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Inside

COMMENTARY

Switching Modalities to Avoid Impasses: A Potential Problem With Psychotherapy Education in Residency Training

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Psychiatry residencies teach multiple psychotherapeutic modalities. The pursuit of proficiency in diverse methods creates a dilemma that is not often encountered by practicing psychotherapists. Trainees select from tools in which they lack proficiency and of which their understanding is incomplete. Training curricula in psychotherapy, trainee comfort and preference, and pressure from supervisors all factor in this decision.

Patients, particularly patients with treatment-resistant symptoms and comorbidities, may not improve according to plan. When meeting an impasse, a frustrated resident may understandably ask, "Am I providing the right treatment?", and seek additional supervision.

Experience shows that psychotherapy supervisors tend to practice almost exclusively in one method (and may disparage other models), and training programs employ supervisors from a variety of therapeutic disciplines. When treatment stalls, trainees may consult a practitioner of a therapy method different from the one they are currently using. The supervisor may insist with seductive confidence that the lack of progress is due to use of an "incorrect" treatment. The promise of better outcomes leads the malleable resident to change course instead of working through deadlocks.

Each psychotherapeutic system incorporates strategies for resolving periods of stagnation. A cognitive-behavioral therapy (CBT) supervisor, for example, can guide a resident conducting CBT through impasses within that theoretical framework. Treatment inertia often hints at core components of the patient's psychopathology; a decision to change methods risks distancing the work from the exact place it needs to be.

Rather than wholesale recommendations to switch to one's orientation of choice, a supervisor is better served to respect the ability of other evidence-based and time-tested therapies to enact change.

Adherence to a psychotherapeutic model is one indicator of treatment outcome, highlighting the risks of mid-treatment changes in a method (1). The patient-therapist relationship is also a key predictor of success (1, 2), and switching of orientation may weaken alliance by altering the way a therapist intervenes in the room (i.e., behavioral-based homework assignments versus insight-oriented interpretation), a change that may be jarring for patients.

Though I acknowledge that there are select times when a change in approach is reasonable, I worry that the decision to abandon one treatment modality for another is too often in the service of an opportunity to learn a new technique or in response to disappointments over the slow, uncertain trajectories that define the psychotherapeutic process. Additionally, I wonder if the idea that therapeutic change remains elusive not because of the resident's clinical inadequacies but because the wrong tools are being used and may be more palatable in the context of the narcissistic vulnerabilities unavoidably cultivated by psychiatric training (3).

When presented a case already in progress, supervisors should be aware of the need for residents to learn to work with resistance in all modalities. Rather than wholesale recommendations to switch to one's orientation of choice, a supervisor is better served to respect the ability of other evidence-based and time-tested therapies to enact change. Encouraging the trainee to work with a supervisor familiar with the current therapeutic mode to challenge resistance, while also discussing how one might approach the patient in an alternative theory, would maximize educational benefit. Working through resistance while adhering to a given therapeutic orientation enhances patient care and resident development.

At the time this commentary was accepted for publication, Dr. Scarella was a fourth-year resident in the Department of Psychiatry, Beth Israel Deaconess Medical Center, Boston; he was also affiliated with Harvard Medical School, Boston.

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ARTICLE

Ketamine as a Rapid-Acting Antidepressant: Promising Clinical and Basic Research

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Suicidal ideation and attempts are a common medical emergency, accounting for about 650,000 adult evaluations per year in emergency settings (1). Depressive disorders are a major driving force behind this, but first-line antidepressants, such as selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs), can take months to work, making them of limited use in acutely suicidal patients. Potentially safe and fast-acting interventions would be invaluable in acute situations until standard antidepressants have time to take effect.

Ketamine, best known as an *N*-methyl-D-aspartate receptor (NMDAR) antagonist commonly used as an anesthetic, has recently drawn attention for possibly filling the role. At lower doses it exhibits strong antidepressant effects in many patients, and it acts on the order of minutes. Despite these promising effects, its use as an antidepressant has been controversial, as ketamine is also a Schedule III controlled substance that is used recreationally for its dissociative and hallucinogenic effects. Furthermore, the full mechanism of action regarding its antidepressant effects has long remained unclear.

In the present article, we review research surrounding ketamine's potential as a fast-acting antidepressant from a "two-pronged" approach: first, summarizing established and new knowledge on its mechanism of action and second, reviewing clinical research addressing its potential to quickly reduce depression and suicidality.

MECHANISM OF ACTION

Ketamine is a chiral compound that operates broadly, having effects on mu, kappa, and delta opioid receptors, as well as dopamine D_2 receptors and the reuptake of serotonin, dopamine, and norepinephrine (2). However, ketamine is best known as an antagonist of the NMDAR, a glutamatergic receptor that allows an influx of cations across neuronal cell membranes (2).

It is ketamine's antagonism of NMDAR that is thought to account for its analgesic and anesthetic effects, with (S)-ketamine binding with greater affinity to NMDAR (2, 3). Until recently, its NMDAR antagonism was thought to account for most of its antidepressant effects as well (4). This hypothesis places ketamine in sharp contrast with traditional antidepressants, whose mechanisms of action generally involve monoamine neurotransmitters (e.g., serotonin or norepinephrine). Despite such putative explanations, limitations of the NMDAR antagonism hypothesis (such as observed antidepressant effects of agents that act as NMDAR agonists, as well as known NMDAR antagonists that lack antidepressant effects) (4) have led to calls for alternative explanations of its mechanism (5, 6).

A recent study published in *Nature* has addressed this call, concluding that the most relevant mechanism of action for ketamine's antidepressant effects is actually due to one of ketamine's metabolites: [2R,6R]-hydroxynorketamine (HNK), a metabolite formed from (R)-ketamine (7). The investigators demonstrated in mice that [2R,6R]-HNK has significantly stronger antidepressant effects than ketamine itself, as well as its enantiomer [2S,6S]-HNK, which only produced effects at much higher doses. This finding was surprising, as (R,S)-norketamine was thought to be the only active metabolite of ketamine (3, 8, 9).

The investigators also showed via mice models that HNK acts not at the NMDA receptors, like ketamine, but rather via the α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPAR), in a sustained, excitatory mechanism (7). This non-NMDA-type ionotropic transmembrane glutamate receptor is thought to play a role in plasticity and synaptic transmission, which are key in sustained antidepressive effects (7, 10, 11). Blockage of this pathway through administration of AMPAR antagonist NBQX showed a loss of antidepressant effects in mice brains upon dosage of ketamine or HNK.

Additionally, it was observed that [2R,6R]-HNK appeared to lack many of the undesirable side effects of ketamine; specifically, mice did not self-administer [2R,6R]-HNK, suggesting lower potential for abuse or addiction (7). Furthermore, [2R,6R]-HNK showed fewer signs of dissociative effects, such as changes in motor coordination and sensory processing (7). This suggests that [2R,6R]-HNK may operate independently of mechanisms that are otherwise active with ketamine. Despite the promise of this recent study, replication in human cells is missing and required for true disease-specific conclusions.

CLINICAL RESEARCH

While standard antidepressants like SSRIs and SNRIs are effective in reducing depression and suicidal ideation, they take weeks to become effective, leaving patients vulnerable to suicidal ideation or behavior. In fact, the first week after standard antidepressant ini-

tiation may be a time of increased suicidality, underscoring the importance of a medication that decreases self-harming behaviors in early stages (12). Ketamine has increasingly attracted interest for potentially filling this role since 2000, when the first randomized controlled trial (RCT) of ketamine as an antidepressant was found to produce significant and rapid improvements of mood lasting 1–2 weeks post-infusion (13).

In a recent meta-analysis of seven RCTs involving 183 patients with major depressive episodes, the effects of ketamine were assessed with respect to clinical response and remission. Clinical remission was defined as a Hamilton Depression Rating Scale (HAM-D) score <7 or a Montgomery-Åsberg Depression Rating Scale (MADRS) score <10. Clinical response was defined as a ≥50% decrease in these scores post-treatment. For clinical remission, pooled odds ratio (OR) and number needed to treat (NNT) were calculated after 24 hours (OR=7.06, 95% confidence interval [CI]=2.50–19.95; NNT=5), 3 days (OR=3.86, 95% CI=1.53–9.74; NNT=6), and 7 days (OR=4.00, 95% CI=1.52–10.51; NNT=6). For clinical response, OR and NNT were also calculated at 24 hours (OR=9.10, 95% CI=4.28–19.34; NNT=3), 3 days (OR=6.77, 95% CI=3.40–13.50; NNT=3), and 7 days (OR=4.87, 95% CI=2.24–10.55; NNT=4) (14). Significantly, adverse events were limited to two patients, one with refractory hypertension and another with hypotension and bradycardia. Other, similar meta-analyses have also concluded that ketamine is fast-acting, effective, and generally well-tolerated (6, 15).

