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Inside

In This Issue

This issue of *The Residents' Journal* includes a special guest section on addiction. Jonathan Avery, M.D., provides valuable information on the pharmacology, safety, dosing, and efficacy of buprenorphine in the treatment of opioid dependence, and Maria Andrea Baez, M.D., discusses the relationship between addiction and overeating.

Editorial

The Role of Psychiatry in Events of Disaster

Sarah M. Fayad, M.D.
Editor-in-Chief

This year marks the 10-year anniversary of 9/11, an infamous date that left a significant impact on the United States. Within psychiatry, it is important to reflect on this tragic event and the impact it had on the many people who were directly and indirectly affected. One study reported that the prevalence of probable posttraumatic stress disorder (PTSD) was 7.4% among New York City firefighters 9 years following the World Trade Center attack (1). Another study demonstrated that the rates of PTSD were as high as 35% among people who were directly exposed to the World Trade Center attack or who had a close associate who was directly exposed (2). The American Psychiatric Association has developed several resources for disaster psychiatry that are readily available online to prepare people for events such as terrorist attacks or natural disasters (3). It is my hope that those in training will use this time to reflect upon and think of how they could best prepare themselves to provide the highest quality care to patients who find themselves experiencing symptoms as a result of such a disaster. As mental health professionals, it is imperative to begin thinking of how we would respond in these types of situations. Those residents who have a particular interest in disaster psychiatry should consider writing a manuscript on this topic. We will also be featuring a section theme on PTSD and traumatic brain injuries in the January 2012 issue. As training psychiatrists, we are the future of psychiatry, and there is no better time to prepare for providing care to disaster victims than now.

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A Resident's Guide to Buprenorphine

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Opioid dependence is a prevalent and difficult-to-treat medical condition. It is characterized by a strong desire to use opioids, difficulty in controlling drug-taking behavior, the presence of withdrawal and tolerance symptoms, the neglect of other pleasures or interests in order to use opioids, and persistent use despite clear evidence of harmful consequences (1, 2). Between one and four million people in the United States are addicted to heroin and other opiates, and the estimated cost of untreated opiate addiction exceeds $20 billion per year (1, 3).

Pharmacotherapy often plays an important role in the treatment of opioid dependence. The standard of care for years has been methadone (an opioid agonist), but other medications, such as naltrexone (an opioid antagonist) and clonodine (an a_2 adrenergic agonist), have also been utilized (4). In 2002, buprenorphine, a partial opioid agonist, was approved in the United States as a schedule-III-controlled substance for treating individuals with opioid dependence (1). Due to the fact that buprenorphine, unlike methadone, can be administered in a clinician's office and because of its proven efficacy and safety, buprenorphine is becoming increasingly popular among clinicians (1). Organizations such as the American Academy of Addiction Psychiatry are now encouraging residents to learn more about this medication and are even offering free training courses.

Pharmacology

Buprenorphine is a partial opioid agonist that binds tightly to and dissociates slowly from opioid receptors; yet it has low intrinsic activity at the receptor site (4). As a consequence of these properties, it has a ceiling effect (and a low risk of overdose) and does not cause significant sedation or euphoria (4). However, it may not be as effective as higher doses of methadone, and it displaces other opioids from endorphin receptors, which can trigger severe opioid withdrawal (4).

Buprenorphine undergoes extensive first-pass metabolism, which results in low oral bioavailability. Sublingual administration has been shown to have much higher bioavailability and is the preferred oral delivery route (5, 6). Buprenorphine is mainly metabolized by cytochrome P450 3A4 (6). There is considerable variation in the reported values of the terminal elimination half-life of this medication, with values ranging from 3 hours to 44 hours (1, 6).

Buprenorphine can be administered alone or in combination with the opioid antagonist naloxone (4). Naloxone has little oral bioavailablility but can cause opioid withdrawal if used intravenously, which discourages individuals with opioid dependence from abusing buprenorphine intravenously (5).

Safety

Common side effects of buprenorphine include sedation, nausea, constipation, and headache (4, 5). More serious side effects include respiratory depression, which can result in death, although there is the ceiling effect that is not present with methadone (4). In pregnant women, some studies have demonstrated that buprenorphine is associated with less neonatal withdrawal than methadone (4). The safety of buprenorphine for mothers who breastfeed is not as certain, however, and this medication has been detected in breast milk in levels similar to maternal serum levels (4). Buprenorphine is relatively contraindicated in patients who are using drugs or receiving medications that cause respiratory depression (ethanol, benzodiazepines) and is absolutely contraindicated in patients who have had hypersensitivity reactions in the past (4, 5).

