant and nonlitigant patients diagnosed with Postconcussional Disorder (PCD) following Mild Traumatic Brain Injury (MTBI). According to Millon Clinical Multiaxial Inventory (MCMI) criteria, four subgroups emerged: (a) 14.7% met criteria for an Axis I disorder, (b) 24.8% for an Axis II disorder, (c) 24.0% for bout Axis I and II disorders, and (d) 36.4% fell below threshold for psychopathology. Thus, 63.5% endorsed emotional pathology. Of the four groups, those with both Axis I and II psychopathology presented the greatest number of emotional complaints in a clinical interview and manifested the lowest neurocognitive test scores. Motor skills, verbal abilities, memory functioning, and IQ were primarily affected. With the exception of post-morbid emotional complaints, no significant differences were identified between litigants and nonlitigants. Our analysis suggests that the combination of both Axis I and II psychopathology is associated with greater impairment following MTBI.

Keywords: mild traumatic brain injury (MTBI); postconcussional disorder (PCD); emotional factors; forensic evaluation

Epidemiological evidence suggests that in the United States alone, more than 1,300,000 individuals sustain a mild traumatic brain injury (MTBI) each year (Sosin, Sniezek, & Thurman, 1996). It is estimated that 10% to 20% of these individuals demonstrate persistence in postmorbid symptoms well beyond the typical initial 3-

month period (Alexander, 1995; Chapman, 1999; Kay, Newman, Cavallo, Ezrachi, & Resnick, 1992; Rutherford, 1989). A major challenge for clinicians who evaluate and treat those with persistent complaints subsequent to MTBI is the question of differential diagnosis. Postconcussional disorder (PCD) is composed of physical, cognitive, and

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emotional residua (American Psychiatric Association, 1994). Physical symptoms include headaches, dizziness, fatigue, a range of pain syndromes, and hypersensitivity to light, sounds, and medication. Cognitive difficulties may include disturbances in continuous information processing (such as multitasking or parallel processing), encoding and retrieval of information (learning new information, immediate memory, and word-finding difficulties), and executive functioning (fluid/flexible thinking, abstraction, and problem solving). Potential emotional symptoms include irritability, anger, depression, anxiety, compulsive behavior, and compromised psychosocial functioning (Gronwall & Wrightson, 1981; Kay et al., 1992; Levin et al., 1987; Rimel, Giordani, Barth, & Jane, 1982). Within the forensic realm, once the diagnosis of PCD has been established, the second critical challenge is differentiating between pre-, co- and postmorbid factors.

Two schools of thought have emerged as to the etiology of chronic symptomatology following MTBI (Ruff & Richardson, 1999). One side focuses on a primarily psychogenic explanation (e.g., Mittenberg & Strauman, 2000), whereas the other side focuses on a neurological one (e.g., Bigler, 2001). Regarding the former, it has been proposed that the response to the trauma itself, disruption of regular activities, and/or the development of a "shaken sense of self" lead to heightened emotional reactions that are responsible for the impairment in functioning beyond the typical 1- to 3-month recovery period (Kay et al., 1992; Ruff, Camenzuli, & Mueller, 1996). Other factors such as premorbid personality and the environmental demands placed on the individual are also potential contributors. Post-traumatic stress disorder (PTSD) is cited as the most prevalent comorbid emotional syndrome followed by depression, anxiety, and conversion disorder (Bryant & Harvey, 1999; Kay et al., 1992; Parker, 1996). Indeed, King (1996) reported a higher correlation for emotional measures than for traditional neuropsychological tests in predicting postconcussive symptoms following MTBI. In addition, Bryant and Harvey (1999) found an increase in postconcussive symptoms and emotional pathology in patients diagnosed with both an MTBI and PTSD. This combination resulted in more concentration deficits, dizziness, fatigue, headaches, sensitivity to sound, and visual disturbances relative to MTBI without PTSD.

