Cataplexy in Anxious Patients: Is Subclinical Narcolepsy Underrecognized in Anxiety Disorders?

Dawn L. Flosnik, M.D.; Bernadette M. Cortese, Ph.D.; and Thomas W. Uhde, M.D.

Objective: Excessive daytime sleepiness, hypnagogic-hypnopompic hallucinations, sleep paralysis, and cataplexy are symptoms associated with narcolepsy. Recent findings indicate that anxiety disorders also are associated with excessive daytime sleepiness, hypnagogic-hypnopompic hallucinations, and sleep paralysis. These observations suggest a possible relationship between anxiety disorders and narcolepsy. Cataplexy is considered the most specific symptom of narcolepsy, but its association with anxiety disorders is unknown. This preliminary investigation examined the prevalence and types of cataplexy in patients with primary anxiety disorders.

Method: Sex- and age-matched patients with anxiety disorders (N = 33) and healthy volunteers (N = 33) were assessed on standardized and validated measures of subjective sleep quality (Pittsburgh Sleep Quality Index) and subclinical narcoleptic events in the form of cataplexy (Stanford Center for Narcolepsy Revised Sleep Inventory). Patients were recruited from October 2006 to January 2007 from 2 programs of the Penn State Behavioral Health Clinic.

Results: Anxiety disorder patients as a group reported poorer sleep quality and endorsed a larger number of different types of situations (e.g., surprise, embarrassment) associated with cataplectic events. Among anxious patients, 33.3% (11 of 33) endorsed events specific for classic cataplexy, as opposed to 9.1% (3 of 33) of healthy volunteers ($\chi^2 = 5.80$, $p = .016$).

Conclusions: Our preliminary findings suggest that anxiety disorders are associated with increased rates of cataplexy. Future research is indicated to elucidate the relationship between anxiety and narcolepsy, with a particular focus on panic and generalized anxiety disorders.

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Received April 3, 2008; accepted July 29, 2008. *From Penn State University College of Medicine, Hershey, Pa. (Dr. Flosnik); and the Department of Psychiatry and Behavioral Sciences, Institute of Psychiatry, Medical University of South Carolina, Charleston (Drs. Cortese and Uhde). Dr. Flosnik is now with the Department of Psychiatry and Behavioral Sciences, George Washington University, Washington, D.C.*

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Corresponding author and reprints: Thomas W. Uhde, M.D., Department of Psychiatry and Behavioral Sciences, Medical University of South Carolina, IOP 5 South, 67 President St., Charleston, SC 29425 (e-mail: uhde@musc.edu).

Anxiety, stress, and trauma are a major public health concern in our society today, costing the United States more than $42 billion dollars a year in psychiatric care, nonpsychiatric care, emergency care, hospitalization, prescription drugs, reduced productivity, and absenteeism from work.[1] Over 15 million adults per year and 30 million people at some point in their lifetime will be afflicted with an anxiety disorder, making these disorders the most prevalent mental health problem in the United States.[1] Anxiety disorders tend to be chronic, lifelong illnesses with recurrent episodes and significant impairment in work and social functions.[1–3]

Anxiety disorders are commonly associated with insomnia[4–6] and have also been linked with excessive daytime sleepiness (EDS).[7,8] In fact, the literature has underscored the high prevalence of chronic intermittent sleep deprivation and resultant EDS in anxiety patients with nocturnal sleep panic attacks.[3,9] Moreover, a recent publication has reported a high prevalence of freezing/immobilization behaviors during acute episodes of anxiety (i.e., panic attacks), which have some phenomenological overlap with sleep paralysis and related immobilization experiences in patients with recurrent sleep paralysis disorder.[10] Also, it has recently been suggested that patients with posttraumatic stress disorder (PTSD) develop not only rapid eye movement (REM)–related flashbacks of prior traumatic events but also hypnagogic and hypnopompic hallucinations, as well as sleep paralysis.[11,12] Thus, anxiety disorders are known to be associated with insomnia and EDS, and emerging new information suggests that freezing and immobilization experiences, as well as sleep-related hypnagogic and hypnopompic hallucinations and sleep paralysis,[12] may be much more common than previously appreciated in some anxiety disorders, perhaps particularly panic disorder (PD) and PTSD.

