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In This Issue



This issue of the *Residents' Journal* begins with a commentary by Matthew Goldenberg, D.O., on tobacco abuse and smoking cessation and the role of psychiatry in identifying treatment options. Vijeta Kushwaha, M.B.B.S., M.D., discusses medication nonadherence and provides important information on measuring adherence, factors affecting adherence, and improving adherence. In their article, Rohit Madan, M.D., and Nathan Bruce, D.O., focus on the relationship between Parkinson's disease and impulse control disorders. Amita D. Mehta, M.D., presents findings of a preliminary study of stigma toward mental illness in an Indian American population. Last, Stefania Prendes-Alvarez, M.D., M.P.H., and Samantha Block, M.D., report a case of recurrent, undiagnosed catatonia in an elderly patient.

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A Psychiatric Perspective on Tobacco Use Disorder and Smoking Cessation

Matthew Goldenberg, D.O.

Despite knowledge of the risks and on-going antismoking efforts, about 43.5 million American adults, or 21% of the population, continue to smoke cigarettes, according to data from the American Lung Association. Today, individuals with psychiatric illnesses are about twice as likely to smoke cigarettes as the general population, and they consume 45% of the total cigarettes smoked in the United States (1).

Tobacco-related illness is now the leading cause of preventable and premature death in the United States and around the world. In 2008, the Centers for Disease Control and Prevention reported that one in five deaths in the United States was attributable to tobacco, and the average smoker will die at least 10 years earlier than the average nonsmoker. As a benchmark, in the year 2000, the mortality rate of smoking-related illnesses surpassed the combined death total from HIV, alcohol abuse and illicit drug use, suicide, murder, and car accidents (2).

If there is a silver lining, it is that health significantly improves after one quits smoking. In 2010, the U.S. Surgeon General reported that 10 years after quitting, the risk of developing lung cancer is cut in half. We also know that 15 years after quitting, the risk of coronary heart disease usually drops to the rate for a nonsmoker (3).

As a profession, we have the potential to serve a valuable role, since patients with mental illness and addictions are particularly vulnerable to the negative effects of tobacco abuse. Helping our patients quit smoking begins with diligent screening for tobacco abuse. In order to increase cessation rates and improve outcomes, we must educate ourselves and our patients about smoking cessation treatment options and the perils of nicotine addiction.

In order to effectively tailor your treatment plan, the first step is to collect a

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thorough history. An understanding and knowledge of what is motivating your patient to quit, whether the patient has ever tried to quit before, and what aids have or have not worked in the past will help you determine the severity of your patient's nicotine addiction and guide you in choosing among the various treatment options available.

Overall, pharmacotherapy, group-behavioral therapy, and intensive physician advice can all at least double quit and abstinence rates. Individual and telephone counseling can increase quit rates by 1.5 times, and nursing and self-help interventions are better than none (4). Local and state agencies can be helpful resources for outpatient follow-up. For example, I refer patients to the Arizona Smokers Help Line, which is a valuable resource.

Patients with schizophrenia and/or mood and anxiety disorders will require more intensive interventions and possibly longer durations of treatment to achieve abstinence. It is also important to note that the recorded quit rates of patients with psychiatric illnesses are similar to those of the general population, even though they smoke more cigarettes and inhale them more deeply (5).

I have concluded that doing nothing is more harmful to my patients than screening, educating, and formulating a specialized smoking cessation treatment plan. I encourage you to take action

because tobacco abuse continues to devastate the health and quality of life of our patients.

Dr. Goldenberg is a third-year resident in the Department of Psychiatry, Banner Good Samaritan Medical Center, Phoenix.

As outlined in an August 2013 American Journal of Psychiatry editorial, the field is [Searching for More Effective Smoking Cessation Treatment](#).

Psychiatric Services published an interesting [study in the January 2014 issue](#) that surveyed clinicians at nine community mental health centers and found that even though many clinicians wanted to help their clients quit smoking, they lacked training in smoking cessation methods, as well as the organizational support to be of much help.

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Medication Nonadherence: A Challenge in Patient Care

Vijeta Kushwaha, M.B.B.S., M.D.

Medication nonadherence is a significant problem in clinical medicine. Patients suffering from psychiatric disorders are less likely to adhere to pharmacotherapy compared with patients receiving medications for nonpsychiatric medical conditions (1–4). A wide range of nonadherence rates have been reported for patients with bipolar disorder (20%–60%), unipolar depression (10%–75%), anxiety disorders (57%), schizophrenia (20%–72%, generally exceeding 60%), and attention deficit hyperactivity disorder (50%–75% over 12 months) (5–11). The variable nonadherence rate in these studies is partly related to lack of consensus about the operative definition and tools for objective measurement (7, 12, 13). We also need to be mindful that nonadherence is not a dichotomous variable or a clinical outcome but a dynamic phenomenon that is determined by the interaction of several variables in a given person at a given point in time. Understanding the causes of nonadherence and improving patients' compliance to psychotropic medications is one of the challenges faced by all mental health professionals with patients across all ages, diagnostic categories, and treatment settings (6, 11, 14, 15).

