For Whom the School Bell Tolls
Helping Youth During the COVID-19 Crisis

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2020 has proven to be a year of challenges and uncertainties thanks to coronavirus disease 2019 (COVID-19). We started the year relatively unaware of the virus, and now it impacts every aspect of our lives. Although the initial concern was aimed at the oldest and most frail segments of the population, we must not forget our younger patients and the unique psychological challenges that children face during this pandemic.

The reopening debate: in-person versus online learning
One of the most contested issues in the country revolves around the reopening of schools. On the one hand, there are concerns over the social and learning needs of children; on the other are the health risks facing students, teachers, other school personnel, and their respective families. In July, the American Academy of Pediatrics

COMMENTARY
Surviving Coronavirus: A Psychiatrist’s Personal Journey

Surviving Coronavirus: A Psychiatrist’s Personal Journey

Amilcar Arnaldo Tirado, MD, MBA

really hope I don’t get coronavirus. These thoughts would later haunt me. On Friday, March 13, 2020, while working at a community hospital in the South Bronx, I began to feel vague, nonspecific symptoms of general malaise, myalgia, and fatigue. When I returned home that evening, I was scared and in denial.

Before the United States was overwhelmed with coronavirus disease-2019 (COVID-19) cases, many of us watched reports on television revealing the plight that people faced in other countries, especially Italy and Spain. It was agonizing to see the frightened facial expressions of health care workers fully clad in personal protective equipment (PPE), working around the clock and under extraordinarily difficult conditions. During the first few months of 2020, there were no clear rules or regulations about face masks or social distancing in the United States as we enjoyed our lives in denial, thinking it could not happen.

ISSUE HIGHLIGHTS
ANXIETY & STRESS DISORDERS
PTSD’s Impact on MDD
Cornel N. Stancliu, MD, MRCP; and Samantha A. Gnanasegaram, MD

ADDICTION
Cannabidiol and Psychosis
Antonio Waldo Zuardi, MD, PhD, and José Alexandre Crippa, MD, PhD

MOOD DISORDERS
Managing Lithium’s Adverse Effects
David N. Osser, MD

COMPLETE CONTENTS, PAGE 5

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COMPLETE CONTENTS, PAGE 5
CONTRAINDICATIONS
• Known hypersensitivity to amphetamines or other ingredients of Vyvanse. Anaphylactic reactions, Stevens-Johnson Syndrome, angioedema, and urticaria have occurred.
• Use with monoamine oxidase inhibitors (MAOIs) or within 14 days of stopping MAOIs (including MAOIs such as linezolid or intravenous methylene blue), because of an increased risk of hypertensive crisis.

WARNINGS AND PRECAUTIONS
• Prior to and during treatment assess for the presence of cardiac disease. Avoid use in patients with known structural cardiac abnormalities, cardiomyopathy, serious heart arrhythmia, coronary artery disease, and other serious heart problems. Sudden death, stroke and myocardial infarction have been reported in adults with CNS stimulants at recommended doses, as well as sudden death in children and adolescents with structural cardiac abnormalities and other serious heart problems while taking CNS stimulants at recommended doses. Further evaluate patients who develop exertional chest pain, unexplained syncope, or arrhythmias while taking Vyvanse.

• CNS stimulants cause increases in blood pressure (mean increase about 2-4 mm Hg) and heart rate (mean increase about 3-6 bpm). Monitor all patients for tachycardia and hypertension.
• Exacerbation of Pre-existing Psychosis: May exacerbate symptoms of behavior disturbance and thought disorder in patients with a pre-existing psychotic disorder.

ADVERSE REACTIONS
• Increased risk of serotonin syndrome when co-administered with serotonergic agents (e.g., SSRIs, SNRIs, MAOIs, tricyclic antidepressants). Discontinue if symptoms occur.
• CNS stimulants, including Vyvanse, are associated with increased risk of weight loss.
• CNS stimulants have been associated with weight loss. Use of other sympathomimetic drugs for weight loss has been associated with serious cardiovascular adverse events. Use with caution in patients with heart disease.

STUDY DESIGN
Multicenter, randomized, double-blind, placebo-controlled, crossover, modifed analog classroom study for risk factors for developing a manic episode (e.g., history of suicide, bipolar disorder, and depression). Prior to initiating treatment, screen for risk factors for developing a manic episode. New Psychotic or Manic Symptoms: May induce a mixed/manic episode in patients with a pre-existing psychotic disorder.

ADVERSE REACTIONS (continued)
• New Psychotic or Manic Symptoms: May exacerbate symptoms of behavior disturbance and thought disorder in patients with a pre-existing psychotic disorder. Induction of a Manic Episode in Patients with Bipolar Disorder: May induce a mixed/manic episode in patients with bipolar disorder. Prior to initiating treatment, screen for risk factors for developing a manic episode (e.g., comorbid history of depressive symptoms, or a family history of suicide, bipolar disorder, and depression). New Psychotic or Manic Symptoms: At recommended doses, may cause psychotic or manic symptoms (e.g., hallucinations, delusional thinking, or mania) in patients with no prior history of psychotic illness or mania. Discontinue if symptoms occur.
• CNS stimulants have been associated with weight loss and slowing of growth rate in pediatric patients (monitor weight and height). Treatment may need to be interrupted in children not growing or gaining weight as expected.
**STUDY 316**

**REVIEW CLINICAL STUDY**

**VYVANSE (LISDEXAMFETAMINE DIMESYLATE) DEMONSTRATED SIGNIFICANT IMPROVEMENT IN ATTENTION IN ADULTS WITH ADHD**¹

**Primary Endpoint:** Average of the PERMP* total scores from all postdose sessions measured, from 2 to 14 hours.¹ ²

**STUDY DESIGN**

Multicenter, randomized, double-blind, placebo-controlled, crossover, modified analog classroom study to simulate a workplace environment in 142 adults aged 18-55 years with ADHD (as defined by Diagnostic and Statistical Manual of Mental Disorders, 4th ed, text revision)⁶, ⁷

During a 4-week, open-label, dose optimization phase, subjects were titrated to an optimal dose of Vyvanse 30, 50, or 70 mg/day in the morning. They were then randomized in the double-blind crossover phase to receive Vyvanse (optimized dose) followed by placebo or placebo followed by Vyvanse, each for 1 week. During the double-blind phase, efficacy assessments occurred at the end of each week using PERMP.¹ ²

*PERMP is an objective, validated, skill-adjusted math test that measures attention in ADHD. The PERMP total score comprises the sum of the number of math problems attempted and the number of math problems answered correctly. Higher scores indicate less severe symptoms. PERMP is not a test of the ability to learn math.*

PERMP® Permanent Product Measure of Performance.

The three core symptoms of ADHD are inattention, hyperactivity, and impulsivity. Symptoms must occur often and appear in two or more settings. Diagnosis should be based on a complete patient history and full clinical evaluation based on the DSM-5.⁶

**ADVERSE REACTIONS (continued)**

- **Adults:** increased appetite, insomnia, dry mouth, diarrhea, nausea, anxiety, and anorexia.

**PREGNANCY AND LACTATION**

Vyvanse may cause fetal harm. Breastfeeding is not recommended during Vyvanse treatment.

Please see Brief Summary of Full Prescribing Information, including Boxed WARNING regarding Potential for Abuse and Dependence, on following pages.

References:
3. Data on file; SPD489-066; Shire US Inc.

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VYVANSE® (lisdexamfetamine dimesylate)
Capsules 10, 20, 30, 40, 50, 60, 70 mg
Chewable tablets 10, 20, 30, 40, 50, 60 mg

CONTRAINDICATIONS
VYVANSE is contraindicated in patients with:
- Known hypersensitivity to amphetamine products or other ingredients of
  VYVANSE. Anaphylactic reactions, Stevens-Johnson Syndrome, angioedema, and
  urticaria have been observed in postmarketing reports.
- Patients taking monoamine oxidase inhibitors (MAOIs), or within 14 days of
  stopping MAOIs (including MAOIs such as linezolid or intravenous methylene blue),
  because of an increased risk of hypertensive crisis.

WARNINGS AND PRECAUTIONS
Potential for Abuse and Dependence (See Above)
Serious Cardiovascular Reactions
Sudden death, stroke, and myocardial infarction have been reported in adults with CNS
stimulant treatment at recommended doses. Sudden death has been reported in children
and adolescents with structural cardiac abnormalities and other serious heart problems
taking CNS stimulants at recommended doses for ADHD. Avoid use in patients with
known structural cardiac abnormalities, cardiomyopathy, serious heart arrhythmia,
coronary artery disease, and other serious heart problems. Further evaluate patients
who develop exertional chest pain, unexplained syncope, or arrhythmias during
VYVANSE treatment.

Blood Pressure and Heart Rate Increases
CNS stimulants cause an increase in blood pressure (mean increase about 2-4 mm Hg)
and heart rate (mean increase about 3-6 bpm). Monitor all patients for potential
tachycardia and hypertension.

Psychiatric Adverse Reactions
Exacerbation of Pre-existing Psychosis
CNS stimulants may exacerbate symptoms of behavior disturbance and thought disorder
in patients with a pre-existing psychotic disorder.

Induction of a Manic Episode in Patients with Bipolar Disorder
CNS stimulants may induce a mixed/manic episode in patients with bipolar disorder.
Prior to initiating treatment, screen patients for risk factors for developing a manic
episode (e.g., comorbid or history of depressive symptoms or a family history of suicide,
 bipolar disorder, and depression).

New Psychotic or Manic Symptoms
CNS stimulants, at recommended doses, may cause psychotic or manic symptoms, e.g.
hallucinations, delusional thinking, or mania in children and adolescents without a prior
history of psychotic illness or mania. If such symptoms occur, consider discontinuing
VYVANSE. In a pooled analysis of multiple short-term, placebo-controlled studies of
CNS stimulants, psychotic or manic symptoms occurred in 0.1% of CNS stimulant-
treated patients compared to 0% in placebo-treated patients.

Suppression of Growth
CNS stimulants have been associated with weight loss and slowing of growth rate in
pediatric patients. Closely monitor growth (weight and height) in pediatric patients
with ADHD, including VYVANSE. In a 4-week, placebo-controlled trial of
VYVANSE in patients ages 6 to 12 years old with ADHD, there was a dose-related
decrease in weight in the VYVANSE groups compared to weight gain in the placebo
group. Additionally, in studies of another stimulant, there was slowing of the increase
in height.

Peripheral Vasculopathy, including Raynaud’s Phenomenon
Stimulants, including VYVANSE, used to treat ADHD are associated with peripheral
vasculopathy, including Raynaud’s phenomenon. Signs and symptoms are usually
intermittent and mild; however, very rare sequelae include digital ulceration and/or soft
skeletal breakdown. Effects of peripheral vasculopathy, including Raynaud’s phenomenon,
were observed in postmarketing reports at different times and at therapeutic doses in
all age groups throughout the course of treatment. Signs and symptoms generally
improve after reduction in dose or discontinuation of drug. Careful observation
for digital changes is necessary during treatment with ADHD stimulants. Further clinical
evaluation (e.g., rheumatologic referral) may be appropriate for certain patients.

Serotonin Syndrome
Serotonin syndrome, a potentially life-threatening reaction, may occur when
amphetamine derivatives are used in combination with other drugs that affect the serotonergic
neurotransmitter systems such as monoamine oxidase inhibitors (MAOIs), selective
serotonin reuptake inhibitors (SSRIs), serotonin norepinephrine reuptake inhibitors
(SNRIs), triptans, tricyclic antidepressants, fentanyl, lithium, tramadol, tryptophan,
bupropion, and St. John’s Wort. Amphetamines and amphetamine derivatives are known
 to be metabolized, to some degree, by cytochrome P450 2D6 (CYP2D6) and display
minor inhibition of CYP2D6 metabolism. The potential for a pharmacokinetic interaction
exists with the co-administration of CYP2D6 inhibitors which may increase the risk
with increased exposure to the active metabolite of VYVANSE (dextroamphetamine). In these
situations, consider an alternative non-serotonergic drug or an alternative drug that
does not inhibit CYP2D6. Serotonin syndrome symptoms may include mental status
changes (e.g., agitation, delusions, delirium, and coma), autonomic instability (e.g.
tachycardia, labile blood pressure, diaphoresis, diaphoresis, flushing, hyperthermia),
neuroparalytic symptoms (e.g., tremor, rigidity, myoclonus, hyperreflexia, incoordination),
seizures, and/or gastrointestinal symptoms (e.g., nausea, vomiting, diarrhea).

ADVERSE REACTIONS
Clinical Trial Experience
Based on data from 4-week parallel-group controlled clinical studies of VYVANSE in
pediatric and adult patients with ADHD.

Adverse Reactions Associated with Discontinuation of Treatment
In the controlled trial in patients ages 6 to 12 years, 8% (18/218) of VYVANSE-treated
patients discontinued due to adverse reactions compared to 1% (1/72) of placebo-treated
patients. The most frequently reported adverse reactions leading to discontinuation (1%
or more and twice the rate of placebo) were ECG voltage criteria for ventricular hypotropy,
tic, vomiting, psychomotor hyperactivity, insomnia, decreased appetite and rash
(2 instances for each adverse reaction, i.e., 2/218 (1%).

In the controlled trial in patients ages 13 to 17 years, 3% (7/233) of VYVANSE-treated
patients discontinued due to adverse reactions compared to 1% (1/77) of placebo-
treated patients. Most frequent adverse reactions leading to discontinuation were
irritability (3/233; 1%), decreased appetite (2/233; 1%), and insomnia (2/233; 1%).
In the controlled adult trial, 6% (21/358) of VYVANSE-treated patients discontinued due
to adverse reactions compared to 2% (1/62) of placebo-treated patients. The most
frequently reported adverse reactions leading to discontinuation (1% or more and twice
the rate of placebo) were insomnia (8/358; 2%), tachycardia (3/358; 1%), irritability (2/358;
1%), hypertension (4/358; 1%), headache (2/358; 1%), anxiety (2/358; 1%), and
dyspnea (3/358; 1%).

Adverse Reactions Occurring at an Incidence of ≥5% or More Among VYVANSE Treated Patients with ADHD in Clinical Trials
Most common adverse reactions (incidence ≥5% and at a rate at least twice placebo) reported
in children, adolescents, and/or adults were anorexia, anxiety, decreased appetite,
delay, decreased weight, diarrhea, dizziness, dry mouth, irritability, insomnia, nausea,
upper abdominal pain, and vomiting.

Adverse Reactions Occurring at an Incidence of 2% or More
Adverse reactions reported in the controlled trials in pediatric patients ages 6 to 12
years, adolescent patients ages 13 to 17 years, and adult patients treated with
VYVANSE or placebo:

Adverse Reactions Reported by ≥2% of Children (Ages 6 to 12 Years) with ADHD
Taking VYVANSE and at least Twice the Incidence in Patients Taking Placebo
VYVANSE (n=218), Placebo (n=72): Decreased Appetite (38%, 4%), Insomnia (22%, 3%), Abdominal Pain Upper (12%, 6%), Irritability (10%, 9%), Vomiting (9%, 4%), Weight Decreased (9%, 1%), Nausea (6%, 3%), Dry Mouth (5%, 0%), Dizziness (5%, 0%), Affect lability (3%, 0%), Rash (3%, 0%), Pyrexia (2%, 1%), Somnolence (2%, 1%), Tic (2%, 0%), Anorexia (2%, 0%),

Adverse Reactions Reported by ≥2% of Adolescent (Ages 13 to 17 Years) Patients
with ADHD Taking VYVANSE and at least Twice the Incidence in Patients Taking Placebo
VYVANSE (n=233), Placebo (n=77): Decreased Appetite (34%, 5%), Insomnia (13%, 4%), Weight Decreased (9%, 0%), Dry Mouth (4%, 1%), Malnutrition (2%, 1%), Anorexia (2%, 0%), Tremor (2%, 0%)

Adverse Reactions Reported by ≥2% of Adult Patients with ADHD Taking VYVANSE
and at least Twice the Incidence in Patients Taking Placebo - VYVANSE (n=358), Placebo (n=62): Decreased Appetite (27%, 2%), Insomnia (27%, 8%), Dry Mouth (26%, 3%), Diarrhea (7%, 0%), Nausea (7%, 0%), Anxiety (6%, 0%), Anorexia (5%, 0%), Feeling Jittery (4%, 0%), Agitation (3%, 0%), Increased Blood Pressure (3%, 0%), Hyperhidrosis (3%, 0%), Restlessness (3%, 0%), Decreased Weight (3%, 0%), Dyspnexa (2%, 0%), Increased Heart Rate (2%, 0%), Tremor (2%, 0%), Palpitations (2%, 0%).
In addition, in the adult population erectile dysfunction was observed in 2.6% of males on VYVANSE and 0% on placebo; decreased libido was observed in 1.4% of subjects on VYVANSE and 0% on placebo.

Postmarketing Experience
The following adverse reactions have been identified during post approval use of VYVANSE. Because these reactions are reported voluntarily from a population of uncertain size, it is not possible to reliably estimate their frequency or establish a causal relationship to drug exposure. These events are as follows: cardiomyopathy, mydriasis, diplopia, difficulties with visual accommodation, blurred vision, eosinophilic hepatitis, anaphylactic reaction, hypersensitivity, dyskinesia, dysgeusia, tics, bruxism, depression, dermatomyositis, alopecia, aggression, Stevens-Johnson Syndrome, chest pain, angioedema, urticaria, seizures, libido changes, frequent or prolonged erections, constipation, and rhabdomyolysis.

DRUG INTERACTIONS

Clinically Important Interactions with Amphetamines

MAO Inhibitors (MAOI)
Clinical Impact: MAOI antidepressants slow amphetamine metabolism, increasing amphetamine effect on the release of norepinephrine and other monoamines from adrenergic nerve endings causing headaches and other signs of hypertensive crisis. Toxic neurological effects and malignant hyperpyrexia can occur, sometimes with fatal results.

Intervention: Do not administer VYVANSE during or within 14 days following the administration of MAOI [see Contraindications].

Examples: selegiline, isocarboxazid, phenelzine, tranylcypromine

Serotonergic Drugs
Clinical Impact: The concomitant use of VYVANSE and serotonergic drugs increases the risk of serotonin syndrome.

Intervention: Initiate with lower doses and monitor patients for signs and symptoms of serotonin syndrome, particularly during VYVANSE initiation or dosage increase. If serotonin syndrome occurs, discontinue VYVANSE and the concomitant serotonergic drug(s) [see Warnings and Precautions].

Examples: selective serotonin reuptake inhibitors (SSRI), serotonin-norepinephrine reuptake inhibitors (SNRI), triptans, tricyclic antidepressants, fentanyl, lithium, tramadol, tryptophan, buspirone, St. John's Wort

CYP2D6 Inhibitors
Clinical Impact: The concomitant use of VYVANSE and CYP2D6 inhibitors may increase the exposure of dextroamphetamine, the active metabolite of VYVANSE compared to the use of the drug alone and increase the risk of serotonin syndrome.

Intervention: Initiate with lower doses and monitor patients for signs and symptoms of serotonin syndrome particularly during VYVANSE initiation and after a dosage increase. If serotonin syndrome occurs, discontinue VYVANSE and the CYP2D6 inhibitor [see Warnings and Precautions and Overdosage].

Examples: paroxetine and fluoxetine (also serotonergic drugs), quinidine, ritonavir.

Alkalizing Agents
Clinical Impact: Urinary alkalizing agents can increase blood levels and potentiate the action of amphetamine.

Intervention: Co-administration of VYVANSE and urinary alkalizing agents should be avoided.

Examples: Urinary alkalizing agents (e.g., acetazolamide, some thiazides).

Acidifying Agents
Clinical Impact: Urinary acidifying agents can lower blood levels and efficacy of amphetamines.

Intervention: Increase dose based on clinical response.

Examples: Urinary acidifying agents (e.g., ammonium chloride, sodium acid phosphate, methemamine salts).

Tricyclic Antidepressants
Clinical Impact: May enhance the activity of tricyclic or sympathomimetic agents causing striking and sustained increases in the concentration of d-amphetamine in the brain; cardiovascular effects can be potentiated.

Intervention: Monitor frequently and adjust or use alternative therapy based on clinical response.

Examples: desipramine, protriptyline

Drugs Having No Clinically Important Interactions with VYVANSE
From a pharmacokinetic perspective, no dose adjustment of VYVANSE is necessary when VYVANSE is co-administered with guanfacine, venlafaxine, or omeprazole. In addition, no dose adjustment of guanfacine or venlafaxine is needed when VYVANSE is co-administered.

