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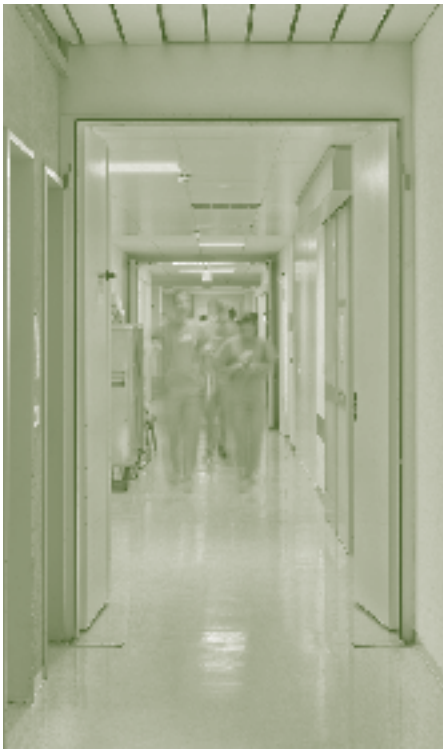

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EDITORIAL

Catatonia and Delirium: A Challenge in General Hospital Psychiatry

Kamalika Roy, M.D.

Catatonia was historically described by Kahlbaum in 1874 as a disease with a cyclic course of melancholy, mania, stupor, and confusion. Kraepelin later postulated that catatonia was a feature of dementia praecox. Evidence has since shown that catatonia is largely associated with bipolar and depressive disorders (43%–45%) and general medical conditions (25%) (1). In clinical settings, catatonia often goes undetected. For example, it was only formally diagnosed in 1.3% of patients in acute psychiatric settings, while two or more catatonic symptoms were present in 18% of cases (2). This disconnect could be due to heterogeneity of clinical presentations wherein catatonic symptoms often overlap with the motoric symptoms of delirium and/or substance withdrawal.

According to DSM-5, delirium prohibits a formal diagnosis of catatonia due to another medical condition. Because of the overlapping symptoms, many remain convinced that one diagnosis necessarily excludes the other. Most accept that catatonia describes motor symptoms and not a disturbed sensorium.

Francis and Lopez-Canino (3) systematically demonstrated the presence of catatonic symptoms in delirium patients. They proposed that catatonia may account for the motor components of hypoactive delirium. In another exploratory study, catatonia was present in 12.7% (using DSM-5 criteria) to 32% (using the Bush-Francis Catatonia Rating Scale) of subjects with Delirium Rating Scale-Revised-98 positive delirium (4). These authors suggested that the requirement of clear consciousness for a diagnosis of catatonia in medical patients is more hypothetical than clinically useful. They also showed a high association between catatonia symptoms and the hypoactive or mixed type of delirium. Another study found that in some cases of alcohol and sedative-hypnotic withdrawal, patients met criteria for "withdrawal delirium" and "withdrawal catatonia" simultaneously (5).

The debate over whether catatonia and delirium exist on a spectrum has treatment implications.

The debate over whether catatonia and delirium exist on a spectrum has treatment implications, as delirium is often treated with antipsychotics, which increase the risk of malignant catatonia. In contrast, benzodiazepines are often the first line of treatment in catatonia despite potentially worsening some components of delirium. Roy et al. (6) described three such cases in which catatonia symptoms worsened with benzodiazepines and ultimately remitted with memantine, which may hold promise for the treatment of patients with such a degree of symptom overlap.

Perhaps antiquated terms such as "agrypnia excitata" or "excited sleeplessness" are more clinically useful and point toward a distinct subtype than delirium or catatonia in such cases (5). Without nosological accuracy, we may delay effective treatments like benzodiazepines and ECT, thereby decreasing treatment responsiveness by the time ECT is initiated (given the delay in catatonia treatment is associated with less responsiveness to ECT), prolong hospitalizations, and increase mortality (7). Furthermore, if we fail to capture the syndrome we hope to describe, research becomes imprecise. Ultimately, our ability to treat catatonia before it becomes malignant will rely on our ability to detect it even when delirium is evident in clinical settings. These complicated questions affect the prognoses of our patients and are the kinds of challenges that psychiatrists in the general hospital face every day.

Dr. Roy is a fellow in psychosomatic medicine in the Department of Psychiatry, University of Michigan, Ann Arbor, Mich., and Guest Editor for this issue of the *Residents' Journal*.

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ARTICLE

Improving Communication Between Patients and Providers Surrounding the Legal Basis for Admission

Robert Loman, M.D.

Communication in health care is critical, more so in psychiatry. It could be called the art of psychiatry if neurobiology is the science of it. Although medical schools encourage learning scientific standards of disease and nomenclature, it is just as important to learn the essentials of effective communication. By refining the skills of communication, the treatment outcome could be positively influenced and a sustained therapeutic relationship can be achieved. Recently, medical education has recognized communication as the area that needs the most improvement. The Accreditation Council for Graduate Medical Education and the American Board of Psychiatry and Neurology have integrated communication as a subsection of the psychiatry milestone project.

In the July 2015 edition of *Psychiatry Milestones*, it is recommended that psychiatry residents recognize failures in teamwork and communication as a cause of preventable patient harm (1). Psychiatry residency programs evaluate the development of communication skills on different levels. To achieve a level 2 in the category of patient safety and health care team, psychiatry residents are expected to use structured communication tools, such as checklists, and safe hand-off procedures to prevent adverse events (1).

It was found that communication skills tend to decline during the time of medical training (2). Contributing factors identified include the "emotional and physical brutality" of medical training, which has been found to erode the pillars of communication (2).

Currently, in the Psychiatry Milestone project 1, "Communication and Patient Safety" is a subheading under "Patient Safety and the Health Care Team." To highlight its importance, a separate section in the milestones should be dedicated to communication.

CASE SCENARIO

A code orange was called as a suicide alert over the hospital speaker on the medical floor. On interview, the patient revealed that he had a specific suicidal plan to hang himself with a rope. After careful evaluation, it was determined that he met the criteria for inpatient psychiatric hospitalization. He was not happy about staying in the hospital and tried to convince the psychiatrist that he did not need to be hospitalized.

The explanation of involuntary and voluntary processes of admission only upset him further. It was difficult to balance the patient's rights and autonomy with concerns about patient safety. Establishing a therapeutic alliance became even more important considering the ongoing disagreement about the need of inpatient psychiatric care.

After reading the voluntary admission form carefully to the patient, he was at first reluctant to sign it and requested to go home. He posed a risk of harm to himself, and therefore inpatient psychiatric hospitalization was required and allowing him to go home was not possible. He was unfamiliar with this process, since he had no history of inpatient psychiatric admission.

In the back of my mind I thought, "How can I communicate this in a way the patient understands the importance of his safety?" The patient felt as though he was being placed in a prison and all his rights were being taken away from him. After some consideration, he agreed to voluntary commitment, and after 5 days of treatment he was ready for discharge.

