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Irritability Following Traumatic Brain Injury

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Abstract

This study was undertaken to identify the clinical and pathoanatomical correlates of irritability in patients with closed head injuries. A consecutive series of 66 patients was assessed in hospital and at 3, 6, 9, and 12-month follow-ups. Patients fulfilling criteria for irritability were divided into 2 groups based on the immediate or delayed onset of their irritability and compared with patients without irritability for background characteristics, impairment variables, and lesion characteristics. There were 12 patients (18.2%) with acute onset irritability and 10 (15.1%) with delayed onset irritability. Acute onset irritability patients had a higher frequency of left cortical lesions. Delayed onset irritability patients showed a strong association with poor social functioning and greater impairment in activities of daily living. The findings suggest that post-brain injury irritability may have different causes and treatment in the acute and chronic stages.

Traumatic brain injury (TBI) accounts for many deaths each year and represents the leading cause of death among young adults. It is estimated that a head injury occurs every 15 seconds resulting in one death every 12 minutes (McGinnis, 1988). In the United States, the numbers of death from TBIs since 1980 have exceeded deaths from wars during the past 200 years (McAllister, 1992). Furthermore, many individuals survive the traumatic event only to be faced with permanent neurologic deficits, frequently requiring lifelong care. Others recover some degree of neurologic function and return to mainstream society but are left with evidence of behavioral and/or cognitive deficits (Long and Webb, 1995; Morton and Wehman, 1995). The neuropsychiatric consequences of TBI include decreased concentration, mood disorder, irritability, insomnia, headache, and fatigue (Chambers et al., 1996; Dikmen et al., 1986; Evans, 1992). Between 50 and 80% of patients who are admitted to a hospital following closed head injury will subsequently complain of one or more of the above symptoms (Evans, 1992; Long and Webb, 1995; Szymanski and Linn, 1992). Although most of these symptoms improve by 3 to 6 months after the injury, persistent behavioral symptoms and cognitive deficits occur in a minority of patients. Risk factors for persisting neuropsychiatric sequelae include age over 40 years, lower premorbid intellect, fewer years of education, lower socioeconomic status, female gender, prior alcohol abuse, prior head injury, and multiple trauma (Evans, 1992). Irritability represents one of the most significant neuropsychiatric consequences of TBI because it may disrupt or prevent the patients from receiving the care that they need. Generally, irritability is thought to have multiple determinants, including both environmental and biological factors (Slagle, 1990). The precipitants and risk factors for irritability associated with TBI have been reported to include sociodemographic factors (*e.g.*, young male between the age of 15 and 34; Evans, 1992), psychosocial factors (*e.g.*, response to loss of job or social isolation; McLean et al., 1993), psychopathological factors (*e.g.*, occurrence of depression; Rohrbaugh et al., 1988), and neuroanatomical correlates (*e.g.*, damage to orbitofrontal lobe or anterior temporal lobes; Gualtieri, 1991; impaired cortical function; Long and Webb, 1995). Although there is a substantial literature examining the short-term and long-term neurobehavioral consequences of TBI, relatively few studies have specifically examined irritable behaviors following TBI. We, therefore, examined patients with TBI to look for factors associated with

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irritability. It has also been hypothesized that early behavioral symptoms following head injury are related to organic factors, whereas psychological factors stimulate the production of later onset symptoms. We thus followed patients with TBI over 1 year to compare the factors associated with acute onset irritability with those associated with delayed onset irritability.

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Methods

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Study Population

Sixty-six patients consecutively admitted to a shock trauma center with acute closed head injuries who did not meet exclusion criteria were included in the study. Patients were excluded if they had open head injuries, spinal cord injuries, multiple non-CNS system injuries such as multiple fractures, intra-abdominal hemorrhage, or collapsed lung or if they had altered consciousness or severe comprehension deficits that interfered with their ability to comprehend questions administered during a verbal interview (*i.e.*, who were not able to follow two-stage commands or who could not complete the first part of the Token Test; De Renzi and Vignolo, 1962). Information about the existence of previous mood disorder, as well as substance abuse or other psychiatric disorder that would fulfill DSM III-R criteria in the family or personal history, was specifically asked of each patient and relatives who were available at the time of interview. The diagnosis was not dependent upon whether or not the patient or relative was receiving treatment. None of the patients had a depressive disorder at the time of the TBI, and none received head injury as a result of a suicide attempt. Severity of brain injury was determined using the 24-h Glasgow Coma Scale (GCS) scores (*i.e.*, mild, GCS 12 to 15; moderate, GCS 8 to 11; and severe, GCS 3 to 7; Teasdale and Jennett, 1974). In addition, patients with GCS scores in the 12 to 15 range but who underwent intracranial surgical procedures or had focal lesions greater than 25 cc were considered to have moderate head injuries (Levin et al., 1987). Of the 66 patients, 22 (33.3%) had severe brain injury, 16 (24.3%) had moderate severity, and 28 (39.4%) had mild head injuries. GCS scores ranged from 3 to 15, with a median of 10 and an interquartile range of 6. Follow-up evaluations were carried out at 3, 6, 9, and 12 months after trauma. The number of patients in whom follow-up evaluations were obtained was 52 (78.8%) at 3 months and 43 (65.2%) at 6, 9, and 12 months. There were 58 patients (87.9%) with at least one follow-up visit, and 51 patients (77.3%) were seen on at least two of four follow-up visits. There were 8 patients (12.1%) who did not have any follow-up evaluations.