Why Is It Important to Know and Address?

Improvements in adherence are shown to reduce hospitalizations, improve patient outcomes, and reduce overall health care costs (4). Medication will work only if it is taken by the patient, and thus it is equally important to understand the factors that improve adherence and employ them to the best of the patient's benefit. Further challenge lies in implementation. Nonadherence in patients with schizophrenia has been found to be associated with increased risk of homelessness, aggression, and property damage. Repeated psychotic relapses may negatively affect course and prognosis, ultimately resulting in resistance to antipsychotic medications and

to the development of chronic psychotic symptoms (10). In bipolar disorder, poor adherence has been found to increase the likelihood of relapse and neuroprogression, re-hospitalization, poor quality of life, and increased risk of suicide (8). Inadequate adherence to antidepressants may lead to increased recurrence, severity, and disability, with poorer response to future treatments (13). In economic terms, it ultimately translates to increase in direct and indirect health care costs. Research suggests that approximately 40% of hospitalization costs for persons who have schizophrenia are attributable to nonadherence to medication (10, 13).

Defining Adherence and Nonadherence

The World Health Organization (WHO) defines adherence as “the extent to which a person's behavior—taking medication, following a diet, and/or executing lifestyle changes, corresponds with agreed recommendations from a health care provider” (9). There is no consensual standard for what constitutes adequate adherence. Most trials consider rates greater than 80% to be acceptable, whereas some studies use a more stringent cut off of 95% (1, 7).

How to Measure

It is important to distinguish between adherence attitudes and adherence behavior because the two are often confused. They may seem to overlap, but they are distinct concepts leading to unique assessment and management issues (13, 16). Adherence attitude can simply be understood as whether the patient wants or does not want to take the medications. Scales of measuring adherence attitude usually cover three domains: subjective response to medication, insight, and comprehensive measures of adherence influences (e.g., the Drug Attitude Inventory, Rating of Medication Influences Scale, and

Brief Evaluation of Medication Influences and Beliefs) (12, 13). Adherence behavior is whether the patient actually takes the prescribed medication or not, which can fluctuate over time. Measures of adherence behavior can be subjective or objective. Objective measures include directly observed therapy, pill count, pharmacy records, an electronic compliance monitor, a medication electronic monitoring system, and plasma concentration levels. Subjective measures, on the other hand, are usually self-reported. Commonly employed subjective measures include clinical interview and self-report questionnaires, such as the Medication Adherence Rating Scale and the Brief Adherence Rating Scale (7, 8, 13, 15). Both subjective and objective measures have been validated, but subjective measures slightly overestimate adherence. Expert guidelines recommend objective methods (9); however, in clinical settings, the use of self-reports with a collaborative relationship is more practical (17). Using more than one measure may give a better estimation of actual adherence.

Factors Affecting Adherence

To improve the patient's ability to follow a medication regimen, all potential barriers to adherence need to be considered. WHO recommends that determinants, which may positively or negatively influence adherence, be categorized into the five dimensions listed below (2).

1. *Patient-related factors:* Patient demographic characteristics such as age, ethnicity, gender, education, employment history, marital status, and living situation have been consistently shown to predict medication adherence among patients with affective disorders but less consistently in schizophrenia. Other patient-related factors that can adversely affect adherence include poor insight, denial of illness, stigma and negative attitudes toward medication, presence of

comorbidity, and lack of social support (7, 9, 15).

2. *Disorder or illness-related factors:* Factors such as nature of illness, age at onset, and number of hospitalizations have been found to affect adherence (8). In schizophrenia and bipolar disorder, symptom severity is negatively correlated, whereas neurocognitive deficits have been inconsistently linked (18).

3. *Treatment or therapy-related factors:* The number of medications (polypharmacy), increased dosing frequency, and side effects, such as sedation, weight gain, akathisia, and sexual dysfunction, have been shown to decrease adherence. The nature of the therapeutic relationship and provider-patient communication are other important determinants (1, 9).

4. *Health care system-related factors:* The availability of the provider, access to health care delivery, and frequency and ease of follow-up are some of the factors that potentially influence adherence (2).

5. *Socioeconomic factors:* Low income, concerns about cost, high co-pay, lack of insurance, lack of family and social support, dysfunctional families, and social stigma of disease are some of the negative predictors of adequate adherence (2, 7).

Improving Treatment Adherence

It is important to specifically understand the facilitators and barriers to adherence in a given patient. Improved physician-patient communication and establishing and maintaining a therapeutic alliance are important steps toward improving adherence. Experts have recommended a three-tiered model-based intervention approach to addressing adherence issues (12). This model proposes interventions to be delivered based on the risk of a patient's nonadherence. This approach assumes that most patients could benefit from an adherence intervention, but some patients may require a more intensive approach. Since adherence problems are multifactorial and complex, the interventions likewise will be multifaceted (1, 7). Methods that have been used to improve adherence can be broadly grouped

under psychological and pharmacological interventions.

