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PERSPECTIVES

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WHAT'S IN THIS ISSUE

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Dreaming and Reality in Narcolepsy

A MAJORITY OF PATIENTS WITH NARCOLEPSY HAVE VIVID DREAMS, OFTEN MISTAKING THEM FOR REALITY

Hypnagogic and hypnopompic hallucinations (vivid dreams upon sleep onset and upon awakening, respectively) in narcolepsy occur during the transitional sleep-wake phase. These dreams are so vivid that they are often mistaken for reality. Dream delusions are defined by Wamsley et al. 2014 as memories of dreams that persist for days from sustained memories about the dream event.

In a collaborative study between academic centers in Boston and Leiden, the Netherlands (Wamsley et al. 2014), researchers studied dream delusions in 46 people with narcolepsy and cataplexy, comparing them with age-matched healthy controls (n = 41). The average age of the patients was 34.2 ± 10.9 years (SD), and 59 percent were female. A structured telephone interview was utilized to assess dream recall. Study participants were asked, "Have you ever had the experience of being unsure whether something was real or if it was from a dream?" The dream events were categorized as delusional episodes if, when fully awake, the subject was uncertain if a memory was dreamed or real or was convinced that a memory was real but later recognized that it was a dream. To define an episode as delusional, the dream had to persist into the waking state.

At the time of the interviews, patients used a variety of medications, including stimulants (72 percent of patients: modafinil, amphetamine, dextro-amphetamine and methylphenidate), antidepressants (15 percent of patients: tricyclic medications, selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, and serotonin agonist and reuptake inhibitors) and sodium oxybate (35 percent of patients). The researchers do not report any differences in medication usage between those with and without dream delusions (chi-square tests of independence: stimulants: p = 0.82, antidepressants: p = 0.64, sodium oxybate: p = 0.69).

Eighty-three percent of patients with narcolepsy reported that they had confused dreams with reality compared with only 15 percent of healthy controls. Thirteen patients rated their dreams as substantially more vivid and more emotional and recalled them more often than the age-matched healthy controls (results were statistically significant). Patients scored higher on the Boundary Questionnaire (BQ) than healthy controls (indicating that patients had "thinner" boundaries; the BQ assesses the personality construct of psychological boundaries. A "thin" boundary score (higher values) is associated with frequent and intense dreams. Among the narcolepsy subjects, neither BQ scores nor any other measure of dreaming differentiated those who did and did not experience delusions (all p > 0.1).

Patients with narcolepsy scored higher than healthy controls on the Prospective-Retrospective Memory Questionnaire (PRMQ) for both retrospective memory problems and prospective memory problems (difficulties in remembering to carry out intentions). Narcolepsy patients with or without dream delusions

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showed no difference in memory impairment as measured by the PRMQ. The scores on dream vividness did not discriminate between patients with and without delusions. The authors state there may be source memory deficits in narcolepsy.

The authors do not mention which treatment, if any, was offered or recommended to the patients.

In our own experience at the Narcolepsy Institute, two remarkable cases come to mind.

One female patient in her 40s with narcolepsy and cataplexy, traveling a great distance to participate in the program, missed several appointments. When asked why she had not kept her appointments, she reluctantly confided that whenever she traveled in the subway, a naked man would sit on her head and no one tried to rescue her. Deeper probing revealed that no one had actually assaulted her, but the fear of an assault had prevented her from keeping her follow-up visits. After a detailed discussion on hypnagogic hallucinations and the availability of medications for this secondary symptom, she agreed to see her physician for treatment. She subsequently kept her appointments—even though she never actually had treatment for her hallucinations. The explanation that her experience was a symptom of narcolepsy assuaged her fear and anxiety, which allowed her to better manage her condition without treatment of her hypnagogic hallucinations.

A second case was a gentleman in his 50s recently diagnosed with narcolepsy and cataplexy, though he reported having symptoms since he was about 40. One of his concerns included having a poor relationship with his family. He shared a recurring incident that was, in fact, a dream episode: During dinner or a family get-together, everyone would tease him about his sleepiness, as he tended to get very sleepy during meals. After a while, his family members pushed him (in his chair) against the wall. The incidents left him feeling weak and drained; he expressed anger with his family over their insensitive behavior.

