A Forensic Neuropsychiatric Approach to Traumatic Brain Injury, Aggression, and Suicide

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Aggression is a common neuropsychiatric sequela of traumatic brain injury (TBI), one which interferes with rehabilitation efforts, disrupts social support networks, and compromises optimal recovery. Aggressive behavior raises critical safety concerns, potentially placing patients and care providers in harm's way. Such aggression may be directed outwardly, manifesting as assaultive behavior, or directed inwardly, resulting in suicidal behavior. Given the frequency of TBI and posttraumatic aggression and the potential medicolegal questions surrounding the purported causal relationships between the two, forensic psychiatrists need to understand and recognize posttraumatic aggression. They also must be able to offer cogent formulations about the relative contributions of neurotrauma versus other relevant neuropsychiatric factors versus combinations of both to any specific act of violence. This article reviews the relationships between TBI and aggression and discusses neurobiological and cognitive factors that influence the occurrence and presentation of posttraumatic aggression. Thereafter, a heuristic is offered that may assist forensic psychiatrists attempting to characterize the relationships between TBI and externally or internally directed violent acts.

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Traumatic brain injury (TBI) is a common problem in the United States. TBIs are sustained by approximately 1.5 million civilians each year,[1,2] 124,000 of whom are expected to experience long-term disability.[3] Current estimates suggest that 1.1 percent of the U.S. civilian population is living with long-term disability from TBI.[4] TBI is also common in soldiers returning from the wars in Iraq and Afghanistan, with an estimated 15 to 20 percent of nearly 2 million deployed U.S. troops having experienced a possible mild TBI.[5,6] The types and severities of symptoms that follow TBI vary widely between and within individuals as they recover. Such variability reflects the broad range of types and severities within this category of clinical diagnosis, as well as the influence of and interaction between preinjury factors and neurotrauma. Variability in symptom development and persistence after TBI also is influenced by postinjury psychosocial factors, including treatments and social supports (or the lack thereof) as well as litigation and other legal entanglements. Posttraumatic cognitive, emotional, behavioral, and physical impairments, irrespective of their causes, are frequent and substantial sources of disability and suffering for both patients and families.[7–11]

Aggression is a common and particularly challenging neuropsychiatric sequela of TBI.[12] It includes externally directed acts (e.g., verbal outbursts, physical violence toward objects, and physical violence toward persons) as well as self-directed violence (e.g., nonsuicidal self-directed violence, suicide attempts, and suicide). Aggression interferes with rehabilitation efforts, disrupts social support networks, and compromises optimal recovery. Caregivers and families have described posttraumatic behavior, including aggres-

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sion, as the most difficult aspect of adjustment in caring for patients recovering from TBI.[13] In addition, aggression raises critical safety concerns, potentially placing patients and care providers in harm's way.

Violence can engender negative social and legal consequences and may further complicate recovery and limit access to care. Participants in legal matters may seek to establish causal relationships between TBI and specific violent behavior, as a defense against criminal culpability, or to establish civil liability. In addition, recent evidence suggests a relationship between TBI and self-directed violence, including suicide.[14–17] Highly publicized litigation against professional sports associations is, in part, predicated on possible associations of multiple mild TBIs and the neuropathological condition termed chronic traumatic encephalopathy with suicide.[18,19]

In light of these findings, it is likely that both criminal and civil litigation surrounding acts of violence performed by individuals with histories of TBI will require analysis and commentary by forensic psychiatrists. Meaningful medicolegal consultation in such matters necessitates avoiding overly reductionistic causal relationships or succumbing to logical fallacies of the *post hoc*, *ergo propter hoc* (after TBI, therefore because of TBI) or *cum hoc*, *ergo propter hoc* (with TBI, therefore because of TBI) varieties. Whether supporting or refuting a causal relationship, the forensic psychiatrist is obliged to base such opinions on a rigorous understanding of the pathophysiology of TBI and on an up-to-date review of the medical literature describing posttraumatic aggression.

With this obligation in mind, this article reviews the relationships between TBI and aggression and discusses neuropsychiatric factors that influence the occurrence and presentation of posttraumatic aggression. Thereafter, a heuristic is offered that may assist forensic psychiatrists attempting to characterize the relationships between TBI and specific acts of externally or internally directed violence.

