

THE AMERICAN JOURNAL OF PSYCHIATRY RESIDENTS' JOURNAL

August 2014

Volume 9

Issue 8

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In This Issue



In this issue of the *Residents' Journal*, Nathan Bruce, D.O., and Rohit Madan, M.D., discuss the clinical features of REM sleep behavior disorder, how it is diagnosed, and treatment options. In a treatment in psychiatry article, Arundhati Undurti, M.D., Ph.D., provides data on the evaluation and management of agitation and aggression in patients with traumatic brain injury. In a case report, Kristin V. Escamilla, M.D., and Isela A. Werchan, M.D., discuss the benefits of a preoperative dose of olanzapine administered to a patient with a history of postoperative delirium. Maria J. Julios Costa, M.D., reports on a case of successful continuation of an ECT course following tardive spontaneous seizure in a patient with major depressive disorder. Suni Jani, M.D., M.P.H., comments on her experience as a psychiatry resident visiting families with children with developmental disabilities in India, the cumulative global impact of mental disorders, and the state of global psychiatry. In a film review, Natosha D. Smith, M.D., M.P.H., discusses *The Perks of Being a Wallflower* and how such movies can help reduce the stigma of adolescent mental illness.

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REM Sleep Behavior Disorder

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

Sleep disturbances are common in primary care and psychiatric practices. Emerging evidence points to REM sleep behavior disorder as one of the most important sleep disorders to identify given its serious implications. The most characteristic symptom of REM sleep behavior disorder is the elaboration of complex behaviors related to patients acting out their dreams as a result of the loss of normal atonia during REM sleep. These behaviors can be violent and/or dangerous, presenting potential risks to both the patient and his or her bed partner. Furthermore, there is an intriguing and emerging link between REM sleep behavior disorder and the synucleinopathies.

REM sleep behavior disorder was first identified in humans in 1986 in five patients in Minnesota who were found to have similar behavior disturbances during REM sleep (1). These five patients, (four elderly men and one woman) all inflicted injuries on themselves and their spouses during REM sleep. Since that time, much has been learned about REM sleep behavior disorder, but it remains a relatively cryptic disorder.

Classification

During normal REM sleep, there is complete, or near complete, atonia on electromyogram (EMG) by way of active inhibition of muscle neurons. REM sleep without atonia is defined by increased EMG tone during REM sleep (2). Dream enactment behavior is seen in REM sleep behavior disorder, but it is not exclusive to REM sleep behavior disorder. Other disorders that have aspects of dream enactment behavior include untreated obstructive sleep apnea, sleep terrors, withdrawal from alcohol or other drugs, and epilepsy (2). REM sleep behavior disorder is only diagnosed in the presence of both REM sleep without atonia and dream enactment behavior.

REM sleep behavior disorder can be classified as idiopathic or secondary. Idiopathic

REM sleep behavior disorder presents as an isolated phenomenon (3). Secondary REM sleep behavior disorder is associated with other neurological or general medical conditions (3).

Clinical Features of REM Sleep Behavior Disorder

The three primary aspects of REM sleep behavior disorder are altered dream mentation, abnormal vocalizations, and abnormal motor behavior (2).

Altered Dream Mentation

In patients with REM sleep behavior disorder, vocal and abnormal behaviors mirror the dream content. The large majority of patients with this disorder recall their dreams as being distressing in nature. They often report a dream in which they need to defend themselves from some type of assault. While most people can recall dreams for only a short period after waking, patients suffering from REM sleep behavior disorder can often recall their dreams for several days, and for some, up to several years (2).

Abnormal Vocalizations

While vocalizing during REM and non-REM sleep is not uncommon, the abnormal vocalizations in REM sleep behavior disorder often include shouting, screaming, and swearing (2). These vocalizations are described as being very different from the patient's normal speech during wakefulness. Additionally, there are cases of REM sleep behavior disorder in which patients have very distinct and seemingly purposeful conversations. For example, in one reported case, a 66-year-old man had a very detailed vocalization involving negotiating prices, mimicking the actions of folding garments, and pointing to different objects. He would later state that he was dreaming of selling fabrics, which was his daily business (4).

Abnormal Motor Behavior

While infrequent limb jerks and movements are common in normal sleep, movements in REM sleep behavior disorder often appear purposeful and more dramatic (2). These abnormal movements range from nonviolent activity, such as coitus-like pelvic thrusting, eating, drinking, urinating, and defecating (4), to violent movements, such as punching, kicking, flailing, running, or jumping out of bed (5). Patients will later report that the dreams were closely related to those movements. A dramatic example is the report of a 69-year-old female patient grabbing her husband's head during sleep. During interview, she stated that she was having a dream about picking apples (4). In another reported case, a patient fell down and exhibited kicking and punching behaviors. He would later remember that he was dreaming of fighting animals in a cave. These two distinct cases illustrate the fact that even if the behaviors are not related to violent dreams, injury can occur. Indeed, serious injuries have been reported, including bruises, pulled hair, limb fractures, and subdural hematomas (2).

Prevalence/Epidemiology

The epidemiology of REM sleep behavior disorder is still under investigation. However, the overall prevalence rate of the disorder has been estimated to be between 0.38% and 0.5% (5). It appears that there is a predominance of men diagnosed with the disorder. However, there is some question whether this is due to true biological factors or to a selection bias (e.g., men may have more violent dreams/behaviors that come to the attention of providers more often) (5). The age of onset for idiopathic REM sleep behavior disorder typically is in the range of 50–70 years old. Before the age of 50, men and women are equally likely to develop REM sleep behavior disorder. Those that are diagnosed at younger ages usually have coexisting narcolepsy (2). A recent study suggests that autoimmune

diseases are common in women (20%) diagnosed with REM sleep behavior disorder (6). Many patients diagnosed with idiopathic REM sleep behavior disorder at a younger age may eventually develop a neurodegenerative disease. Within an average of 7–13 years following a diagnosis of REM sleep behavior disorder, 38%–65% of patients will develop a neurodegenerative disease, typically a synucleinopathy such as dementia with Lewy bodies, Parkinson's disease, or multiple-system atrophy (5). However, published data regarding the prevalence of REM sleep behavior disorder in neurodegenerative diseases are mostly based on convenience samples and may not be truly representative.