83%

of patients with narcolepsy reported that they had confused dreams with reality, compared with only 15 percent of healthy controls

After his diagnosis and improved understanding of his condition, however, the patient understood that his painful experience was a hallucination. He received treatment for his daytime sleepiness and the secondary symptoms of narcolepsy and was able to repair his relationship with his family.

Timely diagnosis, treatment and explanations of the complex symptoms of narcolepsy can alleviate much misery and suffering, empowering patients to better manage their disorder. ■

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Wamsley E, Donjacour CE, Scammell TE, Lammers GJ, Stickgold R. Delusional confusion of dreaming and reality in narcolepsy. Sleep. 2014;37(2):419-422.

RESEARCH NOTE

Researchers in France note high frequency of pain in narcolepsy.

In a study in France, 32.8 percent, or one-third, of patients (*n* = 67) who had narcolepsy with cataplexy (NC) reported pain at least monthly versus 17.9 percent of the normal control group independent of the patients' narcolepsy medication. There was concurrence between patients and family members/friends in reporting pain; however, physicians underestimated patients' pain. Moreover, the study also found that "sleep quantity and depression were determinants for pain, and chronic pain had significant impact on sleep quantity, depression and QoL (quality of life) in NC." ■

Dauvilliers Y, et al. High pain frequency in narcolepsy with cataplexy. Sleep Med. 2011;12(6):572-577.

Ghrelin, Leptin and Sodium Oxybate in Narcolepsy

The hypocretin/orexin system has an important bearing on the sleep-wake cycle and is deficient in narcolepsy. Obesity is a common comorbidity in narcolepsy; however, the cause of this weight gain is not known, although altered states of eating behavior have been observed in narcolepsy.

It is possible that hypocretin deficiency may disturb eating behavior and energy levels due to the role of hormones such as ghrelin and leptin. Ghrelin is a peptide hormone produced in the stomach and the gastrointestinal tract. It regulates energy balance and growth hormones and sends appetite-stimulating signals to the brain. Ghrelin levels rise and fall during the body's 24-hour rhythm with an increase in the early part of the night and a decrease in the morning. The hypocretin system is sensitive to ghrelin, and together they influence eating behavior, including the rewarding or satisfying feeling of eating.

Leptin is also a peptide hormone produced by the subcutaneous white adipose tissue. It signals energy deficiency to the brain, and its blood levels show a circadian rhythm with a rise in the day reaching a peak in the middle of the night. Leptin and hypocretin may interact to influence physical activity and wakefulness. Thus, any loss of hypocretin may disturb the function of leptin as well as ghrelin, as noted above.

Donjacour et al. (2013) conducted a study to examine if the levels of ghrelin and leptin were deficient in hypocretin-deficient narcolepsy patients and to explain the reasons for increased body mass index (BMI) and altered eating behavior in these patients. They also examined the effects of sodium oxybate on these hormones. The subjects were eight men with narcolepsy and cataplexy who were medication free and hypocretin deficient. The control group included eight healthy men matched for age, BMI, body fat percentage and waist-to-hip ratio. The authors found no significant change in the levels of total ghrelin or leptin and "found no evidence to support a bidirectional, integrated feedback loop for total ghrelin or leptin levels and hypocretin."

Each night, three grams of sodium oxybate were administered orally to the subjects and controls at 11:00 pm hours and 3:00 am, a total of six grams per night for five consecutive nights. In both the study and control groups, sodium oxybate treatment showed a significant decrease in stages I/II NREM (non-rapid eye movement) and REM (rapid eye movement) sleep over 24 hours ($p = 0.011$ and $p = 0.009$, respectively). Nighttime awakenings were significantly reduced ($p = 0.002$), and the percentage of SWS (slow wave sleep) more than doubled (narcolepsy: 6.5 percent ± 5.5 percent vs.16.5 percent ± 8.4 percent, controls: 7.1 percent ± 5.5 percent vs. 18.5 percent ± 6.4 percent; $p = 0.001$ for administration effect). Additionally, during the day, time spent in stages I/II NREM and REM sleep ($p = 0.038$ and $p = 0.041$, respectively) was reduced, and there was a trend toward longer periods of wakefulness ($p = 0.098$). There was no effect of sodium oxybate on leptin level.