Psychological Interventions

Psychoeducation is an example of a disease-based intervention. Studies of schizophrenia and psychosis patients have shown that psychoeducation has no benefit over standard care with regard to adherence or clinical outcomes when delivered to patients alone, but improved outcomes resulted when family-focused interventions were made, rather than solely patient-focused interventions (5, 10, 17). Psychoeducation-based interventions have been explored in patients with bipolar disorder, although results thus far have been equivocal. Results have been more promising when a family member has been included (5, 8, 17). Familial involvement has shown modest improvements in individuals with eating disorders, anxiety and panic disorders, addictive disorders, schizophrenia, depressive disorders, and bipolar disorder (12).

Cognitive-behavioral therapy (CBT)-based interventions challenge patients' automatic thoughts regarding their medications. Various models based on CBT have been proposed and empirically tested. The health belief model is one of the earliest treatment adherence models used with adults and children. It provides the prescriber with a guideline to explore individual perceptions about the perceived seriousness of illness, modifying factors, and likelihood of action (11). Another CBT offshoot, the theory of reasoned action, is a cognitive theory to explain the decision to engage in behavior that is based on social norms, as well as attitudes and beliefs about potential outcomes. This theory has some similarities with the health belief model, but it uniquely focuses on the gap between the intention to adhere and actual adherence (11, 12). Compliance therapy is another cognitively based therapy using motivational interviewing techniques that has shown promising results. It has its underpinnings in the trans-theoretical model of change, which describes the process of how individuals move toward accepting treatment recommendations and thus adherence (10).

Behavioral interventions use reinforcement techniques. These are intended to change adherence by targeting, shaping, or reinforcing specific behavioral patterns. These include strategies such as skill building and practice activities, modeling, packaging, and use of pill boxes and reminders (19).

Pharmacological Interventions

Pharmacological interventions can include strategies such as simplifying medication regimens, including minimizing the number and dosage frequency of medications if possible, using depot antipsychotics, or changing antidepressants when sexual side effects are present (1, 20). Second-generation antipsychotics usually have better tolerability over first-generation antipsychotics, but they do not provide any additional meaningful benefits in improving adherence in patients with schizophrenia and schizoaffective disorders (10).

Conclusions

Poor adherence to medications is a universally encountered phenomenon in clinical practice and has serious implications. As part of good clinical practice, patients' preferences and attitudes toward medications and their actual compliance should be explored during treatment. It is useful to involve family when possible. Potential factors contributing to nonadherence should be evaluated on an individual basis, and intervention should be tailored per the need.

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Impulse Control Disorders in Parkinson's Disease: Implications for Psychiatrists

Rohit Madan, M.D.
Nathan Bruce, D.O.

In reviewing your upcoming clinic referrals, you notice that you are going to see a 55-year-old man who is reportedly obsessed with pornography, a 68-year-old woman who has had a recent onset of kleptomania, and a 63-year-old male previous nonsmoker who began smoking obsessively 1 year ago. You further note that all three patients have a diagnosis of Parkinson's disease and suspect that they all have something else in common, an impulse control disorder.

Impulse control disorders are defined by the failure to resist an impulse, drive, or temptation to perform an act that is harmful to the individual or to others (1). Impulse control disorders are an increasingly recognized phenomenon in Parkinson's disease. Over the last decade, these disorders have been discovered to be linked to treatments of Parkinson's disease.

Impulse control disorders include a wide range of heterogeneous neurobehavioral disorders, including pathological gambling, compulsive shopping, hypersexuality, compulsive eating, hobbyism (preoccupation with specific hobbies/activities), punding (stereotyped, repetitive, purposeless behaviors), and dopamine dysregulation syndrome (compulsive dopaminergic medication overuse) (Table 1), as well as many more not formally categorized.

Not surprisingly, impulse control disorders have been conceptualized to be a form of "behavioral addiction" (2). In DSM-5, impulse control disorders are categorized with disruptive, impulse-control, and conduct disorders, whereas in DSM-IV they were categorized under impulse control disorders, not elsewhere classified (1).

Prevalence and Risk Factors

Impulse control disorders in patients with Parkinson's disease are relatively common,

Key Points

- Watch out for newly emerging or worsening pre-existing impulsive or compulsive behaviors.
- Impulse control disorders in Parkinson's disease are strongly linked to dopamine agonists.
- Early recognition and timely appropriate action can prevent worsening and consequences.

with a prevalence range from 5.9% to 13.7% and a point prevalence of approximately 4% (3, 4). A large cross-sectional study of 3,090 patients with Parkinson's disease who were being treated at 46 centers across the United States and Canada identified an impulse control disorder in 13.6% of patients. The most common of these were excessive spending (5.7%), problem gambling (5%), binge eating (4.3%), and aberrant sexual behavior (3.5%). Nearly 4% of patients had two or more disorders, and many patients also had concurrent compulsive behaviors (e.g., hobbyism) (3). Similar prevalence findings have been reported in a large Asian study (5).