Narcolepsy is characterized by episodes of cataplexy (partial or full muscle atonia), sleep paralysis, hypnagogic and hypnopompic hallucinations, and sleep attacks/EDS.[13,14] Although debate surrounds the question of whether cataplexy should be an essential or required diagnostic feature of narcolepsy,[15] Anic-Labat and colleagues,[16] in addition to others,[17] report that a positive history of cataplexy is more predictive of narcolepsy than excessive daytime sleepiness, hypnagogic-hypnopompic

hallucinations, sleep paralysis, or results of a Multiple Sleep Latency Test (MSLT). These findings suggest that a positive history of cataplexy may be sufficient to diagnose narcolepsy.[16–18] The diagnosis of narcolepsy, therefore, can be made without the presence of cataplexy, but cataplexy is the most specific symptom of narcolepsy, and its presence is considered almost pathognomonic for narcolepsy by many physicians.[19] Given that anxiety disorders have already been associated with other key symptomatic features of narcolepsy (e.g., excessive daytime sleepiness, sleep paralysis), this study focused its evaluation on cataplexy.

Many people with narcolepsy, particularly early in the course of illness, report anxiety and fear.[20,21] Whether such narcoleptic anxiety is simply a psychological complication of sleep paralysis or hallucinatory experiences or represents a distinct and separate core feature of narcolepsy remains unknown. Moreover, cataplexy is often triggered by strong emotions, such as anxiety and sudden fear.[14,22–25] Taken together, these observations suggest that biological mechanisms underlying fear or arousal may contribute to a convergent or comorbid relationship between some anxiety disorders and narcolepsy.

To our knowledge, no research team has examined the link between anxiety disorders and narcolepsy. In this preliminary study, the validated Stanford Center for Narcolepsy Revised Sleep Inventory (Stanford-RSI)[16] was administered to patients with primary anxiety disorders and healthy volunteers. Our goal was to examine the proportion of patients with primary anxiety disorders who report cataplexy, and the types of cataplexy, to ascertain whether more comprehensive investigations are justified to study the phenomenological, neurobiologic, and genetic relationships between anxiety disorders and narcolepsy.

METHOD

Subjects

Anxiety patients were recruited from the Outpatient Anxiety Clinic or Partial Hospitalization Program of the Penn State Behavioral Health Clinic from October 2006 to January 2007. Patient charts were examined to identify potential participants based exclusively on a well-documented history and diagnosis of a primary DSM-IV anxiety disorder. Patients were then re-interviewed by a psychiatrist or licensed psychologist to verify the current DSM-IV anxiety disorder. For patients with comorbid psychiatric diagnoses, determination of anxiety as the primary disorder was based on whether the anxiety preceded or developed subsequent to the comorbid condition. Ninety-one percent of the patients were being treated for their anxiety symptoms with psychotropic medications (79% with selective serotonin reuptake inhibitor/serotonin-norepinephrine reuptake inhibitor drugs, 39% with adjunctive benzodiazepines, and 12% with adjunctive second-generation atypical antipsychotic medications). None of the patients was receiving cholinergic agonists.

Sex- and age-matched healthy volunteers were recruited via flyers that were placed within the Penn State College of Medicine and the Milton S. Hershey Medical Center. A telephone screening questionnaire was administered to assess past or present psychiatric, medical, and sleep disorders. Healthy volunteers were excluded if they had a diagnosed psychiatric disorder or major medical disorder or if they were taking psychotropic medications. However, neither anxious patients nor potential volunteers were excluded because of their sleep histories. A majority of the healthy volunteers were medical students, graduate students, or hospital employees.

This study was approved by the Pennsylvania State Hershey Medical Center Institutional Review Board.

All participants gave written informed consent after the study had been explained to them and before they had been enrolled.

Assessment Tools

All participants were given a packet of surveys to complete, which included the Stanford-RSI and the Pittsburgh Sleep Quality Index (PSQI).[26] Thirty-three anxiety patients and 33 age- and sex-matched healthy volunteers who completed the entire packet prior to September 5, 2007, were included in the final analysis.