Defining TBI

TBI is defined as a significant disruption of brain function, structure, or both, resulting from the application of an external physical force (including acceleration/deceleration and blast-related forces) that causes immediate disturbances of cognitive or elementary neurologic function.[20–24] Although trauma to the face and head ought to prompt clinical consideration of TBI, such injury alone is insufficient to establish the diagnosis. Skull fracture, while associated with an increased likelihood of TBI,[25–28] also is insufficiently reliable as a predictor of TBI to enable diagnosis based on this criterion alone.[29] For these reasons, the term head injury as a synonym for TBI is problematic and discouraged.[25]

TBI severity is typically divided into three categories: mild, moderate, and severe. The Glasgow Coma Scale (GCS),[30] although originally developed as a measure with which to assess impaired consciousness after any type of brain injury, is used commonly to estimate initial TBI severity[31] and is a useful tool for this purpose when administered and interpreted appropriately. When GCS assessments are unavailable, the American Congress of Rehabilitation Medicine (ACRM) criteria for mild TBI[23] may help differentiate mild from moderate-to-severe injuries by clinical history. According to these criteria, mild TBI is defined as a mechanically induced physiologic disruption of brain function manifested by any one of the following: a loss of consciousness (LOC); a loss of memory for events immediately preceding or following the injury; an alteration in mental state (feeling dazed, confused, or disoriented) at the time of injury or by focal neurological signs that may or may not be transient. To remain within the category of mild TBI, any associated LOC must be less than 30 minutes in duration, posttraumatic amnesia (PTA) must not exceed 24 hours, and the GCS score must be 13 or higher by 30 minutes after injury. TBI resulting in longer durations of LOC or PTA, or lower GCS scores at 30 minutes after injury, are classified as moderate to severe.

A useful addition to the ACRM criteria is the concept of complicated mild TBI.[32] Most mild TBIs will yield no objective evidence of injury on structural brain imaging (computed tomography (CT) or magnetic resonance imaging (MRI)).[33–35] An injury that otherwise meets ACRM criteria for mild TBI but is associated with intracranial abnormalities on conventional structural neuroimaging consistent with the effects of neurotrauma is referred to as a complicated, mild TBI.[32] This type of mild TBI carries a prognosis more similar to ACRM-defined moderate-to-severe TBI than to uncomplicated, mild TBI.[32,36–38]

TBI and Aggression

Typology of Posttraumatic Aggression

Any effort to elaborate the relationship between TBI and aggression warrants at the outset a few

words of caution. In reviewing the neuropsychiatric complications of TBI, the American Neuropsychiatric Association's Committee on Research[9] noted that interpretation of the literature is challenging as a result of variability in TBI definitions, severity criteria, nosological questions, preinjury psychiatric conditions, the extent to which postinjury psychiatric assessments are considered *de novo*, and the length of follow-up after injury. They argued that the nosology of aggression is particularly problematic, as there is no standardized or universally accepted definition of posttraumatic aggression. It is difficult to determine the extent to which the many terms used to describe behavioral dyscontrol in the literature (i.e., agitation, restlessness, impulsivity, disinhibition, irritability, lability, or explosive behavior) denote aggression, some combination of aggression and other posttraumatic neuropsychiatric problems, or other nonaggressive behavioral disturbances alone. Similarly, posttraumatic aggression is sometimes described by using the DSM-IV-TR[39] diagnosis personality disorder due to general medical condition, aggressive type. As a result of nosological and other methodological problems, accurate estimates of the incidence and prevalence of posttraumatic aggression are lacking.[9]

A related problem is the variability of the clinical phenomena captured by the term posttraumatic aggression. One form of this problem, observed not only in persons with TBI but also other severe neurological disorders,[40] is the organic aggressive syndrome (OAS).[41] This syndrome is characterized by aggression that is reactive (provoked by seemingly trivial stimuli), nonreflective (unplanned), noninstrumental (serves no clear aim or objective), explosive (occurs suddenly and without any apparent build-up), periodic (prolonged periods of relative calm punctuated by aggressive outbursts), and egodystonic (the individual feels bad about the behavior).[40] This type of posttraumatic aggression is uncommon and is generally observed in persons with relatively severe TBI. Common clinical experience suggests that persons with posttraumatic aggression do not typically present with OAS. As aggressive behavior becomes more discordant with the organic aggressive syndrome profile, clinical judgments regarding direct associations and causal relationships between injuries and actions may be more difficult to establish confidently.