Studies have shown a strong association between dopamine agonists (e.g., pramipexole, ropinirole, rotigotine, and pergolide) and impulse control disorders in Parkinson's disease, with an even higher risk when patients receive both a

dopamine agonist and levodopa (6). In contrast, Parkinson's disease patients who receive levodopa as monotherapy, even in high doses, do not seem to be at risk of developing an impulse control disorder, since the prevalence in this group is only slightly higher than that of the general population (3, 4). However, levodopa monotherapy seems more likely to specifically provoke compulsive behaviors, such as punding or dopamine dysregulation syndrome. Data on impulse control disorders in patients with Parkinson's disease treated with deep brain stimulation is currently limited, although cases have been reported (7).

Clinical risk factors for developing impulse control disorders have been identified, including a personal or familial history of alcoholism or gambling, impulsive or novelty seeking traits, younger age at onset of Parkinson's disease, male sex, unmarried status, and current cigarette smoking (8). These characteristics are striking given that the "typical" Parkinson's disease patient's personality profile is usually described as industrious, punctual, inflexible, cautious, lacking in novelty seeking, and being highly harm avoidant (9). It has been suggested that these characteristics reflect the progressive dopaminergic deficit in Parkinson's disease. It follows then that impulse control disorders are more commonly seen in those Parkinson's disease patients

TABLE 1. Types of Impulse Control Disorders in Parkinson's Disease

Behavior	Description
Overspending	Buying unnecessary material items/altruistic spending
Pathological gambling	Traditional forms of gambling, as well as risky investments
Eating disorders	Particularly binge eating
Hypersexuality	From inappropriate sexual demands/advances to obsession with pornography
Punding	Stereotyped, repetitive, purposeless behaviors
Hobbyism	Intense preoccupation with specific hobbies/activities
Dopamine dysregulation syndrome	Compulsive dopaminergic medication overuse

who have a personal or familial history of alcoholism or gambling or impulsive or novelty seeking traits because these patients presumably have higher baseline dopaminergic activity and may be a subgroup more likely to develop these disorders when treated with a dopamine agonist.

Diagnosis and Differential Diagnosis

Impulse control disorders rarely begin soon after initiating medication but rather develop insidiously after years of treatment, often when dopamine agonist doses are increased or when levodopa and dopamine agonists are used together.

These disorders easily go undetected because patients are very unlikely to report these behaviors because of the associated embarrassment. Some behaviors, such as excessive walking or writing, may be difficult for family members and physicians to identify. Many impulse control disorders are conducted in isolation on the Internet (e.g., those related to gambling and pornography) and are often only discovered accidentally or at a time of crisis.

Certain behaviors, such as excessive spending and increased interest in sexual matters, may initially suggest the onset of hypomania or mania and prompt a psychiatric referral. In making a differential diagnosis, history of a mood disorder is important because new onset of mania is less likely in older patients. Patients with impulse control disorders generally do not present with typical symptoms consistent with bipolar disorder, such as irritability, distractibility, decreased need for sleep, or grandiosity.

Superficially, the occurrence of compulsive behaviors may prompt consideration of other primary diagnoses, such as obsessive-compulsive disorder (OCD) or substance use disorders, but careful review of the patient's history usually will exclude these conditions. Another important differential consideration is a disinhibition syndrome, particularly hypersexuality, which can be seen in patients with dementia that affects the frontal lobe, particularly Alzheimer's disease and frontotemporal dementia.

Patients with Alzheimer's dementia often develop some extrapyramidal symptoms later in the course of illness but are unlikely to be treated with dopamine agonists. On the other hand, many patients with Parkinson's disease have comorbid dementia, and impulse control disorders in these patients may be misinterpreted as behavioral manifestations of dementia.

Early recognition and accurate diagnosis is important because impulse control disorders can have serious consequences, such as significant financial loss, strained interpersonal relationships, and psychosocial morbidity. Accurate diagnosis is also essential, since treatment approaches are dictated by the underlying cause.

Pathophysiology/Role of Dopamine Agonists

Dopamine agonists have specific affinity for D₃ receptors, which are also located in limbic nuclei, and are implicated in the dopamine reward pathway. Thus, dopamine agonists stimulate pathways that govern reward behavior, pleasure, and addiction (10). Parkinson's disease patients commonly display a range of impairments in executive abilities, including response inhibition, which contributes to vulnerability for impulse control disorders (11, 12). In addition, dopamine agonists have been shown to impair punishment learning in Parkinson's disease patients, which may lead to engagement in senseless repetitive behaviors (13).

Management

The first step is to identify individuals with active impulse control disorders by routine screening of patients with Parkinson's disease and their family members. All patients with impulse control disorders should be thoroughly assessed for other comorbid neuropsychiatric problems. For those patients whose disorders occur in the context of dopamine receptor agonist treatment, the behaviors often resolve or improve with daily dose reduction, switching to a different agonist, or discontinuing agonist treatment entirely. However, some patients are hesitant to reduce the dosage of dopamine agonists because motor worsening may result. It is also important to be aware that a ste-

reotyped dopamine agonist withdrawal syndrome can occur, which can include anxiety, panic attacks, dysphoria, diaphoresis, fatigue, pain, orthostatic hypotension, and drug cravings (14).