Frequently in the forensic setting, it is suggested that PCD is, in fact, a psychological rather than neurological phenomenon (Mittenberg & Strauman, 2000). However, in response to this, Bigler (2001) noted, "as greater sophistication develops in neuroimaging and neuroimaging protocols to detect structure-function relationships, this type of a position will no longer be tenable" (p. 110). Clearly, positive findings on computed tomography (CT) and/or magnetic resonance imaging (MRI) make the diagnosis of PCD far less controversial. However, in many cases, structural neuroimaging techniques lack the required sensitivity to detect more diffuse or microscopic damage (Alexander, 1995; Bigler, 2001; Gordon et al., 1998; Lucas, 1998; National Institutes of Health [NIH], 2000). As a result, neuropsychological testing continues to play a key role in the determination of MTBI, and it is suggested that the evaluation of emotional factors should be an integral component.

In the context of litigation, the psychogenic explanation faces the following two options: (a) If psychological problems did not exist before the accident, then any acquired emotional problems (either brain based or reactive) should be attributed to the trauma; or (b) if psychological problems existed prior to the MTBI, then they cannot be attributed to the accident. Emotional risk factors, such as personality pathology or depression, may affect the presentation of clinical symptoms following PCD (Hibbard et al., 2000; Kay et al., 1992; Ruff et al., 1996). It has been suggested that people with such preexisting emotional factors may be at greater risk for experiencing more chronic symptomatology following PCD (the so-called eggshell perspective). In one of the few studies to examine the potential impact of preexisting emotional factors, Hibbard et al. (2000) used the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) (American Psychiatric Association) Personality Disorders (SCID-II) to investigate the relationship between emotional variables and PCD. By modifying the SCID-II to reflect premorbid characteristics, the authors found a premorbid incidence rate of 24% for one or more Axis II personality disorders. Of these patients, 15% met the criteria for antisocial, 6% for obsessivecompulsive, 4% for paranoid, 3% for narcissistic, and 1% each for histrionic, borderline, and schizoid. Despite its potential significance, the role of pre- and postmorbid emotional factors is frequently overlooked in both forensic and clinical settings.

The aim of the present study was to explore the incidence and potential influences of emotional residua in patients with PCD. We first explored the relationship between reported emotional complaints presented in an interview format and psychopathology assessed via psychodiagnostic testing. Toward better understanding the effects of premorbid factors on the development of PCD, we also tested the hypothesis that individuals exhibiting evidence of premorbid personality pathology would exhibit more pronounced cognitive residua than patients without significant personality pathology. Finally, we included both liti-

TABLE 1
Demographic Variables for Litigant, Nonlitigant, and Total Sample

	Litigant	Nonlitigant	Total
N	98	31	129
Male	53	17	70
Female	45	14	59
Age	41.2	44.2	42.0
Years of education	14.0	15.5	14.4
Chronicity (months)	17.2	12.0	16.0

gant and nonlitigant cases in our sample to explore the role that litigation may play in the presentation of emotional complaints within the forensic setting.

METHOD

Participants

From our archives of clinical outpatients, we selected 129 patients that were diagnosed with both an MTBI and persistent PCD (see Table 1 for patient characteristics). All of the patients met the following criteria for the diagnosis of MTBI:

- a sustained alteration or loss of consciousness that did not exceed 30 minutes;
- posttraumatic amnesia that did not exceed 24 hours;
- if the Glasgow Coma Scale was administered, scores did not fall below 13; and
- no history of neurosurgical intervention.

For the diagnosis of PCD, the following criteria were met: 3 or more months postinjury and evidence of cognitive deficits on neuropsychological testing. Additional selection criteria included ages between 18 and 80 years and no evidence of malingering based on test profiles (see below) and clinical judgment. Fifty-four percent of the sample was male and 46% was female. The etiology of the MTBI varied in this sample: 57.4% were the result of auto accidents, 14.7% were falls, 11.6% were blows to the head, 7.8% were pedestrians versus auto accidents, 4.7% were motorcycle accidents, 2.3% were sports related, and 1.6% were associated with assaults. The neuropsychological evaluations were administered an average of 16 months (SD = 13.5) after the date of injury, with a range from 3 months to 7 years. Ninety-eight patients (76.0%) were involved in active legal proceedings. The remaining patients represented strictly medical cases that were either selfreferred or referred by their physician (refer to Table 1 for litigant, nonlitigant, and total sample demographics).

