CATATHRENIAS: PARASOMNIA OR OSA SYMPTOM?

by Regina Patrick RPSGT

Catathrenia (derived from the Greek words kata meaning “low” and threnia meaning to “lament”) is a sleep-related phenomenon recently classified in the International Classification of Sleep Disorders Diagnostic and Coding Manual (ICSD-2) as a parasomnia. (A parasomnia is a movement or behavior that manifests either during sleep, during the sleep-wake transition, or during specific sleep stages.) Catathrenia sufferers make a loud, monotonous sound during exhalations while asleep. The sound has a morose quality; hence, it is also called “nocturnal groaning.” A person with catathrenia is not aware of making the noise but bedpartners or family are often frightened or disturbed by it. For some people, continuous positive airway pressure (CPAP) treatment stops the groaning which suggests that catathrenia may result from upper airway constriction. For this reason, some scientists argue that catathrenia should be considered a symptom of obstructive sleep apnea (OSA) rather than a parasomnia.

A person with OSA has an absence of breathing (i.e., apnea) for brief periods during sleep due to blockage (i.e., obstruction) of the upper airway. The obstruction occurs because upper airway tissues such as tonsils, soft palate, and adenoids are drawn into the airway with inspirations. This restricts airflow and as a result causes the blood oxygen level to fall. Ultimately, the person arouses for a few seconds to take some deep breaths and restore the blood oxygen level to normal. Loud snoring typically occurs during inhalations as the person is taking the deep breaths. The snoring noise results from the vibration of the upper airway tissues as air is quickly drawn in. In some people, the vibration continues during exhalations resulting in expiratory snoring. Although catathrenia only takes place during exhalations, it does not appear to be expiratory snoring.

Catathrenia begins with a deep, silent inspiration followed by a prolonged audible exhalation (i.e., groan). Several groans may occur in a catathrenia episode with each groan lasting 2 to 20 seconds. Catathrenia episodes can occur intermittently throughout a sleep period. Since exhalation is prolonged, the person’s breathing rate slows down during a catathrenia episode. Interestingly, there is no respiratory effort during the production of the groaning noise and no oxygen desaturation occurs despite the slower breathing rate. Catathrenia occurs solely or mainly during REM sleep.

In 1983, Belgian scientists de Roeck and van Hoof were the first to describe catathrenia, a phenomenon which they called expiratory groaning. Their subject was a male whose groaning took place primarily during REM sleep and only during exhalations. The subject’s exhalations were prolonged and appeared forced. At the time, the researchers surmised that the noise may have resulted from either (1) vibration of the vocal cords as air is exiting from the lungs; (2) vibration of upper airway tissues above the point of the vocal cords as air is exiting from the lungs; or (3) as a consequence of a brain tumor, epileptogenic focus, or other lesion in brain areas that control respiration. This was the only mention of the phenomenon in medical literature for 18 years. In 2001 an Italian research team, headed by Roberto Vetrugno, and a Belgian research team, headed by Dirk Pevernagie, investigated catathrenia in separate studies. Vetrugno’s team coined the term “catathrenia” for nocturnal expiratory groaning. Both research teams were to conclude that catathrenia is a parasomnia.

The Vetrugno study involved four subjects. In all subjects the groaning started 2 – 6 hours after sleep onset; it occurred in both NREM sleep and REM sleep but particularly in REM sleep; and it occurred during exhalations only. A groan would last 2 – 20 seconds. A catathrenia episode could last 2 minutes to 1 hour. Every catathrenia episode ended with a snort. There appeared to be no respiratory effort during the groans and the noise could not be explained by noises such as wheezing or stridor (i.e., a harsh, high-pitched inspiratory noise) that occur with asthma or other respiratory diseases. Results of fibroscopic examination of the vocal cords and upper airway were normal. Since catathrenia did not appear to be a respiratory-related noise, the authors concluded that it was a new parasomnia.

The Pevernagie study involved ten subjects. The researchers monitored esophageal pressure changes during catathrenia in two of the subjects during sleep. They found that the esophageal pressure slowly decreased with each groan, indicating that the respiratory muscles were not activated during the production of sound. Therefore, the groaning noise could not be explained by increased respiratory effort during exhalations. Since it did not appear to be a respiratory-related disorder, they also concluded that catathrenia was a new parasomnia.