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Psychiatric Examination

After informed consent was obtained from all patients, detailed psychiatric examinations were performed approximately 1 month after injury. A semi-structured psychiatric interview was conducted by a fully trained psychiatrist using a modified version of the Present State Exam (PSE; Robinson et al., 1983; Wing et al., 1974). After this interview was completed, a diagnosis of major depression due to TBI or minor depression (DSM-IV research diagnosis of subsyndromal major depression) was made using DSM-IV criteria. Quantitative mood rating was obtained with the observer-rated Hamilton Depression Rating Scale (HDRS; Hamilton, 1960), a 17-item scale that measures psychological and physiological symptoms of depression. Cognitive function was measured using the Mini-Mental State Exam (MMSE; Folstein et al., 1975), which has been shown to be a reliable and valid means of assessing a limited range of cognitive functions in several medically ill or brain-injured populations (Robinson and Benson, 1981). MMSE scores range from 0 to 30, with scores below 24 indicative of clinically significant cognitive impairment. Impairment in activities of daily living was measured using the Johns Hopkins Functioning Inventory (JHFI; Robinson and Szetela., 1981). Scores on this scale range from 0 to 27, with higher scores indicating a greater degree of functional impairment. Social functioning was quantitatively assessed with the Social Functioning Exam (SFE) and the Social Ties Checklist (STC; Starr et al., 1983). The SFE is a semi-structured clinical interview that assesses the patient's satisfaction with their social functioning. It consists of 28 items exploring 13 dimensions of social functioning: relationships with significant others (identified closest person, spouse, children, and unrelated others in household), performance of home and family responsibilities, social and leisure activities, family solidarity, role performances in the work environment, use of community resources, health and illness experiences, and religion. Scores range from 0.00 to 1.00, with higher scores indicating less satisfaction during the month prior to the evaluation. The Social Ties Checklist assesses the number of social connections available to the patient. Scores may range from 0 to 10, with higher scores indicating less social support. The reliability and validity of each of these instruments has been demonstrated in brain-injured populations in prior publications (Robinson and Benson, 1981; Robinson and Szetela, 1981; Robinson et al., 1985).

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Neuroimaging

CT scans were obtained as part of the standard clinical evaluation of patients admitted to the emergency medical service system that administers the shock trauma center. Scans were usually done within the first day after trauma and repeated 1 or 2 weeks later. All scans were done on a GE-1010 scanner with standard 10-mm axial cuts parallel to the canthomeatal line. The nature of the lesion (*e.g.*, contusion, intraparenchymal bleeding, subarachnoid hemorrhage) was determined from the CT scan. All scans were read by a neurologist (F.M.) who was blind to the results of the psychiatric examination. All lesion locations were determined and transposed onto templates according to the procedure described by Levine and Grek (1984). Subjects with specific lesion locations were defined as patients whose CT scans showed lesions in those locations regardless of whether lesions were also seen in other locations. The size of the lesion (expressed as percentage of total brain volume) was calculated from ratio of the largest cross-sectional area of the lesion on any CT scan slice, divided by the largest cross-sectional area of the whole brain on a CT slice passing through the body of the lateral ventricles. Lesions and whole brain areas were determined by a computerized area calculation program. Reliability of this procedure and correlation with other methods of determining lesion volume have been previously published (Robinson et al., 1985; Starkstein et al., 1987).