Diagnosis

The diagnostic criteria for REM sleep behavior disorder, as described in *The International Classification of Sleep Disorders, 2nd edition* (7), are summarized below:

- 1) Presence of REM sleep without atonia on polysomnogram;
- 2) At least one of the following: sleep-related injurious, potentially injurious, or disruptive behaviors by history (i.e., dream enactment behavior) and/or abnormal REM sleep behavior documented during sleep polysomnographic monitoring;
- 3) Absence of EEG epileptiform activity during REM sleep unless REM sleep behavior disorder can be clearly distinguished from any concurrent REM sleep-related seizure disorder; and
- 4) The behaviors are not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder.

Features of Polysomnogram in REM Sleep Behavior Disorder

In normal REM sleep, one would expect rapid eye movements, mixed alpha and theta activity on EEG, and minimal to no EMG tone. Electrophysiologic findings, including pathologic accentuation of transient muscle activity along with history of characteristic abnormal sleep

behaviors, establish the diagnosis. However, due to the lack of access to sleep laboratories in many parts of the world, questionnaires, such as the Mayo Sleep Questionnaire and the REM Sleep Behavior Disorder Screening Questionnaire, can help to establish a possible diagnosis in the absence of polysomnogram (2).

Differential Diagnosis

Many disorders can have dream enactment behavior and REM sleep without atonia. A comprehensive history taken from both the patient and the patient's bed partner (if one is available) is the first step in ruling out several diagnoses.

The differential diagnosis for REM sleep behavior disorder includes non-REM parasomnias, such as somnambulism, obstructive sleep apnea with confusional arousals, and night terrors. It also includes nocturnal panic attacks, nocturnal wandering associated with dementia, seizures, acute and subacute brain lesions such as cerebrovascular accidents (localized to the pontomedullary areas) (5), demyelinating disorders, and tumors. It has also been seen in CNS diseases such as encephalitis and Guillain-Barré syndrome. Withdrawal from alcohol and benzodiazepines can cause REM sleep behavior disorder (3), and exacerbation of the disorder has been linked to chocolate (8).

As the name suggests, the timing of REM sleep behavior disorder episodes correlates to when the patient is in REM sleep. Since REM sleep usually occurs in the latter third phase of sleep, episodes are more common in the early morning hours. Two important exceptions to this are patients who have REM sleep behavior disorder related to narcolepsy or increased sleep drive (sleep deprivation and untreated sleep apnea). Patients with REM sleep behavior disorder related to narcolepsy tend to enter REM sleep within an hour of sleep onset, resulting in REM sleep behavior disorder episodes throughout the sleep period (2). Patients with high sleep drive also enter REM earlier, manifested by REM sleep behavior disorder episodes earlier in the sleep period. Patients who are untreated can have multiple episodes on a nightly

basis, whereas others may only have one episode per month.

Effect of Psychiatric Medication on REM Sleep Behavior Disorder

To our knowledge, there are a total of 168 patients in 14 reports who have developed what may be acute iatrogenic REM sleep behavior disorder as the result of taking tricyclic antidepressants, selective serotonin reuptake inhibitors, and/or selective serotonin and norepinephrine reuptake inhibitors (9). This has led to speculation that serotonin plays a role in the pathophysiologic process at some level. Another interpretation could be that these serotonergic medications do not actually induce REM sleep behavior disorder but rather unmask what will eventually become REM sleep behavior disorder at a later time point. Either way, in treating patients with depression who also have REM sleep behavior disorder, it may be prudent to use agents with nonserotonergic properties, such as bupropion.

Treatment Options

When treating REM sleep behavior disorder, the goal should be to minimize symptoms and reduce risk of injury. It is important to discuss treatment with both the patient and the patient's bed partner. First steps include moving sharp objects; reorganizing bedroom furniture to decrease the chance of falls; placing a mattress or cushion next to the bed; using a protective barrier on the side of the bed to decrease the risk of falling out of bed; and using a barrier to separate the patient from the bed partner.

Medication can be useful in treating the behaviors. The two most commonly recommended medications in REM sleep behavior disorder are melatonin and clonazepam. A trial of clonazepam (0.25 mg–2 mg) is often cited as a first-line treatment for those patients who do not have a contraindication to its use (obstructive sleep apnea, increase risk of falls, cognitive impairment) (2, 10). For those patients who continue to have REM sleep behavior disorder or have one of

the mentioned contraindications, a trial of melatonin up to 2 mg is appropriate. Other medications that have been used to treat this disorder include donepezil, rivastigmine, pramipexole, sodium oxybate, and levodopa (2, 5, 10).

Conclusions

Restoration of high-quality sleep is always a high priority in the treatment setting. REM sleep behavior disorder is not the most common cause of poor sleep in our patients; however, it is a crucial disorder to recognize and treat. It is also important to remember the potential future implications for patients affected by this illness, since many of them will go on to develop a synucleinopathy.

Dr. Bruce is a fourth-year resident, and Dr. Madan is a fourth-year resident, in the Department of Psychiatry, Creighton University, Omaha, Neb.

The authors thank William Burke, M.D., and William Roccaforte, M.D., for their guidance and assistance in preparing this article.

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New Feature! Residents’ Journal Book Forum

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Interested parties who are available to read one of the three books listed below and write a review (500 words) within 8 weeks should contact Rajiv Radhakrishnan, M.B.B.S., M.D., Senior Deputy Editor (rajiv.radhakrishnan@yale.edu).

The deadline to submit a review is November 3, 2014.

Weary Warriors: Power, Knowledge, and the Invisible Wounds of Soldiers

by Pamela Moss and Michael J. Prince

Classifying Psychopathology: Mental Kinds and Natural Kinds

edited Harold Kincaid and Jacqueline A. Sullivan

Study Guide to Psychiatry: A Companion to The American Psychiatric Publishing Textbook of Psychiatry, 6th ed.

by Philip R. Muskin, M.D.

**Selected reviewers will be permitted to retain the books for their own use.*

Pharmacologic Management of Agitation and Aggression in Patients With Traumatic Brain Injury

Arundhati Undurti, M.D., Ph.D.