Materials and Procedure

Patients were assessed with a clinical interview, a comprehensive neuropsychological evaluation, and the Millon Clinical Multiaxial Inventory–II (MCMI-II) (Millon, 1987) or the MCMI-III (Millon, Davis, & Millon, 1997). Evaluations were typically limited to 8 hours: 1 to 2 hours for the clinical interview, 4 to 6 hours of neurocognitive testing, and 1 to 2 hours of psychodiagnostic testing. The MCMI was used as a psychodiagnostic tool because of its conceptual consistency with *DSM-IV* criteria, attention to long-standing personality pathology, relatively robust psychometric properties, and clinical usefulness (Groth-Marnat, 1997; Millon & Davis, 1996; Millon et al., 1997).

A neuropsychologist with more than 20 years of experience (the second author of this article) conducted each of the 129 clinical interviews. During the interview, patients were asked to describe all physical, emotional, and cognitive symptoms directly attributed to the trauma. In addition, a thorough investigation of premorbid complaints was conducted.

The San Diego Neuropsychological Test Battery was administered, which is an expanded battery based on the core tests utilized in the Traumatic Coma Data Bank (Baser & Ruff, 1987; Lezak, 1995; Ruff & Crouch, 1991). Motivation was formally examined using the Rey 15-Item Test (Rey, 1964), the Dot Counting Test (Rey, 1941), and the Test of Memory Malingering (TOMM) (Tombaugh, 1996). All patients in this sample fell within normal limits on these measures.

RESULTS

Analyses of variance (ANOVAs) revealed no significant differences between litigant and nonlitigant groups with respect to age, education, gender, loss of consciousness, posttraumatic amnesia, neuropsychological test performance, MCMI status, the number of premorbid complaints (emotional, physical, and cognitive), or the number of postmorbid physical and cognitive complaints (all p > .05). The litigant group, however, did report a greater number of emotional complaints following their MTBI (F = 4.79, MS = 10.40, p = .03). In light of the overall consistency between the litigant and nonlitigant groups, the analyses described below were collapsed across groups to provide adequate power in the analyses and for ease of presentation. The difference in the number of postmorbid emotional complaints between litigant groups is explored in the discussion.

Data from the MCMI was analyzed to determine the incidence of emotional risk factors in the current PCD sample (see Table 2). With the aim of reliably capturing

TABLE 2 Frequency of MCMI Axis I and Axis II Elevations Among the Sample

	<u> </u>	
Axis I Disorder	Axis I Only	$Axis\ I + II$
Anxiety	6	3
Somatoform	1	1
ETOH	1	0
Major depression	2	0
Multiple Axis I elevations	9	28
Axis II Disorder	Axis II Only	$Axis\ I + II$
Schizoid	1	2
Depressive	0	1
Dependent	0	5
Histrionic	8	1
Narcissistic	7	2
Sadistic	0	2
Compulsive	9	3
Negativistic	1	2
Paranoid	0	1
Multiple Axis II elevations	6	12

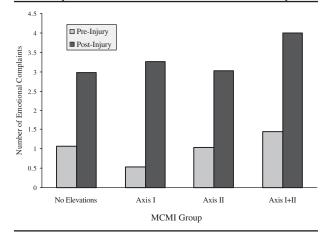
NOTE: MCMI = Millon Clinical Multiaxial Inventory; ETOH = Alcohol Dependence.

psychopathology, a conservative cutoff score of 85 or above was used to classify Axis I and II disorders. Of the 129 patients, 63.5% endorsed significant emotional symptomatology on the MCMI. The subgroups were distributed as follows: 24.0% endorsed both Axis I and Axis II pathology (Axis I + II group), 14.7% endorsed only MCMI Axis I pathology (Axis I group), 24.8% endorsed only MCMI Axis II pathology (Axis II group), and 36.4% of the sample had no base rate score above 85 (No Elevations Group).

