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Emotional Traumatic Brain Injury

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Abstract: The definition of traumatic brain injury (TBI) has expanded to include mild TBI and postconcussive syndrome. This evolution has resulted in difficulty disentangling the physical trauma of mild TBI from the emotional trauma of posttraumatic stress disorder (PTSD). Advances in stress neurobiology and knowledge of brain injury at the macroscopic, microscopic, biochemical, and molecular levels call for a redefinition of TBI that encompasses both physical and emotional TBI. Conceptualizing a spectrum of TBI with both physical and emotional causation resolves the irreconcilable tangle between diagnostic categories and acknowledges overlapping forms of brain injury and shared systemic effects due to hormonal and inflammatory mediators. Recognizing emotional TBI shifts the interpretation of emotional trauma from a confound to a comorbid, related cause of brain injury. The mechanism of emotional TBI includes the intricate actions of stress hormones on diverse brain functions due to changes in synaptic plasticity, where chronically elevated hormone levels reduce neurogenesis, resulting in dendritic atrophy and impaired cognition. The overlapping effects of physical and emotional trauma are seen in neuropathology (ie, reduction of hippocampal volume in TBI and PTSD); fMRI (similar regional activations in physical and emotional pain); and systemic sequelae, including changes in proinflammatory cytokine levels and immune cell function. Accumulating evidence favors a change in the definition of TBI to encompass emotional TBI. The definition of TBI will be strengthened by the inclusion of both physical and emotional trauma that result in diverse and overlapping forms of brain injury with sequelae for physical and mental health.

Key Words: brain trauma, plasticity, psychiatric disorders, neuropsychology/behavior, clinical neurology

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mTBI = mild traumatic brain injury. **PCS** = postconcussive syndrome. **PTSD** = posttraumatic stress disorder. **TBI** = traumatic brain injury.

As brain mechanisms underlying emotion and behavior are discovered, the distinction between neurology and psychiatry grows increasingly tenuous. An example is the definition of traumatic brain injury (TBI), where recognition of mild traumatic brain injury (mTBI) and postconcussive syndrome (PCS) results in difficulty disentangling the physical trauma of mTBI from the emotional trauma of posttraumatic stress disorder (PTSD). A parallel is found in the psychiatric literature, where recognition of the neurobiology of stress results in difficulty categorizing stress-related conditions, including PTSD and complicated grief.

Traditionally, investigations have focused on how physical trauma affects the neural circuitry of emotion, cognition, and behavior; the effects of emotional trauma on the same neural pathways have received less attention. Compelling advances in stress neurobiology and knowledge of brain injury at the macroscopic, microscopic, biochemical, and molecular levels call for a redefinition of TBI that encompasses both physical and emotional TBI.

Conceptualizing a spectrum of TBI with both physical and emotional causation resolves the irreconcilable tangle between diagnostic categories and acknowledges their shared systemic effects due to hormonal and inflammatory mediators. Recognizing emotional TBI shifts the interpretation of emotional trauma from a confound to a comorbid, related cause of brain injury.

The loss of my husband to cancer started me down a path to discover how the emotional trauma of loss affects the brain (Shulman, 2018). In the early days, there was no relief from "the invisible blanket between the world and me," described by C.S. Lewis (1968, chap. 1, p. 3) in *A Grief Observed*. With the passage of time, I viewed my own experience less as the sorrow of grief I had anticipated and more as altered mental status, characterized by disorientation, a fog of confusion, and impaired mental flexibility. There were episodes of dissociation, transient disruptions in perception and consciousness that were triggered by strong emotion.

Dissociation and other defense mechanisms are protective because they distance us from painful memories by day; however, they often result in disturbed sleep at night. A vicious cycle emerges where traumatic memories are too disturbing to be consolidated with past memories during sleep, resulting in unsettling dreams and susceptibility to emotional triggers. These experiences recapitulate many of

the cognitive, affective, and sleep sequelae of physical TBI. As a neurologist and scientist, I have been investigating the relationship between brain function and behavior for decades, never anticipating that personal loss would be a catalyst for new insights.

Historically, different types of emotional trauma were categorized separately. During World War I, for example, Freud conceptualized grief as a normal response as compared to the pathology of depression (melancholia) or traumatic experience (shell shock) (Stroebe et al, 2001). However, these different ways of classifying emotional trauma began to converge with early descriptions of the neurobiology of stress. Initially viewed as a response to acute physical trauma, the stress response was later understood to also be activated by emotional trauma, and its significance was amplified by the discovery that prolonged stress has consequences for both physical and mental health (Sapolsky, 2015).

Until recently, the pathophysiology and psychiatric manifestations of emotional trauma have been conceptualized as having little in common with those of physical trauma. But greater study of the neurobiology of trauma has shown that overlapping brain regions are involved in both TBI and PTSD, and the more recent recognition of mTBI has expanded the overlap of symptoms and sequelae from both physical and emotional trauma. For example, combat-related mTBI doubles the risk for PTSD, and PTSD is strongly associated with persistent postconcussive symptoms (Menon et al, 2010).

Scientific advances continue to prompt redefinition of TBI and mTBI. A 2010 group of experts defined TBI as "*an alteration in brain function, or other evidence of brain pathology, caused by an external force,* where *alteration in brain function* encompassed cognitive–behavioral changes, and *external force* included not only head trauma, acceleration–deceleration and blast injuries, but also *other forces yet to be determined*" [emphasis added] (Menon et al, 2010, p. 1638; Stein and McAllister, 2009).