Pittsburgh Sleep Quality Index. The PSQI was administered to all subjects. The PSQI consists of 7 component scores, each ranging on a scale from 0 to 3. Component scores are added to calculate the PSQI Global score. The Global scale scores range from 0 to 21, with higher scores indicating poorer sleep quality.[26] PSQI Global scores greater than 5 are indicative of a possible sleep disturbance.[26] As expected, the anxiety patients had an overall mean ± SD PSQI Global score of 10.9 ± 5.5, while the mean ± SD PSQI Global score for healthy volunteers was 4.8 ± 2.7. Thus, our anxiety patients and normal controls had scores representative of previously published norms within their diagnostic groups.[16,27–29]

Stanford Revised Sleep Inventory. The Stanford-RSI was derived from the larger 146-item Stanford Center for Narcolepsy Sleep Inventory, and it is a validated 51-item, self-administered questionnaire focused solely on cataplexy. Although the larger questionnaire provides a more complete assessment of narcolepsy, including hypnagogic hallucinations, sleep paralysis, and excessive daytime sleepiness, symptoms of narcolepsy that have been previously reported to be increased in anxious patients,[7,8,11,12] our focus for the present study was Stanford-RSI–assessed cataplexy, a behavior that has not previously been explored thoroughly in anxious patients. Questions 1 to 21 of the Stanford-RSI, which refer to particular situations/triggers that evoke symptoms of cataplexy, were utilized for the analysis. Although the total number of

situations that provoke cataplectic events endorsed by each participant was compared between anxious patients and healthy volunteers, our main focus was the 3 situations/triggers that Anic-Labat and colleagues[16] found to be most predictive for having clear-cut cataplexy. According to Anic-Labat and colleagues,[16] clear-cut, or what we characterize in this article as classic, cataplexy is best distinguished from other, nonspecific types of physiologic muscle weakness when elicited by 3 distinct situations/triggers: "when laughing," "when angry," or "when hearing and/or telling a joke."[16] In fact, the risk of having clear-cut cataplexy approached 92% for subjects who endorsed muscle weakness triggered "when hearing and/or telling a joke" and "when angry."[16] The aim of the present study was to evaluate the responses obtained from anxious patients and healthy volunteers with respect to these 3 classic situations as well as broader-spectrum (i.e., nonspecific) triggers of cataplexy.

Statistical Analysis

Because our primary goal was to assess whether anxiety disorders in general might be associated with increased rates of nonspecific or classic types of cataplexy, we recruited a broad range of participants with primary anxiety disorders. We also examined the rates of cataplexy within anxiety subtypes (e.g., panic disorder, PTSD) but these analyses were exclusively exploratory in nature, and our study was neither designed nor statistically powered to identify significant anxiety subtype × cataplexy interactions. Thus, we first compared all anxiety patients versus healthy volunteers in a primary analysis; then, if there were significant group differences between the total group of anxious patients versus healthy volunteers, we performed a further analysis to explore, in a preliminary manner, whether certain anxiety subtypes appeared to be associated with unusually high rates of cataplexy.

Subjects were divided into 2 groups: anxious patients (N = 33) and healthy volunteers (N = 33). Data were analyzed using the statistical program SPSS version 14 (SPSS Inc., Chicago, Ill.). Data were reported as mean ± SD. The 2 groups were compared by χ^2 analysis or analysis of variance for significant differences in demographic and clinical features. Bonferroni post hoc analyses were conducted as indicated. Two-tailed p values < .05 were used to determine significant differences.

RESULTS

Thirty-three patients being treated in the Behavioral Health Clinic met criteria for at least one DSM-IV primary anxiety disorder. The primary diagnoses of the anxiety patients were posttraumatic stress disorder (PTSD), (N = 5, 15.2%), generalized anxiety disorder (GAD), (N = 14, 42.4%), panic disorder (PD), (N = 9, 27.3%), and social anxiety disorder (SAD), (N = 5, 15.2%). Thirty-three age- and sex-matched participants did not have a psychiatric diagnosis nor were they taking psychotropic medications; this group was designated healthy volunteers.