At the end, he expressed thankfulness to the treatment team for caring for him during this stressful time in his life. In hindsight, one of the difficulties in communication with this patient was the balance between the physician-patient relationship, which might have benefitted from longer explanations to questions the patient posed and directly answering the patient's questions to avoid confusion. While explaining the inpatient hospitalization process to the patient, I found that I provided long responses to the questions the patient had. In hindsight, providing short concise responses may have aided in the understanding of his concerns.

DISCUSSION

Although the legal standards differ from state to state, generally, criteria for inpatient commitment include presence of mental illness, dangerous behavior toward self or others, and the need for treatment (3). Rights in all psychiatric admissions include the right to refuse medications and the right to meet with legal counsel, and in some cases of involuntary admission, committing a patient requires that two separate physicians independently evaluate a patient and conclude that the patient would pose an imminent danger to him- or herself or to others that might be modified with hospitalization (4).

Communication becomes especially important for patients who suffer from mental illness. Psychiatrists especially need the language, communication skills, and empathy to com-

municate effectively with some of the most challenging patients in the population.

Some ways that health care workers can enhance communication include avoiding medical jargon and having the patient repeat back his or her understanding of the intended concept. Simple measures such as offering food or water can help to form sound therapeutic relationships between patients and their health care providers.

A meta-analysis was conducted to study whether enhanced communication skills of the clinician can affect clinical outcomes (5). The investigator found that effective communication exerts a positive influence not only on the emotional health of the patient but also on symptom resolution, functional and physiologic status, and pain control (5). Some of the elements that were considered to be effective communication involved the physician asking many questions about the patient's understanding of the presenting problem, concerns, and expectations (5). Other elements that were considered to be a part of what is regarded as effective communication include showing support and empathy during the encounter. Some limitations of this study include confounding factors, such as response to medications, as well as psychosocial factors, which also influence outcomes. Another study found that poor communication among patients and physicians can result when the physician focuses on technical aspects of diagnosis and treatment without eliciting the patient's values and goals (6). By focusing on the goals of treatment, physicians can enhance communication with their patients.

One way to enhance communication between providers and patients is through implementation of a treatment plan. In the state of Michigan, a treatment plan is referred to as an individual plan of service, and it is an assessment that is tailored to the health and safety needs of the patient. It is developed with the involvement of the patient and, when indicated, the patient's guardian and/or family. Individual plan of service includes the coordination of care by the mental health team, primary care provider, and any other health care providers. A plan is developed with a list of close contacts and the outlined steps to take in the event of a crisis. Dates and frequency of follow-up appointments and support services are arranged and communicated to the patient and/or guardian, as well as to the family (7).

One study evaluated the effect of individualized plan of service and the rate of hospital admissions and readmissions in 24 medically and psychosocially complex patients with high health care utilization. Six and 12 months after the implementation of individualized plan of service, hospital admissions decreased by 56% ($p<0.001$) and 50.5% ($p=0.003$), respectively (8). Thirty-day readmissions decreased by 66% ($p<0.001$) at 6 months and 51.5% ($p=0.002$) at 12 months after care-plan implementation (8). Limitations of this study include its small sample size and the generalizability of the data to psychiatric patients given the studied patients were not on psychiatric units when the treatment plan was formulated.

Communication was found to contribute to better adherence to pharmacotherapy in a study that examined patients with schizophrenia (9). Furthermore, patients with good therapeutic alliance with their physicians were also more likely to be compliant with medications (9). Qualities found in physicians that promote a better relationship with patients include being nonjudgmental and open to addressing the patient's concerns. The physician's understanding of the patient's perspective on his or her illness was found to be essential in establishing the ground work for effective communication (10).

Ways of enhancing communication include incorporating the "five A's" (assess, advise, agree, assist, and arrange) (10), described in detail in Table 1.

TABLE 1. Methods to Enhance Communication

Method	Description
Assess	Assess the situation and what next steps are going to be required.
Advise	After the nature of the situation has been determined, advise the patient of the next steps.
Agree	Agreement on partnership with patient in participation of the patient's care.
Assist	Assist in providing the needs for the patient.
Arrange	Arrange the actions that are required for the patient.

CONCLUSIONS

Incorporating formal teaching in communication skills within medical school and residency training programs will not only promote patient safety and compliance with medication, but also help in earning trust from patients. This will further help to build the basis of an empathic psychiatrist and possibly provide more effective ways of treating distressed minds.

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KEY POINTS/CLINICAL PEARLS

- Communication in the health care setting is in need of improvement.
- Incorporating the "five A's" (assess, advise, agree, assist, and arrange) can improve communication.
- During the involuntary hospitalization process, the patient has the right to refuse treatment.

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Hypokalemia and Psychosis: A Forgotten Association

Ella Hong, M.D.

CASE VIGNETTE

A 41-year-old African American woman with a past diagnosis of schizoaffective disorder and medical history of hypertension and chronic obstructive pulmonary disease (COPD) was brought to the emergency department for auditory hallucinations and paranoid delusions. Upon initial evaluation, she was irritable and agitated and reported active suicidal ideation associated with voices accusing her of being a murderer. She had recently moved into her brother's apartment due to financial hardship and was unemployed. Family history was notable for a brother with schizophrenia. The patient denied a history of illicit substance use. Medications included hydrochlorothiazide for hypertension and albuterol as needed for COPD. The patient had been maintained on clonazapam and paroxetine for depression for the last several years without recent medication adjustment but had run out of medications a week prior to admission. Routine laboratory tests on admission revealed a potassium level of 2.3 mEq/L and negative urine toxicology.

MANAGEMENT AND OUTCOME

Upon arrival, hypokalemia was treated with 60 mEq intravenously and 40 mEq p.o. of potassium chloride. Due to severe agitation and aggression, the patient required pharmacologic and physical restraints. Repeat potassium level was 2.9 mEq/L. Potassium chloride 60 mEq intravenously was again supplemented, and repeat level was 3.5 mEq/L before transfer to the floor. Hydrochlorothiazide was discontinued for possible contribution to hypokalemia. The patient's delusions and auditory hallucinations improved the next day of admission. Paroxetine and clonazepam were restarted based on the history of good response for depressive and anxiety symptoms. Neuroleptics were discontinued, as the patient no longer endorsed auditory hallucinations or paranoid delusions. She received 40 mEq p.o. of potassium chloride each day until her discharge, with a final level of 3.4 mEq/L on the day of discharge.

INDEX EPISODES

From chart review, the patient presented to the emergency department with various symptoms and was found to be hypokalemic in the span of 7 years. There were 11 hospital visits recorded, and in nine instances, a potassium level of 3.5 mEq/L and below was recorded. Out of these nine cases, seven cases were for psychiatric care, with a potassium range of 2.7 mEq/L–3.3 mEq/L. In the first episode of hypokalemia, the patient presented with insomnia and bizarre behavior. Her potassium level was 2.7 mEq/L, and she was on hydrochlorothiazide for hypertension. The second time, the patient presented to the emergency department with paranoia and auditory hallucinations. Her potassium level was 2.7 mEq/L, and the same medical management for hypertension was continued. On the third occasion, she presented with irritable mood and auditory hallucinations. She had been on paroxetine, trazodone, and lorazepam, as well as amlodipine for hypertension. Her potassium level was 3.1 mEq/L. On the fourth presentation she presented with paranoia and auditory hallucinations, and her potassium level was 3 mEq/L. The patient had been on quetiapine, sertraline, paroxetine, and trazodone in the past with recorded noncompliance.