Dosing

The first buprenorphine dose must be administered when the patient's last opioid dose has begun to wear off. The patient's last dose of a short-acting opioid, such as heroin or oxycodone, should be 8–24 hours before initiation of buprenorphine. For longer-acting opioids, such as methadone, the patient's last dose should have been 24–36 hours prior (7). The clinician should wait to begin buprenorphine administration until the patient is in moderate opioid withdrawal, which can be assessed clinically or by scales such as the Clinical Opiate Withdrawal Scale (8). Failure to do so will result in severe opioid withdrawal (4).

The patient is usually given 2–4 mg initially, and approximately every 2–4 hours after this on the first day, the patient should be given additional buprenorphine until he or she no longer displays symptoms of withdrawal (4, 7). Typical first-day doses are 8 mg–16 mg, although lower dosing amounts have been reported, especially for higher-risk patients (i.e. older patients and patients with lower tolerance or using benzodiazepines) (4). Stabilization doses for buprenorphine maintenance can range from 8 mg–32 mg daily, and some patients even respond to less-than-daily dosing schedules (4, 7). Subsequent dosing after the first day of detoxification can vary widely based on patient need and the protocol that is being used (4, 7).

Efficacy

Buprenorphine has been repeatedly shown to decrease opioid use with long-term therapy (9, 10). Compared with placebo, patients receiving buprenorhpine have higher rates of negative urine screens (9, 10). In one small study, 1-year retention in buprenorphine treatment for opiate dependence was 75% in the buprenorhpine group compared with 0% in the placebo group (10). Urine screens were approximately 75% negative for illicit opiates and other substances of abuse in the patients who remained in treatment (10). Among individuals with

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heroin addiciton, buprenorphine use has also been shown to improve social functioning, psychiatric status, and quality of life (11).

However, methadone, at least for now, remains the gold standard of maintenance treatment (4). Several studies (including a meta-analysis) have concluded that while buprenorphine is effective for maintenance treatment, it is not more effective than methadone (9). Still, buprenorphine is safer than methadone; not only does buprenorphine have a lower abuse potential and lower overdose risk, it does not prolong the QT interval and does not cause the same rate of erectile dysfunction (4). Further, patients receiving treatment with buprenorphine score better on tests of cognition and psychomotor abilities compared with patients receiving methadone treatment (4, 12).

Buprenorphine has been shown to be more effective than nonopioid treatments for detoxification in terms of decreasing opioid withdrawal symptoms and keeping patients in treatment (4). However, buprenorphine detoxification is not as effective as buprenorphine maintenance treatment (13).

Resident Training

While most residents (and psychiatrists) never have the opportunity to work in a methadone clinic or prescribe methadone, the relative safety of buprenorphine does allow residents to prescribe buprenorphine to their patients in the office. To be able to prescribe buprenorphine, one must meet the requirements of the Drug Addiction Treatment Act, which includes completion of an 8-hour training course and registration with the Drug Enforcement Administration (3). There are numerous workshops and online training modules that can train and certify a resident physician. Potential barriers to prescribing buprenorphine as a resident include a lack of desire to undergo further training in addition to an already busy schedule and supervising physicians who are not familiar with or not certified to prescribe it.

Resources

American Academy of Addiction Psychiatry (http://www2.aaap.org/)

Substance Abuse and Mental Health Services Administration (http://buprenorphine.samhsa.gov/pls/bwns/training)

National Institute on Drug Abuse (http://www.nida.nih.gov/blending/buptreatment.html)

Conclusions

Buprenorphine is an effective treatment for opioid dependence. While many residents will not choose to become certified to prescribe this medication, they should at least become familiar with its pharmacology and clinical uses, since more and more patients will begin to use it in the community.

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Obesity and Addiction

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Obesity is a complex disorder that often results from a combination of overeating, metabolism, genetics, hormones, environment, and lifestyle. Throughout my career, I have been struck by some patients who claim that they are unable to stop eating and can even experience both tolerance and withdrawal symptoms. They describe their problems with food in a manner similar to that in which an individual with substance dependence might describe his or her substance use, and I have wondered whether these patients may have a food addiction. The interest in the role of addiction in obesity has been increasing in the last several years, although there is no consensus on the topic, and controversy floods discussion panels and most of the existing literature.