From a pharmacokinetic perspective, no dose adjustment for drugs that are substrates of CYP1A2 (e.g. theophylline, duloxetine, melatonin), CYP2D6 (e.g. atomoxetine, desipramine, venlafaxine), CYP2C19 (e.g. omeprazole, lamotrigine, clozapine), and CYP3A4 (e.g. midazolam, pimozide, simvastatin) is necessary when VYVANSE is co-administered.

USE IN SPECIFIC POPULATIONS

Pregnancy
The limited available data from published literature and postmarketing reports on use of VYVANSE in pregnant women are not sufficient to inform a drug-associated risk for major birth defects and miscarriage. Adverse pregnancy outcomes, including premature delivery and low birth weight, have been seen in infants born to mothers dependent on amphetamines. Monitor infants born to mothers taking amphetamines for symptoms of withdrawal such as feeding difficulties, irritability, agitation, and excessive drowsiness.

Lactation
Lisdexamfetamine is a pro-drug of dextroamphetamine. Based on limited case reports in published literature, amphetamine (d-or l, d + l) is present in human milk, at relative infant doses of 2% to 13.8% of the maternal weight-adjusted dosage and a milk/plasma ratio ranging between 1.9 and 7.5. There are no reports of adverse effects on the breastfed infant. Long-term neurodevelopmental effects on infants from amphetamine exposure are unknown. It is possible that large dosages of dextroamphetamine might interfere with milk production, especially in women whose lactation is not well established. Because of the potential for serious adverse reactions in nursing infants, including serious cardiovascular reactions, blood pressure and heart rate increase, suppression of growth, and peripheral vasculopathy, advise patients that breastfeeding is not recommended during treatment with VYVANSE.

Pediatric Use
Safety and efficacy in pediatric patients below the age of 6 years have not been established.

Geriatric Use
Clinical studies of VYVANSE did not include sufficient numbers of subjects aged 65 and over to determine whether they respond differently from younger subjects.

Renal Impairment
Due to reduced clearance in patients with severe renal impairment (GFR <15 mL/min/1.73 m²), the maximum dose should not exceed 50 mg/day. The maximum recommended dose in ESRD (GFR <15 mL/min/1.73 m²) patients is 30 mg/day.

Lisdexamfetamine and d-amphetamine are not dialyzable.

Gender
No dosage adjustment of VYVANSE is necessary on the basis of gender.

DRUG ABUSE AND DEPENDENCE

VYVANSE contains lisdexamfetamine, a prodrug of amphetamine, a Schedule II controlled substance.

OVERDOSAGE
Consult with a Certified Poison Control Center (1-800-222-1222) for up-to-date guidance and advice for treatment of overdose. Individual patient response to amphetamines varies widely. Toxic symptoms may occur idiosyncratically at low doses. Manifestations of amphetamine overdose include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states, hyperpyrexia, and rhabdomyolysis. Serotonin syndrome has been reported with amphetamines, including VYVANSE. Fatigue and depression usually follow the central nervous system stimulation. Other reactions include arrhythmias, hypertension or hypotension, circulatory collapse, nausea, vomiting, diarrhea, and abdominal cramps. Fatal poisoning is usually preceded by convulsions and coma.

Lisdexamfetamine and d-amphetamine are not dialyzable.

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US Pat. No. 7,105,486, 7,223,735, 7,655,630, 7,659,253, 7,659,254, 7,662,787, 7,662,788, 7,671,030, 7,671,031, 7,674,774, 7,678,770, 7,678,771, 7,687,466, 7,687,467, 7,713,936, 7,718,819, 7,723,305
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What I Learned During My Summer Vacation

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ually, summer is about vacations, new adventures and excursions, and time off to explore, learn, and recharge. This year, our summers were a bit different—in fact, they were like no other in recent history. Traditional travel was not an option, but there was still much to learn.

New data poured in about the coronavirus disease-2019 (COVID-19). And, as regions began to re-open, we learned what happens when you do so quickly or without enough caution.

Clinicians like Elliot B. Martin Jr, MD, tackled new challenges associated with the COVID-19 virus. In this issue, he describes the neuropsychiatric sequelae seen on the consultation-liaison service in his hospital. Lessons learned from our frontline C-L psychiatrists will be helpful in the days, weeks, and months to come. Some have been hard lessons, but they can be enlightening and empowering as the next sets of patients arrive at their doorsteps.

We have also learned the importance of tempering enthusiasm with the patience of doing what’s right not just for ourselves, but also for our community. A case in point is the coming school year. The debate over opening in-person versus online must weigh the benefits of protecting children and school officials from the virus against the mental and psychiatric challenges associated with quarantining. Unfortunately, nobody has nobody has a one-size-fits-all answer.

In this issue of Psychiatric Times®, child and adolescent psychiatrist Abhijit Ramanujam, MD, sheds light on the debate, including what we know about the risks to children, what the expert organizations have to say, and the emotional impact of the pandemic and isolation. He also shares resources and guidance to work toward the best outcomes possible, regardless of how we move ahead, especially for children with preexisting psychiatric disorders. The hard-earned lessons of emergency online schooling in the spring and our new knowledge about the impact of the virus on younger patients will enable society and psychiatry to prepare and react better than before.

Although it has been a tough and unusual summer, perhaps it is best to focus on gratitude for what we have learned and what we can do with that information. Knowledge, after all, is power, and so we hope this issue of Psychiatric Times® will inspire you to continue to be successful, safe, and serve your patients well.

Mike Hennessy Sr
Chairman and Founder, MJH Life Sciences
Pandemics Are Not Partisan

John J. Miller, MD | Editor in Chief

A s of this writing, we are well into August, and the COVID-19 pandemic continues to have a stranglehold on our country and the world. Hearing a lot about how this pandemic is starting to compete with the Great Influenza (H1N1) pandemic from 1918 through 1920, I decided to take a deep dive into learning about this widely referenced pandemic that is estimated to have killed 50 million people worldwide, and more than 600,000 people in the United States. I read with curiosity and amazement The Great Influenza, which chronicles in great detail the many subplots of this global pandemic that occurred while the world was also being brutalized by World War I. Remarkably, many of the same challenges we face in combatting COVID-19 existed during the Great Influenza: educating the public about the importance of social distancing; wearing masks; cancelling large gatherings of people; avoiding indoor crowds; quarantining individuals exposed to the virus; the trial and error of potential treatments; and most importantly, tirelessly working to create an effective vaccine.

As of today (August 18, 2020), 171,343 deaths in the United States have been attributed to COVID-19. Unlike the Great Influenza, which basically ended 100 years ago, we really do not know how far we are into the COVID-19 pandemic. Fortunately, we have top notch health care providers, scientists, epidemiologists, computer models, and worldwide information integration that collectively give us a huge advantage over the resources available during the Great Influenza. Significantly, we as a world community have had 100 years to research and understand the Great Influenza, which provides a wealth of guidance as to how to best control and ultimately defeat COVID-19.

I was intrigued by John M. Barry’s conclusion in The Great Influenza:

“Those in authority must retain the public’s trust. The way to do that is to distort nothing, to put the best face on nothing, to try to manipulate no one. Lincoln said that first, and best. A leader must make whatever horror exists concrete. Only then will people be able to break it apart.”

This invitation to embrace the truth of what is serves as the foundational first brick of the edifice of knowledge and problem solving. To be fair, the “truth of what is” is actually only a hypothesis, not objective truth. Any competent scientist will agree with this characterization. In science, we formulate a hypothesis based on observations and, through continued exploration and experimentation, each hypothesis is sculpted to a more sophisticated and refined hypothesis or is discarded. The integrity and confidence in the scientific process results from the countless scientists throughout the world and throughout the millennia that continue to challenge and refine our current hypotheses.

It is likely not a coincidence that 2 of the countries that aggressively implemented the lessons from the Great Influenza, New Orleans and Boston (CONTINUED ON PAGE 9)
Important Safety Information

Boxed Warning: Elderly patients with dementia-related psychosis treated with antipsychotic drugs are at an increased risk of death. CAPLYTA is not approved for the treatment of patients with dementia-related psychosis.

Contraindications: CAPLYTA is contraindicated in patients with known hypersensitivity to lumateperone or any components of CAPLYTA. Reactions have included pruritus, rash (e.g. allergic dermatitis, papular rash, and generalized rash), and urticaria.

Warnings & Precautions: Antipsychotic drugs have been reported to cause:

- **Cerebrovascular Adverse Reactions in Elderly Patients with Dementia-Related Psychosis**, including stroke and transient ischemic attack. See Boxed Warning above.

- **Neuroleptic Malignant Syndrome**, which is a potentially fatal reaction. Signs and symptoms include: hyperpyrexia, muscle rigidity, delirium, autonomic instability, elevated creatinine phosphokinase, myoglobinuria (and/or rhabdomyolysis), and acute renal failure. Manage with immediate discontinuation of CAPLYTA and provide intensive symptomatic treatment and monitoring.

- **Tardive Dyskinesia**, a syndrome of potentially irreversible, dyskinetic, and involuntary movements which may increase as the duration of treatment and total cumulative dose increases. The syndrome can develop after a relatively brief treatment period, even at low doses. It may also occur after discontinuation of treatment. Given these considerations, CAPLYTA should be prescribed in a manner most likely to reduce the risk of tardive dyskinesia. Discontinue CAPLYTA if clinically appropriate.

- **Metabolic Changes**, including hyperglycemia, diabetes mellitus, dyslipidemia, and weight gain. Hyperglycemia, in some cases extreme and associated with ketoacidosis, hyperosmolar coma or death, has been reported in patients treated with antipsychotics. Measure weight and assess fasting plasma glucose and lipids when initiating CAPLYTA and monitor periodically during long-term treatment.

- **Leukopenia, Neutropenia, and Agranulocytosis** (including fatal cases). Perform complete blood counts in patients with pre-existing low white blood cell count (WBC) or history of leukopenia or neutropenia. Discontinue CAPLYTA if clinically significant decline in WBC occurs in absence of other causative factors.
Choose CAPLYTA to help control your patients’ symptoms with low rates of side effects¹

- In clinical trials, the most common adverse reactions were somnolence/sedation (24%) and dry mouth (6%)¹

**Orthostatic Hypotension and Syncope.** Monitor heart rate and blood pressure and warn patients with known cardiovascular or cerebrovascular disease. Orthostatic vital signs should be monitored in patients who are vulnerable to hypotension.

- **Falls.** CAPLYTA may cause somnolence, postural hypotension, and motor and/or sensory instability, which may lead to falls and, consequently, fractures and other injuries. Assess patients for risk when using CAPLYTA.

- **Seizures.** Use CAPLYTA cautiously in patients with a history of seizures or with conditions that lower seizure threshold.

- **Potential for Cognitive and Motor Impairment.** Advise patients to use caution when operating machinery or motor vehicles until they know how CAPLYTA affects them.

- **Body Temperature Dysregulation.** Use CAPLYTA with caution in patients who may experience conditions that may increase core body temperature such as strenuous exercise, extreme heat, dehydration, or concomitant anticholinergics.

- **Dysphagia.** Use CAPLYTA with caution in patients at risk for aspiration.

**Drug Interactions:** Avoid concomitant use with CYP3A4 inducers, moderate or strong CYP3A4 inhibitors and UGT inhibitors.

**Special Populations:** Neonates exposed to antipsychotic drugs during the third trimester of pregnancy are at risk for extrapyramidal and/or withdrawal symptoms following delivery. Breastfeeding is not recommended. Avoid use in patients with moderate or severe hepatic impairment.

**Adverse Reactions:** The most common adverse reactions in clinical trials with CAPLYTA vs. placebo were somnolence/sedation (24% vs. 10%) and dry mouth (6% vs. 2%).

Please see the accompanying Brief Summary of Prescribing Information on the following page.


**SEE ITS EFFICACY AT CAPLYTAHCP.COM**

CAPLYTA™ (lumateperone) capsules 42 mg

CAPLYTA is a trademark of Intra-Cellular Therapies, Inc. © 2023 Intra-Cellular Therapies, Inc. All rights reserved. 08/2023 US-CAP-1088906.
Weight gain has been observed with use of antipsychotics. Monitor weight.

Open-label trial of CAPLYTA for up to 1 year in patients with stable schizophrenia. Initial U.S. Approval: 2019

In pooled data from short-term (4- to 6-week), placebo-controlled trials of adult antipsychotic medication and monitor periodically during long-term treatment.

Esophageal dysmotility and aspiration have been associated with antipsychotic drug use. Antipsychotics, including CAPLYTA, should be used with caution in patients with a recent history of myocardial infarction or unstable cardiovascular disease.

No specific antidotes for CAPLYTA are known. In managing overdose, provide supportive care.

The syndrome can develop after a relatively brief exposure to antipsychotic drugs.

Some neonates recovered within hours or days without specific treatment; others have died.

Increased Mortality in Elderly Patients with Dementia-Related Psychosis

Geoffrey S. Smith, D.M., M.D.

William J. Alhendi, M.D.

National Institute on Aging

Elderly patients with dementia-related psychosis treated with antipsychotic drugs are at increased risk of death. Analysis of 17 placebo-controlled trials (median duration of 10 weeks) revealed a treatment-related increase in mortality of approximately 2.5-fold in patients with dementia treated with antipsychotics compared to placebo controls.

In an open-label trial of CAPLYTA for up to 1 year in patients with stable schizophrenia, the mean change in body weight was approximately -2 kg (SD 6.5) at Day 175 and approximately -3.2 kg (SD 7.4) at Day 350.

Uncommon: Agranulocytosis (including fatal cases) has been reported with other agents in the lumateperone class.

Hepatic Impairment:CAPLYTA should be prescribed in a manner most likely to reduce the risk of adverse events.
South Korea, have had among the best viral containment and lowest numbers of death per capita.3,4 Ideally, science and its ever-evolving hypotheses should remain objective and nonpartisan. In the United States, this has become challenging, as we find ourselves in an extremely partisan political environment with an important presidential election rapidly approaching. As psychiatrists, physicians, and health care providers, let us remember our oath to “do no harm,” and aggressively educate our patients and our nation to modify basic behaviors consistent with the recommendations of the Centers for Disease Control, the Surgeon General, the National Institutes of Health, infectious disease experts, renowned epidemiologists, and current hypotheses about managing a pandemic that have been sculpted from the pain and suffering of past pandemics.

An important question to investigate is why are we Americans so divided and argumentative about the consistent public health recommendations from the experts who have spent their careers studying virology, medicine, and epidemiology to best protect ourselves from the possibility of a pandemic such as the one we are facing? There has always been a subset of the American population that opposes accepted recommended norms: anti-vaccines, no blood transfusions, no Western medical treatment, no need for seatbelts, intolerance for anyone’s views that differ, and no need for government, to list a few. Arguably, discussions that stem from these disagreements ultimately create a more open and sophisticated society and validate the liberty and freedom that the First Amendment fosters. But, in my opinion, the current divisions in America that readily dismiss science, medicine, and the lessons learned from other countries and history have crossed the red line into misinformation and self-righteousness that ultimately increases our illness, pain, suffering, and death. Is the rebelliousness that led to our independence as a nation sewn together in an effort to maintain that independence in the face of any adversity—regardless of the consequences? What is it about American culture that has led us down a lonelier road than the path taken by so many other countries that are more effectively managing the COVID-19 pandemic? I do not know.

I went back and read my May editorial about COVID-19, curious to see where we were, where we have been, and where we are.5 On April 8th there were more than 400,000 cases of COVID-19 and more than 14,000 deaths in the United States; today (August 18, 2020) there are more than 5,400,000 cases of COVID-19 and more than 171,000 deaths.5 I expressed my concern about the misinformation and lack of scientific expert inclusion in policy development around the country—and how symptomatic they are. We still do not know how long antibodies created by viral exposure will render immunity. I am appreciative of the global open-access articles that become available every day from researchers in all specialty fields. The pharmaceutical industries are working aggressively and tirelessly to develop a safe and effective vaccine, and manufacturing facilities around the world are ready to join forces for large scale production once an effective vaccine is created.

Why are we Americans so divided and argumentative about the consistent public health recommendations from the experts who have spent their careers studying virology, medicine, and epidemiology to best protect ourselves from the possibility of a pandemic?

For us in psychiatry, there is currently a well-documented increase in anxiety, depression, trauma-related symptoms, substance abuse, and suicide.6,7 Risk factors include infection by COVID-19, serious illness or death of a loved one infected by COVID-19, loneliness, financial distress, job loss, social isolation, and lingering medical/psychiatric symptoms after recovery from the acute COVID-19 infection. This issue of Psychiatric Times® contains an article on the neuropsychiatric complications by Elliott Martin, MD, who discusses an increase in cases of refractory delirium and persistent encephalopathy at his hospital. A current publication by Li and Wang8 looked at the prevalence of psychiatric disorders and loneliness in the United Kingdom during the COVID-19 pandemic. Of the 15,530 respondents, 29% reported a psychiatric disorder, and 36% reported feeling lonely sometimes or often. Not surprisingly, they also determined, “Having a job and living with a partner are both significant protective factors for general psychiatric disorders and loneliness.”

We have no idea how the next 12 months will transpire. Will there be a second wave of COVID-19 as the Northern Hemisphere moves into the fall and winter months, and the influenza virus joins in the fray? Has the COVID-19 virus mutated during its travels in the Southern Hemisphere creating a more virulent and/or aggressive virus? When will an FDA-approved effective vaccine become available? How can we enhance trust in science so a COVID-19 vaccine will be welcomed with open arms, so that we can quickly achieve herd immunity? Currently, one-third of people in the United States are saying they will not get vaccinated. Will we develop effective treatments for the seriously ill?

There are also the collateral issues that can add to individual and societal stress, increasing the risk of psychiatric symptoms, substance abuse, hopelessness, fear, and loneliness. These include continued social isolation and the merry-go-round of opening—closing—reopening—reclosing of cities and towns as COVID-19 quiets down and then surges in unpredictable manners. Additionally, the struggle continues with economic uncertainty; unemployment; financial distress; childcare; online versus in-classroom education; and relationship stress.

We have been here before. In fact, we have survived more challenging times. Our United States prevails and excels when we are, in fact, united—not divided. So as we find ourselves in the middle of a pandemic, and all of its associated chaos and confusion, let us once again find our common ground and shared strength and unite as a country to show this pandemic—who we are, and how we rise above our partisan differences to defeat COVID-19—and be stronger for it. After all, the world is watching.
Helping youth during COVID-19

Continued from Cover

(AAP) strongly advocated for in-person school learning, since schools provide academic, social, and emotional skills and support that are hard to replicate at home. However, the AAP also affirmed “science should drive decision-making on safely reopening schools,” and noted “a one-size-fits-all approach is not appropriate for return to school decisions.” The AAP added:

“Reopening schools in a way that maximizes safety, learning, and the well-being of children, teachers, and staff will clearly require substantial new investments in our schools and campuses. We call on Congress and the administration to provide the federal resources needed to ensure that inadequate funding does not stand in the way of safely educating and caring for children in our schools. Withholding funding from schools that do not open in person full-time would be a misguided approach, putting already financially strapped schools in an impossible position that would threaten the health of students and teachers.”

The American Academy of Child & Adolescent Psychiatry and the American Psychiatric Association similarly stated, “In these uncertain times, making educational decisions based on science and community circumstances ensures the mental health needs of our children and adolescents are being addressed, allowing them to feel engaged, safe, secure, supported, and loved.” They also acknowledged that “education, including school attendance, is an essential component of successful and healthy development for all children and adolescents” and offered guidelines for reopening. They mentioned ensuring the well-being and educational needs of vulnerable populations like “children with emotional, learning, and physical disabilities as well as those in foster care, poverty, and for whom English is a second language, to mention but a few.”

What is “safe”? Lessons from around the world

The push to open schools in-person was originally based on the theory that children were largely not affected by COVID-19 and that transmission from asymptomatic children was uncommon. However, this assumption came after the strict physical distancing measures and school closures, and the story appears to have changed. The appearance of multisystem inflammatory syndrome, which causes serious, even life-threatening illness in previously healthy children and adolescents, served as a rude wake-up call. By August 6, 2020, the Centers for Disease Control received reports of 570 cases and 10 deaths across 40 jurisdictions (Figure). The affected children were aged 1 to 14 years; their average age was 8 years. Almost all children (96%) with the syndrome tested positive for SARS CoV-2, the virus responsible for COVID-19; the others had been in the vicinity of someone infected with the virus.