DISCUSSION

Hypokalemia is an identifiable, clinically important but often overlooked condition in psychiatric patients. Compared to the general population, the prevalence of hypokalemia (20%) in acute psychiatric patients is surprisingly high (1). The above case highlights the clinical possibility that hypokalemia may cause symptoms of psychosis in psychiatric populations.

Hypokalemia can cause a wide range of clinical manifestations, such as muscle weakness and areflexic paralysis. Cardiac manifestations include arrhythmia and EKG changes (2). Neuropsychiatrically, hypokalemia may present with memory impairment, disorientation, and confusion. Hypokalemia may mimic neurovegetative symptoms, such as weakness, lethargy, apathy, fatigue, and depressed mood (2). Additionally, hypokalemia can mimic anxiety reactions, such as headache, irritability, nervousness, paresthesias, visual disturbances, and muscle discomfort (3).

More importantly, hypokalemia has previously been associated with psychotic exacerbations in patients with schizophrenia. One study revealed that in 259 patients with schizophrenia with acute exacerbation, 6.9% had dehydration, approximately 30% had hypokalemia and leukocytosis, and 66% showed elevated serum muscle enzymes (4). It was postulated in the study that increased endogenous catecholamine levels might lead to a decrease in plasma potassium.

Thus, the mechanisms of hypokalemia in acute exacerbation have been further explored. Based on several reports, antipsychotic agents are believed to cause hypokalemia by changes in adrenergic activity (5). In a study exam-

ining patients with different inpatient psychiatric diagnoses and the variance of hypokalemia, it was postulated that hyperadrenergic state might drive beta-2-recepter stimulation, causing influx of potassium into skeletal muscle, resulting in hypokalemic trend (6). Duration of illness could also affect the sensitivity or density of the beta-2-receptor, which was reflected in the results of one study showing that disorders of relatively short disease duration had lower mean serum potassium values than disorders of longer duration (7). Thus, diagnoses with high acuity like acute psychosis would reflect lower potassium values compared to other diagnoses in the maintenance phase.

It is interesting that several reports suggest hypokalemia was associated with acute decompensation of psychotic symptoms (8). According to a case study, two episodes of acute decompensation of chronic paranoid schizophrenia were related to hypokalemia from use of thiazide diuretics (8). Other variables were also mentioned, such as use of antipsychotic medication upon admission and possible medication noncompliance, which could deter the direct correlation between hypokalemia and psychosis. However, it is still notable to mention that an episode of acute worsening of psychosis was treated successfully by potassium supplement in this case (8). Another case report discussed a patient with a history of psychosis presenting with an acute psychotic decompensation who was treated adequately with intravenous potassium supplement (7). Although other factors were not explained in detail, such reports suggest the importance of recognizing and treating hypokalemia in acute psychotic decompensation and encouraging thorough medical evaluation for such presentations.

KEY POINTS/CLINICAL PEARLS

- In psychiatric patients, hypokalemia may mimic a wide variety of symptoms such as weakness, lethargy, apathy, fatigue, depressed mood, headache, irritability, nervousness, paresthesias, visual disturbances, and muscle discomfort.
- Evidence suggests that hypokalemia may serve as an important clinical condition in acute psychiatric diagnoses such as psychosis.
- Studies suggest hypokalemia results from hyperadrenergic state might drive beta-2-recepter stimulation, causing influx of potassium into skeletal muscle, and duration of illness could affect the sensitivity or density of the beta-2-receptor, reflecting that diagnoses with high acuity like acute psychosis would reflect lower potassium values compared to other diagnoses in the maintenance phase.

CONCLUSIONS

One must heighten the suspicion of all dimensions of illness, including investigating possible metabolic causes for acute decompensation of psychosis. The available literature is not sufficient to clearly justify the association of hypokalemia with psychosis and to impose absolute positive correlation with treating hypokalemia leading to improvement of psychosis. At this time, further systematic controlled studies into the association and causality of hypokalemia and psychosis are needed. However, management of serum potassium level could serve as a valuable way to better the overall outcome of symptoms in acute psychotic exacerbation.

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CASE REPORT

A Complex Case of Suspected Serotonin Syndrome

Anna Kim, M.D.

Serotonin syndrome is a potentially life-threatening state caused by an excess of serotonergic activity in the nervous system. Symptoms include mental status changes, autonomic instability, and neuromuscular hyperactivity (1). The present case report is of an elderly woman with moderate-to-severe presentation following psychotropic medication use.

CASE

"Ms. A" is a 60-year-old single, unemployed woman with a history of hypertension, hypothyroidism, and obstructive sleep apnea and a psychiatric history of bipolar I disorder, borderline personality disorder, and cannabis/alcohol/sedative/hypnotic (benzodiazepine) use disorder. She has had multiple psychiatric admissions since the age of 22 resulting from suicide attempts via overdose. Since this age, she has had 20 sessions of ECT. She was brought to the hospital for suicide attempt via drug overdose in the context of her husband's death.

The patient was medically cleared (temperature=36°C–36.5°C, heart rate=98–104 beats per minute, blood pressure=114–135 mm Hg/63–71 mm Hg, respiratory rate=20, and blood oxygen saturation level=95%–99%), fully oriented, and admitted to psychiatry. Urine toxicology, blood alcohol, acetaminophen, and salicylate levels were unremarkable. It is unclear whether over-the-counter medications were ingested. Past medications include lithium, divalproex, lamotrigine, phenelzine, vorioxetine, risperidone, and olanzapine. Prior to presentation, her psychiatrist placed her on phenelzine after a 3-week wash-out period from olanzapine and vorioxetine due to remission on phenelzine in the past. Phenelzine was titrated to 30 mg every morning and 45 mg every evening. Quetiapine was added for mood stabilization and for treatment of chronic, paranoid delusions that others intensely disliked her. Quetiapine was titrated to 50 mg every morning and 300 mg every evening. She was on these medications for 6 months preceding decompensation.

After admission, phenelzine was increased to 45 mg twice daily. Levothyroxine for hypothyroidism and modafinil for obstructive sleep apnea were continued. Amlodipine was discontinued, since the patient was normotensive. Following further titration, quetiapine was discontinued due to orthostatic hypotension. Risperidone was started, then cross-titrated to haloperidol at 2 mg every morning and 3 mg every evening due to the patient's concern of weight gain side effect and persistent psychotic symptoms on other antipsychotics. She was maintained on benztropine for extrapyramidal symptom prevention. Lamotrigine was started and titrated to a therapeutic level for further mood stabilization.