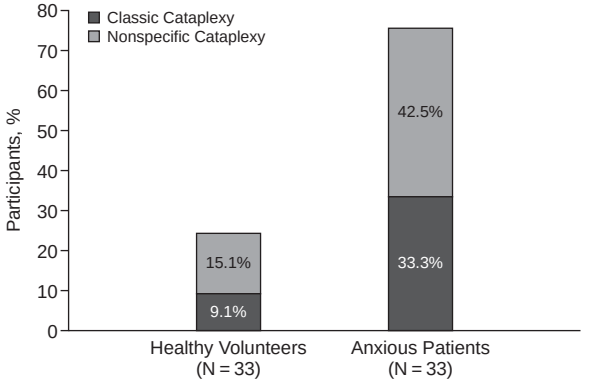
The groups did not differ with respect to sex (χ^2 = 0.00, p = 1.00). The sex distribution for both anxious patients and healthy volunteers was as follows: 78.8% (N = 26) were female and 21.2% (N = 7) were male. Age was not significantly different between the total group of anxiety patients versus healthy volunteers or among any of the anxiety subgroups and healthy volunteers (F = 1.12, df = 4,65; p = .36). Specifically, patients in the total anxiety group were a mean ± SD age of 43.6 ± 13.1 (range, 20–67) years, while healthy volunteers were 43.4 ± 12.8 (range, 22–67) years. Broken down by anxiety diagnosis, mean ± SD age was as follows: PTSD (50.0 ± 6.0 years), GAD (46.6 ± 14.8 years), PD (37.4 ± 8.9 years), and SAD (40.2 ± 16.8 years).

According to the Stanford-RSI, anxiety patients as a group reported a greater mean ± SD number of different types of situations (e.g., surprise, embarrassment) associated with cataplectic events (6.0 ± 5.9; range, 0–23) compared to healthy volunteers [0.8 ± 1.9; range, 0–8; (F = 10.80, df = 4,65; p = .000)]. Post hoc Bonferroni analyses revealed that patients with PD (9.1 ± 5.5) endorsed significantly more situations associated with cataplectic events compared to both patients with SAD (mean ± SD 0.2 ± 0.4; p = .002) and the healthy volunteers (0.8 ± 1.9, p = .000), whereas patients with GAD (6.0 ± 6.3) reported a significantly greater mean ± SD number of different cataplectic-related situations compared to healthy volunteers (p = .001) but not SAD (p = .077) patients. A similar but non-significant increase in the number of different types of cataplectic situations was also found in patients with PTSD (6.2 ± 5.2, p = .07) compared with healthy volunteers.

Overall, 75.8% (N = 25) of anxious patients endorsed at least 1 cataplectic event (classic and/or nonspecific) in contrast to only 24.2% (N = 8) of healthy volunteers (χ^2 = 17.52, p = .000). Since classic cataplexy is believed to be best differentiated from other types of muscle weakness when triggered by laughing, anger, or hearing/telling a joke,[16] we compared the responses of these 3 items between anxiety patients and healthy volunteers. Nearly 4 times the number (N = 11; [33.3%]) of anxiety patients endorsed classic cataplectic events compared to only 9.1% (N = 3) of healthy volunteers, a difference that was statistically significant (χ^2 = 5.80, p = .016; Figure 1).

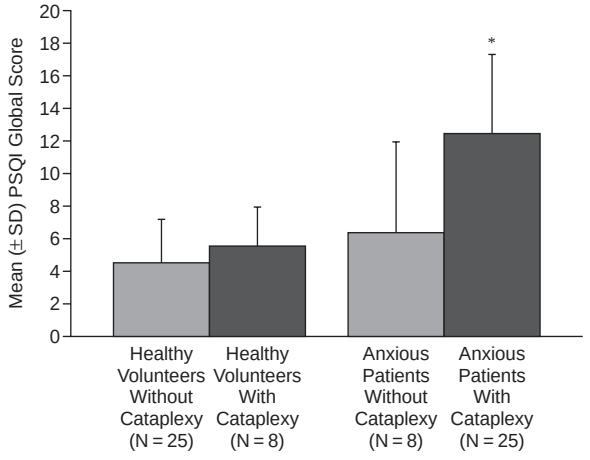
There were significant differences in PSQI Global scores among the 4 groups of subjects (i.e., anxious patients with and without cataplexy, and healthy volunteers with and without cataplexy (F = 17.9, df = 3,65; p = .000; Figure 2). The mean ± SD PSQI Global score among anxious patients endorsing cataplectic events (N = 25) was

Figure 1. Prevalence of Cataplexy[a]



[a]A 3- to 4-fold greater rate of classic and total (classic + nonspecific) cataplexy is reported by patients with anxiety disorders compared to healthy volunteers ($\chi^2 = 5.80$ [classic cataplexy], p = .016; $\chi^2 = 17.52$ [classic + nonspecific], p = .000).

