Are Sleep Disturbances a Risk for Chronic Traumatic Encephalopathy? Only the Shadow Knows

Stuart F. Quan, M.D., F.A.A.S.M.

Editor, Journal of Clinical Sleep Medicine; Division of Sleep Medicine, Brigham and Women’s Hospital and Harvard Medical School, Boston, MA, Arizona Respiratory Center, University of Arizona College of Medicine, Tucson, AZ

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unior Seau, Ollie Matson, John Mackey. What do these individuals have in common? History of National Football League (NFL) stardom? Enshrinement or possible induction into the Pro Football Hall of Fame? Both are true. Yet all share something much more ominous. Death related to the sequelae of chronic traumatic encephalopathy. Chronic traumatic encephalopathy (CTE) is characterized by memory loss, depression, dementia and potentially premature death. In some individuals, such as Junior Seau—and more recently major league baseball player Ryan Freel—it has been posited as the cause of their suicide. Pathologic examination of the brain of those with CTE reveals a degenerative neurologic process which is now attributed to repetitive concussive brain trauma. With the revelation that some living former NFL stars such as Brett Favre and Tony Dorsett are having symptoms suggestive of CTE, there is growing concern about the safety of all sports that can result in concussive brain injury. This level of concern has caused the NFL and college football to change their rules regarding tackling, leading to penalties and possible ejection for tackles that target the head. The NFL in cooperation with the National Institutes of Health also has begun funding research in this area with most of the emphasis on early detection using brain imaging techniques and further studies of the neuropathology. However, is repetitive concussive injury the only risk factor for CTE? Could obstructive sleep apnea (OSA) and other sleep disturbances be contributing or exacerbating factors?

Several recent studies document that many individuals who have suffered acute traumatic brain injury (TBI) are subsequently found to have a variety of sleep disturbances including OSA. In some of these studies, the prevalence of OSA and insomnia has been reported to be as high as 23% and 25% respectively. Furthermore, data suggest that cognitive function is worse in patients who have both OSA and previous TBI than in those with TBI alone. In addition, treatment for sleep disturbances in the setting of chronic TBI may improve cognition and mood. Studies of retired and active NFL football players have found that the prevalence of OSA ranges from 14% to 19%. However, the prevalence may be much higher in retired players. Awareness of the significance of OSA prompted NFL Charities to fund the development of a short online screening application for all players although there has been little interest in teams to use it. In addition to their respective adverse effects on sleep, both OSA and insomnia have been linked to the development of mood disturbances including depression which are also observed with CTE. Recently, epidemiologic studies have identified a link between both OSA and insomnia, and subsequent development of dementia. With respect to OSA, a variety of neuroimaging studies suggest evidence of brain neuronal loss or dysfunction. Gray matter abnormalities also have been observed some, but not all studies of patients with chronic insomnia.

Given the interconnections among TBI, OSA, insomnia, mood disturbances, cognitive decline and neurodegeneration, one can speculate that OSA and insomnia may be additional risk factors or important modifying factors for the development of CTE. For example, could the development, severity or progression of CTE occurring after repetitive brain injury be worsened in the presence of insomnia or OSA? An interesting and provocative question? Hopefully so for sleep and brain injury investigators, parents, athletes and of course sports fans. Perhaps further research will provide enlightenment in this area. Until then, only the Shadow knows.

CITATION


REFERENCES


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