New data also show that children are not invulnerable to COVID-19. In the United States, the number of cases in children rose 44% between July 9 and July 23, according to a report which also found that children accounted for 8.4% of US cases. With 20 states reporting, researchers found between 0.6% and 9.0% of all juvenile COVID-19 cases resulted in hospitalization, confirming that severe illness was possible in children, albeit rarer than in adults. Similarly, in 8 days in July, Florida saw a 34% increase in new cases and a 23% increase in hospitalizations among children; the test positivity rate among children also increased. Recent news reports also tell of thousands of students and teachers quarantined as the number of COVID-19 infections rip through schools in several states after reopening in person.

Some proponents of a wider in-person school opening have noted that children are less likely to spread the virus, but a new report in JAMA Pediatrics contradicts this notion.

The authors found that replication of SARS-CoV-2 in older children resulted in similar levels of viral nucleic acid as in adults, but found significantly greater amounts of viral nucleic acid in children younger than 5 years. Considering behavior and hygiene issues in younger children, the authors said, “Young children can potentially be important drivers of SARS-CoV-2 spread in the general population.”

Lessons can also be learned by looking abroad. Two weeks after reopening schools, Israel saw a spike in COVID-19 cases, with more than 200 cases among students, and 130 cases at a single school. Countries including Singapore and South Korea also experienced spikes in cases when schools were reopened.

Guiding parents and supporting young patients

This is new territory for everyone, and young patients and their parents are likely to experience stress and anxiety as the school year begins. There are several ways parents can prepare themselves and their children for a relatively smooth transition.

First and foremost, clinicians should counsel parents to consider their individual family needs and issues, then develop a clear plan in terms of social distancing. Families with at-risk individuals may prefer more stringent rules, while others may err on the side of socialization. Once parents have determined what works best for their family, they should make the socialization rules very clear to their children, discussing realistic and practical strategies to mitigate the chances of infection. The approach and discussion should be tailored to the age and maturity of the child.

Children need a sense of safety and stability, so navigating these waters can be tricky. Parents should explain that although schools are reopening, socialization will be a step-by-step process. Regroup as needed with children and adolescents to provide updates, share reminders, and allow for questions.

Children in upper elementary and middle school grades are prone to receiving misinformation, either via friends or social media. Parents should facilitate family conversations, listening carefully to their children’s concerns and helping them separate fact from fiction.

Finally, role play may assist children and adolescents as they negotiate this new school year. It can be difficult even for adults to request that someone maintain their distance or ensure that their mask is properly covering their mouth and nose. Share

Table 1. Behavioral Strategies for Patients With ADHD

- Encourage parents to structure each day. Children with ADHD are unable to self-regulate. Hence, structuring the day is very important.
- Encourage parents to have 45-minute bursts of learning activity. Children diagnosed with ADHD can effectively focus up to 45 minutes at a time. Set clear expectations before each learning session.
- Alternate learning activities.
- Use positive reinforcement to change behaviors. Children with ADHD generally respond very favorably to immediate and positive feedback. Remember to be completely present when interacting with the child.
- Advise caregivers to contact their school to monitor their child with ADHD. These students are at an increased level of risk for poor school outcomes. In addition, due to impulsivity and distractibility, they may have problems adhering to social distancing norms and mask protocols.

Source: CDC. https://www.cdc.gov/mis-c/cases/index.html

Figure.

Reported MIS-C cases in the United States as of August 6, 2020

Cases

Source: CDC. https://www.cdc.gov/mis-c/cases/index.html
ways to make the conversations easier. For younger children, you can even make it a game. Act out correct and incorrect behaviors, and see if they can pick out the mistakes.

**Special issues associated with psychiatric disorders**

Children and adolescents with preexisting psychiatric disorders may have additional struggles with social distancing, online learning, and the resulting changes and lack of consistency. Although in-person learning is ideal, not all schools will be able to do so. Similarly, even those schools that initially begin in-person learning may need to move to remote learning. Fortunately, there are strategies to help support students with various psychiatric symptoms and disorders.

**Children with an attention-deficit/hyperactivity disorder (ADHD) diagnosis.** Family confinement and physical distancing may exacerbate ADHD-related risk-taking behavior. As much as feasible, additional medication doses should not be used to manage stress related to confinement. Also, steer clear of using any antipsychotic medication to manage disruptive behavior. Instead, use behavioral parenting strategies that have a beneficial effect for children with ADHD and disruptive behavior (Table 1).

**Children with schizophrenia.** Treatment adherence may become an issue for these patients. Unfortunately, current evidence does not support the use of outpatient telepsychiatry to improve adherence. For children who are acutely ill, daily home visits by a treatment team member is recommended as an alternative to hospitalization. This allows the child and their family to maintain physical distancing and minimize exposure to COVID-19.13

**Children with anxiety disorders.** It is quite likely that socially anxious children will refuse to return to school; even under ideal conditions, a new academic year can be a trigger. It is important to validate their feelings and answer questions truthfully, while not offering unnecessary details. Always try to remain calm during these discussions. Whenever possible, parents should avoid excessive reassurance and instead encourage self-reliance. Parents should model how to manage difficult emotions.

It is also important to frame statements in a positive way. For example, avoid phrases like, “There’s nothing to be afraid of,” “It’s not a big deal,” or “You’ll be just fine.” Instead, try, “I would really like for you to go on a bike ride around the neighborhood. The streets are safe.” Additional tips can be found online at the Child Mind Institute’s website.

Since the world has become a scarier place with so many risks outside of the home, it is important that parents inform their child with anxiety every time they leave the house for work or essential errands. It is also

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**Table 2. Warning Signs of Stress**

<table>
<thead>
<tr>
<th>Preschoolers</th>
<th>Elementary School Children</th>
<th>Adolescents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression in behavior such as return of thumb sucking and bedwetting</td>
<td>Aggression; increased tantrums</td>
<td>Changes in sleep and/or eating habits</td>
</tr>
<tr>
<td>Clinging to parents</td>
<td>Excessive clinginess</td>
<td>Increased anger or aggression</td>
</tr>
<tr>
<td>Sleep disturbance</td>
<td>Frequent nightmares</td>
<td>Isolation</td>
</tr>
<tr>
<td>Loss of appetite</td>
<td>School avoidance</td>
<td>Delinquent behavior</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>Physical complaints</td>
<td>Substance use</td>
</tr>
</tbody>
</table>

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Advising parents that the following warning signs indicate the need for professional help.

PRESCHOOLERS
- Regression in behavior such as return of thumb sucking and bedwetting
- Clinging to parents
- Sleep disturbance
- Loss of appetite
- Withdrawal

ELEMENTARY SCHOOL CHILDREN
- Aggression; increased tantrums
- Excessive clinging
- Frequent nightmares
- School avoidance
- Physical complaints
- Withdrawal from friends

ADOLESCENTS
- Changes in sleep and/or eating habits
- Increased anger or aggression
- Isolation
- Delinquent behavior
- Substance use
Table 3. General Coping Strategies

- Establish and maintain a daily routine. This creates a sense of predictability and reduces anxiety.
- Model basic hygiene and healthy lifestyle practices. This includes frequent and thorough hand-washing, participating in regular physical activity, and eating a balanced diet.
- Help children develop gratitude. Encourage activities like writing letters to health care workers or essential employees. Gratitude promotes well-being.
- Encourage outdoor physical activity. Nature promotes curiosity, and exercise helps improve impulse control. Study results indicate that children who spend more time in natural settings have less anger and aggression.22

helpful to remind them that frontline workers are addressing the situation.

Children with mood disorders. It is important that parents are fully aware of warning signs and symptoms (Table 2). Children with depression have a tendency to isolate, and social distancing can worsen such. Parents should ensure that their child joins them for family meals. Work with the child to come up with a list of family activities and regularly keep them engaged. Have regular check-ins and monitor media content. Discuss news stories that emphasize strength, hope, and positivity.

It also is important to teach children to recognize their health risk behaviors, such as isolating, excessive sleeping, or lethargy. Encourage them to check in with family or friends. Acknowledge and normalize their distress reactions. For example, parents can share, “I can see that you are very stressed, which is very understandable. Many people are feeling that way.” Other coping strategies are listed in Table 3.

Children with eating disorders (EDs). The pandemic is causing specific challenges for children with concerns around eating or body image. Disruption in important daily routines such as grocery shopping and exercise can lead to increased anxiety and worsen unhealthy food-related behaviors. These children should remain connected with their therapists, dietitians, and clinicians to create a relapse prevention plan.

I encourage parents to maintain a structure and schedule around the child’s meals and snacks. Families can get creative and encourage a variety of physical activities. The National Eating Disorders Association has extensive offerings of virtual support groups and live meal support.

COVID’s psychological impact

There is much debate and uncertainty regarding how children and adolescents are impacted by the virus. Current research and data from previous epidemics suggest that the pandemic may be directly or indirectly associated with the onset or exacerbation of psychiatric symptoms such as anxiety, disorders, depressive disorders, insomnia, and posttraumatic stress disorder.14,15 For instance, a study conducted in the United Kingdom by YoungMinds found that 80% of youth surveyed reported that the pandemic made their mental health worse.16 About one-third (32%) of respondents said their mental health was “much worse” in March; this number increased to 41% in their summer follow-up survey.

Research also indicates that the pandemic may influence psychological issues associated with EDs. A study conducted recently in Australia found increased binging, purging, and exercise behaviors in patients with eating disorders.17 Similarly, increased restrictive and binge-eating behaviors were found in those without EDs. More than 50% of participants with EDs also demonstrated moderate to extremely severe levels of depression, anxiety, and stress.

Clinicians also need to be aware of a rapidly increasing subset of issues that include substance abuse and domestic violence and child abuse, which have become more prevalent during stay-at-home orders.18,19 Clinicians should pay special attention to signs of domestic violence or child abuse, including monitoring for parent stress and irritability or harsh and critical responses to child behavior during clinical encounters.

Children who live in foster care, as well as gender or sexual minorities, are at a particular risk for violence and adverse mental health effects; extra care should be taken to screen these patients.20

Clinicians should be familiar with the psychiatric adverse effects associated with the medications used to treat COVID-19. For example, the corticosteroid dexamethasone has benefited some critically ill patients due to its anti-inflammatory and immunosuppressant effects, but its known adverse effects include emotional liability, insomnia, and sudden mood changes. Telepsychiatry options can be useful to minimize virus exposure. Even when children are reluctant to try online therapy or if parents view it as an inferior option, clinicians should suggest they try it anyway. Many study results, encouragingly, indicate that outpatients and their psychiatrists are generally satisfied with telemedicine.11

An extensive review of 24 studies indicated that the negative effects of quarantine can be lessened when clinicians explain its purpose and how to implement it and when they emphasize the altruistic benefit of quarantine in keeping others safe.20 Depending on age and maturity level, clinicians should consider engaging children and adolescents in such discussions.

Parents also should emphasize that social distancing does not mean emotional distancing. They can offer more hugs and loving and supportive messages.21 Children should be encouraged to stay in touch with their grandparents and extended family via video. Depending on their age, children can connect with peers on social platforms and may also have supervised socially distant playdates. For anxious children, the practice of mindfulness should be considered.22 Clinicians should provide parents with additional general coping strategies (Table 3) as well as warning signs of stress (Table 2).

Concluding thoughts

COVID-19 has presented unusual circumstances and challenges, including the need to balance the physical, emotional, and mental well-being of children and their family members. While there is no perfect one-size-fits-all approach to school reopening and social distancing, clinicians should be prepared to support their younger patients and parents as we all navigate through these choppy waters.

Dr Ramanujam is a practicing child and adolescent psychiatrist in Sacramento, California, and the Regional Medical Director of Community Psychiatry.

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Surviving
Continued from Cover

A Dose of Much-Needed Medicine

James L. Knoll IV, MD

In late March 2020, the United States was in full panic mode. What I observed compelled me to write about death terror, the absurd, and whether there were any reasons for optimism or perhaps some positive growth opportunities. It was my hope that we might tap into our creativity to manage the overwhelming death fears, regression, and unhelpful acting out. I wished for our “better angels” to shine forth, leading us away from despair and irrationality and toward hope and gratitude.

Sometimes, life’s lessons appear unexpectedly. Several weeks ago, a forensic psychiatry fellow who graduated from my program—now a friend and colleague—called to tell me that he had almost died from COVID-19. He explained that he now had a completely altered view of life. Of course, I was awestruck and grateful that he reached out to share his story with me. I suggested that Psychiatric Times readers might find his story inspirational and asked if he would feel comfortable sharing it. Consistent with his reenergized sense of humanity and gratitude, he agreed and penned the essay here: what I found to be a beautiful, moving, and inspirational story. Thank you, Dr Tirado. This medicine we all desperately need at this point in time.

Dr Knoll is director of forensic psychiatry and professor of psychiatry at SUNY Upstate Medical University in Syracuse, NY. He is editor in chief emeritus of Psychiatric Times® (2010 to 2014).

REFERENCE


here. My colleagues and I continued with our usual clinical duties. This ignorant bliss was short-lived as the United States soon emerged with the highest number of cases and fatalities in the world, and New York City, where I work and live, became the pandemic epicenter. At that time, it never entered my mind that my life as I knew it would dramatically change.

As COVID-19 statistics began to worsen and news reports grew grimmer, I recall worrying about my safety. When my symptoms first surfaced, I did not even want to consider the possibility that I might have contracted COVID-19. I was frightened by the idea that I might need to be hospitalized.

My symptoms continued to worsen throughout most of my hospitalization, leaving me to ponder a painful potential reality: “This is it. You did everything you could. And this is how your story ends.”

Facing my reality

In time, my mother’s sound advice and my rapidly advancing respiratory distress forced me to set aside these fears in favor of pure survival. I had reached the point of no longer being able to manage from home. On March 17, 2020, I went to a local urgent care center to get tested, yet I still maintained an irrational hope I could be treated as an outpatient. I petted my dog, very worried that I might not see him again. I covered my face with a bandana and took a taxi to the urgent care center; although it was only a few blocks away, I was too weak to walk. I arrived at the urgent care center disoriented and light-headed. The treating physician assistant said, “Dr Tirado, you are very hypoxic and your oxygen saturation is 87. I need to give you oxygen and put an IV [intravenous line] in you. We have to send you to the emergency department (ED) and you will be tested for coronavirus. You are going to be OK.” In that instant, all my hopes and wishful thinking for outpatient treatment were utterly extinguished. I felt dissociative and not entirely conscious. I was able to utter “OK,” and I knew my life was now in the hands of others.

I was transported by ambulance to a nearby ED, where I was held overnight. I received IV antibiotics, supplemental oxygen, and IV fluids, and underwent various procedures, including a chest X-ray and labs, before being admitted to a medical floor. I was diagnosed with pneumonia. After completing a course of IV antibiotics, I was started on oral doxycycline; a few days later this was switched to azithromycin and hydroxychloroquine. Hydroxychloroquine was just beginning to be used for some patients; it wasn’t until after my hospitalization that data indicated this drug was not an effective treatment. I was also given high-flow nasal cannula oxygen therapy to help keep my oxygen saturation above 90%.

My time on the medical floor was emotional and mind-bending. I recall lying in bed staring at the ceiling and thinking, “How did I get here?” and “What happened?” I remained light-headed and fatigued, my mind swirling with the recent events. I had some sense of relief that I had made it to a medical unit. Nonetheless, I intermittently emotionally tortured myself by going through every possible moment in which I might have contracted COVID-19. I continued to have waves of disbelief that this was happening to me. My symptoms continued to worsen, leaving me to ponder a painful potential reality: “This is it. You did everything you could. And this is how your story ends.”

My status was tenuous the first few days of my hospitalization, and I experienced no relief of symptoms. I knew my condition could easily worsen to the point of needing a ventilator. I prayed that would not happen, as I knew it dramatically increased the risk of a poor outcome. Other challenges were mounting—I had no change of clothes and the hospital was low on supplies such as gowns, blankets, towels, and the like.

My mother stayed in my apartment both to care for my dog and because she felt it was important to be close by in case of an emergency. After I informed my mother about the various shortages at the hospital, she offered to drop off items for me. She understood there were restrictions on hospital visitors. I resisted...
because she would be putting her life at risk. As it became clear my hospitalization would not be short, I reiterated, but I felt a knot in my stomach as I knew she would be putting herself in harm’s way.

There is nothing in this world quite like a mom, and I know I am lucky to have a good relationship with mine. She braved the streets of Manhattan—swirling with both imagined and real contagion—and dropped off a bag full of clothing, toiletries, a towel, a blanket, and one of my dog’s toys as a reminder to keep fighting for my life. I remember grabbing the toy in my hand and thinking, “OK, you can do this; you can stay alive.” My symptoms were in full force and I still felt awful, but holding Azul’s toy, I felt a renewed spiritual burst of energy.

Throughout most of my hospital stay, I spiked fevers and experienced shortness of breath, headaches, and a persistent dry cough. It was difficult for me to speak on the phone. Consequently, most of my communication with family and friends was limited to texts. The cycles of sweating, fevers, chills, and muscle aches persisted. At one point, the body aches and joint stiffness were so painful, I required morphine and it brought me only fleeting relief. The pain precluded any restorative sleep. My poor appetite resulted in a 20-pound weight loss. All of these compromised my ability to focus and concentrate on simple things like watching television or playing a game on my phone. I developed new symptoms that seemed strange to me, such as an unquenchable thirst. I drank copious amounts of water yet could not rid myself of an overpowering salty taste. It seemed as though my mouth was full of Himalayan salt, and I had the sensation of salt crystals forming around my mouth, nostrils, and eyes. Of course, I could not see them when I looked at my face in the mirror.

Family or friends supported me virtually through phone calls, emails, and texts. I had moments of feeling sad, depressed, and lonely, but their messages of hope and encouragement helped me stay alive and cheered me up. They also helped distract me from focusing on how truly awful I felt.

Lessons in empathy

In psychiatry, we often discuss the importance of having strong social supports. I can attest to this truth. I am grateful to all the hospital staff who were, directly and indirectly, involved in my care. The nurses who took care of me were fabulous; the respiratory therapist who added a humidifier to my oxygen intake helped me breathe easier; and the custodial staff who insisted on changing the sheets helped ground and humanize me in the hospital. I am forever in their debt, as they cared for me and helped keep me alive. Every staff member who entered my room was fully clad in PPE, and I could only imagine how scared they felt when they entered my room as well as the rooms of other patients.

The most memorable conversation during my hospitalization was with a young Latino resident assigned to my case during the first half of my hospitalization. He reminded me of myself early in my training. He saw I was not eating, and with genuine compassion, asked me, “How are you doing?” It was the first moment I had allowed myself to cry. I said to him, “I am so scared. I don’t want to die.” He put his hand on my shoulder and said, “You are not going to die. People who have coronavirus have walked out of here.” Then he added, “Also, you can’t die because we don’t have enough psychiatrists—and even fewer, when it comes to Latinx! You need to live.” This act of compassion followed by humor gave me hope in a very dark moment. In one of our last interactions, this physician handed me an incentive spirometer and encouraged me to use it to strengthen my lungs, lessen my dependence on supplemental oxygen, and improve my shortness of breath and dyspnea.

In the second half of my hospitalization, a female resident of Middle Eastern descent took over my care. It was with her encouragement that I was able to come off the high-flow nasal cannula oxygen therapy. My body temperature began to normalize, and I had fewer spiking fevers. She was pleased to see my progress and informed me of my likely discharge in the next few days. She was aware that I am a psychiatrist and felt comfortable enough to share her experience of working on a COVID-19 unit. She alluded to not being able to discuss her feelings or reveal how frightened she felt, adding that speaking to me was a brief reprieve before she would have to move on to the next patient. I was able to appreciate that she put back on her mental armor so that she could continue to perform her clinical duties.

She thanked me for the moments of openness and the reprieve our conversation gave her. On March 25, 2020, more than a week after entering the hospital, I was discharged. Although I was no longer experiencing high spiking fevers, I still had dyspnea, cough, fatigue, myalgias, and poor appetite. As part of my discharge instructions, I was told to quarantine at home for an additional 7 days. I remember being brought downstairs in a wheelchair by hospital staff and feeling a combination of exhilaration, fear, and fatigue. The hospital lobby was empty, and the streets of Manhattan were eerily quiet.