The relationship between addiction, overeating (either chronically or in binges), and obesity has been approached from different perspectives. Some have argued that foods with high sugar, fat, or salt content are more likely to be addictive (1). For example, researchers have developed a model of sugar addiction in which rats display "addict-like" behavior, demonstrating withdrawal and cravings, but there is a lack of literature consistently replicating these findings in human subjects (2). Other researchers have proposed an evolutionary theory to explain why some foods can be rewarding. They argue that the intake of high-caloric food may be an adaptive survival strategy, since these foods provide more energy (1). This process of seeking high-caloric food has been shown to be mediated by the mesolimbic reward pathway, which also reinforces other addictive behaviors (1). This is further supported by findings in functional magnetic resonance imaging studies of obese and normal-weight women, which show greater activation of limbic structures in the brains of obese women when they are exposed to pictures of high-caloric foods (3). It has also been shown that individuals who are susceptible to overeating, obesity, and substance use have down-regulation of the dopamine D_2 receptors (2, 3). More recently, other molecules have been implicated in the shared mechanism of addiction and obesity, such as the opiate and endocannabinoid systems (1, 3, 4). Given the shared neurobiological substrates, it is not surprising to find that symptoms experienced by obese people are similar to those described in DSM-IV-TR for substance abuse and dependence (1, 3, 4).

However, many experts argue that associating obesity with addiction could be a mistake. For example, there are documented differences between the relapse patterns and treatment responses of those recovering from substance use compared with those recovering from obesity (2). Additionally, there are peripheral signaling systems, as well as gut- and fat-derived hormones, involved in food consumption that are not necessarily present in other models of addiction (4). Further, there is the risk that people will argue that obesity in and of itself should be regarded as an addiction, which not only ignores the multifactorial nature of obesity but could be quite stigmatizing to the obese population (3). As in all behaviors that are labeled "addictions," there is some concern that highlighting the role of addiction in obesity will negate the role of freewill and personal choice and consequently deemphasize the need for exercise and dieting (1).

While not all obese patients suffer from a food addiction, it is clear that obesity and addiction often share common features. More efforts should be made to identify and study the subset of patients who suffer from both obesity and food addiction, since it could result in better pharmacotherapy and behavioral interventions, expanded research, and implementation of more effective prevention programs. There are already several medications derived from the addiction models of obesity that are underway, but more research is needed in order to understand one of the world's most complex and difficult-to-treat conditions (4).

Dr. Baez is a first-year resident in the Department of Psychiatry at Montefiore Medical Center, Bronx, New York.

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This month, The Residents' Journal *is pleased to debut a new feature, titled "Point-Counterpoint." This feature allows residents to develop and present scholarly ideas relative to a topic that has evidence for two opposing views. It is designed to help residents think critically about these issues and formulate their own thoughts. We hope to offer this feature on a quarterly basis. Residents who would be interested in participating should contact either Dr. Fayad (fayad@ufl.edu) or Dr. Seawell (mseawell@med.wayne.edu). Positions put forth in these articles do not express the viewpoints of* The Residents' Journal *editorial staff.*

Should Ecstasy Be Used Therapeutically?

Article #1

Considering the Use of Ecstasy in the Treatment of Psychiatric Disorders

by Jay Augsburger, M.D.
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[3,4]-Methylenedioxymethamphetamine (MDMA [or ecstasy]) has a complicated history as a therapeutic agent. First synthesized nearly 100 years ago as an intermediate compound to a hemostatic agent, its psychotropic effects were first documented many years later (1). It became popular as an adjunctive agent to psychotherapy in the 1970s and 1980s, although nearly all data to support its use in that era were anecdotal. MDMA was labeled a schedule I drug in 1985, which largely halted its use in therapeutic and research contexts. Recently, however, several studies have suggested that when used in conjunction with psychotherapy, it may still have some value in treating psychiatric disorders.