There are no clear-cut guidelines or recommendations regarding further options for patients with persistent impulse control disorder symptoms after reducing or eliminating dopamine agonists. Atypical neuroleptics, mood stabilizers, and antiepileptics have been tried with some success based on case reports and some nonrandomized clinical trials (8). The value of selective serotonin uptake inhibitors, although effective in palliating many OCDs, is uncertain in treating impulse control disorders triggered by dopaminergic therapies. Naltrexone, an opioid antagonist, is also being evaluated for potential efficacy in this condition (15).

Other therapeutic strategies may include interventions such as participation in Gamblers Anonymous, support groups, and behavioral interventions. It may be important for the family to limit the patient's access to money or the Internet to reduce the patient's ability to gamble, shop, or pursue illicit sexual activity. This may be particularly challenging in patients who appear otherwise cognitively intact and capable of self-determination.

In some patients, deep brain stimulation might be helpful by improving control of dyskinesia and motor fluctuations, thereby making it easier to reduce the dosage of dopaminergic therapies. However, there have been case reports of impulse control disorder behaviors and compulsions beginning after patients underwent deep brain stimulation for Parkinson's disease (7). Further research is required to elucidate the role that deep brain stimulation may play as a potential treatment.

Conclusions

The group of behaviors characterized as impulse control disorders are often considered the province of psychiatrists, and consultation to evaluate these behaviors is likely. Patients and their caretakers should be informed about the risk of these disorders before initiation of medication. Treatment involves reducing the dosage, switching, or discontinuing the dopamine

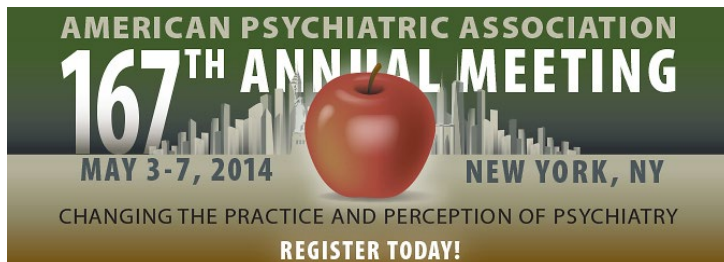
agonist. Alternatives include use of atypical antipsychotics, reduction in levodopa dosage, behavioral interventions, and deep brain stimulation. Future research is required to explore the in-depth relation of impulse control disorders with dopaminergic agonists, as well as the prevention and treatment of these disorders in Parkinson's disease.

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A Preliminary Study of Stigma Toward Mental Illness in the Indian American Population

Amita D. Mehta, M.D.

Mental health disorders are present in approximately 10% of the adult population worldwide. In many countries, families bear a significant portion of economic costs due to limited government-funded mental health services, while also experiencing a dampened quality of life from the emotional burden of looking after disabled family members (1). These economic and social burdens contribute to the stigma of mental illness among all cultures.

Mental illness stigma has been defined as the “devaluing, disgracing and disfavoring by the general public of individuals with mental illnesses.” Scrambler (2) used two terms to define stigma: felt stigma and enacted stigma. Felt stigma (internal stigma or self-stigmatization) refers to the shame and expectation of discrimination that prevents people from talking about their experiences and stops them from seeking help. Enacted stigma (external stigma, discrimination) refers to the experience of unfair treatment by others. Ultimately, stigma can prevent mentally ill individuals from seeking treatment, adhering to treatment regimens, finding employment, and living successfully in community settings (3).

The objective of this study is to provide understanding and knowledge of various sources of stigma toward mental illness in the Indian population living in the United States.

Indian Culture

Traditional Indian values are complex, focusing on interpersonal harmony, filial piety, hierarchical family, and social structure. There is a strong emphasis on emotional restraint and self-control, with extreme shame resulting from aberrations from the cultural norm (4). Feelings of shame strongly influence behavior and serve as a motivator to reach the high expectations set by family members. If unable to deliver, family members are often overwhelmed with guilt, which

negatively affects behavior, both at home and in society. Furthermore, attainment of harmony (both intrapsychic and interpersonal) through compromise between individual and group needs is highly valued.

Interestingly, Indian patients express psychiatric symptoms predominantly through somatization. Conversion disorders, histrionic symptoms, and compulsive behaviors are the few presentations that have been well described (4).

Family and Allocentricism

Indians are allocentric (group oriented) rather than idiocentric (self-oriented). There is an expectation that the individual will make sacrifices for the family, including investing money toward the education of various family members and participating in arranged marriages that unite families. Furthermore, families are also the primary source of emotional, social, and financial support. Interdependence is fostered, while self-identity is discouraged. A conservative orientation is ultimately rewarded. The traditional Indian family does not reward competitiveness, achievement, or self-orientation within the family. This value, however, has become increasingly controversial because Indian Americans are beginning to adopt more Western ideals that focus on independence and personal achievement (5).

Privacy within the family is valued, with reluctance to trust nonfamily members with familial secrets. Taking this into consideration, seeking professional help for psychiatric issues outside of the extended family is usually not accepted and highly stigmatized (5).