As I waited for a taxi, I called my mother to let her know that I was on my way home and that she should leave. We were both aware that I could not be around her as I was potentially contagious. As my taxi pulled up, I saw my mother standing out front waiting for her taxi. I wanted to hug her and talk to her, yet I was gripped with fear about exposing her to the virus. I was so exhausted that I only managed to wave to her and say “Hello!” before heading up to my apartment. My mother would later tell me that she could see I looked tired, scared, and pale, and that my breathing was labored. I did not start to feel like myself until about 2 to 3 months after hospitalization.

Most of my symptoms have resolved, but I continue to have occasional lingering shortness of breath. Since my discharge, I have reflected on my near-death experience. It was profoundly humbling, and I recognize how lucky I am to be alive. I am left with a feeling of being blessed with a second chance at life, as well as a desire to discover why I was given it and what I can do with it. I am still working on this.

COVID-19 has been life-changing for everyone. It has and will continue to impact how we think, feel, and interact with each other, and how we live our lives personally and professionally.

As my survival as a physician, and specifically a psychiatrist, has been spiritual and emotional. The experience has motivated me to share my story, as well as some practical advice I learned (Table). My sincere hope is that my words might help bring comfort and hope to others who must confront this tragic disaster or may be dealing with a similar situation during this trying time.

Dr Tirado is a forensic psychiatrist at Lincoln Medical Center in the Bronx, NY, and assistant clinical professor of psychiatry at Weill Cornell Medicine, New York, NY. The author reports no conflicts of interest concerning the subject matter of this article.

Table. 9 Practical Tips

1. Adapt to the “new normal” of face masks and social distancing.
2. If you become symptomatic, do not delay seeking medical care.
3. Bring your wallet, keys, phone, and phone charger if you are going to the hospital.
4. Have a “go bag” ready in the event you need to seek medical care, as you may require hospital admission. Consider including changes of comfortable loose-fitting clothes, several pairs of underwear and socks, toiletries, a towel, a blanket, water bottles, a small box of snacks, and something meaningful for you like a family photo.
5. Determine who will care for your child(ren) and pet(s) should you require hospitalization. This is also applicable if you are the caretaker for an adult loved one.
6. Request a copy of your COVID-19 lab results for your records, especially for any return-to-work requirements.
7. Once you return home, make meals a simpler process. Consider food delivery services so you have nutritious and already prepared or easy-to-prepare meals. Similarly, disposable plates and utensils means one less chore.
8. After hospitalization and quarantine, gradually take walks around your neighborhood to strengthen your lungs and overcome any fear of being outside. Learn to respect the limits of your body and what it needs in order to heal.
9. During dark moments, keep an appreciation for living in the present, the value of family and friends, the beauty of life, and the importance of gratitude.

Specification

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Specification
FORENSIC PSYCHIATRY
Bad Men Do What Good Men Dream

**James L. Knoll IV, MD**

We are pleased to offer a broad sampling of fascinating subjects by experts in forensic psychiatry. This 2-part Special Report focuses on pyromania, court testimony, avoiding malpractice, and psychopathy. Psychopathy is a particularly interesting subject in that it has been heavily researched, yet it remains something of a puzzle, and most certainly a huge burden on society. In forensic psychiatry, psychopathy is generally thought of as a more aggressive, narcissistic, predatory form of antisocial personality. French physician Philippe Pinel introduced the concept of the psychopathic personality at the turn of the 19th century. He described characteristics such as impulsive violence in the absence of appreciable deficits in intellect or cognition. In 1941, the classic text by Hervey Cleckley, MD, *The Mask of Sanity*, provided more detailed clinical descriptions. Building upon the work of Cleckley, Robert D. Hare, PhD, has approached the clinical construct by developing a research tool—the Psychopathy Checklist (PCL-R). Nevertheless, there remain multiple conceptualizations of psychopathy, as well as problems with reliability. While a large body of research has been generated on the subject, the heterogeneous nature of psychopathy renders many conclusions speculative. In fact, the epidemiology of what I would like to call “antisocial spectrum” disorders is complicated by their heterogeneity. While it appears that antisocial behavior is heritable, its expression varies by age, definition, and adverse life circumstances—among other factors. Psychopathy research, conducted primarily on caught or “unsuccessful” psychopaths, continues to seek clarity via grouping characteristics into various facets or models.

For example, the triarchic model of psychopathy is a more recently developed model identifying 3 primary domains: they include boldness, meanness, and disinhibition (Table). Boldness relates to fearlessness, tolerance of danger, and high self-confidence. Meanness encompasses lack of empathy, disregard for close attachments, and exploitative tendencies. Disinhibition refers to poor impulse control, inability to delay gratification, and possibly frontal executive dysfunction.

Throughout the centuries, we have continued to puzzle over our capacity for antisocial behavior. All the while, it may be helpful to keep in mind the aphorism, “Bad men do what good men dream.” Or as the great psychoanalyst, Mick Jagger, aptly noted, “Just as every cop is a criminal, and all the sinners saints . . .”

Dr Knoll is director of forensic psychiatry and professor of psychiatry at SUNY Upstate Medical University in Syracuse, NY. He is editor in chief emeritus of *Psychiatric Times* (2010 to 2014).

**REFERENCES**


**SPECIAL REPORT**

**Also in this special report**

**16 The Right Way to Avoid Malpractice Lawsuits**

William H. Reid, MD, MPH, and Skip Simpson, JD

**26 Tips for Testifying in Court**

Ashley VanDercar, MD, JD, and Phillip J. Resnick, MD

**COMING IN PART II IN OCTOBER PSYCHIATRIC TIMES**

**Assessing Competency to Stand Trial**

Barry Wall, MD, and Ruby Lee, MD

**Psychopathy: Insights for General Practice**

Barbara Burton, MD, and Fabian M. Saleh, MD

**Understanding Adult Fire Setting, Pyromania, and Arson**

Josephine Collins, MSc, Nichola Tyler, PhD, MSc, and Magali Fleur-Barnoux, PhD, CPsychol
The Right Way to Avoid Malpractice Lawsuits

William H. Reid, MD, MPH, and Skip Simpson, JD

Many psychiatrists are sued for malpractice every year. Some deserve it; some do not. We offer some practical tips for helping patients (most important), decreasing the chance of being sued, and increasing the odds of winning if you are. These are not legal tricks, but practice errors that plaintiffs’ lawyers look for, and ways to correct them.

SIGNIFICANCE FOR PRACTICING PSYCHIATRISTS

Psychiatrists can significantly decrease their chances of being sued for malpractice by remembering several key points—not of defensive practice but of good patient care and communication.

- Suicide is by far the most common factor leading to psychiatric malpractice lawsuits.
- Recognizing and managing risk, not “predicting suicide,” is the relevant issue.
- Certain specific measures are effective in reducing that risk and keeping the clinician within the standard of care.

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INDICATION

SECUADO® (asenapine) transdermal system is indicated for the treatment of adults with schizophrenia.

IMPORTANT SAFETY INFORMATION

WARNING: INCREASED MORTALITY IN ELDERLY PATIENTS WITH DEMENTIA-RELATED PSYCHOSIS

Elderly patients with dementia-related psychosis treated with antipsychotic drugs are at an increased risk of death. SECUADO is not approved for the treatment of patients with dementia-related psychosis.

Contraindications: Severe hepatic impairment (Child-Pugh C) or a history of hypersensitivity reactions to asenapine or any components of this formulation.

Cerebrovascular Adverse Events, Including Stroke: Elderly subjects with dementia had a higher incidence of stroke (including fatal stroke) and transient ischemic attack in clinical trials with antipsychotic drugs. SECUADO is not approved for the treatment of patients with dementia-related psychosis.

Neuroleptic Malignant Syndrome (NMS): NMS, a potentially fatal symptom complex, has been reported with antipsychotics. NMS can cause hyperpyrexia, muscle rigidity, altered mental status, and evidence of autonomic instability. If NMS is suspected, immediately discontinue SECUADO and provide intensive symptomatic treatment and monitoring.

Please see additional Important Safety Information and Brief Summary of full Prescribing Information, including BOXED WARNING, on following pages.
To be successful in a malpractice action, the plaintiff must prove 4 things (Table). If any of those 4 elements is not proved, then no malpractice has occurred. The level of proof required is fairly low, compared with criminal matters: preponderance of the evidence (merely more likely than not).

The authors have been involved in the clinical, legal, and administrative aspects of psychiatric malpractice cases, particularly those related to suicide, for more than 3 decades and in more than 30 states. We see the importance, to patients and courts alike, of practicing well. Neither medicine nor the law asks for perfection, but both demand competence and mindfulness of the concepts we will illustrate. Our comments are not meant to preach about clinical practice—many readers should already know most or all of what we describe—but to alert clinicians to the things that influence lawyers and juries as they consider alleged malpractice.

Most lists of the reasons people sue for psychiatric malpractice are misleading. The great majority of such lawsuits are related to one thing: suicide. Many apparently non-suicide topics on those...
lists—such as inadequate assessment, treatment or follow-up—refer to cases in which suicide occurred. Others, such as violating confidentiality, lack of informed consent, failure to monitor lab results, adverse medication reactions, and danger to others are far less common than readers might think. Another, sex with patients, is almost always adjudicated as a nonmalpractice damage issue or a criminal and/or licensure offense, rarely as “malpractice” (and rarely covered by malpractice insurance).

Finally, some of the examples below allude to a combination of clinician and hospital responsibility.

It is important to note that the standard of care (SOC) in malpractice cases requires physicians to recognize, within reason, unsafe treatment environments and procedures to which their patients are exposed, to protect patients from them, to bring them to others’ attention, and to advocate for their elimination.1

Treat patients as you want your family members to be treated, and give time. Be sure to give the time needed in assessments, personal contact, family feedback, and follow-up. Take time explaining things, and time documenting in sentences rather than acronyms or quick words that do not reveal your thinking process and judgment. (More about medical record notes later.)

Understand the assessment, recognition, and management of suicide risk. Competently assess patients who are potentially suicidal, do so completely and regularly, not just on admission and discharge.

The point is risk assessment, not prediction. Suicide

IMPORTANT SAFETY INFORMATION, Continued

Falls: SECUADO may cause somnolence, postural hypotension and motor or sensory instability, which may lead to falls, and consequently, fractures or other injuries. For patients with diseases, conditions, or medications that could exacerbate these effects, complete fall risk assessments when initiating antipsychotic treatment and recurrently for patients on long-term antipsychotic therapy.

Leukopenia, Neutropenia, and Agranulocytosis: Leukopenia, neutropenia, and agranulocytosis (including fatal cases) have been reported with antipsychotics, including asenapine. Monitor complete blood count in patients with pre-existing low white blood cell count (WBC) or absolute neutrophil count or history of drug-induced leukopenia or neutropenia. Discontinue SECUADO at the first sign of a clinically significant decline in WBC and in severely neutropenic patients.

GT Prolongation: Sublingual asenapine was associated with increases in Qtc interval from 2 to 5 m/sec versus placebo: There were no reports of GT prolongation exceeding 500 m/sec for SECUADO and placebo. The use of SECUADO should be avoided in patients with a history of cardiac arrhythmias and in circumstances that may increase the risk of the occurrence of torsade de pointes and/or sudden death in association with the use of drugs that prolong the QT interval.

Hyperprolactinemia: SECUADO can elevate prolactin levels and the elevation can persist during chronic administration. Long-standing hyperprolactinemia when associated with hypogonadism may lead to decreased bone density in both female and male subjects.

Seizures: Use SECUADO with caution in patients with a history of seizures or with conditions that lower the seizure threshold.

Potential for Cognitive and Motor Impairment: Somnolence was reported in patients treated with SECUADO. Caution patients about operating hazardous machinery, including motor vehicles, until they are reasonably certain that SECUADO does not affect them adversely.

Body Temperature Regulation: Use SECUADO with caution in patients who will experience conditions that increase body temperature (strenuous exercise, extreme heat, dehydration and concomitant anticholinergics).

Dysphagia: SECUADO should be used cautiously in patients at risk for aspiration. Esophageal dysmotility and aspiration have been associated with antipsychotic drug use.

External Heat: Avoid direct external heat sources while wearing SECUADO.

Application Site Reactions: During wear time or immediately after removal of SECUADO, local skin irritation may occur. Instruct patients to select a different patch application site each day to minimize the occurrence of skin irritation.

Adverse Reactions: Commonly observed adverse reactions (incidence >5% and at least twice the rate of placebo) were extrapyramidal disorder, application site reaction and weight gain.

Drug Interactions: Monitor blood pressure and adjust antihypertensive drugs when taken with SECUADO. Based on clinical response, SECUADO dose reduction may be necessary when used with strong CYP3A4 inhibitors (fluvoxamine). Reduce paroxetine (CYP2D6 substrate and inhibitor) dose by half when taken with SECUADO.

Pregnancy: Studies have not been conducted with SECUADO in pregnant women. Advise patients to notify their healthcare provider of a known or suspected pregnancy. The National Pregnancy Registry for Atypical Antipsychotics monitors pregnancy outcomes in women exposed to antipsychotics, including SECUADO, during pregnancy. For information, contact 1-866-961-2388 or http://womensmentalhealth.org/clinical-and-research-programs/pregnancyregistry/.

To report suspected Adverse Reactions, contact Noven at 800-455-8070 or FDA at 800-FDA-1088 or www.fda.gov/medwatch.

Please see brief summary of full prescribing information on following pages.

**Write more, not less. Short notes are very often inadequate, and abbreviated terms say little to malpractice juries and (clinical readers). How did you assess suicide risk and arrive at your decisions? What was your thinking?**

**SECUADO® (asenapine) Transdermal System**

**Dosing Summary**

<table>
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<th>Tablet Form</th>
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<td><strong>SECUADO</strong></td>
<td>5 mg/24 hours</td>
<td>10 mg/24 hours</td>
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</table>

**Weight Gain**

Weight gain has been observed with atypical antipsychotic use, including SECUADO. Monitor weight at baseline and frequently thereafter. Data on mean changes in weight and the proportion of patients meeting weight gain criteria of 7% of body weight from the placebo-controlled schizophrenia trials presented in Table 5.

**Hypersensitivity Reactions**

Hypersensitivity reactions have been observed in patients treated with antipsychotics, including SECUADO. In several cases, these reactions occurred after the first dose. These hypersensitivity reactions included angioedema, anaphylaxis, hypotension, tachycardia, urticaria, wheezing, angioedema, urticaria, dyspnea, and laryngeal edema. Instruct patients to discontinue treatment if they experience adverse events or if the reactions are severe. If necessary, seek medical assistance.

**Elderly Patients**

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**Table 1: Changes in Baseline in Adult Patients in the 6-Week, Placebo-Controlled Trials with Inadequate Response to Treatment**

<table>
<thead>
<tr>
<th>Tablet Form</th>
<th>Baseline Change (mg/kg)</th>
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<tbody>
<tr>
<td>Placebo</td>
<td>3.9 (21)</td>
<td>4.4 (22)</td>
</tr>
<tr>
<td>10 mg/24 hr</td>
<td>5.3 (24)</td>
<td>5.0 (23)</td>
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**Table 2: Changes in Plasma Levels of Dopamine and Norepinephrine in Adult Patients with Schizophrenia**

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<th>Plasma Norepinephrine (ng/mL)</th>
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<tbody>
<tr>
<td>Placebo</td>
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<td>1.1 (24)</td>
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<tr>
<td>SECUADO</td>
<td>1.7 (24)</td>
<td>3.5 (24)</td>
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**Table 3: Changes in Baseline in Adult Patients with Schizophrenia**

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**Table 4: Changes in Baseline in Adult Patients with Schizophrenia**

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<tr>
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**Study Limitations**

The data presented in this publication are from SECUADO clinical trials. The effectiveness of SECUADO in clinical practice may differ from that observed in clinical trials. The results of SECUADO clinical trials do not necessarily reflect the results that may be obtained in clinical practice.

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**References**

Please refer to the full clinical trial for a complete list of references.
Document your judgment. Write more, not less. Short notes are very often inadequate, and abbreviated terms such as “No SI/HI” say little to malpractice juries (and clinical readers). How did you assess suicide risk and arrive at your decisions? What was your thinking?

Malpractice liability rarely comes from honest errors in judgment; it comes from lack of adequate examination and failure to use intelligent reasoning (e.g., Tschekoff Roberts’). If there isn’t clear evidence of reasonable professional judgment in the medical record, lawyers and jurors often assume there wasn’t any. Efforts to convince them by testifying months or years later sound hollow. If there is reasonable evidence supporting your actions in the record, lawyers usually do not file suit.

So-called “positive” or “protective” risk factors are worthless for assessing individual patient risk. Loving families, good jobs, status, certain age groups, and other conditions may be statistically associated with lower group risk, but patients are individuals. Suicide occurs in all social strata, ethnic groups, religions, and age groups; in close families and broken ones; and in people with jobs and without them.
“Dr. Martin,” a successful scientist at a large government laboratory, was being treated for depression and severe anxiety by his family doctor. He declined referral to a psychiatrist, fearing his security clearance and position would be affected. One evening after dinner with his wife and children, he went into his study and shot himself in the head. The suit filed against the family doctor alleged that he had not adequately assessed the patient and inappropriately allowed his status to interfere with her pursuing necessary psychiatric referral.

“Dr. Boris,” a prominent physician, was hospitalized after a large overdose that he said was “accidental.” The psychiatrist who assessed him strongly recommended inpatient psychiatric evaluation. The patient refused, saying he had “learned his lesson.” His wife was adamantly against inpatient treatment as well, referring to their close family and promising “We’ll take good care of him.” The psychiatrist asked for a second opinion. The second psychiatrist concurred with the first. An involuntary hold was accomplished and the patient was allowed to go to a distant facility for further care. At the end of the initial patient detention (5 days), the convinced inpatient psychiatrist, who was aware of the overdose but never contacted the earlier consultants, was discharged and died by suicide within 24 hours. Malpractice suits against the distant facility and psychiatrists were settled for an undisclosed amount.

Get consultation. Getting competent consultation
before denying hospital admission, decreasing level of observation, or ordering discharge when a patient is at risk of suicide not only decreases the odds of a bad decision, it also reduces the chances of being sued if a tragedy occurs.

Don’t rely solely on the patient for information about suicidal thoughts and behaviors. If there is no other information source, be very cautious. Many patients lie or hide the truth. Even those who answer truthfully often do not understand their own symptoms, and cannot predict their future behavior.

Never rely on “contracts for safety” (CFS, no harm contracts). Many hospitals and some psychiatrists still use these forms, despite the many studies that show their uselessness (and sometimes the damage they can do) for patients who are suicidal. Never rely on these forms when making decisions about admission, monitoring, or discharge.

“Mr Young” was brought to a general hospital emergency department by police officers, who had found him intoxicated and sitting precariously on an overpass railing above a busy highway. He said he was planning to jump in front of “the next 18-wheeler that comes along down there.” He was admitted for intoxication with suicide risk. Mr Young’s wife arrived as he was being transported to the psychiatric unit, but she was not allowed to accompany him. Staff added her contact information to the chart.

A patient psychiatrist saw him the next morning and reviewed the admission. Mr Young was anxious to go home, assuring the doctor that he had been drunk and had “acted stupid” the night before. “I was clearing my head, thinking about getting a divorce, not about jumping off the wall.” When the doctor asked about the police report, he said “I don’t remember that…they were trying to help, but I’m sober now and I don’t believe here."

The psychiatrist went through the same suicide risk checklist that the emergency department social worker had used, relying on the patient’s own responses. Mr Young denied prior psychiatric history, suicidal thoughts, and past attempts; the checklist score was well below “high risk.” The psychiatrist ordered discharge for that morning with a note that said, “Pt. wants disch. No Si/Hi apparent. No intent to die. Mar. probs./intox…. Own transportation. Call PRN.”

Mr Young left the hospital via a hired car service and went to a bar. Several hours later he was struck and killed by a truck on the same highway where he had been found the prior night.

His widow sued the psychiatrist and the hospital. Past records and her deposition testimony indicated recent treatment for both substance abuse and depression, and a serious suicide attempt while intoxicated a few weeks before this incident. His wife testified that she had tried to talk with the admitting physician but was told to contact the psychiatric unit in the morning. She “thought he was safe in the hospital….and that they’d help him and call me and I’d tell them what he’d been doing…but they didn’t call, and when I finally called them, they’d already discharged him and nobody knew where he was.”

The attending psychiatrist settled out of court. The hospital, which employed the emergency department physician, lost at trial.