CONCLUSION

In this study, the results showed that increased BMI of narcolepsy patients may not be influenced by changes in total ghrelin or leptin levels. Although sodium oxybate may influence body weight, ghrelin or leptin secretion levels may not be influencing these changes. ■

REFERENCE

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Influence of Sleep Apnea on Cardiovascular Structure and Function

By Pramod K. Mohanty, MD, FACP, FACC, FAHA, Professor Emeritus of Medicine, Virginia Commonwealth University, Richmond, VA

Sleep apnea (SA) is a common disorder with an estimated prevalence rate of almost 20 percent among middle-aged adults in North America. Despite increasing negative impact, true incidence of SA may be underestimated, because people with this entity are often unaware of their problem. As a result, the adverse consequence of SA on cardiovascular diseases (CVD) is becoming more prevalent with major impact on morbidity and mortality in at-risk adults. Obstructive sleep apnea (OSA), the most common form of SA, is being recognized as an independent risk factor for a variety of cardiovascular disorders. Central sleep apnea (CSA), the less common and less well appreciated form of SA, occurs in less than 1 percent of the general population, although it occurs with increased frequency in patients with congestive heart failure (CHF), stroke and a common heart rhythm disorder known as atrial fibrillation (AF). In heart failure, CSA is manifested as Cheyne-Stokes respiration (CSR), a form of periodic breathing with a crescendo-decrescendo pattern of tidal volume accompanied by long periodic cycle duration. Combinations of both OSA and CSA have been observed in patients with advanced CHF and stroke.

SLEEP APNEA IN NARCOLEPSY

Narcolepsy may coexist with features of OSA and CSA. However, the overall incidence of SA in narcolepsy remains poorly defined. In one observational study, of the 133 patients with narcolepsy, 33 demonstrated features of OSA, with an estimated incidence of 25 percent. Thus, it is important to actively rule out diagnosis of narcolepsy in patients with OSA to minimize the delay in the diagnosis of narcolepsy. Moreover, treatment with CPAP will improve the levels of daytime sleepiness that are high in people who have both OSA and narcolepsy.

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PATHOPHYSIOLOGY OF CARDIOVASCULAR RESPONSE TO OSA

The adverse influence of OSA on cardiovascular function is based on complex overlapping mechanisms. Both cardiac vagal withdrawal and increased sympathetic nerve activity (SNA) result in tachycardia. In addition, sympathetic overactivation stimulates neuro-hormones known as the renin-angiotensin aldosterone system, which, in turn causes peripheral edema, vasoconstriction, increased afterload and cardiac rhythm disorders—both supra-ventricular and ventricular types. There is now compelling clinical evidence to suggest that patients with OSA and atrial rhythm disorder manifest adverse cardiac structural remodeling characterized by increased heart wall thickness, left atrial size and right ventricular volume/size, and that treatment with nasal continuous positive airway pressure (nCPAP) tends to reverse cardiac structural remodeling, decrease blood pressure (BP) and lower the risk of abnormal rhythm reoccurrence. These observations emphasize the importance of initiating appropriate and timely treatment of OSA to minimize and or prevent cardiac morbidity due to sleep apnea.

OBSTRUCTIVE SLEEP APNEA AND CARDIOVASCULAR CLINICAL ENTITIES

A variety of cardiovascular disturbances, including pulmonary and systemic arterial hypertension, left ventricular hypertrophy (LVH), right ventricular hypertrophy (RVH), coronary artery disease (CAD), heart failure and heart rhythm disorders, and increased risk of sudden cardiac death during sleep and stroke, have all been linked to OSA. It is important to reemphasize that OSA is an independent risk factor for arterial hypertension and also directly contributes to poor control of blood pressure despite maximal medical therapy with multiple antihypertensive medications. Often, treatment with CPAP renders refractory (resistant to therapy) hypertension to well-controlled blood pressure and thus has the potential to prevent cardiac structural changes, the key factor that influences progressive cardiovascular disease (CVD) and increased cardiovascular morbidity and mortality. In addition, OSA plays a role in the worsening of nocturnal angina, ischemic electrocardiographic (ECG) changes, triggering plaque rupture and precipitating acute heart attack in patients with CAD.