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Statistical Analysis

Statistical analysis included both one- and two- way analysis of variance models for ratings on continuously measured scales. For discrete variables, we used chi-square tests. When sample sizes were prohibitively small, we used Fisher's exact test. Survival analysis was utilized to examine the duration of irritability and the depressions. Kaplan-Meier (product-limit) estimators were used to estimate the time to recovery from irritability and depression, with nonparametric log-rank chi-squares being utilized to test for group differences in survival times. These methods account for right-censored data for those not recovering before the end of the study (Kalbfleisch and Prentice, 1980). Logistic regression was used to test for an association between the presence of irritability and lesion location. Again, an overall test was used to control for alpha inflation and interrelations among the lesion locations. Following the significant likelihood ratio test for the full model, backward selection was used to reduce the number of lesion locations considered.

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Results

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Sample Characteristics

Based on the PSE, irritability was present when the patient acknowledged feelings of irritability and the examiner observed the patient showing irritability during the interview and/or if the patient's relatives reported that they had seen repeated episodes of irritable behavior characterized by uncooperative, angry, hostile, shouting, or antagonistic language or behavior. Of the original 66 TBI patients, 12 (18.2%) met the irritability criteria at the time of initial evaluation and constituted the acute onset irritability group, whereas 10 (15.1%) patients met the criteria at either the 3, 6, 9, or 12-month follow-up evaluation and constituted the delayed onset irritability group. All of the acute onset and all of the delayed onset cases were based on information from their families of irritable behavior, and 3 patients were also observed by the examiner to be irritable. There were 44 patients (66.7%) who did not show irritability and were not reported by their families to be irritable either at the initial evaluation or during the 1-year follow-up period and they constituted the nonirritability group.

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Background Characteristics

Demographic characteristics of the 66 patients, grouped by presence and time of onset of irritability, are shown in Table 1. Patients were predominately in their 20s and 30s, of lower socioeconomic class, right-handed, white, and male. There were no significant intergroup differences in these characteristics or in their educational level, Glasgow Coma Scale score, family history of psychiatric disorder, personal psychiatric disorder, or personal alcohol and/or other substance abuse history. There were also no significant differences in the use of analgesic or sedative medications (Table 1). The mean duration of irritability was 2.2 months in the acute onset group and 1.5 months in the delayed onset group. There were no intergroup differences in the estimated mean duration of irritability. Patients without irritability had a significantly higher frequency of severe head injury ($[chi]^2$ = 7.8, *df* = 1, *p* = 0.019), as compared with patients with acute or delayed onset irritability. There were no intergroup differences in the frequency of moderate or mild head injury.


Table 1

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Psychiatric Findings

We compared the HRSD, MMSE, JHFI, STC, SFE, and PSE scores of three groups at the initial exam and at 3, 6, 9, and 12-month follow-ups (Table 2). The acute onset irritability group showed no significant differences from the delayed onset or nonirritability groups for any of the impairment scales except that their PSE scores

were significantly higher at the initial evaluation (F = 6.1, df = 1; 54, p = 0.016). On the other hand, the delayed onset irritability group had poorer social functioning as measured by the SFE at both 3 months (F = 5.9, df = 1; 43, p = 0.018) and 9 months (F = 5.5, df = 1; 34, p = 0.023). The delayed onset group also had greater impairment in activities of daily living as measured by JHFI at 6-month (F = 8.9, df = 1; 35, p = 0.005) and 9-month (F = 7.1, df = 1; 34, p = 0.011) follow-ups. Finally, there were significantly higher PSE scores in the delayed onset group at 3- (F = 8.4, df = 1; 43, p = 0.005), 6- (F = 6.4, df = 1; 35, p = 0.015), 9- (F = 10.7, df = 1; 34, p = 0.002), and 12-month (F = 5.5, df = 1; 34, p = 0.023) follow-ups. There were no significant intergroup differences in depressive symptoms as measured by the HDRS, intellectual impairment as measured by the MMSE, or impairment in social connectedness as measured by the STC.

Table 2

To check which aspects of social impairment were strongly correlated with irritability, individual items of the SFE were analyzed. The patient's perception that family members were inadequate in coping with illness (F = 3.2, df = 2; 44, p = 0.046) was significant at 3 months, whereas fears of loss of job (F = 7.9, df = 2; 30, f = 0.001) and inadequacy of spiritual beliefs (F = 4.2, df = 2; 39, p = 0.020) were significantly associated with the presence of delayed onset irritability at 9 months. We next examined the potential interaction of irritability with depression. Of the 66 patients who were examined at the initial evaluation, 17 (25.7%) had DSM-IV-defined major depression and 2 (3.0%) had DSM-IV-defined minor depression. Among the patients who had not developed depressive disorders in the initial evaluation, the incidence of delayed onset (*i.e.*, any follow-up evaluation) major and minor depression was 14.8% (7/47) for major and 6.4% (3/47) for minor depression. A total of 29 patients with depressive disorders at some time during the study included 8 (66.7%) of 12 acute onset irritability, 5 (50.0%) of 10 delayed onset irritability, and 16 (36.3%) of 44 nonirritability patients. There were no statistically significant intergroup differences in the frequency of depressive disorders. However, the estimated mean duration of depression was 4.6 months in acute onset irritability group, 6.0 months in delayed onset group, and 2.8 months in nonirritable group (Table 1). Thus, the delayed onset irritability group had a significantly longer mean duration of depression ($[chi]^2$ = 4.4, df = 1, p = 0.035) compared to nonirritability group.