This vignette illustrates several critical errors. The patient had showed no objective signs of decreased suicide risk before 1:1 was discontinued; he had merely spent a few days without talking about suicide. Neither the psychiatrist nor the nursing staff had documented evidence of adequate suicide risk assessments after his admission. The record contained little description of the physician’s judgment in allowing release from 1:1 and deciding that Q15 was adequate for a greatly shamed man who had tried to shoot himself just a few days before. There were physical inadequacies with the patient’s room and bedding that made it easy for him to hang himself. In addition, posttrial juror questioning revealed that several faulted the physician for “going along with” a hospital CFS policy he should have known was wrong.

When considering discharge or decrease in monitoring level, be able to document what has changed for the patient. Make sure your documentation is more than a description of the time spent on the unit without an attempt. If there are no substantial, reliable changes, be very careful.

Beware of 2 substantial and common risk factors that are not on most checklists: instability and unpredictability. Because suicide is unpredictable, it is important to be cautious. Patients who are unstable, whether from psychosis, severe depression, bipolar disorder, severe anxiety, substance abuse, borderline syndromes, recent loss, or something else, are inherently at higher risk.

16 TENETS FOR AVOIDING MALPRACTICE

- Treat patients as you want your family members to be treated, and give time.
- Understand the assessment, recognition, and management of suicide risk.
- The point is risk assessment not prediction.
- Be the doctor.
- Document your judgment, not just your findings.
- So-called “positive” or “protective” risk factors are worthless for assessing individual patient risk.
- Get consultation.
- Don’t rely solely on the patient for information about suicidal thoughts and behaviors.
- Never rely on “contracts for safety” (CFS, no harm contracts).
- When considering discharge or decrease in monitoring level, be able to document what’s changed for the patient.
- Beware of 2 substantial and common risk factors that aren’t on most checklists: instability and unpredictability.
- Fifteen-minute checks (Q15) are below the SOC for patients with even moderate suicide risk.
- Communicate with corroborating sources, including family members.
- Communicate with co-treaters.
- Know your referral resources and co-treaters.
- Never assume that a patient’s family can or will monitor the patient as well as hospital staff.
“Ms Reynolds,” a young woman with a history of intensive but mercurial relationships, volatile moods, and episodes of overdose and cutting her wrists, stopped her medications and was hospitalized after stabbing herself during an argument with her boyfriend. She had accused him of causing her tomiscarry and then cut herself across the abdomen.

Ms Reynolds seemed to respond to medication and support over 4 days of close observation. She denied suicidal thoughts and participated in groups. She talked with her boyfriend by phone, sometimes calmly but sometimes angrily that he had not visited her. When he finally came to visit, she was inappropriately affectionate.

She requested discharge on her fifth hospital day. Nursing notes said her insight, judgment, and impulse control were “normal,” based primarily on her agreeing to take medication and denying suicidal thoughts. Her psychiatrist, relying on the nursing notes and brief daily interviews, discharged her within hours of removing her from continuous observation, and without a systematic risk assessment. She was given a supply of medication and a clinic appointment in 3 weeks.

Ms Reynolds was found dead from an intentional overdose a week before her outpatient appointment. In the malpractice action, the defense argued that “You can’t protect patients from themselves; you have to discharge them sooner or later,” and said her prior overdoses and cutting were not “real” suicide attempts.

The case was settled before trial.

This vignette illustrates the need to keep patients safe until treatment response is well established, then carefully reassess their risk. In addition, transition to aftercare is important, easing the change from 24-hour observation and support to virtually none. Discharge planning should include contact with the outpatient caregiver before discharge and a first outpatient appointment within days, not weeks.

The vignette also illustrates the folly of calling self-destructive behavior “minor” or “gestures.” There is no such thing as a suicide “gesture.”

Fifteen-minute checks (Q15) are below the SOC for patients with even moderate suicide risk. It is easy to kill oneself in 15 minutes, or 5 minutes for that matter, even if the checks are carried out as ordered (sometimes they are not). Jayaram et al., describing very successful Johns Hopkins risk management procedures, are among the many who have established continuous observation as the SOC. Chassin and Loeb agree, citing Joint Commission on the Accreditation of Healthcare Organizations experience and a large professional literature.

“Mr Kane” was placed on Q15 for suicide risk. He was found dead during shift change early the next morning. His time of death was determined to be around 3:00 AM, hours before he was found, but the Q15 record sheet indicated that an aide had observed him sleeping every 15 minutes through the night shift. At deposition, the aide admitted falsifying the Q15 shift record, with the excuse that “We do that all the time. I check the patient every 15 minutes, then fill everything in at the end.” He couldn’t explain why he had not noticed the patient hanging in the room for hours.

In the subsequent lawsuit, lack of continuous observation was found to be the main cause of Mr Kane’s death. The psychiatrist was found liable for not ordering continuous observation. The hospital settled out of court.

Falsified Q15 records are not rare. Nevertheless, the primary lesson is that if Mr Kane had been on continuous observation, his death would almost certainly have been prevented.

Communicate with corroborating sources, including family members. We have long taught 1 way of gathering of important clinical and safety information does not require a release of information (so long as the informant knows that the patient is in your care). Here is how it works: The doctor tells the relative or other person, “I cannot share what (the patient) has told me, but I would very much like for you to answer a few questions and tell me what you can.” Relatives and friends have no duty of confidentiality to the patient.

Do not let false confidentiality concerns threaten a patient’s life or limb when they are at risk for suicide or other substantial harm.

Communicate with co-treaters. Regular contact with co-treaters is important to meeting the SOC. Unless you have terminated care entirely, you are still the patient’s doctor. Be sure that counselors and programs give you regular follow-up, document it, and do the same for them. There is no issue of clinical confidentiality between co-treaters.

Know your referral resources and co-treaters. You are responsible for knowing the general qualifications of clinicians and facilities to whom you refer. If you reasonably should have known that they were incompetent or otherwise inappropriate for the referral, you may be partially responsible for a negative outcome.

Never assume that a patient’s family can or will monitor the patient as well as hospital staff. Asking family to carry out hospital responsibilities is below the SOC. (Like several of the previous examples, this caveat applies to both hospital discharge and failure to admit an outpatient who is at substantial risk.)

After several days of psychiatric hospitalization following a hanging attempt, “Mr Frank” demanded release. His psychiatrist and the hospital discharge planner spoke with Mr Frank’s family, who promised to watch him “24/7.” Mr Frank was found hanging in their garage early on the second day after discharge to their care.

In the subsequent lawsuit, the family testified that they indeed had said they would monitor Mr Frank, and part of the psychiatrist’s defense was that he had essentially discharged Mr Frank to another “care environment”—his family home—as agreed. A jury found, however, that the psychiatrist was not entitled to rely on the family. They found that the family had no duty and no reasonable expectation of an ability to treat the patient as he would have been treated in a hospital, and releasing Mr Frank to the family was below the SOC and caused Mr Frank’s death.


Dr Reid is a clinical and forensic psychiatrist who has worked with attorneys in scores of malpractice actions. He is professor of psychiatry at the University of Texas Dell Medical School and clinical professor at Texas Tech University Health Sciences Center. Mr Simpson is a malpractice attorney for psychiatric and mental health organization matters, with cases throughout the United States. He is on the Board of Directors of the American Association of Suicidology.

REFERENCES

With the emergence of new therapeutic strategies, we now have more options for improving the quality of life for people diagnosed with psychiatric disorders. As the field continues to progress it is important for you and your colleagues to understand the current clinical data and best practices when caring for your patients.

The Annual Psychiatric Times® World CME Conference™ is an event specifically designed to help you apply new evidence-based clinical concepts in psychiatry to real-world clinical practice. This 3-day meeting will feature expert presentations and panel discussions addressing emerging psychiatric treatments and newest clinical trial data, and best practices for managing challenging psychiatric diagnoses including mood disorders, anxiety disorders, eating disorders, postpartum depression, substance abuse disorders, and more. This experience provides the ideal curriculum with a focus on challenging patient case scenarios, integrating relevant evidence into real-world patient management, and comparing your practices with those of experts and peers.

Overview:

The future of psychiatry is evidence based

Agenda:

Thursday, October 15, 2020

10:00 AM Registration and Lunch
11:40 AM Presession Survey
12:00 PM Interventions for Sleep Disturbances in Psychiatric Disorders Karl Doghramji, MD
1:00 PM The Interplay of Migraines and Psychiatric Disorders : Treatment Implications Heidi Mosawi, MD
1:45 PM Clinical Psycho-Oncology: Assessment and Management Guy Maytal, MD
2:45 PM Postsession Survey and Audience Q&A
3:00 PM BREAK
4:20 PM Hot Topics
4:25 PM Presession Survey
4:45 PM Cannabis in Psychiatry: Clinical, Legal, and Ethical Issues David A. Grofick, MD, PhD, DAPA, FASAM
5:05 PM The New Role of Psychedelics Shannon Clare Carlin, MA, AMFT
5:30 PM Postsession Survey and Audience Q&A
6:00 PM Special Lectures Moderator:
6:30 PM Diagnosing and Treating PTSD Mark Hamner, MD
7:00 PM Educator of the Year Lecture Sidney Zisook, MD
7:30 PM ADJOURN

Benefits of Attending:

Learn About Emerging Treatments
Interact With Psychiatric Experts
Understand Strategies for Early Diagnosis

Ask questions to our faculty via our custom interactive platform!
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Tips for Testifying in Court

ASHLEY H. VANDERCAR, MD, JD, AND PHILIP J. RESNICK, MD

In law school, students are taught how to act, dress, and talk in court. They are told where to stand, what color suit to wear, what to say, and how to say it. They practice in lifelike simulations, held in courtrooms.

Psychiatrists—often called to testify in court—receive little if any of this training. They can feel unprepared when they first step on a witness stand. This article will help psychiatrists feel more comfortable with the testifying process by providing some necessary vocabulary, an outline of the judicial process, and practical tips on courtroom behavior.

Types of Proceedings
There are many types of courtroom proceedings: criminal (e.g., murder), civil (e.g., medical malpractice), and federal (e.g., immigration or bankruptcy). Psychiatrists are often asked to testify in personal injury cases or in medical malpractice suits.

Types of Witnesses
When a psychiatrist testifies, it is either as a party or non-party. In criminal court, there are defendants and prosecutors. In civil court, there are plaintiffs, who file suit, and defendants, who are being sued. If a psychiatrist is sued by their patient, the patient becomes the plaintiff; the psychiatrist becomes the defendant. If that psychiatrist testifies at trial, they testify as a party.

More commonly, psychiatrists testify as a non-party, either as a fact witness or as an expert witness. A fact witness testifies about what they personally experienced. An expert witness testifies about their opinion. For example, after a car accident, a pedestrian who saw the collision would be a fact witness. A psychiatrist hired by the plaintiff to assess psychological damages would be an expert witness.

Another example would be a bank teller injured in a bank heist who sues their employer for emotional distress. If the bank teller (as the plaintiff) subpoenaed their treating psychiatrist to testify about the bank teller’s pre-existing psychiatric condition, that psychiatrist would be a fact witness. If the bank teller’s attorney hires a psychiatrist to conduct an independent medical examination (after the robbery), that psychiatrist would be an expert witness.

Types of witnesses are usually unpaid. Expert witnesses are paid by the retaining attorney or the court. Expert witnesses can serve in a consulting role (reviewing a case and giving advice about trial strategy or chances of success) or a testifying role (reviewing a case and forming an opinion, and then testifying about a specific issue)—or both. In the bank teller example, a testifying expert witness could be asked to form an opinion on whether the bank teller had posttraumatic stress disorder and, if so, whether it was due to the bank robbery. In many states, physicians are required to offer their opinions with reasonable medical certainty. This is a legal standard that most often means more likely than not. For a summary of some of the roles, and responsibilities, of psychiatrists in court, see the Table.

Courtroom Procedure
As a witness, you should take advantage of your attorney’s expertise. Attorneys are trained in courtroom procedure. If you are a defendant, your attorney is on your side and should be your advocate. If you are a non-party witness, you still avail yourself of the attorney who hired or subpoenaed you to ask logistical questions.

When you are asked to appear at a court proceeding, make sure you know where you need to be and at what time. Judges do not tolerate lateness. Plan to arrive at the courthouse at least 30 minutes early. Bring a government issued identification card. Know which courtroom in which you will be testifying and where you should wait (inside or outside of the courtroom). As a witness, you will not know exactly when you will be called to testify; you might be given a window of several hours. Bring relevant materials to review or a book to read.

When your name is called, go straight to the witness stand. At some point, either on the way there or once you sit down, you will be sworn in. The courtroom is the judge’s domain. Unlike a hospital, you are a guest. Act accordingly.

During a trial, questions are asked in a very stylized format. Your attorney will begin with direct examination. You should have discussed your testimony with your attorney before the trial and educated them on pertinent psychiatric concepts. Their ability to ask the right questions will depend on how well you have helped them understand your analysis and psychiatric opinions.

During direct examination, your attorney will guide you through a series of open-ended questions. Your fundamental responsibility is to present your opinions cogently and convincingly. On cross-examination, the opposing attorney will ask specific questions based on what was covered during direct examination.

The American court system is adversarial. One of the main purposes of cross-examination is to discredit you and/or your opinion. The key

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TABLE: Examples of a Psychiatrist’s Role Testifying in Court Proceeding

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<th>Type of Case</th>
<th>Role</th>
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<td>Civil commitment</td>
<td>As a fact witness (the treating psychiatrist); explaining your observations of the patient.</td>
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<td></td>
<td>As an expert witness: giving your opinion on whether the criteria for civil commitment are met.</td>
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<tr>
<td>Medical malpractice</td>
<td>As a defendant: explaining how your actions were clinically appropriate.</td>
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<td>As an expert witness: giving your opinion on whether the defendant physician met (or did not meet) the standard of care.</td>
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<tr>
<td>Psychological damages</td>
<td>As a fact witness: describing your past treatment of the plaintiff (who, is, or was, your patient) and their symptoms before the accident.</td>
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<td></td>
<td>As an expert witness: giving your opinion on a plaintiff's psychiatric impairment, and whether it was caused by the accident.</td>
</tr>
<tr>
<td>Criminal</td>
<td>As a fact witness: describing your past treatment of the defendant (who was previously your patient) and their symptoms before the crime.</td>
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<td></td>
<td>As an expert witness: giving your opinion on competence to stand trial or sanity at the time of the offense.</td>
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role of an opposing attorney is to knock you off balance. After cross-examination, attorneys have a chance for redirect, to help rehabilitate you (in the event any damage was done) and to give you an opportunity to clarify your answers. Then there is re-cross—the opposing attorney’s second chance to discredit you.

Credibility

Your value as a witness is not just in what you say, but how you say it and how you come across (how credible you are). No matter how smart you are, if the judge or jury do not believe you, you will be an ineffective witness.

You always need to tell the truth, even when it goes against the side for which you are testifying. You need to be professional and likeable. Particularly with jury trials, it helps to act like the jury expects you (as a doctor) to act and look like they expect you to look.

Juries decide whether to believe a witness, including an expert witness, based on what they deem credible. Credibility is judged on a variety of factors. This includes your professional credentials (where you went to school, how long you have practiced, what you have published, and so on), whether you appear biased, and your conduct.

Just like we evaluate our patients’ demeanor and appearance, judges and juries evaluate a witness’s demeanor. An important aspect of demeanor is attitude. It is okay to be nervous. It is not okay to be grandiose, entitled, or overly professorial. The worst sin is to appear arrogant. Do your best to be likeable.

Likeability is hard to teach. Even people who are likeable in real life can become hostile on cross-examination. It is helpful to view cross-examination as a tennis match, a series of volleys between 2 sides. Wait for the entire question to be asked, then pause and think. Only answer the question that is asked. Do not feel as if you need to keep talking. Embrace the silence, or pregnant pause, which opposing attorneys may use strategically to get a witness to keep talking.7

Do not let the opposing attorney provoke you,8 which they often try to do. For instance, if you are a defendant in a medical malpractice claim, the plaintiff’s attorney might lead you through a series of yes or no questions (eg, whether you ordered a test, series). For instance, if you are a defendant in a medical malpractice claim, the plaintiff’s attorney might lead you through a series of yes or no questions (eg, whether you ordered a test or series)6—knowing that you will answer “no” on each question. This can feel uncomfortable. Maintain your poise and calm demeanor. Take the high road. Be your professional self and avoid getting into arguments with the cross-examining attorney.

Your professional identity should come across in your appearance. Dress conservatively. Allow opposing counsel to finish their question before you answer. Prepare thoroughly. Know where you will be testifying and get there early. Tell the truth. Be respectful toward both the direct and cross examiner.

- DON’T -

Wear distracting, bright clothing. Wear heavy makeup or loud jewelry. Be late or unprepared. Be cavalier or too casual. Be argumentative. Use jargon.

The bottom line

It takes years of practice to become a stellar witness. But, even their first time, a psychiatrist can do a good job testifying.10 Just prepare adequately, remain calm, tell the truth (in easy-to-understand language), and remember that you are there as a doctor. The courtroom is designed to be adversarial. To be an effective witness, credibility is key. Witness credibility hinges not just on what the witness says, but how they say it and their overall demeanor.

Dr VanDercar is a forensic psychiatry fellow, University Hospitals Cleveland Medical Center. Dr Resnick is professor of psychiatry, Case Western Reserve University School of Medicine. He serves on Psychiatric Times’ editorial board.

REFERENCES

Exploring the Relationship Between Cannabidiol and Psychosis

Antonio Waldo Zuardi, MD, PhD, and José Alexandre Crippa, MD, PhD

The association between Cannabis sativa (cannabis) and psychosis goes back at least as far as the Pen-ts’ao Ching, the world’s oldest pharmacopoeia, which is attributed to the Chinese emperor Shen-Nung (circa 2700 BC). It says, “ma-fen [the fruit of cannabis] if taken in excess will produce visions of devils... over the long term, it makes one communicate with spirits.” In the 19th century by pioneering French psychiatrist Jacques-Joseph Moreau, and it continues to be used to this day.1

Cannabis’ acute psychotomimetic effect is transitory; however, consistent evidence indicates that the chronic and intense use of the plant, especially if started in adolescence, contributes to the occurrence of schizophrenia.2

We know that cannabis contains about 100 cannabinoïd compounds and that the psychotomimetic effect of the plant are induced by tetrahydrocannabinol (THC). However, we have also known since the early 1970s that the effects of cannabis cannot be attributed solely to THC.3 Other cannabinoids, including cannabidiol (CBD), have intrinsic actions as well.4

Antipsychotic properties

In 1982, a study of the interactions between THC and CBD in healthy volunteers provided the first evidence that CBD might have antipsychotic properties. Our group administered oral CBD concomitantly with a high dose of THC to investigate whether CBD could attenuate THC-induced anxiety. Surprisingly, in addition to alleviating anxiety, CBD reduced the psychotic symptoms commonly induced by THC.5

More recently, this observation was confirmed in a study with THC administrated intravenously after oral pretreatment with CBD or placebo.6 In addition to blocking the psychotic symptoms induced by THC, CBD and THC presented opposite effects relative to placebo in terms of activation of the striatum during verbal recall, as assessed by functional MRI. The early observation that CBD reduced THC-induced psychotic symptoms led us to carry out a pioneering study to test the effects of CBD in a model commonly used to identify drugs with an antipsychotic profile in laboratory animals.7 The stereotypy induced in rats by a dopaminergic agonist was clearly reduced by CBD, without producing the catalepsy that is associated with the extrapyramidal adverse effects (AEs) of classical antipsychotic drugs.8 This finding suggests that CBD has an atypical antipsychotic pattern. A series of other tests with different animal models confirmed and expanded the antipsychotic profile of CBD.9

Effects on schizophrenia

The next step was to evaluate the effects of CBD in a patient with schizophrenia. The patient was a woman with chronic psychosis who experienced many AEs with traditional antipsychotics, which provided the ethical justification for this first clinical test. After 4 weeks of treatment, the patient had a marked reduction in her psychotic symptoms as assessed by standardized rating scales. This published case report stimulated the undertaking of randomized controlled clinical trials (RCTs).10

To date, 3 RCTs have evaluated the therapeutic effects of CBD in patients with schizophrenia. The first one, using a double-blind procedure, included 39 patients treated with either CBD (800 mg/day; n = 20) or the atypical antipsychotic amisulpride (800 mg/day; n = 19) for 4 weeks. The 2 drugs led to similar significant reductions in both positive and negative psychotic symptoms, but fewer AEs were seen in the CBD group.11

In the other 2 RCTs, CBD was administered as an adjunctive treatment for 6 weeks with placebo control; however, the results were contradictory. CBD treatment (1000 mg/day; n = 42) was associated with a significant reduction in positive symptoms from baseline to the end point compared with placebo (n = 44) in one study (Figure 1).12 Results from the other RCT, which used a very similar methodology but a lower CBD dose (600 mg/day; n = 20), found no significant symptomatic differences between treatments with CBD and placebo (n = 19).13 A possible explanation for the contradictory results may be the 400-mg difference in daily CBD doses.