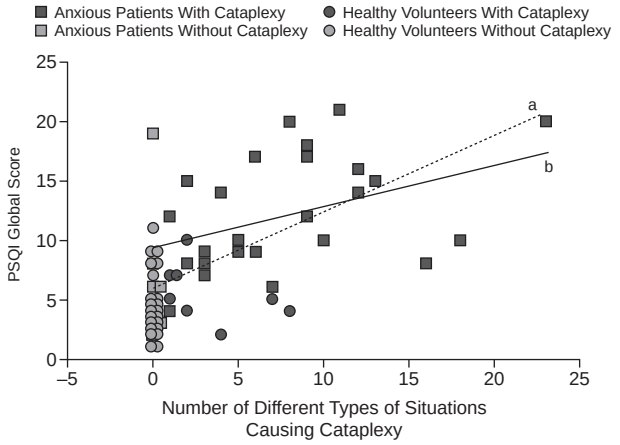
Figure 2. Mean ± SD Pittsburgh Sleep Quality Index Global Score of Participants With and Without Cataplexy[a,b]



[a]Anxious patients with cataplexy had significantly greater PSQI Global scores compared to anxious patients without cataplexy (p = .003).
[b]PSQI Global scores of anxious patients with cataplexy were also significantly higher than PSQI Global scores of healthy volunteers with (p = .000) or without cataplexy (p = .000).
*p < .01.
Abbreviation: PSQI = Pittsburgh Sleep Quality Index.

12.4 ± 4.8. Post hoc analyses indicated that anxiety patients with cataplexy had significantly greater mean ± SD SQI Global scores compared to anxious patients without cataplexy (N = 8; 6.4 ± 5.5; p = .003). Mean ± SD PSQI Global scores of anxious patients with cataplexy were also significantly higher than PSQI Global scores of healthy volunteers with (N = 8; 5.5 ± 2.4; p = .000) or without cataplexy (N = 25; 4.5 ± 2.7; p = .000; Figure 2). Of interest, there was no difference in PSQI Global scores

Figure 3. Pittsburgh Sleep Quality Index Global Scores and Cataplexy[a,b]



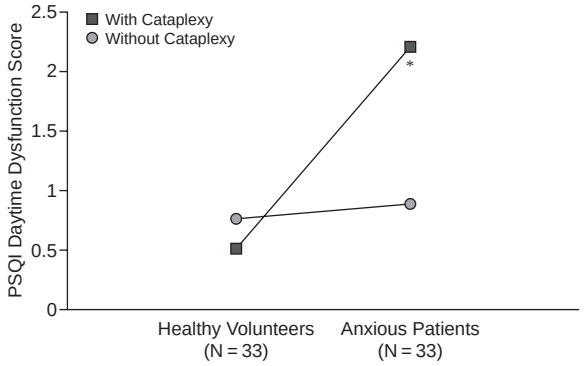
[a]Subjects with the greatest number of different types of situations that trigger cataplexy were significantly more likely to report more severe sleep disturbances (r = 0.62, N = 66, p = .000).
[b]The relationship between number of different types of situations triggering cataplexy and degree of sleep disturbance remains significant within all subjects reporting a positive lifetime history of cataplexy (r = 0.54, N = 33, p = .000).

between anxious patients without cataplexy compared to healthy volunteers, including those with a history of cataplexy. Thus, there was a significant interaction between diagnosis (anxiety versus healthy volunteers) and cataplexy (positive versus negative lifetime history) status (F = 4.82, df = 1,62; p = .032), suggesting that the coexistence of anxiety plus cataplexy is associated with the most severe global sleep problems.

In addition to the interactive effects of anxiety and history of cataplexy on global sleep quality, there also was a significant relationship between the number of different types of situations associated with cataplectic events and severity of subjective sleep disturbance on the PSQI (r = 0.62; p = .000; Figure 3). This association remained significant when subjects without cataplexy were excluded from the analysis (r = 0.54; p = .000; (Figure 3).