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Lesion Characteristics

CT scans demonstrating focal brain lesions were available for 49 patients (74.2%). There were 10 scans (83.3%) from the 12 acute onset patients, 8 (80.0%) from the 10 delayed onset patients, and 31 (70.5%) from the 44 nonirritability patients. Of the 49 patients, there were 37 (75.5%) who presented with mass lesions on their CT scans. There were no significant between-group differences in the nature of their lesions such as frequency of diffuse or focal brain injury or frequency of hemorrhages, contusions, hydrocephalus, or brain atrophy, and there were also no significant intergroup differences in the size of lesions. There were 21 patients (56.8%) with left hemisphere lesions and 12 patients (32.4%) with right hemisphere lesions. There were 28 patients (75.5%) with cortical lesions and 9 patients (24.3%) with subcortical lesions (Figs. 1 and 2). A logistic regression model included the following variables: presence or absence of frontal (right, left, or bilateral), temporal (right, left, or bilateral), left anterior (lesions involving the left dorsolateral frontal cortex and/or left basal ganglia lesion), left and right cortical (lesions involving cerebral cortex plus adjacent subcortical white matter), left and right subcortical (lesions involving deep white matter, basal ganglia, brain stem, and cerebellum), or other brain injury. There were no significant intergroup differences in lesion location associated with the development of irritability in overall test of significance. Left cortical lesions, however, approached significance (Wald $[chi]^2$ = 5.8, df = 2, p = 0.054), perhaps indicating that cortical lesions were more frequent in the irritability patients compared with the no irritability patients. A backward selection procedure was then performed to remove the nonsignificant variables. The presence of left cortical lesions (Wald $[chi]^2$ = 4.6, df = 1, p = 0.032) was associated with an increased probability of developing acute onset irritability. There were 13 (72.2%) of the 18 acute or delayed onset irritability patients with cortical lesions and only 1 (5.6%) with a subcortical lesion. On the other hand, 13 (41.9%) of the 31 nonirritable patients had cortical lesions and 7 (22.6%) had subcortical lesions (Figs. 1 and 2). The frequency of cortical lesions was significantly higher in the irritable compared with nonirritable patients (Wald $[chi]^2$ = 4.6, df = 1, p = 0.031).


Fig. 1


Fig. 2

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Discussion

This study found that 33.3% of the patients with TBI demonstrated irritability at some time throughout the 1-year follow-up period. This frequency is within the range reported by other investigators (Chambers et al., 1996; Long and Webb, 1995; Taylor and Price, 1994; van Zomeren and van den Burg, 1985). Acute onset irritability patients had a significantly higher frequency of left cortical lesions, and both acute and delayed onset irritability patients had a higher frequency of cortical lesions than nonirritable patients. Furthermore, we found that delayed onset irritability patients had significantly poorer social functioning and impairment in

activities of daily living and had a longer mean duration of depression than patients without irritability. Finally, patients with irritability had less severe head injury than nonirritable patients.