Anxiolytic effects of CBD

The CBD dose-response relationship appears to have a particular feature. The anxiolytic effects of CBD, described in the early 1980s and confirmed by later animal and human studies, clearly follow this dose-response pattern.14 In 1990, CBD was tested in a range of doses in rats with the elevated plus-maze model and was found to act according to a bell-shaped dose-response curve. CBD induced an anxiolytic-like effect only at intermediate doses.15

This dose-response curve was also observed in healthy volunteers subjected to anxiety via a public speaking test and by public speaking in real-world settings.16 In both situations, treatment with CBD 300 mg was associated with significant decreases in anxiety symptoms, but this effect was not observed with lower or higher doses.

The same response pattern was observed in preclinical tests using other models of induced anxiety, cognitive impairment, and schizophrenia-like behavior.17 The findings suggest that this inverted U-shaped curve response pattern may be extended to other therapeutic effects of CBD, with different effective doses and therapeutic windows for each condition.

Accordingly, data from the 3 previously mentioned CBD RCTs suggest that the dose range to reduce psychotic symptoms (probably between 800 and 1000 mg/day), but not cognitive symptoms, should be high.

Figure 1. Change in the Positive and Negative Syndrome Scale (PANSS) positive scores from baseline to the end of the treatment (mean plus standard error) in patients with schizophrenia treated with cannabidiol (1000 mg/day; n = 42) or placebo (n = 44), as an adjunctive treatment for 6 weeks.

Asterisk (*) indicates a statistically significant difference (P = .019) between the two groups.

Data obtained and adapted from McGuire et al.18
er than that used to induce anxiolytic effects (between 200 and 400 mg/ day). However, precise dose ranges for each condition or symptom are yet to be determined in future RCTs with larger samples, different clinical populations, and multiple doses.

**Parkinson psychosis**

It seems that the antipsychotic effect of CBD is not limited to patients with schizophrenia. Antipsychotic effects of CBD (150 mg to 400 mg/day) were seen in patients with Parkinson disease who, for at least 3 months, had psychotic symptoms that could not be controlled by reducing antiparkinsonian drug regimens. We observed a reduction in psychotic symptoms (Figure 2) in addition to a significant improvement in global functioning (as assessed with the Unified Parkinson’s Disease Rating Scale and the Clinical Global Impression Scale).

This important study paved the way for a new, double-blind, placebo-controlled RCT with parallel groups of patients with Parkinson treated with 2 doses of CBD (75 mg and 300 mg/day) for 6 weeks. Patients with dementia or psychotic symptoms were excluded from the study to avoid the influence of psychotic symptoms. The results showed that CBD induced a significant improvement in nonmotor symptoms, including daily life activities and rapid eye movement sleep behavior disorder.

CBD presents multiple actions in the central nervous system that may have a crucial role in the pharmacotherapy of the motor and nonmotor effects of Parkinson disease. This cannabinoid was shown to have neuroprotective properties by enhancing the recycling of old damaged cell components via the facilitation of autophagic action. Moreover, CBD exerts antioxidant activities, stimulates neurogenesis, regulates mood and sleep, improves cognition and motor activity, and restores dendritic arbor and brain-derived neurotrophic factor levels in the hippocampus.

**Mechanisms of action**

The specific pharmacological mechanisms underlying the antipsychotic action of CBD are not fully understood, as this compound seems to interfere with neurotransmitter systems in diverse ways. For instance, CBD inhibits the reuptake and metabolism of anandamide, which may be implicated in the antipsychotic effect of CBD, as the concentrations of this endocannabinoid in the cerebrospinal fluid of patients with schizophrenia are higher than in those of controls and are inversely correlated with psychotic symptoms. It suggests a feedback mechanism in which the anandamide increase outweighs the occurrence of psychotic symptoms. Moreover, patients with prodromal psychosis present higher anandamide levels in cerebrospinal fluid (CSF) than do controls. The clinical improvement produced by CBD in the patients with schizophrenia has been linked with increased CSF anandamide levels. The effects of CBD on anandamide regulation in different brain regions associated with the pathophysiology of schizophrenia could explain its antipsychotic action.

Further mechanisms that could clarify the antipsychotic action of CBD include its ability to increase hippocampal neurogenesis and neuroprotection; its interaction with SHT1A, GPR55, and TRPV1 receptors; and its antioxidant and anti-inflammatory effects. For instance, some second-generation antipsychotics, such as ziprasidone and antiparkinsonian, also activate 5-HT1A receptors, which, at least in part (in addition to the noninnvolvement of dopaminergic antagonism), explain the absence of extrapyramidal AEs of CBD.

**Conclusions**

Research has been conducted on CBD and its potential use for neuro-psychiatric conditions such as schizophrenia, anxiety, Parkinson disease, depression, and dystonia, as well as for diseases and conditions that involve other organs and systems, such as inflammation, immune response, ischemia, diabetes, cancer, and many others. Most of this are preclinical studies in laboratory animals. The only CBD formulation approved by the FDA is Epiplex (GW Pharmaceuticals) to treat rare forms of epilepsy in children and adolescents; it is not currently available for use in schizophrenia. Although there are controlled good laboratory and manufacturing practices, and purified synthetic analogues for CBD exist, further safety and efficacy studies are needed.

CBD can be easily purchased throughout most of the United States, which raises concerns about the use of handcrafted, uncontrolled, cannabis-enriched CBD. However, there are well-known harmful long-term effects of THC on the developing brain, cognitive impairments associated with cannabis, and worsening of psychotic symptoms and antipsychotic response, particularly in younger patients. Therefore, well-designed RCTs with larger patient samples, using high-quality and reliable CBD, are needed to ascertain the effectiveness and safety of CBD as an antipsychotic medication and to ultimately receive FDA approval for use in schizophrenia.

**Dr Zuardi is a full professor, Department of Neuroscience and Behavior, Ribeirão Preto Medical School, University of São Paulo, Brazil. Dr Crippa is a full professor, Department of Neuroscience and Behavior, School of Medicine of Ribeirão Preto, University of São Paulo, Brazil, and a member of the National Institute of Science and Technology for Translational Medicine, Brazilian National Council for Scientific and Technological Development, Brasilia, Brazil. In addition, Dr Crippa is a member of the International Advisory Board of the Australian Centre for Cannabinoid Clinical and Research Excellence.**

The authors report that they are coinventors of the patent “Phurinol CBD compounds, compositions and uses therefor.” They are also recipients of Conselho Nacional de Desenvolvimento Científico e Tecnológico productivity fellowships. In addition, Dr Crippa has received travel support from BSPG Laboratories, Ltd, and has received a grant from the University Global Partnership Network to establish global priorities in cannabinoid research excellence.

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JOURNAL CLUB

Then and Now: Addressing Comorbid PTSD and MDD

Cornel N. Stanciu, MD, MRO, and Samantha A. Gnanasegaram, MD

Major depressive disorder (MDD) is a prevalent and debilitating chronic condition for which first-line standard antidepressant treatment modalities lead to remission in only one-third of sufferers. As psychiatrists, we are faced with tough decisions on how to proceed in managing non-response in the remaining two-thirds. Strategies include antidepressant switching or augmentation with various agents. To complicate matters, a highly comorbid condition for those with MDD is posttraumatic stress disorder (PTSD) with national surveys showing a 0.5 correlation. Such co-occurrence is associated with greater disease burden severity and very different challenges in terms of management compared with MDD alone.

In a secondary analysis of a 2017 study, Mohamed and colleagues tackled a timely question: Should a PTSD comorbidity affect our decision of how to manage MDD non-response to a first-line antidepressant treatment?

**Structured Question**

**QUESTION.** Should a concurrent PTSD diagnosis affect whether to augment or switch medications when MDD has not responded to a prior antidepressant trial?

**TYPE OF STUDY.** Secondary analysis of a randomized, single-blind, parallel-assignment trial (ClinicalTrials.gov identifier: NCT01421342). (Note: This clinical trial is the 2017 study that generated the data based on which the 2020 secondary analysis was conducted.)

**POPULATION.** Participants included 1522 Veterans Health Administration (VA) patients from 35 study sites who experienced suboptimal response to at least 1 course of selective serotonin reuptake inhibitor/serotonin-norepinephrine reuptake inhibitor/mirtazapine antidepressant treatment meeting minimal standards for dose and duration. Patients were primarily men (85.2%) with a mean age of 54.4. A total of 717 participants (47.1%) had a co-occurring PTSD diagnosis. Patients all had a diagnosis of non-psychotic MDD and were referred by their VA clinicians.

**EXPOSURES.** Study assignment was random (1:1:1) to 3 treatment strategies: switch to bupropion (switch-BUP); augment current antidepressant with bupropion (aug-BUP); or augment current antidepressant with aripiprazole (aug-ARI).

**METHOD.** Between December 2012 to May 2015, participants underwent 12 weeks of treatment with either switch-BUP (n = 511), aug-BUP (n = 506), or aug-ARI (n = 505). Blinded raters with the 16-item Quick Inventory of Depressive Symptomatology—Clinician Rated (QIDS-C16) determined remission (less than or equal to a score of 5) and response (reduction in score from baseline by 50% or more) by 12 weeks and relapse after remission (score of 11 or more for those who previously achieved remission). Long-term follow-up lasted up to 24 weeks. Survival analyses compared treatment effects in patients with concurrent PTSD and those without it.

**STATISTICAL ANALYSIS.** Remission and response outcomes for patients with and without comorbid PTSD were compared using Cox regression models with comorbid PTSD and treatment as independent variables with an interaction term for treatment and PTSD. Multivariable Cox regression models were developed using stepwise regression in which significant predictors of outcome that were associated with PTSD were added to the models to adjust estimates of PTSD and treatment effects for covariates. Comparisons were expressed by relative risk ratios (RR) estimated from hazard ratios from Cox models and 95% confidence intervals (CI), reporting the Wald test P values.

**Results**

From the main study, 1137 of the 1522 randomized veterans (74.7%) completed the treatment phase. Remission rates at 12 weeks (primary outcome) were only 30% for all groups, with the highest scores from the aug-ARI group (Figure). Similarily, response (secondary outcome) was greatest for the aug-ARI group (74.3%). Anxiety was more frequent in the 2 bupropion groups. Adverse effects were more frequent in the aug-ARI group and included somnolence, akathisia, and weight gain.

**Comparing the impact of a PTSD comorbidity**

A greater proportion of African American and Hispanic patients were found in the comorbid PTSD group. Overall, patients with comorbid PTSD had more severe depressive symptoms on the QIDS as well as global clinical severity indexes and lower quality of life scores.

In terms of remission, at 12 weeks there was a 43% lower risk of remission for those with comorbid PTSD (RR = 0.567; 95% CI, 0.462–0.697) with all 3 treatments, irrespective of the severity of MDD. Kaplan-Meier plots of cumulative probability of remission for each treatment group by PTSD status show remission rates are lower in the PTSD group; the switch-BUP treatment group had the lowest rate of remission.

Relative risk of response was also significant for those with PTSD (RR = 0.745; 95% CI, 0.658–0.843) and for each of the 3 treatments, reflecting a 25% lower symptom improvement among those with PTSD. For relapse after remission, the risk was not significantly greater among those with PTSD. The results across treatment groups were mixed, with significantly greater likelihood of relapse among participants with PTSD in the aug-BUP group but not among those in the switch-BUP or aug-ARI groups.

**Comparing the impact of various treatment interventions**

A comparison of the magnitude of
In this population of older, veteran males, with nonpsychotic moderate to severe MDD that was unresponsive to antidepressant treatment, those with comorbid PTSD showed greater disease burden at baseline and significantly lower remission and response with 3 next-step 12-week treatments. Augmentation with aripiprazole resulted in a small, yet statistically significant, increase in response likelihood at 12 weeks when compared with switching to bupropion irrespective of a comorbid PTSD diagnosis.

There were no significant differences in response between augmentation with aripiprazole and augmentation with bupropion in either patients with PTSD or patients without PTSD. The analysis of relapse showed no significant differences in comparative treatment effectiveness on remission between patients with and without PTSD. In the analysis of response, augmentation with aripiprazole was associated with significantly greater likelihood of achieving response (68.4%) than switching to bupropion (57.7%) in patients with PTSD (HR = 1.26; 95% CI, 1.01–1.59) and also in patients without PTSD (HR = 1.29; 95% CI, 1.05–1.57) (78.9% response with augment with aripiprazole versus 66.9% response with switching to bupropion).

There were no significant differences in response between augmentation with aripiprazole and augmentation with bupropion in either patients with PTSD or patients without PTSD. The analysis of relapse showed no significant differences between pairs of treatment groups with or without PTSD and minor interaction of treatment group and PTSD diagnosis (P = .15).

The bottom line
In this population of older, veteran males, with nonpsychotic moderate to severe MDD that was unresponsive to antidepressant treatment, those with comorbid PTSD showed greater disease burden at baseline and significantly lower remission and response with 3 next-step 12-week treatments. Augmentation with aripiprazole resulted in a small, yet statistically significant, increase in response likelihood at 12 weeks when compared with switching to bupropion irrespective of a comorbid PTSD diagnosis.

Comments
There were several limitations of the 2020 study. First, it was conducted within the VA system with a predominately older male population with likely combat-related trauma histories. It is thus unclear whether results would generalize to the broader population with other underlying PTSD forms.

Second, despite inclusion of a greater proportion of participants with non-psychotic psychiatric comorbidities, there were still a number of exclusion criteria, including active substance use, which represents a significant comorbidity found in patients with MDD.

Third, all individuals included experienced a moderate degree of depression severity; hence, these findings cannot be extrapolated in the care of patients with mild and severe MDD.

Fourth, 26% of participants did not complete the study, resulting in a substantial dropout rate. It is also possible that the group that switched to bupropion may have experienced withdrawal from previous treatment, putting them at a disadvantage for dropout or other adverse outcomes.

Fifth, only 1 antidepressant and 1 antipsychotic were evaluated. Generality to other agents is unknown, especially to factors such as behavioral interventions, tricyclic antidepressants, monoamine oxidase inhibitors, electroconvulsive therapy, transcranial magnetic stimulation, and lithium augmentation.

Lastly, although there was no financial support from industry, authors disclosed some pharmaceutical affiliations, which may potentially lead to biases.

Dr. Stanciu is assistant professor of psychiatry at Dartmouth’s Geisel School of Medicine and Director of Addiction Services at New Hampshire Hospital, Concord, NH. He is Addiction section editor for Psychiatric Times.

Dr. Gnanasegaram is instructor in psychiatry at Dartmouth’s Geisel School of Medicine and Attending Psychiatrist at New Hampshire Hospital, Concord, NH. The authors report no conflicts of interest concerning the subject matter of this article.

REFERENCES
What’s in Your Mouth?

The Oropharyngeal Microbiome in Schizophrenia and Mania

By Brian Miller, MD, PhD, MPH

There is evidence for immune system dysfunction and inflammation in major psychiatric disorders, including schizophrenia, bipolar disorder, and major depressive disorder. The microbiome—composed of microbes that inhabit mucosal surfaces—is an important determinant of the immune system and inflammation that can influence brain and behavior. Most previous studies of the human microbiome have focused on fecal samples, but the oropharynx also has a microbiome that is easily sampled. One pilot study found evidence for alterations in the oropharyngeal microbiome in patients with schizophrenia compared with controls who were not being treated for psychiatric illness.

Yolken and colleagues investigated the oropharyngeal microbiome in a cohort of patients with schizophrenia (n = 121), mania (n = 62), major depression (n = 48), and controls (n = 85). Participants with a psychiatric diagnosis were identified from inpatient or day hospital programs. Patients with schizoaffective disorder who met criteria for mania were analyzed in the mania group. Controls were screened for psychiatric disorders with the Structured Clinical Interview for DSM-IV Axis I Disorders, Non-Patient Edition. Participants were aged 18-65 years, English-speaking, and did not have a history of intravenous drug abuse, intellectual disability, HIV, serious medical disorders affecting cognition, or a primary diagnosis of alcohol or other substance use disorder.

All participants were administered the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS). Those in the psychiatric groups were assessed for symptoms using the Brief Psychiatric Rating Scale (BPRS). Participants also had a throat swab to assess the oropharyngeal microbiome. The composition and abundance of the oral microbiome was measured by the weighted UniFrac distance. Statistical differences among diagnostic groups were determined by permanova analyses, where q values represent P values that remained significant after correction for multiple comparisons. Individual taxa of the oropharyngeal microbiome were evaluated among diagnostic groups using analysis of variance. Relationships between these taxa and cognitive function were assessed using regression models. The participants’ mean age was 36 years, mean body mass index (BMI) was 30, and 50% of the total sample was male. The study groups were significantly different from each other regarding age, sex, race, smoking, and BMI, and the authors controlled for these variables in the analyses.

The oropharyngeal microbiome of individuals with schizophrenia and mania were significantly different from that of controls in both composition and abundance. Three of the 5 taxa that were significantly different among groups—Neisseria subflava, Weeksellaceae, and Prevotella—were decreased in schizophrenia and mania versus controls, while Streptococcus were increased in these groups. One taxon, Schlegelella, was only found in individuals with mania. Neisseria subflava was also positively associated with cognitive functioning (with a small-to-moderate effect size of approximately 0.3). However, the researchers did not find taxa significantly altered in individuals with major depression.

The authors noted that a number of environmental exposures might be more prevalent in patients with psychiatric disorders—including exposure to respiratory viruses, alcohol, poor dentition, and diet—that were not measured in the present study. Future studies might also consider assessment of the oropharyngeal microbiome in individuals with prodromal psychosis.

The bottom line

Patients with schizophrenia and mania had an altered oropharyngeal microbiome compared with controls in terms of both composition and abundance, after considering multiple potential confounding factors. An increased understanding of biological pathways by which the oropharyngeal microbiome potentially affects brain function might lead to novel treatment approaches.

Dr Miller is professor in the department of Psychiatry and Health Behavior at Augusta University in Augusta, Georgia. He is the Schizophrenia Section Chief for Psychiatric Times.

The author reports that he receives research support from Augusta University, the National Institute of Mental Health, the Brain and Behavior Research Foundation, and the Stanley Medical Research Institute.

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**BIPOLAR UPDATE**

**Management of Lithium’s Adverse Effects: Weight Gain**

David N. Osser, MD

Let’s begin with the fact that lithium causes less weight gain than competing mood stabilizing medications including valproate, olanzapine, and quetiapine—as demonstrated in head-to-head comparison studies with these other options. Moreover, risperidone causes more weight gain than lithium in children and adolescents. Lithium definitely causes some, and sometimes a lot of weight gain, but lithium should not be avoided on this account over these other often more popular choices, and the weight gain may be manageable. If weight gain is a primary consideration, then we have carbamazepine, ziprasidone, lurasidone, lamotrigine and cariprazine, and aripiprazole to consider.

There are at least 4 mechanisms for the weight gain from lithium. All should be anticipated and may require separate management strategies. First, there is the increased thirst associated with ingesting this salty inorganic compound. The key here is to avoid caloric beverages including sugary sodas and fruit juices. The latter have just as much sugar as the sodas. Water, sugar-free sports beverages with more solute load than water, and sugar-free sodas are preferred.

Next, there is the water retention secondary to the salt retention. Some patients put on weight very quickly, and this is the likely explanation; adipose-based weight would increase much more slowly. There may be increased urination, which eliminates some of the water but there is a net retention of fluid in some patients. A diuretic may help with this problem; treatment may be required with replacement thyroid.

Finally, the toughest contributor is carbohydrate craving. Patients will crave bread, candy bars, cookies, and other sugary baked goods. This problem is associated with many other psychiatric medications (eg, the famous “munchies” from quetiapine), but finding alternative ways to manage the hunger is always tough. Advanced warning about this is important so patients recognize it when it starts and have a management strategy ready to implement rather than having the problem identified only after the patient has gained 50 pounds.

**Lithium causes less weight gain than competing mood stabilizing medications.**

Dr Osser is associate professor of psychiatry, Harvard Medical School, and Consulting Psychiatrist, US Department of Veterans Affairs, National Telemental Health Center, Bipolar Disorders Telehealth Program, Brockton, Massachusetts. He is a member of Psychiatric Times Editorial Board.