After 3 months of the above medication adjustments, Ms. A was noted over a couple of days to be oriented to herself only and resistant to passive flexion and extension of extremities. These symptoms, in combination with the recent addition of a neuroleptic, raised concern for neuroleptic malignant syndrome. However, the patient's vital signs were unchanged. Delirium workup revealed only a positive urinalysis that was subsequently treated. Catatonia was suspected, but the patient did not respond to a trial of benzodiazepines. Haloperidol was decreased to 2 mg at bedtime.

Overnight, the patient developed spontaneous, bilateral ocular and ankle clonus, mild diaphoresis, tremulousness, and diffuse 4+ hyperreflexia more pronounced in the lower extremities. Her 24-hour vitals were as follows: temperature=36.2°C–37.4°C, heart rate=77–107 beats per minute, blood pressure=108–158 mm Hg/52–105 mm Hg, respiratory rate=17–46, and blood oxygen saturation level=92%–98%. The rapid emergence of hyperreflexia and spontaneous clonus were suggestive of serotonin syndrome. Additionally, the patient remained afebrile, whereas temperatures of more than 38°C are typical in 87% of neuroleptic malignant syndrome cases. The patients' creatine phosphokinase levels oscillated between 200 IU/L and 400 IU/L and peaked at 693 IU/L, lower than the 1,000 IU/L–100,000 IU/L in neuroleptic malignant syndrome (2).

Psychotropic medications were discontinued. Supportive management with intravenous fluids was started with lorazepam as needed. The clonus and diaphoresis resolved. The patient's laboratory workup was normal. Video EEG showed nonspecific generalized slowing. However, the patient became intermittently tachycardic to 110 beats per minute and hypertensive to 170/90. She remained afebrile.

The patient was transferred to an intensive care unit for administration of cyproheptadine by nasogastric tube. Over several days, her heart rate and blood pressure normalized. Over the following week, her symptoms resolved, and she returned to baseline mental status. She was transferred back to psychiatry where she was stabilized on divalproex, vortioxetine, haloperidol, lorazepam, and benztropine.

DISCUSSION

Serotonin syndrome was first described in the 1960s in a patient with tuberculosis who had received meperidine with iproniazid (3). Since the death of Libby Zion, the daughter of a powerful attorney, in 1984 (4), there have been hundreds of case re-

TABLE 1. Common Drugs Associated With Serotonin Syndrome[a]

Drug	Mechanism of Action
Amphetamine, amphetamine derivatives	CNS stimulant: blocks presynaptic reuptake of norepinephrine and dopamine
Analgesics: fentanyl, meperidine, tramadol	CNS opioid receptor agonist, NRIs, SRIs
Antidepressants	SSRIs, SNRIs, MAOIs
Mood stabilizers	Lithium: unknown, alters nerve and muscle cell Na+ transport VPA: unknown, increases CNS concentrations of GABA
Antimigrane drugs: ergots, triptans	Ergots: 5-HT1D agonist; triptans: 5-$HT_{1B/1D}$ agonist
Antiemetics: metoclopramide, ondansetron	CNS and PNS: antagonizes dopamine receptors; modulates serotonin receptors
Dextromethorphan	Centrally acting antitussive; active at multiple receptor types
Linezolid	Oxazolidinone antibacterial, reversible MAOI
Methylene blue	Potent MAO-A inhibitor, MAO-B inhibition at higher levels
Cocaine	CNS dopamine transporter protein blockade. 5-HT_3 antagonist
St. John's wort	Multiple mechanisms, including reuptake inhibition of multiple monamines
Tryptophan supplements	Amino acid, involved in biosynthesis of serotonin

[a] For further details, see references 13–17. CNS=central nervous system; GABA=gabaaminobutyric acid; MAO=monoamine oxidase; MAOI=monoamine oxidase inhibitor; Na+=sodium; SNRI=serotonin-norepinephrine reuptake inhibitor; SSRI=selective serotonin reuptake inhibitor; PNS=peripheral nervous system; VPA=valproate.

ports describing serotonin syndrome. A literature review published in 2000 examined 226 cases from the 1960s to the 1990s (5). The age of individuals with serotonin syndrome spanned from 5 years old to 82 years old but occurred commonly between ages 20 and 50. The male-to-female ratio was 1:1.7, and 90% of patients had at least one of the following disorders: depression (65.8%), headaches (19%), obsessive-compulsive disorder (9.7%), alcohol abuse (7.3%), and generalized anxiety (7.3%) (5).

Ingestion of multiple drugs that block serotonin reuptake can cause serotonin syndrome (see Table 1). Patients with no exposure to a selective serotonin reuptake inhibitor (SSRI) who acutely ingest an SSRI in large doses are at higher risk (6). Hunter's Criteria is one instrument used in making this clinical diagnosis and captures 84% of cases. Diagnosis using Hunter's Criteria requires any of the following symptoms in the setting of serotonergic agent use: spontaneous clonus, clonus induced by sudden dorsiflexion of the ankle plus agitation or diaphoresis, ocular clonus (slow continuous lateral eye movements) plus agitation or diaphoresis, tremor plus hyperreflexia, or hypertonia plus temperature above 38°C plus ocular clonus or inducible clonus (1). These symptoms may develop later, and presenting symptoms can include lethargy and disorientation (5).

There is no specific test for serotonin syndrome. Laboratory abnormalities are nonspecific but include elevated creatine phosphokinase, leukocytosis, transaminitis, and low serum bicarbonate (7). Measurement of serotonin levels has not been shown to be helpful (4).

Clinical presentation can range from mild to moderate to severe symptoms. Intensity of symptoms reflects the degree of serotonin toxicity. Consequently, intensity of treatment depends on clinical findings. Mild cases with hyperreflexia, intermittent tremor and without fever or tachycardia can be managed by discontinuing the offending agent and supportive care. Lack of improvement warrants transfer to a medical unit. Moderate cases involving tachycardia, hypertension, fever, diaphoresis, or clonus and severe cases involving rigidity, seizures, and unstable hemodynamics require additional treatment: hydration, sedation, and intubation (4, 8). Controlling repetitive isometric muscle contractions in episodes of agitation with benzodiazepines is crucial to prevent morbidity and mortality (4, 8). Restraints are contraindicated and may contribute to mortality. Cyproheptadine, a 5-HT_{2A} antagonist, is often given, although efficacy has not been clearly established (4, 8).

The presentation of the patient in the above case was moderate to severe. Additionally, the patient was taking phenelzine, a monoamine oxidase inhibitor (MAOI) strongly associated with severe cases due to irreversible inhibition of the enzyme (1). Because of this, the body must regenerate monoamine oxidase to resume prior levels of enzymatic activity, a process that may take weeks. Effects of the MAOI can persist after the drug has been cleared (9). A limitation of the present case re-

KEY POINTS/CLINICAL PEARLS

- Symptoms of serotonin syndrome include mental status changes, autonomic instability, and neuromuscular hyperactivity.
- Hunter's Criteria (sensitivity 84% and specificity 97%) is a measure commonly used to diagnose serotonin syndrome.
- Laboratory abnormalities seen are nonspecific but can include elevated creatine kinase, leukocytosis, transaminitis, and low serum bicarbonate.
- Treatment includes removal of the offending agent and supportive care.; cyproheptadine, a 5-HT_{2A} antagonist, is often given, although efficacy has not been clearly established.

port, however, is that discontinuation of all psychiatric medications would treat both serotonin syndrome and neuroleptic malignant syndrome and that timing of the improvement would correspond to both neuroleptic malignant syndrome (10) and severe serotonin syndrome with MAOI involvement (9). Moreover, it is possible that the patient had co-occurring substance use on the unit or an acute ingestion. Urine toxicology repeated on readmission to psychiatry was negative, but this test does not detect agents such as synthetic opiates and SSRIs (11).