Before discussing the possible explanations or implication of this study, several methodological limitations should be acknowledged. The major methodological limitation of this study is that our definition of irritability was based on the result of family report and observation of a psychiatrist and not on behavioral measures. There are many self-report questionnaires that seem to emphasize behavior rather than mood and all conceptualize "irritability," "aggression." and "anger" in different ways, some emphasizing contextual cues (Novaco, 1975, 1985), some separating scores for aggression directed at others or self (Caine et al., 1967), and others focusing on arbitrary types of hostility (Buss and Durkee, 1957). In addition, The Schedule for Affective Disorders and Schizophrenia (Spitzer and Endicott, 1979) uses the word "irritability" for overt behavior, which can also be associated with other dysphoric moods such as depression. But the PSE (Wing et al., 1974) rates irritability as present only if the patient subjectively experiences irritability independent of behavior. As a consequence, we required both statements of irritability by patients and confirmatory observations of their families. It seems unlikely, however, that patients and/or their families would falsely admit to irritability. Another limitation in this study is that the population of TBI patients was primarily lower socioeconomic class, white, and young male with a history of alcohol and/or drug abuse. It should also be recalled that patients with severe comprehension deficits or altered consciousness were excluded. Although this demographic profile is typical of the population reported in studies of TBI, our findings may not be applicable to all patient populations with TBI. In addition, we examined only 49 CT scans demonstrating brain lesions; furthermore, 12 of the patients did not have a lesion on their scan. Magnetic resonance images would likely have identified more lesions. Finally, although we examined 87.9% of the original population at some time during follow-up, the percentage of patients lost at one or more of the 6, 9, and 12-month follow-up interviews was 34.8%. This may have decreased the number of delayed-onset irritability patients and skewed the associations of impairment in social functioning and activities of daily living (ADL). However, there were no significant differences in background characteristics, initial psychiatric variables scores, or type and location of brain lesions between patients who were followed (N = 58) and dropped out (N = 8) and the significant associations of delayed onset irritability with social and ADL impairment were found on more than one follow-up evaluation which included slightly different groups.

Given these limitations, how might these findings be construed? One of the unexpected, but perhaps the most important finding that needs to be explained is the fact that left cortical brain injury was significantly associated with irritability during the acute posttrauma period.

Few other investigators have looked for an association between lateralization of lesion and occurrence of irritability. Gualtieri (1991) reported severe damage to orbital frontal lobes or anterior temporal lobes (amygdala) resulted in a syndrome including irritability, disinhibition with socially inappropriate sexual or impulsive behaviors, and restlessness. In addition, they found that minor damage to the basal frontal lobes as a result of TBI could produce irritability that was part of a postconcussion syndrome, whereas Long and Webb (1995) postulated that impaired cortical functions leading to minor difficulties in concentration and performance may cause irritability. They suggested that investigation of subtle cortical dysfunction may provide an understanding of the behavioral and emotional sequelae following head trauma. Galbraith (1985) wisely points out that the frustrating experience of mental inefficiency may well contribute to irritability following head injury. He also notes that it could result from direct damage to the limbic system although no site has been identified. In a study of stroke patients with anger or violent behavior, Paradiso et al. (1996) reported that left cortical lesions were more frequent in violent patients compared with the general stroke population.

Although the nature of this association between acute onset irritability and left cortical lesion location is not known, this finding suggests that left cortical function may be critical in preventing irritability. We have previously reported that lesions involving the left cerebral hemisphere, particularly the dorsolateral frontal cortex or left basal ganglia, were associated with an increased frequency and severity of depression (Fedoroff et al., 1992; Jorge et al., 1993a, 1993b; Morris et al., 1996; Paradiso et al., 1996). This finding raises the question of whether irritability could be a manifestation of depression. Although depression may have contributed to irritability in some patients, depression was not significantly associated with irritability.

The fact that impaired social functioning and activities of daily living had the most consistent relationship with delayed onset irritability is in agreement with our previous findings of different clinical correlates for acute onset and delayed onset depression (Fedoroff et al, 1992; Jorge et al., 1993a, 1993b). The finding that irritability was associated with patients' perceptions that their families could not cope with chronic illness, fear of job loss, and inadequate spiritual life suggests that these factors may produce the greatest fears and frustrations following head

injury and therefore ultimately lead to irritability.

The other interesting finding from this study was that patients without irritability had greater severity of head injury than irritability patients. This finding is consistent with a previous report that neurobehavioral consequences after TBI were inversely correlated with severity of injury (Lidvall et al., 1974). Although many explanations might be posed for this finding, it might be speculated that irritability requires more intact motor and cognitive processes and that the mildly injured patients experience the most frustration, which they act out as irritability.

In summary, this study has found that, although acute onset and delayed onset irritability share some factors such as cortical lesion location, there are important differences in these disorders. Some acute onset irritabilities are related to lesion characteristics and may have their etiology in biological responses of the injured brain, whereas delayed onset irritabilities may be mediated by psychosocial factors, suggesting psychological reaction to physical and social impairment. Finally, these findings suggest that the response of these disorders to treatment may be different. Early after injury, irritability might respond better to pharmacological treatment while delayed onset irritability may respond better to social and rehabilitation interventions.

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Conclusions

This study found that irritability is frequent in patients with TBI occurring in one third of patients sometime during the first year after injury. Irritability during the acute postinjury period was associated with left cortical injury, whereas irritability at 3 or more months after injury was associated with impairment in social functioning and activities of daily living. These findings suggest that the cause of irritability in patients with TBI may change over time and therefore the most appropriate interventions may also change over time.

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