Why might MDMA enhance the efficacy of psychotherapy? For many years, explanations were primarily psychodynamic in nature, but Johansen (2) summarized three proposed neurobiological mechanisms by which this might occur:

> 1) MDMA increases oxytocin levels, which may strengthen the therapeutic alliance; 2) MDMA increases ventromedial prefrontal activity and decreases amygdala activity, which may improve emotional regulation and decrease avoidance; and 3) MDMA increases norepinephrine release and circulating cortisol levels, which may facilitate emotional engagement and enhance extinction of learned fear associations." (2)

The most convincing study of MDMA enhancing the efficacy of psychotherapy was conducted by Mithoefer et al. in 2010 (3). In this study, 20 patients with treatment-refractory posttraumatic stress disorder (PTSD) were given either MDMA (125 mg [N=12]) or placebo (N=8) during two 8-hour therapy sessions and then assessed 4 days after each session as well as 2 months after completing the second therapy session. The most common cause of PTSD was sexual trauma or abuse, which was reported by 80% of participants. At the final assessment, the response rate on the primary outcome measure (the Clinician-Administered PTSD Scale) was 83% in the active treatment group, compared with 25% in the placebo group. The study has several important limitations. First, a drug with such a clear psychotropic effect is difficult to blind. Second,

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Article #2

Ecstasy: Illicit Substance or Therapeutic Treatment?

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[3,4]-Methylenedioxymethlyamphetamine (MDMA), or "ecstasy," is a unique drug with a long history of use by psychiatrists, patients, and the general public. Aside from those individuals who use the drug for recreational purposes and claim that it is "life-affirming" and helps them to find their "true self," researchers and clinicians have attempted to show that it can improve interactions and insights in therapy sessions as well as alleviate symptoms of major depressive disorder, posttraumatic stress disorder (PTSD), anxiety disorders, and even schizophrenia (1–3). However, despite the purported good of this drug, the clinical data have not been strong enough to support its use by clinicians and patients, and more and more studies are revealing its negative effects. In fact, the U.S. Drug Enforcement Administration has placed MDMA in schedule 1, citing the side effects and the lack of positive studies on the drug with an "appropriate methodological design" (2).

While acute MDMA use can result in an increase in positive emotional states by increasing serotonin, oxytocin, and prolactin and by modulating other neurotransmitters, it can also increase negative emotional states, which can cause psychiatric distress in individuals with and without mental illness (1). For this reason, most of the positive outcomes noted in therapy were under highly controlled and safe settings, which can be difficult to replicate, and even experienced therapists have had adverse outcomes (1, 4). Other acute side effects include disinhibition, confusion, anxiety, perceptual disturbances, teeth clenching, elevated heart rate, seizures, and dehydration (2, 5). Additionally, MDMA can be lethal in overdose (2).

Subacutely, numerous studies have demonstrated that there is a period of "neurochemical recovery " after MDMA is used, which can result in depression, anxiety, irritability, fatigue, insomnia, and gastrointestinal symptoms, all of which can be countertherapeutic (1, 5). This hangover effect is called the "mid-week blues" or "blue Wednesday" by abusers of the drug and has been shown to occur in both recreational use and in controlled laboratory settings (2). Most short-term positive studies of MDMA do not observe patients after they discontinue the drug and therefore ignore this hangover period (1, 2).

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the therapy sessions were 8 hours in length, which is far from a typical standard practice. And finally, the study was funded by the Multidisciplinary Association for Psychedelic Studies, which could introduce bias.

Additionally, an open-label 2008 study demonstrated some benefit of the drug as an adjunct to psychotherapy in six women with PTSD secondary to sexual assault (4). A follow-up study of MDMA-assisted psychotherapy sessions, conducted from 1988 to 1993, for patients with a variety of diagnoses in Switzerland revealed that 91% of responders felt that they had benefitted from treatment with MDMA, and patients experienced relatively few side effects (5). Ongoing research in this area is underway.

In summary, MDMA has demonstrated that it can enhance the efficacy of psychotherapy for PTSD. The current data are not sufficient to conclude that MDMA is both safe and efficacious for this indication, but with further research, it might prove to be a helpful option in caring for patients with difficult-to-treat disorders such as PTSD. MDMA has risks that are well-known and not to be minimized. However, this is true for many psychiatric agents, and we should fairly consider the potential benefits of this drug along with its risks.

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Further, individuals with a psychiatric illness may be especially susceptible to these subacute effects (1).

The chronic side effects of MDMA are less clear. Some have argued that chronic use may be neurotoxic and can result in problems with executive decision making and memory (2, 6, 7). One study examined 466 regular MDMA users and showed that while 31% felt more open toward people, 59% developed tolerance, 38% suffered from an impaired ability to concentrate, and 37% suffered from depression (7). Among recreational users, there are high rates of depression, anxiety, and other substance use (1).