IMPORTANCE OF TREATMENT OF SLEEP APNEA

Effective treatment of OSA with CPAP has shown to be associated with lower BP and left atrial size, lower cardiac mass or wall thickness, and a lower risk of abnormal rhythm recurrence after catheter radio-frequency treatment (ablation). Nocturnal arrhythmias seen in OSA also decrease significantly with CPAP therapy. In another study, both systolic and diastolic left ventricular (LV) dysfunction in patients with OSA reversed as early as three months into CPAP therapy, with progressive improvement in cardiovascular structural remodeling over a period of one year. Taken together, the clinical, epidemiologic and cardiovascular imaging studies convincingly support the causal relationship between OSA and adverse changes in cardiovascular structure and function. The fact that the structural cardiac remodeling associated with OSA can be modified favorably by CPAP therapy further substantiates the powerful influence of sleep apnea on cardiac structure and function. What about atherosclerotic CAD? Arterial structural remodeling induced by OSA is considered early atherosclerotic disease characterized by increased carotid arterial wall thickness and increased arterial stiffness. Furthermore, CPAP treatment has shown to decrease both arterial wall thickness and arterial stiffness, supporting a causal link between OSA and atherosclerosis.

SUMMARY AND CONCLUSIONS

Currently, there is compelling evidence to suggest that OSA can initiate, sustain and contribute to refractory hypertension modifiable by effective CPAP therapy. Adverse cardiovascular structural remodeling has been well documented in patients with OSA. These structural changes can be favorably modified and or reversed by timely and effective therapy of OSA.

Early diagnosis of OSA in patients with CVD and prompt initiation of CPAP therapy has great potential to modify adverse cardiovascular symptoms, thereby preventing progression of CVD.

Accordingly, early diagnosis of OSA in patients with CVD and prompt initiation of CPAP therapy has great potential to modify adverse cardiovascular symptoms, thereby preventing progression of CVD. Based on the above observations, it is important to emphasize that the clinical provider community must make serious efforts to encourage public awareness of sleep apnea that in turn may accelerate early diagnosis and implementation of appropriate therapy to minimize its impact on OSA-related cardiovascular disease. ■

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Kasai T, et al. Sleep apnea and cardiovascular disease: a bidirectional relationship. Circulation. 2012;126:1495-1510.

Colish J, et al. Effects of continuous positive airway pressure on cardiac remodeling as assessed by cardiac biomarkers, echocardiography and cardiac MRI. Chest. 2012;141:674-681.

Comorbidity in Narcolepsy Is High

In a national, controlled, retrospective and prospective survey, scientists found several comorbidities. Data were gathered from the National Patient Registry (Danish) on 757 patients diagnosed with narcolepsy on whom information was available at least three years prior to and after diagnosis. Results were compared with data from randomly selected citizens (n = 3,013) matched for age, gender and socioeconomic status from the Danish Civil Registration System Statistics.

Prior to getting a diagnosis for narcolepsy, increased morbidity* included disorders of the endocrine, nutritional and metabolic systems, as well as of the nervous and musculoskeletal systems. After the diagnosis of narcolepsy, specific diagnoses were: **diabetes, obesity, sleep apnea, other sleep disorders, chronic obstructive pulmonary disease, lower back pain, arthrosis/arthritis,** neurological diseases, other diseases and rehabilitation. The results show that, compared with the normal population, persons with narcolepsy have higher comorbidities before diagnosis. These comorbidities increase after diagnosis.

These results indicate that thorough history-taking, comprehensive management and timely referral are paramount to successful management of narcolepsy. ■

odds ratio, 95 percent confidence interval

***diabetes** (2.4, 1.2-4.7, p < 0.01), **obesity** (13.4, 3.1-57.6, p < 0.001), **sleep apnea** (19.2, 7.7-48.3, p < 0.001), **other sleep disorders** (78.5, 11.8-523.3, p < 0.001), **chronic obstructive pulmonary disease** (2.8, 1.4-5.8, p < 0.01), **lower back pain** (2.5, 1.4-4.2, p < 0.001), **arthrosis/arthritis** (2.5, 1.3-4.8, p < 0.01), **observation of neurological diseases** (3.5, 1.9-6.5, p < 0.001), **observation of other diseases** (1.7, 1.2-2.5, p < 0.01) **and rehabilitation** (5.0, 1.5-16.5, p < 0.005)*

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Jennum P, et al. Comorbidity and mortality of narcolepsy: a controlled retro- and prospective national study. Sleep. 2013;36(6):835-840.