Analogous to the typologies of violence described by Reid and Thorne,[42] posttraumatic aggression may take several forms. Violent acts that are purposeful (i.e., showing intent, premeditation, determination, and resolve) and instrumental (i.e., serving as a means to a specific end) fall at the end of the spectrum of posttraumatic aggression opposite that of OAS. Prototypical examples of purposeful, instrumental aggression are violence for revenge, violence for hire, or violence in the defense of self or others.[42] Somewhere on the middle of this proposed spectrum of aggressive behavior is nonpurposeful (i.e., impulsive) but instrumental violence, wherein unplanned aggressive behavior is directed at a specific person in response to a perceived threat[42] or toward a specific end (e.g., object, person, or experience). Common clinical experience suggests that aggression of this type is more common than either OAS or purposeful instrumental violence in neurobehaviorally impaired TBI survivors and particularly in those with generalized impairments of impulse control (i.e., disinhibited behavior), as well as those with comorbid severe cognitive impairments, depression, mania, anxiety, or psychosis.

It also is important to recognize that aggression, in any form, may arise in persons with TBI in the absence of other neuropsychiatric comorbidities. Such behavior in persons with recent or remote TBI may bear no direct relation to that condition at all. In the latter circumstance, aggression may arise as a function of intoxication, concurrent medical conditions (e.g., delirium due to non-TBI causes), premorbid personality traits and disorders (e.g., antisocial, borderline, or narcissistic), or a context-dependent isolated act of purposeful, instrumental violence in a healthy individual.

Reports of Aggression in Persons With TBI

Offenders

TBI is associated with intimate partner violence (IPV)[43–46]; in a recent meta-analyses TBI was identified in 53 percent of IPV perpetrators.[47] Associations between TBI and aggression also are observed in forensic populations.[48–54] For example, Schofield *et al.*[51,52] identified self-reported histories of at least one prior TBI in 82 percent of men entering the Australian prison system; 43 percent endorsed a history of four or more prior TBIs. These reports establish an association, but not a causal relationship, between TBI and aggression.

Children, Adolescents, and Young Adults

Links between TBI and aggression also are described in children and adolescents.[55–61] Cole *et al.*[62] followed 97 children (age 4–19 years) for one year after severe TBI. Verbal aggression, physical aggression toward others, and physical aggression toward objects were reported to occur more often in the postinjury period than the preinjury period. Preinjury aggression, anxiety, attention problems, and postinjury disability were risk factors for postinjury aggression.

In a prospective study of children with oppositional defiant disorder (ODD),[55] injury severity predicted change in ODD symptoms at two years after injury. Patients with more severe injury, lower socioeconomic status, preinjury ODD symptoms, and preinjury family dysfunction developed more severe aggressive symptoms. Max *et al.*[58] identified personality change after childhood TBI in 59 percent of children with severe TBI and in 5 percent of those with mild-to-moderate TBI. Labile, aggressive, and disinhibited subtypes were most common. These observations reveal a relationship between TBI and aggression in children, but they also highlight the role of pre- and postinjury factors in the development of such behavior.

In young adults with mild TBI, higher levels of emotional distress, including higher hostility and interpersonal sensitivity, which are potential proxies for posttraumatic aggression, are observed than in healthy young adults and (as a control group) in those with a history of general anesthesia exposure.[63] Despite their differences in affectivity, these groups did not differ on tests of cognition. This observation suggests the potential for dissociation between posttraumatic cognitive function and affectivity/behavioral control.

Veterans with Penetrating TBI

Grafman *et al.*[64] studied 279 Vietnam veterans with penetrating TBI and 57 uninjured veteran controls. They observed elevated aggression in persons with TBI and ventromedial frontal lesions when compared with those with TBI and lesions elsewhere and with the control subjects. In the ventromedial frontal lesion group, aggression was not explained by concurrent impairments of general intelligence. These findings further suggest either the potential dissociation of posttraumatic aggression and cognitive function or the relative insensitivity of current assessments to the types of cognitive impairments that are associated with posttraumatic aggression.[9,65]

Adults With Nonpenetrating TBI

Tateno *et al.*[66] compared aggressive behavior between adults with nonpenetrating TBI and those in an injured (without nervous system involvement) control group and observed higher levels of aggression, (on the overt aggression scale) in the TBI group, despite a lack of between-group differences in functional status.[67] Posttraumatic aggression was observed in 33.7 percent of the TBI participants during the first 6 months after injury. Those with aggressive behavior were more likely to have histories of mood disorders, alcohol and substance abuse, and legal intervention for prior aggressive behavior. Major depression was more frequent in aggressive subjects, who, as a group, had poorer social functioning. Analogous to the observations by Grafman *et al.*[64] of veterans with penetrating TBIs, subjects with frontal lesions had higher levels of aggression than those with lesions elsewhere.

