Study identifies how muscles are paralyzed during sleep

July 11 2012

Two powerful brain chemical systems work together to paralyze skeletal muscles during rapid eye movement (REM) sleep, according to new research in the July 11 issue of The *Journal of Neuroscience*. The finding may help scientists better understand and treat sleep disorders, including narcolepsy, tooth grinding, and REM sleep behavior disorder.

During REM sleep — the deep sleep where most recalled dreams occur — your eyes continue to move but the rest of the body's muscles are stopped, potentially to prevent injury. In a series of experiments, University of Toronto neuroscientists Patricia L. Brooks and John H. Peever, PhD, found that the neurotransmitters gamma-aminobutyric acid (GABA) and glycine caused REM sleep paralysis in rats by "switching off" the specialized cells in the brain that allow muscles to be active. This finding reversed earlier beliefs that glycine was a lone inhibitor of these motor neurons.

"The study's findings are relevant to anyone who has ever watched a sleeping pet twitch, gotten kicked by a bed partner, or has known someone with the sleep disorder narcolepsy," said Dennis J. McGinty, PhD, a behavioral neuroscientist and sleep researcher at the University of California, Los Angeles, who was not involved in the study. "By identifying the neurotransmitters and receptors involved in sleep-related paralysis, this study points us to possible molecular targets for developing treatments for sleep-related motor disorders, which can often be debilitating," he said

The researchers measured electrical activity in the facial muscles responsible for chewing of sleeping rats. Brain cells called trigeminal motor neurons communicate the brain's message to move to these muscles. Previous research suggested neurotransmitter receptors called ionotropic GABAA/glycine receptors in the motor neurons caused REM sleep paralysis. However, when the researchers blocked these receptors, REM sleep paralysis still occurred.

The researchers found that to prevent REM sleep paralysis, they had to block both the ionotropic receptors and metabotropic GABAB receptors, a different receptor system. In other words, when the motor cells were cut off from all sources of GABA and glycine, the paralysis did not occur, allowing the rats to exhibit high levels of muscle activity when their muscles should have been inactive. The data suggest the two neurotransmitters must both be present together to maintain motor control during sleep, rather than working separately.

The finding could be especially helpful for those with REM sleep disorder, a disease that causes people to act out their dreams. This can cause serious injuries to patients and others around them. It is also often an early indicator of neurodegenerative diseases, such as Parkinson's.

"Understanding the precise mechanism behind these chemicals' role in REM sleep disorder is particularly important because about 80 percent of people who have it eventually develop a neurodegenerative disease, such as Parkinson's disease," study author Peever added. "REM sleep behavior disorder could be an early marker of these diseases, and curing it may help prevent or even stop their development," he said.

Provided by University of Toronto