While the literature on ketamine's potential as an antidepressant is growing, there is now research examining ketamine's anti-suicidal properties specifically. One of the first group of investigators to assess ketamine's anti-suicidal properties found that in 33 subjects with major depressive disorder, intravenous ketamine reduced suicidal ideation in all four measures used (HAM-D, MADRS, the Scale for Suicide Ideation, and the Beck Depression Inventory). These reductions were significant ($p<0.001$ for all measures) up to 230 minutes post-infusion, the latest measurement taken, suggesting significant promise for reducing suicidal ideation acutely (16). Limitations of this work, however, include small sample size and open-label administration, accounting for possible bias. Additionally, the 230-minute time point may only be capturing the expected highs and lows of suicidal thoughts.

In an RCT comparing intravenous ketamine to midazolam placebo in patients with treatment-resistant major depression, suicidality assessment in patients 24 hours post-infusion showed that ketamine significantly reduced suicidal ideation in comparison to midazolam (17). These changes, however, were mediated by reduction in non-suicidal depressive symptoms, raising the question of whether ketamine's effect on reducing suicidal ideation is merely a by-product of its antidepressant actions.

Ketamine's antidepressant effects, however, are relatively short-lived, with one infusion generally lasting around 1 week (18). Consequently, research has begun to examine the safety and efficacy of repeated infusions. In one study, Murrough et al. (19) gave 24 patients with treatment-resistant depression intravenous ketamine thrice weekly over a 12-day period, up to six times. Responders (defined as a ≥50% improvement in MADRS) deviated from non-responders by 4 hours post-first treatment (mean MADRS score: 10.35 compared with 19.0, $p=0.013$), and the deviation increased by 24 hours (8.35 compared with 18.8, $p=0.002$). Overall, 70.8% of patients responded to treatment. Of patients who responded, the median time to relapse was 18 days after last infusion (defined as <50% improvement in MADRS compared to baseline, for two consecutive visits). Four patients remained relapse-free until 83 days later, the last day assessed (19). Thus, there is initial evidence to suggest that repeated infusions may be a safe and effective way to extend the protective effects of ketamine in acute settings.

CONCLUSIONS

In 2014, Thomas Insel, former Director of the National Institute of Mental Health, declared that ketamine might be "the most important breakthrough in antidepressant treatment in decades" (20). Since then, research has largely substantiated this enthusiasm. Most meta-analyses and reviews published within the last 2 years have found ketamine to be fast-acting, effective, and well-tolerated, although the duration of its effects is somewhat limiting (14, 18). Furthermore, recent research from basic science has finally shed light on what is likely the primary mechanism of action of ketamine's antidepressant properties—its metabolism to [2R,6R]-HNK, with subsequent activation of AMPA receptors (7).

Limitations

There remains much to learn before ketamine can be approved for treatment of suicidal ideation. Ketamine's known effects include both stimulant and opiate-like properties, both of which are known to transiently improve mood but are limited by the possibility of misuse and physiological dependence (5, 21). Fur-

KEY POINTS/CLINICAL PEARLS

- Ketamine has shown promise as a safe and fast-acting antidepressant, but effectiveness of single doses is limited by short duration (around 1 week).
- Groundbreaking new research in mice models suggests that it is not ketamine itself but hydroxynorketamine (HNK; a metabolite of ketamine) that primarily accounts for ketamine's antidepressant effects.
- HNK, especially (2R,6R)-HNK, appears to exert its strong antidepressant effects by activation of α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors, not inhibition of *N*-methyl-D-aspartate receptors, like ketamine; furthermore, it seems to lack the addictive or dissociative effects of ketamine.
- Future research on ketamine as a rapid-acting antidepressant should address the safety and efficacy of extending its effects through repeated infusions and explore the potential of (2R,6R)-HNK as an antidepressant in humans.

thermore, while adverse events in clinical trials have been rare, the long-term effects of repeated administration are relatively unknown (4).

Perhaps the significance of ketamine research to date has been the discovery of its metabolite, HNK, which may provide a mechanism for the treatment of depressive-like behaviors in mice. This could prove to anchor our quest for safe, efficacious, and rapidly acting therapies for the treatment of human depression and suicidal ideation.

Andrew Tuck is a second-year medical student, and Danish H. Ghazali is a second-year medical student at Columbia University College of Physicians and Surgeons, New York.

Andrew Tuck and Danish H. Ghazali contributed equally to this study.

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ARTICLE

Survey of EKG Monitoring Practices: A Necessity or Prolonged Nuisance?

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Psychotropic drugs delay cardiac repolarization prolonging the QT interval on EKGs. These medications bind cardiac potassium channels, thus blocking potassium efflux from cardiomyocytes. A prolonged QT arouses concerns in clinical practice, as it can lead to, in some cases, a life-threatening polymorphic ventricular tachyarrhythmia called torsades de pointes (1). Torsades de pointes is usually self-limited and typically manifests in convulsions, dizziness, and syncope; however, it can also result in ventricular fibrillation and sudden cardiac death (2). EKG warning signs that precede torsades de pointes include marked QTc prolongation, premature ventricular contractions, and T-wave changes. Cardiac repolarization is faster at higher heart rates, hence there is need for a rate-corrected adjustment of the QT interval (QTc) (2, 3). The most commonly used formula, Bazett's formula, defines QTc as QT/RR0.5, where RR is heart rate. This formula produces accurate calculations for heart rate in the narrow range of 60–80 beats per minute. For higher heart rates, there is the Fridericia correction formula, which defines QTc as QT/RR0.33 (3). Normal QTc range differs based on age and sex: for men it varies between 440 and 450 msec, and for women it varies between 460 and 470 msec (4, 5). The Food and Drug Administration defines threshold for potential discontinuation of a medication at a QTc >500 msec, or 60 msec above baseline, or >5-msec increase in mean value (6). The American Heart Association and American College of Cardiology mirror this threshold. QTc prolongation is not an ideal predictor for risk of torsades de pointes but rather modest at best (7).

The objective of the present study was to determine the extent to which evidence-based practices have affected clinical QTc monitoring in those prescribing antipsychotics and how closely American Psychiatric Association (APA)-outlined guidelines are being followed.

RISK

There is no threshold for QTc prolongation at which torsades de pointes is certain to occur. Data from congenital long QT syndrome studies report that a QTc >500 msec is associated with a 2-to 3-fold higher risk for torsades de pointes (5). Incidence is difficult to obtain. Sudden death is assumed to be secondary to arrhythmias; however, patients are not monitored at the time of death, and we cannot assume association with torsades de pointes. Number needed to screen would be needed. Risk factors for QTc prolongation can be divided into three categories: genetic, medication-related, and individual dependent. Genetic risk includes congenital long QT syndromes that can be attributed to mutations in potassium and sodium channels, occurring in 1/2,500 individuals in the population (8). Medication risk manifests in several drugs known to prolong QTc by way of cumulative effect or via inhibition of the cytochrome system; these most frequently inhibit potassium channels (9). Examples include antiarrhythmic class IC and III (sotalol, flecainide, and propafenone), diuretics (especially potassium sparing), all antidepressants to some degree, antipsychotics (10–12), antihistamines, macrolides, alcohol, caffeine, cocaine, and methadone. Aside from electrolyte abnormalities (hypokalemia, hypomagnesemia), individual risk factors also include female gender, 65 years of age or older, and patients with cardiac risk factors such as congenital long QT syndromes, ischemic heart disease, congestive heart failure, myocarditis, hypertension, bradycardia, and sinoatrial/atrioventricular blocks (9) The APA also mentions a personal history of syncope, as well as family history of sudden death at an early age (less than 40 years old, especially if both parents had sudden death) as risk factors (13).

