|  |
| --- |
| **Name:** |
| Cameron Young |
| **Group:** |
| 5A-5 |
| **Pathology Question:** |
| What are the contributing factors for bruxism? |
| **Report:** |
| According to Lobbezoo et al., bruxism can be defined as “repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible.” Bruxism can be described as having two distinct circadian manifestations, sleep bruxism which occurs during sleep and awake bruxism which occurs during wakefulness. The exact etiology of bruxism is complex, controversial and most likely multifactorial, however many of these factors can be attributed to central disturbances involving variations in neurotransmitter release. Contributing factors of bruxism can be categorized into drug-associated factors, psychosocial factors, and physiologic factors. Prevalence of sleep bruxism is higher among females than males and is greatest in children and decreases in the adult population. Enough of the population is impacted where better understanding of the contributing factors is important in its diagnosis and treatment.  Recent studies have mainly focused on sleep bruxism due to the ability to reliably track sleep activity through polysomnography. The study of awake bruxism relies on questionaires and anecdotal evidence from patients which limits the information that can be gathered. From the limited studies that have been performed pertaining to awake bruxism, there may be a general association between increased stress, anxiety and fatigue and increased bruxism. Specific neurologic disorders such as Parkinson’s disease and Huntington’s disease may also play a role in bruxism.  Multiple studies have shown an association between drug use and sleep bruxism. Alcohol, caffeine, and nictotine tend to be the drugs most studied. Intake of large quantities of alcohol in short periods of time have toxic effects on the brain and have a role in CNS disturbances which may initiate or make sleep bruxism worse. Consumption of large amounts of caffeine have been shown to be associated with sleep bruxism due to the fact that caffeine acts as a CNS stimulant. Nicotine has been shown to have a significant association with bruxism in a dose-dependent manner through the drug’s influences on central dopaminergic activities. Other prescription drugs such as antidepressants, antipsychotics, and amphetamines also have some possible causal relationship with bruxism. Finally, there have been several studies that suggest an association between the use of the illicit drug MDMA and bruxism but the evidence is weak, mostly relying on narrative reviews, select case studies and anectodal evidence.  Recent studies have made suggestions that sleep bruxism may play some role in normal physiology. One proposed hypothesis is that sleep bruxism plays a protective role in gastroesophogeal reflux (GER). When pH of the esophagus is decreased from increasing acidic contents during GER, rhythmic masticatory muscle activity epidsodes increase in frequency. This may suggest that GER has a casual relationship with sleep bruxism and may even be protective to prevent aspiration or mucosal injury from acidic secretions. |
| **References:** |
| Bertazzo-Silveira, Eduardo, et al. “Association between Sleep Bruxism and Alcohol, Caffeine, Tobacco, and Drug Abuse.” The Journal of the American Dental Association, vol. 147, no. 11, 2016, doi:10.1016/j.adaj.2016.06.014.  Feu, Daniela, et al. “A Systematic Review of Etiological and Risk Factors Associated with Bruxism.” Journal of Orthodontics, vol. 40, no. 2, 2013, pp. 163–171., doi:10.1179/1465313312y.0000000021. |