Given the aforementioned short- and long-term side effects of MDMA use, there would have to be significant clinical data detailing that it can be effective in order for it to be used therapeutically. However, the data are underwhelming at this point, with psychotherapists perhaps providing the best (albeit small and inconsistent) evidence for its use in MDMA-assisted therapy (1, 2). The literature on its use for PTSD is also increasing, but it too remains inconsistent (2, 3). Further, there have been many negative studies of MDMA, many of which demonstrate more of the aforementioned adverse effects than positive effects (1, 2, 7). Large placebo-controlled studies of MDMA with positive results are nonexistent, and proponents of this drug cling largely to results from small and short-term studies (1, 2).

The psychiatric community is always looking for newer and more effective ways to treat patients. While MDMA has at times appeared as if it could significantly help certain individuals, the many adverse effects of this drug and the lack of positive research data indicate that it is not a viable option for clinicians and patients at this point. Future work to modify MDMA or its dosing and administration, as well as research to indicate which type of patients may benefit from it, is needed before it becomes a part of psychiatric practice.

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Physician Suicide: A Brief Review

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Suicide rates among physicians are higher than those for the general population. The overall suicide rate for physicians in the United States is nearly double the rate for Caucasian American men (1). Per year, the number of physicians who kill themselves is approximately equivalent to two average-sized graduating medical school classes (2). It has been shown that when all causes of mortality are compared between physicians and the general population, suicide is the only cause of death in which the risk for doctors is higher than that for the general population (3). When further explored from a gender perspective, male physicians have a rate of suicide that is 70% higher than the rate for men in the general population. Perhaps even more shocking, female physicians have a particularly highly elevated rate of suicide (4), shown to be between 250%–400% higher than the rates found among their nonphysician counterparts (5).

Two of the greatest risk factors for physician suicide are a history of mental illness (6) and substance use disorders (6–8). Similar to risk factors among nonphysicians, diagnosis of an axis I mood disorder, such as depression, is a key risk factor for physician suicide (8, 9). One study found that the rate of depression among physicians increased dramatically during the intern year of training (10). The study revealed that prior to internship, only 3.9% of subjects met criteria for depression. However, this number sharply increased at quarterly intervals, with an average of 25.3% of subjects endorsing depression. Surprisingly, even when physicians are admitted to inpatient psychiatric facilities for treatment of psychiatric disorders, they do not improve but instead continue to have an almost fourfold greater risk for suicide compared with nonphysicians admitted under similar circumstances (11).

With regard to substance use disorders, estimates show that 40% of physician suicides are associated with alcohol use and 20% with drug abuse (12). Although studies are conflicting as to whether or not there is a link between increased suicide risk and the choice of medical specialty, drug abuse has been found to be particularly prevalent among anesthesiologists, emergency physicians, and psychiatrists (13, 14). Interestingly, anesthesiologists face unintentional second-hand environmental exposure to addictive drugs during procedures, which put them at increased risk for drug addiction (13). Of particular note, intentional drug ingestion has been shown to be one of the most common methods of suicide by medical doctors (1). The opportunity to self-medicate and the knowledge of lethal medication doses may play a role in the increased rates of suicide completions by overdose among physicians (2).

Other characteristics that may contribute to physician suicide include occupational demands, lack of personal support, and competing life responsibilities (15). In particular, physicians have trouble with limit setting (2). Long hours are to be expected, and there is often a strong sense of obligation to their profession and patients. Delayed gratification is common among medical doctors, and they often put off enjoyment of what life has to offer in exchange for professional development. This combination impinges on having a balanced lifestyle, possibly putting physicians at increased risk.

Few physicians seek help. Perhaps a reflection of stoicism, some evidence has shown that doctors feel uncomfortable approaching their colleagues for assistance (16) and instead isolate themselves through the use of alcohol or illicit drugs. Once help is sought, doctors are often given "special treatment," which does not always correlate with the standard of care (2). For example, the treating doctor may use less aggressive techniques when caring for another physician. It may be difficult for physicians to suggest inpatient care for their colleagues who were previously viewed as being strong and healthy. Another explanation for the lack of help-seeking is that there are potentially negative professional consequences for pursuing psychiatric assistance. For example, medical licensing boards may discriminate against physicians with diagnosed psychiatric disorders (9). In addition, personality features associated with physician suicide include excessive self-reliance, high expectations of self, and nondisclosure of personal stress, which may contribute to the lack of help-seeking behaviors demonstrated by physicians (15). Perfectionism, a trait that many physicians possess, which has helped to propel their education and careers forward, may in turn provide a barrier to help-seeking with regard to their own health.