Concerns have arisen about the increasing overlap between physical and emotional traumatic pathologies based on the inclusion of milder trauma with subtle cognitive and affective symptoms (Menon et al, 2010). Potential confounds to the diagnosis of mTBI are cited (Ruff et al, 2009) because objective, focal neurologic deficits and abnormalities on neuroimaging are rarely seen, and deficits that fulfill mTBI criteria (eg, confusion, fatigue, sleep disorders) are less specific and may be caused by stress, anxiety, PTSD, or depression. Challenges to the development of diagnostic criteria for mTBI include disentangling physical and emotional trauma and differentiating altered mental status related to physical trauma from that related to strong emotional reaction (Ruff et al, 2009).

A minority of people with mTBI experience chronic postconcussive symptoms. Many of these symptoms overlap with PTSD, including sleep disturbance, trouble concentrating, anxiety, and depression (Menon et al, 2010). A comparison of individuals with mTBI and a control group of those with physical trauma (without brain injury) showed that acute posttraumatic stress and pain, or preinjury anxiety or depressive disorder, increased the risk of PCS. Overall, postconcussive symptoms were similar in the mTBI group and the trauma control group (Meares et al, 2011). In a study of US soldiers returning from Iraq, mTBI resulted in increased psychological and functional problems, yet these effects were not significant when PTSD and depression were considered (Bryant, 2008; Hoge et al, 2008). These results can be interpreted from two different perspectives, either where emotional trauma is a confound resulting in misdiagnosis of mTBI and PCS, or where both TBI and emotional trauma result in overlapping brain injury with comorbid causation of PCS.

The mechanism of emotional TBI has been investigated extensively by stress neurobiologists. These insights have extended the previous narrow concept of fight or flight (ie, the hypothalamic-pituitary-adrenal feedback loop) to include the intricate actions of stress hormones on diverse brain functions, from memory to learning to mood and to other aspects of behavior. The structural and functional plasticity of the brain is remarkably heightened by stress, with remodeling of neural architecture occurring over minutes to hours (McEwen, 2016). A U-shaped *dose*-response curve occurs, wherein moderate levels of stress hormones facilitate memory, and high levels of stress hormones suppress memory (Sapolsky, 2015).

Acutely elevated hormone levels have been shown to increase synaptic plasticity, in turn enhancing cognition, but chronically elevated stress hormone levels have been shown to reduce neurogenesis and cause dendritic atrophy, thereby impairing cognition (Sorrells et al, 2009). The anatomic localization of these effects corresponds with the neurobehavioral consequences. Chronic stress reduces neural connections in the hippocampus and medial frontal cortex, resulting in less inhibition of the fear center in the amygdala (McEwen, 2016; Sapolsky, 2015).

TBI commonly involves damage to the frontal and prefrontal cortices due to shearing forces of the brain being brought against the rough inner surface of the skull. Here again, structural and emotional mechanisms are intertwined and are corroborated by neuropathology showing a reduction of hippocampal volume in both individuals with TBI and those with PTSD (Menon et al, 2010; Stein and McAllister, 2009), and fMRI showing similar regional activations in both physical and emotional pain (O'Connor, 2012).

Both physical and emotional TBI have systemic effects due to hormonal and inflammatory mediators, including changes in proinflammatory cytokine levels, immune cell function, and heart rate variability. For example, reduced T-cell functioning and poorer lymphocytic response to pathogens have been shown in bereaved individuals versus controls, and the immunologic dysfunction was found to be more pronounced in early bereavement rather than late (O'Connor, 2012; O'Connor et al, 2009).

Proinflammatory cytokines including interferon gamma, interleukin IL-6, and tumor necrosis factor alpha have also been shown to be associated with the level of an individual's grief severity (Fagundes et al, 2019). Takotsubo cardiomyopathy (ie, broken heart syndrome) is a vivid example of these interactions, where stress hormones cause abnormal movement of the heart muscle, resulting in typical angina symptoms and, in some cases, infarction. Recent reports have described the co-occurrence of Takotsubo syndrome and transient global amnesia, where both are triggered by stressful experiences

associated with activation of the stress response (Sajeev et al, 2017).

Seven years have passed since the loss of my husband, and the signs of altered mental status secondary to emotional trauma continue to diminish. Stress-induced neural changes have given way to restored function as neural connections have been remodeled based on new experiences. Disproportionate emotional responses to various triggers and sleep disturbances are also resolving as overactive and sensitized neural pathways settle down.

Resistance to the concept of emotional TBI is likely to be based on several factors, including the historical definition, which emphasizes the type of inciting event rather than similar mechanisms and sequelae. The jury is still out on the reversibility of mTBI, PCS, PTSD, and emotional trauma. For example, mTBI was associated with a twofold increase in the risk of dementia in veterans, raising concern about risks of neurodegeneration (Barnes et al, 2018). Many different medical disciplines focus on TBI (eg, neurorehabilitation, psychiatry, neurosurgery, sports medicine), impeding the development of broader perspectives. Some will find emotional TBI a bridge too far, but it was not long ago that acceleration injury was unknown and concussion was considered benign.

Accumulating evidence favors a change in the definition of TBI to encompass both physical and emotional TBI. The definition of TBI will be strengthened by the inclusion of both physical and emotional trauma that result in diverse and overlapping forms of brain injury with sequelae for physical and mental health.

After recovery from the acute symptoms some would attempt to distinguish cerebral contusion from neurosis following the injury …. Difficulty arises from the facts that focal signs are usually absent in cerebral contusion and that certain mental symptoms are common to both cerebral contusion and anxiety neurosis. In practice it is impossible to make the distinction, and the attempt to do so is both unprofitable and undesirable. The patient must be regarded and treated as a psychophysiological unit.

W. Russell Brain, 1947

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