Sleep researchers Christian Guillenmaut et al. suspect that, despite these findings, catathrenia may indeed be the result of sleep-disordered breathing. In their 2008 study, they reported their experience with seven women with catathrenia whose symptoms resolved when treated with CPAP or with sleep apnea surgery (e.g., uvulopalatopharyngoplasty [UPPP]). All of the women had a small oropharynx as evidenced by their small jaws and a narrowed upper airway.
During polysomnographic monitoring, Guilleminault et al. observed increased respiratory effort during the production of the groaning noise. However, the scientists did not objectively validate this by measuring abdominal/thoracic effort. The nasal cannula recording on the polysomnogram (PSG) indicated decreased airflow during each groan. Since increased respiratory effort with decreased airflow indicates airway obstruction and since a small oropharynx is a risk factor for OSA, Guilleminault et al. concluded that catathrenia may be a respiratory-related sleep disorder – in other words, a symptom of OSA – rather than a parasomnia.

Conflicting research findings have made effectively treating catathrenia a frustrating problem. Based on current findings, catathrenia should be approached either as a parasomnia with a possible central origin or as a sleep-related breathing disorder. However, antidepressants or other drugs aimed at altering the activation of the respiratory centers in the brain and CPAP therapy or apnea surgery aimed at correcting the anatomical problems involved in sleep-disordered breathing have had inconsistent or results.

Pevernage treated two of the subjects with CPAP therapy. The treatment reduced catathrenia episodes in one patient but it had no effect on catathrenia in the second patient. On the other hand, CPAP therapy stopped catathrenia episodes in all of Guilleminault’s female subjects. Pevernage treated the other eight subjects with the benzodiazepine clonazepam or antidepressants drugs (e.g., paroxetine, dosulepine, and trazadone). The subjects had one of three outcomes with drug therapy: (1) the catathrenia episodes remained but the loudness of the noise was reduced; (2) the catathrenia episodes were temporarily eliminated; or (3) the catathrenia episodes continued at their pretreatment level.

Scientists are further frustrated by the inability to determine exactly how the noise in catathrenia is produced. As a result, scientists have proposed theories which have yet to be investigated in research studies.

One theory is that the natural recoil of the lungs may play a role. In other words, as the lungs slowly deflate in the absence of respiratory effort, air exiting from the lungs vibrate the vocal cords and produce the noise. A second theory is that catathrenia may be a “grown up” version of the baby sigh (a deep inspiration followed by prolonged audible exhalation). Like a baby sigh, catathrenia involves a deep breath on inhalation, prolonged exhalation, and vocalization during exhalation. Infants sigh frequently during sleep which scientists suspect may play a role in maintaining the distensibility and surface tension of lung tissue and airway walls and in stabilizing the rhythmicity of respiratory movements (i.e., inhalation and exhalation). The slowed breathing pattern in a catathrenia episode may serve the same purposes.

On a PSG, catathrenia looks like a central apnea since there is no airflow or thoracic effort on the respiratory channels. However, one distinguishing feature of catathrenia is that it always involves the production of sound during the apparent lack of breathing; a central apnea never does. This distinction can mean the difference between diagnosis and misdiagnosis. For this reason, it is important for a recording technologist to note sounds and the quality of sounds occurring during what appears to be a central apnea.

Catathrenia is a rare phenomenon. Just slightly more than 35 people with catathrenia have been described in the medical literature since 1983. Existing studies of catathrenia have left scientists with more questions than answers. For example, are the different responses to CPAP, surgery, and drug treatment an indication that there may be more than one type of catathrenia? Is the disorder the result of an anatomical problem in the airway or is it the result of a central dysfunction or both? Is there any significance to catathrenia that occurs during REM sleep vs. catathrenia that occurs during NREM sleep? Answers can not come soon enough for a person with catathrenia since it can have socially negative effects. The noise can be embarrassing for a catathrenia sufferer; disruptive to the sleep of a bed partner; or frightening for a person witnessing it for the first time. Future studies hopefully may soon settle the controversy over whether catathrenia is a sleep-related breathing disorder or a parasomnia. But more importantly, increased scientific focus on the disorder may lead to improved treatment for catathrenia sufferers.

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