APA GUIDELINES

Current APA guidelines recommend checking EKG and serum potassium before treatment with thioridazine, mesoridazine, or pimozide, as well as prior to treatment with ziprasidone in the presence of cardiac risk factors. It is also recommended to obtain a follow-up EKG any time a significant change in dose of thioridazine, mesoridazine, or pimozide occurs in the presence of cardiac risk factors, ziprasidone use, or with addition of other medications that can alter QTc interval. Discontinuation of any antipsychotic should be considered at a QTc >500 msec, or 60 msec above baseline, or >5 msec increase in mean value (13).

METHODOLOGY

A survey was distributed via Qualtrics Survey Software (https://www.qualtrics.com) to 52 members of the faculty/academic residency program at East Carolina University, Department of Psychiatry. Design of the survey aimed to best characterize trends in EKG monitoring practices. Survey conductors had no disclosures, and this study was exempt from institutional review board review. Target participants included a varied sample of physicians in training at any level, fellows, and faculty treating both adults

and adolescents at both inpatient and outpatient levels. Level of expertise and age was very varied. Affiliate and adjunct faculty were excluded, as were any faculty members unable to write prescriptions. Initial distribution was on September 11, 2015, and was available for 1 month with three weekly reminders sent out to non-responders. Responses were completely anonymous, with no Internet protocol address or any identifiers collected. Data were gathered and fur-

FIGURE 1. Survey of Provider Screening for Risk Factors

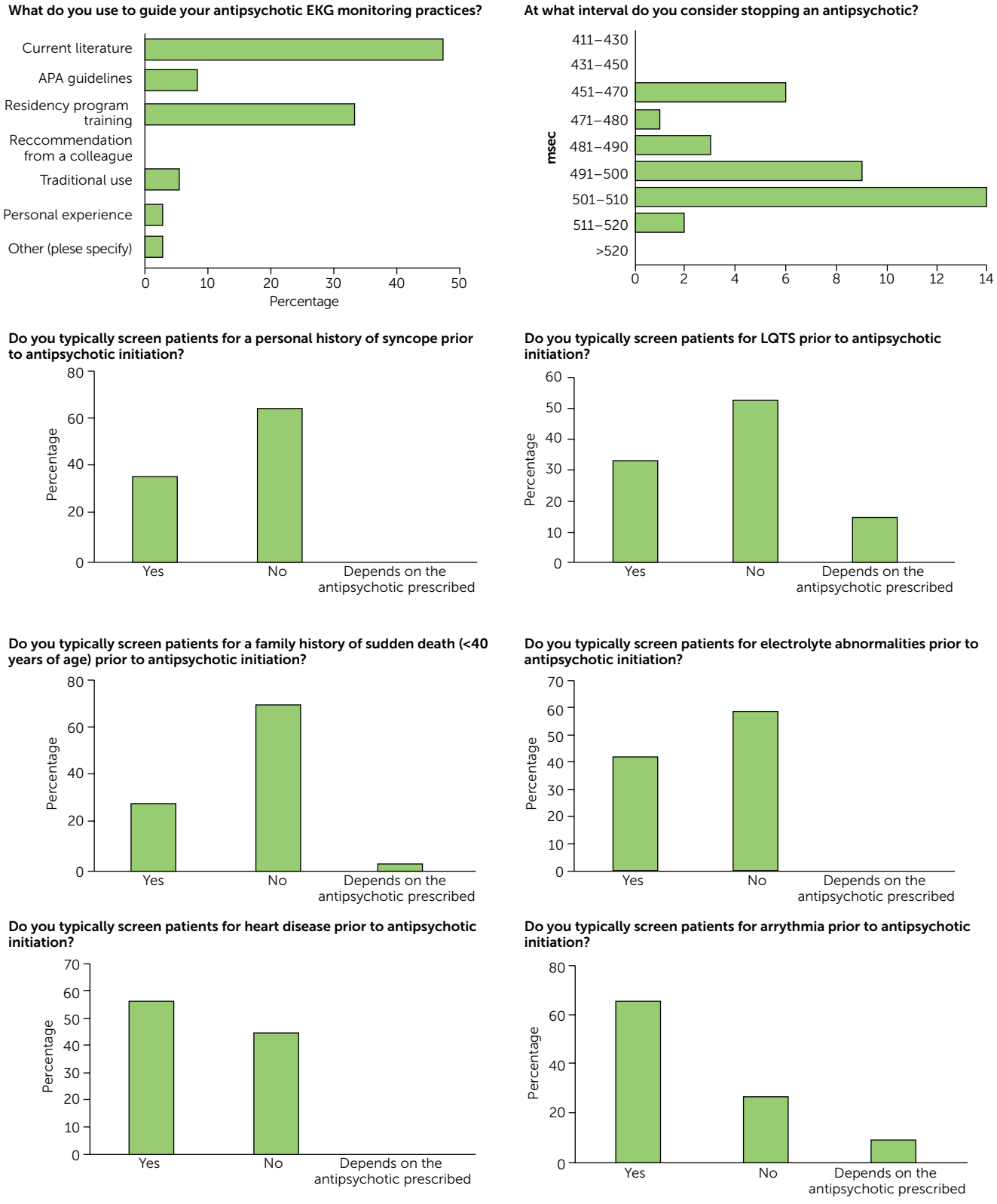
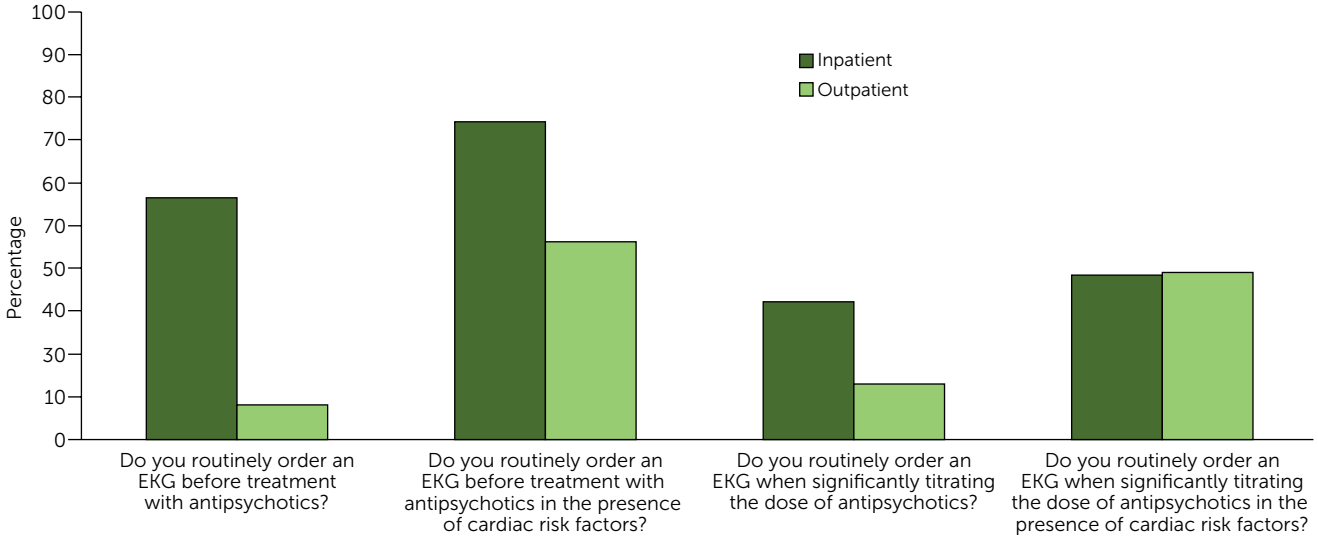


FIGURE 2. Survey of EKG Assessment With Antipsychotic Treatment



ther analyzed by Qualtrics. The response rate was 55%, with a 24% drop-out rate (i.e., participants who started the survey but did not complete it). Participants who reported practicing in both inpatient and outpatient settings completed 27 questions, whereas participants who reported only one practice setting completed a survey of 22 questions. The length of the survey may have been a factor in the drop-out rate. The survey maintained complete confidentiality, and the surveyors remained blinded.

RESULTS

Upon consulting with a statistician (although there was no statistically significant difference between those practicing in outpatient versus inpatient settings), there was a significantly large proportion of psychiatrists who did not screen for personal history of syncope, family history of sudden death, electrolyte abnormalities, and long QT syndrome (see Figure 1 and Figure 2).