**References**


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Richard M. Berlin, MD


Dr Berlin is Instructor in Psychiatry, University of Massachusetts Medical School, Worcester.

The haiku “COVID-19 death” was first presented as a recording for “Social Distancing, Haiku, and You,” a digital sound collage created by Alan Nakagawa for the Orange County Museum of Art (OCMA). Nakagawa asked participants from around the world to write and digitally record haikus inspired by their own personal experiences during the pandemic. Nakagawa subsequently created a sound collage that interweaves the multitude of voices (around 500) into a single composition.
Ms Zorn was a 45-year-old woman with no previous psychiatric history who presented with a 2-month period of “feeling like I am losing myself” and seeing “visually distorted faces.” Faces of people she was looking at would appear to be hideously elongated, with facial components distorted, as if it was melting. Cars would also appear to be more round than usual.

She was admitted to an inpatient psychiatry unit for unspecified psychosis, but after 2 days she was transferred to a general medical unit for additional medical evaluation. She was started on 2.5-mg quetiapine bid, which was titrated to 200 mg bid. Lumbar puncture was completed to rule out central nervous system infection or autoimmune encephalopathy, with results pending at the time of transfer to an academic medical center for higher level of multispecialty care.

On interview, she said that she felt like she was “losing” herself and reported visual distortions of “faces melting.” Ms Zorn was able to recognize faces and colors. She reported photophobia. She had headaches, which were relieved with ketorolac. Her husband reported that she had new onset of episodes of shaking her arms in a circular motion. She would also walk around the room in circular patterns while saying to herself “I am losing myself” and seeing “faces melting.” Ms Zorn’s thought process was linear and coherent. Affect was mildly anxious, perplexed, non-labile, and non-terrful. Speech was normal. Insight and judgment were marginal. Her Montreal Cognitive Assessment (MoCA) score was 10/30. Her decisional capacity was impaired, and the screening neurological examination was non-focal.

On hospital day 2, Ms Zorn reported sleeping from 4:30 am to 12:30 pm. She reported the visual phenomena of pixels and faces melting. Ms Zorn was only able to sleep for 1.5 hours after receiving quetiapine and mirtazapine. Ms Zorn was able to sleep for 1.5 hours after receiving quetiapine and mirtazapine. On exam, she reported feeling better compared with the previous day. She no longer reported any distortions of faces or objects in the room, although she did report some “blue dots” in her field of view. She had 3 hours of sleep the previous night. Her husband noted that the she had decreased episodes of her aimless pattern of waving her arms in a circular motion. Her MoCA score was 14/30. Due to the significant improvement in her hallucinations following the first dose of methylprednisolone, the scheduled quetiapine was held.

When seen on hospital day 3, Ms Zorn said that she could fall asleep, but had trouble staying asleep. She had an episode where she got up and walked around the room purposelessly. Her MoCA score was 13/30 and decisional capacity was impaired.

She felt like she was “losing” herself and reported visual distortions of “faces melting.”

Due to the atypical presentation of psychotic disorder, the full workup included a neurology consultation; brain MRI and lumbar puncture results were normal, except for autoimmune encephalitis results that were pending. Serum HIV, syphilis, vitamin B12, lead, rapid plasma regain (RPR), anti-nuclear antibody titer (anti-dsDNA), thyroid stimulating hormone (TSH) levels were normal; EEG was unremarkable. Pelvic ultrasound and thyroid scan was normal. The cerebrospinal (CSF) encephalopathy panel came back positive for anti-NMDA receptor antibody R1, confirming a diagnosis of anti-NMDA (N-methyl-D-aspartate)-receptor encephalopathy (ANMDARE). Neurology started plasmapheresis for 7 treatments, every other day. She had no hallucinations. She reported that she was getting “sleep sleep.” MoCA score was 16/30. A quetiapine 50 mg am dose was added.

Plasmapheresis started on hospital day 11. The patient denied any visual distortions. She said that she felt significantly better than the previous week; MoCA score was 16/30. When next seen on hospital day 15, Ms Zorn reported sleeping from 11:00 pm to 5:30 am. She denied visual distortions. MoCA score had improved to 21/30; however, decisional capacity continued to be impaired.

On hospital day 5, her husband reported that she had had continued visual phenomena of pixels and faces melting. Ms Zorn was only able to sleep for 1.5 hours after receiving quetiapine and mirtazapine. On exam, she reported the visual hallucinations and had a MoCA score of 12/30. Continuous EEG was reported as normal and abdomen CT was negative for ovarian teratoma. To address continued psychosis and poor sleep, quetiapine was increased to 100 mg am and 300 mg pm. Mirtazapine was increased to 15 mg pm.

Ms Zorn was seen again on hospital day 6. She reported that she had been able to sleep for 8 hours. She denied any visual “melting” of faces but stated that the “world looked pixelated at times.” MoCA score was 14/30. Subsequently, when seen on hospital day 8, she stated that she was still seeing pixels in the periphery of her visual field. MoCA score improved to 17/30. Quetiapine was consolidated to 400 mg pm.

She was next seen on hospital day 10. The cerebrospinal (CSF) encephalopathy panel came back positive for anti-NMDA receptor antibody R1, confirming a diagnosis of anti-NMDA (N-methyl-D-aspartate)-receptor encephalopathy (ANMDARE). Neurology started plasmapheresis for 7 treatments, every other day. She had no hallucinations. She reported that she was getting “sleep sleep.” MoCA score was 16/30. A quetiapine 50 mg am dose was added.

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Plasmapheresis started on hospital day 11. The patient denied any visual distortions. She said that she felt significantly better than the previous week; MoCA score was 16/30. When next seen on hospital day 15, Ms Zorn reported sleeping from 11:00 pm to 5:30 am. She denied visual distortions. MoCA score had improved to 21/30; however, decisional capacity continued to be impaired. On hospital day 18, she was doing well. She had declined her scheduled quetiapine the previous day “to give it a try.” She no longer saw distorted faces. She had no guilt disturbance. MoCA score improved further to 25/30. Given her improved mental status, the psychiatry team decided that she had recovered her decision-making capacity. Mirtazapine was decreased to 7.5 mg pm and quetiapine held.

Ms Zorn was seen for the last time on day 22 of hospital admission. After her final plasmapheresis treatment, she continued to be free of psychotic symptoms. She stated that she was “doing better than last week,” and that she was “80% back to normal.” She was sleeping well.
and was increasingly physically active during the day. She was excited to return home; MoCA score was 22/30. Decisional capacity continued to be intact. Mirtazapine 7.5 mg bedtime was continued.

As she was medically stable for discharge, she was referred for psychiatric follow-up near her home. She was advised that any future recurrence of psychosis should first be addressed as if it was a recurrence of delirium and/or ANMDARE.

**Discussion**

ANMDARE is a relatively recently described illness that may present with psychiatric symptoms, neurologic symptoms, or both (either simultaneously or sequentially). While the exact mechanism remains obscure, antibodies to the NMDA receptor (on CSF and/or serum assay) is confirmatory. Management includes empiric treatment of manifest symptoms and immunomodulation systemic therapies.

ANMDARE is important to the consultation-liaison psychiatrist for a number of reasons. First, its presentation lies in the “neuropsychiatric consultation-liaison psychiatrist for a neurologic patient.” Clinical suspicion for ANMDARE was confirmed with reference laboratory (Mayo Clinic) results of an R1 antibody to NMDA receptor on CSF analysis. This was accomplished in the context of a thorough laboratory evaluation for other esoteric causes of atypical psychotic illness.

Empiric treatment of the psychiatric symptoms with mirtazapine and quetiapine, concurrent with 2 rounds of immunomodulation therapy, led to amelioration of symptoms and near complete cognitive recovery in a period of 2.5 weeks. Her cognitive status based on objective measure with the Montreal Cognitive Assessment (MoCA) was initially in the moderate to severely impaired range. With comprehensive treatment, Ms Zorn’s cognitive status improved to a level of only mild impairment. Correspondingly, her decisional capacity improved from significantly impaired to largely intact over the same period. Psychiatrists assessing and treating patients with atypical presentation of psychotic illness should actively consider a diagnosis of ANMDARE as explanatory. In female patients with suspicion of ANMDARE, pelvic ultrasonography and/or CT scan should be ordered to rule out commonly co-occurring ovarian teratoma. Patients with ANMDARE who are treated with high-dose intravenous corticosteroids should be monitored (and treated for) any corticosteroid-associated psychiatric adverse effects, which can overlap with the atypical psychotic symptoms at illness onset. Empiric treatment with psychopharmacology for psychotic and/or depressive symptoms should be pursued, although such medications may not be necessary indefinitely once definitive immunomodulation therapy is completed.

ANMDARE should be on the differential for atypical psychosis, with an expectation of a thorough search for laboratory and systemic clinical (especially neurologic) findings, empiric treatment of psychiatric symptoms, and reassessment of the patient if immunomodulation therapy is completed. Prospective study of ANMDARE illness cohorts are needed to quantify recurrence risk, need for ongoing intervention, and ultimate prognosis.

Consultation-liaison psychiatrists working in academic medical centers may be in a position to assist multispecialty teams in the identification, diagnosis, management, and ongoing follow-up of these patients. As with any illness that affects cognitive function, attention to and serial assessment of decisional capacity is a critical part of C-L psychiatry care, especially as pertains to patient consent for major immunomodulation therapies.

**Dr Bourgeois** is Chair, Department of Psychiatry, Baylor Scott & White Health, Central Texas Division, and clinical professor for medical education, Texas A&M University Health Science Center, Temple, TX. Mr Li is a medical student, Texas A&M University Health Science Center, Temple, TX. The authors report no conflicts of interest concerning the subject matter of this article.

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**Table. Progression of Symptoms and Care**

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<td>1-g methylprednisolone</td>
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<td>9</td>
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<td>16</td>
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<td>400-mg quetiapine PM 15-mg mirtazapine PM</td>
<td>Plasmapheresis (7 treatments; 1 every even numbered hospital day [10-22])</td>
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<tr>
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<td>Plasmapheresis</td>
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<tr>
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<tr>
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<tr>
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<td>14</td>
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<td>Yes</td>
<td>7.5-mg mirtazapine PM</td>
<td>Plasmapheresis</td>
</tr>
</tbody>
</table>

MoCA, Montreal Cognitive Assessment; DC, decisional capacity; VH, visual hallucinations.
Delirium, Encephalopathy, and COVID-19: An Update From the Field

Elliot B. Martin Jr, MD

“ ‘You guys must be twiddling your thumbs these days, huh?’ This was the ill-informed question posed to me by one of our outpatient primary care doctors moonlighting as a hospitalist in this “all-hands-on-deck” time of ours. I looked at him and only laughed.

During the early stages of COVID-19 in my state of Massachusetts, we experienced frequent emergency department referrals of brief psychosis related to severe anxiety in the context of what was then COVID-19 anticipation. Now that we are in a full-blown surge, the 2 most striking pathologies we are seeing on the medical side are a much greater volume than usual of refractory delirium, both in COVID-19 and patients without-COVID-19, punctuated by um, both in COVID-19 and patients uume than usual of refractory delirium.

The biggest challenge we have faced thus far has been the requests for assistance in managing persistent delirium, which have increased about three-fold and continue to rise. The requests have been split roughly in half between patients with COVID-19 and those without COVID-19, mirroring where we are at this point with regard to inpatient populations, with actually only about a quarter having been intubated at some point in the ICU. Almost all the patients are older than 75, and about three-quarters are male. About three-quarters have pre-existing cognitive decline. About a quarter are non-native English speakers. Many have required significantly higher doses of antipsychotic medications than are typically prescribed. Most have required augmenting agents such as antiepilepsy drugs, benzodiazepines, barbiturates, dexmedetomidine, opiates, and/or ketamine.

The question, of course, is why are these cases so refractory? Never in my experience have we had to resort to such trial and error, and really for the safety of the patients and staff, as many patients have been unwittingly violent.

We have collected the following contributors to this phenomenon:

1. The hospital has not been allowing any visitors. There have been no family or other social supports at the bedside on a consistent basis to orient and reorient patients appropriately.

2. Staff are required to wear masks at all times. On the medical and surgical floors, most are wearing much more, including all manner of goggles, face shields, and surgical caps. In other words, every person these patients see looks like something out of a science fiction movie. There are no faces to read and no way to gain a sense of the situation.

3. There is no touch. I never noticed how much I touch older patients until now. We all catch ourselves as we instinctively reach to touch a shoulder, to hold a hand, to press someone back down gently into bed. None of this happens any more.

4. The lack of technological experience in the older population has made communication between patients and their spouses, siblings, and peers difficult. Many family members with whom we have spoken do not have smartphones or computers. This has also made applications for Medicaid and other assistance exceedingly slow and difficult to arrange.

5. Bottlenecking on discharge continues to be a problem. With the COVID-19 crisis, there have been very few available skilled nursing facilities and long-term care facilities for patients. This also results in much longer hospital stays.

6. Persistent encephalopathy, although much less common than the delirium, has been more difficult to explain and thus far unresponsive to all attempted treatments. The 5 cases we have seen were aptly described by an ICU team as “different than the typical different we have been seeing.” (This piece explores the 2 earliest cases, but all cases have been similar.) In the 2 earliest cases, the patients were admitted for COVID-19 infection with subsequent complications. One patient was female in her late 50s, and the other was male in his early 60s. Both patients had significant psychiatric histories with previous psychotic symptoms and had been on longstanding chronic antipsychotic medications. The patients were both intubated during their respective hospital courses—3 days for our female patient and 3 weeks for our male patient—and both required tracheostomy due to complications. Both had type 2 diabetes, hypertension, and chronic obstructive pulmonary disease, and both were smokers. They both had been living in skilled nursing facilities and had previous strokes. The patients had remote substance use history, having used alcohol and cannabis. At baseline, prior to their hospitalizations, both had been fully cognizant and conversant as well as fully ambulatory with only minimal motor deficits.

The differences in their behavior were noted soon after extubation. As their pulmonary function improved and organs generally recovered, they remained mentally altered, mostly staring off and non-interactive. The patients appeared conscious and awake, but they would not eat or drink on their own or would not respond to any commands. Their pu-
The list of prohibited donors is long and detailed, so for the purposes of this discussion, some key details will be highlighted.

Who can make political contributions?

Individuals and certain specific business entities, including some LLCs and partnerships depending on how those entities are taxed. (But not your “professional corporation”; details follow.) Individuals can contribute to candidates, parties, and a variety of political organizations in different amounts. Both minors and adults can give, but you are not allowed to give your minor child money specifically to make contributions that you are directing. It needs to be their own money and their choice.

What is a contribution?

A contribution can take many forms:

- Direct monetary contributions
- In-kind contributions of goods and services offered free or below market rate; or
- Funds obtained through sales, fundraisers and purchases of political items or event tickets.

Who can’t make contributions?

The list of prohibited donors is long and detailed, so for the purposes of this discussion, some key details will be highlighted.

Campaigns are prohibited from accepting contributions from certain types of organizations and individuals:

- Your “professional corporation.” Although law firms, doctors’ practices, and similar businesses are often organized as partnerships, some of these businesses may instead be professional corporations. Unlike a partnership, a professional corporation is prohibited from making any contributions because contributions from corporations are unlawful.
- Corporations, including nonprofit corporations. However, contributions from a separate segregated fund are permissible.
- Labor organizations. Yet, funds from a separate segregated fund are permissible.
- Federal government contractors.
- Foreign nationals. There is an exception for green-card holders as permanent resident aliens.
- Contributions in the name of another.

Can I donate anonymously?

Probably not, at least not directly to a specific candidate. An anonymous contribution of cash is limited to $50, per the Federal Elections Commission (FEC). Any amount in excess of $50 must be promptly disposed of and may be used for any lawful purpose unrelated to any federal election, campaign, or candidacy.

Federal law requires disclosure of most significant contributions over $200. This includes donors’ names, addresses, occupations, and employer information, which then become public record freely available and tracked by the FEC. Many organizations report all contributions to stay on the safe side of disclosure laws.

Federal law requires that all political action committees, political parties, and federal candidates disclose any and all contributions received that are more than $200. This information then becomes public record.

How much can I contribute?

Many are surprised that individuals are most limited in giving money to a specific candidate directly ($2,800), but they can make very large contributions ($100,000 or more) to a national party committee account. They also can give away millions through other methods. The FEC has a very detailed chart that breaks down contribution limits based on the type of donor and the type of recipient. Cash (actual currency) contribution limits are most specifically limited by the FEC. A campaign may not accept more than $100 in cash from a particular source with respect to any campaign for nomination for election or for election to federal office.

Dr. Devji has practiced law exclusively in the areas of asset protection, risk management, and wealth preservation for the last 16 years.}

REFERENCES


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Psychiatry’s Intellectual Crisis
Giovanni Fava, MD

FAVA: I agree; psychiatry is going through an intellectual crisis. This crisis is shared by other areas of clinical medicine and stems from a narrow concept of science that neglects clinical practice as a source of fundamental research questions. Fewer and fewer academic psychiatrists actually assess and treat patients. Most of published research has no relevance to practice. The progress of neuroscience in the past 2 decades has often led to the belief that clinical problems in psychiatry were likely to be solved by this approach. Such hopes are understandable in terms of massive propaganda operated by biotechnology and pharmaceutical corporations. An increasing number of psychiatrists are wondering, however, why the cures and clinical insights promised by neurosciences have not come to fruition. Biological reductionism has resulted in an idealistic framework into psychiatry much more than serving as an investigative tool.

FAVA: Like Molière’s bourgeois who was surprised to speak in prose, the clinician may discover they are constantly faced with iatrogenic comorbidity. In pharmacological terms, let us think of selective serotonin reuptake inhibitors (SSRIs) as a prime example. One patient who has been unsuccessfully in analysis for many years and is offered a course of cognitive behavioral therapy (CBT); their response is likely to be affected by the previous psychotherapeutic experience in terms of expectation, adherence, and motivation. The concept of iatrogenic comorbidity is simply an attempt to conceptualize the problem in a practical way. Regrettably, psychiatrists, unlike other specialists, have been taught to consider comorbidity only in terms of diagnoses, and not as problems and treatment experiences. And they are unable to think “iatrogenic” in interpreting clinical problems, simply because they have not been exposed to the concept, which has been submitted to tight censorship by mainstream psychiatry.

FAVA: The term discontinuation syndrome applied to antidepressants, versus withdrawal syndromes with benzodiazepines, was a very smart method of the pharmaceutical industry to deny the problem. It is sad that most academic psychiatrists followed these leads. The evidence, based on systematic reviews and a large body of literature, is now pretty clear and the tide is turning. Selective serotonin reuptake inhibitors and serotonin and norepinephrine reuptake inhibitors may cause withdrawal reactions (ie, new symptoms that were not present before) despite slow tapering; these reactions may be severe and do not necessarily subside in a few weeks. When I see a patient whose symptoms have been treated with paroxetine or venlafaxine, I am always looking for manifestations of iatrogenic comorbidity, which, unfortunately, I am very likely to find.

This crisis is shared by other areas of clinical medicine and stems from a narrow concept of science that neglects clinical practice as a source of fundamental research questions.

AFTAB: I feel psychiatrists are in a very tough spot. The way they are educated and trained, they are unaware of the systematic ways in which research studies and guidelines are heavily biased and misrepresented. They have to learn to critically appraise research literature for guidance, which is a hard thing to do for clinicians without research training, especially since it often means challenging the wisdom of their colleagues, conclusions of top researchers, and “key opinion leaders,” as well as established guidelines in the field. To make matters worse, there is very little unbiased and generalizable research. This means psychiatrists have to rely on their clinical experience when working with individual patients, which is subject to biases and distortions of its own. Despite mountains of research data, it feels like we are in an impoverished state of knowledge. Am I being too nihilistic, or do you share this view?

FAVA: Like Molière’s bourgeois who was surprised to speak in prose, the clinician may discover they are constantly faced with iatrogenic comorbidity. In pharmacological terms, let us just think of the case of switch in to mania of a patient with allegedly unipolar depression treated with antidepressant drugs. You are faced with a modification of illness that is largely drug-induced and that is likely to affect treatment. Or, in psychotherapeutic terms, let us think of psychiatrists better differentiate between withdrawal and illness relapse, and how can maintenance studies be better designed to parse out this issue?