Baguley *et al.*[12] observed aggressive behavior in 25 percent of individuals discharged from a brain injury rehabilitation service at 6, 24, and 60 months after injury. In this group of individuals with moderate-to-severe TBI, depression and age at time of injury were the most significant predictors of violence at all study time periods. There were no observed associations among aggression and injury pattern, history of substance use and psychiatric conditions, and cognitive function.

Rao *et al.*,[68] using a prospective observational study to determine the prevalence of aggression in the 3 months after TBI followed by a nested case-control analysis, observed posttraumatic aggression in 28.4 percent of 107 individuals with first-time TBI. Verbal aggression predominated in this group; only one subject displayed aggression against objects. Posttraumatic aggression was associated with new-onset depression, postinjury social impairment, and dependence on others for activities of daily living (ADLs).

Despite variation in the study populations and injury types among these studies, the results collectively support the thesis that TBI is a risk factor for aggression, including violence toward others. However, these studies also highlight the importance of preinjury and postinjury factors as modifiers of that risk.

Neuroanatomy of Aggression

The literature suggests that aggressive behavior is a common problem after TBI, especially during the first year after injury. Posttraumatic aggression also appears to be associated most closely with frontal lobe lesions and may occur despite normal cognitive function.[69–80] This relationship usefully contributes to our understanding of posttraumatic aggression regardless of whether such behavior is directed externally (toward others) or internally (toward oneself, as in suicide).[69–80]

The frontal and temporal lobes are particularly susceptible to the injurious effects of contact and inertial forces to which the brain is subjected during biomechanical trauma.[81–84] This regional susceptibility has been demonstrated in autopsy-based research[82,83] and with modern-day neuroimaging techniques.[85,86] Shearing and straining forces also injure white matter,[10,86–91] which is most affected in the brainstem, cerebral parasagittal white matter, corpus callosum, and gray-white junctions of the cerebral cortex.[81,84,92,93] TBI also at least transiently disrupts the structure and function of major modulatory neurotransmitter systems, including cholinergic, dopaminergic, noradrenergic, and serotonergic projections (see Arciniegas and Silver[94,95] and Bales *et al.*[96]). These combinations of structural and neurochemical changes increase the likelihood of clinically significant early and late posttraumatic disturbances in frontally mediated cognition, emotion, and behavior.

The frontal lobes are the most evolutionarily recent and distinctively human portion of the brain and play a crucial role in higher cognitive processes and the regulation of emotion and behavior. The anterior frontal, or prefrontal, cortical areas subserving these functions are organized into five discrete but parallel and reciprocally interactive frontal-subcortical circuits: the premotor subcortical circuit, involved in the organization of voluntary motor function; the frontal eye field subcortical circuit, which facilitates voluntary eye movements; the anterior cingulate subcortical circuit (ACC), which subserves motivation and aspects of attention; the dorsolateral prefrontal subcortical circuit (DLPFC), which subserves executive function; and the lateral orbitofrontal subcortical circuit (LOFC), which subserves comportment and social intelligence.[97–99]

The DLPFC subserves executive function, a term that denotes cognitive processes such as retrieving, categorizing, organizing, and sequencing information, problem solving, abstraction, judgment, and insight, all of which facilitate autonomous (i.e., self-directed) behavior and decrease behavioral dependency on environmental contingencies.[98–100] This circuit facilitates flexible and adaptive responses to the challenges of everyday life. The LOFC appears to play the key role in suppressing aggression by supporting socially appropriate behavior, imbuing limbically driven appetites and emotions with social insight and judgment, and putting the brakes on contextually inappropriate, limbically driven behavioral responses.

Neurobehavioral manifestations of LOFC injury include irritability, impulsiveness, lability, tactlessness, environmental dependency, and aggression.[98–100] As noted by Grafman *et al.*,[64] traumatically acquired ventromedial lesions of the mediofrontal and orbitofrontal cortices are associated with posttraumatic aggression. A more recent follow-up investigation[101] suggests that lesion location and genetics (MAO-A genotype) interact in mediating posttraumatic aggression, suggesting that integrity of the prefrontal cortex is necessary for modulating genetic susceptibility to aggressive behavior.