There are no clear guidelines as to which psychiatric medications to restart in patients with a history of serotonin syndrome (12). The patient in the above case report was restarted on a regimen without an MAOI.

Most of the literature on serotonin syndrome dates from the 1990s to the 2000s. Future areas of investigation include the creation of risk assessments for serotonin syndrome, which include details correlating morbidity and mortality with symptom timing, type and dose of drug ingested, and diagnoses. Objective measures such as vital signs warrant inclusion.

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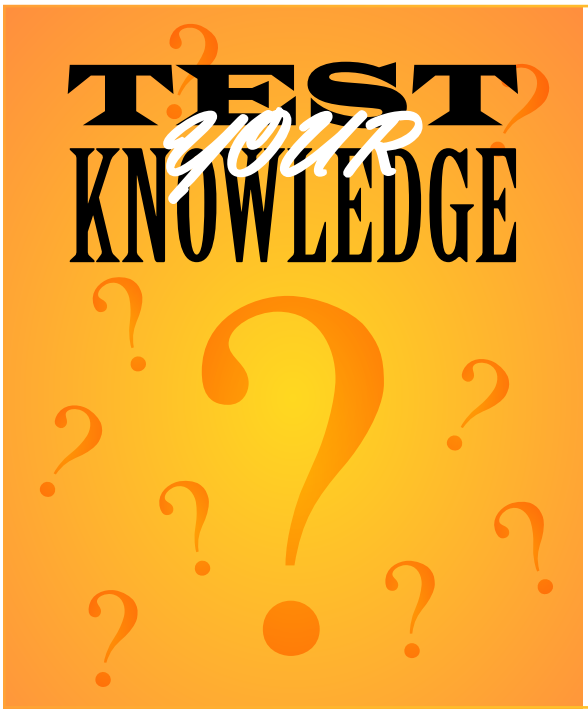

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**Please direct all inquiries to Rachel Katz, M.D., Senior Deputy Editor (rachel.katz@yale.edu).*

ARTICLE

Revisiting Decision-Making Capacity

Aparna Atluru, M.D.

Currently, a patient's right to self-determination is protected by the legal doctrine of informed consent, which comes with a very important caveat. As Justice Schroeder of the Kansas Supreme Court in the case of *Natanson v. Kline* (1960) (1) wrote: "Anglo-American law starts with the premise of thorough-going self-determination. It follows that each man is considered to be master of his own body, and he may, if he be of sound mind ...." This simply means that a person should have decision-making capacity in order to give informed consent. Hence, a thorough understanding of decision-making capacity is needed. Medical trainees of all levels are trusted with assessing decision-making capacity while obtaining informed consent, which is needed on three occasions: in treatment/procedure, when a patient refuses treatment/procedure, and when a patient decides on an alternate treatment/procedure. It is often believed that an experienced physician can assess decision-making capacity simply by following a gut feeling. However, when a physician encounters a case of questionable decision-making capacity, a focused assessment is necessary.

ASSESSMENT OF DECISION-MAKING CAPACITY

The first and foremost criterion for decision-making capacity is that a patient can make a decision and be able to convey it. Additionally, the patient should be able to acknowledge the problem and possible solutions. Decision-making capacity reflects functional abilities that a person needs to possess in order to make a specific decision (2). The patient should be able to provide a rationale for his or her decision. The patient can reject medical advice if he or she can understand the consequences and be able to express them. It is not wrong for physicians to persuade the patient toward an optimal treatment. However, coercion or deceptions should be avoided.

CASE EXAMPLE 1

"Mr. P" is a 58-year-old man recently diagnosed with prostate cancer. Urology had already recommended surgery. The patient wanted more options, which were given to him. He then had a conversation with radiation oncology but was still unsatisfied, and he requested an ethics consult. The patient refused to decide whether he wanted any treatment at all. He requested more information. In three subsequent meetings over the period of a month, more and more information was provided to him, but he still could not make a decision.

The patient in case 1 does not have decision-making capacity because a person who has such capacity should be able to communicate a decision when enough information is provided to them.

Many house staff only assess capacity when a patient's decision differs from either their own or the generally accepted medical practice. Rarely does a physician say that a patient agrees with the medical recommendation and therefore a capacity assessment is needed. It is important to remember that determining decision-making capacity involves evaluating the process the person uses to make the decision, not whether the final decision is in accord with the team's recommendation.

CASE EXAMPLE 2

"Martha" is a 52-year-old attractive woman who was just diagnosed with breast cancer. Surgery has the best chance of cure in her case, but she refuses surgery. When asked why, she responds that she wants to get into modeling and reports trying to break into the modeling industry for the last 20 years. She feels that now she has a real chance to make it and worries this can be affected by surgery.

Does the patient in case 2 have decision-making capacity?

Yes, this patient has decision-making capacity. She has a rationale for her decision, and though it may be far-fetched, she has an explanation.

CASE EXAMPLE 3

A 57-year-old HIV patient is admitted to the medical floor with what appears to be a bacterial infection. He refuses blood draws and blood cultures, as well as intravenous antibiotics, saying that he has fought HIV for the past 25 years and is tired of his quality of life. He does not want to be poked or prodded any longer. He says he knows there is a chance that he could die, but he prefers death, as he says he has lived a satisfactory life, and he prefers to die comfortably, even if the length of his life is reduced.

The resident physician tries to persuade the patient to agree to blood draws and intravenous antibiotics, explaining the need for these interventions and the chance of grave consequences if these interventions are not carried out.

The patient says he understands the consequences of not receiving treatment, says he knows he could die, and says he still does not want treatment. When the patient is asked why he decided to come to the hospital if he did not want any treatment, he replied, "to die peacefully."

Patients have a right to refuse life-saving treatment if they are not affected by depression, mental illness, or any other treatable conditions affecting the decision, including uncontrolled pain. Refusing life-saving treatment does not affect their decision-making capacity.

CAPACITY VS. COMPETENCE

It is of paramount importance that the concept of decision-making capacity is differentiated from "competence," as the

terms are frequently used interchangeably. The complexity of the issue, with the aid of clinical examples, is explained in this article.

Decision-making capacity is decided by clinicians regarding a specific question, while competence is decided by a court and is implemented over a functional domain, such as finances or medical decisions. Decisions regarding competence are legal decisions, which take medical evaluations into account, and are binding for the duration specified in a court order.