In the Axis I group, 47% were elevated on two or more Axis I scales. The remaining patients were elevated on a single Axis I scale with anxiety disorder being the most frequent (see Table 2). Of the 32 patients endorsing solely Axis II pathology, the most frequent elevations were on the Histrionic, Narcissistic, and Compulsive scales. A smaller number of these individuals (23%) were elevated on multiple Axis II scales. However, in the Axis I + II group, 90% were elevated on multiple Axis II scales. Trends were observed for this group with respect to personality pathology, and the most frequent elevations were on the Dependent and Compulsive scales.

Next, we analyzed whether there were differences in the number of physical, emotional, and cognitive complaints among the four MCMI diagnostic groups. ANOVAs revealed no differences among the four groups with respect to the frequency of reported premorbid physical (F = 2.26, MS = 2.20, p = .11) or cognitive complaints (F = 0.96, MS = 4.25, p = .40). However, there was a signif-

FIGURE 1 **Number of Pre- and Postmorbid Emotional Complaints Across the Four MCMI Groups**



NOTE: MCMI = Millon Clinical Multiaxial Inventory.

icant difference in the frequency of reported premorbid emotional difficulties across the groups (F = 2.86, MS =4.25, p = .04; see Figure 1). Post hoc Tukey tests revealed a significantly higher number of emotional complaints by the Axis I + II group (M = 1.4) compared to the Axis I group (M = 0.5). The Axis II and No Elevations groups fell in between (M = 1.1 and 1.0, respectively).

We also analyzed the frequency of postmorbid physical, emotional, and cognitive complaints across the four MCMI groups. Regarding physical complaints, an ANOVA indicated a significant difference among the MCMI groups (F = 4.21, MS = 21.07, p = .01). Post hoc Tukey tests revealed that the Axis I group reported a significantly higher number of physical problems (M = 6.1)than the Axis II group (M = 4.1). Falling in between, the Axis I + II group had a mean of 5.5 complaints whereas those without MCMI elevations reported an average of 4.6 complaints.

There was also a significant difference among the MCMI groups with respect to the number of postmorbid emotional complaints (F = 3.43, MS = 7.38, p = .02). Numerically, the Axis I + II group had the greatest number of emotional complaints (M = 4.0), followed by the Axis I group (M = 3.3), the Axis II group (D = 3.0), and the No Elevations group (M = 3.0). Post hoc Tukey tests revealed that the Axis I + II group endorsed significantly more postmorbid emotional symptoms than the Axis II group.

With regard to the number of postmorbid cognitive complaints, the four MCMI groups did not significantly differ from each other (F = .84, MS = 2.20, p = .47). The Axis I group reported an average of 3.3 complaints, the Axis I + II group 3.2 complaints, the No Elevations group 3.1 complaints, and the Axis II 2.7 complaints.

TABLE 3 Mean Performance for MCMI Subgroups Across **Cognitive Domains**

	No Axis I or II $(N = 47)$	Axis I (N = 19)	Axis II $(N = 32)$	Axis I and II $(N = 31)$
Perception	3.81	3.94	3.95	3.74
Motor	3.52	3.22	3.55	2.57
Spatial	3.72	3.81	3.78	3.52
Verbal	3.73	3.69	3.75	3.33
Attention/				
concentration	3.48	3.30	3.53	3.09
Learning	3.08	2.97	2.95	2.74
Memory	3.54	3.10	3.24	2.83
Planning	3.74	3.57	3.63	3.32

NOTE: MCMI = Millon Clinical Multiaxial Inventory.

The four MCMI groups were also compared in terms of neuropsychological test performance. To reduce the likelihood of spurious significance among the more than 150 neuropsychological test variables, tests were categorized according to eight different cognitive domains (see Table 3). Test scores within each cognitive domain were ranked as follows: 0 = < 1st percentile, 1 = < 1st through 4th percentile, 2 = 5th through 9th percentile, 3 = 10th through 24th percentile, 4 = 25th through 75th percentile, and 5 = >75th percentile. Because of scoring differences, the domains of spatial abilities and verbal abilities were averaged and ranked as follows: 1 = seriously deficient, 2 = deficient, 3 = borderline, 4 = intact. Kruskal-Wallis tests were used to analyze differences between the four MCMI groups across the neurocognitive domains.