DISCUSSION AND TREATMENT RECOMMENDATIONS

In our practice, the use of psychotropic medications that affect repolarization cannot be avoided. Physicians should be aware of the risk associated when combining multiple drugs that prolong QTc and avoid these prescribing habits. Personalized risk assessments should also be undertaken with a goal of risk reduction. Electrolyte evaluation and EKG recording appear to be warranted prior to treatment initiation and later under steady conditions. Slow dose titration and regular EKG monitoring of patients at high risk or of those prescribed additional medications that can prolong QTc are recommended. Throughout the course, it is prudent to be mindful of potential electrolyte imbalance during episodes of diarrhea, sweating, malnutrition, diuretic therapy, alcohol/drug use, and eating disorders. Arrhythmia should be considered when patients present with palpitations, dizziness, syncope, or convulsions. In the event of a markedly elevated QTc, magnesium sulfate, either orally or intravenously, should be considered. Medication discontinuation should always be considered when QTc is >500 msec, despite normal serum potassium, normal QRS duration, and lack of symptoms.

CONCLUSIONS

At present, no quantitative multivariate risk index exists for the prediction of torsades de pointes. Providers in our survey share differing opinions on the exact QTc interval at which discontinuation of antipsychotics is necessary. Our survey shows that most providers turn to current literature to guide their treatment decisions, indicating the older existing APA guidelines are lagging behind current literature. Current APA guidelines need to be updated to reflect data relevant to today's prescribing practices. Our survey demonstrated that antipsychotics that appear to have providers concerned about QTc prolongation were identified as ziprasidone, haloperidol, prolixin, thorazine, clozapine, and

KEY POINTS/CLINICAL PEARLS

- Antipsychotic medications delay cardiac repolarization resulting in QTc prolongation and, in severe cases, torsades de pointes.
- Regardless of practice setting, prior to institution of such agents we need to individually risk-stratify our patients by obtaining baseline EKGs and screen for predisposing factors of QTc prolongation.
- During course of treatment, especially in those labeled high risk, it is prudent to slowly titrate medications, routinely check EKGs and electrolytes, and discontinue the agents whenever QTc is >500 msec, despite normal serum potassium, normal QRS duration, and lack of symptoms.

quetiapine. The fact that thioridazine, medoridazine, and pimozide were not among them is likely attributed to the lack of current utilization. One common finding based on literature review is apparent—the importance of cardiac risk stratification. Our survey demonstrated that providers do not routinely screen for cardiac risk factors. Only 64% and 55% of those surveyed report screening for arrhythmias and heart disease, respectively, and more concerning is the lower proportion of providers screening for any personal history of syncope (33%), family history of sudden death (26%), or long QT syndromes (34%). Only 42% screen for electrolyte imbalance. An independent, nonprofit organization (www.crediblemeds.org) developed a risk stratification process—the Adverse Drug Event Causality Analysis [ADECA]—where drugs are placed into one of four risk categories based on their relative potential for QTc prolongation and/or cause of life-threatening ventricular arrhythmias. This, along with cardiac risk stratification, can provide guidance in treatment decisions. According to our data, outpatient providers are less likely to order EKG when prescribing antipsychotics. The literature suggests that this is attributed to financial issues and lack of access to EKG resources, especially when not practicing in a tertiary care setting. Further evaluation comparing tertiary care setting outpatient psychiatrists, as well as those with access to EKG resources, would be beneficial in identifying barriers to practice.

Limitations

This study was conducted strictly in the setting of an academic institution. Whether there is generalizability to the practices of outside providers cannot be confirmed. The number of responders participating is relatively low; however, the high response rate increases the validity.

Dr. Broszko and Dr. Stanciu are fourth-year residents in the Department of Psychiatry and Behavioral Medicine, East Carolina University, Greenville, N.C.

Starting July 2017, Dr. Stanciu will be entering an Addiction Psychiatry fellowship offered by the Dartmouth-Hitchcock Department of Psychiatry, Lebanon, New Hampshire.

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CASE REPORT

Recurrent Foreign Body Ingestions Following Rapid Methadone Taper: Neurological Aspects of Self-Injury and Opioid Therapy

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The following case details the treatment of a patient with opioid use disorder and borderline personality traits hospitalized for recurrent foreign body ingestion after rapid taper of methadone. Methadone is a leading treatment for opioid-addicted patients, reducing relapse and other substance use, and shown to have anti-manic and anti-psychotic effects (1). The medical literature equivocates about methadone-taper versus long-term therapy, and the risks cessation can present to patients with psychiatric comorbidities (2, 3). Although usefulness of naltrexone and anti-psychotic therapy for curbing self-mutilation has been demonstrated, the relationship between self-harm and methadone is largely unstudied, despite high concordance between parasuicidality and opioid abuse (4–6). The present case highlights the neurological relationship between methadone and parasuicidal behavior in patients with borderline traits and illustrates the risks of inadequate methadone therapy in patients with these psychiatric comorbidities.

We present a case with discussion surrounding the relationship between self-harm and opioid pathways in patients with borderline personality disorder. Informed consent was obtained from the case patient, and our institutional review board opined that this report is exempt from institutional review board inspection.

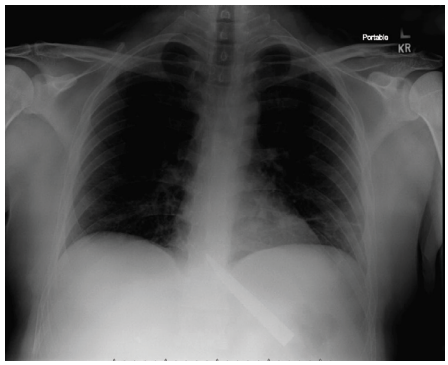
CASE

"Mr. A" is a homeless, 39-year-old Caucasian man, who presented to the emergency department of an urban academic medical center after he swallowed a 6-inch knife (see Figure 1). He endorsed a history of opioid use disorder, depression, psychotic episodes, and childhood sexual abuse but denied suicidality. He described episodes of cutting and self-electrocution with an electric fence in his teens, calling these behaviors a means of emotional release. He reported trying foreign body ingestion once as a manipulative behavior 13 years before, learning it from fellow inmates while incarcerated on drug charges. His vitals and physical examination were benign, without signs of opioid withdrawal. Urinary toxicology showed marijuana, cocaine, and opioids. Mini-Mental State Examination and Montreal Cognitive Assessment scores were normal (29 and 30, respectively). Mental status examination demonstrated a well-dressed male, cooperative to interview, with appropriate eye contact and normal speech. He described his mood as depressed and angry, with congruent affect. The patient's thought content showed no suicidal or homicidal ideation, with linear, goal-oriented process. No perceptual distortions were elicited.

Collateral from prior treatment programs revealed a 3-year period of stability at daily doses of methadone (190 mg–210 mg), sertraline (100 mg), and quetiapine (200 mg). Since he had inpatient-restricted Medicaid, his outpatient methadone treatment had not been paid. Subsequently, he had an administrative discharge from a methadone maintenance treatment program (MMTP) and a 6-week taper to 0 mg. After several weeks without treatment, he presented to our hospital for foreign body ingestion.

After endoscopic removal of the knife, he was started on sertraline (100 mg), quetiapine (200 mg), and methadone. The daily methadone dose was gradually increased, and we planned to refer him to an MMTP. Despite 2-to-1 observation, the patient locked himself in his bathroom and ingested three screws and a broken light bulb, since his physician only prescribed a 35-mg dose of methadone, whereas the consultant psychiatrist had recommended 40 mg (dosage confirmed by chart review). The objects perforated his gastric wall, requiring immediate laparotomy and repair. Even with this injury, when his demands for medication were not heeded or he feared that he might be discharged without referral to an MMTP, he would again ingest, with items ranging from screws and cabinet knobs to a pencil.

FIGURE 1. Emergency Room X-Ray of Patient Who Swallowed a 6-Inch Knife[a]



[a] The image is a portable chest X-ray of the patient, showing a 6-inch linear opacity overlying the spinal column and gastric bubble. (Courtesy of the SUNY Downstate Department of Radiology.)

We hypothesized that Mr. A's recurrent foreign body ingestion was the result of exacerbation of his borderline personality traits secondary to the cessation of his opioid-agonist therapy. Once his methadone was titrated to 100 mg daily, episodes of foreign body ingestion ceased. He was discharged with appropriate Medicaid and referred to an MMTP.