Since narcolepsy itself is especially characterized by daytime disturbances (i.e., excessive daytime sleepiness/sleep attacks), we assessed component 7 of the PSQI (range, 0–3), a subscale that specifically targets daytime dysfunction. On this measure, there was a significant interaction between diagnosis (anxiety patients versus healthy volunteers) and cataplectic status (F = 17.2, df = 1,62; p = .000). Similar to results found with the total PSQI score, post hoc analyses indicated that the anxiety disorder patients with cataplexy (N = 25; 2.2 ± 0.6) reported significantly greater mean ± SD daytime sleep disturbances, as reflected on the PSQI component 7 subscale measure, compared with non-cataplectic anxious

Figure 4. Interactive Effect of Diagnosis and Cataplexy on Pittsburgh Sleep Quality Index Daytime Dysfunction Score[a,b]



[a]There is a significant interaction between diagnosis (healthy volunteer versus anxiety disorder [D] and history of cataplexy [C]; [D x C; p < .0001]).
[b]Anxiety disorder patients with cataplexy report the most severe levels of sleep disturbances on both the total PSQI (not shown) and component 7, daytime dysfunction subscale score (shown here).
*p < .01.
Abbreviation: PSQI = Pittsburgh Sleep Quality Index.

patients (N = 8; 0.9 ± 0.4; p = .000) as well as compared to both the non-cataplectic (N = 25; 0.8 ± 0.7; p = .000) and the cataplexy positive (N = 8; 0.5 ± 0.8; p = .000) healthy volunteers (Figure 4). Of interest, the interactive effects of diagnosis (anxiety vs. healthy volunteer) × cataplexy history (positive vs. negative) on excessive daytime sleepiness remained significant even after applying analysis of covariance to take global PSQI effects into account (F = 11.3, df = 1,61; p = .001).

DISCUSSION

To our knowledge, this study represents the first analysis of anxiety disorder patients with respect to narcolepsy, with a special focus on cataplexy. Overall, a little more than three quarters of all anxious patients endorsed at least one cataplectic event (classic and/or nonspecific) in contrast to only approximately one quarter of healthy volunteers. Although the overall rate of all types of cataplexy found in our healthy controls may appear high, it is consistent with other studies showing prevalence rates up to 20% in the general population.[13] Moreover, Anic-Labat and colleagues[16] reported that 46% of their non-narcoleptic sample endorsed episodes of nonspecific muscle weakness associated with triggers such as athletic activity or while tense or stressed.

This type of nonspecific muscle weakness is differentiated from the type of muscle atonia thought to be most clearly reflective of classic, or clear-cut, cataplexy reported in patients with narcolepsy, i.e., muscle weakness triggered by laughing, anger, or hearing/telling a joke.[16] Thirty-three percent of our anxiety patients reported classic cataplexy, which was significantly higher than that reported in our healthy control group (9.1%) but within the range found in patients with well-defined narcolepsy.[13] That a small number of healthy controls reported episodes of classic cataplexy at some point in their lifetimes suggests that diagnosing narcolepsy on the basis of muscle atonia alone, even classic cataplexy, may lead to false positives. As suggested by Anic-Labat and colleagues,[16] classic cataplexy combined with other core symptoms of narcolepsy may confer greater diagnostic specificity. Within this context, it is noteworthy that, if we employ the combined items of classic cataplexy plus excessive daytime drowsiness (> 1 on component 7 of the PSQI) as an index of core narcolepsy-like symptoms, then 0% and 33% of healthy controls and anxiety patients, respectively, meet these criteria. While our measure of excessive daytime drowsiness is only an indirect measure of sleepiness, our more rigorous criteria still indicate that an unexpectedly high percentage of anxiety disorder patients report lifetime symptoms suggestive of subclinical narcolepsy.

Current DSM-IV diagnostic criteria for narcolepsy require that subjects report "irresistible attacks of refreshing sleep." Our clinical experience is that daytime fatigue and excessive daytime drowsiness are familiar complaints among patients with anxiety disorders, whereas multiple, daily sleep attacks are less commonly reported as core problems. Moreover, even after a full night of sleep or after daytime naps, patients with primary anxiety disorders do not, in our experience, typically report being refreshed.[3] In the present study, we did not obtain multiple sleep latency tests (MSLT) and did not obtain sleep-EEG evidence for REM-onset sleep. Thus, it is unclear what percentage of our patients would meet unequivocal criteria for all 4 of the classic features of narcolepsy (sleep attacks, cataplexy, sleep paralysis, and hallucinations). Nonetheless, cataplexy is considered by most investigators and clinicians to be the most definitive clinical and diagnostic feature of narcolepsy.