Out of the eight cognitive domains analyzed, the pattern of performance across the four MCMI groups differed for motor skills (p = .01), verbal abilities (p = .02), and memory (p = .01). The Axis I + II group performed consistently lower across all these domains (see Table 3). Post hoc tests (Mann-Whitney U) revealed that the Axis I + II group performed significantly lower than the Axis II and No Elevation groups on motor skills and verbal abilities (all p < .01). On the memory domain, the Axis I + II group as well as the Axis I group performed significantly lower than the No Elevation group (p = .01 and p = .03,respectively).

IQ scores from the Wechsler Adult Intelligence Scale-III were also analyzed across the four MCMI groups using one-way ANOVAs. There were significant differences across the groups with respect to verbal IQ, F(3,128) = 2.76, p = .04; performance IQ, F(3, 128) = 2.82, p = .04; and full scale IQ, F(3, 128) = 3.30, p = .02. Again, the Axis I + II group evidenced consistently lower scores. Post hoc Tukey tests revealed significantly lower scores in the Axis I + II group relative to the Axis II group across all three IQ measures (p = .04, .03, and .02 for verbal, performance, and full scale IQ, respectively). The Axis I + II group had averages of 98.9, 95.6, and 97.4 for verbal, performance, and full scale IQ, respectively; the Axis II group 108.6, 106.0, and 108.0, respectively; the Axis I group 103.4, 100.3, and 102.6, respectively; and the No Elevations group 106.7, 103.4, and 105.8, respectively.

Because the above results indicated the poorest performance in the Axis I + II group, we explored whether the number of elevated MCMI scales correlated with poorer neuropsychological performance. Significant correlations (Pearson product-moment correlation) between neuropsychological domain scores and the number of elevated MCMI factors were in the following areas: motor (r = -.29, p = .01), spatial skills (r = -.22, p = .04), verbal skills (r = -.29, p = .01), attention/concentration (r = -.24, p = .01)p = .01), memory (r = -.30, p = .01), planning (r = -.21, p = .01).04), verbal IQ (r = -.23, p = .01), performance IQ (r = .04) -.23, p = .01), and full scale IQ (r = -.25, p = .01). Thus, individuals with more MCMI elevations performed lower across a number of cognitive domains.

In light of the potential that the differences observed between the four MCMI groups stemmed from the severity of MTBI, an ANOVA was also conducted to determine if there were differences among the four MCMI groups with respect to LOC or PTA. No significant differences were observed for either the duration of LOC (F =.27, MS = .44, p = .83) or the length of PTA (F = .04, MS = .04) .06, p = .99).

DISCUSSION

In the current study, a majority of patients (63.5%) with MTBI and PCD endorsed significant emotional symptomatology on the MCMI. This finding represents a significantly greater percentage of psychopathology than found in the general public (American Psychiatric Associatrion, 1994). Our findings are consistent with previous studies of emotional factors in TBI (Hibbard et al., 2000; Van Reekum, Bolago, & Finlayson, 1996). In the current study, four groups emerged: patients with significant Axis I pathology, patients with significant Axis II pathology, patients with Axis I and Axis II pathology, and patients with no significant elevations. Consistent with the literature, the most common Axis I disorders were anxiety and depression (Bryant & Harvey, 1999; Hibbard et al., 2000; Parker, 1996). Patients with Axis II elevations were most frequently classified as compulsive, histrionic, narcissistic, or had multiple elevations. Patients with combined Axis I and II elevations endorsed a wider range of Axis II pathology overall.