Traumatic brain injury (TBI) is caused by blunt force trauma, acceleration/deceleration injury, or by penetrating head injury. This type of injury disrupts the normal functioning of the brain and can lead to changes in memory, cognition, mood, and behavior. The severity can range from mild to severe TBI. A mild TBI or concussion is defined as a temporary change in neurological functioning and can affect mood, cognition, and memory. A severe TBI involves loss of consciousness for greater than 30 minutes or altered mental status for greater than 24 hours. Altered mental status is characterized by confusion, disorientation, amnesia, agitation, and perceptual disturbances, and this constellation of findings is also referred to as posttraumatic amnesia (1). According to the Centers for Disease Control and Prevention, there are 1.7 million new TBIs annually, and TBIs contribute to 30% of all injury-related deaths in the United States. While falls are the leading cause of TBI, motor vehicle accidents are the leading cause of death from TBI (2). TBI is also a growing concern in the military, with mild TBI seen in 20% of soldiers returning from Iraq or Afghanistan (3).

Neurocognitive sequelae of TBI depend on age, baseline cognitive functioning before the injury, extent of the injury, and concurrent injuries. In 80%–85% of patients who suffer from mild TBI, cognitive deficits, typically in attention and memory, resolve within 3–6 months. Those who suffer from severe TBI can have long-lasting impairment in memory, awareness, language, and visuospatial processing (1). Executive dysfunction is common after TBI, since the frontal lobes are particularly vulnerable to injury. Executive dysfunction involves impairment in recall, planning, judgment, motivation, and impulsivity. Patients with TBI have increased dif-

ficulty with organizing memory rather than with forming new memories (4). Apart from neurocognitive sequelae, TBI patients also report disturbances in energy and sleep, and as many as 10%–70% of patients report depressive symptoms (5). Furthermore, posttraumatic stress

Agitation and aggression are perhaps some of the most distressing consequences of TBI, and both pharmacologic and nonpharmacologic strategies are crucial.

disorder (PTSD) is a common co-occurring disorder with TBI (6). Perhaps the most distressing symptom after TBI is agitation and aggression. According to a Cochrane review, long-term aggression (60 months after injury) continues in approximately 25% of TBI patients who are discharged from a rehabilitation facility (7). The terms agitation and aggression are often used interchangeably, but TBI researchers distinguish between the two. Agitation occurs primarily in the setting of posttraumatic amnesia. Aggression, on the other hand, occurs in the later stages of recovery and is characterized by irritability, hostility, and violent or assaultive behavior (7). There is no evidence to suggest that pharmacologic management of agitation should differ from that of ag-

gression, and the present review does not differentiate between the two.

Evaluation of Agitation in TBI

It is important to evaluate for additional causes of agitation, including delirium, Wernicke-Korsakoff syndrome, akathisia, increased intracranial pressure, and agitation secondary to PTSD, psychosis, or other psychiatric disorders. Initial assessment includes an assessment of the severity of TBI (loss of consciousness, neurological deficits), of premorbid level of functioning, and of substance use to rule out intoxication and withdrawal syndromes, as well as review of current medications. Screening laboratory tests are important to assess for metabolic abnormalities as well as infection. Imaging (head CT/brain MRI) should be obtained if indicated.

Management of Agitation and Aggression in TBI

If identifiable, the primary cause (metabolic abnormalities, infection, substance withdrawal) should be treated.

Nonpharmacologic Management

Delirium prevention precautions, such as frequent orientation, minimizing disturbances during the night by clustering care, opening the blinds during the day, and regular toileting schedules, are very important. Structured behavior programs are also efficacious, which consist of providing information to patients and their families about their cognitive status, as well as improving self-awareness (8).

Pharmacologic Management

All studies referenced in this review have examined the efficacy of pharmacologic interventions to treat agitation and aggression specifically in TBI patients.

A young man sustains traumatic brain injury (TBI) in a motor vehicle accident.

A 39-year-old man with no previous psychiatric history is admitted after sustaining subarachnoid and intraventricular hemorrhage in a motor-vehicle accident. The patient sustained loss of consciousness for greater than 90 minutes. At the time of psychiatry consult, his care was disrupted by frequent agitation, characterized by attempts to remove his intravenous line, cervical collar, and G-tube. Upon initial evaluation, he was not oriented to place or time and had significant expressive and receptive aphasia. His vital signs and results of a physical examination and screening laboratory tests did not show evidence of infection. A urine toxicology screen was negative. The patient's agitation was considered likely secondary to delirium from TBI, and quetiapine (25 mg b.i.d.) was scheduled after ensuring that his QTc was normal. The consult service also worked with nursing staff to create a behavioral care plan to prevent and manage his agitation. Over the next 2 months, the patient's quetiapine dosage was gradually titrated upward with improvement in agitation. At discharge, the daily quetiapine dose was 450 mg, and the patient successfully transitioned to a rehabilitation facility.

Antipsychotics. Antipsychotics are perhaps the most frequently used medications to control agitation and aggression. While there is some concern that antipsychotics may prolong recovery and rehabilitation, the general consensus is that antipsychotics are effective in managing agitation and aggression (9). Haldol is the most commonly used typical antipsychotic, and the availability of an intravenous formulation makes its use convenient in the acute setting. Dosage varies depending on the patient's age, comorbid medical conditions, and previous exposure to antipsychotics, but typical doses range from 2 mg to 15 mg daily. Side effects include development of extrapyramidal symptoms, neuroleptic malignant syndrome, and prolongation of QTc. Atypical antipsychotics, such as olanzapine, quetiapine, and risperidone, are also used, and while clinical experience suggests that they can be effective in controlling agitation and aggression, evidence in the literature remains weak. It is important to frequently reassess whether a patient requires antipsychotics and to attempt to discontinue the medication as early as feasible.

Mood stabilizers. Good evidence exists for valproic acid and carbamazepine. Retrospective chart review and clinical experience suggest that valproic acid may be considered as a frontline agent for treatment of agitation and aggression with additional benefit of seizure prophylaxis (10). Its onset of action is rapid (typically within 7 days), and common side effects include sedation, nausea, weight gain, and hair loss. The most serious side effects include hepatitis, pan-

creatitis, and thrombocytopenia, and it is important to monitor for this on a regular basis. Doses up to 2,500 mg daily have been used successfully. Regarding use of carbamazepine, evidence from a prospective open trial and several case reports suggests that it improves agitation and aggression at daily doses ranging from 300 mg to 800 mg (11). Rare but serious side effects include hepatitis, severe bone marrow suppression, and Stevens-Johnson syndrome. Case studies suggest that lithium can also be used to treat aggression at serum levels of 0.6–1.0 (11). Side effects from lithium include gastrointestinal distress, sedation, cognitive dulling, and tremor. Generally, TBI patients may be more vulnerable to the CNS effects of lithium, making this a less attractive option.