There have been several steps suggested for the prevention of physician suicide (9, 17). Starting in medical school, students should be trained to better recognize the symptoms of depression not only in their patients but in themselves as well. Additionally, physician licensing boards should base decisions on professional performance as opposed to psychiatric diagnoses. Given the high comorbidity of substance use disorders with physician suicide, impaired physicians should be encouraged and incentivized to attend programs for substance rehabilitation. Certain groups, such as the American Foundation of Suicide Prevention, have been actively involved in the prevention of physician suicide (18). In particular, they have launched a documentary film series that increases awareness about physician depression and suicide, which is currently being expanded to also include medical students. A pilot project through the foundation to further develop outreach methods that would encourage students and physicians to seek treatment is underway. The combination of improving education about physician suicide and decreasing barriers to care may help combat this devastating and serious problem.

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Mood Disorders in Women: A Focus on Premenstrual Dysphoric Disorder

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Our modern notion of premenstrual symptoms originated in the early 1930s, with Robert Frank's observational studies of women (1). Frank noted the cyclic occurrence of negative mood symptoms, which he termed "premenstrual tensions," that would disappear shortly after the onset of menstruation (1). This term was used until the 1950s, when it was replaced by premenstrual syndrome or PMS. In 1994, DSM-IV distinguished PMS from a more severe form of symptoms called premenstrual dysphoric disorder (PMDD).

It is estimated that approximately 80% of all women have some form of premenstrual symptoms (2), although PMDD is relatively uncommon, affecting 3%–5% of menstruating women (3). PMDD differs from PMS in that the symptoms of the former are more severe and cause marked impairment in functioning. Symptoms often interfere with family and social relationships and can impair functioning at work. Severe premenstrual symptoms can lead to thoughts that life is not worth living, and approximately 15% of symptomatic women ultimately attempt suicide (4). Despite the fact that the burden of disease from PMDD is similar to that of dysthymic disorder, and not much lower than that of major depressive disorder, it remains an under-recognized disorder by health care providers (5).

Diagnosis

PMDD is currently not an official DSM diagnosis; rather, it is categorized as an example of depressive disorders not otherwise specified and remains a diagnosis "requiring further study." It is described in the DSM-IV appendix as a constellation of symptoms (e.g., depressed mood, marked anxiety, marked affective lability, decreased interest, and lack of energy as well as physical symptoms such as breast tenderness and bloating) occurring during the last week of the luteal phase. Symptoms cannot be the complication of another mental health condition and must remit within 1 week of the onset of menses.

A diagnosis of PMDD requires daily recordings demonstrating symptoms during a minimum of two consecutive menstrual cycles. Evidence suggests that daily ratings often fail to support the subjective, retrospective reports of premenstrual symptoms (6). The Daily Record of Severity Problems is a well-designed scale that can be used for recording premenstrual symptoms (6). The scale consists of 11 items derived from the definition of PMDD symptoms, as presented in DSM-IV, and three additional items describing specific types of functional impairment. Patients are instructed to rate items on a daily basis using a 6-point severity scale and to indicate days of "spotting" or "full flow of menses." This scale has shown high test-retest reliability as well as moderate to high correlation with other measures of PMDD severity. Scores were also found to reflect symptomatic changes with treatment (7). The scale is available online (http://www.pmdd.factsforhealth.org/have/dailyrecord.asp).

Etiology

The etiology of PMS and PMDD remains uncertain at present. Women who develop premenstrual symptoms may have a particular sensitivity to normal cyclical hormonal changes. Several central neurotransmitters have been implicated in the etiology of PMS and PMDD, including serotonin, -aminobutyric acid (GABA), glutamate, and beta-endorphins. For instance, women prone to PMS symptoms experience less inhibitory GABAA activity. These women are thought to have decreased sensitivity to allopregnanolone, a progesterone metabolite that increases during the luteal phase and is known to interact with GABAA receptors. Symptomatic women have also been found to have a lower density of serotonin transporter receptors relative to comparison subjects. It has been hypothesized that estrogen's association with positive mood and well-being may be related to its influence on the seratonergic system (4).

It is unlikely, however, that PMDD symptoms are caused simply by luteal phase levels of estrogen and progesterone. Estrogen and progesterone have a large number of downstream effects, some of which may depend on the change in concentration of these hormones, not just on their absolute levels. Moreover, women with PMS may have abnormalities in the secretion of other hormones, such as melatonin, cortisol, thyroid-stimulating hormone, and/or prolactin. (4). Additional research is needed to understand the complex set of interactions between hormones and the CNS activity involved in PMDD and to elucidate possible biological differences between PMDD and PMS.