Our study found no significant differences between the four MCMI groups with respect to the number of premorbid physical and cognitive complaints in a clinical interview. However, the Axis I + II group endorsed a higher number of pre- and postmorbid emotional difficulties in the clinical interview. These findings generally support the notion that premorbid emotional factors play a role in the clinical presentation of PCD (Cicerone & Kalmar, 1997). In the current study, we also found that patients with a combination of Axis I and Axis II pathology achieved overall lower neuropsychological test scores compared to the other groups. Specifically, scores in the domains of motor skills, memory, verbal abilities, and IQ were affected. Moreover, there was a significant relationship between emotional pathology and cognitive performance across the patient sample; the greater the number of elevated MCMI scores, the lower the neuropsychological performance scores.

The current study included both litigants and nonlitigants. Across a wide range of demographic, psychodiagnostic, and neuropsychological test scores, there were no significant differences between the groups. The one factor on which the two groups differed was the number of postmorbid emotional complaints present during the clinical interview. Caution has been raised regarding the use of self-reported evidence in TBI litigant populations. Two studies (Iverson, King, Scott, & Adams, 2001; Lees-Haley & Brown, 1996) reported higher base rates of cognitive complaints among litigants than nonlitigants in head injury cases. However, Lees-Haley and Brown (1996) also noted the possible effects of the emotional response to the litigation processes itself, citing a high frequency of both depression and stress in TBI litigant populations. This study is consistent with our findings of increased emotional, but not cognitive or physical, complaints in litigants and underscores the need for careful consideration of comorbid emotional factors.

In litigation, preexisting emotional maladjustments are of particular relevance because the patient should not be compensated for premorbid problems. The diagnosis of premorbid characterological problems is most challenging because neuropsychologists administer their examinations following the TBI. Nonetheless, once Axis II psychopathology is revealed on MCMI or MMPI profiles, it is then typically applied in two disparate directions. On one side, defense experts may argue that the preexisting emotional problems caused the PCD. Conversely, plaintiff experts may argue the eggshell perspective, pointing to the same preexisting emotional problems as vulnerabilities or risk factors for postmorbid difficulties.

Although no single profile was revealed in our study, three personality disorders did emerge in the Axis II group: compulsive, histrionic, and narcissistic. With respect to personality styles, patients with compulsive personality disorders tend to be disciplined and perfectionist. Setbacks are viewed as unacceptable and must be overcome by working even harder. Because the healing process following MTBI is not directly under their control, this coping mechanism is frequently ineffective. A prolonged lack of progress is readily viewed as a personal failure, or even a crisis, that renders the individual powerless. Thus, acceptance of reduced functioning levels is intolerable, and their coping strategy of working hard is ineffective. Patients with histrionic personality disorders are motivated to seek support and recognition from others. Yet PCD often results in negative attention and family members, friends, and health care professionals are frequently unable to adequately satisfy their needs. Indeed, their coping style of dramatic presentation is commonly viewed as somatization by forensic evaluators. The perceived lack of support received during forensic evaluations can result in an increase in their coping response of dramatic symptom presentation. Patients with narcissistic personality disorders may cope by being overly competent, grandiose, or even manipulative to compensate for feelings of insecurity. With high expectations for themselves and a history of avoiding the acknowledgment of personal weaknesses, they are typically ill-equipped to cope with the personal challenges that may arise immediately following their MTBI. Displacing perceived weaknesses on an external event, such as the MTBI, rather than personal culpability or failure may stave off insecurity and intolerable "narcissistic injuries."

There were several limitations in the current study. First, our patient sample was not representative of the MTBI population as a whole because many patients fell within the so-called miserable minority. That is, they experienced symptoms beyond 3 months post-MTBI. Second, because the data were collected over a 7-year period, two different versions of the MCMI were utilized. Perhaps the most significant limitation of the current study was that Axis II disorders were assessed following the MTBI. Despite its frequent clinical use in neuropsychological evaluations, the MCMI was not normed for the assessment of brain-injured patients, raising the question of possible validity issues. It is possible that acute emotional turmoil may have exacerbated responses to those questions that loaded on long-term personality pathology. However, this is the typical practice that neuropsychologists employ. To a degree, this problem was addressed in the current study with the addition of a clinical interview in which patients were asked to enumerate their pre- and postmorbid emotional, physical, and cognitive problems. We are currently developing a clinical questionnaire that measures pre- and postmorbid symptoms separately to address this challenge in the assessment of PCD (Ruff Neurobehavioral Inventory) (Ruff, 2003). It is hoped that this tool will be beneficial to the evaluation of MTBI patients in both the forensic and nonforensic setting.