Beta blockers. The mechanism of action of beta blockers, such as propranolol, is poorly understood, although a dampening of the autonomic response is thought to contribute (12). A randomized double-blind placebo-controlled trial showed that propranolol reduced the intensity of agitation but not necessarily the frequency. The starting dose of propranolol is typically 60 mg daily and can be increased by 60 mg every 3 days, not to exceed a maximum daily dose of 420 mg (12). Side effects can include hypotension, bradycardia, sedation, and depression.

Benzodiazepines. The mechanism of action of benzodiazepines is by increasing activity at the GABA receptor. The general consensus is that benzodiazepine use be limited to episodes of acute agita-

tion. Long-term use is not recommended given their effects on cognition and other side effects, such as respiratory depression, anterograde amnesia, disinhibition, paradoxical agitation, tolerance, and dependence (11, 13).

Selective serotonin reuptake inhibitors (SSRIs). SSRIs are primarily used to treat depression, although some studies have suggested improvements in agitation and aggression as well. In an open clinical trial, citalopram was combined with carbamazepine to successfully treat depression, as well as behavioral disturbance (14). The data with sertraline are mixed. One prospective placebo-controlled randomized double-blind study found no significant benefit of sertraline in TBI patients, while a nonblinded study found that sertraline decreased agitation in patients within 1 week of treatment (13). The rapid response to sertraline in the latter study suggests that the mechanism of action may be distinct from that involved in its antidepressant action. The dose of sertraline in that study was 50 mg/day–200 mg/day. A confounding factor in both studies was the small sample size. Side effects from antidepressants include headache, nausea, and sexual dysfunction.

Neurostimulants. There is evidence from a randomized double-blind placebo-controlled trial to suggest that amantadine at daily doses up to 300 mg can be efficacious in the management of agitation and aggression (11). It may also enhance cognitive functioning (15), thus making it an attractive candidate for TBI patients.

Conclusions

The neuropsychiatric consequences of TBI include problems with memory, executive functioning, mood, and impulse control. Agitation and aggression are perhaps some of the most distressing consequences of TBI, and both pharmacologic and nonpharmacologic strategies are crucial. Further research is required to assess the efficacy of pharmacologic treatments and to identify which agents are most successful in controlling symptoms with minimal side effects.

Dr. Undurti is a third-year resident in the Department of Psychiatry, University of Washington Medical Center, Seattle.

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Preoperative Dose of Olanzapine in the Prevention of Postoperative Delirium

Kristin V. Escamilla, M.D.
Isela A. Werchan, M.D.

Delirium is defined as impaired attention, with changes in cognition or perceptual disturbances. Postoperative delirium or confusion has been reported to range from 8% to 78% (1). Pandharipande et al. (2) found the prevalence of delirium to be 73% in a surgical population, with an average duration of 3 days. Overall, delirium is a strong predictor of longer hospitalization, mechanical ventilation use, and increased mortality (3). This condition is a growing burden on the cost of health care, with estimates ranging from \$38 billion to \$152 billion per year in the United States alone (4). In a study conducted by Franco et al. (5), multiple expenditures were higher for patients with delirium, including technical, nursing, pathology, medication, and medical professional costs.

While antipsychotics are well known to treat delirium, using them perioperatively for prevention is increasingly being reported in the literature. Larsen et al. (4) conducted a large double-blind placebo-controlled trial in which 5 mg of olanzapine was administered to elderly patients pre- and postsurgery for joint replacement and found that olanzapine lowered the incidence of delirium but not its severity or duration. A 2005 randomized controlled trial showed that 1.5 mg of haloperidol did not decrease the incidence of delirium, but it did decrease the severity and duration of delirium and reduced the total number of hospital days, without any major side effects (6). A second trial published in 2008 confirmed these findings using 0.5 mg t.i.d. of haloperidol (7). Additionally, Kaneko et al. (8) examined the use of postoperative haloperidol in patients undergoing gastrointestinal surgery and found that it significantly decreased the incidence of postoperative delirium. A recent meta-analysis on antipsychotic prophylaxis in

at-risk elderly patients indicated that there was a 50% reduction in the relative risk of delirium among those receiving treatment (9).

We report the case of a nongeriatric patient who had several episodes of delirium after multiple hand reconstructive surgeries, until a 10-mg preoperative dose of olanzapine was administered before her last surgery.

Case

“Ms. D” is a 47-year-old Caucasian woman who was admitted to the trauma service at our hospital after experiencing left-hand third-, fourth-, and fifth-digit amputations from a rollover motor-vehicle collision. The patient’s medical history included asthma, diabetes mellitus type 2, gastroesophageal reflux disease, hypertension, migraines, peripheral neuropathy, arthritis, and adverse reaction to anesthesia with past procedures. She was receiving pregabalin, budesonide, verapamil, rosuvastatin, lidocaine patch, and metformin. Her past psychiatric history included nonspecific anxiety disorder, without mania, psychosis, or substance abuse. Our psychiatry service was consulted after she awoke from surgery extremely agitated and disoriented.

Throughout her hospitalization, the patient underwent seven surgeries to repair her injured hand. After each procedure using general anesthesia, she awoke delirious, requiring multiple, minimally effective antipsychotics (as occasion required). She occasionally emerged from anesthesia combative, biting staff, and pulling out her Foley catheter, posing not only a risk to herself but to others.

Prior to the patient’s final hand surgery under general anesthesia, we decided to use a preoperative dose of olanzapine to prevent her recurring postoperative

delirium. Three hours before her final procedure, the patient was given 10 mg of olanzapine, instead of an alternate antipsychotic, because of its efficacy in treating delirium (4) and because she had tolerated previous doses well. Immediately postsurgery, the patient was noted to be alert and oriented to person, place, time, and situation. She did not present with symptoms of delirium or agitation in the following 6 days and was successfully discharged.

Discussion

Based on our experience with the patient discussed in the present case, using a preoperative dose of an antipsychotic such as olanzapine seemed to reduce the incidence of delirium postoperatively, which corroborates previous findings from research on this topic. It further reinforces the need to conduct more studies, preferably randomized controlled trials because current trials have indicated conflicting outcomes, with some showing decreased incidence, severity, and duration of delirium and others showing no significant change. Clarifying these outcomes would be helpful in determining how delirium can be treated and, more importantly, prevented.