How We Forgive Others Can Affect Our Well-being

Most great religions of the world teach the virtue of forgiveness. One of the basic principles of Christianity is repentance for transgressions and forgiveness (Krause and Ellison 2003). As mentioned in the 2013 spring/summer issue of *Perspectives*, forgiveness involves letting go of resentment and negative thoughts, feelings and behaviors in the face of transgression toward others or oneself. The concept of forgiveness is multidimensional and includes forgiving others, asking for forgiveness from others, and asking God for forgiveness. Often not covered in certain cultures and in religious discourse is forgiving oneself. In both clinical practice and in personal life, we know that harboring guilt, remorse, resentment and bitterness can cause physical and mental anguish. What is the mechanism that allows relationships to withstand these challenges? Now, there is reliable research revealing the positive and negative consequences of different ways of forgiving and how forgiveness influences our relationships and overall well-being.

Forgiving unconditionally is based on the belief (e.g., the Christian model) that people should forgive as God does—unconditionally, thereby avoiding constant thinking about the offense. Some researchers question this process of unconditional forgiveness because the offense is not resolved and the offended party may harbor resentment and anger. A survey by Krause and Ellison (2003) examines the reasons why some require contrition and others do not.

In their nationwide probability survey, the researchers set out to examine forgiveness in older adults (Krause and Elison 2003). The aims of this study were threefold: To compare the effects of forgiving others with receiving forgiveness from God; to assess if forgiving others unconditionally is more beneficial to psychological well-being than requiring transgressors to perform acts of contrition; and to understand why some people require transgressors to perform acts of contrition. The sample consisted of 748 older whites and 752 older African Americans. The overall response rate was 62 percent. The average age of these individuals was 74.5 years (SD = 6.4 years). Forty-two percent were men, 50 percent were married, and all had completed an average of 11.5 years of schooling (SD = 3.4 years).

Forgiveness of others was measured through the following items: feeling resentful toward others, holding a grudge, and the ability to forgive others for things they had done. Acts of contrition were measured by the following items: offering an apology, promising not to do the same thing again, and repaying or compensating the respondent in some way.

Depressive symptoms were measured by seven indicators taken from the Center

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for Epidemiologic Studies Depression Scale (CES-D). These measures reflected two underlying dimensions. The first, depressed affect, assessed the cognitive-affective aspects of depressive symptoms, including feeling sad, blue and depressed. The second dimension measured somatic symptoms (related to body), such as difficulty sleeping, poor appetite and low energy.

Life satisfaction is one's perception of having acquired desired goals. Is there a gap between one's aspirations and one's achievement? Another dependent variable measured was death anxiety. Religious control measures included frequency of church attendance and frequency of private prayer.

Acts of contrition may include awareness of the error, admission of the error, resolve not to repeat error, and making restitution. The authors point to benefits and disadvantages of acts of contrition. Acknowledging an error could pave the road to effective communication, as often the transgressor may not even be aware of the error or may be in denial, commonly observed in daily life. Another benefit is the assurance that the error will not be committed again. The authors argue that negative effects on psychological well-being may ensue. "Expecting the transgressor to make amends may set in motion a vicious cycle of recrimination and revenge because transgressors may not perform the anticipated acts of contrition, or they may not perform them in a way that is satisfactory to the victim."

RESULTS

Forgiving others tends to enhance psychological well-being, and these salubrious effects are greater than those associated with forgiveness by God.

Older people who expect transgressors to perform acts of contrition have higher depressed affect scores (beta = 0.130; b = 0.150; p < 0.001) and also have more somatic symptoms of depression, but the relationship is not as strong (beta = 0.071; b = 0.110; $p < 0.01$); they have lower levels of life satisfaction (beta = –0.160; b = –0.160; $p < 0.001$) and are more anxious about dying than respondents who do not require acts of contrition to forgive (beta = 0.276; b = 0.281; $p < 0.001$). Moreover, respondents who feel they are forgiven by God are about two and a half times more likely to feel that offenders should be forgiven unconditionally than those who feel they are not forgiven by God (odds ratio = 2.582). Additionally, the relationship between forgiveness by God and the odds of forgiving unconditionally (beta = 0.715; b = 0.949; $p < 0.001$) was stronger than the effects of any other variable, including church attendance (beta = 0.194; b = 0.073; $p < 0.05$) and the frequency of private prayer (beta = 0.065; b = 0.036; n.s.). Altogether, some methods of forgiveness may have deleterious effects on one's well-being.