Yurgelun-Todd *et al.*,[102] in a sample of 15 veterans with TBI and 17 healthy controls, demonstrated a relationship between cingulum white matter integrity and measures of impulsivity and current suicidal ideation. The cingulum comprises the dorsal limbic pathway, which connects mid-dorsolateral prefrontal areas with the orbitofrontal cortex and medial surface of the frontal lobe.[103] Hence, it serves as a neurobehaviorally critical pathway through which LOFC and DLPFC functions are integrated. When its structure is compromised, the risk for both externally directed and internally directed impulsive aggressive behavior may be increased.

Injury to the DLPFC impairs problem solving and increases the tendency toward environmentally bound behavior. Mega and Cummings[99] postulate that impairment of this circuit compromises the ability to maintain an adequate repertoire of adaptive responses to challenging or stressful situations and therefore increases the likelihood of maladaptive responses, including aggression. In our clinical experience, and as suggested by the observations in the

study by Grafman *et al.*,[64] posttraumatic aggression is uncommonly associated with isolated DLPFC injury. This association, at least in part, reflects the relatively uncommon occurrence of isolated DLPFC injury in persons with TBI.[81–84] It also accurately reflects the more common connection of posttraumatic aggression with injury to the LOFC, with or without concomitant DLPFC injury or dysfunction.

At the same time, it is equally important to recognize that most individuals who sustain a TBI and injury to these areas do not become violent. As noted in the Aspen Neurobehavioral Conference Consensus Statement on violence,[104] all human behavior is variably governed by the interaction of numerous factors, including genes, early life experience, acquired brain damage, learned behavior patterns, and situational contingencies. Violence derives from either normal or abnormal operation of the brain. Although TBI is associated with increased risk of aggression and violence, and TBI with frontal dysfunction appears to threaten the capacity to inhibit violent behavior, it is crucial to appreciate that illness is not destiny. A host of preinjury and postinjury factors, as well as violent act-specific factors (i.e., context, precipitants, purposefulness, and instrumentality) are necessary considerations in any given individual performing a specific violent act toward self or others.[104]

TBI and Suicide

Several studies have shown that the risk of self-directed violence, specifically suicide, is often, but not invariably, increased in persons with TBI.[14,15,105–108] Although the relationship between TBI and suicide is necessarily a complex one, it may be similar in its psychological and neurobiological bases to externally directed violence.

Kerr *et al.* [109] suggest that aggressive impulses may be directed toward the self, in which case they manifest as suicidal ideation, self-injurious behavior, suicide attempts, and completed suicide. Aggression may fuel depression, which may lead to suicidal ideation and attempts. Aggression may lead to behavior that has negative consequences, thereby worsening depression and suicidal ideation. Aggression also may directly lead to suicidal ideation and attempts. This model provides a useful conceptual framework for considering the relationship between TBI and suicide, although neuropsychiatric comorbidities relevant to the link between TBI and suicide are likely to extend beyond depression, and include comorbid substance use,[15,105] among others.

Since traumatically induced ventral frontal injury is associated with aggressive behavior, and since aggression may be either externally or internally directed, it is plausible that TBI survivors with injuries involving LOFC and its connections to other behaviorally salient neural networks may be at increased risk for suicidal behavior. The observations of Yurgelun-Todd *et al.*,[102] described earlier in this article, are consistent with this suggestion. In addition, findings from a recent familial study on suicide identified impulsive aggression as an endophenotype for suicidal behavior and a potential role for impulsive aggression in familial transmission of suicide risk. Oquendo *et al.*[110] studied the relationship between TBI, suicidal behavior, and other risk factors in depressed patients and offered a hypothesis framed in a stress-diathesis model for suicide. In this model, TBI potentially plays a dual role: it may serve as a stressor, causing psychiatric illness and suicidality, or it may serve as diathesis via frontal lobe injury, precipitating disinhibition, impulsivity, and aggression. In this formulation, TBI and suicide share many antecedents, including aggression, but such antecedents alone do not explain the relationship between TBI and suicide. Compared with persons without TBI, those with TBI had larger increments in adult aggression scores, leading to the supposition that the occurrence of TBI may have furthered aggression, thereby increasing the risk for suicide.[110]

Reports of Suicide in Persons with TBI

Silver *et al.*,[105] using the New Haven portion of the National Institute of Mental Health (NIMH) Epidemiologic Catchment Area program, identified a higher frequency of suicide attempts in individuals with a history of TBI when compared with those without TBI (8.1% versus 1.9%). The combination of TBI and alcohol abuse increased the odds ratio (OR) for suicide attempts by 5.7 times, relative to that of the general population; the OR for suicide attempts remained elevated (4.5 times) even after adjustment for alcohol abuse. Teasdale and Engberg[111] conducted a large population-based study in Denmark in which the reference for standardized mortality ratios (SMRs) was suicides in the general population (i.e., SMR = 1). Concussion was associated with an SMR of 3.02, and injury involving cerebral contusion or traumatic intracranial hemor-

rhage (i.e., complicated mild or greater severity) was associated with an SMR of 4.05. Substance abuse dramatically increased the risk of suicide across all levels of TBI severity.