KEY POINTS/CLINICAL PEARLS

- Decision-making capacity and competence are not synonymous.
- Some patients may have decision-making capacity for some decisions but not others.
- The sliding scale of competence describes differences in the threshold or "level of capacity needed," depending on the risks and benefits with which a decision may come.
- All patients, even those with established psychiatric diagnoses, have decision-making capacity until proven otherwise.

THE VARIED THRESHOLD FOR ASSESSMENT OF DECISION-MAKING CAPACITY

Decision-making capacity is assessed at three levels of complexity depending on the clinical situation and the patient's acceptance or refusal of the treatment. Additional issues that must be considered when assessing decision-making capacity are the urgency of the situation and the risk versus the benefit involved. The sliding scale of competence, originally discussed by Dr. James Drane, describes differences in the threshold or "level of capacity needed" depending on the risks and the benefits. For example, a simple intervention with a low risk and high benefit (e.g., drawing blood to measure the hematocrit) may require only simple assent; however, a procedure with substantial risk and uncertain benefit requires great understanding on the patient's part.

Decision-making capacity is not an all or none phenomenon. All of us can have decision-making capacity for some decisions but not for other decisions.

CASE EXAMPLE 4

"Mr. A," a 62-year-old man with chronic alcoholism, is admitted after a fall. He underwent detoxification and was ready to go home when he was diagnosed with an aortic dissection. An ethics consultant determined that he did not have decision-making capacity to consent for surgery. Once recovered from surgery, the patient was ready to go home again. The case manager noted that the patient did not have decision-making capacity, hence he should be sent to a nursing home, where it would be safer for him. The same ethics consultant determined that that the patient did have decision-making capacity for the decision to go home.

Was the ethics consultant right the first time or the second time?

The consultant was right both times, as the assessment for questions of different magnitude will be of a different complexity. All patients have decision-making capacity until proven otherwise.

THE ROLE OF PSYCHIATRISTS

Mental illnesses may affect decision-making capacity, and in those patients, psychiatrists are best suited to assess capacity. They can be consulted in all other cases as well, at the discretion of the attending physician.

CASE EXAMPLE 5

A 49-year-old patient is receiving ECT on a regular basis, and the patient's anesthesiologist requests an ethics consult because he feels uncomfortable continuing to assist in performing ECT due to the patient experiencing multiple episodes of arrhythmia during an anesthesia induction.

In this patient, a decision-making capacity assessment should be done by a psychiatrist, who will understand the need for ECT and the effect of ECT, as well as the effect of the underlying mental illness on capacity.

CONCLUSIONS

The patient has a right to refuse a capacity assessment by a consultant, and his or her refusal must be respected. Patients should not be compelled or coerced into agreeing to an assessment. In cases when patients refuse to be assessed for decision-making capacity by psychiatrists or ethics consultants, the responsibility will fall upon the primary physician.

The possession of capacity has been described as a gateway to the exercise of autonomy. On occasion, a patient may refuse an essential treatment as an autonomous choice. In our society, even in situations in which autonomy is in conflict with beneficence, liberty and freedom for the patient should be protected. Therefore, everyone has capacity until proven otherwise.

A status approach, the fact that a patient has an established psychiatric diagnosis, should not solely dictate the presence or absence of decision-making capacity. A focused assessment of these patients is necessary. The values and beliefs of the patient may not necessarily be shared by the clinician, but they should be consistent, stable, and affirmed by the patient, and this value system should serve as a sieve through which the criteria of decision making is filtered (3).

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CASE REPORT

ANNA-1 Paraneoplastic Encephalomyelitis Presenting as Rapidly Progressing Dementia in a Man With Undiagnosed Small Cell Lung Cancer

Patrick K. Reville, M.P.H.
Catherine Steingraeber, M.D.

Patients with paraneoplastic encephalomyelitis may present with cognitive dysfunction, memory loss, confusion, psychiatric disturbances, rapidly progressive dementia, myoclonic jerks, and/or seizures. The present case report is of an elderly man with rapidly progressive cognitive decline and positive anti-neuronal nuclear antibody-1 prior to small cell lung carcinoma diagnosis.

CASE

"Mr. B" is a 64-year-old man with a past medical history of hypertension presented to our emergency department with his wife for safety concerns in context of rapid behavioral changes, mood lability, impulsivity, and memory problems. Review of his outside records showed that he initially presented to his primary care physician 2 months prior due to a 1-month history of memory deficits, confabulations, confusion, and executive function deficits in the context of an unintended 20-pound weight loss. Additionally, he had alternating irritability and euphoria, psychomotor agitation, nonspecific paranoia, increased speech, and impaired judgment. Neurologic examination was concerning for frontal and temporal lobe deficits. The patient's Montreal Cognitive Assessment score was 12/30. His vitamin B12 and thyroid-stimulating hormone levels were normal. His primary care physician started him on bupropion (150 mg twice daily) and mirtazapine (7.5 mg at night) for mood. An MRI of the patients' head revealed several foci of T2 hyperintensity within the periventricular white matter and centrum semiovale. Subsequently, an [^{18}F]FDG PET [^{18}F-fluorodeoxyglucose positron emission tomography] scan of the brain showed reduced metabolic activity in the posterior cingulate, precuneus, and lateral temporal-parietal lobes suggestive of Alzheimer's disease. Two days prior, the patient was seen at an outside emergency department because he was becoming too difficult to manage; olanzapine (5 mg a day) was added to his medication regimen.

The patient is a former smoker with a 50-pack/year history and drinks alcohol occasionally. He lives at home with his wife and worked as a repairman until 2 months prior when he had to resign due to cognitive decline. His family history is significant for a mother with depression, anxiety, and acute lymphoblastic leukemia as a child, as well as a sister with depression and anxiety.

Laboratory investigation in the emergency department revealed a hemoglobin level of 12.1 g/dl with normal mean corpuscular volume, hyponatremia with a sodium level of 134 mEq/L, a negative toxicology screen, an unremarkable urinalysis, and Montreal Cognitive Assessment score of 12/30. The patient was admitted for further workup. The consulting psychiatry service recommended discontinuing bupropion, mirtazapine, and olanzapine in favor of 50 mg of quetiapine at night.

Initially, the patient had episodes of increasing agitation and confusion that was managed with an as-needed quetiapine dose of 50 mg. About a week into his hospitalization, his agitation worsened, necessitating the addition of haloperidol (2 mg p.r.n.) to control his agitation. As his delirium was subsequently better controlled, it remained clear that his major neurocognitive disorder remained, with serial Montreal Cognitive Assessment examinations demonstrating consistent levels of cognitive impairment.

