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Polysomnographic features of sleep paralysis (SP) captured in a patient with narcolepsy
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Introduction
Humans rely on a mechanism known as muscle atonia during REM sleep in order to protect against injurious behavior that may occur during emotionally-charged dream enactment [1]. SP is a transient episode of muscle atonia that occurs during the transition between sleep and wakefulness, clinically manifesting as inability to perform voluntary movement while consciousness is entirely preserved [2,3]. Isolated SP is known to occur in healthy individuals [1], however, recurrent SP is known as a well-documented feature of narcolepsy [4]. There is limited literature describing the polysomnographic features of SP, partly owing to its rarity and likelihood of being captured during a single night’s study [3]. We aim to illustrate the key neurophysiological features in a patient experiencing SP on polysomnogram.

Methods
We report a 24-year-old female who presented to our sleep clinic for excessive daytime somnolence and recurrent SP episodes upon awakening from naps. Despite sleeping 12 hours per night, she reported an irresistible urge to nap during the day. She also reported experiencing a vivid “dream-like” state and inability to move her body voluntarily upon awakening from naps. During such times, she experienced a sensation of fear, causing her to hyperventilate. She denied having episodes of cataplexy.

Multiple sleep latency test (MSLT) was consistent with narcolepsy. She reported an episode of SP arising from her final sleep-onset REM period (SOREMP). Figure 1 displays a 30-second epoch marking the transition from REM sleep to wakefulness. Initially, she is in REM sleep, characterized by theta-predominant EEG background and chin EMG atonia. Phasic eye movements are not captured in this tracing. The arrow depicts the transition to wakefulness, characterized by alpha-predominant EEG, however EMG tone remains suppressed, indicating SP.

Results
Figure 2 displays a 30-second epoch marking resolution of SP during wakefulness. The arrow depicts gradual increase in EMG tone, followed by bursts of muscle tone enhancement, indicating resolution of SP.

Discussion/Conclusion
Our patient’s MSLT tracing displays mutual existence of REM and wake EEG features occurring in tandem with persistent muscle atonia on chin EMG [3]. This phenomenon underlies the clinical experience of immobility despite transitioning from REM sleep to wakefulness. In narcolepsy, there is loss of hypocretin-mediated suppression of REM-sleep [5], which may explain the inappropriate intrusion of REM-sleep features into wakefulness. SP is most common within the first two hours of sleep-onset or within the hours prior to awakening, corresponding to when timing of SOREMPs and REM-sleep predominate, respectively [2,6].

References