Simpson and Tate,[15] based on 48 articles evaluated in a systematic review-like manner, conclude that the risk for suicide, suicide attempts, and suicidal ideation is increased in TBI survivors when compared with the general population, even after adjustment for psychiatric comorbidities. Suicidal ideation occurred in 21 to 22 percent of persons with TBI across all severities, and the suicide attempt rate was approximately 18 percent in persons with severe TBI. The risk of completed suicide in men and woman was elevated in comparison to that of the general population, with SMRs of 3.9 and 4.7, respectively. Similar to the findings of Teasdale and Engberg,[111] suicide risk increased with injury severity: the SMR for concussion was 3 whereas the SMR for severe (lesional) TBI was 4.1. Consistent with the observations of Silver *et al.*[105] and Teasdale and Engberg,[111] substance abuse increased the risk of suicide in persons with TBI, with an SMR for suicide of 7.4 in this subgroup. The influence of substance abuse on post-TBI suicidal ideation is supported by the findings of Tsaousides *et al.*[112] in community-dwelling adults with TBI; they also observed a strong and consistently increased risk of post-TBI suicidal ideation in persons with postinjury emotional distress, psychopathology, and relatively poor psychosocial functioning.

Brenner *et al.*[14] conducted a Cox proportional hazards survival analysis to compare suicide rates in veterans with and without TBI. Adjustment for psychiatric comorbidities and demographics showed that veterans with a history of TBI were 1.55 times more likely to die by suicide than those without a history of TBI. Veterans with a history of TBI involving concussion or fracture were 1.98 times more likely to die by suicide than were veterans without a TBI history, whereas veterans with history of TBI involving contusion or traumatic intracranial hemorrhage were 1.34 times more likely to die by suicide. This study adds to the body of literature associating history of TBI with increased risk for suicide, and extends such findings to our veteran population. Notably, these findings are discordant with the dose effect of injury severity on suicide risk observed in other studies.[15,111] However, and as noted by Brenner *et al.*,[14] unexplored preexisting factors or additional covariates (such as pain) may have contributed to the inverse relationship between injury severity and suicide risk reported.

TBI, Aggression, and Suicide in the Medicolegal Context

The literature reviewed for this article revealed a compelling relationship of TBI and aggression with suicide. However, the literature also revealed complex relationships involving preinjury factors, details of the TBI itself (i.e., severity and injury location), and postinjury psychosocial factors. Clinical formulations recognizing a generalized increased tendency toward aggression subsequent to TBI may be sufficient for treatment purposes. However, medicolegal formulations necessitate attending more precisely to the myriad of factors that may contribute to such behavior. In addition, medicolegal formulations also necessitate commenting on a specific act of violence in an individual with a history of TBI and not merely on generalized behavioral tendencies observed in groups of persons with TBI.

Unequivocal attribution of violent behavior to TBI rather than to purposeful, instrumental violence should be undertaken with caution, and only after careful consideration of the totality of circumstances surrounding such acts. Considerations include, but are not limited to, specific details of the TBI, preinjury and postinjury psychosocial factors, the context in which the particular violent act occurred, and any potential precipitants and possible objectives of the act. Hence, cogent medicolegal formulations typically require close attention to details pertaining to the individual (i.e., the person who sustains a TBI), the TBI sustained by that individual, the pre- and postinjury psychosocial context, and the particular act of violence that has brought the individual to medicolegal attention.

Preinjury Factors

Aggression and self-directed violence may arise in persons with TBI as a consequence of neuropsychiatric comorbidity. At a minimum, preexisting conditions frequently contribute to the mental state and psychosocial circumstances that culminate in any given act of violence. In many instances, acts of aggression that occur subsequent to a TBI are more directly related to preexisting conditions than to neurotrauma. Cogent formulations thus require meticulous attention to the individual's preinjury neuropsy-

chiatric status. Of particular relevance are preexisting conditions involving externally or internally directed aggressive behavior (including prior suicidal ideation and attempts), impulsivity, substance abuse, mood disorders, personality traits and disorders (e.g., antisocial, borderline, or narcissistic), seizures, and cognitive impairment and decline. Such conditions have the potential to contribute significantly to acts of aggression, or the psychosocial situations in which such behavior is apt to arise.