How does forgiveness by God affect the way people forgive others? What are the practical implications of these findings? The authors suggest that the psychosocial atmosphere in places of worship and empathy and level of expressiveness among members may contribute to members' receptivity to practicing forgiveness. Family values are likely to play an important role in contributing to individuals' willingness to forgive. Therapeutic modules designed to provide services at the individual level or in support groups would be beneficial in promoting forgiveness. The gift of forgiveness can enhance positive channels of communication and improve relationships and thus promote peace of mind and loving kindness. "A wise man will make haste to forgive, because he knows the true value of time, and will not suffer it to pass away in unnecessary pain" (Samuel Johnson, *The Rambler*, December 24, 1751). ■

REFERENCE

Krause N, Ellison CG. Forgiveness by God, forgiveness of others, and psychological well-being in late life. J Sci Study Relig. 2003;1:42(1):77–94.

Mazindol Is Effective in Narcolepsy

Mazindol is a non-amphetamine stimulant and a tricyclic antidepressant that is prescribed for narcolepsy and, because of its anorectic effect, sometimes recommended for obesity. A study in Paris, France, evaluated the efficacy and side reactions of Mazindol in 139 patients. Ninety-four people had narcolepsy and 66 percent had cataplexy, 37 had idiopathic and eight had symptomatic hypersomnia refractory to modafinil, methylphenidate and sodium oxybate. The most common side effects were dry mouth (13 percent); palpitations (10 percent), including one with ventricular hyperexcitability, headaches (6 percent), nervousness (6 percent) and anorexia (6 percent). Treatment with Mazindol for an average of 30 months (dosage: 3.4 ±1.3 mg/day, 1-6 mg) showed a decrease in the Epworth Sleepiness Scale (ESS) score from 17.7 ± 3.5 to 12.8 ± 5.1, with an average decrease of -4.6 ± 4.7 (p <0.0001). The frequency of cataplexy attacks were reduced from 4.6 ± 3.1 to 2 ± 2.8 episodes per week; cataplexy was absent after treatment in 14.5 percent of the patients, it was reduced in 27.5 percent, and 29 percent showed no change. ■

REFERENCE

Nittur N et al. Mazindol in narcolepsy and idiopathic and symptomatic hypersomnia refractory to stimulants: a long-term chart review. Sleep Med. 2013;14(1):30-36.

Patients' Corner

My name is Shirley Wilson. I am 55 years old, and I have had narcolepsy all of my life. It has interfered with my education, job, and social and personal life and self-esteem issues.

When I was 11, I noticed I had problems staying awake. As I got older, especially in my teen years, I felt confused, defeated and isolated.

One day I lost my job. I remember my sister Sheila saying that I have a high school diploma and a college background, but I can't keep a job. She picked up the phone and called Montefiore, getting in touch with Dr. Meeta Goswami.

When I met Dr. Goswami, she told me I had the symptoms of narcolepsy and referred me for a sleep test. My narcolepsy was confirmed. She also told me about Montefiore's support group meetings for narcolepsy patients. When I attended the meetings, I was amazed to find that there were people who had experienced problems similar to mine. It changed my life. I love the narcolepsy support groups at Montefiore. Now I know I am not alone. I have received services from the Narcolepsy Institute since 1991, when I was diagnosed.

Dr. Goswami has worked diligently for many years to help people like me with narcolepsy. She has helped me to better manage narcolepsy and love myself as I am.

My name is Rosemarie Laurencin and I am 75 years old. I have narcolepsy. I learned all about the symptoms of narcolepsy, especially cataplexy, from an article I obtained from the Narcolepsy Institute called "A Counseling Service for Narcolepsy: A Sociomedical Model."