KEY POINTS/CLINICAL PEARLS

- Opioid agonist maintenance can be an essential treatment for people receiving it, and too rapid taper risks psychiatric behavioral decompensation.
- In addition to reducing opioid abuse and other drug use, methadone may curb impulsive and self-injurious behavior in patients with borderline personality traits.
- Although withdrawal can be mitigated with as little as 20 mg of methadone, high-dose treatment (>80 mg) has been shown to more greatly reduce incidence of relapse and other drug use.

DISCUSSION

The above patient's initial foreign body ingestion was a pathologic manipulative response to ensure access to methadone, but it generalized to situations in which he felt frustrated, such as confrontations with nurses, surgical teams, and even housekeeping. A differential diagnosis for foreign body ingestion includes malingering, psychosis, and self-harm in patients with cluster B personality traits (7). Although Mr. A's aggression, drug history, and criminality suggested antisocial personality, his affective instability, idealization, and devaluation of medical staff and impulsive self-harm (in excess of what was necessary to remain hospitalized) indicated significant borderline personality traits.

Case reports have described cessation of self-mutilation of borderline patients using naltrexone and atypical antipsychotics, but to our knowledge the efficacy of methadone for this purpose has not been demonstrated (4, 5). Opioid addiction, manipulative parasuicidality, and psychiatric symptoms share neurological underpinnings. Self-injury can lead to release of endogenous opioids, creating an antinocioceptive state via altered μ-opioid receptor activity (8). Oyefeso et al. found 49% prevalence of alexithymia and self-harm (most commonly cutting) in a cohort of 80 opioid-addiction treatment patients, pointing toward a commonality between drug use and self-injurious methods for endogenous opioid release (6). A 2010 neuroimaging study of 18 sober, female borderline personality disorder patients showed increased μ-opioid receptors in the prefrontal cortex, nucleus accumbens, and amygdala (suggesting a resting opioid deficit), with paradoxically elevated endogenous opioid system activation during times of induced stress (9). Though the exclusion of male subjects and those with comorbid substance abuse disorders (within 3 months of positron emission tomography scan) limits this study's generalizability, it corroborates the model of opioid-deficiency in borderline personality disorder and suggests a mechanism for these patients' exaggerated stress response.

The neurological models previously illustrated beg the question of whether methadone could stabilize the opioid receptors of our borderline personality disorder patient. From his own reporting, as well as collateral information obtained from multiple sites (hospitals, outpatient treatment centers, police records, family members), it was clear that Mr. A was stable when receiving high doses of methadone. By restarting the high-dose methadone therapy, he returned to his non-impulsive, non-self-injurious baseline. Frankenberg et al. demonstrated opioid prescription rates in borderline personality disorder populations to be double that of patients with other axis II diagnoses. Though limited by size in the comparison group (borderline personality disorder patients: N=264 vs. axis II patients: N=63), this study suggests a neurological convergence of physical and psychical pain unique to the borderline personality disorder pathology that is assuaged by opioid therapy (10). These studies all offer possible explanations for Mr. A's period of psychiatric stability and substantiate the idea that he could return to baseline by resuming high-dose methadone.

A neurological commonality between parasuicidality and opioid use is tempered by the fact that by increasing Mr. A's methadone in response to his foreign body ingestion, we positively reinforced a maladaptive behavior. Nevertheless, this case makes clear the consequences of inappropriate methadone maintenance treatment cessation and the benefits of high-dose methadone maintenance treatment. Case studies of prisoners show elevated risk of re-incarceration and resumption of opioid use by patients previously on methadone maintenance treatment (1). Although withdrawal can be mitigated with as little as 20 mg of methadone, high-dose treatment (>80 mg) has been shown to more greatly reduce incidence of relapse and other drug use (8, 10) Methadone tapering is most successful in patients with stable housing, finances, and psychiatric well-being, conditions that Mr. A clearly did not meet (3). With the help of a multi-faceted team of social workers, physicians, and nursing staff, Mr. A was able to acquire temporary housing and was discharged to an MMTP, where it was agreed another taper would not be attempted.

CONCLUSIONS

Rapid taper of high-dose methadone therapy led a man with borderline personality traits to engage in repeated foreign body ingestion until his treatment was reinstated. This case illustrates the danger of behavioral destabilization if opioid agonist maintenance therapy is abruptly discontinued in such patients.

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CASE REPORT

Kratom (*Mitragyna speciosa*) Use in a Veteran With Chronic Pain

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Kratom (*Mitragyna speciosa*) is a plant indigenous to Thailand, Myanmar, and Malaysia, with opioid receptor activity traditionally used by Southeast Asian farmers to combat fatigue and to improve work productivity. Amidst a backdrop of increasing opioid misuse and overdose in the United States, kratom has steadily gained popularity among individuals with chronic pain due to its legality, analgesic properties, and ability to mitigate withdrawal symptoms from opioids. Kratom has recently become the focus of Drug Enforcement Agency (DEA) scrutiny. We present the case of a veteran's use of kratom to mitigate symptoms of opioid withdrawal in the context of physical dependence to prescription opioids.

CASE

"Mr. B" is a 34-year-old divorced, unemployed, Caucasian male veteran with a history of opioid use disorder, posttraumatic stress disorder (PTSD), and major depressive disorder (MDD), who presented to a Veterans Administration (VA) hospital requesting opioid detoxification.

The veteran had an extensive combat trauma history, serving 18 months in Iraq and Afghanistan as a machine gunner. In addition, he was exposed to improvised explosive device blasts but was never rendered unconscious. He was diagnosed with MDD and PTSD in 2009 after returning from Afghanistan. Prior to being discharged from military service, he was prescribed hydrocodone/acetaminophen and tramadol for back pain related to an injury sustained in Iraq. He denied taking more hydrocodone/acetaminophen than prescribed but acknowledged the possibility that he was taking it for reasons other than treatment of his back pain.

When he returned from deployment in 2010, he began using illicit oxycodone/acetaminophen (300 mg by mouth daily) and denied other routes of administration. He was prescribed buprenorphine/naloxone by a non-VA provider in February 2011 and did not use any illicit opioids for 1 month. He did not follow-up with his non-VA provider and resumed episodic illicit oxycodone/acetaminophen from March 2011 until July 2011. His pattern of use ranged from 30 mg to 150 mg of oxycodone/acetaminophen by mouth daily for 1–2 weeks, followed by abstinence for 1–2 weeks.

In August 2011, the patient entered an intensive outpatient substance use treatment program and discontinued oxycodone/acetaminophen. However, he began brewing kratom tea to mitigate symptoms of withdrawal from oxycodone/acetaminophen. He escalated his use of kratom to 10 grams twice daily to avoid withdrawal symptoms due to development of physiological tolerance to kratom. He denied feelings of euphoria while using kratom, instead experiencing relief from the elimination of withdrawal symptoms. He purchased kratom from online vendors and at specialty stores. When he was unable to procure kratom, he would substitute with illicit buprenorphine/naloxone (4 mg/1 mg). Prior to presenting for detoxification at the VA, he transitioned from kratom to buprenorphine/naloxone under the presumption that medical professionals would be more familiar with detoxification from buprenorphine/naloxone than kratom.

On admission to the VA inpatient detoxification unit, standard admission laboratory results were within normal limits. Urine toxicology was positive for opioids. The veteran was found to have no clinically relevant electrolyte abnormalities. He completed a 3-day uncomplicated detoxification from opioids. Withdrawal symptoms included restlessness, anxiety, sleep disturbance, sweats, chills, and diarrhea. He was discharged to a residential addiction program upon completion of his detoxification.

DISCUSSION

Native to Southeast Asian countries, kratom is a large tropical tree whose leaves have been traditionally chewed or made into a tea by laborers and farmers for decades to help combat fatigue and improve work productivity (1). As early as the 1800s, it has been used for the treatment of muscle pain, diarrhea, and cough, as well as to enhance productivity (2). Kratom preparations have also been used for centuries to treat morphine dependence in Thailand and to serve as an opium substitute in Malaysia during cultural and religious ceremonies (3). In 1979, kratom was placed under Schedule 5 of the Thai Narcotic Act, making it illegal to buy, sell, import, or possess. In Malaysia, kratom has been placed

under the Poisons Act 1952 since 2003, resulting in a penalty or jail sentence for selling it. In recent years, however, kratom has been legally exported to North America and Europe from Indonesia for processing and redistribution (4).