Religion

About 80% of India’s population is Hindu. Regardless of religion, most Indians believe in fatalism and animism. Psychological distress and disorders are often explained in a religious framework, in terms of a “curse from God” or

a punishment for “sins of the past life.” Psychiatric interventions conducted by witch doctors, priests, or family members can include chaining up the mentally ill, chanting spells, or poking the mentally ill with pins or beating them “to force the spirit out” (6). As help is sought, every precaution is taken to ensure anonymity in order to protect families from unwanted attention and stigma.

Migration

Indian people in India are putatively different from Indian people in America, since individuals who migrate develop weakened customs and social support systems as they adjust to their new culture. Consequently, such changes may lead to transformations in cultural identity, which often translates into higher rates of mental illness in migrant groups (6).

Study Design

Participants were recruited through the identification of Indian origin populations through local community groups or social media, with subsequent e-mail contact to solidify participation. An e-mail script was sent to all participants, which explained the purpose of the study, along with a link to complete an anonymous 18-question survey. Survey questions were obtained through analysis and consolidation of multiple mental health stigma surveys available online (7–9). The study was approved by the institutional review board of Allegheny Health Network.

Results

Ninety individuals were contacted, and 40 responded. The study demonstrates that few participants were likely to discuss their mental health issues with a physician. Perhaps even more alarming, very few were willing to discuss their psychiatric problems with close family and friends, despite living in a culture

centered on family. Interestingly, many would be uncomfortable seeking psychiatric help if they were suicidal. None of the participants reported feeling discriminated against or ashamed. Details of the questionnaire results are summarized in Tables 1 and 2.

Discussion

Together, these findings suggest that Indian Americans would risk their quality of life in exchange for preserving the image they present to family, friends, and professionals. In hiding their sickness, they temporarily bury their shame and suffer the debilitating consequences. Understanding this aspect of Indian culture is paramount in identifying and consequently treating mental illness in this population.

Psychotherapy and counseling are rarely utilized in the Indian culture. General physicians, however, are held in high regard, necessitating that serious referrals for psychiatric help originate from primary care providers. Ultimately, psychiatrists should expect requests for a “quick fix,” since patients desire to hide symptoms before they are revealed to the public. Long-term treatment plans are therefore rarely successful (5); when the Indian patient does not see immediate results, he or she will usually terminate treatment. For this reason, contractual short-term interventions targeted at relatively rapid symptom relief are preferred.

Expectations for psychiatrists are strongly shaped by the traditional Indian belief in the guru-disciple relationship. To therapists, this will feel like an unhealthy dependency but should be recognized as an expression of cultural norms. The patient will often test the psychiatrist’s personal integrity and spiritual depth because the Indian culture is heavily influenced by Hindu spiritual values (4). Psychiatrists are cautioned to focus on their own personal integrity and to let go of any desire to succeed or see results.

There are several limitations to this study. First, the study consisted of a small sample size. A larger sample survey would be helpful in obtaining a more comprehensive picture of stigma in this population. Second, demographic information (e.g.,

TABLE 1. Results of Personal Attitudes Toward Mental Illness in an Indian American Population (N=40)

Item	Participants Who Were Uncomfortable	
	N	%
If you experience mental/emotional conflicts or had a mental health diagnosis, how comfortable would you be with the following:		
Talking with your doctor	21	52.5
Taking medication	12	30.0
Discussing with your immediate family	21	52.5
Discussing with your extended family	36	90.0
Discussing with your current employer	34	85.0
Seeking professional help for your substance abuse problem or that of a loved one	14	35.0
Seeking professional help for suicidal thoughts	18	45.0
Seeking professional help for emotional problems related to experience of sexual abuse	20	50.0
Sharing the experience of psychiatric hospitalization with friends/colleagues	36	90.0
Marrying a person with established psychiatric problems	32	80.0
Giving your child medication for psychiatric problems	12	30.0
Sending your child to a day care when the baby sitter has psychiatric problems	33	82.5
Giving your business to a banker/cook who has a psychiatric problem	21	55.5

age, gender, and educational background) was not collected. Third, since this was a preliminary study, there was no control group and no corresponding statistical analysis was performed.

The benefits to individuals and families in tackling stigma include a reversal of the negative effects of discrimination. It is likely that people would present for treatment sooner, with increases in treatment

adherence, if stigma were reduced. Society would not only benefit from greater social cohesion but would reap financial gains by rehabilitating people into paid employment.

To expand upon our preliminary findings in Indian Americans, we are administering the survey to Indians living in India to perform a cross-cultural study between these two populations. The hope is to

TABLE 2. Results of Social Attitudes Toward Mental Illness in an Indian American Population (N=40)

Item	Participants Who Agreed	
	N	%
Someone with emotional problems (crying excessively) needs to be hospitalized.	12	29.5
Someone with psychosis problems needs to be hospitalized.	26	64.5
Mental health diagnoses are considered an excuse for crimes.	27	67.5
People with psychiatric illness are dangerous and more liable to commit crimes.	17	42.5
One should discourage a friend from marrying someone with psychiatric problems.	23	57.5

gain a better understanding of the origin of stigma and to identify future goals to combat stigma in this ethnic group.