It is possible that the anxiety patients over-reported somatic symptoms, including muscle atonia (i.e., cataplexy) in response to questions on the Stanford Narcolepsy Scale. Theoretically, an increased prevalence of cataplexy in our anxiety disorder sample might be a partial by-product of nonspecific endorsement. While such an interpretation would be consistent with reports in the literature demonstrating increased somatic complaints in patients with PD,[30,31] PTSD,[32] GAD,[33] and SAD,[34] there is an equally robust literature indicating that anxiety patients report distinct, internally reliable, syndrome-specific physical complaints that align with different anxiety, mood, and sleep states.[35,36] Thus, our finding of an increased prevalence of cataplexy in anxiety patients is unlikely to be explained exclusively on the basis of over-reporting somatic symptoms or anxiety sensitivity.

Taken together, our preliminary findings suggest that classic cataplexy (or perhaps even narcolepsy) may be an underappreciated condition in patients with primary anxiety disorders. Of interest, cataplexy in well-diagnosed patients with narcolepsy has been associated with avoidance of social situations in which embarrassment or harm may occur, possibly leading to social isolation or withdrawal.[37,38] These observations suggest possible overlapping diatheses in some anxiety disorders with narcolepsy. To the extent that there is a pathophysiologic overlap, there are a number of neuroanatomic (e.g., locus ceruleus, dorsal raphe) and neurotransmitter-neuromodulatory systems (e.g., dopaminergic, noradrenergic, serotonergic, GABAergic-benzodiazepine-chloride) that could be plausibly implicated; perhaps most intriguing is the theoretical role of the hypocretin system.[39–41] Hypocretin neurons innervate brain regions involved in the biologic functions of wakefulness-promotion and arousal-vigilance-fear. Hypocretins appear to maintain wakefulness and inhibit transitions into REM-stage sleep, and they have been proposed as a model for maintaining vigilance during sleep.[42] Hypocretins also reduce unconditioned fear responses.[42] Thus, the loss of hypocretin neurons, which has been found in narcolepsy,[43] or deficient release of hypocretins or defects in hypocretinergic receptors, might result in parallel increases in anxiety and the intrusion of REM sleep, with its associated cataplexy, during conscious wakefulness.

Anxious patients, as a whole, were found to have increased Global PSQI scores compared to healthy volunteers. Interestingly, anxious patients with cataplexy had the most severe disturbances in sleep quality. Compared to anxious patients without cataplexy, they had approximately double the mean PSQI Global score, suggesting that the combination of anxiety plus cataplexy is likely to be associated with particularly disabling sleep problems. Within this context, one of the characteristic symptoms of narcolepsy is excessive daytime sleepiness, which is also a complaint of patients with primary anxiety disorders.[7,13] The role of excessive daytime sleepiness in anxious patients with cataplexy, however, has not been explored by the scientific community. An examination of the PSQI component 7 score, which measures the amount of daytime dysfunction,[26] may provide some insight into the impact of cataplexy on daytime dysfunction related to sleep problems in patients with anxiety disorders. Specifically, this component asks about trouble staying awake during daytime activities, such as driving, eating meals, and social activities, essentially an index of excessive daytime sleepiness. Anxiety patients with cataplexy exhibited the greatest amount of daytime sleepiness compared to anxious patients without cataplexy and healthy volunteers with or without cataplexy. This finding raises an interesting question regarding the relationship between daytime sleepiness, anxiety, and cataplexy. Perhaps excessive daytime sleepiness and anxiety are distinct by-products of a common disorder; in this case, narcolepsy. Or possibly, anxiety and narcolepsy may be separate entities with common features (i.e., EDS and cataplexy).

From a practical perspective, it is useful for clinicians to know that anxiety disorder patients with cataplexy may be at greater risk for having daytime dysfunctions such as having difficulty staying awake or lacking enthusiasm for carrying out daily work and social functions.

Our preliminary findings suggest that further research is warranted to investigate PSG and MSLT relationships between primary anxiety disorders and narcolepsy. This may be particularly relevant to patients with generalized anxiety and panic disorders. Until more definitive investigations are conducted in well-controlled studies of patients with anxiety disorders, narcolepsy, and healthy controls, our findings suggest that clinicians should evaluate patients with anxiety disorders for the presence of cataplexy and excessive daytime drowsiness and, if they are present, consider pursuing a more formal sleep evaluation and possibly HLA-typing to rule out narcolepsy.

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