Injury Factors

When considering the relationship between any given act of violence and any given TBI, the severity of the TBI becomes an essential consideration. Posttraumatic aggression is associated with TBIs of relatively greater severity. Injuries exceeding ACRM criteria for uncomplicated, mild TBI are more routinely associated with acts of aggression than those falling within the uncomplicated, mild TBI range. Such findings intuitively make sense, suggesting that there is a dose effect wherein a greater degree of neurotrauma results in increased likelihood of neuropsychiatric symptoms, including aggression.

A related consideration is the temporal relationship between any given TBI and any particular act of violence. The natural history of TBI is generally one in which impairment is most severe in the immediate postinjury period, with subsequent improvement of symptoms, leading to a plateau in symptom evolution or symptom resolution, depending on the severity of the initial injury. Although this point pertaining to temporal relationships does not feature prominently in the posttraumatic aggression literature at present, such themes are well developed within the more general TBI literature, particularly that relating to uncomplicated, mild TBI.

Uncomplicated, mild TBI carries a very favorable prognosis for most individuals who sustain such injuries.[113–117] The 2004 meta-analysis conducted by the WHO Collaborating Center Task Force on Mild TBI[114] demonstrated that, for both children and adults, complete recovery following mild TBI is the norm and that such recovery generally occurs within weeks or months of injury. Accordingly, posttraumatic aggression also is expected to remit spontaneously during that timeframe in most individuals with single, uncomplicated, mild TBI.

When aggressive behavior, with or without other postconcussive symptoms, fails to remit spontaneously in persons with such injuries, alternate neuropsychiatric conditions (i.e., depression, posttraumatic stress disorder, substance abuse, medication effects, litigation effects, and other) must be considered as possible explanations for their development and persistence. Phrased another way, positing causal relationships between neurotrauma and acts of violence becomes more difficult with uncomplicated, mild TBI, especially in the late postinjury period, and the relevance of premorbid neuropsychiatric conditions, other postinjury neuropsychiatric disorders, and psychosocial factors grows commensurately. These potential complications surrounding injury severity and temporal relationships highlight the need for careful case-by-case analysis and the avoidance of overly broad generalizations. For instance, although attribution of an aggressive act to an uncomplicated, mild TBI appears to be a tenuous proposition based on the preceding discussion, aggressive behavior can manifest during an extended period (<24 hours by definition) of PTA, and consideration for a pertinent role played by neurotrauma may be appropriate under such circumstances.

Psychosocial Context

Postinjury psychosocial factors mandate consideration. The provision (or absence) of appropriate treatment, education, and social supports may influence the course of postinjury symptoms more generally, including the occurrence and course of posttraumatic aggression specifically. Environmental precipitants (e.g., job loss, financial stressors, relationship dissolution, and incarceration) are often readily identifiable, and such precipitants necessarily interact with an individual's mental state. The psychosocial import of environmental precipitants relates to some extent to the prominence of pathological neuropsychiatric conditions (TBI or other) in understanding the etiology of any given act of violence. In other words, dire circumstances invoking extreme distress may reasonably be expected to precipitate aggressive behavior in individuals with otherwise modest neuropsychiatric burden, whereas relatively trivial occurrences might lead to aggressive acts in those persons who have substantial neuropsychiatric illness.

For the forensic psychiatrist, consideration of litigation and other legal entanglements in the postinjury psychosocial environment is necessary. The aforementioned WHO Collaborating Center Task

Force[114] identifies litigation and compensation as among the only consistent predictors of long-standing symptoms after mild TBI. Similar findings are reported in a more recent meta-analysis conducted by Belanger *et al.*[113] Collectively, they took care to point out that the relationship between litigation and compensation and persisting mild TBI symptoms remains poorly understood, such that conscious symptom exaggeration (i.e., malingering) should not be assumed in any given case. A host of conscious and unconscious factors are potentially at play, such that complex, multidetermined behavior may warrant a differential diagnosis that is unique to the litigation environment.[118] At the same time, the potential for malingering is very real when litigation is a factor, and various authors have reported significant base rates (up to 40%) of malingering in brain injury litigation.[119,120] The performance of meaningful and objective forensic evaluations necessitates consideration of litigation effects (including malingering) when the reported clinical history comports poorly with the known natural history of TBI.[114,121]