Conclusions

An understanding of the Indian American's way of life (i.e., cultural practices, belief systems, and attitudes toward mental illness) is essential for mental health professionals working with Indian immigrants. Being informed about their customs and traditional values can help professionals be more sensitive to their specific mental health needs.

Dr. Mehta is a fourth-year resident in the Department of Psychiatry, Allegheny General Hospital, Pittsburgh.

The author thanks Drs. Misty Richards, Gary Swanson, and Anjali Medhekar for their assistance.

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A Case of Recurrent, Undiagnosed Catatonia

Stefania Prendes-Alvarez, M.D., M.P.H.
Samantha Block, M.D.

Catatonia is a neuropsychiatric syndrome characterized by three or more symptoms of stupor, catalepsy, waxy flexibility, mutism, and negativism (among others). It was first described by Karl Ludwig Kahlbaum in 1874 and historically has been associated with psychosis and subsequently schizophrenia. However, catatonia is a syndrome with multiple organic and psychogenic causes beyond schizophrenia.

In the present report, we present an uncommon manifestation of catatonia in a patient being evaluated for seizures and discuss the different causes of catatonia.

Case

“Mr. K” is a 71-year-old right-handed Caucasian man with poorly controlled posttraumatic stress disorder (PTSD), depression, diabetes mellitus type 2, hypertension, peripheral neuropathy, and coronary artery disease. He was electively admitted to a Veterans Administration (VA) hospital for continuous electrographic monitoring for evaluation of a possible seizure disorder. He had a previous similar admission, with diagnostic workup that was nondiagnostic for a seizure disorder. His clinical presentation included a 20-plus year history of recurrent episodes of “passing out and waking up in a hospital.” The patient never recalled any details surrounding these episodes. His wife had witnessed multiple episodes and stated that he would become bradykinetic (lying in the same position and only occasionally changing postures) and respond inappropriately.

During this admission, Mr. K did not have a typical event. Instead, he was preoccupied with speaking about his service years with a medical team. Forty-eight hours of continuous EEG monitoring only showed rare sharp transients over the left anterior temporal region, and he was discharged home.

On the following day, he fell and was found lying on the sidewalk by a friend who subsequently called 911. He was initially evaluated at a local hospital and then referred and transported to the VA hospital because of persistent decreased responsiveness. Upon arrival to the emergency department, Mr. K's vital signs were normal, and he was noted to be clinically catatonic; he remained lying on his back without voluntary movements and with his eyes opened (midline) but without eye contact. He would blink in response to threat but would not track any visual stimuli or movement. Additionally, he was mute and displayed a waxy flexibility; he maintained his four limbs elevated against gravity until an examiner lowered them. The remainder of the neurological examination revealed no focal deficits, and the general examination was only significant for a laceration of the forehead and nose. Stat CT scan of the head and cervical spine were unremarkable. Complete blood count, arterial blood gas, serum ethanol levels, and complete metabolic panel were all normal. A stat EEG while the patient remained clinically catatonic revealed similar findings as the baseline EEG from the previous day's admission. The background consisted of a very low amplitude mixture of beta and alpha rhythms.

The patient was transferred to the EEG monitoring unit, and he was given 1 mg of lorazepam. The continuous EEG continued to show a mixture of alpha and beta activity, and interestingly the alpha rhythm appeared better modulated. He then progressed into stage 2 and slow-wave sleep, and the recording was continued for a complete 24 hours.

The patient's wife arrived shortly after he had received lorazepam, and she verified that this event resembled his classical events. During the following 3 hours, the patient was inconsistently able to grasp his wife's hand but not the hand of the

clinical provider. He was also able to track his wife visually but not the doctor.

The following morning, he was back at his baseline; he was sitting up and conversing with his wife when the medical team arrived. He reported only a minimal recollection of the previous day's events. He described an experience of hearing people's voices, not knowing whether they were speaking to him, and seeing the world as though through a “rounded glass.” He felt an unexplainable distance between himself and everything happening. On this morning, he persisted on speaking with the team about detailed experiences from war, and he mentioned how a recent attendance at an Army graduation had made him reflect on his service. He became tearful when sharing details of his service years.

Discussion

Catatonia is a motor and mood dysregulation syndrome that occurs in men and women of all ages (1). In 1980, the DSM-III identified catatonia as a type of schizophrenia (2). It was not until 1994 that DSM-IV recognized catatonia outside of schizophrenia and added “catatonia secondary to a medical disorder” (2). This acknowledged the organic causes of catatonia that include neurologic disorders (stroke, epilepsy, and traumatic brain injury), metabolic disturbances, endocrine disorders, infections, intoxication, and pharmacologic agents. These organic causes were excluded in our patient, and we do not believe that his episodes could be secondary to a seizure disorder because continuous monitoring during the event showed no interictal or ictal discharges. Although it is well known that a patient with frontal lobe seizures can have a normal EEG recording, our patient's clinical presentation of catatonia is unlikely ictal given the prolonged duration of the state and the inconsistency of the neurological examination. Previous studies monitoring

catatonic patients on EEG have reported an increase in alpha frequency and a decrease in alpha amplitude (3), which was observed in our patient during the overnight recording.