Kratom has a unique pharmacological (see Table 1) and toxicological (see Table 2) profile and is commonly used for its anxiolytic and antidepressant effects (5). For chronic users, withdrawal symptoms are consistent with that of opioids such as morphine (Table 2) (6). According to the National Institute on Drug Abuse, the number of deaths from heroin has increased 6-fold from 2001 to 2014 (7). While kratom has been associated with several deaths in combination with other substances, there is no solid evidence that it was the sole contributor to an individual's death (Table 2) (6). In 2010, only one case of kratom use was reported in U.S. drug data bases. In 2011, there were 44 reports of kratom use and 81 reports in the first 6 months of 2012. However, as kratom use is not monitored by any national drug use surveys in the United States, it is difficult to quantify the prevalence of its use among the U.S. population (1). Obtaining kratom is a relatively simple undertaking in the United States, as it can be ordered on many websites and shipped directly to an individual's home. It is sold as tablets, capsules, concentrated extracts, or chopped leaves. The fresh or dried leaves of kratom are traditionally brewed into a tea or smoked (5). However, kratom has recently garnered much attention in the media after the DEA announced its intention, on August 31, 2016, to designate it as a Schedule I substance. Following public outcry, the DEA withdrew its notice of intent and instead opened an official public comment period until December 1, 2016. The DEA has also requested that the Food and Drug Administration hasten scientific research, which would be halted if kratom is assigned Schedule I status.

A recent study examining mitragynine pseudoindoxyl, synthesized by an oxidative rearrangement of mitragynine, showed promise for the compound as a potent analgesic. The study found that mice administered mitragynine pseudoindoxyl developed analgesic tolerance more slowly than morphine and showed limited physical dependence, respiratory depression, and constipation and displayed no reward or aversion in conditioned place preference, as well as conditioned place aversion assays (8). Despite the promise of this study, there is a dearth of controlled clinical human studies to better understand the risks and benefits.

TABLE 1. Pharmacological Profile of Kratom

Primary constituents	Mitragynine (60%) and 7-HMG (2%) (6)
Potency	7-HMG > mitragynine; 7-HMG up to 17 times more potent than morphine (10)
Receptor activity	Mitragynine and 7-HMG: selective and full agonists of mu-opioid subtype receptors (6); mitragynine: mu, kappa, delta opioid receptor agonist with activity noted at adenosine-2a, postsynaptic alpha-2, dopamine-2s, and various serotonin receptors (2); descending noradrenergic and serotonergic anti-nociceptive activity (9)
Dose-dependent effects	Low dose (1–5 g): stimulant-like effects High dose (5–15 g): opioid-like effects (5)
Time to onset of effects	Following ingestion, noticeable in 10–20 minutes; full experience 30–60 minutes (10)
Duration of effects	Strongest effects at about 2–4 hours after ingestion, usually lasts 5–7 hours; weak after-effects can be felt as late as the next day (6)
Half-life	Mitragynine: 3.4 hours; 7-HMG: 2.5 hours (6)
Metabolism	Mitragynine: phase I and II; inhibits CYP450: 3A4, 2D6, 1A2 (11)
Elimination	Mitragynine and 7-HMG primarily by urine (11)

[a] Abbreviation: 7-HMG=7-α-hydroxymitragynine.

The above case highlights the difficulty faced by millions of Americans with opioid use disorder in identifying ways to manage their addiction. It is incumbent upon physicians to educate themselves about the properties of kratom, the limited ability of standard toxicology screens to detect this substance, and the potential to treat patients with addiction and chronic pain. Given the DEA's recent delay in designating kratom as a Schedule I substance, the translation of these findings to clinical practice is of imminent importance as researchers are searching for ways to treat pain and addiction while addressing the opioid epidemic.

TABLE 2. Toxicological Profile of Kratom

Withdrawal symptoms	Generally range from non-existent to mild. Physical symptoms: dysphoria, nausea, hypertension, irritability, myalgia, diarrhea, arthralgia, yawning, and insomnia for chronic users (5); psychological symptoms: agitation, anxiety, irritability, and depression for chronic users (14)
Short-term side effects	Sleep problems, nausea, constipation, itching, sweating, and temporary erectile dysfunction (6)
Long-term side effects	Tremor, hyperpigmentation, and weight loss (6)
Infrequent effects	Liver toxicity, seizure (alone or combine with other substances), coma, intrahepatic cholestasis, psychotic symptoms, adult respiratory distress syndrome, and hypothyroidism (5)
Fatalities	Reports of death when mixed with tramadol metabolite O-desmethyltramadol, propylhexedrine, and various psychiatric medications (5); no solid evidence exists that kratom was the sole cause of an individual's death (13)

[a] Abbreviation: 7-HMG=7-α-hydroxymitragynine.

KEY POINTS/CLINICAL PEARLS

- Kratom (Mitragyna speciosa) is a drug of abuse increasingly utilized in the United States due to its current legality, analgesic properties, and ability to mitigate opioid withdrawal symptoms.
- Kratom is now a focus of Drug Enforcement Agency scrutiny amidst the increasing opioid epidemic in the United States.
- Kratom is unique in its ability to produce stimulant-like effects at low doses (1–5 grams) and opioid-like effects at higher doses (5–15 grams).
- The compound mitragynine pseudoindoxyl, synthesized from the kratom alkaloid mitragynine, shows promise in treating pain and opioid dependence without the adverse effects typical of opioids.

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CASE REPORT

Fugue-Like State and Psychosis in the Context of Chronic Cannabis Use

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Occurring in 0.2%–7.2% of the general population, dissociative amnesia is one of three disorders listed in the DSM-5 under dissociative disorders (1). The disorder refers to a heterogeneous collection of psychological presentations, distinctive in the dimensions of content and timing (2). It is characterized by an inability to recall autobiographical information that may occur after an inciting traumatic event (3). The amnesia can be complete (generalized amnesia) or limited to a life event (localized amnesia). It is usually retrograde with an identifiable onset and termination. Dissociative amnesia involving purposeful travel or arbitrary wandering is termed dissociative fugue, a DSM-5 specifier that occurs in approximately 0.2% of the general population (1). Although the ICD-10 lists a dissociative fugue as a unique diagnosis, clinical and neuroimaging research findings support its new designation in the DSM-5 (4).

Retrospective and prospective studies tend to support a pathopsychological model implicating antecedent psychological stressors (5). It is commonly diagnosed in survivors of child abuse, sexual abuse, natural disasters, and war. A time lag may exist between the inciting trauma and the dissociative amnesia. Functional neuroimaging studies, which compared patients during amnesic episodes to age-matched healthy controls, have demonstrated hypometabolism in the right inferolateral prefrontal cortex in patients, implicating this region in the neural basis of dissociative amnesia (6). Dissociative amnesia usually remits spontaneously after removal from the traumatic situation. Hospitalization is required for patients who pose a danger to themselves or others and also offers separation from the stressful situation. Psychotherapy is the cornerstone of management for persistent cases. Hypnosis or drug-facilitated interviews may be helpful to recover lost memories and to manage the impact of resurfaced ones (2).

CASE

"Mr. A" is a 51-year old Caucasian male brought in by police after being found sleeping outside a 7-Eleven 200 miles from his home in a neighboring state. According to initial police statements, the patient was unable to recall his name, home address, or the date. Throughout the interview, the patient was tearful, hostile, delusional, and tangential, often forgetting the initial question. The patient endorsed recent auditory hallucinations but refused to provide details. He denied visual hallucinations.

The patient had a history of aggravated sexual assault on a minor for which he served 20 years in prison. During his time in prison, he reported having experienced auditory hallucinations and was treated with risperidone. He refused to go into further detail about his time in prison but did endorse increased stress and the onset of numerous gaps in his memory. He explained that he lives with his ex-wife and works at a local car wash. He refused to discuss his childhood or family life, including any emotional, physical, or sexual abuse. The patient endorsed daily cannabis use since 15 years old.

Upon arrival, his dress and personal hygiene were poor. His eye contact and motor activity were normal. His behavior was threatening and restless. When attempting to complete a Mini Mental State Examination, the patient was unfocused, easily distracted, and uncooperative, refusing to answer several questions. He was disoriented to name, date, location, and situation. His physical examination was insignificant, and he was found to be in good physical health. Laboratory results were significant for an AST of 53 IU/L and ALT of 59 IU/L. His serum alcohol level was negative. His urine drug screen was positive for cannabis but did not detect other substances. An initial diagnosis of other psychotic disorders was made based on the presence of auditory hallucinations of unknown duration based on DSM-5 criteria. Additionally, the patient was diagnosed with cannabinoid use disorder based on patient history and a positive urine drug screen.

