EDITORIAL

Is the Silent Epidemic Keeping Patients Awake?

Andreas A. Theodorou, M.D.1; Sydney A. Rice, M.D., M.S.2

1Pediatric Critical Care Medicine, University of Arizona, Tucson, AZ; 2Children’s Clinics for Rehabilitative Services, University of Arizona, Tucson, AZ

Approximately 1.4 million people sustain a Traumatic Brain Injury (TBI) each year in the United States according to statistics collected by the Centers for Disease Control and Prevention (CDC).1 Of these, 1.1 million individuals are seen, treated and released from an emergency department and 235,000 are hospitalized. Countless others sustain a mild TBI and never seek medical attention. Traffic accidents and falls make up the majority of TBIs, though the impact of injuries acquired on the athletic field may be as significant. The dramatic presentation of moderate to severe TBI draws initial attention but the long term consequences of even mild traumatic brain injury, including headaches, memory problems, personality changes, and attention deficits, are poorly understood and often go unidentified. Because of the staggering numbers and the potentially subtle cognitive, emotional and behavioral consequences, TBI has been referred to as the “silent epidemic.” Due to publicity surrounding repetitive head injuries to high profile athletes and to the large number of casualties in our overseas conflicts, this epidemic is no longer silent. This epidemic now calls out for our attention.

Wars have often been linked to a specific “signature” injury. World War II, with the use of atomic bombs, was associated with radiation-induced cancer. The Vietnam War will be remembered for the use of the defoliant Agent Orange and the physical ailments suspected by exposure to it and for the identification of Post Traumatic Stress Disorder (PTSD) in war veterans. Unfortunately, the signature injury for the current conflicts in Afghanistan and Iraq is the TBI.2 The use of advanced protective gear such as Kevlar helmets and body armor has reduced the risk of penetrating trauma and has improved survival rates. Kevlar helmets, though, do not completely protect from closed head injuries resulting from blasts such as those generated by improvised explosive devices (IEDs). It has been noted at Walter Reed Army Medical Center in Washington, D.C, that 59% of blast-exposed patients have a TBI, a figure representing thousands of veterans who will need ongoing care for those injuries.

For a successful outcome in a patient with TBI, more is required than the good work of intensivists, acute care nurses or neurosurgeons in an inpatient setting. That is just the beginning. Long-term success, as measured by the best quality of life possible, mandates the use of an interdisciplinary team that can assess and manage a complex individualized rehabilitation process. The three articles presented in the current issue address the poorly understood and underappreciated relationship of sleep disorders and TBI.

Verma and colleagues3 retrospectively studied patients with chronic TBI, defined as three months to two years following the injury, who presented with sleep complaints, and evaluate the etiology of those complaints. Mild, moderate and severe TBI patients were included as defined by the Global Assessment of Functioning (GAF) scale. Results suggest that disordered sleep is related to poor quality of sleep documented by polysomnogram (PSG) and associated anxiety and depression. Forty-five percent of the subjects in this study had a BMI of over 30 kg/m2 and reported significant weight gain after their injury. The authors speculate on mechanisms for weight gain including hypothalamic injury, inactivity due to neurological impairment, and medications. They suggest that the correct diagnosis and treatment of the sleep disturbance may contribute to the successful rehabilitation of the patient.

Watson and colleagues4 conducted a prospective cohort study utilizing 514 consecutive patients admitted to a level 1 trauma center, collecting data at one month and at one year. Injury severity was defined using the motor component of the Glasgow Coma Scale (GCS) and sleepiness was evaluated using a portion of the Sickness Impact Profile. This article suggests that there is an increased incidence of daytime sleepiness during the first year after TBI, but that this symptom improves by one year after injury. Controls for this study included individuals who had experienced a traumatic injury exclusive of head trauma. All individuals with traumatic injury reported sleepiness at one year after injury, but this may be an underestimation of the true prevalence of sleep disorders in TBI patients due to inability of the more severely injured patients to respond. The identification of increased sleepiness in the trauma controls may suggest a non-cerebral source of sleep problems such as pain and weight gain with resulting obstructive sleep apnea related to inactivity and medications. It should be noted that even though more severe injuries resulted in greater sleepiness, it was common even in mild TBI.

Castriotta and his colleagues5 prospectively studied subjects that were at least three months post-TBI without specific complaints of sleep disorder. TBI severity was graded by GCS and CT scan. Almost half of these subjects had evidence of some form of sleep...
disorder as tested by PSG with no relationship to severity of injury. In contrast to the Verma study, an association with mood disorder was not found. Sleepy subjects had slower reaction times and better self-reported quality of life. The authors speculate that differences in reported quality of life may reflect lack of insight; this seems reasonable and could be supported by reporting severity of injury or level of function along with reported quality of life.

As clinicians who treat acute and chronic TBI, we are intrigued by the combined evidence in the three presented articles demonstrating the variety of sleep disorders suffered by TBI patients. Though the pathogenic mechanism for the sleep disorders was not the purpose of any of the studies, several possibilities were presented including direct or indirect injury to various parts of the brain and brainstem, alterations in neuroendocrine function, and physical changes such as obesity. It was also not studied, yet speculated, that untreated sleep disorders adversely affect outcome in TBI patients. This premise should be tested in well-designed studies. Meanwhile, as the “silent epidemic” spreads and we search for every treatment option to minimize the consequences and optimize quality of life for those who have been affected, it seems that we have an important new colleague on the interdisciplinary TBI team – the sleep specialist.

REFERENCES