FAVA: Psychopathology, the basic neglected method of psychiatry, allows differentiation of withdrawal syndromes from relapse. Specific diagnostic criteria have been developed by Guy Chouinard, MD, and Virginie-Ann Chouinard, MD, in 2015. These criteria allow differentiation of withdrawal syndromes from relapse, recurrence, rebound, and persistent post-withdrawal disorders. Maintenance studies with antidepressants are likely to confound withdrawal and relapse unless specific criteria are used. We need new studies with these criteria.

AFTAB: It has been interesting that the psychiatric establishment insists on using the term antidepressant discontinuation syndrome, while researchers such as yourself and critics in the general public favor antidepressant withdrawal. Does this battle of terminology have any larger significance?
SNRI discontinuation. I do not see anything similar happening in the United States.

AFTAB: Many clinicians seem to hold the view, implicitly or explicitly, that it is better to identify and treat depression when it is mild or even subthreshold because untreated depression will progress in severity. This is coupled with the assumption that at worst, the medication may not help but it certainly won’t cause disease progression. Do such clinicians have an unfairly malignant view of the natural history of untreated depression and an unfairly benign view of psychopharmacological treatment?

FAVA: These clinicians perceive a state of distress in their patients but have been taught only to think in terms of harmless medications. They are often unaware of the major advances that have been made in psychotherapy in the past decades, which are far superior to the pharmacological ones. Mild or subthreshold depression should be primarily addressed with effective and short-term psychotherapies, such as CBT. Unfortunately, the prescribing clinician is driven by an overestimated consideration of potential benefits, paying little attention to the likelihood of spontaneous remission and to potential vulnerabilities in relations to the adverse effects of treatment, one of the spectacular achievements of “evidence-based medicine,” which has been transformed into the marketing arm of the pharmaceutical industry. These latter components outweigh potential benefits of antidepressants in mild depression.

AFTAB: You seem to interpret the results of STAR*D as indicating that the use of switching and/or augmenting strategies propels depressive illness into a phase characterized by low remission, high relapse, and high intolerance to medications. You tie the poor outcomes seen in STAR*D to the use of psychopharmacological strategies while conventionally the poor outcomes have been viewed as a result of patient characteristics or characteristics of disorder pathophysiology. Is there anything in the data that supports one interpretation over another?

FAVA: The findings of STAR*D were pretty clear. The aim of the trial was to apply the best pharmacological strategies for obtaining remission in major depression. Those who did not recover after a trial with citalopram were submitted to 4 sequential steps involving switching, augmentation, and combination strategies, based on available literature.

The results were rather disappointing. The cumulative rate of remission after 4 sequential steps was 67%. However, when sustained recovery (taking into account relapse rates while on treatment) was considered, the cumulative rate was 43%. This means the strenuous efforts after step 1 (open treatment with citalopram) yielded an additional 6% of sustained recovery. Remission rates decreased after each treatment step. Rates of relapse increased after each treatment step in patients who achieved remission. Further, intolerance (dropouts for any reason during the first four weeks, or adverse effects afterwards) increased after each treatment step.

STAR*D’s message should have been clear: Do not do what we have done. Regrettably, just the opposite came out. The findings can be interpreted in light of the oppositional model of tolerance. Continued drug treatment may recruit processes that oppose the initial acute effects of a drug. This may explain loss of treatment efficacy and the fact that some side effects (such as increased appetite and weight gain) tend to ensue only after a certain time. These processes may also propel the illness to a more malignant and treatment-unresponsive course, as with a bipolar manifestation or paradoxical reactions. When drug treatment ends, oppositional processes may encounter no more resistance, resulting in appearance of new withdrawal symptoms, rebound symptomatology, persistent post-withdrawal disorders, hypomania, resistance to treatment if it is reinstalled.

In the long run, antidepressants may increase chronicity, vulnerability to depressive disorders, and comorbidity. There is now extensive literature supporting this mechanism. In practice, you may encounter patients whose symptoms have been treated with a number of psychotropic drugs, particularly in combination, and who end up as in the STAR*D. What to do is a big problem. You can blame the illness and the patient, it is easy, but you should also think iatrogenic.

AFTAB: You wrote, “Current diagnostic methods in psychiatry, both DSM-5 and the forthcoming ICD-11, refer to patients who are drug-free and do not take the issue of iatrogenic comorbidity into adequate consideration. They are suited for a patient who no longer exists.” How do you think the diagnostic manuals can take iatrogenic comorbidity into account?

FAVA: Most of the patients we see in practice are already taking psychotropic drugs. If a primary care physician refers a patient to me, it is because the patient’s symptoms did not respond to the pharmacological treatment (generally an SSRI or SNRI) that had been started. Medications may affect clinical presentation of symptoms. In a naïve implicit formulation that runs counter to any modern insights into the plasticity of the brain, we may believe that after discontinuing an antidepressant everything goes back to the pre-treatment state. It does not, as any physician in the real world knows.

Treatment is a one-way street. We even subscribe to the hope of finding pre-treatment predictors (biomarkers), and we waste money on it. Everything changes in the course of treatment. This means we need to supplement DSM with a more comprehensive assessment that takes the issues of medication history and development of tolerance into consideration, using methods that pertain to clinimetrics, the science of clinical measurements, such as macroanalysis and staging.

AFTAB: It is very common for psychiatrists to encounter patients with complex presentations of multiple psychiatric comorbidities who are on multiple psychotropic medications but remain symptomatic with a mix of symptoms that does not fit into diagnostic categories and their psychopharmacological treatment history is a mess of one trial after another, consisting of medications that once worked but no more, medications that never worked, and medications poorly tolerated. Do you have any general clinical recommendations regarding how to approach evaluation and treatment in such cases?

FAVA: These are exactly the patients who do not fit into any DSM category. You need to take into account the iatrogenic comorbidity into account and spend a good deal of time for a full clinimetric assessment and use diagnoses only as temporary transit stations that can be verified in the course of time. There is almost no research on these issues, as it happens with most of the truly important matters in psychiatry. I can share with you what I do clinically. First, I use clinimetric assessment to capture clinically relevant information that is not included in the diagnosis. Based on the assessment, I try to taper and discontinue, very slowly, the multiple medications starting from those I judge most responsible for the patient’s condition.

More Conversations in Critical Psychiatry

**Reading Linehan in the Age of Critical Psychiatry**

To read Marsha M. Linehan’s memoir is to realize that she underwent a crucifixion and resurrection of her own, and that the Pontius Pilate in her story is psychiatry itself. [https://www.psychiatrictimes.com/view/reading-linehan-age-critical-psychiatry](https://www.psychiatrictimes.com/view/reading-linehan-age-critical-psychiatry)

**Chaos Theory With a Human Face: Niall McLaren, MBBS, FRANZCP**


**Social Constructionism Meets Aging and Dementia**

As less important. I am also looking for counter-therapeutic elements (behavioral components) and attitudes that may affect response to treatment at the individual, family, and work levels using a framework that Karl Rickels, MD, developed in the ‘60s and that we have recently updated. I also pay a lot of attention to psychological well-being (ie, is there anything I can build on?). Then I introduce the type of psychotherapy (eg, CBT, brief dynamic psychotherapy, family therapy, well-being therapy) that may affect the targets I have identified. It is simply wishful thinking to believe that one course of treatment may entitle the solution to the problems that we encounter in these complex cases. We need the sequential combination of different treatments, and this should be planned from the very beginning and subsequently modified as needed.

AFTAB: What percentage of patients prescribed antidepressants would you say experience clinically significant effects related to oppositional tolerance? Why is there so much variability in how individuals are impacted by these medications?

FAVA: It very much depends on the type of practice. In my practice, which is very much shifted to treatment-resistant, difficult, unusual cases, the percentage of cases who present with the various manifestations of tolerance (eg, loss of clinical effects, paradoxical reactions, switching, persistent post-withdrawal disorders, refractoriness) is very high and may approach 1 case out of 2. But you do not have the same crowd, the percentage may be much lower. We need studies addressing these issues in a comprehensive manner in outpatient populations.

AFTAB: Psychiatry is continuing its path of aggressive pharmacological treatment of “treatment resistance” in depression, and medications such as ketamine are being hailed as new miracle drugs. What is your perspective on these developments?

FAVA: These types of studies reveal the methodological frailty and the narrow perspectives of much of current psychiatry. You test a drug in a highly heterogeneous sample of patients who do not respond to a certain treatment (the reasons for which could be so different) without any discrimination in terms of tolerance and questioning whether the treatment was appropriate (you just go by the book). Because you use a controlled design, you are convinced you are performing high-level clinical science. But the results are likely to be spurious and the outcome is monitored for a very short time.

For a clinician, a short-lived response does not mean much. It can also be achieved with non-specific ingredients, such as high expectations. One course of treatment, again, is unlikely to yield solutions to the complex situations of these patients. The claim of “miracle drugs” such as ketamine are only a sign of the intellectual crisis of psychiatry. I would like to see adequate assessments and articulated courses of treatment be tested for enduring results.

AFTAB: Have you read or are familiar with Robert Whittaker’s book Anatomy of an Epidemic? If so, do you have any thoughts on what the book gets right and what it gets wrong?

FAVA: Yes, of course. I think it was an excellent piece of investigative journalism that was highly effective in fighting the tight censorship that mainstream psychiatry endorsed against any vulnerabilities induced by psychotropic drugs. Subsequent developments (eg, Mad in America) had the recognized merit of addressing clinical problems, such as the withdrawal syndromes from SSRI and SNRI, that were and are still denied by professional organizations and scientific societies, as well as issues related to financial conflicts of interest.

However, the risk is forgetting that, if used properly, antidepressants, antipsychotics, and lithium are life-saving drugs, and benzodiazepines offer a rapid and safe relief for anxiety. The problem is their inappropriate application, like any other medication (eg, antibiotics).

AFTAB: As you have discussed, forces and incentives in the world of academic publishing converge to reward mediocrity and discourage truly innovative research. What would be your advice to early career psychiatrists about surviving in academia without giving up independent thinking and creative integrity?

FAVA: A young psychiatrist who reads our conversation may become convinced that this is the worst of times for psychiatry. As Dickens teaches us, however, it may also be the best of times. There are entire areas of clinical research, such as those related to the iatrogenic effects of medications and psychotherapy, that are largely unexplored. Neuroscientific methods applied to specific problems (eg, what characterizes withdrawal syndromes) may offer unprecedented opportunities but should be associated with clinimetrical opinion, we may be close to a change of paradigms in psychiatry. Either you persist on the current mainstream road to nowhere that leads to frustrating clinical experiences or realize that there is a world of new possibilities and pathways.

AFTAB: Thank you!

Dr Aftab is a psychiatrist in Cleveland, Ohio, and clinical assistant professor of psychiatry at Case Western Reserve University. He is a member of the executive council of Association for the Advancement of Philosophy and Psychiatry and has been actively involved in initiatives to educate psychiatrists and trainees on the intersection of philosophy and psychiatry. He is also a member of the Psychiatric Times Advisory Board. He can be reached at awaisaftab@gmail.com or on Twitter @awaisaftab. Dr Fava, MD, completed his medical education as well as his psychiatry residency training at the University of Padova, Italy. After working for several years in the United States, he returned to Italy in 1988, where he established an Affective Disorders Program at the University of Bologna. He is currently clinical professor of psychiatry at the State University of New York (SUNY) at Buffalo, NY. A full version of this article can be found online at www.psychiatric times.com.

Dr Aftab and Dr Fava have no relevant financial disclosures or conflicts of interest.

The opinions expressed in the interviews are those of the participants and do not necessarily reflect the opinions of Psychiatric Times.

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Assessing Malingered Voice-Hearing

Joseph M. Pierre, MD

“I have no lid upon my head, but if I did
You could look inside and see what’s on my mind.”

— Let You Down, Dave Matthews Band

The benefit of the doubt?

“Mr. Arnold,” a 50-year-old Army veteran with a
history of chronic depression and alcohol use dis-
order (in remission) was admitted to the hospital after complaining of “voices telling me to bayonet my wife… or someone else.” His outpatient psy-
chiatrist, who had been treating him with mirtazapine
45 mg/day and quetiapine 300 mg/day over the previous year, documented new onset command hallucinations and admitted the patient involun-
tarily based on homicidal ideation.

On subsequent exam with the inpatient team, Mr. Arnold did not appear acutely depressed. His chief complaint was that he was hearing the voices of 3 military personnel—including a lieutenant, sergeant, and corporal”— who were all telling him to “kill people.” He then stated that he had “struck a deal with the corporal” such that he would not have to
kill anyone if he killed himself, Mr. Arnold, there-
fore, planned to do so by refusing all food other
than milk and yogurt.

If the infamous social experiment
by the late psychologist David Ros-
shen, PhD, published in 1973 with the title “On Being Sane in Insane Places” taught us anything, it was that psychiatrists working in clinical settings may not always be the best at detecting malingered voice-hearing. Although a recent book has claimed that Rosenhan misrepresented what actually hap-
peneed when he and 7 others were
hospitalized based on false reports of hearing a voice saying words like “empty,” “hollow,” and “thud,” the take-home message of his experiment remains the same.

When help-seeking individuals present with fabric-
cated claims about hearing voices, psychiatrists
will often respond in kind, offering antipsychotic therapy based on a provisional diagnosis of psychosis.

Without the ability to look inside
the minds of patients who en-
dose hearing voices, and in the
absence of any established biolog-
ical marker or reliable neuroimag-
ing blueprint for auditory verbal hallucinations, clinicians often re-
sort to a benefit-of-the-doubt ap-
proach that assumes a help-seeking
patient is ill. However, colluding with false claims of psy-
chosis and disability and un neces-
sary exposure to antipsychotic side
effects is not beneficial to patients.

In order to distinguish between
real and feigned psychotic symp-
toms, a generation of psychiatrists has followed the guidelines of for-
rensic psychiatrist Philip Resnick,
MD, who first published specific
criteria more than 20 years ago to alert clinicians
of suspicion of malingered auditory verbal hali-
Table 1. Inquire About These Details When Assessing Malingered Auditory Verbal Hallucinations

<table>
<thead>
<tr>
<th>VOICE IDENTITY</th>
<th>Number of voices</th>
<th>Age</th>
<th>Gender</th>
<th>Familiarity</th>
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<tbody>
<tr>
<td>VOCAL CHARACTERISTICS</td>
<td>Volume</td>
<td>Localization</td>
<td>clarity</td>
<td>Second- or third-person speech</td>
</tr>
<tr>
<td>INTERACTION</td>
<td>Commands</td>
<td>Talking back to voices</td>
<td>Compliance with commands</td>
<td></td>
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<tr>
<td>INSIGHT</td>
<td>Associated delusional thinking</td>
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Table 2. Differences Between Malingered Voice-Hearing in Forensic Versus Clinical Settings

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<th>Forensic</th>
<th>Clinical</th>
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<tbody>
<tr>
<td>Presentation</td>
<td>Calls attention to voices initially, but vague, evasive, and inconsistent with detailed questioning</td>
<td>Calls attention to voices initially, but vague, evasive, and inconsistent with detailed questioning</td>
</tr>
<tr>
<td>Secondary gain</td>
<td>Exculpation from criminal charges</td>
<td>Obtaining services</td>
</tr>
<tr>
<td>Voice characteristics</td>
<td>“Atypical” and cartoonishly exaggerated</td>
<td>Overlapping with drug-induced symptoms or non-hallucinatory experiences such as depressive ruminations</td>
</tr>
<tr>
<td>Voice content</td>
<td>Commanding violence or other criminal behavior</td>
<td>Commanding suicide (less commonly with violence towards others)</td>
</tr>
<tr>
<td>Compliance</td>
<td>Claimed due to malingered delusions</td>
<td>Claimed due to mood-congruence</td>
</tr>
<tr>
<td>Clinical course</td>
<td>Persistent while still in a forensic setting</td>
<td>Voices and contingent claims of suicide resolve once services are obtained</td>
</tr>
</tbody>
</table>

“Finding the pony”
 Attempts to complete neuropsychological testing were initially met with resistance, including threats to kill members of the treatment team. (“The deal with the corporal is off… I’m going to take everyone out!”) However, after Mr Arnold was told that he would be discharged in response to these threats, he retracted them, became apologetic, and agreed to complete testing. He scored well above the cut-off scores for malingering on the SIMS, M-FAST, TOMM, and VSTV based on gross over-endorsement of unusual symptoms.

Without directly confronting Mr Arnold about his test results, a subsequent interview was held to ascertain potential motives for secondary gain. In doing so, the team learned that he made several unsuccessful attempts to obtain service-connected disability and had been recently told that his case was closed. The team responded empathically and guided him to resources about applying for Social Security Disability Insurance for legitimate medical complaints. He soon stopped endorsing psychotic symptoms and homicidal self-injury and self-discharged the next day.

Clinicians should be mindful that confrontations about suspected or detected malingering may result in patients upping the ante with threats of violence or self-injury. It is therefore recommended that clinicians allow patients to save face when providing diagnostic feedback instead of accusing them of lying. Clinicians should also strive to “find the pony” by assessing patients in a thorough but non-confrontational manner with a goal of empathic and compassionate understanding of legitimate sources of personal distress.

Uncovering the secondary gain motives of malingering allow clinicians to identify the biopsychosocial origins of distress and offer interventions in kind, including psychotherapy, housing, disability income support, and treatment of substance use disorders. This holistic approach can be useful in both diagnosing and treating malingering through disincentivization. Non-antipsychotic pharmacotherapy targeting symptoms of insomnia, anxiety, and substance withdrawal can also help resolve co-occurring claims of hearing voices.19

Countertransference and treatment success
 After discharge, Mr Arnold made no further mention of hearing voices to his outpatient psychiatrist, who had been given a “warm hand-off” by the inpatient team. However, after transferring care to a new psychiatrist the next year, Mr Arnold renewed complaints about hearing voices along with novel endorsement of posttraumatic stress disorder (PTSD) symptoms that he attributed to having a heavy object fall on him during military service. He was prescribed chlorpromazine, sertraline, and prazosin based on a diagnosis of atypical psychosis and PTSD.

After a few more years in treatment, Mr Arnold’s psychiatrist revised his diagnosis to reflect malingering and discontinued antipsychotic medication in favor of antidepressant monotherapy. However, Mr Arnold eventually decided that he no longer wanted to take any psychiatric medication and dropped out of psychiatric care. Over the next 5 years—12 years after his original hospitalization—he did well without psychiatric treatment and did not endorse psychiatric symptoms during primary care visits.

Successfully managing malingering requires clinicians to maintain awareness of countertransference issues. Physicians are not generally taught in medical school to consider malingering within a differential diagnosis or how to withhold requests for diagnoses and interventions that are requested but are harmful or not indicated.20 Psychiatrists may, therefore, find it difficult to diagnose malingering and are often more concerned about missing real mental illness (eg, false negatives) than the problem of inappropriate diagnosis (eg, false positives).4 Longitudinal assessment can often help resolve diagnostic ambivalence.

When a diagnosis of malingering is ultimately made, psychiatrists and other mental health workers must manage their reactions to the fact that the patient lied to them. This is best accomplished by understanding the ubiquity of lying as a normal and adaptive human behavior—the average person lies once or twice a day—that is further incentivized by the limited resources for help available to people in distress due to difficult life circumstances. Like any clinical diagnosis, a diagnosis of malingering should ideally guide appropriate interventions, not steer patients away from mental health care altogether. Over time, however, resolution of malingering’s “symptoms” and the motivations that underlie them should be considered a treatment success.

### Table 3. Neuropsychological Tests That Assess Malingering

<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Structured Interview for Reported Symptoms (SIRS)</td>
<td></td>
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<tr>
<td>Structured Inventory of Malingered Symptomatology (SIMS)</td>
<td></td>
</tr>
<tr>
<td>Miller Forensic Assessment of Symptoms Test (M-FAST)</td>
<td></td>
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<tr>
<td>Test of Memory Malingering (TOMM)</td>
<td></td>
</tr>
<tr>
<td>Victoria Symptom Validity Test (VSTV)</td>
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</tbody>
</table>

Concluding thoughts
 Auditory verbal hallucinations are a common claim when people malinger psychiatric symptoms since they suggest the presence of a severe mental illness and are largely unverifiable beyond clinical judgment. Careful clinical interviewing, identification of secondary gains, and supplemental psychometric testing provide the best approach to assessment. Once a diagnosis of malingering is made, clinical management that recognizes iatrogenic causes and disincentivizes misrepresentation by addressing legitimate sources of distress can be therapeutic and can help to confirm the diagnosis.

### References
Pain Management Medical Director
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