Once admitted to the unit, the patient was started on olanzapine (10 mg p.o. daily) for psychosis. Later that day, he had an episode of acute psychosis requiring chemical restraints, including haloperidol (10 mg via intramuscular injection once), diphenhydramine (50 mg via intramuscular once), and lorazepam (1 mg via intramuscular once).

The following day, the patient was tearful as he revealed details of an emotionally distressing altercation with a work associate that resulted in his manager threatening to fire him. He recalled waking up days later and "needed to do something important," which led to his cross-state trip. Over the subsequent

KEY POINTS/CLINICAL PEARLS

- The DSM-5 diagnosis of dissociative amnesia is characterized by an inability to recall autobiographical information that may occur after an inciting traumatic event; dissociative amnesia involving purposeful travel or arbitrary wandering is termed dissociative fugue, a DSM-5 specifier to dissociative amnesia.
- While cannabis and other substances are exclusion criteria for dissociative amnesia and schizophrenia in the DSM-5, it is important to consider alternate diagnoses so that patients can be managed appropriately.
- Further research into the role of cannabis in possible common neurobiological mechanisms underlying both dissociative symptoms and psychotic symptoms may enhance our management of these patients.

days, the patient slowly began to regain pieces of his amnesic episode. He was able to recall driving a distance, abandoning his car once it broke down, and hitching a ride from "evil individuals."

Olanzapine was changed to an oral disintegrating form at 10 mg p.o. q.h.s. Over the following 3 days, the patient's mood and cognition improved, as did his acute psychosis. He attended several therapy sessions aimed at developing coping skills. He continued to refuse to discuss his past auditory hallucinations in detail or his time in prison and was never able to regain complete memory of the events during his fugue-like state. The patient met his treatment goals and was discharged in stable condition.

DISCUSSION

The presence of a fugue-like state in the context of psychosis and cannabis use is rarely discussed in recent literature. A differential diagnosis of dissociative amnesia with dissociative fugue must be considered despite cannabis use and as an explanation for what were originally considered fugue-like symptoms. The patient in the above case was found far from his hometown, initially unable to recall important autobiographical information, which caused significant distress and impairment, resulting in his mental health detainment. It has been demonstrated that cannabis along with 3,4-methylenedioxy-methamphetamine, cocaine, and other substances can cause and increase dissociative symptoms (7) and that cannabis and other substances can elicit psychotic symptoms (8). The literature has discussed a possible link between dissociative symptoms and psychotic symptoms in patients with a history of sexual child abuse (9) or certain medications (10). A 2013 case report described a 43-year-old male who entered a fugue-like state during an acute psychotic episode after initiating tacrolimus (10). After treating the acute psychotic episode and discontinuing tacrolimus, his condition improved.

In our case, the patient's history of regular cannabis use precludes a DSM-5 diagnosis of dissociative amnesia due to criterion C of dissociative amnesia; however, his dissociative symptoms do meet criteria for unspecified dissociative disorder. In addition, due to his cannabis use and psychotic symptoms, he meets all criteria in the DSM-5 for substance-induced psychotic disorder. While cannabis use has been shown to independently cause either psychosis or dissociation in patients (7, 8), we propose further exploration into the role of cannabis, as it may highlight common neurobiological mechanisms underlying both dissociative and psychotic symptoms. Understanding the mechanism by which cannabis produces these symptoms might enhance our current understanding of both and aid in the management of these patients.

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Call for Applications to Join the 2017 Editorial Board

The *American Journal of Psychiatry—Residents' Journal* is now accepting applications to join the 2017–2018 Editorial Board for the following positions:

SENIOR DEPUTY EDITOR POSITION 2017

Job Description/Responsibilities

- Frequent correspondence with AJP-Residents' Journal Editorial Board and AJP professional editorial staff, including a monthly conference call.
- Frequent correspondence with authors.
- Peer review manuscripts on a weekly basis.
- Make decisions regarding manuscript acceptance.
- Work with AJP editorial staff to prepare accepted manuscripts for publication to ensure clarity, conciseness, and conformity with AJP style guidelines.
- Coordinate selection of book review authors and distribution of books with AJP professional editorial staff.
- Recruit authors and guest editors for the journal.
- Manage the *Test Your Knowledge* questions and work closely with authors in developing Board-style review questions for the *Test Your Knowledge* section.
- Collaborate with the Editor-in-Chief in selecting the 2018 Senior Deputy Editor, Deputy Editor, and Associate Editors.
- Attend and present at the APA Annual Meeting.
- Commitment averages 10–15 hours per week.

Requirements

- Must be an APA resident-fellow member.
- Must be starting as a PGY-3 in July 2017, or a PGY-4 in July 2017 with plans to enter an ACGME fellowship in July 2018.
- Must be in a U.S. residency program.

Selected candidate will be considered for a 2-year position, including advancement to Editor-in-Chief.

DEPUTY EDITOR POSITION 2017

Job Description/Responsibilities

- Frequent correspondence with Residents' Journal Editorial Board and AJP professional editorial staff, including a monthly conference call.
- Frequent correspondence with authors.
- Peer review manuscripts on a weekly basis.
- Make decisions regarding manuscript acceptance.
- Work with AJP editorial staff to prepare accepted manuscripts for publication to ensure clarity, conciseness, and conformity with AJP style guidelines.
- Prepare a monthly *Residents' Resources* section for the Journal that highlights upcoming national opportunities for medical students and trainees.
- Recruit authors and guest editors for the journal.
- Collaborate with the Editor-in-Chief in selecting the 2018 Senior Deputy Editor, and Associate Editors.
- Attend and present at the APA Annual Meeting.
- Commitment averages 10 hours per week.

Requirements

- Must be an APA resident-fellow member.
- Must be a PGY-2, PGY-3, or PGY-4 resident starting in July 2017, or a fellow in an ACGME fellowship in July 2017.
- Must be in a U.S. residency program or fellowship.

This is a 1-year position only, with no automatic advancement to the Senior Deputy Editor position in 2018. If the selected candidate is interested in serving as Senior Deputy Editor in 2018, he or she would need to formally apply for the position at that time.

ASSOCIATE EDITOR POSITIONS 2017

(two positions available)

Job Description/Responsibilities

- Peer review manuscripts on a weekly basis.
- Make decisions regarding manuscript acceptance.
- Recruit authors and guest editors for the journal.
- Collaborate with the Senior Deputy Editor, Deputy Editor, and Editor-in-Chief to develop innovative ideas for the Journal.
- Attend and present at the APA Annual Meeting.
- Commitment averages 5 hours per week.

Requirements

- Must be an APA resident-fellow member.
- Must be a PGY-2, PGY-3, or PGY-4 resident in July 2017, or a fellow in an ACGME fellowship in July 2017.
- Must be in a U.S. residency program or fellowship.

This is a 1-year position only, with no automatic advancement to the Deputy Editor or Senior Deputy Editor position in 2018. If the selected candidate is interested in serving as Deputy Editor or Senior Deputy Editor in 2018, he or she would need to formally apply for the position at that time.

MEDIA EDITOR POSITION 2017

(one position available)

Job Description/Responsibilities

- Manage our Twitter and Facebook accounts
- Oversee podcasts
- We are open to many suggestions within reason
- Collaborate with the associate editors to decide on content
- Collaborate with Senior Deputy Editor, Deputy Editor, and Editor-in-Chief to develop innovative ideas for the Journal.
- Attend and present at the APA Annual Meeting.
- Commitment averages 5 hours per week.

Requirements

- Must be an APA resident-fellow member.
- Must be an upcoming PGY-2, PGY-3, or PGY-4 resident in July 2017, or a fellow in an ACGME fellowship in July 2017.
- Must be in a U.S. residency program or fellowship.

This is a 1-year position only, with no automatic advancement to the Deputy Editor or Senior Deputy Editor position in 2018. If the selected candidate is interested in serving as Deputy Editor or Senior Deputy Editor in 2018, he or she would need to formally apply for the position at that time.

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For all positions, applicants should e-mail a CV and personal statement of up to 750 words describing their reasons for applying, as well as any ideas for journal development to Rachel.Katz@yale.edu.

The deadline for applications is 3/31/2017.