Based on the present case report, it appears that there are several risk factors that may assist in identifying which patients will benefit from a preoperative intervention. Throughout her hospital stay, our patient was also being prescribed opiates and benzodiazepines, which are known risk factors for delirium and could have contributed to her postoperative delirium. It is helpful to monitor medications in this at-risk population because virtually every surgical patient is prescribed opiates. A concomitant anxiety disorder, which our patient had, may also serve as a risk factor, as evidenced by

the Kimball study on delirium risk factors for open-heart surgery (10). Additionally, our patient had a history of prior delirio-genic episodes after anesthesia; therefore, if a patient presents with a history such as this, consideration of dosing an anti-psychotic before surgery should be made.

Although the bulk of the literature today is based on the geriatric population, it ap-pears that a younger population may also respond very well to this type of preven-tive measure. Therefore, future research and preventive treatment should also be targeted toward younger patients with a history of postoperative delirium, con-comitant benzodiazepine and narcotic use, and a history of anxiety disorders.

The limitations of our case report further highlight the need for future research that is much more controlled and uses measures to examine efficacy. Because this is a post hoc case report, our patient's mental status examination was subjective and not evaluated with any quantitative objective measures. Hours before the last procedure, we decided to decrease her ex-posure to narcotics and benzodiazepines because these agents are known to be de-liriogenic, which could have affected the outcome itself.

Preventing delirium after this last proce-dure allowed for a safer and faster recovery for our patient, suggesting that prophylaxis with an atypical antipsychotic like olanzapine could be an effective and eas-ily employed primary prevention strategy targeting certain at-risk populations, such as those with pre-existing mental illness, prior history of delirium, and concurrent exposure to narcotics and benzodiaz-

epine. We hope that further research in this area will consist of randomized con-trolled trials that focus on nongeriatric cohorts, controlling for deliriogenic med-ications, evaluating typical versus atypical antipsychotic efficacy, and using objective measures.

Even one episode of delirium can cause increased mortality, morbidity, length of hospital stay, health care costs, and symp-toms that possibly persist up to a month later. Emphasis should be placed on pre-venting delirium, not just on treating it (9). Antipsychotics appear to be an excel-lent option for doing so, especially since they have been shown to result in only mild side effects in prophylaxis for de-lirium (9).

Dr. Escamilla is a first-year fellow at the University of Texas Southwestern Psycho-somatic Medicine Fellowship Program, and Dr. Werchan is a third-year resident at the University of Texas Southwestern Psychia-try Residency Program in Austin, Tex.

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Successful Continuation of an ECT Course After a Tardive Spontaneous Seizure

Maria J. Julios Costa, M.D.

ECT was first administered more than 70 years ago, and it is still the single most effective intervention for severe depression (1). ECT is considered a safe treatment, with a low mortality estimated at 1–2 deaths per 100,000 sessions, or approximately 1 death for every 10,000 patients treated (2).

Although uncommon, adverse events such as prolonged seizures, tardive spontaneous seizures, and status epilepticus may occur during a course of ECT (2). We report a case of tardive spontaneous seizure and the successful continuation of ECT in a patient with major depressive disorder without a history of epilepsy. This report is aimed to add to the limited available literature on what is the best course of action when these complications arise so that physicians can recognize and treat such events that could otherwise lead to increased ECT-related morbidity and mortality.

Case

“Mr. M” is a 39-year-old Caucasian man with a history of recurrent major depressive disorder with severe current major depressive episode (defined as a Beck Depression Inventory-II [BDI-II] scale score of 38, range: 0–63 [a score ≥ 30 is considered severe depression]) and unspecified anxiety. His past psychiatric history was notable for a treatment-resistant course (prior adequate medication trials of sertraline, fluoxetine, paroxetine, and bupropion were without benefit). His first ECT course (15 sessions of unilateral ECT) was successfully administered at age 36 without complications in the context of severe depressive episode with psychosis. The patient had no history of non-ECT-related seizures, epilepsy, or febrile seizures, and he was taking duloxetine, clonazepam, and aripiprazole when he began receiving ECT for current major depressive episodes.

The patient’s pre-ECT evaluation included a normal physical examination, ECG, and routine laboratory tests. A MECTA Spectrum 5000 Q device (MECTA Corp., Tualatin, Ore.) was used to deliver right unilateral (according to d’Elia electrode placement) ultra-brief pulse stimulation. Methohexital (150 mg), succinylcholine (100 mg), ketorolac (30 mg), and glycopyrrolate (0.2 mg) were administered during the first session (with the dosage based on the patient’s weight). Stimulation (at settings of 0.30-msec pulse width, 60-Hz frequency, 1.5-second stimulus duration, and 800 mA of current) achieved an adequate seizure in quality and duration (synchronic EEG epileptiform activity with a duration of 38 seconds peripherally and centrally) without complications.

Vitals before the second ECT session were reported as normal (blood pressure: 111 mmHg/73 mmHg; heart rate: 60 beats/minute; and O₂ saturation on room air of 97%). The patient received the same pre-procedure medications except for methohexital, which was reduced from 150 mg to 120 mg. Settings were increased as indicated for a second session with right unilateral (d’Elia) electrode placement (0.30 msec-pulse width, 120-Hz frequency, 2-second duration, and 800 mA current). A 47-second peripheral seizure and a 59-second central seizure were induced without intraprocedure complications. Seizure termination was confirmed with bifrontal EEG. The patient regained consciousness and was transferred to recovery in stable condition. Approximately 30 minutes after the procedure ended, he developed a generalized tonic-clonic seizure with incontinence that lasted 120 seconds. His seizure activity subsided spontaneously, and he remained slightly confused, with a mild and transient headache.

A multidisciplinary evaluation, including a neurology consult, was obtained. Results from the patient’s mental status evaluation, physical examination, laboratory tests (including complete blood count and basic metabolic panel) and imaging studies (CT and MRI with and without contrast) were all within normal limits. An EEG while the patient was awake revealed a normal posterior dominant rhythm with a slightly abnormal record due to delta slowing over the left posterior temporal region that was considered to be of unclear significance. No epileptiform discharges or seizures were recorded. It was concluded that although seizure activity was likely related to ECT, this treatment could be continued safely because no abnormalities were found on the physical examination, laboratory results, or brain imaging. The patient agreed to continue the ECT sessions given the severity of his depression and history of previous response to ECT. Multidisciplinary consensus was to continue the index ECT course with administration of lorazepam (2 mg intravenously) immediately after termination of each seizure induced by ECT.