The current findings strongly suggest that emotional risk factors should be differentiated when diagnosing patients with PCD. Because proposed diagnostic criteria for PCD were first introduced in the DSM-IV, we believe that neuropsychologists should utilize, investigate, and refine this construct. Because PCD is not a neurocognitive deficit per se but can affect multiple functions, we recommend that neuropsychologists, as a rule, use the multiaxial approach of the DSM. Concurrent Axis I disorders, such as generalized anxiety disorder and major depression, for example, should be identified during the examination process. External stressors, such as strained family relations, inadequate finances, and unemployment, should be noted on Axis IV. Physical residua are frequently a part of a PCD and the compounding effects of pain or orthopedic injuries should be identified on Axis III. Attention to these designations will also encourage clinicians to recommend treatments for these coexisting problems.

The current findings support the cumulative model of PCD (Ruff, et al., 1996; Ruff & Richardson, 1999). This model views physical, emotional, cognitive, psychosocial, vocational, financial, and recreational setbacks as cumulative stressors that exacerbate premorbid factors. For the 10% to 20% who fall in the miserable minority, pre- and postmorbid symptoms combine to a level that prevents these individuals from returning to their premorbid functioning. We believe a comprehensive approach that incorporates the understanding of the entire individual (cognitive, physical, and emotional history) is critical to the evaluation, treatment, and continued understanding of PCD.

REFERENCES

- Alexander, M. P. (1995). Mild traumatic brain injury: Pathophysiology, natural history, and clinical management. *Neurology*, 45, 1253-1250.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Baser, C. N., & Ruff, R. M. (1987). Construct validity of the San Diego Neuropsychological Test Battery. Archives of Clinical Neuropsychology, 2, 13-32.
- Bigler, E. D. (2001). The lesions in traumatic brain injury: Implications for clinical neuropsychology. Archives of Clinical Neuropsychology, 16, 95-131.
- Bryant, R. A., & Harvey, A. G. (1999). Postconcussive symptoms and posttraumatic stress disorder after mild traumatic brain injury. *Journal of Nervous and Mental Diseases*, 187, 302-305.
- Chapman, E. H. (1999). Homeopathic treatment of mild traumatic brain injury: A randomized, double-blind placebo-controlled clinical trial. *Journal of Head Trauma Rehabilitation*, 14(6), 222-231.