Although I was diagnosed with narcolepsy in my early 40s, I had symptoms from the time I was an infant. My mother recalled how, when I was playing with my twin sister in the playpen, I would fall asleep quite frequently.

Attending the narcolepsy support group meetings has been very beneficial to me. The group discussions allow all members to ask questions and express their opinions. We learn about nutrition and exercise, and we share our experiences about living with narcolepsy. Thank you, Narcolepsy Institute, for being there for us. ■

DID YOU KNOW?

One study showed that short rapid eye movement latency (REML) (≤15 minutes)—that is the time it takes to go into REM sleep, during nocturnal polysomnogram had a high predictive value, was specific for narcolepsy, and may be a useful diagnostic tool without a multiple sleep latency test (MSLT). Absence of REML, on the other hand, calls for an MSLT. ■

REFERENCE

Andlauer O, et al. Nocturnal rapid eye movement sleep latency for identifying patients with narcolepsy/hypocretin deficiency. JAMA Neurol. 2013;70(7): 891-902.

Body mass index (BMI) in subjects with narcolepsy and cataplexy (N+C) was significantly higher (BMI > 30) (39.0 percent) compared with those who had narcolepsy without cataplexy (N-C) (13.8 percent). N-C subjects did not have a higher BMI nor a greater incidence of obesity compared with the general population. ■

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Adderall® (amphetamine, dextroamphetamine) is prescribed for attention deficit hyperactivity disorder (ADHD) and, in some cases, in narcolepsy. Overdose may lead to any of the following symptoms: hyperactivity, hyperthermia (increased body temperature), tachycardia (increased heartbeat), tachypnea (increased rate of breathing), mydriasis (increased dilatation of the pupil of the eye), tremors and seizures. Contact your physician if you experience any of these symptoms. ■

REFERENCE

Fitzgerald KT, Bronstein AC. Adderall® (amphetamine-dextroamphetamine) toxicity. Top Companion Anim Med. 2013;28(1):2-7.

Functional neuroimaging modalities indicate structural and functional abnormalities compatible with a deficit in the hypocretinergic system. These tests also reveal that other brain structures are involved such as the amygdala, nucleus accumbens, midbrain, thalamus, hippocampus and fronto-temporal cortical areas. ■

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To speak gratitude is courteous and pleasant, to enact gratitude is generous and noble, but to live gratitude is to touch heaven. — JOHANNES A. GAERTNER

Wherefore I say unto thee, her sins which are many are forgiven, for she loved much: but to whom little is forgiven, the same loveth little. — LUKE 7:47

Christ said, "Father, forgive them, they know not what they do." — LUKE 23:34

This time is a very good time if we but know what to do with it. — RALPH WALDO EMERSON

PERSPECTIVES FROM THE NARCOLEPSY INSTITUTE

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ABOUT THE INSTITUTE

Narcolepsy is a chronic sleep disorder of neurological origin. Its main symptoms are (1) excessive daytime drowsiness with a tendency to sleep at inappropriate times, (2) cataplexy (sudden loss of strength in the muscles generally provoked by strong emotions or stress), (3) sleep paralysis and (4) hypnagogic hallucinations (extremely vivid dreams or images). Disturbed nighttime sleep, problems with memory, and fatigue are common complaints of people with narcolepsy.

The Narcolepsy Institute, initiated in 1985, is committed to providing comprehensive care to people with narcolepsy by integrating the medical, social, psychological and spiritual dimensions of health in a spirit of kindness and respect toward all, irrespective of race, creed, ethnicity or social class; that the recipients of care may realize their potential and live productively in joy, peace, harmony and dignity and thus improve the quality of their lives.

Activities of the Narcolepsy Institute include: screening, counseling, conducting professionally led support groups, advocacy, and public and professional education. Counseling entails comprehensive management of the symptoms of narcolepsy and strategically applying behavioral and non-pharmacological approaches to ally the devastating impact of narcolepsy on the personal, social, educational and occupational lives of affected individuals and their families. Our patients benefit most by a family-centered and person-centered approach to improving the quality of their lives.

The contents of this publication are not intended to provide advice for individual problems nor to replace medical advice. Readers are urged to consult with their professionals before initiating self-therapy. We welcome comments and suggestions about the contents of the newsletter.

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