The Violent Act

Violent behavior varies widely in several regards, some of which are particularly relevant to formulations invoking posttraumatic aggression and suicide. Given that compelling neuroanatomic relationships and associated medical literature suggest that TBI threatens the capacity to inhibit aggressive impulses, violent behavior referable to posttraumatic aggression ought to tend toward the impulsive. Hence, in considering a particular act of violence and its relationship to TBI, the behavior at issue should be viewed along a continuum featuring highly impulsive acts at one end and planned or premeditated acts at the other. A similar continuum may be constructed in considering the instrumental nature of any specific act of violence. Again, given the neurobehavioral consequences of TBI, violent acts referable to TBI are expected to tend toward the noninstrumental (i.e., not serving clearly as a means to a specific end) rather than the deliberate and goal directed.

This construct for analyzing a given violent behavior and its relationship to TBI can be visually represented via a three-dimensional graph (Fig. 1). Although not intended to be taken too literally in its practical application and certainly not capturing all the applicable dimensions, this figure offers a heuristic for violent behavior and the possible causal relationship with TBI. As illustrated, the violent act meticulously planned over weeks to months (i.e., high purposefulness) and serving obvious objectives (i.e., high instrumentality) is unlikely to be meaningfully associated with a single, uncomplicated, remote, mild TBI, regardless of whether the violent act is assault, homicide, or suicide (Fig. 1, object A). At the other extreme, violent acts that are highly impulsive and without discernible objectives are more apt to be causally related to TBI, especially when those injuries are severe and involve damage to the ventral frontal networks (Fig. 1, object D). Violent acts involving relatively modest degrees of purposefulness and instrumentality and that are associated with injuries of intermediate severity (Fig. 1, objects B and C) will present greater challenges to the forensic examiner attempting to describe their causal relationships. We suggest that this construct is applicable to the forensic examination of violent acts, regardless of whether those acts are internally or externally directed.

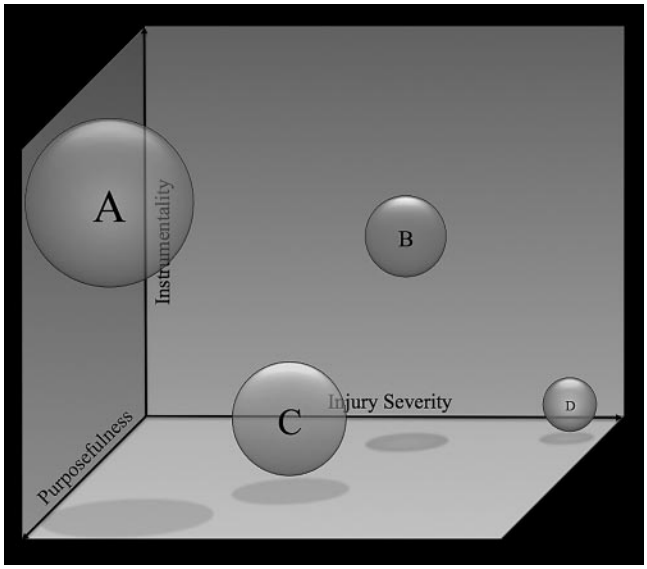

Figure 1. The *x*-axis represents TBI severity, moving from mild to severe. The *y*-axis represents the continuum of instrumentality, and the *z*-axis the continuum of purposefulness.

Conclusions

There remains much to be learned about the complex relationships between TBI, aggression, and suicide. However, those relationships are sufficiently described within the medical literature, such that arguments surrounding causal relationships between violent acts and TBI feature prominently in medico-

legal proceedings at the present time, and these legal matters cannot await definitive answers to lingering questions surrounding these complicated neuropsychiatric phenomena. Experts offering opinions must strive to combine relevant and reliable formulations predicated on an integrated understanding of the neuropsychiatric sequelae of TBI and the neuropsychiatry of violence. Moreover, the expert must assiduously avoid overly reductionistic cause-and-effect representations that fail to pay deference to the multitude of confounding factors that invariably contribute to the occurrence of real-world instances of post-traumatic violence.

It seems likely that, as the literature linking TBI and violence continues to develop, causal relationships between the two will be featured with increasing frequency in criminal and civil litigation involving persons with histories of TBI. The heuristic offered in this article may serve as an initial guide for forensic evaluators offering medicolegal formulations in such matters. Future investigations, critiques, and analyses of this subject are needed to provide concrete direction to forensic experts asked to address the medicolegal complexities of violent acts performed by persons with TBI.

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