- Cicerone, K. D., & Kalmar, K. (1997). Does premorbid depression influence post-concussive symptoms and neuropsychological functioning? *Brain Injury*, 11, 643-648.
- Gordon, W. A., Brown, M., Sliwinski, M., Hibbard, R., Patti, N., Weis, M., et al. (1998). The enigma of "hidden" traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 13(6), 39-56.
- Gronwall, D., & Wrightson, P. (1981). Memory and information processing capacity after closed head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 44, 889-895.
- Groth-Marnat, G. (1997). *Handbook of psychological assessment*. New York: John Wiley.
- Hibbard, M. R., Bogdany, J., Uydal, S., Kepler, K., Silver, J. M., Gordon, W. A., et al. (2000). Axis II psychopathology in individuals with traumatic brain injury. *Brain Injury*, 14, 45-61.
- Iverson, G., King, R., Scott, J., & Adams, R. (2001). Cognitive complaints in litigating patients with head injuries or chronic pain. *Journal of Forensic Neuropsychology*, 2(1), 19-30.
- Kay, T., Newman, B., Cavallo, M., Ezrachi, O., & Resnick, M. (1992). Toward a neuropsychological model of functional disability after mild traumatic brain injury. *Neuropsychology*, 6, 371-384.
- King, N. (1996). Emotional, neuropsychological, and organic factors: Their use in the prediction of persisting postconcussion symptoms after moderate and mild head injuries. *Journal of Neurology, Neurosurgery, and Psychiatry*, 61, 75-81.
- Lees-Haley, P., & Brown, R. (1996). Neuropsychological complaint base rates of 170 personal injury claimants. Archives of Clinical Neuropsychology, 8(3), 203-209.
- Levin, H. S., Mattis, S., Ruff, R. M., Eisenberg, H. M., Marshall, L. F., Tabaddor, K., et al. (1987). Neurobehavioral outcome following minor head injury: A three-center study. *Journal of Neurosurgery*, 66, 234-243.
- Lezak, M. D. (1995). Neuropsychological assessment (3rd ed.). New York: Oxford University Press.
- Lucas, J. (1998). Traumatic brain injury. In P. Synder & P. Nussbaum (Eds.), Clinical neuropsychology (pp. 243-265). Washington, DC: American Psychological Association.
- Millon, T. (1987). MCMI-II manual. Minneapolis, MN: NCS.
- Millon, T., & Davis, R. D. (1996). *Disorders of personality: DSM-IV and beyond*. New York: John Wiley.
- Millon, T., Davis, R. D., & Millon, C. (1997). Manual for the Millon Clinical Multiaxial Inventory–III (MCMI-III) (2nd ed.). Minneapolis, MN: NCS.
- Mittenberg, W., & Strauman, S. (2000). Diagnosis of mild head injury and the postconcussion syndrome. *Journal of Head Trauma Rehabili*tation, 15, 783-791.
- National Institutes of Health. (2000). NIH consensus statement on rehabilitation of persons with traumatic brain injury. Available at http://consensus.nih.gov
- Parker, R. (1996). The spectrum of emotional distress and personality changes after minor head injury incurred in a motor vehicle accident. *Brain Injury*, 10, 287-302.
- Rey, A. (1941). L'examen psychologique dans les cas d'encephalopathie traumatique. Archives de Psychologie, 28, 286-340.
- Rey, A. (1964). L'examen clinique en psychologie. Paris: Presses Universitaires de France.
- Rimel, R., Giordani, B., Barth, J., & Jane, J. (1982). Moderate head injury: Completing the clinical spectrum of brain trauma. *Neurosur*gery, 11, 344-351.
- Ruff, R. (in press). *Ruff neurobehavioral inventory*. Odessa, FL: Psychological Assessment Resources.
- Ruff, R. M., Camenzuli, L. F., & Mueller, J. (1996). Miserable minority: Emotional risk factors that influence the outcome of a mild traumatic brain injury. *Brain Injury*, 10, 551-565.
- Ruff, R. M., & Crouch, J. A. (1991). Neuropsychological test instruments in clinical trials. In E. Mohr & P. Brouwers (Eds.), *Handbook of clinical trials: The neurobehavioral approach* (pp. 89-119). Amsterdam: Swets & Zeitlinger.

- Ruff, R. M., & Hibbard, K. M. (2003). Ruff Neurobehavioral Inventory (RNBI). Lutz, FL, PAR.
- Ruff, R. M., & Richardson, A. M. (1999). Mild traumatic brain injury. In J. J. Sweet (Ed.), Forensic neuropsychology: Fundamentals and practice. Studies on neuropsychology, development, and cognition (pp. 315-338). Bristol, PA: Swets & Zeitlinger.
- Rutherford, W. H. (1989). Postconcussion symptoms: Relationship to acute neurological indices, individual differences, and circumstances of injury. In H. S. Levin, H. M. Eisenberg, & A. L. Benton (Eds.), Mild head injury (pp. 217-228). New York: Oxford University Press.
- Sosin, D. M., Sniezek, J. E., & Thurman, D. J. (1996). Incidence of mild and moderate brain injury in the United States, 1991. Brain Injury, 10,
- Tombaugh, T. N. (1996). Test of Memory Malingering (TOMM). Toronto, Ontario: Multi-Health Systems.
- Van Reekum, R., Bolago, I., & Finlayson, M. A. (1996). Psychiatric disorders after traumatic brain injury. Brain Injury, 10(5), 319-327.

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