For the patient’s third session, methohexital was increased to 200 mg, and along with the same previous dose of succinylcholine (120 mg), 30 mg of ketorolac and 0.2 mg of glycopyrrolate were administered. Right unilateral electrode placement and stimulus settings were identical to the second treatment, and a prolonged seizure was induced with ECT. Lorazepam (2 mg intravenous) was administered approximately 2 minutes into the seizure given the patient’s history of prolonged ECT-induced seizure duration. Although our initial plan was to administer lorazepam immediately after termination of each ECT-induced seizure, at this point we were forced to administer the medication before the

seizure ended spontaneously. Status epilepticus was ruled out by EEG.

Continuation of the index course of ECT with postictal administration of lorazepam (2 mg) was still favored given the severity of the patient's symptoms and the potential for further improvement of his depression with ECT (mild improvement was evident after session 3).

In light of the above-mentioned complications and trying to avoid a new prolonged ECT-induced seizure, both the frequency and duration of stimuli were decreased to 60 Hz and 1.5 seconds, respectively, for the fourth ECT session. Although this change in settings may seem drastic, it was considered adequate in the context of a prolonged seizure in a patient who had already experienced two different seizure-related complications but had a documented adequate seizure duration on the proposed settings (i.e., well above 25 seconds). With these settings, an adequate seizure (synchronic EEG epileptiform activity with a duration of 23 seconds peripherally and 66 seconds centrally) was achieved. Postictal suppression and degree of muscle relaxation were considered excellent, and no intraprocedure or postictal complications were reported.

The patient received a total of 22 ECT sessions (10 right unilateral, five bifrontal, and seven bitemporal electrode placements) without further prolonged or tardive spontaneous seizures. He was able to continue the ECT course with no further complications as long as he received intravenous prophylactic lorazepam upon termination of each ECT-induced seizure. His depressive symptoms markedly improved after session 22 (BDI-II score of 12), and an office follow-up 1 month after the course was completed showed sustained benefit, with a BDI-II score of 7.

Discussion

Our patient experienced a prolonged seizure during the fourth session. ECT-related prolonged seizures are defined as seizures lasting longer than 3 minutes by motor or EEG manifestations (3–5). The incidence ranges from 1% to 2% (6). The risk of developing prolonged seizures

increases with the concomitant use of medications (such as theophylline, lithium, or trazodone) or stimulants such as caffeine, as well as with the presence of comorbid medical conditions that lower seizure threshold (e.g., electrolyte disturbances) (3–5). Prolonged seizures may result in increased postictal confusion and memory impairment (3, 7) and can progress to status epilepticus (8, 9) if not managed promptly. Prolonged seizures can be terminated by intravenous administration of anticonvulsant medications, including benzodiazepines (e.g., midazolam, lorazepam), or a repeat dose of an anesthetic agent (e.g., methohexital) (10).

Our patient also experienced a tardive spontaneous seizure after the second session. Tardive spontaneous seizures have been described as seizures occurring minutes, hours, or even months after an ECT-induced seizure has terminated (11). Tardive spontaneous seizures are fortunately rare in ECT practice, and no causal relationship between ECT and the development of epilepsy has been identified (12–15). Literature on this topic is particularly scarce, and no consensus exists when considering the use of ECT in patients with a history of ECT-induced tardive spontaneous seizures.

Ongoing use of anticonvulsants during a course of convulsive therapy is counterintuitive, and they are generally tapered off or discontinued before starting an ECT course (16). This is done not only because of possible interactions with anesthetic agents and muscle relaxants but also because of the potential for decreasing ECT's therapeutic effectiveness by altering the convulsive threshold and seizure duration (17). However, this remains a controversial issue. ECT has been safely administered to patients treated with various anticonvulsant drugs, and adequate seizures can be obtained, although occasional dose reduction may be required (17, 18).

Both prolonged seizures and tardive spontaneous seizures in the context of ECT should be treated expeditiously with anticonvulsive medication. In the event of tardive spontaneous seizures, a prompt interdisciplinary approach, including a neurology consultation, must

take place. The evaluation should include assessment of risk factors (e.g., family history of epilepsy, medications that lower seizure threshold, and past brain injuries), EEG, brain imaging, and blood work (e.g., electrolytes, complete blood count, blood glucose) in order to diagnose, evaluate potential causes, and assess the safety of ECT continuation.

As shown in the above case, as well as in available evidence, even in the presence of prolonged seizures or tardive spontaneous seizures, ECT can be safely delivered with modifications to the standard procedure so as to avoid new complications without compromising its effectiveness. For tardive spontaneous seizures that occur soon after administration of ECT, we recommend use of prophylactic, intravenous benzodiazepine (midazolam or lorazepam) after each ECT session in order to prevent delayed seizure activity and to allow for continuation of ECT. However, these recommendations may not be generalized to certain populations, including older patients or patients with significant comorbid medical conditions.

Dr. Julios Costa is a third-year resident in the Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, Philadelphia.

The author thanks Mario A. Cristancho, M.D., Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, John P. O'Reardon, M.D., Department of Psychiatry at the University of Medicine and Dentistry of New Jersey, Newark, N.J., and Mahendra T. Bhati, M.D., Associate Medical Director, Department of Psychiatry, University of Pennsylvania.

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**Please direct all inquiries to Rajiv Radhakrishnan, M.B.B.S., M.D., Senior Deputy Editor (rajiv.radhakrishnan@yale.edu).*

On Becoming a Local Healer of Global Mental Health Problems

Suni Jani, M.D., M.P.H.

This was a standard, unpredictable international trip. Twenty-seven hours of travel, a jilted circadian clock, and a landing in one city where we discovered that the connecting flight for the city we were meant to be in was 3 hours later in an airport across town. There were reunions with families and weddings to attend, and I was not sure how I would survive such a full schedule. Hours later at nightfall, just as I thought I would experience a full night of horizontal sleep, my mother woke me up. It was a medical emergency, she said. I had to leave our hotel and take a cab three boroughs away immediately.

“But, I’m a psychiatry resident,” I said. “We tell suicidal people to go to the emergency room.” “This is India, where you do not tell people that,” my mother admonished. I rushed over to discover 10 families huddled around my mother who directed me to a chair next to her.