An initial EEG demonstrated focal spike and wave discharges from the right temporal chain, but a 24-hour video EEG re-demonstrated epileptiform abnormalities without clear electrographic seizures. Levetiracetam was loaded but discontinued after the results of the video EEG. Initial head CT was unremarkable, and a head MRI showed T2-fluid attenuated inversion recovery hyperintensity in the bilateral mesial temporal lobes. Initial laboratory workup included a heavy metal screen that was negative; iron studies demonstrating elevated ferritin, normal iron, total iron binding capacity, and transferrin indicating anemia of inflammation; and CSF analysis. Basic CSF analysis demonstrated normal cell count and glucose and an elevated level of CSF protein to 75 mg/dl. CSF gram stain and cultures were negative. CSF polymerase chain reaction tests were negative for herpes simplex virus, enterovirus, and Epstein-Barr virus. CSF angiotensin converting enzyme levels were normal. CSF serology demonstrated positive anti-neuronal nuclear antibody-1 (ANNA-1), with a titer of 1:512, no anti-*N*-methyl-D-aspartate receptor antibodies, and a normal Creutzfeldt-Jakob screen. A prostate-specific antigen screen was normal. Given the positive ANNA-1 antibody, a CT of the chest, abdomen, and pelvis was performed, which showed enlarged right paratracheal lymph nodes

but no other abnormalities. Subsequent bronchoscopy with endobronchial ultrasound-guided fine-needle aspiration of the paratracheal lymph nodes was performed. Initial cytology smear was suspicious for malignancy. Final pathology demonstrated cells positive for synaptophysin, chromogranin, and thyroid transcription factor-1 consistent with small cell lung carcinoma. A subsequent PET-CT performed showed hypermetabolic activity in the right paratracheal lymph nodes, mildly hypermetabolic nodes involving the right hilum, pretracheal, and paraesophageal nodes, and moderate uptake in the right fourth and tenth rib with sclerosis, suggestive of extensive-stage small cell lung carcinoma.

The patient's paraneoplastic syndrome was initially managed with 5 daily doses of intravenous immunoglobulin (400 mg/kg/day) and five plasmapheresis treatments three times weekly. His first cycle of chemotherapy with cisplatin and etoposide was started in the hospital. He was discharged home after a 23-day hospital stay. During initial follow-up with a lung oncologist, the decision was made to pursue curative intent with chemotherapy plus radiation. The patient's behavioral agitation is improved; however, his cognitive function remains diminished.

KEY POINTS/CLINICAL PEARLS

- Patients with paraneoplastic encephalomyelitis may present with cognitive dysfunction, memory loss, confusion, psychiatric disturbances, rapidly progressive dementia, myoclonic jerks, and/or seizures.
- Paraneoplastic encephalomyelitis occurs commonly in small cell lung, ovarian, and breast carcinomas, as well as thymomas and Hodgkin's lymphomas.
- While uncommon, paraneoplastic encephalomyelitis is an important diagnostic consideration in patients presenting with rapidly progressing dementia, as the cognitive symptoms usually present prior to detection of the malignancy.

DISCUSSION

Paraneoplastic syndromes are autoantibody-mediated disorders associated with underlying tumors (1). Neurological paraneoplastic syndromes occur in approximately 1 in 10,000 cancer patients (2). These conditions arise when systemic tumors express antigens normally found on neural tissue. Tumor directed immune responses recognize these antigens as non-self, leading to production of antibodies against the neural antigen. Paraneoplastic encephalomyelitis symptoms are often subacute and often precede detection of the tumor (2). Cancers associated with paraneoplastic encephalomyelitis include small cell lung carcinoma, ovarian carcinoma, breast carcinoma, thymoma, Hodgkin's lymphoma, and, rarely, prostate cancer (3).

The patient in the above case presented with records showing imaging suggestive of Alzheimer's dementia. On PET, the metabolic pattern characteristic of Alzheimer's disease is hypometabolism in parietotemporal areas, the posterior cingulate cortex, and the precuneus, as well as medial temporal lobes, mostly the entorhinal cortex and hippocampus (4). Interestingly, PET has utility in the diagnosis of paraneoplastic neurologic syndromes as well. In clinically suspected paraneoplastic encephalomyelitis, PET has a sensitivity and specificity of 75% and 87%, respectively, in diagnosing paraneoplastic encephalomyelitis (5). Clear indications for PET in patients with major neurocognitive disorder are not currently established, but the American College of Radiology and the American Society for Neuroradiology suggest it can be useful in patients with unexplained major neurocognitive disorder (6).

A case series of patients with ANNA-1 paraneoplastic encephalomyelitis shows similarities with the above case (7). The authors reported that the median age was 63 years old, 75% were men, and paraneoplastic encephalomyelitis preceded diagnosis of the malignancy in 73% of cases (7). Treatment of the primary tumor with or without immunotherapy was a predictor of improvement or stabilization of the neurologic sequelae (7). Similarly, in another case series of ANNA-1 paraneoplastic encephalomyelitis, tumor complete response was the only predictor of paraneoplastic encephalomyelitis stabilization, and immunotherapy did not modify the outcome regarding the tumor or paraneoplastic encephalomyelitis (8). The probability of survival at 30 months has been shown to be higher in small cell lung carcinoma patients with paraneoplastic encephalomyelitis than those without paraneoplastic encephalomyelitis (8). Similarly, a report in patients without clinically evident paraneoplastic encephalomyelitis showed that small cell lung carcinoma patients with detectable ANNA-1 antibodies in their serum were more likely to have: limited stage disease, a complete response to therapy, and longer overall survival compared with patients without ANNA-1 antibodies (9). Despite more positive prognosis for small cell lung carcinoma with ANNA-1 paraneoplastic encephalomyelitis, reports suggest that up to 65% of patients with ANNA-1 paraneoplastic encephalomyelitis and small cell lung carcinoma die of neurological complications rather than tumor progression (9). It is unclear whether paraneoplastic encephalomyelitis preceding small cell lung carcinoma diagnosis allows for earlier detection of disease and a potential survival benefit through lead-time bias or there is a biological relationship between these autoimmune processes and small cell lung carcinoma outcomes.

Despite the initial uncertainty in the above case, a relatively rapid diagnosis of small cell lung carcinoma was made. Initial diagnostic workup for rapidly progressive dementia should be broad and include testing for metabolic, infectious, and autoimmune causes through blood, urine, and CSF; neuroimaging with MRI is also recommended (10). This workup can be expanded based on results of a complete initial evaluation. Early diagnosis of paraneoplastic encephalomyelitis and tumor and early tumor treatment are necessary. However, the overall functional outcome is poor, and >50% of patients are confined to bed or a wheel-

chair in the chronic phase of the disease, highlighting the therapeutic challenge these patients face (7, 8).

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Opioids and Cannabis: Myths and Misperceptions

Rasna Patel, M.D.
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WHAT WE KNOW

Dr. Nora Volkow, Director of the National Institute on Drug Abuse, discussed the opioid epidemic, a public health issue that plagues America. The opioid epidemic was put into the limelight by the media when opioid overdose mortalities surpassed mortalities by motor vehicle accidents in the 1990s, a time in which opioid prescriptions increased drastically. This was fueled by the growing concern of inadequate assessment and treatment of the "fifth vital sign": patients' pain. The ubiquity of pain, easy access to narcotic medications, and lack of public awareness about their addictive potential led to widespread misuse. Since 1999, opioid prescriptions have quadrupled, and opioid overdose is on the rise, especially in women ages 45–55 years-old (1).