Treatment

Treatment for PMDD includes both nonpharmacological and pharmacological options. Although there are not many studies specific to PMDD, data on PMS is often used to inform treatment decisions. Clinicians often recommend minimizing or eliminating caffeine, alcohol, and nicotine and ensuring adequate sleep. Exercise has also been shown to improve emotional and physical symptoms associated with PMS (8–10). A large, randomized-controlled trial found that calcium carbonate (600 mg twice daily) helped to relieve mild to moderate mood and physical symptoms associated with PMS, as measured by daily symptom

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ratings (11). Several trials have suggested that light therapy may improve depression symptoms during the premenstrual phase. However, given the methodological limitations of the trials and small sample size, the efficacy of bright-light therapy for the treatment of PMDD remains uncertain (12).

Herbal remedies may have some role in the treatment of premenstrual symptoms. One double blind, placebo-controlled trial among women with PMS concluded that agnus cactus fruit extract (1 tablet per day), also known as chasteberry, significantly decreased self-assessment of irritability, anger, headache, and breast fullness when compared with placebo (13). Another small single-blind, randomized, placebo-controlled study of women with PMS found that Ginkgo biloba improved symptoms, particularly breast tenderness and fluid retention, as measured by a daily symptom rating questionnaire (14). Early evidence suggested that evening primrose oil was a useful treatment for PMS. However, a recent review of studies found that it was no more effective than placebo (15).

Selective serotonin reuptake inhibitors (SSRIs) are the primary pharmacological treatment for PMDD. Evidence from large randomized-controlled trials supports the use of fluoxetine, sertraline, paroxetine, escitalopram, and venlafaxine in the treatment of mood symptoms associated with the disorder (16, 17). SSRIs are effective only during the luteal phase of the menstrual cycle. However, for more severe PMDD symptoms, they are generally required throughout the month. A recent randomized-controlled trial comparing intermittent and continuous dosing in women with PMDD found that they were equally effective for treating mood swings and irritability but that continuous dosing was more effective in improving social functioning, somatic symptoms, and depressed mood (18). Given the high rate of comorbidity between PMDD and lifetime major depressive disorder (30%–70%), women receiving intermittent dosing often eventually require continuous dosing (4).

Another potential treatment option is the oral contraceptive. In one study, an oral contraceptive containing ethinyl estradiol and the progesterone analogue drospirenone was found to significantly reduce premenstrual emotional and physical symptoms in women with PMS when compared with placebo (19), as measured by the Daily Record of Severity of Problems. It has been suggested that the efficacy of this particular oral contraceptive may be due to its administration regimen (24 days of active pill followed by a relatively short 4-day hormone-free interval), which provides more stable hormone levels, as well as the unique antimineralocorticoid and antiandrogenic properties of drospirenone (20).

Conclusions

While PMS symptoms are quite common, PMDD is a relatively rare but often debilitating disorder. PMDD is not yet considered an official DSM diagnosis, since it was only recently defined in the DSM appendix and requires further investigation. However, there is a growing body of evidence that supports the existence of this discrete disorder, which differs from PMS in terms of its severity and effect on function. Several large trials have found effective pharmacological and nonpharmacological treatments for PMDD symptoms. Whether PMDD is included as an official DMS diagnosis or not, diagnosing and treating symptoms can significantly improve and even save patients' lives.

Dr. Broudy was a clinical fellow in women's mental health in the Department of Psychiatry, Columbia University College of Physicians and Surgeons, New York, at the time this article was accepted for publication. *The author thanks Drs. Elizabeth Fitelson and Sylvia Fogel for their insightful comments and excellent mentorship.*

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CALL FOR PAPERS

The Residents' Journal is looking to publish articles on the topic of Psychiatry and the Military.

Sample topics are as follows:

- A review article discussing posttraumatic stress disorder (PTSD) in a historical context (i.e., the various terminology that has been used to describe PTSD (e.g., "shell shock") and any corresponding evolution in the field's understanding of this disorder.
- A review article discussing military sexual trauma and sexual harassment.
- A commentary on one's experiences serving as military personnel, for example, as a resident training in a military residency, as a medical student training at the Uniformed Services University, or as an active duty serviceman or servicewoman.
- A review article discussing mental health sequelae among children of deployed military personnel.