This was not an emergency in the traditional sense, but we were in town for the night, while these families had lived here for generations and were in desperate need of a psychiatrist. They all had children with intellectual disabilities and developmental disorders that had prevented them from enrolling or remaining in standard education or vocational training. Each family told a distressing story. One man thought he would get his intellectually disabled son married so he would have a permanent caretaker. He gave a substantial dowry to a family, and on the wedding night, the girl discovered that the son was “off” and ran away. Another mother described witnessing countless seizures that prevented her child from ever going to school. Unlike thousands of families with disabled children living in the slums in India, these families were fortunate in the fact that they were reasonably wealthy and had befriended several artists who had organized a grassroots campaign to teach

basic vocational skills to their children. Now they had reached a collective block: What happens after we die? What happens to the next generation who do not have our resources? The families wanted

Participating in global outreach efforts ... requires empathy, compassion, and a willingness to seek out the underserved.

advice from a doctor who was working with the American health system on how to create an evidence-based vocational rehabilitation program for their children. This experience and others enabled me to gain more understanding of the unmet needs of this vulnerable population.

I asked about the local government and their understanding of the impact of poor mental health care on the economy, explaining that the cumulative global impact of mental disorders in terms of lost economic output is estimated to be \$16.3 million (U.S. dollars) between 2011 and 2030 (1). I imagined this was a solid argument for the school system in this neighborhood to have an individual educational program with outcome reviews through formal evidence-based testing. These ideas were met with blank stares and very little hope.

This is the state of global psychiatry, where untreated mental disorders with an ineffective treatment infrastructure

frequently result in impoverishment, homelessness, and occupational and legal problems. Even for wealthier families like the ones surrounding my mother and I in Vadodara, it is exceptionally challenging to get assistance in helping to meet their children’s potential because treatment is stigmatized in this setting. Their children were unable to make decisions about their own treatment, vote, participate in public life, begin a family, or expect the highest attainable standard of health. In the slums outside and the world beyond, individuals with mental illness are more likely to live in unhygienic conditions and to experience physical and sexual abuse, neglect, and harmful and degrading treatment practices in health facilities (1).

This was a challenging world when I entered it 7 years ago as a Masters in Public Health student. At that time in my coursework in global mental health, the numbers and challenges of mental illness disabilities in international settings seemed overwhelming. The burden of global mental illness was a Sisyphean task, and I had no understanding of where to begin. I therefore concluded that the most hands-on way to understand the limitations of providing transcultural psychiatric aid to underserved global populations would be through my own forays into the system.

I began a 501(c)3 nonprofit organization and named it Global Health Linkages, Inc. (2). I designed an online telepsychiatry electronic medical record site that allowed for inclusion of an integrated global community and for health promotion and screening. This project remains ongoing in development as Global Health Linkages expanded into a multitude of other mental health advocacies and training work, as well as from community organizing and education to teaching grant writing and providing resources for grassroots organizations.

Resources were provided to hospitals, domestic violence shelters, and orphanages in several cities in India. In America, students would come to meetings and learn to write grants and presentations from their research of international data. It was after many trials and tribulations with Global Health Linkages that I could sit in Vadodara being slightly less terrified of what it meant to become a global psychiatrist.

As residency allows me to develop my identity as a psychiatrist, Global Health Linkages and the serendipitous encounters like the one I had in Vadodara gradually informed my understanding of global and cultural psychiatry. I have learned that participating in global

outreach efforts does not require a jet-setting lifestyle, a nonprofit, or proficiency in numerous languages. Instead, it requires empathy, compassion, and a willingness to seek out the underserved. I want readers to know that they do not have to travel in order to contribute to international mental health care. The trainees with whom I have had the privilege of working through these grassroots organizations have provided well-researched expertise to these vulnerable populations. Such local efforts can help to reduce the global burden of mental health problems.

Dr. Jani is a third-year resident in the Department of Psychiatry, Baylor College of Medicine, Houston.

The author thanks Dr. John Coverdale, as well as her parents, and Drs. Niranjani and Sushma Jani.

This year, the American Journal of Psychiatry introduced a new feature called Perspectives in Global Mental Health. See this month's column, by Sasan Vasegh, M.D., on the case of a depressed Shia Muslim woman living in a city in western Iran near the Iran-Iraq border.

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The Perks of Being a Wallflower

by Stephen Chbosky

“If you really want to understand me, watch *The Perks of Being a Wallflower*,” my adolescent patient stated when I informed her I finally watched *Girl, Interrupted*. Intrigued about her connection to this movie, I discovered it won the Teen Choice Award for best movie drama, as well as best drama actress and best drama actor. It grossed \$31,420,327 worldwide in 2013 (1, 2). I became aware of the positive regard adolescents had for this movie.

The film is set in 1990s Pittsburgh and features Charlie, a socially awkward but insightful high school freshman whose best friend, Michael, recently committed suicide. On his first day of school, he connects with his English teacher, Mr. Anderson, who mentors him through English literature. Charlie navigates his first weeks of high school quite isolated. He is befriended by two senior misfits, fearless Patrick and his beautiful stepsister Sam. Charlie falls in love with Sam but hesitates to pursue her, as she is dating a college guy. Soon he too is, reluctantly, in a relationship. Charlie is exposed to partying, alcohol, drugs, sexuality, bullying, and true friendship. He processes the thoughts and feelings these experiences evoke by writing to an unknown “friend.”

These memorable experiences, along with the realization that his friends will soon leave him to go away to college, contribute to his breakdown. Charlie’s unconscious past is uncovered as he has more frequent flashbacks. By now, both he and Sam have broken up with their respective partners, and he is able to express his love for her the night before she travels to college. Through this sexual encounter, Charlie’s repressed memories of his beloved aunt sexually abusing him are finally brought to his

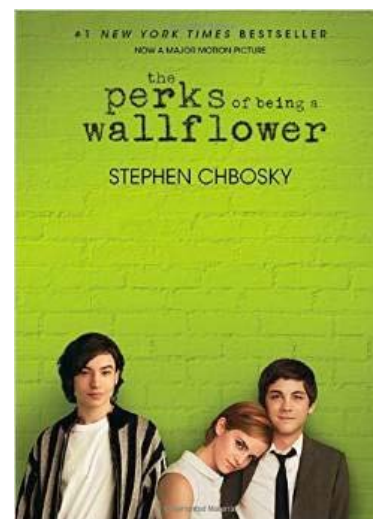
conscious mind. As a young boy, he always blamed himself for his aunt’s traumatic death. Soon after his friends move to college, he decompensates and becomes depressed and suicidal. He spends the majority of his summer in a psychiatric hospital where he is able to

This movie was unlike most of the current pop culture teenage movies because it highlighted the impact that normal adolescent development has on mental illness.

process feelings of loneliness, guilt, and confusion. He gains an understanding about his past and develops an overall stronger self-concept.