The treatment team was left to consider psychogenic causes of catatonia. In DSM-5, catatonia is not a subtype of schizophrenia but a specifier for schizophrenia and other psychiatric diagnoses. This change comes from the study of psychogenic causes of catatonia, including schizophrenia, reactive psychosis, major depression, bipolar disorder, conversion disorder, and dissociative disorders. Our patient has no history of psychosis or bipolar disorder, and his underlying diagnosis of depression has no clinical correlation to the catatonic events.

Several cases of conversion catatonia have been described in the literature, always with an identifiable stressor preceding the catatonic episode (4). Identification of a stressor before the captured event was difficult with our patient, but we have not completely eliminated conversion disorder from our differential. There have also been cases of dissociative disorder presenting as catatonia (5), and our patient's description of hearing people's voices, not knowing whether they were speaking to him, and seeing the world as though through a "rounded mirror" could suggest a dissociative disorder manifesting as catatonia.

Additionally, our patient had poorly controlled PTSD manifested as daily nightmares and flashbacks. Although little is known regarding an association between catatonia and PTSD, one case

describes a catatonic presentation as being the extreme of the avoidance, numbness, and motor responses encountered in PTSD (6). This raises the question whether our patient's catatonic presentation could be related to his PTSD.

Lastly, there is little published in the literature relating catatonia to factitious disorder. In factitious disorder, a person acts as though he or she has an illness in order to assume the sick role and gain the attention of a patient. Perhaps this could explain the small laceration on our patient's face and his selective responsiveness to his wife as soon as she arrived. Unspecified catatonia was added to DSM-5 for patients showing catatonia of uncertain origin (7). For the time being, we will say that our patient has unspecified catatonia until further interviewing can assist us with understanding which psychogenic cause is most likely contributing to his recurrent catatonic episodes.

Conclusions

Catatonia, a marked psychomotor disturbance, can have a puzzling clinical presentation that leaves many physicians with more questions than answers. Individuals presenting with catatonia should not be immediately considered to have psychosis. Multiple medical conditions and mental disorders can explain a patient's catatonia, and physicians should perform a thorough investigation to determine the underlying medical or psychiatric diagnoses in order to better understand and treat the patient.

Drs. Prendes-Alvarez and Block are both first-year psychiatry residents in the Department of Behavioral Sciences, University of Miami.

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

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 David Hsu, M.D., a fellow in geriatric psychiatry at Massachusetts General Hospital/McLean/Harvard, Boston, and Associate Editor of the Residents Journal.

Question 1.

Which of the following is true of major or mild frontotemporal neurocognitive disorder?

- A. The three language variants are semantic, agrammatic/nonfluent, and logopenic.
- B. The behavioral variant must include five core symptoms: behavioral inhibition, apathy, loss of empathy, ritualistic behavior, and hyperorality.
- C. It is a common cause of dementia in people over age 65.
- D. The course of frontotemporal neurocognitive disorder is slower than the course in typical Alzheimer's disease.

Question 2.

A 65-year-old man without significant medical or psychiatric history presented to the emergency department after falling at home. His medical tests do not demonstrate any marked abnormality. You are the consulting psychiatrist. After interviewing him, you find that he has had recurrent episodes of unexplained falls, autonomic dysfunction, fluctuating cognition, and difficulty sleeping. He notes that there is a boy that follows him "everywhere and is in this room," but it does not bother him. You note significant cogwheeling on examination, and his cognitive testing is somewhat impaired. What is the diagnosis?

- A. Major neurocognitive disorder due to Alzheimer's disease
- B. Major neurocognitive disorder with Lewy bodies
- C. Major frontotemporal neurocognitive disorder
- D. Major vascular neurocognitive disorder

ANSWERS TO JANUARY QUESTIONS

Question #1.

Answer: B. Animism

Animism refers to the attribution of human emotions or characteristics to animals or nonliving objects.

It is present in the pre-operational stage of Piagetian development (ages 2–7 years). Object permanence, the understanding that objects continue to exist even though they cannot be seen, develops during the Piagetian sensorimotor stage (birth to 2 years old). Reversibility is the ability to understand that objects can be returned to their original state (e.g., a deflated ball can be re-inflated), and it develops during the Piagetian concrete operations stage (7–11 years old). Mindfulness is not a Piagetian concept.

Reference

1. Cook JL, Cook GL: Child Development: Principles and Perspectives. Upper Saddle River, NJ, Pearson, 2005

Question #2.

Answer: C. Earlier age at onset

A later onset of symptoms has been associated with a better prognosis in schizophrenia, as opposed to early illness. Female gender, good premorbid functioning, rapid symptom onset, and lack of negative symptoms are all good prognostic indicators.

Reference

1. Weinberger DR, Harrison P (eds): Schizophrenia. Hoboken, NJ, Wiley-Blackwell, 2011

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Submissions should include the following:

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- *Please direct all inquiries and submissions to Dr. Hsu: davidhsu222@gmail.com.

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