Manuscripts outside of this theme are also welcome

TEST YOUR KNOWLEDGE

In preparation for the PRITE and ABPN Board examinations, test your knowledge with the following questions.
(answers will appear in the next issue)

This month's questions are courtesy of Dawn L. Flosnik, M.D., who is a second-year resident in the Department of Psychiatry and Behavioral Sciences at George Washington University, Washington, DC. (Please see the accompanying article on physician suicide in this issue.)

Question #1
Compared with the suicide rate among Caucasian American men, the overall suicide rate for physicians in the United States is:

A. One-third
B. One-half
C. Same
D. Double
E. Triple

Question #2
What are the two greatest risk factors for physician suicide?:

A. Mental illness history and substance use disorders
B. Mental illness history and the physician's medical subspecialty
C. Lack of personal support and substance use disorders
D. Occupational demands and lack of personal support

ANSWERS

Answers to August Questions. To view the August Test Your Knowledge questions, go to http://ajp.psychiatryonline.org/cgi/data/168/8/A26/DC2/1.

Question #1.
Answer: D. Antidepressant-induced mania
The patient is most likely experiencing antidepressant-induced mania, a phenomenon in which treatment for depression leads to manic symptoms in susceptible individuals. It is estimated that one-quarter to one-third of patients with bipolar disorder may be susceptible to antidepressant-induced mania. Goldberg and Truman (1) emphasized that "bipolar patients with a strong genetic loading for bipolar illness whose initial illness begins in adolescence or young adulthood may be especially at risk." "Exposure to multiple antidepressant trials" is an additional risk factor for antidepressant-induced mania (1). The risk for developing antidepressant-induced mania may heighten between the ages of 10 and 14 years (2).

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Question #2
Answer: D. Chronic difficulties in mood, emotion, and behavior regulation
There is considerable debate regarding differences in adult and pediatric manifestations of bipolar disorder. However, there is some consensus that pediatric bipolar disorder is often characterized by chronic mood dysregulation and impairment in baseline functioning (1).

Reference
1. McClellan J, Kowatch R, Findling RL; Work Group on Quality Issues: Practice parameter for the assessment and treatment of children and adolescents with bipolar disorder. J Am Acad Child Adolesc Psychiatry 2007; 46:107–125

We are currently seeking residents who are interested in submitting Board-style questions to appear in the Test Your Knowledge feature. Selected residents will receive acknowledgment in the issue in which their questions are featured.
Submissions should include the following:
1. Two to three Board review-style questions with four to five answer choices.
2. Answers should be complete and include detailed explanations with references from pertinent peer-reviewed journals, textbooks, or reference manuals.
***Please direct all inquiries and submissions to Dr. Seawell; mseawell@med.wayne.edu.**

Author Information for *The Residents' Journal* Submissions

The Residents' Journal accepts manuscripts authored by medical students, resident physicians, and fellows; manuscripts authored by members of faculty cannot be accepted.

1. Commentary: Generally includes descriptions of recent events, opinion pieces, or narratives. Limited to 500 words and five references.

2. Treatment in Psychiatry: This article type begins with a brief, common clinical vignette and involves a description of the evaluation and management of a clinical scenario that house officers frequently encounter. This article type should also include 2-4 multiple choice questions based on the article's content. Limited to 1,500 words, 15 references, and one figure.

3. Clinical Case Conference: A presentation and discussion of an unusual clinical event. Limited to 1,250 words, 10 references, and one figure.

4. Original Research: Reports of novel observations and research. Limited to 1,250 words, 10 references, and two figures.

5. Review Article: A clinically relevant review focused on educating the resident physician. Limited to 1,500 words, 20 references, and one figure.

6. Letters to the Editor: Limited to 250 words (including 3 references) and three authors. Comments on articles published in *The Residents' Journal* will be considered for publication if received within 1 month of publication of the original article.

7. Book Review: Limited to 500 words and 3 references.

Abstracts: Articles should not include an abstract.

Upcoming Issue Themes

Please note that we will consider articles outside of the theme.

October 2011
Section Theme: Interventional Psychiatry
Guest Sectionv Editor: Adam Stern, M.D.
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November 2011
Section Theme: Autistic Disorders
Guest Section Editor: Arshya Vahabzadeh, M.D.
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December 2011
Section Theme: Sleep
Guest Section Editor: Dawn Flosnik, M.D.
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January 2012
Section Theme: PTSD and Traumatic Brain Injuries
Guest Section Editor: Brandon Cornejo, M.D., Ph.D.
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February 2012
Section Theme: Family Psychiatry
Guest Section Editor: Michael Ascher, M.D.
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