WHAT IS NEW

In March 2016, the Centers for Disease Control and Prevention released new opioid prescribing guidelines related to chronic noncancer pain (2). Many states have now mandated the use of prescription monitoring programs before prescribing opioids and other controlled substances in efforts to reduce misuse and make physicians aware of patients' prescription habits. The biggest impact has come from building awareness of the issue. At the National Prescription Drug Abuse and Heroin Summit in March 2016, President Barack Obama spoke regarding the opioid epidemic. He at once acknowledged the gravity of the problem and rallied America to come together to address the epidemic.

FUTURE DIRECTIONS

Ongoing research is advancing our understanding of addictive pathways and biochemistry, as well as the identification of biomarkers to recognize which patients are at greater risk for developing addictive disorders (3). The development of opioid formulations that are tamper resistant and the development of pro-drugs might further reduce and deter abuse of prescription opioids (4). Dr. Volkow concluded by challenging physicians everywhere to address pain with more than their prescription pads. She asked that we become advocates for change and build awareness and provide education.

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The authors thank Dr. Nora Volkow for her continued work in the field of addiction psychiatry.

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Twilight, Chickens, and a Return to Humanity: A Letter to the Interns

Linda B. Drozdowicz, M.D.

The room is dark and cool. The dog is still sleeping, now having inched himself into the warm spot I left in the bed. My husband, also unconscious, bears a facial expression suggesting mixed palsies of multiple cranial nerves. There is drool. From the bedroom I can smell the savory, salty chicken broth seeping into the pearled barley as it boils on the stove in my "charming, compact" [read: tiny] Manhattan kitchen. I walk to the kitchen and peer down into the deep, glinting aluminum of the pot to see the soup simmering below. The microwave clock shows 6:00 a.m., and the sun is just starting its morning commute.

Growing up, I could never understand why my mother would rise out of bed before the sun just to cook. I mean, come on—she would be awake, dressed, and productive before our backyard chickens even had a chance to start puttering around and clucking bloody murder. Who wakes up before the chickens?

Today, 1 ½ years into residency, I wake up before the chickens. Unlike the frantic days of intern year, when each morning would bring the promise of endless to-do lists and general bottom-of-the-totem-pole-ism, today I rise feeling rested and calm. Like my mother, I savor the morning twilight and delight in the ability to prepare something good for later. Reflecting on intern year, I delight in the ability to even think about later. Next to the stove, the coffee pot drips and clicks as it brews my drug of choice.

Your first year as a physician is challenging at best and grueling at worst.

Taking a moment to think about the day to come, I envision the busy psychiatric emergency service. I recall my heightened (to put it lightly) anxiety the first few times I was assigned to patients with alcohol withdrawal, thinking only of the helpful medical school axiom that "alcohol withdrawal will kill your patients!" I am pleased that, with time and training, I no longer suffer a temporary arrhythmia when a patient comes in for alcohol detox; instead, I calmly formulate a taper schedule and engage the patient in motivational interviewing. My heart thanks me for the change. The coffee is ready.

I am grateful to care for people in their times of crisis and relieved to feel like I finally have some competence in doing so. While my doctoring skills are far from perfect, I no longer bear the intern's cross of complete unfamiliarity and fear. I pour the coffee into my favorite tacky mug and continue to swirl my stovetop concoction. The barley becomes soft, gradually absorbing the flavors of the broth that surrounds it. And I—having absorbed over a year of knowledge and experience in my chosen field—I slowly exhale and smile.

To the interns: Your first year as a physician is challenging at best and grueling at worst. Have faith and keep your chins up. You too will return to humanity in time.

Dr. Drozdowicz is a third-year resident in the Department of Psychiatry, Icahn School of Medicine at Mount Sinai, New York; she is also an APA Leadership Fellow.

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).*

NOVEMBER DEADLINE

Fellowship/Award and Deadline	Organization	Brief Description	Eligibility	Contact	Website
Research Colloquium for Junior Investigators **Deadline: November 30, 2016**	American Psychiatric Association (APA)	The colloquium provides guidance, mentorship, and encouragement to young investigators in the early phases of their training. Held during the APA Annual Meeting.	• Psychiatrists who are senior residents, fellows or junior faculty; • MD degree or be a member of the APA or eligible to become a member of the APA; • Must be receiving their training in the U.S. or Canada.	Keila Barber colloquium@psych.org (e-mail) or 703-907-8690 (phone)	https://www.psychiatry.org/psychiatrists/practice/research/research-colloquium

DECEMBER DEADLINE

Fellowship/Award and Deadline	Organization	Brief Description	Eligibility	Contact	Website
Association for the Advancement of Philosophy and Psychiatry (AAPP) Karl Jaspers Award **Deadline: December 15, 2016**	AAPP	This award is given for the best solely authored, unpublished paper related to the subject of philosophy and psychiatry. Appropriate topics for the essay include, among others, the mind-body problem, psychiatric methodology, nosology and diagnostic issues, epistemology, biopsychosocial integration, the philosophy of science, philosophical aspects of the history of psychiatry, psychodynamic, hermeneutic and phenomenological approaches, and psychiatric ethics.	Resident or fellow in psychiatry, graduate students and post-doctoral students in philosophy, psychology, or related fields.	Christian Perring cperring@yahoo.com (e-mail)	https://philosophyandpsychiatry.org/jaspers-award/

JANUARY DEADLINE

Fellowship/Award and Deadline	Organization	Brief Description	Eligibility	Contact	Website
American Psychiatric Leadership Fellowship Program **Deadline: January 30, 2017**	APA	The aim of the American Psychiatric Leadership Fellowship is to develop leaders in the field of organized psychiatry by providing opportunities for residents to engage, interact, and participate at a national level and further develop their professional leadership skills, networks, and psychiatric experience. The program creates prospects for fellows to expand relationships with peers and national thought-leaders in the field of psychiatry. This fellowship has graduated many participants who have gone on to assume leadership roles that have helped guide the field of psychiatry.	• APA member; • Enrolled as PGY-2 in an accredited U.S. or Canadian psychiatry residency program; • Passed appropriate board examinations (i.e., USMLE 1-3; COMLEX 1-3; Canadian QE; LMCC 1-2); • Need not be a U.S. citizen or permanent resident, or a graduate of a U.S. medical school	Sejal Patel Telephone: (703) 907-8579 or e-mail: psychleadership@psych.org	http://www.americanpsychiatricfoundation.org/get-involved/fellowships/american-psychiatric-leadership-fellowship

Author Information for *The Residents' Journal* Submissions

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

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 on 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

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

Mental Health of Healthcare Providers

If you have a submission related to this theme, contact the Section Editor Charles Johnson, M.D. (charles.a.johnson@ucdenver.edu)

Childhood Medical Conditions and Psychopathology

If you have a submission related to this theme, contact the Section Editor David Saunders, M.D. (david.saunders@yale.edu)

LGBT Mental Health

If you have a submission related to this theme, contact the Section Editor Mark Messih, M.D., M.Sc. (Mark.Messih@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 Katherine Pier, M.D., Editor-in-Chief (katherine.pier@mssm.edu).