AMERICAN PSYCHIATRIC ASSOCIATION

APA/APAF FELLOWSHIPS

APA/APAF Leadership Fellowship
The aim of the APA/APAF Leadership Fellowship is to develop leaders by providing opportunities for residents to engage, interact and participate in organized psychiatry at a national level and further develop their professional leadership skills, networks and psychiatric experience.

Child and Adolescent Psychiatry Fellowship
The Child and Adolescent Psychiatry Fellowship is designed to promote interest and a career in child and adolescent psychiatry.

Diversity Leadership Fellowship
The Diversity Leadership Fellowship is designed to develop leadership to improve the quality of mental health care for minority groups at risk and underrepresented in psychiatry.

Jeanne Spurlock Congressional Fellowship
The aim of the fellowship is to provide an opportunity for a senior psychiatry resident with significant interest in child and/or minority mental health advocacy to work in a congressional office.

Public Psychiatry Fellowship
The aim of the Public Psychiatry Fellowship is to create the next generation of leaders in public psychiatry. This program creates opportunities for residents to engage in several mentorship sessions, conduct public psychiatry program site visits, and interact with thought leaders in the field of publicpsychiatry.

SAMHSA-Funded Minority Fellowship Program
The goal of the APA SAMHSA Minority Fellowship is to enhance the knowledge and capabilities of racial and ethnic minority psychiatry residents to teach, administer, conduct services research and provide culturally competent, evidence-based mental health services to minorities and underserved populations. Minorities and applicants interested in serving minority and/or underserved populations are encouraged to apply.

SAMHSA-Funded Substance Abuse Minority Fellowship Program
The goal of the APA SAMHSA Substance Abuse Minority Fellowship is to enhance the knowledge and capabilities of racial and ethnic minority psychiatry residents to teach,administer, conduct services research and provide culturally competent, evidence-based mental health and substance abuse services to minorities and underserved populations. Minorities and applicants interested in serving minority and/or underserved populations are encouraged to apply.

Psychiatric Research Fellowship
The fellowship provides funding for an early research career psychiatrist to design and conduct a health services/policy-related research study using national data housed at the APA. The fellow's research activities will be carried out under the supervision and guidance of a mentor at his/her institution in collaboration with his/her mentor(s) at the APA Division of Research.

FOR MORE INFORMATION VISIT US AT ONLINE AT WWW.PSYCHIATRY.ORG/FELLOWSHIPS

Residents' Resources

Here we 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 Oliver Glass, M.D., Deputy Editor (glassol@ecu.edu).*

MARCH DEADLINES

Fellowship/Award, Organization, and Deadline	Brief Description and Eligibility	Contact and Website
American Academy of Child and Adolescent Psychiatry (AACAP) Pilot Research Award for Child Psychiatry Residents and Junior Faculty AACAP **Deadline: March 30, 2017**	Offers $15,000 for child psychiatry residents and junior faculty who have an interest in beginning a career in child and adolescent psychiatry research. Recipients have the opportunity to submit a poster presentation on their research for AACAP's 64th Annual Meeting in Washington, DC, 2017. The award also includes the cost of attending the AACAP Annual meeting for 5 days. • Enrolled in a child psychiatry residency or fellowship or have a faculty appointment in an accredited medical school but no more than 2 years of experience following graduation from training. Candidates must not have any previous significant, individual research funding in the field of child and adolescent mental health. • AACAP member	Department of Research, Training, and Education E-mail: research@aacap.org Phone: (202) 587-9664 http://www.aacap.org/AACAP/Awards/Resident_and_ECP_Awards/Pilot_Research_Award_Child_Psychiatry_Residents_Junior_Faculty.aspx
AACAP Pilot Research Award, supported by Pfizer, Inc. AACAP, Supported by Pfizer **Deadline: March 30, 2017**	Offers $15,000 for general psychiatry residents who have an interest in beginning a career in child and adolescent mental health research. Recipients have the opportunity to submit a poster presentation on their research for the AACAP 64th Annual Meeting in Washington, DC, 2017. The award also includes the cost of attending the AACAP Annual Meeting for 5 days • Candidates must be enrolled in a general psychiatry residency. Candidates must not have any previous significant, individual research funding in the field of child and adolescent mental health. • AACAP member.	Department of Research, Training and Education E-mail: research@aacap.org Phone: (202) 587-9664 http://www.aacap.org/aacap/Awards/Resident_and_ECP_Awards/AACAP_Pilot_Research_Award.aspx
AACAP Pilot Research Award for Learning Disabilities for Child Psychiatry Residents and Junior Faculty AACAP, Supported by the Elaine Schlosser Lewis Fund **Deadline: March 30, 2017**	Offers $15,000 for child and adolescent psychiatry residents and junior faculty who have an interest in beginning a career in child and adolescent mental health research. The recipient has the opportunity to submit a poster presentation on his or her research for the 64th Annual Meeting in Washington, DC, 2017. • Enrolled in a child psychiatry residency or fellowship or have a faculty appointment in an accredited medical school but no more than 2 years of experience following graduation from training. • Candidates must not have any previous significant, individual research funding in the field of child and adolescent mental health. • AACAP member	Department of Research, Training, and Education E-mail: research@aacap.org Phone: (202) 587-9664 http://www.aacap.org/AACAP/Awards/Resident_and_ECP_Awards/AACAP_Pilot_Research_Award_for_Learning_Disabilities.aspx
American Psychiatric Association (APA) Resident Recognition Award APA **Deadline: March 31, 2017**	The Resident Recognition Award is presented annually to outstanding psychiatry residents or fellows from each department or institution who exemplifies one or more APA values. Multiple awards are given each year. Psychiatry residents or fellows who: • Are APA members; • Are in good standing in their general psychiatry or fellowship program.	Claire Van Wagner E-mail: cvanwagner@psych.org https://www.psychiatry.org/psychiatrists/awards-leadership-opportunities/awards/resident-recognition-award

Author Information for *The Residents' Journal* Submissions

The Residents' Journal accepts manuscripts authored by medical students, resident physicians, and fellows; attending physicians and other members of faculty cannot be included as authors.

To submit a manuscript, please visit http://mc.manuscriptcentral.com/appi-ajp, and select a manuscript type for AJP Residents' Journal.

1. **Commentary:** Generally includes descriptions of recent events, opinion pieces, or narratives. Limited to 500 words and five references.
2. **History of Psychiatry:** Provides a historical perspective on a topic relevant to psychiatry. Limited to 500 words and five references.
3. **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. This article type should also include a table of Key Points/Clinical Pearls with 3–4 teaching points.
4. **Clinical Case Conference:** A presentation and discussion of an unusual clinical event. Limited to 1,250 words, 10 references, and one figure. This article type should also include a table of Key Points/Clinical Pearls with 3–4 teaching points.
5. **Original Research:** Reports of novel observations and research. Limited to 1,250 words, 10 references, and two figures. This article type should also include a table of Key Points/Clinical Pearls with 3–4 teaching points.
6. **Review Article:** A clinically relevant review focused on educating the resident physician. Limited to 1,500 words, 20 references, and one figure. This article type should also include a table of Key Points/Clinical Pearls with 3–4 teaching points.
7. **Drug Review:** A review of a pharmacological agent that highlights mechanism of action, efficacy, side-effects and drug-interactions. Limited to 1,500 words, 20 references, and one figure. This article type should also include a table of Key Points/Clinical Pearls with 3–4 teaching points.
8. **Perspectives in Global Mental Health:** This article type should begin with a representative case or study on psychiatric health delivery internationally, rooted in scholarly projects that involve travel outside of the United States; a discussion of clinical issues and future directions for research or scholarly work should follow. Limited to 1,500 words and 20 references.
9. **Arts and Culture:** Creative, nonfiction pieces that represent the introspections of authors generally informed by a patient encounter, an unexpected cause of personal reflection and/or growth, or elements of personal experience in relation to one's culture that are relevant to the field of psychiatry. Limited to 500 words.
10. **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.
11. **Book and Movie Forum:** Book and movie reviews with a focus on their relevance to the field of psychiatry. Limited to 500 words and 3 references.

Upcoming Themes

If you have a submission related to the themes shown at right, contact the Section Editor listed below the topic. ***Please note that we will consider articles outside of the theme.***

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 Katherine Pier, M.D., Editor-in-Chief (katherine.pier@mssm.edu).

LGBT Mental Health

Mark Messih, M.D., M.Sc., mark.messih@gmail.com

War, Terror, and Psychopathology

Anna Kim, M.D., anna.kim@mountsinai.org