Although the story is focused on Charlie, all of the other characters also learn that “we accept the love we think we deserve.” Not obviously about mental illness, it features symptoms of depression and posttraumatic stress disorder. This movie was unlike most of the current pop culture teenage movies because it highlighted the impact that normal adolescent development has on mental illness, and vice versa. I watched this movie and marveled at how my patient was essentially

Natasha D. Smith, M.D., M.P.H.



sending me a message through a form of media. I appreciated each character and imagined which one, or ones, my patient identified with. I empathized with her struggle. I am thankful for films such as these that not only seem to reduce the growing stigma of adolescent mental illness but serve as a means of communication between myself and the population that I treat.

At the time this commentary was accepted for publication, Dr. Smith was a second-year child and adolescent psychiatry fellow at the National Capitol Consortium-Walter Reed National Military Medical Center, Child and Adolescent Psychiatry Clinic, Bethesda, Md., and a Capt. in the United States Air Force.

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Residents' Resources

We would like to welcome all our readers to this new feature of the Journal! Here we hope to highlight upcoming national opportunities for medical students and trainees to be recognized for their hard work, dedication, and scholarship.

**To contribute to the Residents' Resources feature, contact Tobias Wasser, M.D., Deputy Editor (tobias.wasser@yale.edu).*

October Deadlines

Fellowship/Award and Deadline	Organization	Brief Description	Eligibility	Contact	Website
The Group for the Advancement of Psychiatry (GAP) Fellowship Deadline: October 1, 2014	GAP	Each fellow: Attends four GAP meetings over 2 years; Becomes a member of one of the working GAP committees; Collaborates with other fellows on a plenary presentation to the general GAP membership; Learns about group process in their GAP committee and with their fellowship group; Benefits from close interaction with peers and mentors.	PGY 2 or later at an accredited psychiatry residency program in the United States or Canada; Have at least 2 years of training ahead (e.g., PGY-2 or 3, or first year of a 2-year fellowship)	frda1@airmail.net 972-613-0985	http://ourgap.org/
Geriatric Mental Health Foundation's Scholars Fund Deadline: October 1, 2014	The American Association for Geriatric Psychiatry (AAGP)	Provide trainees with the following benefits: One year of membership within the AAGP; Registration to the AAGP Annual Meeting; A travel stipend to help defray travel expenses to attend the AAGP Annual Meeting.	Medical students in an LCME or COCA accredited school; PGY 1-4 residents in ACGME or AOA accredited program	main@aagponline.org 301-654-7850	http://www.aagponline.org/index.php?src=gendocs&ref=ScholarsApply&category=Main
American Psychiatric Foundation (APF) Schizophrenia Research Fellowship Deadline: October 15, 2014	APF	A 1-year psychiatric research fellowship for three postgraduate psychiatry trainees specifically to focus on research and personal scholarship. Minimal time (less than 15%) will be devoted to teaching, patient care, consultation, or other duties. The protection of time for research should be assured by the department chairperson.	APA Resident-Fellow Member (RFM); Not already an established investigator	Marilyn King schizophrenia@psych.org 703-907-8653	http://www.psychiatry.org/researchers/research-training-and-career-distinction-awards/schizophrenia-research-fellowship
APA/Lilly Psychiatric Research Fellowship Deadline: October 15, 2014	APA	This fellowship provides funding for a postgraduate psychiatry trainee, under the supervision and guidance of his/her mentor, to design and conduct a research study on a major research topic.	APA RFM; Not already an established investigator	psychresearch@psych.org	http://www.psychiatry.org/researchers/research-training-and-career-distinction-awards/psychiatric-research-fellowship

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The American Psychiatric Association would like to invite you to attend our **APA JobCentral Virtual Career Fair!** APA JobCentral has been connecting employers and exceptional psychiatrists since 2012. By creating this virtual event, we hope to start that initial conversation between your recruiting department and psychiatrists searching for that perfect career without anyone having to leave their home or office!

Employer Benefits

As an employer, you are allowed up to four recruiters to showcase your open positions in your customized virtual booth. Your team will be able to engage in one-on-one online chats, view attendees' resumes, work histories, and view optional profile pictures. A rating system is in place for you to score your interactions and record notes about each candidate! **Early-bird discounted booths and sponsorship packages available!**

Job Seeker Benefits

As a job seeker, you will be able to interact live with recruiters and HR departments at each virtual booth. You are able to share your resume, experience, and schedule second-round interviews. By learning about all of the new opportunities available in one 3 hour time slot, you increase your job pool as you continue on the search for your first or next career opportunity!

For participating in our virtual fair, job-seekers will receive a 20% discount on any APP product! A coupon code will be emailed at the conclusion of the fair with instructions for use.

With APA JobCentral, job seeker registration is and always will be FREE.

For detailed information on how you can register as an employer, contact Eamon Wood at ewood@pminy.com or 212-904-0363. **If you are a job seeker, please visit APA JobCentral's web page at jobs.psychiatry.org for more information.**



jobs.psychiatry.org

Author Information for *The Residents' Journal* Submissions

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Misty Richards, M.D., M.S.
(UCLA)

Senior Deputy Editor

Rajiv Radhakrishnan, M.B.B.S., M.D.
(Yale)

Deputy Editor

Tobias Wasser, M.D.
(Yale)

The Residents' Journal accepts manuscripts authored by medical students, resident physicians, and fellows; manuscripts authored by members of faculty cannot be accepted. To submit a manuscript, please visit <http://mc.manuscriptcentral.com/appi-ajp>, and select "Residents" in the manuscript type field.

- 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 Themes

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

Women's Health

If you have a submission related to this theme, contact the Section Editor, Kathleen Mary Patchan, M.D. (kpatchan@psych.umaryland.edu).

Violence and Mental Health

If you have a submission related to this theme, contact the Section Editor, Ijeoma Chukwu, M.D., M.P.H. (ichukwu@uci.edu).

Addiction Psychiatry

If you have a submission related to this theme, contact the Section Editor, Juliet Muzere, D.O. (jmuzere@gmail.com).

*If you are interested in serving as a **Guest Section Editor** for the *Residents' Journal*, please send your CV, and include your ideas for topics, to Misty Richards, M.D., M.S., Editor-in-Chief (mcrichards@mednet.ucla.edu).